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ABOUT THIS SPECIAL ISSUE

Oral and systemic health are closely related to each other. oral diseases are potentially associated with different general health conditions. Thereby, an influence of oral conditions on systemic health or vice versa as well as different bidirectional relationships have been uncovered. Moreover, medications can show distinct side effects in the oral cavity, such as xerostomia or gingival overgrowth, or affect the patient's immune system as well as bone metabolism. oral diseases can affect the initiation and progress of various systemic diseases such as cardiovascular, neurological, and respiratory diseases; on the other hand, systemic diseases can increase the susceptibility of suffering from oral diseases. Both oral and systemic diseases share several common risk factors, which contribute to the incidence of both diseases, for example, aging, smoking, alcohol abuse, gender, education and socioeconomic status, and genetic susceptibility.

This Special Issue will focus on these different aspects of oral conditions, dental care, and quality of life in the context of the relationship between oral and systemic health.

SP-1

Acceptance and Hesitancy of Covid -19 Vaccine by I-Mbbs Studentsin Chennai

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Abstract: On January 2021, in India launching of COVID-19 vaccine was done. Priority was given to front-line health care providers that include medical students also, who are likely to be exposed to COVID-19 patients. In this pandemic situation, it is mandatory to increase the vaccine coverage rate among medical students, as they are the future physicians having role in educating the public regarding vaccine efficacy and providing vaccine recommendations. This project is done by using online questionnaire to evaluate acceptance and hesitancy of COVID -19 vaccine by I-MBBS students in Chennai. Majority of the student group have bright outlook onCOVID-19 vaccine and acceded that chance of them getting exposed toCOVID-19 disease is more. Vaccine hesitancy is found among n=32(16%) of participants. Those students who are willing to take the vaccine trust the public health experts and they agree with the vaccine mandates. (P<0.05).The students who showed hesitancy on COVID-19 vaccine has received standard information regarding vaccine from media platform and website source. Hesitancy towards vaccine by MBBS students can be reduced by conduction of awareness campaigns and release of safety and efficacy data about COVID-19 vaccine in scientific journals and including the same in the educational curriculum.

Keywords; Vaccine hesitancy and acceptance, COVID-19 vaccine, students studying medicine.

INTRODUCTION

One of the world-wide ailments is COVID-19, resulted in a greater number of illnesses and death. From January 2021. Vaccines for COVID -19 diseases released, as apart of control measure to the disease spread. As vaccines are effective and protects us from severe illness and death. Mass vaccination have been emerged as a preventive strategy. Covaxin and Covishield are two vaccines manufactured in India.[1] Through CO-WIN portal online registration for vaccination is done. Medical professionals and also the students studying medicine are frequently taken as the study groups to evaluate the vaccine hesitancy.[2] Due to the heave in COVID-19 ailment, it is important to do a study about hesitancy towards COVID-19 vaccine among the student group studying medicine. The present study aim is to evaluate acceptance and hesitancy of COVID -19 vaccines by I-MBBS students in Chennai.

MATERIALS AND METHODS

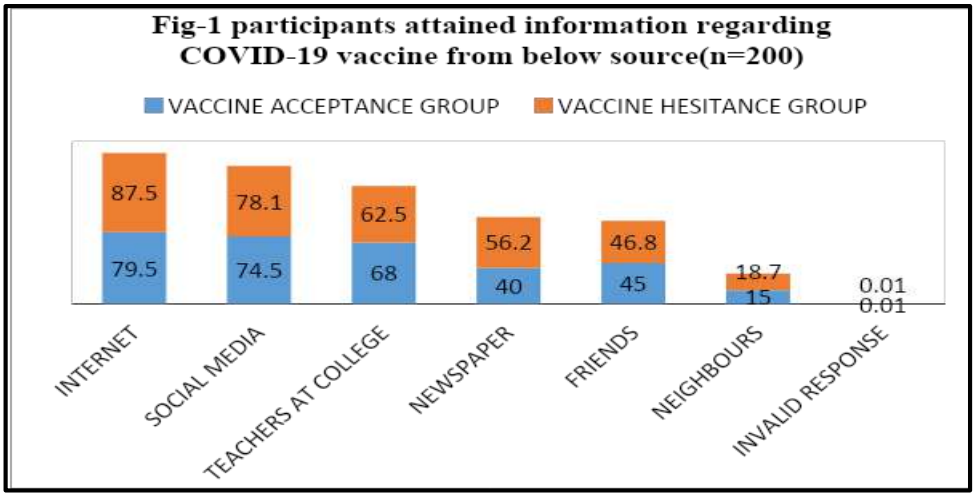
An online structured questionnaire based study for the students studying medicine in Chennai for duration of one month. Sample size was calculated using the previous study with 95% CI and P<0.05. Sample size = 200. A structured questionnaire is done using previous study evidence on hesitancy toCOVID-19 vaccine by students studying medicine.[3,4] Study questions was developed to get their basic details, general attitudes to vaccine, general opinions on COVID-19 vaccine, their personal views on COVID-19 and vaccine, personal vaccination behavior etc., Questionnaire is circulated through WhatsApp groups which included students in I MBBS. Ethical permission for doing this study was obtained from sree balaji medical college and hospital, Chennai – Institutional Ethical Committee.

STATISTICAL ANALYSIS

On completion of data collection, data analysis was done using the online software (SPSS V 23.0). Categorical variables were set out and P- value calculated for each. P< 0.05 is taken as significant range.

RESULT AND DISCUSSION

Data collected from the student groups=200 (response rate=100%). female participants=55%. Majority of the students accepted with the statement 'for COVID -19 disease, vaccine is needed to reduce the disease spread'. For the question "I am willing to participate in vaccine trial" 10(31.2%) responded "no" and 11(34.3%) responded "none of the above". 32(16%) students were hesitant towards COVID -19 vaccine. In students who responded YES, 80(57.5%) was already vaccinated and 59(42.4%) were not vaccinated at the time of data collection. In table-1results are set out based on whether the students have responded yes or no to receive the vaccine. Students of both the acceptance and hesitance group got the standard information about COVID-19 vaccine from Internet, social media, peer groups, neighbors and teachers at medical colleges (figure-1)



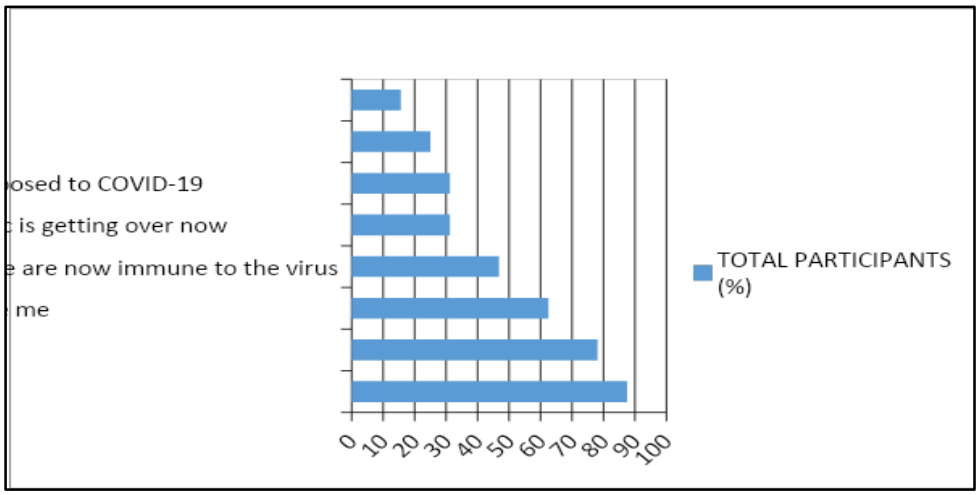


Fig-2Reasons why students studying medicine showed hesitance towards COVID-19 Vaccine(n=32).

TABLE-1 Acceptance and hesistance towards COVID-19 VACCINE-Survey response n=200

SURVEY QUESTIONS	APPROVING RESPONSE OF PARTICIPANTS (YES/NO)			
	ALL RESPONDENTS (N=200)n(%)	COVID-19 VACCINE ACCEPTANCE GROUP (n=168) n(%)	COVID-19 VACCINE HESITANT GROUP (n=32) n(%)	P VALUE
PERSPECTIVE TOWARDS COVID-19 VACCINE				
TO STAY HEALTHY AS A FUTURE PHYSICIAN, VACCINES ARE MANDATORY	196(98)	165(98.2)	31(96.8)	0.6199
IT IS EACH MEDICAL PROFESSIONAL ROLE TO LEARN ABOUT VACCINES	196(98)	165(98.2)	31(96.8)	0.6199
GENERAL VIEWS TOWARDS COVID -19 VACCINE				
TO REDUCE THE COVID-19 DISEASE SPREAD, VACCINE IS NEEDED	193(96.5)	162(96.4)	31(96.8)	0.8997
FOR OVERALL PUBLIC HEALTH COVID-19 VACCINATION IS IMPORTANT	193(96.5)	162(96.4)	31(96.8)	0.8997
COVID-19 VACCINATION IS ESSENTIAL FOR THE GENERAL COMMUNITY	140(70)	117(69.6)	23(71.8)	0.8006
COVID-19 VACCINATION IS MUST FOR ALL MEDICAL PROFESSIONALS	160(80)	136(80.9)	24(75)	0.4404
COVID-19 VACCINE -				

PERSONAL OPINION				
AS AFUTURE PHYSICIAN, I WILL BE PRONE TO COVID-19	192(96)	162(96.4)	30(93.7)	0.4785
COVID-19 VACCINE IS NEEDED FOR ME ,AS AHEALTH CARE PROFESSIONAL	192(96)	161(95.8)	31(96.8)	0.6038
I AM WILLING TO BE A PART IN COVID-19 VACCINE TRIAL	150(75)	139(82.7)	11(34.3)	<0.0001
I CARE ABOUT THE EFFICACY OF COVID-19 VACCINE	150(75)	119(70.8)	31(96.8)	0.0018
I CARE ABOUT THE REACTION IN BODY AFTER COVID-19 VACCINATION	117(58.5)	89(52.9)	28(87.5)	0.0002
ABOUT COVID-19 VACCINE, I NEED EXTRA INFORMATION	172(86)	141(83.9)	31(96.8)	0.053
I BELIEVE ABOUT THE INFORMATION FROM PROFICIENT PUBLIC HEALTH PROFESSIONALS REGARDING COVID-19 VACCINE	170(85)	140(83.3)	30(93.7)	0.1304
I AGREE TO GET VACCINATED FOR COVID-19 ONLY IF IT BECOMES MANDATORY	35(17.5)	13(7.7)	229(68.7)	<0.0001
MY OWN MEETS RELATED TO COVID-19				
I GOT AFFECTED WITH COVID-19 INFECTION	47(23.5)	44(26.1)	3(9.3)	0.0397
I HAD TAKEN CARE OF SOMEONE INFECTED WITH COVID-19	31(15.5)	26(15.4)	5(1.5)	0.9829
I MYSELF KNOW PEOPLE WHO ARE INFECTED WITH COVID-19.	137(68.5)	117(69.6)	20(62.5)	0.4253
I MYSELF KNOW PEOPLE WHO LOST THEIR LIFE DUE TO COVID-19	46(23)	39(23.2)	7(21.8)	0.8689
PERSONAL VACCINATION BEHAVIOUR				
HAVE YOU POSTPONED VACCINATION SCHEDULE FOR REASON OTHER THAN HEALTH ISSUE	20(10)	14(8.3)	6(18.7)	0.0718

DISCUSSION

Finding of the Study

Nearly 16% of students fall under vaccine hesitancy category. Vaccine hesitancy has been reduced over time, when compared to the earlier ones.[5-9] Hesitancy contributing factors include vaccine side effects, safety and efficacy of vaccine and reduced level of belief in the data got from the public health professionals.[10-15] the reasons why students studying medicine are showing hesitance towards COVID-19 are listed in figure 2. Upgrading of more trustable finding regarding vaccine through scientific journals, their educational curriculum and official websites, will further reduce the hesitancy level.[16-18] Majority of those who accepted the vaccine, consider the COVID-19 vaccination as good measure which will help them to get back from the confined life-style during COVID-19 outbreak.84% of students consider vaccines are essential for everyone in the community.

What This Study Adds?

Medical student's perception level towards vaccination is must, as our health system has made the COVID-19 vaccine as essential one. This study was conducted to evaluate the acceptance and hesitancy of COVID -19 vaccine by I-MBBS students in Chennai.

Study Limitation:

Data have been collected from single college which may influence the study conclusion. It is an online study, so it fails to obtain data extensively, which will be possible through other methods of observational study.

CONCLUSION

Majority of the students group had bright attitude on COVID-19 vaccination, and they know the importance of vaccination for themselves and also for the public. To enhance the knowledge among students regarding vaccine, information related to vaccination can be included in the educational curriculum, and the same can be released in scientific journals.[19-21] Students concern regarding vaccination can be given priority, as they are the future physicians.

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ETHICAL STANDARDS

Study was conducted after obtaining ethical clearance from SBMCH -Institutional Ethical Committee, Chennai.

CONFLICT OF INTEREST

Conflict of interest declared none.

REFERENCES

1. Kumar VM, Pandi-Perumal SR, Trakht I, Thyagarajan SP. Strategy for COVID-19 vaccination in India: the country with the second highest population and number of cases. npj Vaccines. 2021 Apr 21;6(1):1-7.
2. Biotech B. Bharat Biotech announces phase 3 results of COVAXIN: Indias first COVID-19 vaccine demonstrates interim clinical efficacy of 81%. Hyderabad, TS. 2021 Mar;3.
3. Kernéis S, Jacquet C, BannayA,. Vaccine education of medical students: a nationwide cross-sectional survey. American journal of preventive medicine. 2017 Sep 1;53(3):e97-104.
4. Afonso NM, Kavanagh MJ, Swanberg SM, Schulte JM, Wunderlich T, Lucia VC. Will they lead by example? Assessment of vaccination rates and attitudes to human papilloma virus in millennial medical students. BMC public health. 2017 Dec;17(1):1-8.
5. Lucia VC, Kelekar A, Afonso NM. COVID-19 vaccine hesitancy among medical students. Journal of Public Health. 2021 Sep;43(3):445-9.
6. Mo PK, Luo S, Wang S, Zhao J, Zhang G, Li L, Li L, Xie L, Lau JT. Intention to receive the COVID-19 vaccination in China: application of the diffusion of innovations theory and the moderating role of openness to experience. Vaccines. 2021 Feb;9(2):129.
7. Larson HJ, Jarrett C, Schulz WS, Chaudhuri M, Zhou Y, Dube E, Schuster M, MacDonald NE, Wilson R. Measuring vaccine hesitancy: the development of a survey tool. Vaccine. 2015 Aug 14;33(34):4165-75.
8. Kernéis S, Jacquet C, Bannay A, May T, Launay O, Verger P, Pulcini C, Abgueguen P, Ansart S, Bani-Sadr F, Bernard L. Vaccine education of medical students: a nationwide cross-sectional survey. American journal of preventive medicine. 2017 Sep 1;53(3):e97-104.
9. Onello E, Friedrichsen S, Krafts K, Simmons Jr G, Diebel K. First year allopathic medical student attitudes about vaccination and vaccine hesitancy. Vaccine. 2020 Jan 22;38(4):808-14.
10. National Academies Release Frame work for Equity Allocation of a COVID-19 Vaccine for Adoption by State, Tribal, Local, and Territorial Authorities. National Academieswebsite. Updated October 2, 2020. (26 October 2020, date last accessed).
11. Betsch C, Wicker S. E-health use, vaccination knowledge and perception of own risk: drivers of vaccination uptake in medical students. Vaccine 2012;30(6):1143–8.
12. Fisher KA, Bloomstone SJ, Walder J. Attitudes toward apotential SARS-CoV-2 vaccine: a survey ofU.S. adults. Ann Intern Med . Published online September4,2020
13. Dror AA, Eisen bach N, Taiber SV accine hesitancy: the next challenge in the fight against COVID-19. Eur J Epidemiol2020;35(8):775–9.
14. Reiter PL, Pennell ML, Katz ML. Acceptability of a COVID-19 vaccine among adults in the United States: how many people would get vaccinated? Vaccine 2020;38(42):6500
15. National Vaccine Advisory Committee. Recommendation from the National Vaccine Advisory Committee: standards for adult immunization practice. Public Health Rep 2014;129(2):115–23..
16. Edwards KM, Hackell JM. Committee on infectious diseases, the committee on practice and ambulatory medicine. Countering vaccine hesitancy. Pediatrics2016;138(3):2016–146.
17. Schaffer De Roo S, Pudalov NJ, Fu LY. Planning for a COVID-19 vaccination program. JAMA 2020;323(24):2458–9.

18. MoHFW (2021) Frequently asked questions: COVID-19 vaccination. New Delhi: Ministry of Health and Family Welfare, Government of India. Available at (AccessedMay2021).
19. Bhuyan A India begins COVID-19 vaccination amid trial allegations. Lancet (London, England) 2021;397,264.
20. Sun S, Lin D and Operario D (2021) Interest in COVID-19 vaccine trials participation among young adults in China: willingness, reasons for hesitancy, and demographic and psycho social determinants. Preventive Medicine Reports 22,101350.
21. Jain J, Saurabh S, Kumar P, Verma MK, Goel AD, Gupta MK, Bhardwaj P, Raghav PR (2021). COVID-19 vaccine hesitancy among medical students in India.
Epidemiology and Infection 149, e132, 1–10.

SP-2

Burst Fracture Lumbar Vertebra Treated with Posterior Stabilisation with Pedicle Screw Fixation- A Case Report

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Abstract: Injury to spine is more serious and life-threatening condition. Almost ninety percent of injuries involve thoracolumbar region. Road traffic accident, fall from height and crush injuries and all other type of trauma lead to lumbar spine injuries. Twenty-five years old female came with alleged history of fall from stairs sustained injury to her lower back region, following which she had difficulty in getting up, standing & walking. X-Ray Antero-Posterior (AP) and a lateral view of the Dorso-lumbar spine showed Burst fractures first lumbar vertebra. Computed tomography (CT) and Magnetic resonance imaging (MRI) spine taken. Posterior stabilisation with pedicle screws and rods from D 11 to L 3 with Posterior fusion done for burst fracture first lumbar vertebra. Postoperative period uneventful without any complications.

Keywords: Burst Fracture, Posterior Stabilisation, Pedicle Screw Fixation, Fracture Lumbar Vertebra.

INTRODUCTION

One among the leading problem in the world today is spine fracture. Current individual lifestyle made them more prone for spine fracture.[1, 2] In orthopaedic practice it is one of the most common problems encountered. In the current adult population fracture of the thoracolumbar spine is the major cause of disability Although 2 recent prospective studies reported similar clinical and radiologic outcomes for operative and nonoperative treatments, they are limited by the fact that the loss of kyphosis correction in the surgical group was significant.[3, 4] It produces socio-economic burden to the country apart from disability. Road traffic accident, fall from height and crush injuries and all other type of trauma lead to lumbar spine injuries.[5] Neurological deficit are found in twenty per cent of spine injury patient leading to increased morbidity and mortality. Compression to flexed spine, shear or rotational component produces most of the spinal fractures. In some cases, extension type of force produces different pattern of fracture. In our population fall from height is most common mode of spinal injuries followed by motor vehicle accidents. Painter and mason are more prone for spinal injurie. In rural side fall from tree is common cause of spinal injuries. Spinal injuries are more common in young adult active earning person in family leading to financial burden in the family thus lower the socio-economic status of the country in general. Due to recent advancement in radiological imaging and more stable fixation and intra operative monitoring the results of spinal injury are better compared to olden days. Steroid injection in spinal injuries has been proven effect in reducing oedema thus preventing secondary injuries. Managing spinal fracture is still challenging for an orthopaedic surgeon despite various advancements. Pedicle screw rod system provide 3 column fixations in fracture stabilization including only less motion segment in the fusion.[6-8] Short segment posterior pedicle screw fixation provides good stability. It has advantaged that patient can be made ambulant at the earliest without pain and neurological recovery is expected if compressing body over cord is removed. Complications like bed ulcer and DVT can be avoided and individual can resume his regular activities at the earliest.The current prospective study aimed to evaluate burst fracture lumbar vertebra treated with posterior stabilisation with pedicle screw fixation.

CASE PRESENTATION

A 25-year-old female came to Sree Balaji Medical College And Hospital with an alleged history of fall from stairs about 10 feet height at her work place and landed on her buttock and sustained injury to her lower back region, following which she had difficulty in getting up, standing and walking. Patient was able to move her both lower limbs and she is able to feel her clothes. There is no history of head injury, loss of consciousness, vomiting, ENT bleed, seizures. No loss of bowel & bladder control. She is a known case of hypothyroidism for past 2 years and is on irregular treatment. On examination Tenderness present at dorsolumbar junction from D11-L2. There is no motor and sensory deficit. Superficial and deep reflexes are found to be normal. Plain radiograph Anteroposterior (AP) and a lateral view of the dorsolumbar showed Burst fractures first lumbar vertebra. Computed tomography spine taken to get completely picture of fracture and Magnetic resonance imaging spine taken to know the amount of spinal cord compression.

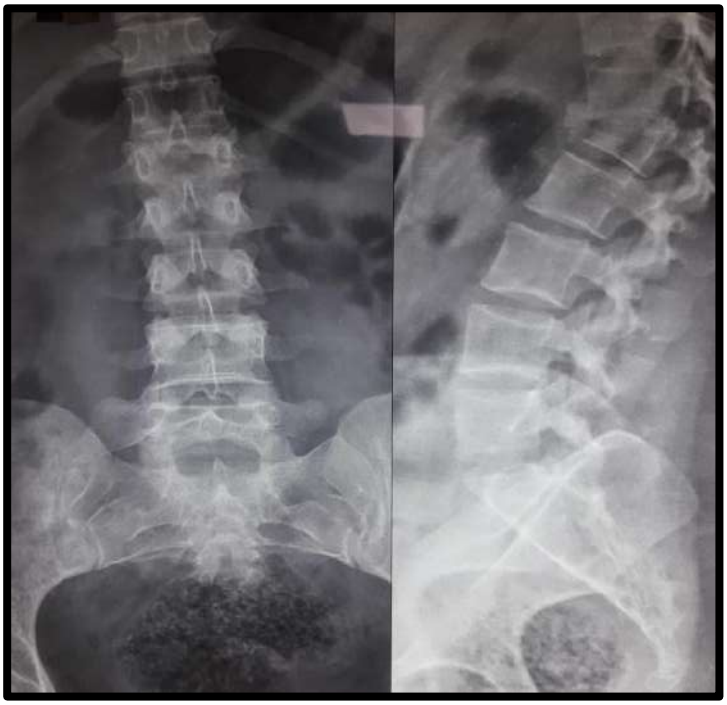

Figure 1: Plain X-ray AP and Lateral view of lumbosacral spine showing burst fracture L1 vertebra.

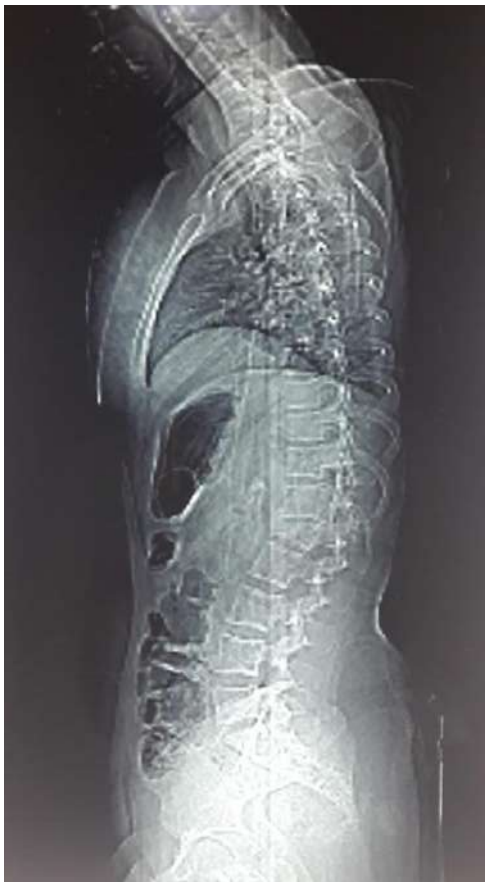

Figure 2: CT showing burst fracture L1 vertebra.

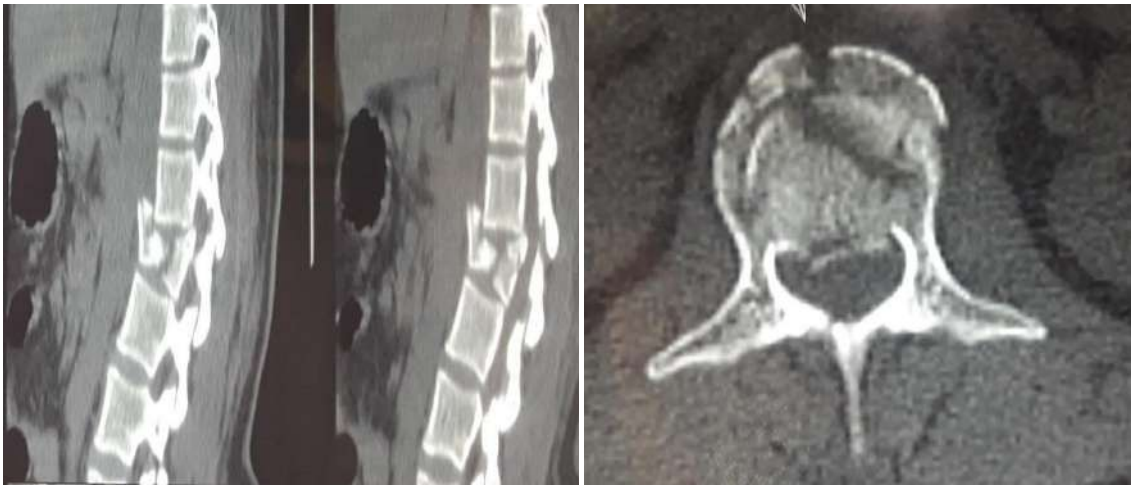

Figure 3: CT sagittal and axial plane of lumbar spine showing burst fracture L1 vertebra.

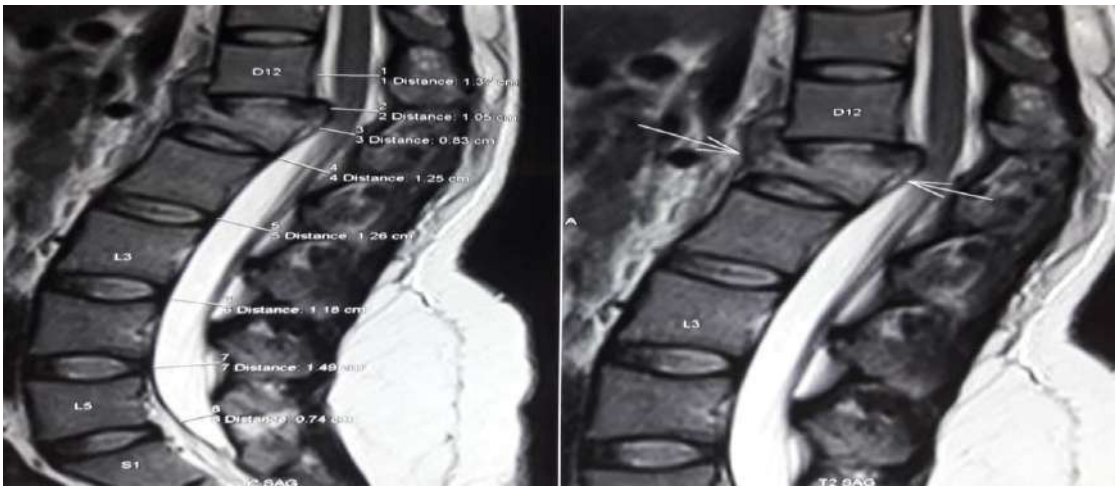

Figure 4: MRI lumbar spine showing compression of spinal cord by fractured L1 vertebra.

Procedure

All Pre-operative investigations done and anaesthetic fitness obtained. Patient posted for posterior stabilisation with pedicle screws and rods from D 11 to L 3. Patient under General Anaesthesia, in prone position, the intra-abdominal pressure declines in prone position and hence it decreases venous stasis and there by the bleeding. Adequate padding should be given for bony prominences. After complete draping, the skin, subcutaneous tissue and paraspinal muscles are infiltrated with 1:50000 epinephrine along with lignocaine solution in order to achieve minimal bleeding. We used standard posterior approach. Through posterior approach a 10cm mid longitudinal incision was made centring over L 1 spinous process. Para spinal muscle erased from D 11 to L 3 level, D12 spinous process was found to be unstable. Laterally the dissection is done till facet and mammillary process visualized. Tapping of the hole till the pedicle body junction to obtain a good purchase at the body. Depth gauge is used for measuring the length of the screw and an appropriate screw is inserted. Posterior stabilisation done using pedicle screws, left side over D11, D12, L1, L2, L3and right side over D11, D12, L2, L3 vertebral bodies. Pedicle screws were connected using connecting rods over both sides.L1 burst fracture level height was restored, facectectomy done on both sides from D11 to L2 ,fusion of vertebra done by using bone graft , harvested from spinous process, Wound wash was given , & closed in layers with drain kept &sterile dressing was done.

Postoperative:

Patient was given post-operative intravenous antibiotics for three days followed by oral antibiotics for five days. Physiotherapy is initiated from day one. Patient is made to turn on either side from day two. Alternate staples are removed on twelfth day. Complete staples removal done on fourteenth day. Patients allowed sit upright with thoracolumbar support and mobilized from day three or four observing closely the neurological status. Brace is support is encouraged for first 3 months. Radiological and neurological parameters are carefully recorded. Postoperative radiograph has taken as a routine prior to discharge.

***Follow up*:**
patient is asked to come for follow-up every 4th week after surgery for the first 6 months. During which clinical, neurological and radiological examinations were performed to assess the stability of the spine.

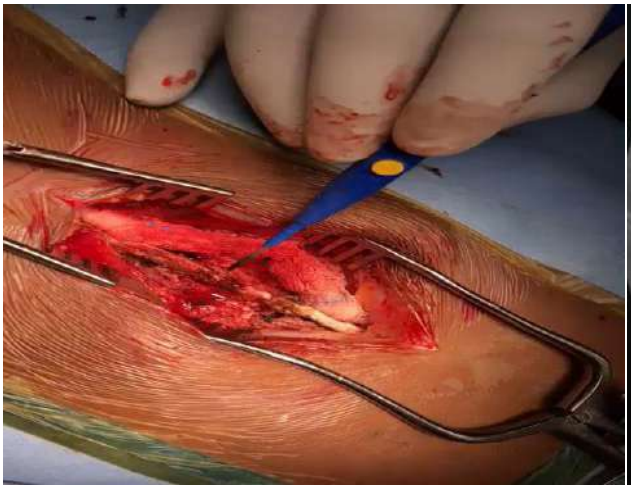

Figure 5: Intraoperative Clinical Picture.

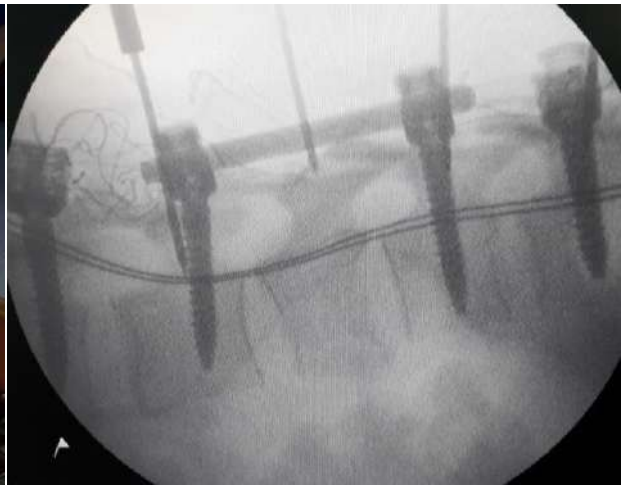

Figure 6: Intraoperative C-Arm picture.

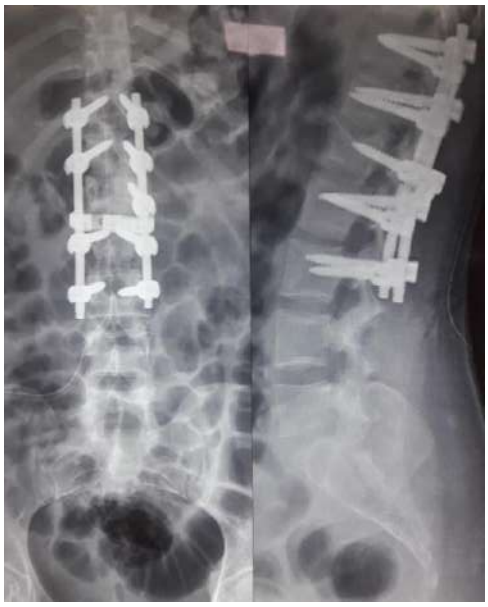

Figure 7: Post-operative X-ray – Plain X-ray AP and Lateral view of lumbosacral spine with posterior stabilization done using pedicle screws and rods for L1 vertebral fracture.

DISCUSSION

The vertebra fracture had been big health problem and is being studied for ages and its occurrence is around 6% of all the fractures of human being and about 60% of these occurring at the dorso-lumbar junction, which is region of relatively high motion and lies between thoracic and lumbar segments. The above statement directly matches with our case where fracture involve L1 vertebra (dorso-lumbar junction). Most common cause of lumbar vertebral body fracture is fall from height followed by motor vehicle accident and crush injuries.[9] In our case trauma is due to fall from height which has been listed as most common mode of lumbar spine injuries. A significant amount of 15% to 20 % are associated with neurological deficits but in our case, there is no neurological deficit. This type of fracture destroys spinal column and affect neurological function hence the main goal of treatment is removing the compression effect promoting recovery of nerve and restoring normal anatomy of spinal column.[10,11] Not all the spinal fractures are treated surgically; the unstable fractures are the ones who require surgical treatment. To achieve the goal invention of new fixation method and various studies on the spinal stability have been done. Decompression is the primary indication for surgery in burst fracture. Neurological status of the patient will improve following surgical decompression and this has been documented that both experimentally and clinically.[12] Direct removal of compressing fragment or indirectly realigning the spine decompression can be achieved. Posterior stabilization uses the ligament taxis principle in fracture reduction and thereby restoring the sagittal contour and indirectly producing the decompression. Posterior stabilization with conventional short fixation has been a very effective method for the management of lumbar fractures.

CONCLUSION

Dorso-lumbar spine fracture can occur in all type of trauma such as road traffic accident, fall from height and crush injuries. Posterior stabilization with pedicle screw and rod for lumbar spine bursts fracture has given good results in short term follow-up. Anterior column height restoration in posterior stabilization is important factor determining outcome. However, a larger sample study and an extended follow up is needed to validate our conclusion further.

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CONFLICT OF INTEREST

Conflict of interest declared none.

REFERENCE

1. Dias JJ. Chapman's Orthopaedic Surgery 3rd edn (4 volumes) Edited by Michael W. Chapman et al. Publisher: Lippincott Williams & Wilkins, Philadelphia, 2001 ISBN 0781714877 Price£ 242.00. Journal of Hand Surgery. 2002 Apr;27(2):208.
2. Dai LY, Jiang SD, Wang XY, et al: A review of the management of thoracolumbar burst fractures. Surg Neurol. 2007, 67: 221-231.
3. Shen W-J, Liu T-J, Shen Y-S. Nonoperative treatment versus posterior fixation for thoracolumbar junction burst fractures without neurologic deficit. Spine 2001;26: 1038-45.
4. Wood K, Buttermann G, Mehbod A. Operative compared with nonoperative treatment of a thoracolumbar burst fracture without neurological deficit. A prospective, randomized study. J Bone Joint Surg Am 2003; 85:773-81.
5. Bucholz RW. Rockwood and Green's Fractures in Adults: Two Volumes Plus Integrated Content Website (Rockwood, Green, and Wilkins' Fractures). Lippincott Williams & Wilkins; 2012 Mar 29.
6. Hwang JH, Modi HN, Yang JH, Kim SJ, Lee SH. Short segment pedicle screw fixation for unstable T11-L2 fractures: with or without fusion? A three-year follow-up study. Acta Orthop Belg. 2009 Dec 1;75(6):822-7.
7. Parker JW, Lane JR, Karaikovic EE, Gaines RW. Successful short-segment instrumentation and fusion for thoracolumbar spine fractures: a consecutive 4½-year series. Spine. 2000 May 1;25(9):1157-70.
8. Butt MF, Farooq M, Mir B, Dhar AS, Hussain A, Mumtaz M. Management of unstable thoracolumbar spinal injuries by posterior short segment spinal fixation. International orthopaedics. 2007 Apr;31(2):259-64.
9. Welsey W. Applied anatomy of spine. Chapter. 1992; 2:29-73.
10. Alizadeh A, Dyck SM, Karimi-Abdolrezaee S. Traumatic spinal cord injury: an overview of pathophysiology, models and acute injury mechanisms. Frontiers in neurology. 2019 Mar 22; 10:282.
11. Dyck S, Kataria H, Alizadeh A, Santhosh KT, Lang B, Silver J, Karimi-Abdolrezaee S. Perturbing chondroitin sulfate proteoglycan signaling through LAR and PTPσ receptors promotes a beneficial inflammatory response following spinal cord injury. Journal of neuroinflammation. 2018 Dec;15(1):1-26.
12. Li Y, Walker CL, Zhang YP, Shields CB, Xu XM. Surgical decompression in acute spinal cord injury: a review of clinical evidence, animal model studies, and potential future directions of investigation. Frontiers in biology. 2014 Feb;9(2):127-36.

SP-3

A Study of the Functional Outcomes of Cemented Vs Uncemented Bipolar Hemiarthroplasty in Displaced Fracture Neck of Femur in Adults

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Abstract: To determine outcomes of cemented versusun cemented bipolar hemiarthroplasty in fractures of neck of femur based on functional outcome, operative time, pain, blood loss and complications. Seventy patients with displaced neck of Femur fracture operated with bipolar hemiarthroplasty were included in this study, of which 30 patients were in the un-cemented group and 40 in the cemented group. Functional outcomes were evaluated using Harris Hip score, blood loss and complication. Post-operative status of the patients was assessed using Harris Hip Score (HHP) and Visual Analogue Score (VAS).The cemented group had significantly less mean pain score compared to the un-cemented group. Functional outcomes based on HHS was better in the cemented group. Mean operative duration and blood loss were more in the cemented group when compared to the un-cemented group. The un-cemented group had higher intraoperative and postoperative complication rates. Based on our study, Un-cemented bipolar hemiarthroplasty is preferred choice in relatively young patients without comorbidities. But despite increased intraoperative bleeding,Cemented is preferred in old age people because of osteoporotic bone, better patient functional levels and less residual pain, thus overall better outcome.

Keywords: Hemiarthroplasty, Neck of femur fractures, un-cemented, cemented

INTRODUCTION

Hip joint is a synovial joint of the ball and socket variety. The ball is the femoral head and acetabulum is the socket. The head and neck make an angle of 130(±7) with long axis of the shaft. Femoral neck fractures are more common among females. Majority of fractures among the elderly occur because of low energy fall, such as fall from standing position, but fall direction is also a key factor.[1] Hemiarthroplasty of the hip is better than internal fixation for displaced neck of femur fractures, as it enables early mobility, lesser rate of reoperations, and better functional outcomes at one year. However, there exists acontroversy whether to use cemented or uncemented HA for elderly patients. Cemented hemiarthroplasty is based upon a solid bone-implant interface, which is created using PMMA bone cement. Cementation aids firm fixation of femoral stem within the femur, there by lesser postoperative mid-thigh pain,[2] however, there is a higher risk of complications like pulmonary embolism due to PMMA particles or contents of bone marrow.[3-5] Uncemented Hemiarthroplasty is based on press-fit technique, which provides primary stability in the femur. Endosteal micro fractures occurring while preparation and resultant bone in-growth leads to long-term stability while being associated with higher rates of per prosthetic fractures. The purpose of our study was therefore to compare the results of cemented vs uncemented bipolar hemiarthroplasty in terms of functional outcome, operative time, pain and blood loss.

MATERIALS AND METHODS

Prospectively analysed the postoperative and 1year follow-up of 70 patients with displaced femoral neck fracture, operated with bipolar hemiarthroplasty. Patients of the age group 65-85yrs: 30 un-cemented and 40 cemented were included in this study, performed at our institute between Aug 2019 to Aug 2020. Patients were evaluated based on functional outcomes by using Harris Hip score (HHS), Visual Analogue Score (VAS) and blood loss. Patients were selected based on Dorr's classification. Dorr's classification attempts to guide indications for cemented or un-cemented femoral component fixation.

Table 1: DORR'S CLASSIFICATION

DORR CLASSIFICATION	*RATIO*	*CHARACTERISTICS*	*SUGGESTTED FIXATION*
Type A	< 0.5	Cortices seen on both AP and lateral Xray	Uncemented
Type B	0.5 to 0.75	Thinning of posterior cortex on lateral XR	Cemented /uncemented
Type C	>0.75	Thinning of cortices on both views	Cemented

Inclusion Criteria

- Age group: 65 to 85 years of both sexes.
- Patients with displaced neck of femur fracture presenting within 2weeks of injury.
- Patients willing and fit for surgery.

Exclusion Criteria

- Patient not conforming to the aforesaid age group

- Patients failing anaesthetic fitness.
- Patient with injury to ipsilateral femur, tibia and knee joint, being treated surgically concomitantly.
- Patient with a pathological femoral neck fracture.

Our Surgical Procedure

Preparation of Patient

On the day of surgery, parts are prepared using povidone-iodine solution and covered with sterile drapes. Prophylactic antibiotics are given on the table. We prefer Cefopera zone with Sulbactam 1.5g IV along with an amino glycoside for a minimum period of 5 days.

Operation Theatre

All the surgeries were done in the OT with laminar air flow.

Anaesthesia Used, Positioning and Approach

Patients are put on epidural/spinal/ general anaesthesia. In our study, all the patients were operated through posterior approach[9]. In this approach, the patient is placed in the true lateral position with the affected limb uppermost. A 10 to 15 cm curved incision is made on the posterior aspect of the greater trochanter. Fascia lata is incised on the lateral aspect of the femur to uncover the vastus laterals. Fascial incision is lengthened superiorly in line with the skin incision and fibres of the gluteus maximus are split by blunt dissection. The hip is internally rotated to put short external rotator muscles on a stretch and to pull the operative field away from the sciatic nerve. Muscles close to the femoral insertion are reflected backward. The posterior aspect of the hip joint capsule is now fully exposed. The hip joint capsule is incised in a T-shaped fashion. Dislocation of hip is achieved by internal rotation, flexion, and adduction. Femoral head with fractured neck is removed and excellent exposure of the acetabulum is obtained. As a routine, swabs were taken both from acetabular and femoral side. Modern bipolar hemiarthroplasty allows different combinations of stem and neck length, and head. These provide a better fitting prosthesis for many patients as the leg length discrepancy and femoral offset are equalized. Muscle tension is adjusted accordingly, which gives better functional outcome and lesser risk of dislocation.

Medullary Preparation

Medullary cavity is prepared and reamed for cementing (not done in uncemented group). The femoral awl is inserted laterally in neck of femur and then rotated to match the ante version of the femoral neck (approx.15°). Such a lateral starting point helps prevent Varus malalignment. With the help of a series of rasps, the intramedullary cancellous bone is removedtill the prosthesis fits appropriately within the medullary canal. In next step, the femoral stem size is confirmed by the fitment of appropriate size rasp.

Introduction of Prosthesis

The prosthesis cemented or uncemented[7] is inserted in valgus alignmentwith laterally placed proximal stem, so that its distal tip is close to the medial femoral cortex. For an uncemented implant, it is ensured that the stem of the prosthesis snugly fits into the medullary canal.

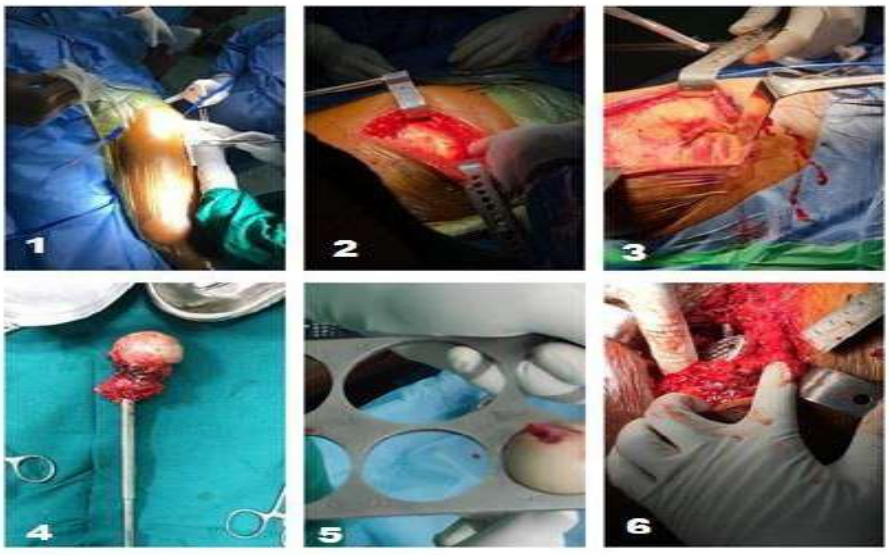

Fig:1 Positions and draping of the patient, Fig: 2 Visualisation via posterior approach of the trochanteric bursa, Fig: 3 The short external rotators, Fig: 4 Extracted head, Fig: 5 Measurement of head size, Fig: 6 Post reaming – checking of measurement after fixation of trial.

Cementation

Before inserting the cement, the canal is meticulously cleaned and irrigated. A dry sponge is kept in the canal, temporarily. A cement restrictor is placed 1-2 cm below the prosthesis. This permits the cement to be pressurised. The polymer and monomer components of the cement are mixed[8].With the help of a cementation gun, the medullary canal is filled from bottom to top. Care is taken to avoid air or blood mixing with cement. Now the cement is compressed with the prosthesis. This pushes the cement into the surrounding bone, ensuring better anchorage. The prosthesis is inserted before the cement hardens, in ante version and valgus position. It is then secured in the befitting predetermined depth. Once the stem is placed, the cement is allowed to set. Excess cement is pared from the hip joint and the wound. Trial femoral head is used on the cemented stem to determine the diameter and neck length. Soft tissue tension and hip stability are affected by the neck length. With reduction, the stability is checked and once satisfied, the definitive femoral head is inserted to the stem.

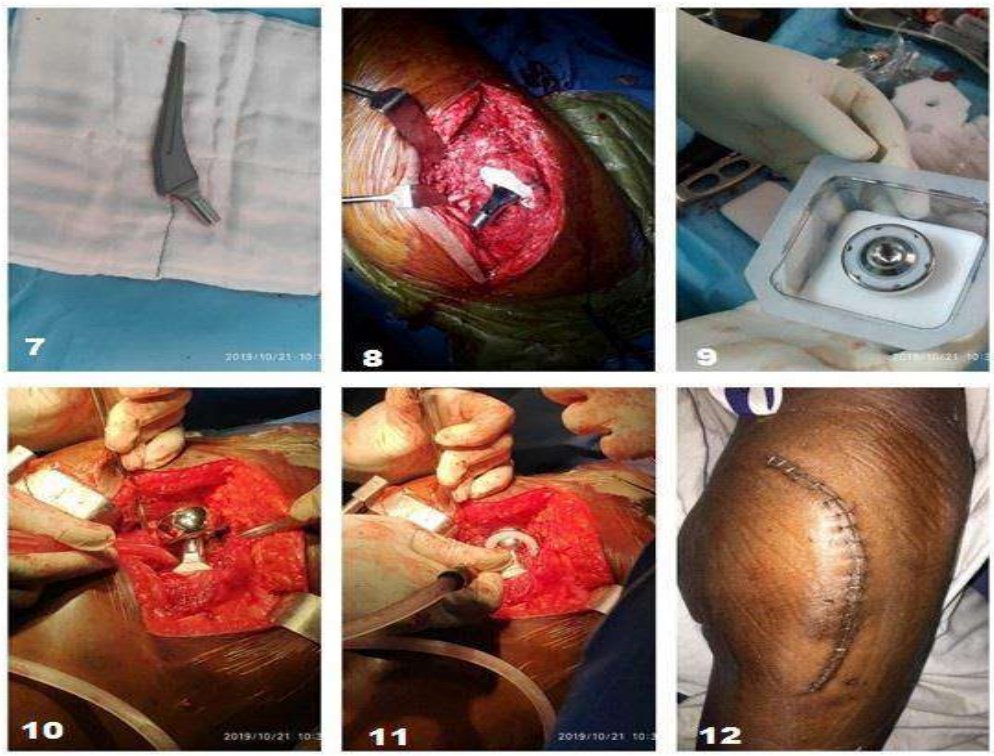

Fig: 7 Prosthesis – Stem component, Fig: 8 Fixation with cementing, Fig: 9 Prosthesis – Head with polyethylene component, Fig: 10 Reduction of head, Fig: 11 Reduction of head, Fig: 12 Post-operative day 2 dressing after drain removal.

After the hip is reduced, posterior soft tissue envelope is repaired. Capsule (if preserved) is repaired using non absorbable sutures. Previously labelled tendons of short external rotators are reattached to the greater trochanter, in the posterior aspect. Post-operative hip stability depends on careful reconstruction of the posterior soft tissue envelope. Two closed suction DTs are inserted, one deep to the TFL and the other in the subcutaneous plane and brought out through stab wounds separately. The fascial incisionis closed with closely approximated sutures, with hip in 10°abduction. The subcutaneous layer is closed with interrupted absorbable sutures. Skin is closed in routine fashion.

Postoperative Care and Rehabilitation

Antibiotics

The patient is given IV antibiotics for 5 days.

Post operative care

Necessary aseptic precautions are taken in the postoperative ward. An abduction pillow is placed between the legs and a small pad is placed beneath the knee to maintain it in slight flexion. Drain tubes are removed after 48 hrs.

Rehabilitation protocol

This is started pre-operatively, the exercises to be done - Ankle dorsiflexion and plantar flexion, Quadriceps, and gluteal exercises, are trained by the physiotherapist. The exercises are started as soon as the painsubsides. Upper limb and chest physiotherapy are also done.On POD 1, patients areallowed to sit in bed. After removal of drain, patient isencouraged to stand and walk with support. On POD 12, sutures are removed and patient isadvised to full weight bear after 4 weeks. The patient is

advised against adduction, flexion, and internal rotation. The patient isalso instructed to avoid squatting, sitting cross legged and is to adapt to a table and chair lifestyle.

Follow up

Patients were reviewed regularly at 6 weeks, 3months, 6 months, 1year. Patients were assessed for clinical evaluation of hip function (According to HHS) and intensity of pain (Based on VAS), blood loss, operative time and postoperative complications.

RESULT

70 patients operated with bipolar hemiarthroplasty at SBMCH were followedup, among which 40cemented, and 30 uncemented were evaluated. The mean age of the patients was 72(65-85) years in cemented group and 70(65-85) years in uncemented group. The mean operativeduration was 90 minutes in cemented group and 70 minutes in uncemented group. The mean intraoperative blood loss was 300cc and 265cc in cemented and uncemented groups, respectively (P<0.05). Duration of admission was 11 days in cemented group and 10 days in the uncemented. The meaning of pain, according to VAS criteria was 1.3 ± 0.2 after one month in cemented group that was 1.7 ± 0.3 in uncemented group, respectively and there were significant differences.The mean of HHS in cemented group was 86 at 1 yr, uncemented group was 81 at 1 yr.

Table 2: HARRIS HIP SCORE

HHS (at 6 months)	Uncemented (30)	Cemented (40)
100-91 (Excellent)	10	22
90-81(Good)	6	7
80-71 (Fair)	13	10
<70 (Poor)	1	1

Table 3: PRE AND POST SURGICAL HARRIS HIP SCORE

	Uncemented	Cemented
Mean pre op	36.2	36.2
Mean latest	81.5	85.8

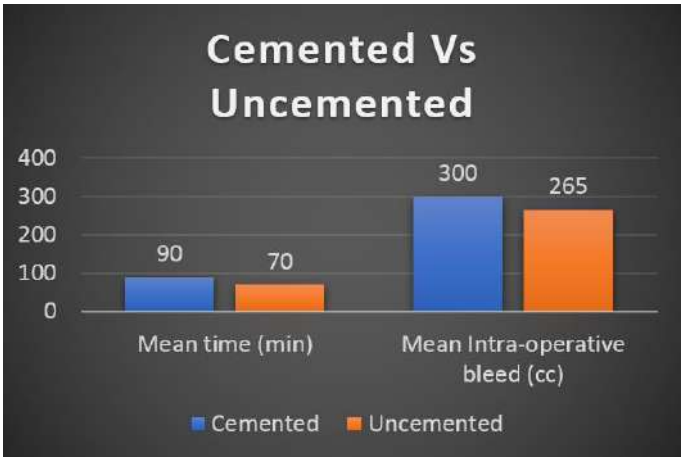

Fig.13. In cemented group, Mean time is 90 min and mean intraoperative bleed is 300cc and In uncemented group Mean time is 70 min and mean intraoperative bleed is 265cc.

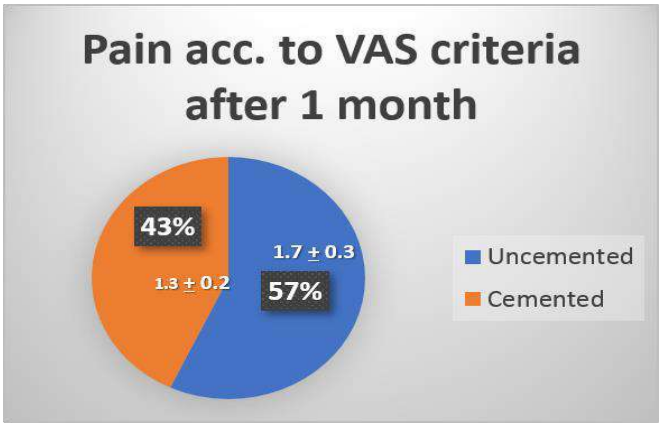

Fig 14. In cemented group, pain according to VAS after 1month is 1.3±0.2 and In uncemented group, it is 1.7±0.3

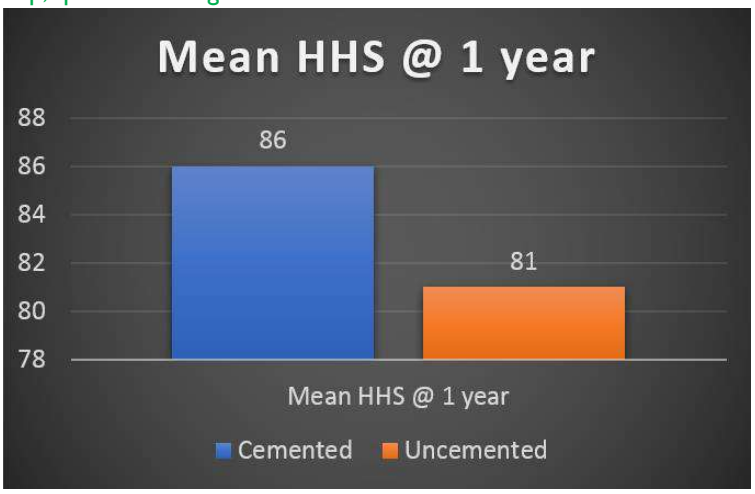

Fig 15: In cemented group, Mean HHS @ 1 year is 86 and In uncemented group, Mean HHS @ 1 year is 81

DISCUSSION

Fracture femoral neck has a higher incidence in elderly age people and is associated with high morbidity and mortality. Hemiarthroplasty is recently most practiced, performed with unipolar& bipolar prosthesis. Bipolar HA is an effective treatment modality for femoral neck fractures, which is beneficial in that it could help early ambulation andprovides satisfactory functional recovery, thus is increasingly being performed by the Ortho surgeons. In bipolar prosthesis, there is movement between metal head and polyethylene cover and movement between metal cup and the acetabulum (outer bearing). This is advantageous in that it causes less erosion and protrusion in acetabulum. Also, length of femoral neck and size of the head are variable and thus can be converted to THA. According to some studies, the use of bipolar prosthesis has shown better outcomes for management of femoral neck fracture in the elderly.[6] Hemiarthroplasty can be performed with or without the use of cement, both of which have different outcomes Thus, indications have been evaluated by some studies.The hemiarthroplasty is either cemented into the femoral canal or uncemented with press-fit technique[10]. In this study we compared patients who underwent hemiarthroplasty with cemented and uncemented bipolar prosthesis (HHS) to assess the outcome.[7,8] The study showed significant improvement in patients operated with cemented hemiarthroplasty with mean HHS 86% compared to uncemented group with HHS 81%, Cemented group had better mobility and less pain compared to cemented group. The study showed more bleeding in cemented group when compared to uncemented group[11-14].Pain according to VAS criteria was significantly less in uncemented group. In the present study, the average duration surgery was 92 minutes (81-118minutes) the average blood loss was 321ml (275-375ml) and the average hospital stay was 16 days (ranges 14-23 days).

CONCLUSION

Based on our study, Uncemented bipolar hemiarthroplasty is preferred choice in relatively young patients without comorbidities. But despite higher intraoperative bleeding, Cemented is preferred in old age people because of osteoporotic bone, better patient functional levels and less residual pain, thus overall better outcome.

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CONFLICT OF INTEREST

Conflict of interest declared none.

REFERENCES

1. Parker MI, Pryor G, Gurusamy K. Cemented versus uncemented hemiarthroplasty for intracapsular hip fractures: a randomised controlled trial in 400 patients. The Journal of bone and joint surgery. British volume. 2010 Jan;92(1):116-22.
2. Langslet E, Frihagen F, Opland V, Madsen JE, Nordsletten L, Figved W. Cemented versus uncemented hemiarthroplasty for displaced femoral neck fractures: 5-year followup of a randomized trial. Clinical Orthopaedics and Related Research. 2014 Apr;472(4):1291-9.
3. Learmonth ID, Young C, Rorabeck C. The operation of the century: total hip replacement. The Lancet. 2007 Oct 27;370(9597):1508-19.
4. Figved W, Opland V, Frihagen F, Jervidalo T, Madsen JE, Nordsletten L. Cemented versus uncemented hemiarthroplasty for displaced femoral neck fractures. Clinical Orthopaedics and Related Research. 2009 Sep;467(9):2426-35.

5. Khan RJ, MacDowell A, Crossman P, Keene GS. Cemented or uncemented hemiarthroplasty for displaced intracapsular fractures of the hip—a systematic review. Injury. 2002 Jan 1;33(1):13-7.
6. Fallon KM, Fuller JG, Morley-Forster P. Fat embolization and fatal cardiac arrest during hip arthroplasty with methylmethacrylate. Canadian Journal of Anesthesia. 2001 Jul;48(7):626-9.
7. Ries MD, Lynch F, Rauscher LA, Richman J, Mick C, Gomez M. Pulmonary function during and after total hip replacement. Findings in patients who have insertion of a femoral component with and without cement. The Journal of bone and joint surgery. American volume. 1993 Apr 1;75(4):581-7.
8. Skyrme AD, Jeer PJ, Berry J, Lewis SG, Compson JP. Intravenous polymethyl methacrylate after cemented hemiarthroplasty of the hip. The Journal of arthroplasty. 2001 Jun 1;16(4):521-3.
9. Campbell's operative orthopaedics: ed: Terry Canale and James H. Beaty; 12th ed. Elsevie, St. Louis, Mo. 2013. Pp:181
10. Jaimo Ahn MD, PhD, Li-Xing Man MD, MSc, SangDo Park MD, Jeffrey F. Sodl MD, Systematic Review of Cemented and Uncemented Hemiarthroplasty Outcomes for Femoral Neck Fractures, Clin Orthop Relat Res (2008) 466:2513– 2518
11. Bell KR, Clement ND, Jenkins PJ, Keating JF. A comparison of the use of uncemented hydroxyapatite-coated bipolar and cemented femoral stems in the treatment of femoral neck fractures: A case-control study. Bone Jt J. 2014;96 B(3):299–305.
12. Taylor F, Wright M, Zhu M. Hemiarthroplasty of the Hip with and without Cement: A Randomized Clinical Trial. J BONE Jt Surg [Internet]. 2012;94(7):577–83. Available from: papers2://publication/uuid/49E47BEB-E56B-4F89-BE82-A0D4F3BECB4E
13. Kapoor U, Chug A, Baranwal G, Patil S, Kumar S. Comparing outcomes in cemented vs uncemented hemiarthroplasty in femoral neck fractures. Int J Res Orthop. 2018;5(1):152.
14. Mohabey A V, Warjukar PR, Ravikumar M. Functional outcome of cemented versus uncemented modular bipolar hemiarthroplasty in proximal femoral neck fractures. Int J Orthop Sci. 2017;3(4i):609–11.

SP-4

An Unusual Case of Suspected Brodie's Abscess in Midshaft of Tibia

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Abstract: Brodie's abscess is a form of subacute pyogenic osteomyelitis, which may progress to chronic osteomyelitis if not managed properly. Since its first appearance in medical literature in 1832, numerous cases have been described. It usually occurs in the cancellous parts of the long bones, and especially the tibia being the most frequently affected. The most common pathogen is staphylococcus aureus, in around 30-60 percent of the cases. However in 20-50 percent of the cases there would be no organisms present. This report is on a discussion about the case of Brodie's abscess occurring on the mid shaft diaphysis of the tibia. Brodie's abscess usually affects the cancellous part of the long bones. But it can be said that brodie's abscess can also occur on diaphysis of long bones as well. Therefore it is necessary to have brodie's abscess as differential diagnosis in mind. To prove the possibility of occurrence of brodie's abscess in the diaphysis of long bones. A 20-year-old male presented with symptoms of pain in the right leg for 3 months. History of trauma 3 months ago (fall from motorcycle). Diagnosis was made with a combination of imaging modalities: plain X-ray, MRI and CT-scan. Treatment consisted of surgery and conjunction with long term antibiotics. Staphylococcus aureus was the pathogen most often found in the culture (67.3%). Brodie's abscess usually occurs on metaphysis of the long bones. Therefore the differential diagnosis of brodie's abscess in the diaphysis of the long bones to be considered.

Keywords: Brodie's abscess, operative management, metaphysis, diaphysis, long bones, staphylococcus aureus

INTRODUCTION

Brodie's abscess is a form of subacute pyogenic osteomyelitis, which may progress to chronic osteomyelitis if not managed properly. Since its first appearance in the medical literature in 1832, numerous cases have been described. In immunocompetent individuals, acute osteomyelitis is limited and walled off by granulation tissue, resulting in Brodie's abscess.[1, 2] It usually occurs at the cancellous part of the long bones especially in the lower limbs and particularly the tibia is more frequently affected.[3]The commonest pathogen is Staphylococcus aureus in around 30-60% of cases. However, in 25-50% of cases no organisms will be present.[4, 5] Making an accurate and timely diagnosis is usually a challenge as pain or swelling are generally the most stereotypical and vague complaints at presentation. therefore, in the absence of any physiological or Hematological signs of illness other then pain, many of these cases can end up being symptomatically treated until definitive testing and management takes place.[6,7] Some authors reported that systemic antibiotics alone might be effective in treating primary subacute osteomyelitis in children and suggested that surgery should be reserved for aggressive lesions and those not responding to antibiotic therapy.[8-10] Prolonged pharmacological therapy may result in high antibiotic serum concentration associated with nephrotoxic and ototoxic effects and allergic complications. Here, we present a similar case with all the hallmark features of Brodie's abscess. What makes this case especially interesting is that the patient developed pain and sclerosis around the diaphyseal region of the tibia. The sclerotic part was identified and treated successfully with surgical saucerization and curettage, followed by a course of long-term antibiotics.

CASE HISTORY

***Patients Presenting Complaint*:** Complaints of pain in Right Leg for Past 3 months

History of Presenting Illness

A 20 year old male presented with symptoms of pain in the right leg for 3 months. History of trauma 3 months ago (fall from motorcycle). Pain was insidious in onset post trauma, non radiating pain, dull aching type of pain, constant in nature, and no aggravating or relieving factors.

Past History

Patient had no history of any medical comorbidities.
Patient has no relevant past history.
History of no surgeries in the past.

Family History

Nil significant family history.

Observation

On examination, there was tenderness 2-3 cm below tibial tuberosity.

Mild Warmth was present over the junction between the proximal and middle third of the shaft of tibia.
No visible abnormalities on the overlying skin. No distal neurovascular deficit.

Special test

Not applicable.

Investigations

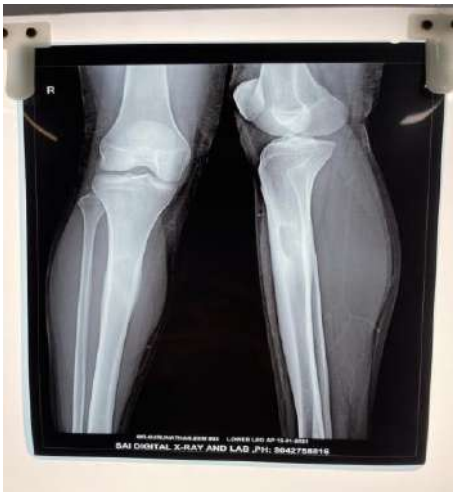

Fig.1. X ray shows a well defined lytic lesion surrounded by a rim of sclerosis, cortical thickening at this same level is noted in the proximal ⅓ rd of the shaft of the right tibia .

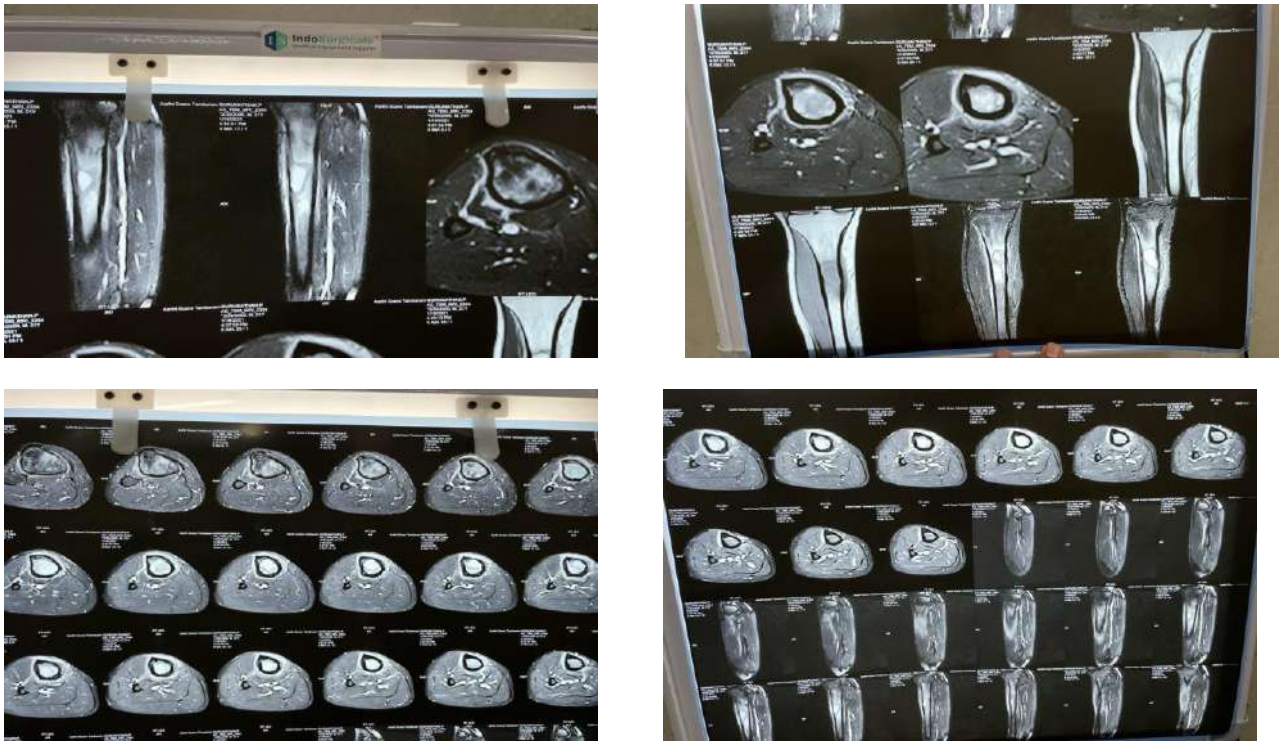

Figure: 2 ABCD MRI T2WI and STIR images in the coronal and axial sections shows a well defined hyperintense lesion with hypointense margin noted in the midshaft of the tibia. Surrounding intramuscular edema noted.

Diagnosis

Brodie's abscess on the proximal 1/3rd of the diaphyseal region of the right tibia.

Prognosis

Brodie's abscess responds well to surgical curettage of the abscess, cancellous bone grafting and long term antibiotic therapy.[11]

Our patient had a satisfactory outcome, with a new bone formation in the cavity. Patient didn't report of any reoccurrence

Treatment Plan

Based on the fact that it is a well demarcated osteolytic lesion with surrounding sclerosis, it is highly likely to be of infective etiology. Hence bone saucerization and curettage was planned with biopsy.

Procedure Done

The treatment option would usually involve surgery followed by long term antibiotics.[6] A 5 cm incision made over the antero medial aspect of the proximal mid shaft of tibia. Periosteum and other soft tissues were retracted with the aid of a periosteal elevator. Entry point for bone was confirmed with fluoroscopic guidance. With a drill bit, serial burr holes were made. A gush of yellowish green pus was noted once the burr holes were made. With the help of an osteotome an oval window was made. A cavity was made by doing serial curettage and with the help of a bone nibbler. Serial saline, hydrogen peroxide and povidone iodine wash was given. After serial thorough wound washes, closed in layers with drain in situ and a sterile dressing was done.

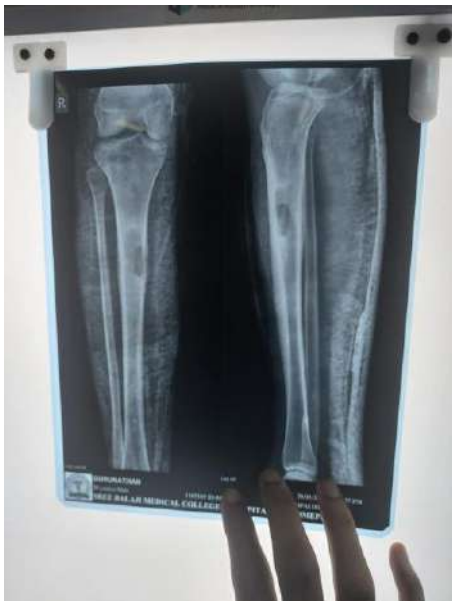

Figure: 3 Post Operative X ray

DISCUSSION

This paper presents a case of brodie's abscess which has occurred on the right shaft of tibia. Brodie's abscess was described as subacute osteomyelitis without any acute symptoms.[7] It appears more commonly in lower extremities, especially in the metaphyseal/ cancellous part of long bones [12-15]. It is difficult to diagnose osteomyelitis by imaging investigations. Several studies report satisfying results (up to 100% success rates), very low recurrence rates and few complications of surgical debridement[8-12]. A plain radiograph is less helpful than MRI in distinguishing it from other diseases such as tumours. Regarding treatment of brodie's abscess, several treatments have been reported[13-15]. It has been reported that medical cost, length of stay and complications are lower than that of conservative treatment and antibiotics. Treatment consists primarily of surgery (94%) often in combination with antibiotics(97%)[16,17].Patients with Brodie's abscess respond well to surgical curettage of the abscess, cancellous bone grafting and antibiotic therapy. In our case we planned for sauceriation and curettage with biopsy and done.[18-21]

CONCLUSION

Brodie's abscess usually occurs on metaphysis of the long bones. But it can be said that brodie's abscess can also occur on diaphysis of long bones as well. Therefore it is necessary to have brodie's abscess as differential diagnosis in mind.

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FUNDING SOURCE

None

CONFLICT OF INTEREST

Conflict of interest declared none.

REFERENCE

1. Brodie BC. An account of some cases of chronic abscess of the tibia. Medico-chirurgical transactions. 1832;17:239.
2. Harris NH, Kirkaldy-Willis WH. Primary subacute pyogenic osteomyelitis. The Journal of bone and joint surgery. British volume. 1965 Aug;47(3):526-32.
3. Labbé JL, Peres O, Leclair O, Goulon R, Scemama P, Jourdel F, Menager C, Duparc B, Lacassin F. Acute osteomyelitis in children: the pathogenesis revisited?. Orthopaedics & Traumatology: Surgery & Research. 2010 May 1;96(3):268-75.
4. Ruttan TK, Higginbotham E, Higginbotham N, Allen CH, Hauger S. Invasive Kingella kingae resulting in a brodie abscess. Journal of the Pediatric Infectious Diseases Society. 2015 Jun 1;4(2):e14-6.
5. Trueta J. The three types of acute haematogenous osteomyelitis: a clinical and vascular study. The Journal of Bone and Joint Surgery. British volume. 1959 Nov;41(4):671-80.
6. der, N. v., Smeeing, D. P.J., Houwert, R. M., Hietbrink, F., Govaert, G. A.M., and der, D. v.: Brodie's Abscess: A Systematic Review of Reported Cases, J. Bone Joint Infect., 4, 33–39, https://doi.org/10.7150/jbji.31843, 2019.
7. Gulati Y, Maheshwari AV. Brodie's abscess of the femoral neck simulating osteoid osteoma. Acta Orthop Belg. 2007 Oct;73(5):648-52. PMID: 18019923.
8. Hamdy RC, Lawton L, Carey T, Wiley J, Marton D. Subacute hematogenous osteomyelitis: are biopsy and surgery always indicated?. Journal of Pediatric Orthopaedics. 1996 Mar 1;16(2):220-3.
9. Ezra E, Cohen N, Segev E, Hayek S, Lokiec F, Keret D, Wientroub S. Primary subacute epiphyseal osteomyelitis: role of conservative treatment. Journal of Pediatric Orthopaedics. 2002 May 1;22(3):333-7.
10. Ross ER, Cole WG. Treatment of subacute osteomyelitis in childhood. The Journal of bone and joint surgery. British volume. 1985 May;67(3):443-8.
11. Olasinde AA, Oluwadiya KS, Adegbehingbe OO. Treatment of Brodie's abscess: excellent results from curettage, bone grafting and antibiotics. Singapore Med J. 2011 Jun;52(6):436-9. PMID: 21731997.
12. Gaillard, F., Fahrenhorst-Jones, T. Brodie abscess. Reference article, Radiopaedia.org. (accessed on 27 Jan 2022) https://doi.org/10.53347/rID-1019
13. Conterno LO, Turchi MD. Antibiotics for treating chronic osteomyelitis in adults. Cochrane database of systematic reviews. 2013(9).
14. Kamio S, Arai M, Matsumoto S, Saito M, Asano N, Nakayama R. Radiological changes in the formation of Brodie's abscess by sequential magnetic resonance imaging: a case report. Radiology Case Reports. 2021;16(10):2993-7.
15. Boriani S. Brodie's abscess. A study of 181 cases, with special reference to radiographic diagnostic criteria . Ital J Orthop Traumatol 1980;6:373–83.
16. Miller WB Jr, Murphy WA, Gilula LA. Brodie abscess: reappraisal. Radiology 1979;132:15–23. 10.1148/132.1.15
17. Bogoch E, Thompson G, Salter RB. Foci of chronic circumscribed osteomyelitis (Brodie's abscess) that traverse the epiphyseal plate. J Pediatr Orthop 1984;4:162–9. 10.1097/01241398-198403000-00003
18. Davis JM, Peel MM. Osteomyelitis and septic arthritis caused by Kingella kingae. Journal of clinical pathology. 1982 Feb 1;35(2):219-22.
19. Tan K, Yoong P, Marshall TJ, Martin C. Percutaneous drainage as a novel approach for the treatment of Brodie's abscess. Clinical radiology. 2012 Oct 1;67(10):1030-3.
20. Jaramillo D, Dormans JP, Delgado J, Laor T, St Geme III JW. Hematogenous osteomyelitis in infants and children: imaging of a changing disease. Radiology. 2017 Jun;283(3):629-43.
21. Gillespie WJ, Moore TE, Mayo KM. Subacute pyogenic osteomyelitis. Orthopedics. 1986 Nov 1;9(11):1565-70.

SP-5

A Case Report of Chronic Osteomyelitis after Tibia Nailing Treated by Implant Removal with Biodegradable Antibiotic Coated Beads

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Abstract: Chronic bone osteomyelitis is a complicated disease to treat. Various treatment modalities are available for osteomyelitis. We report a case of chronic osteomyelitis after tibia nailing treated by implant removal and local biodegradable antibiotic coated beads. Outcome was studied. 40 years male operated 5 years ago for right tibia fracture with tibial intramedullary nail now presented with complaints of discharge from the right leg for the last eight months. Patient treated with removal of intra medullary nail, wound debridement and IV antibiotics. After 1week of IV antibiotics, post-operatively patient had a recurrent purulent discharge. Patient was treated with medullary canal serial reaming, followed by the treatment of the infectious tibial medullary canal with biodegradable antibiotic coated beads. In our case ,after treatment of the infectious tibial medullary canal with biodegradable antibiotic coated beads, post-operative discharge decreased and stopped over time. The patient recovered well and was mobilized prior to discharge. Hence we conclude absorbable local antibiotic is very effective in the treatment of chronic osteomyelitis. Biodegradable antibiotic coated beads offers a more patient-friendly treatment compared with other treatment options

Keywords: antibiotic beads, osteomyelitis, biodegradable, IV antibiotics

INTRODUCTION

Chronic osteomyelitis is a one of the worst complications after trauma or orthopaedic surgery.[1] Patients usually have multiple unsuccessful treatment history because of poor soft tissues , multi-resistant organisms and multiple comorbidities[2-5]. Staged surgical treatment is common. Patient undergo repeated wound debridement and delayed skin closure.[2,6-14]Further ,surgeries may be required to reconstruct bone defects or to remove polymethyl methacrylate (PMMA) antibiotic-loaded beads.[15,16] Recently, along with multiple debridement, negative pressure wound therapy (NPWT), has been combined .But it only increases the number of revision procedures and not improving the rate of resolution of osteomyelitis.[17,18] Implant-associated infections are still represent one of the major problems. Though systemic antibiotic prophylaxis are accepted; recent literature highlights the importance of local antibiotic therapy at the fracture site. Branstetter et al[19] conducted animal studies and he showed that when compared with calcium sulphate alone, local antibiotics in calcium sulphate eradicated bacteria better after wound debridement Rand, Penn-Barwell and Wencke[20], proved that local antibiotics into an infected bone defect was superior to systemic antibiotics alone. Parental antibiotics required to penetrate and destroy bacteria enclosed in the bio-film is approximately 10 to 100 times the normal bacterial concentration, thus making intravenous therapy ineffective in such cases. Parental antibiotics are also associated with allergic and nephrotoxic side effects.[4-6] Osteomyelitis associated with in situ implants are caused > 90 % of the time by Staphylococcus epidermidis. Chronic bone infection decreases the cortical blood supply and may lead to sequestra formation. Necrotic cortical bone pockets are also difficult to treat. In reaction to the sequestrum, new bone formation is normally seen and the sequestra is surrounded by the involucrum. If osteomyelitis is in a chronic stage of sequestra development, it typically needs surgical intervention. For decades, the treatment of osteomyelitis using local antibiotic delivery has been used.[8-12] Various modalities of treatment are developed for controlling infection of bone. Various treatment modalities have been developed to control bone infections.[21] Biodegradable materials such as calcium sulphate have recently become successful because they have different advantages and excellent results in the treatment and control of bone osteomyelitis.[22]Therefore, we present a case report of an infected tibia with an intramedullary nail in situ that we treated with implant removal along with antibiotic coated calcium sulphate beads.[15]

CASE REPORT

40 years male operated 5 years ago for right tibia fracture with tibia intramedullary nail in situ now presented with complaints of discharge from the right leg for the last eight months. The patient was assessed and scheduled for implant removal with all baseline investigations and wound culture. Implant removal and wound debridement of the sinus discharge were performed. Intravenous antibiotics started. After one week of intravenous antibiotics, post-operatively patient had a recurrent purulent discharge. Thus, the patient was scheduled to receive local antibiotics. We took patient for surgery; we did medullary canal serial reaming with reamer and filled the tibia intramedullary canal with calcium sulphate beads coated with antibiotics. MEROPENEM was used as an antibiotic and we used 500mg of antibiotics for every 10cc of calcium sulphate. Based on the cultural sensitivity of the patient's wound-prior to surgery, antibiotics were determined. The post-operative discharge decreased and stopped over time. The patient recovered well and was mobilized prior to discharge.

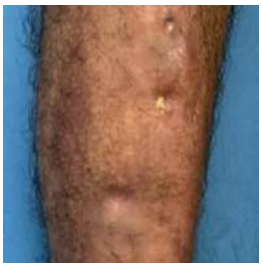

Figure:1 Clinical picture with active sinus discharge

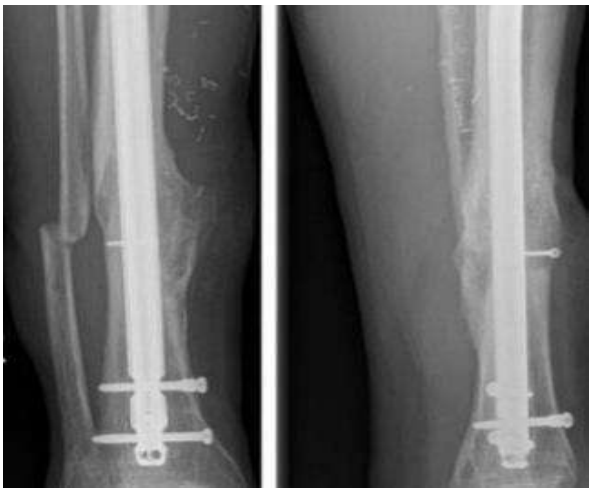

Figure: 2 pre op x ray of the patient with implant

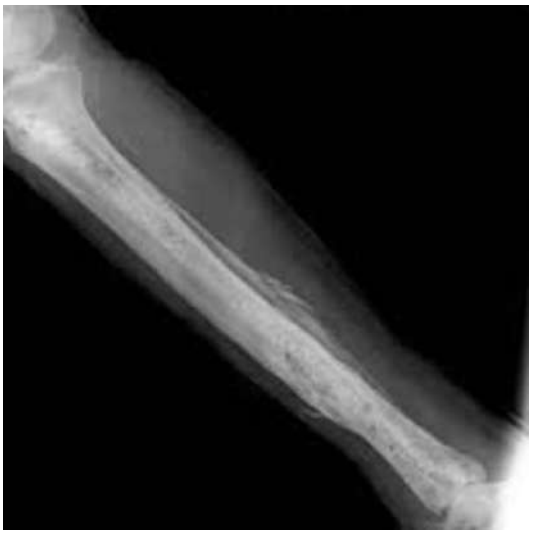

Figure: 3 Post operative x-ray

RESULTS AND DISCUSSION

Chronic osteomyelitis is a difficult infection to treat due to both the multidrug resistance of the typical pathogenic microorganism and the low penetration of antibiotics in the bone. A frequent issue associated with systemic antibiotic therapy and often even local drug delivery systems is the insufficient release of the antibacterial agent to the contaminated bone site. The main drawback of PMMA is that the material is non-biodegradable and that subsequent invasive operations are required to remove the implant. In addition, PMMA has a weak elution profile, characterized by an initial release of relatively high concentrations of bolus accompanied by a rapid decline in sub-inhibiting concentrations.[23] they must be removed when bone graft is inserted in a further surgical procedure. Biodegradable antibiotic beads are a synthetic hemi hydrate form of Calcium sulphate. It is manufactured using a synthetic process resulting in 100% purity with no traces of potentially poisonous impurities associated with naturally occurring mineral sources of Calcium Sulphate. Biodegradable antibiotic-coated beads often have the

advantage of having a broader variety of antibiotic combinations. It cures at a low temperature, enabling the mixing of heat-sensitive antibiotics with biodegradable beads coated with antibiotics. Synthetic calcium sulphate provides the benefit of predictability in the elution of antibiotics over a duration of three to four weeks, buffering the pH of the local wound (towards physiologic), elimination/reduction of dead space and compatibility with a variety of antimicrobials. Subsequent procedures for removing the inserted material and recreation of dead space are avoided due to the resorb ability of the beads. In the treatment of osteomyelitis, surgical debridement, obliteration of dead space resulting from debridement and a long course of antibiotics remain the keystone.[22] A number of studies have shown that combining debridement with the use of antibiotic impregnated material would increase the eradication of infection and potentially minimize the period of the required systemic antibiotics.

CONCLUSION

Chronic osteomyelitis has different treatment modalities such as: obtaining multiple bacteriological samples, administering culture sensitive systemic antibiotics, performing implant removal, thorough wound debridement, eliminating the dead space, providing adequate vascularised soft-tissue cover and ensuring adequate osseous stabilisation. But the use of local biodegradable antibiotic coated beads has been a recent development in the field of orthopaedics, which has the benefit over PMMA of not requiring more surgical procedures to extract the same. Our initial experience shows that local biodegradable antibiotics offer a patient-friendly treatment which merits further study.

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CONFLICT OF INTEREST

Conflict of interest declared none.

REFERENCES

1. Salvana J, Rodner C, Browner BD, et al. Chronic osteomyelitis: results obtained by an integrated team approach to management. Conn Med 2005;69:195–202.
2. McNally M, Nagarajah K. Osteomyelitis. Orthop Trauma 2010;24:416–429.
3. Romanò CL, Romanò D, Logoluso N, Drago L. Bone and joint infections in adults: a comprehensive classification proposal. Eur Orthop Traumatol 2011;1:207–217.
4. Berendt AR, McNally M. Osteomyelitis. In: Warrell DA, Cox TM, Firth JD, eds. Oxford Textbook of Medicine. Oxford: Oxford University Press, 2010:3788–3795.
5. McNally M, Sendi P. Implant-Associated osteomyelitis of long bones. In: Zimmerli W, ed. Bone and Joint Infections: From Microbiology to Diagnostics and Treatment. Chichester, West Sussex: John Wiley & Son, 2014:303–323.
6. Cierny G III. Chronic osteomyelitis: results of treatment. Instr Course Lect 1990;39:495–508.
7. Cierny G III, DiPasquale D. Treatment of chronic infection. J Am Acad Orthop Surg 2006;14:S105–S110.
8. Walenkamp GH, Kleijn LL, de Leeuw M. Osteomyelitis treated with gentamicinPMMA beads: 100 patients followed for 1-12 years. Acta Orthop Scand 1998;69:518– 522.
9. McNally MA, Small JO, Tofighi HG, Mollan RA. Two-stage management of chronic osteomyelitis of the long bones. The Belfast technique. J Bone Joint Surg [Br] 1993;75-B:375–380.
10. Spangehl MJ, Masri BA, O'Connell JX, Duncan CP. Prospective analysis of preoperative and intraoperative investigations for the diagnosis of infection at the sites of two hundred and two revision total hip arthroplasties. J Bone Joint Surg [Am] 1999;81-A:672–683.
11. Krenn V, Morawietz L, Perino G, et al. Revised histopathological consensus classification of joint implant related pathology. Pathol Res Pract 2014;210:7
12. Evans RP, Nelson CL. Gentamicin-impregnated polymethylmethacrylate beads compared with systemic antibiotic therapy in the treatment of chronic osteomyelitis. Clin Orthop Relat Res 1993;295:37–42.
13. Ilizarov GA. The principles of the Ilizarov method. Bull Hosp Jt Dis Orthop Inst 1988;48:1–11.
14. Lazzarini L, Mader JT, Calhoun JH. Osteomyelitis in long bones. J Bone Joint Surg [Am] 2004;86-A:2305–2318.
15. Cho SH, Song HR, Koo KH, Jeong ST, Park YJ. Antibiotic-impregnated cement beads in the treatment of chronic osteomyelitis. Bull Hosp Jt Dis 1997;56:140–144.
16. Tiemann AH, Schmidt HGK, Braunschweig R, Hofmann GO. Strategies for the analysis of osteitic bone defects at the diaphysis of long bones. Strategies Trauma Limb Reconstr 2009;4:13–18.
17. Diefenbeck M, Mennenga U, Gückel P, et al. Vacuum-assisted closure therapy for the treatment of acute postoperative osteomyelitis. Z Orthop Unfall 2011;149:336–341. (In German).
18. Timmers MS, Graafland N, Bernards AT, et al. Negative pressure wound treatment with polyvinyl alcohol foam and polyhexanide antiseptic solution instillation in posttraumatic osteomyelitis. Wound Repair Regen 2009;17:278–286.
19. Branstetter JG, Jackson SR, Haggard WO, Richelsoph KC, Wenke JC. Locally administered antibiotics in wounds in a limb. J Bone Joint Surg [Br] 2009;91-B:1106– 1109.

20. Rand BC, Penn-Barwell JG, Wenke JC. Combined local and systemic antibiotic delivery improves eradication of wound contamination: an animal experimental model of contaminated fracture. Bone Joint J 2015;97-B:1423–1427. Gogia J.S., Meehan J.P., Di Cesare P.E., Jamali A.A. Local antibiotic therapy in osteomyelitis. Semin Plast Surg. 2009;23(2):100–107.
21. McNally M.A., Ferguson J.Y., Lau A.C. Single-stage treatment of chronic osteomyelitis with a new absorbable, gentamicin-loaded, calcium sulphate/hydroxyapatite biocomposite: a prospective series of 100 cases. Bone Joint Lett J. 2016;98-B(9):1289–1296..
22. Buchholz H.W., Engelbrecht H. Uber die depotwirkung einiger antibiotica bei vermischung mit dem kunstharz Palacos. Chirurg. 1970;41(11):511–515.
23. Ferguson J.Y., Dudareva M., Riley N.D., Stubbs D., Atkins B.L., McNally M.A. The use of a biodegradable antibiotic-loaded calcium sulphate carrier containing tobramycin for the treatment of chronic osteomyelitis: a series of 195 cases. Bone Joint Lett J. 2014;96-B:829–836.

SP-6

A Clinical and Functional Outcome of Bisphosphonate Therapy in Management of Osteoporosis- A Short Term Study

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Abstract: Osteoporosis is a silent disease and without prevention and screening, the cost of osteoporotic fracture related morbidly and mortality is a concern and quite often the treatment is initiated after the fracture has occurred. The aim of this study was to assess osteoporosis and treat the high risk patients with anti-resorptive medications such as bisphosphonates with closed regular monitoring and follow-up clinically with the help of Dual-Energy X-ray Absorptiometry (DEXA for measuring bone mineral density (BMD) Patients were selected from SreeBalaji medical college OPD based on clinical assessment of T-Score of -1 to <- 2.5 SD for bone mineral density as assessed using DXA scan.They were divided into two groups. One group of 25 patients who received a single dose of I.V zolendronic acid infusion yearly, after optimal rehydration and evaluation of Renal and blood parameters annually and other group received oral alendronate 70 mg weekly regimen for 6 months. Increase in BMD as measured by t-score using DXA scan was seen in both group patients- oral alendronate and intravenous zolendronic acid group. In patients receiving oral alendronate, mean T-score of pre-treatment is -2.22, at 12 months is -2.014 and at the end of 24 month is -1.871. Total increase in t-score values was 0.35. In patients receiving iv zolendronic acid mean T-score of pre-treatment is -2.408 at 12 months -2.116, and at 24 month is -1.668. Total increase in t-score values was 0.74. Both alendronate and zolendronate therapy increased mean t-score values as measured by DXA scan and reduced risk of osteroporosis and fragility fractures with zolendronate being almost twice as effective than alendronate in increasing t-score values.

Keywords: Osteoporosis, Dual-Energy X-ray Absorptiometry, Bone Mineral Density

INTRODUCTION

Peak bone mass, which can be defined as the amount of bony tissue present at the end of the skeletal maturation that becomes an important determination for potential osteoporosis.[1]Our bone mass is determined by the age of 30 for rest of our lives[2]. Osteoporosis is characterized by low bone mass, structural deterioration, and porous bone, which are associated with higher fracture risk[3]. Usual high-risk patients are post-menopausal women, who make up the majority of osteoporosis cases[4]. Dual-energy X-ray Absorptiometry (DXA) is the most widely used validated technique to measure Bone mineral density (BMD).[5] BMD is reported as a T-score, defined as the difference in number of standard deviations (SDs) from the mean BMD of a normally distributed, healthy adult reference population; itis expressed as a negative number[6]. The World Health Organization (WHO) defines osteoporosis as a BMD greater than 2.5 SDs below the average. Normal bone is no more than 1 SD below this value, and osteopenia is 1 to 2.5 SD below average. Severe osteoporosis is BMD greater than< - 2.5 SD.[7] Patients with osteopenia and osteroporosis are at a higher risk of fractures which can be prevented with anti-resorptive bisphosphonates therapy which can be clinically monitored with help of non-invasive quick reliable BMD monitoring. Role of bisphosphonates as 1st line of treatment is well documented. It is pyrophosphate analogue that works by inhibiting osteoclastic activity and resorption of bone, there by slowing the deterioration and allowing osteoblastic activity, to increase the BMD.[8] Bisphosphonates binds to bone mineral with no affinity for other tissues. About 40–60% of the dose distributes to bone, the remainder is excreted unchanged in the urine, and there is no substantial metabolism.[9] This preferential uptake into bone affords bisphosphonates a high degree of target organ specificity. As in 3 generations of bisphosphonates, Alendronate is the 2nd generation orally consumed in 5-10mg OD or 35-70 mg weekly regimen. Whereas Zolendronate is the 3rd generation Intravenous 5mg slow infusion after optimal rehydration administered annually. Both the medications are given after the general parameters of Renal function test, Serum calcium, phosphorus and serum electrolytes are found to be normal. Long term therapy has shown the specific side effects like esophageal irritation, acute phase response, osteonecrosis of the Jaw and atypical femur fracture[10]. These complications can be decreased with proper care and appropriate patient selections. To assess the value of monitoring response to bisphosphonate comparing pre-& post treatment clinically measuring BMD T-scores from DXA scan.

MATERIALS AND METHODS

Subjects

34 Patients were selected from SreeBalaji medical college OPD based on clinical assessment of T-Score of -1 to <- 2.5 SD for bone mineral density as assessed using DXA scan.

Study design

Subjects were clinically evaluated for secondary causes of osteoporosis and were checked for any abnormal blood parameters. They were divided into two groups(table 1). One group of 25 patients who received a single dose of I.V zolendronic acid infusion yearly, after optimal rehydration and evaluation of Renal and blood parameters annually and other group received oral alendronate 70 mg weekly regimen for 6 months yearly advised to be taken on empty stomach, sitting upright position for at

least 30mins after optimal oral rehydration . Both received calcium carbonate 500 mg/day with vitamin D 400 I.U/ day for three months along with primary treatment.

Table 1: Two study groups based on treatment received

Groups	Treatment received	Number of patients
1	Alandronate treatment	7
2	Zolondronate treatment	25

Outcome Measurements

Serial T-score measurements were done at 12 and 24 months using DEXA clinically.

RESULTS

The change of BMD T-Score after 12 and 24 months of therapy was compared with the BMD T-score values before treatment.

MEAN CHANGE IN BOTH GROUPS

The mean BMD T-score values of both groups together before treatment was -2.36, the mean BMD T-score after 12 months of both groups together improved to -2.09 and after 24 months further improved to -1.71 (Table 2). Thus there was a net improvement of 0.65 due to treatment with alendronate and zolendronic acid. This was compared statistically with a paired t-test & was found to be statistically significant with a p value of <0.05.

Table 2: Mean BMD T-score values before treatment, 12 & 24 months after treatment of both groups together

	preT-Score	T-Scorepost 12 months	T SCORE post 24 months	Age
Number of patients	32	32	32	32
Mean	-2.3688	-2.0938	-1.7125	64.53
Std. Deviation	.43436	.29286	1.00923	9.239
Range	2.00	1.20	4.50	35
Minimum	-3.90	-2.80	-2.60	45
Maximum	-1.90	-1.60	1.90	80

ALENDRONATE VS ZOLENDRONATE GROUPS

Higher increase in BMD was seen in group 1 patients who were treated with I.V Zolendronic acid rather than Oral Alendronate. In patients receiving oral alendronate, mean T-score of pre-treatment is -2.22, at 12 months is -2.014 and at the end of 24 month is -1.871. Total increase in t-score values was 0.35. In patients receiving iv zolendronic acid mean T-score of pre-treatment is -2.408 at 12 months -2.116, and at 24 month is -1.668. Total increase in t-score values was 0.74. Both alendronate and zolendronate therapy increased mean t-score values as measured by DXA scan and reduced risk of osteroporosis and fragility fractures with zolendronate being almost twice as effective than alendronate in increasing t-score values. (figure 1 & 2)

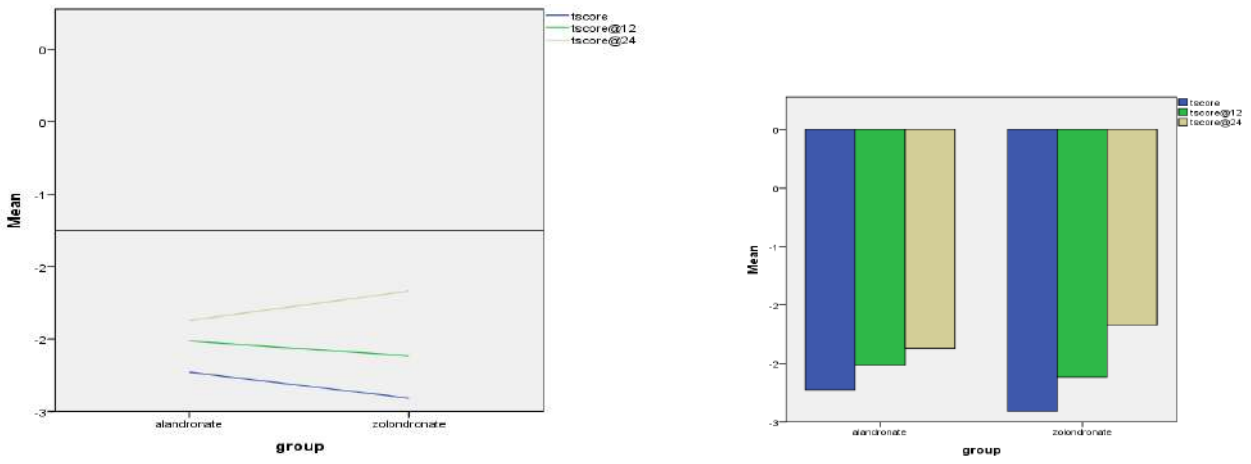

Figure 1:increase in BMD t-score of alendronate and zolendronate at 12 & 24 months, withhigher increase at both 12 & 24 months from zolendronate.

Here line graph is used to represent the change in T-score of two injection treatment Alandronate and Zolondronate from baseline to follow-up of the treatment to 24month.

There is improvement of mean T-score from pre to post treatment. The mean T-score is -2.36 before treatment, it changes to -2.09 at 12th month and it reaches to -1.712

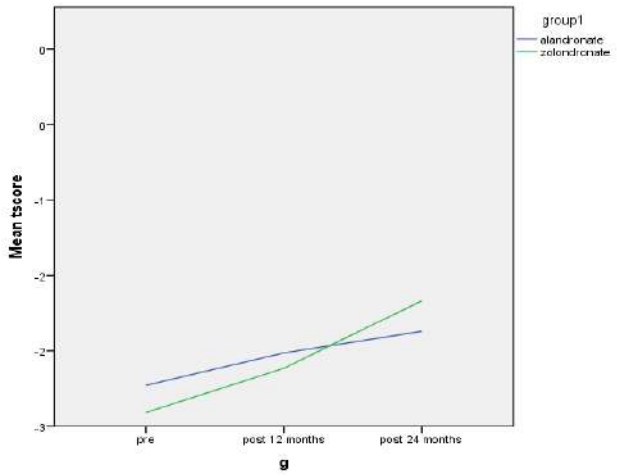

Figure 2: Line graph is used to represent the change in T-score with Alandronate and Zolondronate treatment from baseline to follow-up of the treatment to 24month.

Compliance & complications

Compliance for oral bisphosphonates was determined by the tablet counts during follow-up at every 3 months period. Compliance was around 95%. Iv bisphosphonate had compliance of 100% which was administered under monitoring. Thirty four patients had base line measurement of T-score of <-2.5 SD at the beginning of the treatment. A repeat T – score done at the end of 12 & 24 months showed minimum significance in the BMD and did not show any further deterioration either. None of the subjects incurred any type of fragility fracture due to self-fall throughout the treatment.

DISCUSSION

Osteoporosis as a silent disease that if not detected and treated early and serious complications like fragility fractures for the patients.[11] The prevalence of osteoporosis and thus fragility fractures is going to increase in coming time with ageing global population.[12] Osteoporosis associated fragility fractures increases the morbidity and mortality of the elderly population.[13] Therefore there is an urgent need for early detection and treatment of osteroporosis as this can prevent complications like fragility fractures and increase the quality of life of the patients.Dual-Energy X-ray Absorptiometry (DXA) scan is an excellent modality for early detection of osteoporosis.[14] It is an non-invasive test and can be performed on outpatient basis for early detection of osteoporosis.It is also helpful in management of therapy especially as a reliable modality to assess the efficacy of the treatment and follow-up.[15] Bisphosphonates can be used to treat osteoporosis which can be used as a short term therapy with just ones yearly treatment or weekly regimen that can be tailored accordingly [16, 17] The condition picked up early can be treated efficiently with minimum efforts for both the patients and doctors. In our study, bisphosphonates both alendronate and zolendronate produced an increase in bone mineral density at 24 months of follow-up.These results confirm the important role played by bisphosphonates in treatment of osteoporosis and preventing fragility fractures.[18] Once the T-score is near normal, drug holiday can be initiated with regular follow ups and BMD monitoring which can also be used as a self-assessment tool.

CONCLUSION

Osteoporotic treatment with Bisphosphonates reduces the fracture risk and is recommended for patients with T-score of <-2.5 SD. BMD using DEXA is economical, cost effective and reliable measurement that can be utilized as a drug monitoring tool that is significant in a long term usage. Bisphosphonates are generally well tolerated and are considered first line treatment modality to reduce the fracture risk thereby improving the functional quality as well.

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CONFLICT OF INTEREST

Conflict of interest declared none.

REFERENCES

1. Rizzoli R, Bonjour JP. Determinants of peak bone mass acquisition. InOsteoporosis 2020 (pp. 115-137). Humana, Cham.

2. Lu J, Shin Y, Yen MS, Sun SS. Peak Bone Mass and Patterns of Change in Total Bone Mineral Density and Bone Mineral Contents From Childhood Into Young Adulthood. J ClinDensitom. 2016;19(2):180-191.
3. Obid P, Conta A, Drees P, Joechel P, Niemeyer T, Schütz N. Minimally invasive lumbopelvic stabilization of sacral fragility fractures in immobilized geriatric patients: feasibility and early return to mobility. Archives of Orthopaedic and Trauma Surgery. 2020 Sep 26:1-6.
4. Ji MX, Yu Q. Primary osteoporosis in postmenopausal women. Chronic Dis Transl Med. 2015;1(1):9-13. Published 2015 Mar 21.
5. Who Scientific Group On The Assessment Of Osteoporosis At Primary Health Care Level Summary Meeting Report Brussels, Belgium, 5-7 May 2004.
6. Xue S, Zhang Y, Qiao W, Zhao Q, Guo D, Li B, Shen X, Feng L, Huang F, Wang N, Oumer KS, Getachew CT, Yang S. An Updated Reference for Calculating Bone Mineral Density T-Scores. J ClinEndocrinolMetab. 2021 Jun 16;106(7):e2613-e2621.
7. World Health Organization (WHO) Study Group. Assessment of fracture risk and its application to screening for postmenopausal osteoporosis. Report No. 843. Geneva, Switzerland: World Health Organization, 1994;1–134.
8. Kinoshita M, Ishijima M, Kaneko H, Liu L, Nagao M, Sadatsuki R, Hada S, Arita H, Aoki T, Yamanaka M, Nojiri H, Sakamoto Y, Tokita A, Kaneko K. The increase in bone mineral density by bisphosphonate with active vitamin D analog is associated with the serum calcium level within the reference interval in postmenopausal osteoporosis. Mod Rheumatol. 2019 Jan;29(1):157-164.
9. Cremers S, Papapoulos S. Pharmacology of bisphosphonates. Bone 2011; 49:42–9.
10. Kennel KA, Drake MT. Adverse effects of bisphosphonates: implications for osteoporosis management. Mayo Clin Proc. 2009;84(7):632-638.
11. Liscum B. Osteoporosis: the silent disease. OrthopNurs. 1992 Jul-Aug;11(4):21-5.
12. Sànchez-Riera L, Wilson N, Kamalaraj N, Nolla JM, Kok C, Li Y, Macara M, Norman R, Chen JS, Smith EU, Sambrook PN, Hernández CS, Woolf A, March L. Osteoporosis and fragility fractures. Best Pract Res ClinRheumatol. 2010 Dec;24(6):793-810.
13. Sànchez-Riera L, Carnahan E, Vos T, Veerman L, Norman R, Lim SS, Hoy D, Smith E, Wilson N, Nolla JM, Chen JS, Macara M, Kamalaraj N, Li Y, Kok C, Santos-Hernández C, March L. The global burden attributable to low bone mineral density. Ann Rheum Dis. 2014 Sep;73(9):1635-45. doi: 10.1136/annrheumdis-2013-204320. Epub 2014 Apr 1.
14. Makhdoom A, Rahopoto M, Siddiqui KA, Qureshi GA. Early Detection of Osteoporosis by Dual Energy X-ray Absorptiometry. Pak J Med Sci. 2014;30(6):1265-1269.
15. Dennis M Black 1, Pierre D Delmas, Richard Eastell, Ian R Reid, Steven Boonen, Jane A Cauley, Felicia Cosman, PéterLakatos, Ping Chung Leung, Zulema Man, Carlos Mautalen, Peter Mesenbrink, Huilin Hu, John Caminis, Karen Tong, Theresa Rosario-Jansen, Joel Krasnow, Trisha F Hue, Deborah Sellmeyer, Erik Fink Eriksen, Steven R Cummings, HORIZON Pivotal Fracture Trial Pivotal. Once-yearly zoledronic acid for treatment of postmenopausal osteoporosis. N Engl J Med. 2007;358:1809–1822.
16. Anderson FH, Francis RM, Peaston RT, Wastell HJ. Androgen supplementation in eugonadal men with osteoporosis: Effects of six months treatment on markers of bone formation and resorption. J Bone Miner Res. 1997; 12:472–478.
17. Liberman U, Weiss S, Broll J, Minne HW, Quan H, Bell NH, Rodriguez-Portales J, Downs RW Jr, Dequeker J, Favus M. Effect of oral alendronate on bone mineral density and the incidence of fractures. N Engl J Med.1995;333(22):1437-43.
18. Watts NB, Lewiecki EM, Bonnick SL, Laster AJ, Binkley N, Blank RD, Geusens PP, Miller PD, Petak SM, Recker RR, Saag KG, Schousboe J, Siris ES, Bilezikian JP. Clinical value of monitoring BMD in patients treated with bisphosphonates for osteoporosis. J Bone Miner Res. 2009 Oct; 24(10):1643-6. doi: 10.1359/jbmr.090818. PMID: 19712042.

SP-7

A Complex Case of a Segmental and Comminuted Fracture of Shaft of Femur with Ipsilateral Neck of Femur Fracture and Associated Patellar Fracture

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Abstract: Many treatment protocols are being followed based on the fracture pattern and the level of the fracture and there is no singe consensus regarding the type of implant that needs to be used in these complex situations. The conundrum faced by surgeons in the treatment of multifocal level femur fractures is that different surgical constructs are used for the individual treatment at each level of the femur, but one implant which may be preferred for one region and might not be the choice of fixation at another level. Along with this is the difficulty posed to the surgeons in choosing the order of fixation. In this case study we have used a single Femoral Long reconstruction nail as the implant of choice along with an cerclage wiring for the patella.

Keywords: Femur fractures, Femoral long reconstruction, cerclage wiring, patella, fixation.

INTRODUCTION

Rare is the occurrence of a multilevel fracture of femur involving the segmental shaft of femur with ipsilateral neck of femur fracture and an associated fracture of patella of the same limb. Femoral shaft fractures occur usually as an isolated injury, but 1-9% of the cases they are associated with a neck fracture.[1,2] Around 10-20% of such occurrences, the neck fracture is usually missed. This type of trauma was first described in 1993 by Kach.[3] The injury is often the result of high energy trauma such as motor vehicle accidents, falls from height, and pedestrian versus motor vehicle accidents and usually occurs in young patients.[4,5]

CASE REPORT

31/M patient sustained a road traffic accident 2-wheeler vs lorry, patient had hit a lorry and fell into the gutter and hit his right knee on a concrete wall and presented to the emergency department of Sree Balaji Medical college and hospital 4 hours after the injury. The patient was evaluated at the emergency department, general condition was stable, Vitals Were found to be normal, head injury and abdominal injury was ruled out, Right thigh severe swelling and deformity noted. There was an associated right knee swelling and an open wound around the anterior aspect of the knee measuring around 4X3 cm, which was contaminated. Patient was evaluated for other injuries. There was no neuro vascular injury in the affected limb. Trauma series X-rays were taken and was diagnosed to have severely comminuted and segmented femur fracture almost involving the entire femur from the sub trochanteric area to the distal metaphysis of the femur. There was an associated un-displaced neck of femur fracture and a comminuted patella fracture. There were no other bony injuries noted. Wound wash was given with 3 litres of Saline for the wound and thorough debridement was done at the emergency ward and dressing was applied. Temporary splinting was done to immobilise the limb and was planned for a CT scan of the affected hip and the thigh area to evaluate the fracture fragments and plan for surgery. Patient was shifted to the operation theatre after suitable pre op planning and investigations. The patient was planned for a single implant procedure for the entire femur fracture. A long reconstruction nail was decided as the implant of choice. Patient was positioned on a traction table under epidural anaesthesia. Using a lateral approach entry was taken under C - arm guidance through the tip pf greater trochanter. The guide wire was passed into the sub trochantericregion. Considering the segmentation, comminution and displacement of the fracture fragents incision was put near the major fragments to manipulate the fragments and hold the reduction using SS wires in the form of a cerclage fixation. Gradually the guide wire was passed under the C arm guidance. Reaming was limited only to the entry area of the nail. Long recon nail of size 42*10mm was inserted and guided cautiously into the multiple segmented fracture fragments. To avoid rotation of the fragments Schanz pin 4.5mm was used to hold the intermediate fragments.

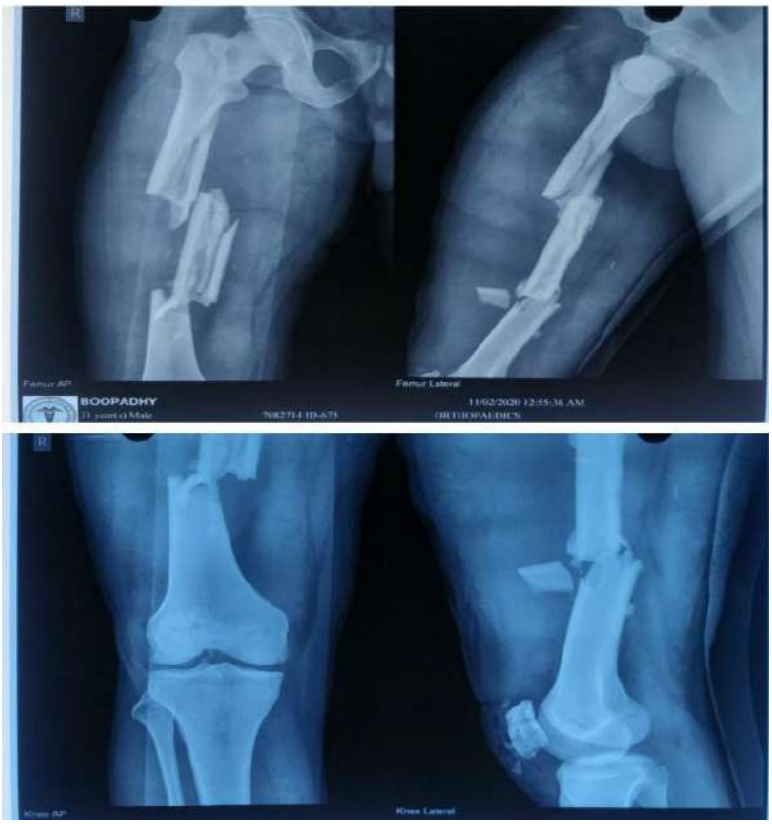

Fig .1 comminuted and segmented femur fracture

The neck of femur fracture was undisplaced and hence the neck screw was applied to hold the reduction. Locking bolts 2 nos were applied distally of sizes 45mm and 40mm. The knee was debrided and thorough wound wash given, the wound was contaminated and hence the patella procedure was deferred for 5 days. Dressing was applied and above knee slab was applied in extension for immobilising the limb. Immediate postoperative period was uneventful. Parentral antibiotics were administered and suitable analgesia advised according to the pain threshold. Once the wound was clean after serial debridement. Patient was planned for Circlage fixation of the patella.

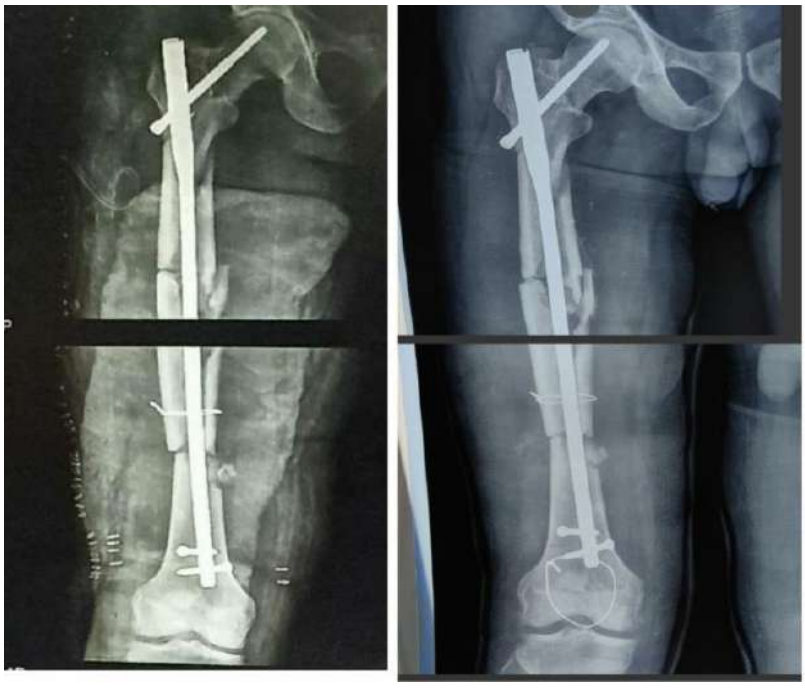

Fig.2 Long recon nail of size 42*10mm

Immediate Post Op Xray ***8 Weeks Follow Up***

Postoperatively patient was advised for strict non-weight bearing for 6 weeks. Gradual knee mobilisation was started after 3 weeks. Teriparatide injection was given for3 months to facilitate fracture union.

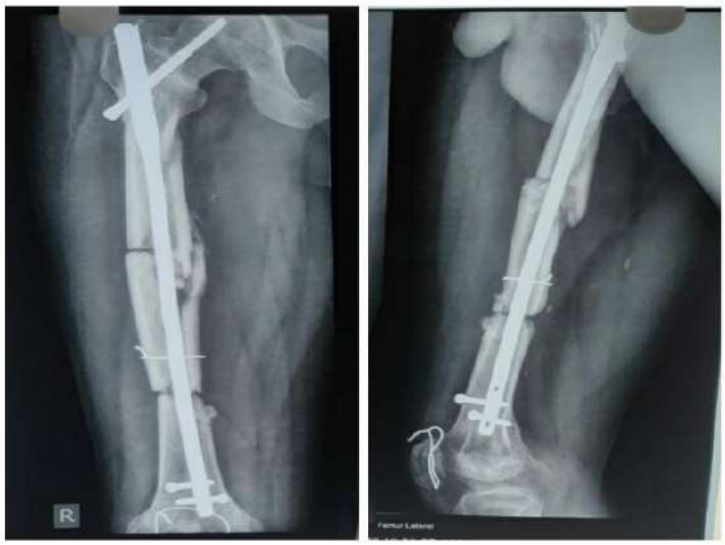

FIG.3. 6 Months follow up

At six months follow up currently patient is able to weight bear without support, callous formation is noted at multiple places in the femur shaft in the Follow up Xray. Neck of femur and patella fracture were found to be united. A secondary Bone grafting procedure was advised to the patient to improve the rate of union and to facilitate complete fracture union, but the patient was not willing for the procedure as he was able to take care of his day to day activities functionally and walk without any discomfort.

RESULTS

Range of Motion at 6 months
Hip flexion – 80 degrees
Hip Extension – 20 degrees
Hip abduction – 40 degrees
Hip adduction - 30 degrees
Knee flexion - 35 degrees

Patient has got back to his work and is active and able to take care of his day to day activities. He is following physiotherapy for improving knee range of motion. The knee open wound had healed well, the patient was functionally happy and there was no pain.

DISCUSSION

With the increase in incidence of high velocity trauma, prompt surgical management of multilevel long bone fracture is the need of the hour. No single method is agreed to be appropriate for fixing multilevel shaft with neck of femur fractures.[1]
In a recent paper, Tsai et al.[6] recommend against the use of cannulated screws with antegrade intramedullary nailing in ipsilateral fractures of femoral neck and shaft; they felt that they were associated with a higher incidence of complications. It is appropriate to use reconstruction nails for osteosynthesis of the proximal femur and femoral shaft, since they can stabilise both fractures very well. There are multiple advantages for fixation of Multi level femoral fracture using a single implant like a long reconstruction nail. They are less soft tissue damage, lesser dissection, minimal loss of blood and less surgical time. Also using multiple implants can cause stress riser in the intermediate fragment which can lead to secondary fractures. The study done by Wang and his associates regarding Usage of cephallomedullary nails for Ipsilateral shaft with neck fracture shows favourable results.[7] The main key to achieve good functional result is to achieve adequate reduction to the neck of femur fracture and provide stability.[8-10] In case of severely displaced femoral neck fractures additional stability might be required. Intracapsular neck fracture usually required additional implant usage. But usually in Femoral neck fractures associated with neck fractures, the commonest type is usually a Basicervical type of Fracture.[11] The order of fixation for managing trifocal injuries has been a matter of debate.[9] Our cases suggest that good outcomes can be achieved with initial treatment of the proximal fracture, followed by fixation of the distal fractureas suggested by Barei et al.,[12] The poor outcome associated with delayed treatment of a shaft of femoral fracture is likely less than that of a proximal fracture with the potentially devastating complication of avascular necrosis of the femoral head.[8] However, as with the choice of implants, the order of fixation should be dictated by surgeon's preference, equipment availability, and fracture configuration.[13] Our patient underwent a single implant procedure as we believed in decreasing the hardware usage due to the severely comminuted and extensive nature of fracture pattern. We hoped to provide a better chance for fracture union by using a minimally invasive procedure and lesser implants, along with a delayed patella fracture fixation and prolonged immobilisation. Our patient was followed up for 6 months and found to have adequate fracture union with good range of movement and minimal to nil pain while performing activities of daily living.[14-16]

CONCLUSION

Multi-level femur fractures with femoral neck fracture and patella fracture is a very rare and complex injury and there Is no consensus on any single mode of treatment. We had used a single long reconstruction nail for the fixation and were able to achieve satisfactory union and Functional recovery in this case. As done in this case adequate pre-op imaging and planning, immediate surgical fixation with a minimally invasive approach and restricting the hardware usage to minimum, proper reduction of the neck of femur fracture are the key to achieve good outcome and along with an early mobilisation protocol after an adequate period of immobilisation will ensure a good post-operative outcome clinically, radiologically and functionally.

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CONFLICT OF INTEREST

Conflict of interest declared none.

REFERENCES

1. Wu KT, Lin SJ, Chou YC, Cheng HH, Wen PC, Lin CH, Yeh WL. Ipsilateral femoral neck and shaft fractures fixation with proximal femoral nail antirotation II (PFNA II): technical note and cases series. Journal of orthopaedic surgery and research. 2020 Dec;15(1):1-7.
2. Kang L, Liu H, Ding Z, Ding Y, Hu W, Wu J. Ipsilateral proximal and shaft femoral fractures treated with bridge-link type combined fixation system. Journal of Orthopaedic Surgery and Research. 2020 Dec;15(1):1-0.
3. Angelini A, Mavrogenis AF, Crimì A, Georgoulis J, Sioutis S, Bekos A, Igoumenou VG, Cerchiaro MC, Berizzi A, Ruggieri P. Double fractures of the femur: a review of 16 patients. European Journal of Orthopaedic Surgery & Traumatology. 2021 Jan 26:1-0.
4. Barei DP, Schildhauer TA, Nork SE. Noncontiguous fractures of the femoral neck, femoral shaft and distal femur. J Trauma. 2003;55:80–86.
5. Papaioannou I, Baikousis A, Korovessis P. Trifocal Femoral Fracture Treated With an Intramedullary Nail Accompanied With Compression Bolts and Lag Screws: Case Presentation and Literature Review. Cureus. 2020 May;12(5).
6. Tsai CH, Hsu HC, Fong YC, Lin CJ, Chen YH, CJ HSU. Treatment for ipsilateral fractures of femoral neck and shaft. Injury. 2009;40:778–782.
7. Wang WY, Liu L, Wang GL, Fang Y, Yang TF. Ipsilateral basicervical femoral neck and shaft fractures treated with long proximal femoral nail antirotation or various plate combinations: comparative study. J Orthop Sci. 2010;15:323–30.
8. Bennett FS, Zinar DM, Kilgus DJ. Ipsilateral hip and femoral shaft fractures. ClinOrthopRelat Res. 1993;296:168–77.
9. Griffin, Michelle; Dick, Alastair G; Umarji, Shamim. Outcomes after Trifocal Femoral Fractures. Case Reports in Surgery; New York Vol. 2014. DOI:10.1155/2014/528061
10. Yanbin, Lin, Renbin Li, Yan et al. Treatment of middle up part long-segment femoral fracture with long PFN A: JPMA. 2014.
11. Park J, Yang KH. Correction of malalignment in proximal femoral nailing reduction technique of displaced proximal fragment. 2010; 41(6):634-8.
12. Zheng ZL, Yu X, Xu GQ et al. Four pins assisted reduction of complex segmental femoral fractures; a technique for closed reduction. J Huazhong univ sci technolog med sci. 2014; 34(6):912-916.
13. Shukla S, Johnston P, Ahmad MA et al. Outcome of traumatic subtrochanteric femoral fractures fixed using cephalo-medullary nails. 2007: 38(11):1286-93.
14. Chen CH, Chen TB, Cheng YM et al. Ipsilateral fractures of the femoral neck and shaft injury. 2000;31(9):719-22.
15. Wu c, Chen W: Healing of 56 segmental femoral shaft fractures after locked nailig. Acta orthop scand 68:1997,pp.494-500.
16. Sojbjerg J, Eiskjaer S, Moller-larsen F: Locking nailing of communited and unstable fractures of femur. J Bone joint surg Br 72:1990, pp.23-25.

SP-8

Malunited Proximal Ulna Fracture and Radial Head Dislocation

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Abstract: Neglected Monteggia fracture dislocation is one of the challenging case and it becomes more of a therapeutic dilemma especially in adults. Though several surgical techniques are described, the complication rate following surgery is high.
We report a case of 17yrs old male with alleged history of slip and fall 3 months back and sustained injury to the left forearm. Patient went for native treatment. Now the patient came with complaints of restriction of movements and deformity of left forearm for past 1month, He was diagnosed asBado's type IMonteggia fracture-dislocation. Patient was managed surgically. Ulnar fracture was fixed with Dynamic compression plate (DCP) followed by reduction of radial head was done. The dilemma remains whether radial head preservation or excision gives better functional range of motion in adult neglected monteggia fracture dislocation. Very few reports of chronic monteggia in adults have been published and they have described different management options. In our case, posterior subcutaneous approach was used to fix the ulna with a plateand extendedkochers approach to the previous incision was used to reduce the radial head .Ulna was not lengthened and plate was applied in compression mode. Treatment of chronic radial head dislocation is still controversial. In adults where no growth or remodelling potential is left, procedures like fixation of ulna in angulation, ulna lengthening is not required

Keywords: Neglect, Monteggia, DCP

INTRODUCTION

Monteggia fracture is a rare fracture and it is observed in only 0.4% of all forearm fractures.[1] The condition is named after Giovanni Battista Monteggia.He reported 2 patients with fractures of the proximal third of the ulna with anterior dislocation of the radial head in 1814.[2] The modern definition of the Monteggiafracture includes any fracture of the forearm with dislocation of the proximal radioulnar joint.These lesions are further classified in accordance with the Bado classification system.[3] Proximal ulna fractures range in severity from simple olecranon fractures to severe Monteggia fractures, which involve damage to the elbow's stabilising components (i.e. coronoid process, radial head).[4] Upper extremity fractures can occur at any age, but they are most common in adults over the age of seventy. Anatomical restoration of ulnar alignment must be the primary goal of surgical treatment for restoring unrestricted elbow function (in length, rotation, and axis). As a result, in order to assist early (active) rehabilitation and avoid elbow stiffness, the surgeon must carefully address all aspects of the injury. An incorrect osseous reconstruction of the ulna, as well as a failed/missed reattachment of elbow stabilising tissues, would result in persistent pain, poor function, and progressive joint degeneration due to prolonged elbow in stability.[5] As a result, efficient treatment of proximal ulna fractures remains a challenge for orthopaedic surgeons. The purpose of this review paper is to demonstrate how to correctly treat these injuries using modern osteosynthetic implants and procedures while taking into account the elbow joint's specific biomechanics.

CASE PRESENTATION

17-year-old male presented with a complaint of restriction of movement and deformity of the left forearm for the past one month; patient has non pathological alleged history of a slip and fall three months prior and sustained injury to the forearm; patient received native treatment; patient now presented to Sree Balaji Medical College and Hospital for further management.
On examination of the left forearm, there is no tenderness, range of movement of the left elbow is limited to 0-90 degrees of flexion, extension, supination full and free, pronation 0-90 degrees, all finger movements are normal, and shoulder movements are normal. Radiography revealed anterior dislocation of the radius and malunited of the proximal 1/3rd of the shaft of the ulna (Figure 1) This fracture-dislocation resembled type-1 bado's classification of monteggia because of the fracture of the proximal third of the ulna and anterior dislocation of the head of the radius. The procedure was performed the next day, with the patient in a supine posture with his left upper limb held in place on an arm board. under Axillary block and ASP, Through the posterior subcutaneous approach, a 15cm midline incision is made just distal to the olecranon and up to the mid forearm. Cut and retracted skin and subcutaneous tissues (Figure 2) An osteotome was used to shatter the callus and shape the bone after a fracture site with callus was identified.

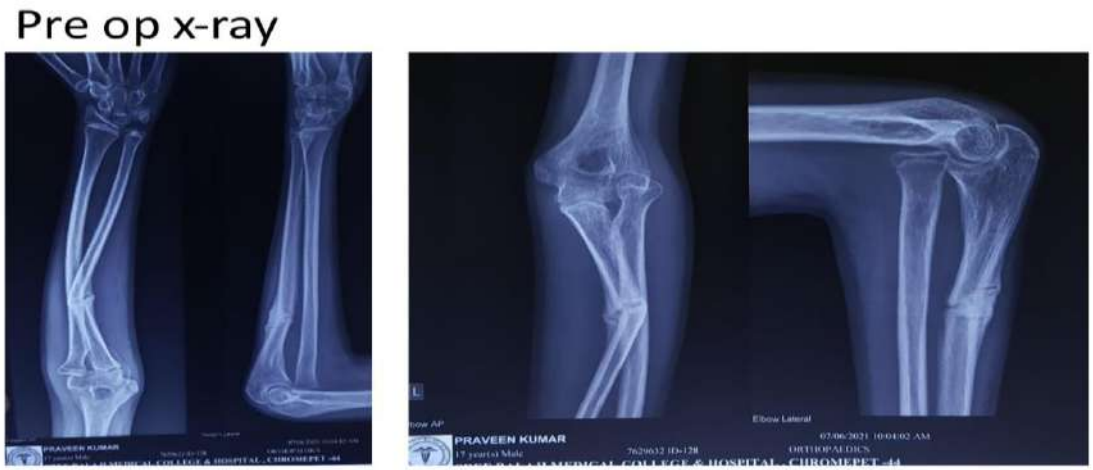

Figure 1- Anteroposterior and lateral view of left full length forearm and elbow showing proximal 1/3 rd ulna fracture , displaced laterally and radial head dislocated anteriorly.

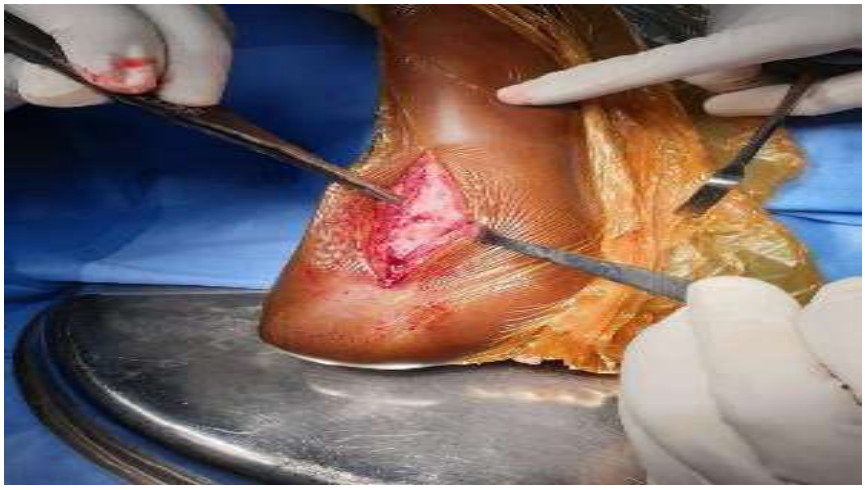

Figure -2 intra operative picture showing malunited ulna bone after subcutaneous incision

To reduce the fracture, a 7holed Asian DCP was used with a bone clamp and 3proximal and 3 distal screws were used. The radial head was exposed, soft tissue adhesions were released, the annular ligament was cut, and the radial head was reduced through an extended kochers approach to this previous incision. To reinforce the radial head in its reduced position, a tendon constraint was used. After a thorough wound wash, the skin was closed in layers, the drain was kept in place and functioning, a sterile dressing was applied, and an Above elbow slap applied ,post operative radiological follow up don(Figure 3) After two weeks, the AE slap was removed, elbow range of movement was initiated.

Figure-3 post operative anteroposterior x ray of full length forearm and elbow joint

DISCUSSION

Proximal ulna fracture with anterior dislocation of radial head was described by Monteggia GB in 1814. In 1909, Perin J described these patterns of fractures as monteggia fracture dislocation[6]. FurtherBado JL classified it into four types according to angulation of the ulna fracture and direction of the radial head dislocation. Bado's type II is more common (80%) in adults. In children Bado'sType I lesions are more common .In this case report, we describe our experience of managing neglected

monteggia fracture dislocation.[6] All Monteggia fractures are considered unbalanced and need intervention. Emergent orthopedic consultation is essential for open fractures and vascular compromise. Urgent orthopedic consultation is indicated for neurologic deficits without vascular compromise. Monteggia fractures and dislocations are divided into four categories by Bado et al.,[7] (Figure-4)

Type I ulna fracture involving the proximal two-thirds of the ulna, with anterior dislocation of the radial head and anterior angulation of the ulna.
Type 2 ulna proximal two-thirds Fracture with angulated posteriorly, posterior dislocation, and radial head fracture.
Type 3 -Ulna Fracture with lateral dislocation of the radial head just distal to the coronoid process.
Type 4: proximal two-thirds ulna fracture, anterior dislocation of radial head, and proximal one-third radial bone fracture.

In this case fracture occurred as a result of a slip and fall accident, and it is similar to type I fractures.

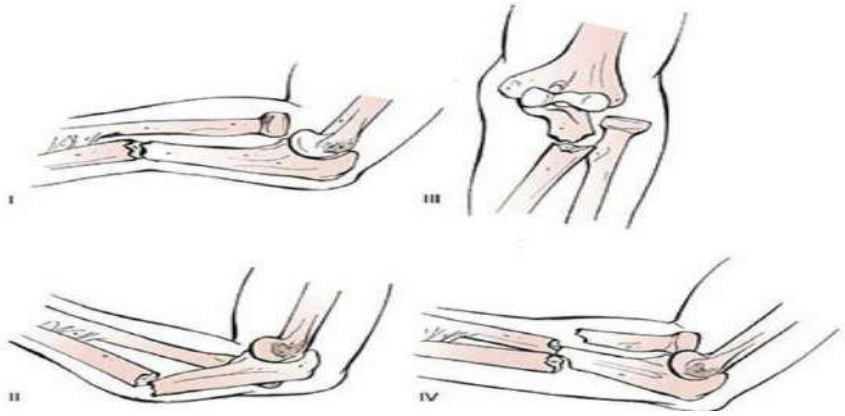

Figure-4 bado's classification showing four types of monttegia fracture

Very few reports of chronic monteggia in adults have been published and they have described different management options. In our case, posterior subcutaneous approach was used to fix the ulna with a plateand extendedkochers approach to the previous incision was used to reduce the radial head .Ulna was not lengthened and plate was applied in compression mode. Treatment of chronic radial head dislocation is still controversial. Chronic monteggialesions are managed with many techniques like ligament reconstruction and ulnar osteotomy, In complex monteggia fracture dislocations, the proximal ulna including the olecranon and coronoid may require reconstruction. One of the most important factors which would affect the functional outcome is ulna length. This is becauseduring supination and pronation radius and ulna act as one unit, Hence in management of monteggia fracture dislocation, ulnar osteotomy plays an important role.In type I and IV monteggia fracture dislocation ,three dimensional kinematics of radial head dislocation has been studied in vivo.[8] According to them due to isometricity of the interosseous membrane, radial head was relatively stablein type I lesions. This supported the concept of ulnar bending osteotomy to maintain ulna length. Another topic of debate in management of chronic lesions is annular ligament reconstruction.[8] In a comparative study,those managed with and without annular ligament reconstruction were compared.Itstates that annular ligament reconstruction is not always necessary.It is advised only when there is coronal translation of the radial head. In our case, there was anterior dislocation of radial head.[9] There was no coronal translation of the head after reduction and fixation of ulna intraoperatively. After reduction and fixation of ulna, usuallyradial head gets reduced. But sometimes the radial head does not get reduced, in such cases we have to suspect buttonholing through the annular ligament or anconeus muscle and a separate radial approach is advised. Higher risk of radioulnar synostosis is observed in combined approach to proximal ulna and radius. Posterior interosseous nerve palsy are reported inneglected montaggia fracture dislocationcases.As it is a neuropraxia injury, after anatomical reduction of radial head, this usually recovers.[10] In a cadaveric study, it has been found that ,tension in the interosseous membrane almost doubledafter radial head excision. And it states that the radial head has to be excised only in those monteggiafracture -dislocationswith radial head fracture.[11] The radial head was not excised in our case.In neglected cases,further research neededto find out whether there is any benefit or notin excising the radial head at the time of ulna fixation . But such neglected cases are rare in present day scenario, it is difficult to do a comparative study between two (radial head excised/ not excised) larger groups of patients.

CONCLUSION

A patient with a malunited proximal ulna fracture and head of radial bone dislocation was treated with open reduction for radial head dislocation and open reduction internal fixation with DCP plating for ulna. Three weeks after surgery, the patient showed functional improvements with 120 degrees of flexion, full extension, and an 80 degree pronation and full supination. He had no neuropathic complaints on physical examination, and his median, radial, and ulnar nerves were all intact. With continued treatment, the patient's condition improved even more, allowing for 135 degrees of flexion, complete extension, 90 degrees of pronation, and full supination. In adults,as there is no growth or remodelling potential , procedures like fixation of ulna in angulation, ulna lengthening are not required. The dilemma stillremains whether radial head preservation or excision gives better functional range of motion in adult neglected monteggia fracture dislocation.It requires further research.

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CONFLICT OF INTEREST

Conflict of interest declared none.

REFERENCE

1. A. S. Shah and P. M. Waters, ""Monteggia-fracture dislocation in children," in Rockwood and Wilkins," in Fractures in Children, J. M. Flynn, D. L. Skaggs, and P. M. Waters, Eds., pp. 527–563, Wolters Kluwer, Philadelphia, PA, USA, 2015.
2. G. B. Monteggia, InstituzioneChirurgiche, Maspero, Milan, Italy, 2nd edition, 1814.
3. J. L. Bado, "The Monteggia lesion," Clinical Orthopaedics and Related Research, vol. 50, pp. 71–86, 1967.
4. Siebenlist S, Buchholz A, Braun KF. Fractures of the proximal ulna: current concepts in surgical management. EFORT Open Rev. 2019 Jan 7;4(1):1-9. doi: 10.1302/2058-5241.4.180022. PMID: 30800474; PMCID: PMC6362340.
5. Sanchez-Sotelo J, Morrey M. Complex elbow instability: surgical management of elbow fracture dislocations. EFORT Open Rev 2016;1:183–190.
6. Perrin J. Les fractures du cubitus accompagnees de luxation de l'extremitesuperieur du radius. In: Perrin J, editor. These de Paris. G Steinheil: Paris, France; 1909.
7. Bado JL. The Monteggia lesion. Clin OrthopRelat Res. 1967;50:71–86.
8. Miyake J, Moritomo H, Kataoka T, Murase T, Sugamoto K. In vivo three-dimensional motion analysis of chronic radial head dislocations. Clinical Orthopaedics and Related Research® 2012;470(10):2746–55.
9. Bhaskar A. Missed Monteggia fracture in children: Is annular ligament reconstruction always required? Indian Journal Of Orthopaedics. 2009;43(4):389.
10. Strauch RJ, Rosenwasser MP. The assessment and treatment of nerve dysfunction after trauma around the elbow. Clinical Orthopaedics And Related Research. 2000;370:138–53.
11. Jepegnanam TS. Salvage of the radial head in chronic adult Monteggia fractures. Report of four cases. J Bone Joint Surg Br. 2006;88(5):645–48. [PubMed]

SP-9

A Prospective Study to Analyze the Clinical, Radiological and Functional Outcome of Sanders Type I and Type II Closed Displaced Intra-Articular Calcaneal Fractures Treated Conservatively.

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Abstract: Purpose is to analyse the outcome of Sanders type I and type II closed displaced intra-articular calcaneal fractures treated conservatively. Method used is the prospective analysis of 20 patients with Sanders type 1 and type 2 Calcaneal intra-articular fractures were treated conservatively between the period from August 2018 to September 2020. Patients with age above 21-50 yrs old were chosen as the inclusion criteria and patients with closed intra-articular fractures were chosen for the study. Patients were treated with immobilisation with P.O.P slab and cast and was reviewed periodically in the interval and was assessed at 8 months post injury to evaluate the clinical, radiological and functional outcome with the help of AOFAS scoring. Results obtained were that Sanders type1 and type 2 fractures treated conservatively showed good outcome with AOFAS mean score being 77.25 at 8 months follow-up. All patients were comfortable performing their day to day activity without major issues. Concluding that Calcaneal fractures is a controversial topic for operative and non operative management with very less known outcome to predict the result. This study concludes with a good overall outcome with patients suffering from Sanders type I and type II fractures treated conservatively.

Keywords: Calcaneal fracture, Non-operative management, Sanders classification, Bohler's angle, Gissane's angle.

INTRODUCTION

Calcaneal fractures are about 3% of all the fractures in the body. Out of all the tarsal fractures, calcaneal fractures account for upto 65%, with 70% of being intra articular.[1] Most classical history of calcaneal fractures results from fall from height which leads to the pathology being bilateral in significant individuals with calcaneal fractures. Many of these patients are construction site workers who would be the sole member of the family earning, hence leading to financial, social and mental burden on the family. The optimum management of calcaneal fractures is still not clear. The treatment option varies from conservative management with immobilisation to aggressive open surgical approach. Out of these treatment options carefully choosing patients for surgery should be a key importance in getting a good outcome, hence surgical intervention for calcaneal fractures has increased. From the year 1990 to 2000the debilitating complications has been reduced due to proper intervention.[2] Mechanism of injury: The downward transmission of energy through the talus with the calcaneum attached to the ground leads to inferomedial movement of the sustentacular fragment and the calcaneal tuberosity fragment moves more laterally and gets elevated. The talus pushes the lateral part of posterior facet into the cancellous bone of the fragment with the tuberosity.[3] On the basis of length of the fragment over the supero- lateral aspect with the remaining small fragments and the posterior facet with the articular surface makes the types of the Essex-Lopresti classification. The commonest type being: -Joint depression type. The key presentation of Intra articular calcaneal fractures are fall from height in a young individual age ranging from 25-55 yrs from a height of 6 feet or more than that, can be also seen in lesser velocity in osteoporotic elderly individual.[4,5] Other mode of injuries like motor vehicle collision directly on the heel might cause intra-articular fractures.[6]

METHODS AND MATERIALS

This study was conducted at Sree Balaji Medical College and Hospital, BIHER, Chromepet from August 2018 to September 2020. Patients were classified by Sander's CT classification for displaced Calcaneal intra-articular and the type 1type 2 group was treated conservatively with slab and cast. Most of these cases were a result of a fall from a height. Few had a history of traffic accidents. The cases presented with swelling and heel pain and inability to walk. All patients were evaluated with calcaneal radiography - Axial, lateral and AP views along with computed tomography.

Inclusion criteria

All patients with the age of 21 - 50 years both male and female with traumatic closed displaced intra-articular fractures were involved in the study.

Exclusion criteria

Patients not falling under the above age criteria are excluded. Patients with Pathological fractures due to tumours are excluded. Previous treated calcaneal fractures are excluded from the study. Traumatic conditions presenting after a delay of 2 weeks or those which have undergone native treatment are excluded.

METHOD

Patients were treated with above knee slab for 2 weeks following with 4 weeks of Cast and immobilisation with rest and immobilization.All patients had regular follow-up of 2 weeks, 3 months and 8 months . AOFAS scoring for all patients were done at 8 months to analyse the outcome of the conservative management.The Bohler's angle and Gissane's angle was assessed on day 0 of injury and at 8 months to compare the radiological outcome.The mean score of all the patient's AOFAS score was calculated to find out the collective outcome in conservative management of type I and type II calcaneal fractures.

Observation

Table 1. Patient classification

Treatment modality	Total number
Sander's typeI	10
Sander's typeII	10

Table 2: Sex distribution

Sex	No.
Male	12
Female	8

Table 3: Mode of injury

Mode of injury	No.
Fall from height	16
RTA	4

Table 4: AOFAS scores for individual patients.

Sr.no	Name	Sex	Age	8months follow-upAOFAS score
1	DK	M	28	80
2	SB	M	42	76
3	SS	M	32	75
4	FD	M	33	75
5	AS	M	25	82
6	PH	M	47	78
7	BL	M	26	83
8	LR	M	32	72
9	GH	M	41	78
10	MS	M	44	72
11	RP	M	29	79
12	DE	M	31	85
13	MA	F	34	79
14	TP	F	32	77
15	RS	F	26	84
16	BA	F	48	68
17	CC	F	33	75
18	SV	F	32	80
19	RL	F	34	73
20	FB	F	47	74

Analysis of Data

The data collected at the interval of 8 month follow-up was recorded and the mean of the scores of all 20 patients were taken up to analyse the outcome. The mean value of AOFAS is calculated and found to be 77.25. Hence the mean value falls at a good score with 77.25. Showing good outcome for patients with Sander's type I and II fractures treated conservatively.
The T value calculated for a single T test for our study comes to 58.03 and the p value comes to less than 0.001 which shows good significance of our study.

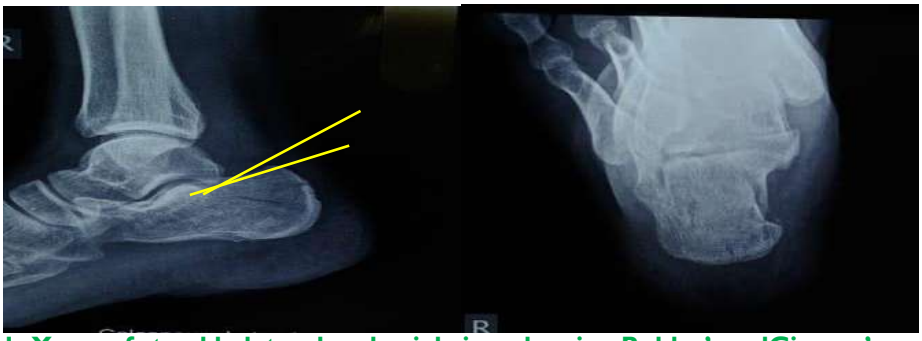

Fig1: X-ray of rt ankle lateral and axial view showing Bohler'sandGissane's angle

Figure 1 shows a decrease in the Bohler's angle of about 12 degrees and axial X-ray showing intra-articular fracture. (Normal 20-40 degrees)

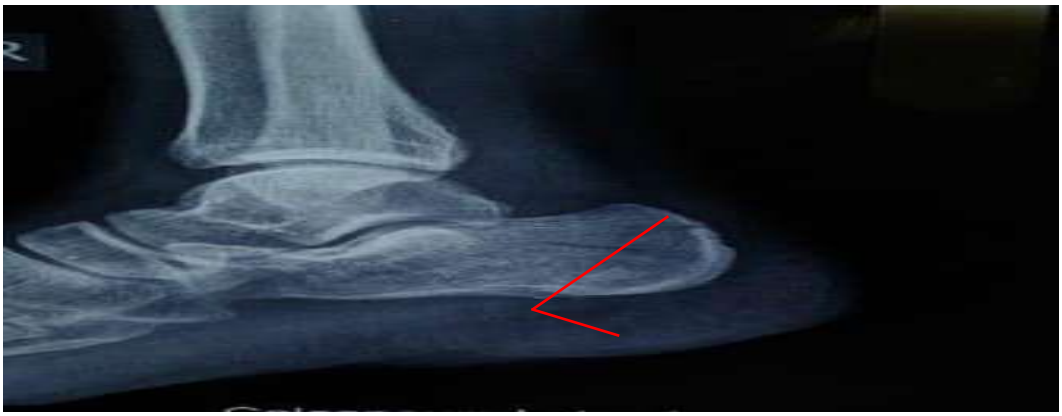

Fig2.: X-ray of rt ankle lateral view.

Fig. 2 describes a decreased F angle of gissane of about 110 degrees. (Normal 130-145degrees)

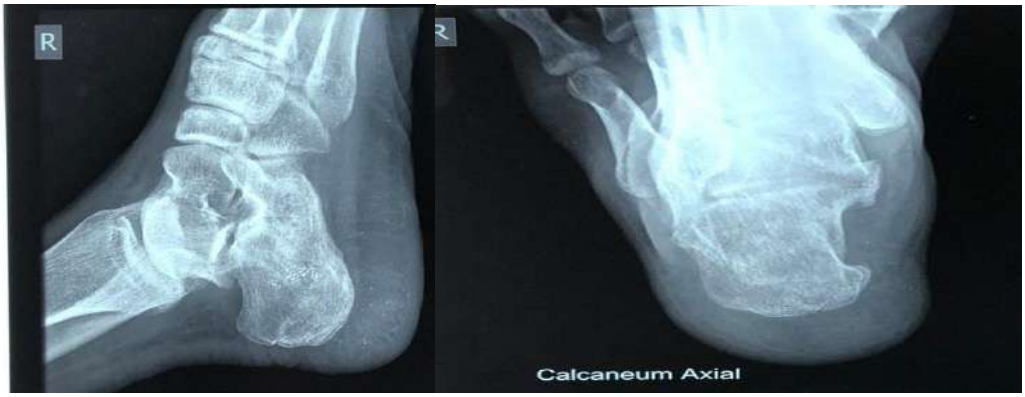

Fig. 3 Eight month post injury X-ray ankle lateral and foot axial view.

Fig. 3 Shows no change in the radiological angles at 8 month follow-up of conservative management.

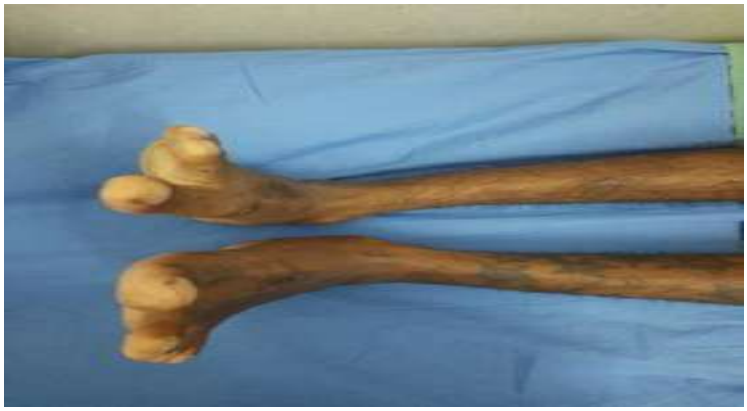

Fig. 5 Eight month clinical outcome.

RESULTS

Calcaneal fractures can be intra-articular and extra-articular. Non-union is not a major complication in calcaneal fractures. Intra-articular fractures are problematic as it causes pain and problems in alignment and causes arthritis leading to decreased motion and function. In past literature due to unavailability of contoured plates, anatomical plates, antiseptic methods and the plate being subcutaneous causes much more skin breakage and infection was a major issue. The newer implant with LCP fashion has a good hold of the fragments and gets malleable according to the shape of the bone and provides a good stable construct. Implant removal post fracture union is the must to get back the maximal outcome. Implant removal should be planned post 12 months of surgery. Our study emphasises the need of conservative management in patients with type 1 and type 2 calcaneal fractures and to restrict the use of surgical management to avoid the debilitating complications arising from calcaneal fractures. Our study shows a mean AOFAS score of more than 75 which is a good clinical outcome for patients with calcaneal fractures.

DISCUSSION

Calcaneal fractures are the commonest fracture in a patient with fall from height and landing directly on the heel. Elderly patients might land up in calcaneal fractures even if the velocity of the injury is too low.[7] In patients at 20-30 yrs old the velocity of injury should be proportionately high to get a calcaneal fracture. This is all due to osteoporosis in the elderly population. Male patients are seen in majority in calcaneal fractures due to their physical activity in the occupation. The following shows several studies conducted worldwide according to AOFAS score. A study done by Palmer in 1948 showed 90% good result in treating calcaneal fractures with conservative management.[8] Mc Reynolds et al. stated 82.5% outcome at 1982 for Sander's type 1 calcaneal fractures.[9,10] Benirschke et al. stated in his study with an outcome of 75.2% in calcaneal fractures falling under Sander's type 1 and type 2 fractures.[11] Thordarson & Krieger et al. had a similar study which showing 75% outcome.[13] Our study shows an outcome of 77.25% for Calcaneal closed intra-articular falling under Sander's type 1 and 2 fractures which was treated conservatively.

CONCLUSION

This concludes help in decision making for each calcaneal fracture whether to operate or to opt for conservative management to provide the patient with optimum final results and to manage with the patients without causing or expecting for other complications which are related to operative management.

ACKNOWLEDGEMENT

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None

CONFLICT OF INTEREST

Conflict of interest declared none.

REFERENCES

1. Fitzgibbons TC, McMullen ST, Mormino MA. Fractures and dislocations of the calcaneum. In: Bucholz RW and Heckman JD Eds Rockwood and Green's Factures in adults, 5th ed. Philadelphia: Lippincott Williams & Wilkins. 2001;3: 2133-2179
2. Rodrigues RC. Traducao, adaptacao cultural e validacao para lingua portugues ao American Orthopaedic Foot and Ankle Society (AOFAS) Ankle-Hindfoot Scale.
3. Asik, M. Sen C. Surgical management of intraarticular fractures of the calcaneus. Arch. Orthop. Trauma. Surg. 2002, Jul: 122(6) : 354-9.
4. Barei, DP, Bellabarba C, Sangeorzan BJ, Benirschke 5K. Fractures of the calcaneus. Orthop. Clin. North Am. 2002 Jan 33(1) : 263-85. p
5. ESSEX-LOPRESTI P: The mechanism, reduction technique, and results in fractures of the oscalcis. Br J Surg. 1952, 39: 395-419. 10.1002/bjs.18003915704.
6. Potter MQ, Nunley JA: Long-term functional outcomes after operative treatment for intra-articular fractures of the calcaneus. J Bone Joint Surg Am. 2009, 91: 1854-1860. 10.2106/JBJS.H.01475.
7. Harvey EJ, Grujic L, Early JS, Benirschke 5K, Sangeorzan BJ. Morbidity associated with ORIF of intra-articular calcaneus fractures using a lateral approach. Foot Ankle Tnt. 2001 Nov. 22(11) 868-73.
8. Illert T, Rammelt S, Drewers T. Stability of locking and non locking plates in an osteoporotic calcaneal fracture model. Foot angle int .2011; 32(3):307-13
9. Blake MH, Owen JR, Sanford TS. Biomechanical evaluation of a locking and non locking reconstruction plate in an osteoporotic calcaneal fracture model. Foot Ankle Int.2011 ;

10. Shen C, Shen Y, DaiL Z, Locking compression treatment for intra articular calcaneal fractures. Zhongguo Gu Shang. 2010;23(3).225-7
11. Stoffel K, Booth G, Rohrl SM. A comparison of conventional versus locking plates in intra articular calcaneal fractures. A biomechanical study in human cedavers. Clin Biomech 2000 ;22(1);100-5
12. Wagner M General principles for the clinical use of the LCP. Injury. 2003;34 suppl2:B31-42
13. Wagner M, Frenk A, Frigg R. New concepts of bone fracture treatment and the locking compression plate. Surgtechnolint 2004 ;12;271-7
14. Epstein N, Chandran S, Chou L. Current concepts review: intra-articular fractures of the calcaneus. Foot & ankle international. 2012 Jan;33(1):79-86.
15. Hansen JMiller MD, Thompson SR, Hart J. Review of Orthopaedics E-Book. Elsevier Health Sciences; 2012 Apr 23.
16. T. Netter's Clinical Anatomy-E-Book. Elsevier Health Sciences; 2022.
17. Sanders R. Current concepts review-displaced intra-articular fractures of the calcaneus. JBJS. 2000 Feb 1;82(2):225-50.
18. Sanders R. Intra-articular fractures of the calcaneus: present state of the art. Journal of orthopaedic trauma. 1992 Jun 1;6(2):252-65.
19. Benirschke SK, Sangeorzan BJ. Extensive intraarticular fractures of the foot. Surgical management of calcaneal fractures. Clinical orthopaedics and related research. 1993 Jul 1(292):128-34.
20. Ballinger PW, Frank ED. Merrill's atlas of radiographic positions and radiologic procedures. Vol. 3. Mosby; 1999.
21. Hildebrand KA, Buckley RE, Mohtadi NG, Faris P. Functional outcome measures after displaced intra-articular calcaneal fractures. The Journal of bone and joint surgery. British volume. 1996 Jan;78(1):119-23.
22. Rammelt S, Sangeorzan BJ, Swords MP. Calcaneal fractures—should we or should we not operate?. Indian journal of orthopaedics. 2018 Jun;52(3):220-30.
23. Li Y, Bao RH, Jiang ZQ, Wu HY. Complications in operative fixation of calcaneal fractures. Pakistan journal of medical sciences. 2016 Jul;32(4):857.
24. Buckley RE, Tough S. Displaced intra-articular calcaneal fractures. JAAOS-Journal of the American Academy of Orthopaedic Surgeons. 2004 May 1;12(3):172-8.
25. Wei N, Zhou Y, Chang W, Zhang Y, Chen W. Displaced intra-articular calcaneal fractures: classification and treatment. Orthopedics. 2017 Nov 1;40(6):e921-9.
26. Swanson SA, Clare MP, Sanders RW. Management of intra-articular fractures of the calcaneus. Foot and ankle clinics. 2008 Dec 1;13(4):659-78.
27. Sanders R. Current concepts review-displaced intra-articular fractures of the calcaneus. JBJS. 2000 Feb 1;82(2):225-50.
28. Sanders R. Intra-articular fractures of the calcaneus: present state of the art. Journal of orthopaedic trauma. 1992 Jun 1;6(2):252-65.

SP-10

Collagen Patch Repair of Tympanic Membrane Perforation

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Abstract: Chronic perforations of the tympanic membrane (TM) represent a significant source of morbidity worldwide. It is still one of the most prevalent otology problems. These complications include conductive hearing loss, middle ear infections, acquired cholesteatoma, or even intracranial complications if left untreated. Acute perforations of the TM usually heal without treatment, with up to 80% undergoing spontaneous closure. Those that persist and become chronic usually result from infection (e.g., otitis media) or traumatic injury. The management of TM perforation has been completely transformed thanks to new biomaterial designs and, more recently, tissue-engineered composites and grafts. In this study we wanted to investigate as to how fibrinogen-based collagen graft repair of central perforation, performed endoscopically improves both hearing and ear discharge. This study was done to see as to how effective collagen patch would be in closing small perforations in the tympanic membraneand to findout the difference between postoperative Air-Bone gap and the pre-operative ABG this was performed on 30 patients. This procedure done under local anesthesia an office procedure, and all patients had given consent to participate in the study after they were informed about the demerits and merits of fibrinogen-based collagen patch closure of tympanic membrane perforations. The expected result was to find the usage of collagen patch as a graft material for the repair of small tympanic membrane perforations is effective and avoid Type I Tympanoplasty in case of small perforation.Recent advances in biomaterials research and tissue engineering have provided alternative materials for TM regeneration. Biomaterials such as silk, collagen, AlloDerm, chitosan, and calcium alginate have beeninvestigated as potential TM grafting materials, and have shown promising results in animal models and clinical studies.

INTRODUCTION

Perforation of tympanic membrane is a common problem encountered by otorhinolaryngologists. If untreated, it may result in hearing loss and persistent otorrhoea. Most of the small perforations may heal spontaneously with time, but some persist because of infection.[1]

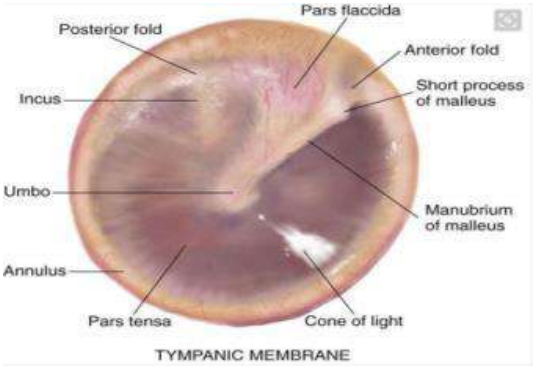

Figure 1: Normal tympanic membran

Over the years, a variety of graft materials, with different success rates has been used. An array for graft materials have been tried some of which are: temporalis fascia, perichondrium, fat, tragalorconchal cartilage.[2] Biomaterials like Gel foam, derivatives of hyaluronic acid and paper-patch, xenografts like submucosa of porcine small intestine and genetically-engineered biomaterials e.g., chitosan, calcium alginate, silk fibroin, and collagen have been experimented with[3]. This study was done to see as to how effective collagen patch would be in closing small perforations in the tympanic membrane. The fibrinogen-based collagen is a sponge like patch. It is made of collagen from equine source and is covered with a mixture of bovine-aprotinin, human-fibrinogen and bovine-thrombin. It attaches itself strongly to tissue, forming a waterproof membrane.[4-8] Patients experiencing tympanic membrane perforation usually complain of sudden onset of pain accompanied by hearing loss, bloody otorrhea, hearing loss, vertigo, or tinnitus. In the study from Nigeria, the most common presenting symptom was otorrhea (81.5%), followed by otalgia (72.8%) and tinnitus (55.7%). Unless there is associated inner ear injury, vertigo and tinnitus are typically fleeting. The physical exam must include otoscopy for direct visualization and a general assessment of vestibular function and hearing[9-14]

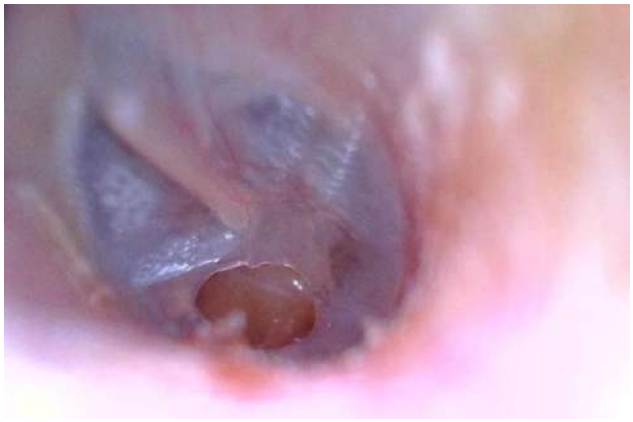

Figure 2: Left tympanic membrane with small central perforation (anteroinferior - most common location of cp)

Case History

- The duration of the study was one year (June 2019 – June 2020) in the ENT Department of Sree Balaji Medical College and Hospital, to see the effectiveness of collagen patch in the closure of small tympanic membrane perforations. History was taken, clinical examination, and preoperative investigations were done. Patients didn't have any co morbidity.
- Preoperatively and postoperatively, Pure tone audiometry (PTA) was done. Air – Bone (A-B) gap was determined at 0.5kHz,1kHz, and 2 kHz in all the study participants.
- There were 30 patients who were included in this study,all of whom had consented to participate after hearing the merits and demerits of fibrinogen based collagen patch closure of tympanic membrane perforations.

Technique

- This procedure is done under local anaesthesia as an office procedure.
- First, a cotton ball soaked in 4% Xylocaine solution was kept inside the external-auditory canal.
- Patients were made to lie down in thesupine position, with the head over the head ring,turned to the opposite side of perforation.
- Strict aseptic precautions were followed;Hopkins 0^0 endoscope was used; local anaesthesia was given in EAC using 2% Xylocaine mixed with Adrenaline.
- Margin of perforation was freshened using a sharp dissector.
- Collagen sheet was cut approximately to twicetheperforation size and placed in sterile saline solution.
- The collagen patch was inserted into perforation, such that it sticks to the overlying surface; to keep it in position and to promote healing, Gel foam pieces were kept around and on the collagen patch.
- All the study participants were advised to avoid straining or nose blowing and to keep the ear dry, for a minimum period of 1 month. They were asked to visit the OPD after 7, 14, 30 and 60 days.
- Dimeric tympanic membrane was evaluated in every follow up.
- After 1 month, and 2 months, audiometry was done.

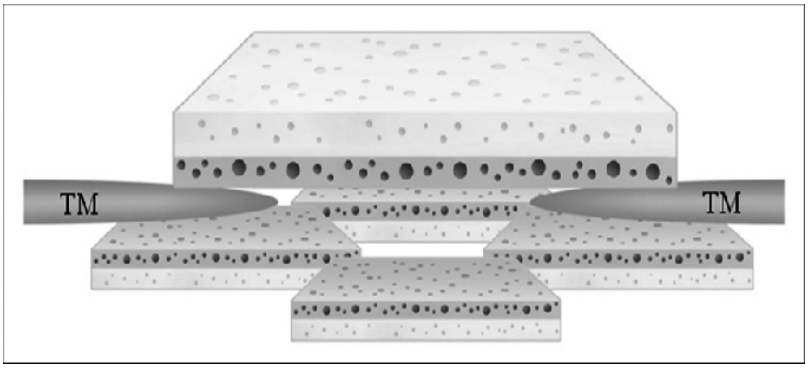

Fig 3: Schematic illustration of placement of the fibrinogen-based collagen.

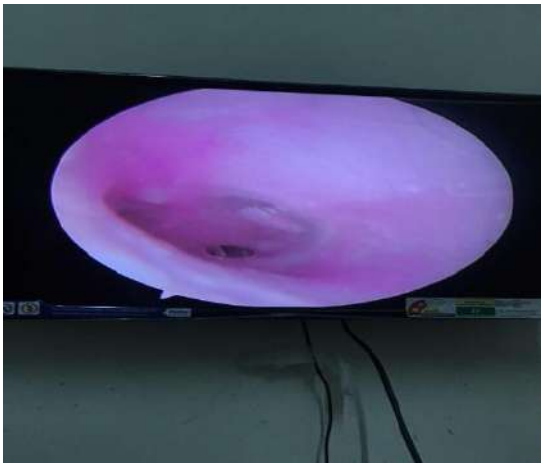

Figure 4: Pre op - left ear cp

Figure 5: post op - collagen patch repair of left ear cp

OBSERVATIONS

- This procedure has an advantage of causing the least amount of pain and trauma to the ear drum as there were no meatal flap dissections, skin incisions and a smaller number of complications. Edges of the perforations can be viewed by the surgeon easily as collagen grafts are fully transparent and hence, post-operative events can be easily monitored. Through this procedure, maximum improvement of the air-bone gap can be achieved. However, it cannot always be an alternative to the conventional surgical myringoplasty.
- The study consisted of 30 patients, of which 11 were male and 19 were female between the age group 15-50 years. Patients with smaller size perforations were chosen as part of the study. The outcome measures of this study were post-operative Air Bone gap, perforation closure, absence of complications like, persisting tympanic membrane defect, infection, otomycosis, and extrusion of the collagen patch. Of the study population, 63.3% were female and 36.7% were male (FIG 6).
- In our study, majority of the perforations were seen in the anterior quadrant, mostly in antero-inferior quadrant (50%). 10% cases comprised of Antero –superior quadrant perforation. Superior quadrant perforation was comparatively less in our study. 6.7% cases comprised of posterosuperior quadrant perforation and posteroinferior quadrant perforations were found in 33.3% cases.
- Infective (50%) & traumatic causes (33.3%) of perforation were found at a higher quadrant. Post-myringoplasty cases (16.7%) were found to be less common.

- **CHI SQUARE TEST**

	Value	df	Asymptomatic. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	30.000[a]	2	.000	.000	
Likelihood-Ratio	36.652	2	.000	.000	
Fisher's Exact-Test	29.017			.000	
LinearbyLinear Association	20.471[b]	1	.000	.000	.000
Number of Valid Cases	30				

Chi-Square Tests	
	Point Probability

Pearson Chi-Square	
Likelihood-Ratio	
Fisher's Exact-Test	
LinearbyLinear Association	.000[b]
Number of Valid Cases	

1. 4 cells (66.7%) had expected count of < 5. Minimum expected count is 2.10.

2. Standardized statistic -4.524.

2nd MONTH

		Frequency	Percent	Valid-Percent	Cumulative-Percent
Valid	Healed cp	7	23.3	23.3	23.3
	Complete Closure	14	46.7	46.7	70.0
	Incomplete Closure	9	30.0	30.0	100.0
	Total	30	100.0	100.0	

Outcome

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Failure	9	30.0	30.0	30.0
	Success	21	70.0	70.0	100.0

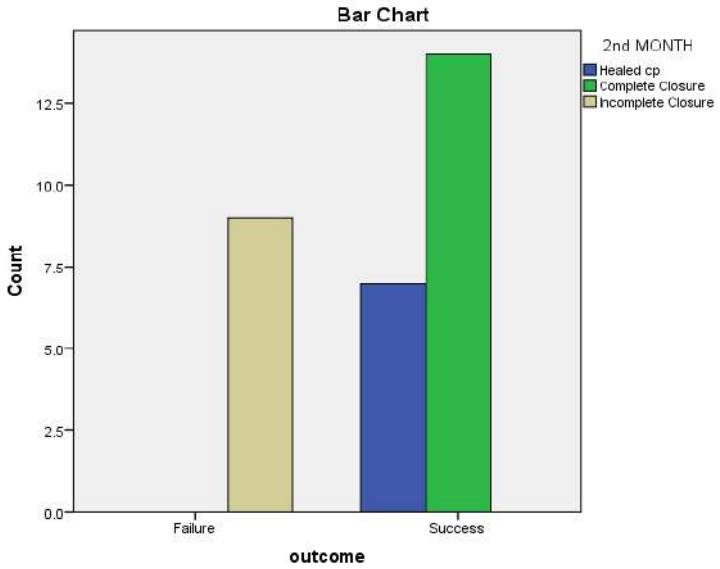

Figure: 6 Bar Graph Showing Outcomes

- In this study, collagen patch closure had a success rate of 70%and is a viable graft material for small perforations (FIG 6).
- The success rate was higher in traumatic (38.1%) and post-myringoplasty (19%) cases as compared to infective group (42.9%).
- Infective causes showed a higher failure rate (66.7%) when compared to other causes (traumatic – 22.2%, post myringoplasty – 11.1%).
- In this study, extrusion of collagen was the reason for 30 % of failure cases.
- Audiogram of a patient with 45dB hearing loss. Two months after collagen patch closure of the perforation a gain of 20dB was noted, indicating improvement in hearing.

Table showing initial and 2nd month ab gap

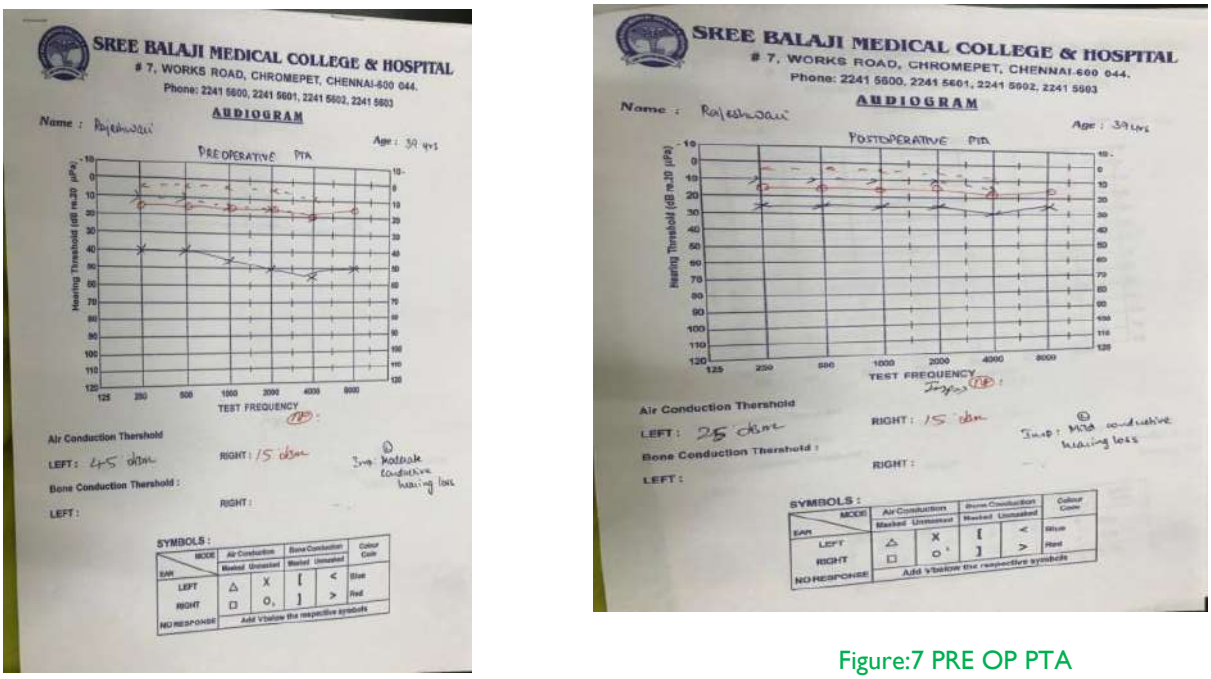

Figure:7 PRE OP PTA

Table: 1 POST OP PTA (after 2 months)

		AGE	INITIAL AB GAP (dB)	AB GAP (Db) 2nd MONTH
N	Valid	30	30	30
	Missing	0	0	0
Median		36.00	22.00	14.00
Range		34	8	10
Minimum		16	18	10
Maximum		50	26	20
Percentiles	25	26.25	20.00	12.00
	50	36.00	22.00	14.00
	75	42.00	24.00	16.00

RESULTS

While analyzing the collagen patch uptake, and the preoperative and postoperative air-bone gap, it was observed that 46.7% of the patients (n=14), showed complete resolution of the tympanic membrane perforation following collagen patch application. 23.3% of the patients (n=7) showed healed perforation after the 2 month follow up period. Whereas, 30% of the patients (n=9) showed incomplete closure following the procedure. When statistically comparing the uptake of collagen patch between the outcome groups, chi square value was found to be 30.00 which was significant, with a df = 2.

DISCUSSION

Collagen patch closure is a simple, short, cost effective procedure. Fibrinogen-based collagen attaches itself strongly to tissue, forming a waterproof membrane.[15] Collagen helps maintain the integrity, toughness, and the ability of the tympanic membrane to recover, which is necessary for maintenance of the physiological functions of normal tympanic membrane[13]. According to this study, collagen was found to have 66.7 % success, making it a viable option for repair of small perforations. A higher success rate was seen in traumatic (38.1%) & post-myringoplasty (19%) cases as compared to infective group (42.9%).[7] The failed cases were mostly of infective origin and 30% was due to extrusion of collagen patch. However these can be minimized with proper postoperative antibiotic cover and care.[6] The hearing improvement was assessed by comparing the A-B gap during follow up PTA with pre-operative Air Bone gap, the use of collagen patch in the defect showed significant improvement in hearing following the procedure.[8] There were no complications in this study. Patient compliance was better as compared to that of myringoplasty as it less time consuming and was less traumatic.[8] Collagen patch technique is cosmetically better as t is incisionless and hence less painful.[9] As the collagen grafts are fully transparent, margins of the perforations can be easily evaluated by the surgeons, and post-operative events can be easily monitored.[10-13] The results of collagen patch repair are better than those of conventional myringoplasty, or paper-patching as seen in the literatures. Some studies of patch materials to treat the tympanic membrane perforation are listed below (Table-2).

Author, year	Country	Model	TMP duration	Cause of TMP	Patch material (patients number)	Control group	Assessment methods	Result
Lee, et al., 2008[33]	South Korea	Human	Chronic	COM	Paper patch after trimming with CO2 laser (90)	None	Endoscopy, audiometry	Healing rate (%): 52.2 Improvement of ABG in all the cases where the TM healed to normal status
Hakuba, et al., 2010[34]	Japan	Human	Chronic	COM	Silicone film with bFGF and atelocollagen (87)	None	Endoscopy, audiometry	Healing rate (%): 92 Hearing threshold improvement by 10 dB or more in 51 patients, 13.4 dB HL of average hearing improvement
Lou and He, 2011[17]	China	Human	Acute, ≤ 3 days	Trauma	Gelfoam patch (30), Perforation edge-approximation with gelfoam patch (30)	No treatment	Endoscopy, healing time, infection rate	Healing rate (%): 97, 97 (study groups), 85 (control group) (p>0.05) Healing time (days): 16 ± 5.6, 18 ± 4.7 (study groups) & 30 ± 10.1 (control group) (p<0.05) Infection rate (%): 3, 3 (study groups), 7 (control group)
Saliba and Woods, 2011[18]	Canada	Human	Chronic, > 6 months	COM	Hyaluronic acid fat graft myringoplasty (131)	Underlay technique with TF/TP, overlay technique with TF/TP	Endoscopy, audiometry	Healing rate (%): 92.7 (study groups), 92.2 & 92.6 (control groups) (p>0.05) ABG (dB HL): clinically and statistically significant improvement in hyaluronic acid fat graft myringoplasty
Araujo, et al., 2012[41]	Brazil	Human	Chronic	COM	Myringoplasty with polylysine latex biomembrane (39)	Myringoplasty only, myringoplasty with silicone film	Endoscopy, audiometry	Healing rate (%): 74.4 (study group), 70 & 57.1 (control groups) (p>0.05) Vascularization: significantly greater in myringoplasty with polylysine latex biomembrane ABG (dB HL): 23.5 → 12.9* (study group), 25.2 → 11.9* & 28.5 → 10.1* (control groups) (p>0.05)
Jun, et al., 2014[20]	South Korea	Human	Acute, ≤ 3 months	Trauma	Egg shell membrane (39)	Perforation edge-approximation	Endoscopy, healing time	Healing rate (%): 92.3 (study group), 89.7 (control group) (p=0.74) Healing time (days): 42.8 ± 19.8 (study group), 87.2 ± 41.3 (control group) (p=0.02)
Simsek and Akin, 2014[21]	Turkey	Human	Acute, ≤10 days	Trauma	Paper patch (33)	No treatment	Endoscopy, audiometry	Healing rate (%): 90.9 (study group), 76.7 (control group) (p>0.05) ABG (dB HL): 23.6 → 1* (study group), 26.1 → 5* (control group) (p<0.001)
Present study	South Korea	Human	Acute & chronic, > 14 days	Trauma	Fibrinogen-based collagen fleece (29)	None P**	Endoscopy, audiometry	Healing rate (%): 100 ABG (dB HL): 12.9 → 2.4*

Table: 2 STUDIES OF PATCH MATERIAL USED IN THE TREATMENT OF TYMPANIC MEMBRANE PERFORATION
(TMP: Tympanic Membrane Perforation, Pre-Operative Average ABG → Post-Operative Average ABG, ABG: Air-Bone Gap, TF: Temporalis Fascia, TM: Tympanic Membrane, TP: Tragal Perichondrium, bFGF: basic Fibroblast Growth Factor, COM: Chronic Otitis Media)

CONCLUSION

In this study, we found that using collagen patch as a graft material for the repair of small tympanic membrane perforations is effective. There was a significant difference between postoperative Air-Bone gapand the pre-operative ABG. Other advantages of the procedure are the no requirement of hospitalization, and avoidance of traditional tympanoplasty in cases with small perforations.

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CONFLICT OF INTEREST

Conflict of interest declared none.

REFERENCE

1. WarmaldPJ.Myringoplasty. In: Bleach N, Milford C, Van Hasselt A, eds. A Operative Otolaryngology. Ch.9: Blackwell Science Ltd.; 1997:44–51
2. Kristensen S. Spontaneous healing of traumatic tympanic membrane perforations in man: a century of experience. J Laryngol Otol. 1992;106:1037–1050.
3. Chun SH, Lee DW, Shin JK. A clinical study of traumatic tympanic membrane perforation. Korean J Otolaryngol-Head Neck Surg. 1999;42:437–441.
4. Lee SH, Jin SM, Lee KC, Kim MG. Paper-patch myringoplasty with CO2 laser for chronic TM perforation. Eur Arch Otorhinolaryngol. 2008;265:1161–1164.
5. Hakuba N, Iwanaga M, Tanaka S, Hiratsuka Y, Kumabe Y, Konishi M, et al. Basic fibroblast growth factor combined with atelocollagen for closing chronic tympanic membrane perforations in 87 patients. OtolNeurotol. 2010;31:118–121.
6. Lou ZC, He JG. A randomised controlled trial comparing spontaneous healing, gelfoam patching and edge-approximation plus gelfoam patching in traumatic tympanic membrane perforation with inverted or everted edges. Clin Otolaryngol. 2011;36:221–226.
7. Saliba I, Woods O. Hyaluronic acid fat graft myringoplasty: a minimally invasive technique. Laryngoscope. 2011;121:375–380.
8. Araujo MM, Massuda ET, Hyppolito MA. Anatomical and functional evaluation of tympanoplasty using a transitory natural latex biomembrane implant from the rubber tree Hevea brasiliensis. Acta Cir Bras. 2012;27:566–571.
9. Jun HJ, Oh KH, Yoo J, Han WG, Chang J, Jung HH, et al. A new patch material for tympanic membrane perforation by

trauma: the membrane of a hen egg shell. Acta Otolaryngol. 2014;134:250–254.

10. Levenson MJ. The Management of traumatic perforation of tympanic membrane. In: Blitzer A, Pillsbury HC, Jahn AF, Binder WJ, eds. Office based surgery in otolaryngology. Ch.15: Thieme Medical Publisher; 1998:95–107.
11. L Macri, D Silverstein, RA Clark,Growth factor binding to the pericellular matrix and its importance in tissue engineering,Adv Drug Deliv Rev, 59 (2007), pp. 1366-1381
12. Farhadi Mohammad, Maryamjalessi, (2011) Collagen immobilized patch for repairing small tympanic membrane perforations: invitro and invivo assay. SociBiomater 100(3):543–549 Adegbiji WA, Olajide GT, Olajuyin OA, Olatoke F, Nwawolo CC. Pattern of tympanic membrane perforation in a tertiary hospital in Nigeria. Niger J Clin Pract. 2018 Aug;21(8):1044-1049.
13. Van Hoecke H, Calus L, Dhooge I. Middle ear damages. B-ENT. 2016;Suppl 26(1):173-183
14. Levin B, Rajkhowa R, Redmond SL, Atlas MD. Grafts in myringoplasty: utilizing a silkfibroin scaffold as a novel device. Expert Rev Med Devices 2009;6:653–664.
15. Teh BM, Robert MJ, Shen Y, Friedland P, Dilley RJ, Atlas MD. Tissue engineering of the tympanic membrane. Tissue Eng Part B Rev 2012; doi:10.1089/ten.TEB.2012.0389. Epub ahead of print.

SP-11

Comparison of Body Mass Index with Pulmonary Function Test in Obese Individuals

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Abstract: Body mass index (BMI) is the estimation of body fat based on height and weight.BMI is considered an important measure of obesity. A rise in BMI leads to a greater risk of developing serious health conditions, such as respiratory illness, heart disease, high blood pressure, liver disease, osteoarthritis, Diabetes Mellitus, stroke, gallstones, certain cancers, including breast, colon and, kidney cancers. Pulmonary function tests (PFT)using Spirometry is an non invasive technique used mainly to diagnose lung pathology as well as differentiate the Obstructive from Restrictive lung disease..In this study we tried to hypothesize that increase in BMI causes decline in the pulmonary function so that. we can derive an correlation between BMI and PFT obtained by spirometry of the obese individual. To Compare and correlate Body Mass Index with Pulmonary Function Test in Obese Individuals .To measure the Body Mass Index, in different age groups of both genders in obese individuals. To measure PFT using a Koko computerized spirometer. To compare and correlate Pulmonary Function Tests with BMI.METHODOLOGY :This study is a cross-sectional study conducted among 300 individuals of both genders. The study group included were normal subjects who satisfied my inclusion and exclusion criteria.BMI was measured by the Quetelet index, which is weight in kilograms divided by square of height in meters (kg/m2).The pulmonary function test was done by a Koko spirometry.The spirometry values were compared with the BMI values for correlation using statistical analysis .There is no significant negative linear relationship between Body Mass Index and all the spirometric values with p-value>0.05. This study result shows that BMI alone can't be taken into account for comparing pulmonary physiology

Keywords: Body mass index, pulmonary function, spirometry.

INTRODUCTION

Obesity is one of the most common and most frustrating disorders in medical practice which is difficult to manage. In adults increase in body weight may lead to an increase in adipose tissue or accumulation of fluid or both.[1] Obesity is having too much body fat. It is different from overweight. Being overweight is the pre-obesity. But both the terms overweight and obesity are said to be that the person's weight is greater than ideal weight which is not an indicator for good health.[2] Physical inactivity, overeating, genetics, hormonal, psychological factors, medications had become the major factors of Obesity.[3] Weight distribution may be either upper (manly) or lower (womanly).[4] To distinguish between these two is important because, compared to female obesity; male obesity is associated with greater risks of hypertension, cardiovascular disease, Diabetes Mellitus, and stroke.[5] Men who are obese are more likely to have the android kind of obesity and hence are at higher risk of obesity complications when compared with women. [6] The ideal BMI for an adult is 18.5kg/m2-24.9kg/m2. Below 18.5kg/m2 is considered as underweight and between 25kg/m2-29.9kg/m2 as overweight and more than 30kg/m2 as obese.[7]A high BMI is assumed to be a sign of too much fat on the body, while a low BMI is a sign of too little fat on the body. According to the National Institutes of Health, more than two in three adults are considered overweight and one in three is considered obese. About 17 percent of children and teenagers (2 to 19 years) are considered to be obese. [8] A rise in BMI leads to a greater risk of developing serious health conditions, such as respiratory illness, heart disease, high blood pressure, liver disease, osteoarthritis, Diabetes Mellitus, stroke, gallstones, certain cancers, including breast, colon and, kidney cancers. [9] Spirometry measures the amount of air we inhale and exhale. Spirometry finds out the integrated mechanical function of the lung, chest wall, and respiratory muscles by measuring the total volume of air exhaled from a full lung. Pulmonary function test is classified as static lung function test and dynamic lung function test. Static lung function tests include Inspiratory reserve volume (IRV), Expiratory Reserve Volume (ERV), Residual volume, and Tidal volume. Dynamic lung function tests include Forced vital capacity (FVC), Forced expiratory volume (FEV), Maximum voluntary ventilation (MVV), Peak Expiratory Flow Rate (PEFR). These pulmonary function tests are mainly used to differentiate the Obstructive from Restrictive lung disease. Depending upon the Spirometry values the status of the lung is diagnosed. BMI is considered an important measure of obesity. In this study we tried to hypothesize that increase in BMI causes decline in the pulmonary function test values. This study was conducted in the Department of Physiology of SreeBalaji Medical College and Hospital, Chrompet, Chennai.

STUDY DESIGN

This study is a cross-sectional study conducted among 300 individuals of both genders. The study group included were normal subjects who satisfied my inclusion and exclusion criteria.

Sample size

To calculate the sample size:
1. Estimation of the mean formula was used
2. 95% confidence interval i.e.1.96
3. S.D- 0.84
4. d-margin error- 10%
5. The sample size was calculated to be 278 based on the previous study
The sample size rounded off as 300.

Inclusion criteria

1. Age group -18-40years.
2. Both male and female gender.
3. Volunteers with no history of medical illness

Exclusion criteria

1. Subjects suffering from the following disease are excluded from the study
• Pre-existing respiratory disorders
• Acute upper or lower respiratory infections
• Individuals with cardiac disease, renal disease, and any other systemic illness like Diabetes and Hypertension.
2. Smokers and alcoholics were excluded.

Data collection

History taking

Each subject was asked to fill up the questionnaire (ANNEXURE IV) regarding the general information, socio-economic status, and previous medical history regarding treatment taken for any respiratory disorder.

Anthropometric parameters

Height
Height was measured to the nearest of 0.1 cm while the subject was standing in an erect position with feet on a flat surface in Stadiometer and head straight.

Weight

The body weight was measured in kilograms using a digital weighing scale with the subject standing on the center of the scale barefoot.

Body Mass Index

BMI was measured by the Quetelet index, which is weight in kilograms divided by square of height in meters (kg/m2). BMI in the range of 18.50 to 24.99 kg/m2 is considered to be normal. BMI is classified as follows
Underweight: <18.5 kg/m2
Normal weight: 18.5- 24.9 kg/m2
Overweight: 25.0- 29.9 kg/m2
Obesity 1: 30.0- 39.9 kg/m2
Obesity 2: >40 kg/m2

Pulmonary Function Test

The pulmonary function test was done by a Koko spirometry. Before starting the test the apparatus was calibrated with a 3lpiston. The Subject was asked to sit in an upright position and to hyperventilate and then asked to take a deep breath and blow in the mouthpiece of the spirometer continuously for 6 seconds and then take a deep inhalation till the curve is obtained. In the same manner, three more trials were taken and the best of the three efforts were taken as the final value. The directly evaluated parameters were lung volume and capacities. The Forced Vital Capacity, Forced Expiratory Volume in the first second, and FEV1/FVC ratio was the value documented. The Normal values: FEV1 – 3.5l-2.5l, FVC -4.8l- 4.0l, and FEV1/FVC-80%. The spirometry values were compared with the BMI values.

Statistical Analysis

1. Analysis is done using IBM SPSS (Statistical Package for Social Sciences) version 23.
2. Data collected was entered in Microsoft Excel sheet.
3. Descriptive statistics given in the form of Means, Standard Deviation, frequency, and graphs.

4. Linear relationship between two continuous variables is found using the Pearson correlation.
5. Non- parametric tests Kruskal Wallis is applied to find the mean difference between the spirometric values.

RESULTS

• Out of 300 patients who participated in the study 53% were male and 47% were female.
• Among them, 56% were in the age group 17- 20years and 16% were in the age group 20-25years.
• Pearson's correlation is applied to check the linear relationship between BMI and the spirometric values ie.FEV1, FVC, FEV1/FVC.
• 34% of the population is overweight and 64% of them are moderate obese category.
• The weight, height, BMI, is described by Mean ± SD.
• There is no significant negative linear relationship between Body Mass Index and all the spirometric values with p-value>0.05.

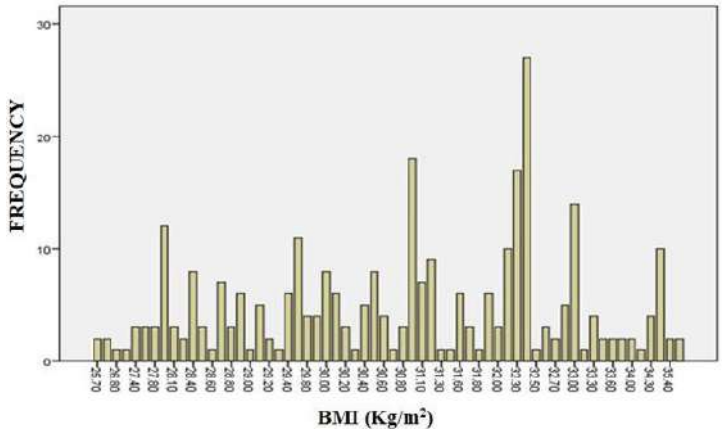

Figure: 1 Frequency bar diagram of Body Mass Index

This bar diagram shows frequency of BMI with mean 31.2±4.25kg/m2. In this study population 10% of subjects had BMI of 32.45kg/m2 ; 12% of subjects had BMI of 32.30kg/m2 and 31.10kg/m2 together; 32% of subjects had BMI of 34.30kg/m2 and 26% of subjects had BMI of 27.40kg/m2, 33.60kg/m2 and 35.40kg/m2 together. This shows that the range of BMI in study subjects are more.

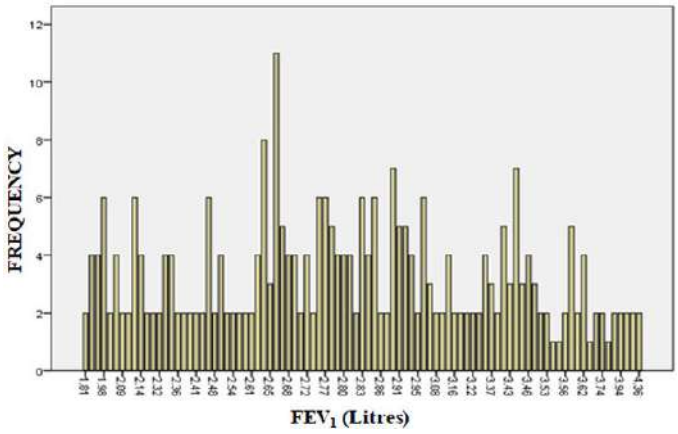

Figure: 2 Frequency bar diagram of Forced Expiratory Volume in one second

This bar diagram shows the frequency of FEV1 with mean 2.84 ± 0.5188l. In this study population 12% of subjects had the FEV1 values of 2.67 and 2.68 together; 22% of subjects had the FEV1 values of 3.16, 3.37, 3.22 together; 25% of subjects had the FEV1 values of 3.56, 3.64, 3.53 together; 26% of subjects had the FEV1 values of 2.14, 2.09 together; 15% of subjects had the FEV1 values of 1.81, 1.98, 4.36, 3.94 together. The normal value of FEV1 for male ranges between 3.5l-2.5l and for female ranges between 2.5l-3.25l. This shows that in this study population 48% of the subjects had reduced FEV1 values when compared to normal.

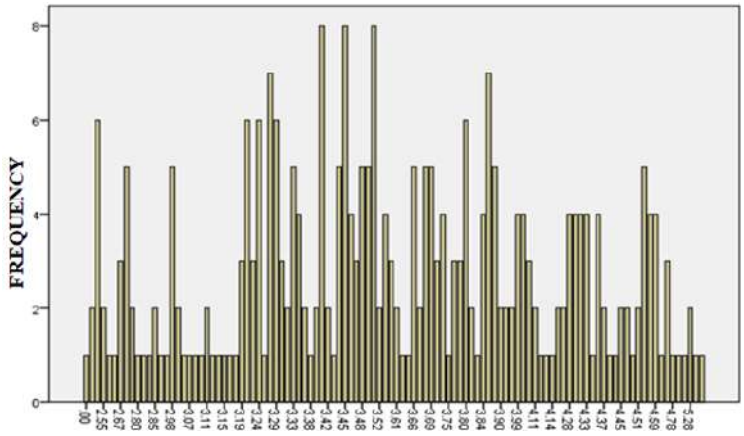

Figure: 3 Frequency bar diagram of Forced Vital Capacity

This bar diagram shows the frequency of FVC with mean 3.626 ± 0.630l. In this study population 24% of subjects had the FVC values of 3.84l, 3.48l, 3.45l together; 40% of subjectshad the FVC values of 3.19l , 3.15l, 3.07l together; 36% of subjects had the FVC values of 2.98l, 2.95l, 2.55L together. The normal value of FVC ranges between 4.8L-4.0L. This shows that in this study population 50% of subjects had reduced FVC when compared to normal.

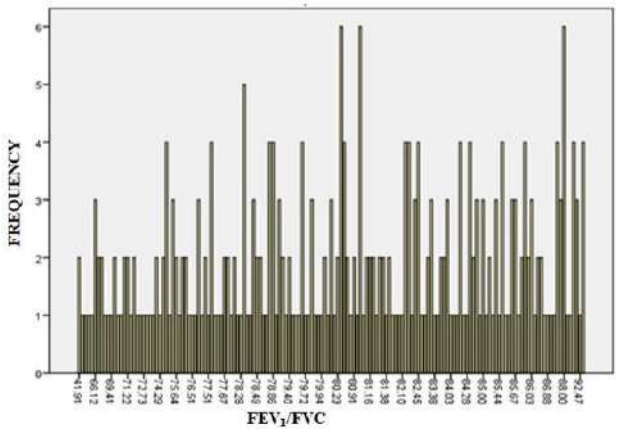

Figure : 4 Frequency bar diagram of FEV1/FVC

This bar diagram shows the frequency of FEV1/FVC with mean 80.32 ± 6.37. In this study population 18% of subjects had FEV1/FVC ratio of 80.23, 80.91 and 88.00 together; 40% of subjects had FEV1/FVC ratio of 79.72, 78.28 and 77.51 together; 42% of subjects had FEV1/FVC ratio of 69.41, 66.12 and 41.91together. The normal value 0.75-0.80.This shows that in this study population 42% of subjects had reduced FEV1/FVC which means that there decline in the pulmonary function.

Table: 1 Comparison of Body Mass Index with the Spirometric values

Parameters		FEV1 /FVC	FEV1(Litres)	FVC(Litres)
		Mean± Standard Deviation	Mean± Standard Deviation	Mean± Standard Deviation
BODY MASS INDEX Kg/m^2	OVERWEIGHT	81.16±4.83	2.85±0.53	3.58±0.72
	OBESE [MODERATE]	79.87±7.04	2.86±0.51	3.66±0.58
	OBESE [SEVERE]	78.73±6.21	2.74±0.11	3.49±0.41
	MORE SEVERE	82.98±8.22	2.05±0.11	3.29±0.75
P-VALUE		0.07	0.08	0.06

P significance taken as < 0.05.
Normal values: FEV1 – 3.5l-2.5l
FVC – 4.8l- 4.0l
FEV1/FVC-0.75-0.80

This table shows the comparison of Body Mass Index with all the spirometric values like FEV1, FVC and FEV1/ FVC .Based on BMI the subjects are classified as overweight, moderately obese, severely obese and more severely obese. The p value (0.07, 0.08, and 0.06) for FEV1/ FVC, FEV1 and FVC respectively shows there is no significance between the BMI and the Spirometric

values. Compared to normal values it is shown that although there is decline in pulmonary function it is not statistically significant.

Table: 2 Correlation between the Body Mass Index and the Spirometric values.

		FEV1 (Litres)	FVC Litres)	FEV1 /FVC	BMI
BMI Kg/m^2	Pearson Correlation	-0.101	-0.018	0.007	1
	Sig. (2-tailed)	0.081	0.763	0.908	
	N	299	299	299	299

. Correlation is significant at the 0.01 level (2-tailed).

This table shows the correlation between the BMI and the Spirometric values. There is no significant correlation between the BMI and the Spirometric values. In this study population the increase in BMI has notalteredthe pulmonary function which is reflected in the PFT values.

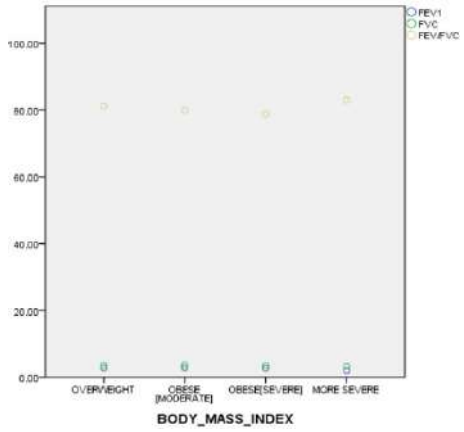

Figure :5 Scatter plot of Body Mass Index with the Spirometric values

Figure 5 shows the scatter plot which shows that there is no significant linear relationship between BMI and all the spirometry values. The spaced variable shows that there is no significant relationship.

DISCUSSION

Obesity has altered the pulmonary functions as evidenced by the Spirometricvalues.In this study, 46% were females and 53% were males of whose mean and standard deviation of BMI were 31.24 ± 4.40 and 31.24± 4.127 respectively as shown in table 2. Here we can see that 56% of patients are in age-interval between 15-20 years, and 17% of patients are between age-interval 35-40 years and 16% of them are in between the interval 20-25 years. In individual patients, the distribution of fat may be more important than BMI. Further research has to be done on the distribution of fat, dyspnoea, central breath timing, and chest wall mechanics in obese individuals.[10] Ranjanasinghne et al study observed pulmonary function decline mainly depends on the movement of lungs, thoracic wall muscles, and diaphragm in which BMI increase leads to a decrease in FEV1, FVC, and FEV1/FVC.[11] Whereas in this study BMI is not correlating much with the Spirometric values. David Madden's study had set the opinion that depending on the threshold level of BMI, obesity can be determined.[12] Likewise in this study obesity has been classified as obese, moderately obese, and severely obese based on the BMI threshold which is shown in table 1. BMI alone won't be the deciding factor of obesity, as there are many pit's and fall in the values.[13] The same thing was noted in this study, that BMI does not play a major role in analyzing the pulmonary function. Whereas in some studies they found that there was a strong association between lung function and BMI. Mainly, FVC and FEV1 were generally decreased over a 10 year period both with higher baseline BMI.[11] Cheryl M Salome study states that the presence of adipose tissue around the rib cage and abdomen reduces the functional residual capacity and expiratory reserve volume. Obesity even has a little direct effect on airway caliber, thus reduce respiratory well-being.[15] HarpreetRanu's study showed pulmonary function tests as an important tool in the assessment of patients with suspected or known respiratory disease. In this study, fig.2,3, and 4 show the mean and standard deviation of spirometric values, FEV1-2.84 ± 0.5188; FVC-3.626 ± 0.630; FEV1/FVC- 80.32 ± 6.37. Many studies showed that overweight and obesity lead to a reduction in lung volume and capacities. Besides, there was a diminution in forced expiratory volume and forced vital capacity. Miller et al's study stated the standard method (normal respiratory maneuver) of measuring the spirometric values like FVC, TLC, MMEF, MMV, FRC, and all the lung volumes. Mohammed Al Ghobain et al study showed that there was no significant difference between the obese and non-obese subjects in FEV1, FVC, FEV1/FVC ratio, and FEF25-75. The study done by UmusOsbey et al found that as the BMI, WC, and WHR increases there is a decrease in the spirometric values like FEV1, FVC, MEF25, MEF25-75, and MEF50 with significant p-value < (0.05).[16] This shows that in this study population BMI does not correlate with the spirometric values.

CONCLUSION

Many studies had shown that definitely there is a decline in pulmonary function in obese individuals. For which BMI was mainly taken into account, this study result shows that BMI alone can't be taken into account for comparing pulmonary physiology. BMI is the measure of height and weight, which alone doesn't cause much change in lung function. This study had shown that BMI can't be the important measure of obesity to correlate with the PFT values.

CONFLICT OF INTEREST

Conflict of interest declared none.

ACKNOWLEDGEMENT

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ETHICAL STATEMENT

The study was approved by the Institutional Ethics Committee for human research. The procedure and purpose of the study were clearly explained in detail to the subjects. Written informed consent was obtained from the subjects in their own language.

REFERENCES

1. Bhaskara Rao Thirunavalli, Usha Rani Chadalawada. Preventive Cardiology. Text Book of Community Medicine.2015; 3:522-524.
2. M C Gupta, B K Mahajan. Food and nutrition.Text Book of Preventive and Social Medicine.2005; 3:359-360.
3. K Park. Non – Communicable Disease .Preventive and Social Medicine.2005; 18:316-319.
4. A H Suryakantha. Epidemiology of Non- Communicable Diseases. Community Medicine with Recent Advances.2017; 4:569-571.
5. PreglerDecherney. Obesity. Women's Health Principle and Clinical Practice.2002; 1-16.
6. https://www.hopkinsmedicine.org/health/treatment-tests-and-therapies/pulmonary-function-tests;1:991-1005.
7. https://www.mayoclinic.org/tests-procedures/spirometry/about/pac-20385201.
8. Lukaski HC, Johnson PE.Assessment of fat free mass using bio electrical impedance measurements of the human body. Am J clin Nutr.1945; 41:810-7
9. Rexrode KM, Hennekens CH et al. A prospective study of Body Mass Index, weight change and risk of stroke in women. JAMA 1997; 277: 1539-45.
10. Miller MR. Standardisation of spirometry. EurRespir J 2005; 26: 319–338.
11. David Madden. Body Mass Index and the Measurement of Obesity. January 2007; 1-16
12. Dirceu Costa. The Impact of obesity on pulmonary function in adult women clinics. 2008; 63(6):719-24.
13. Frank Q. Nuttall, Body Mass Index Today. 2015; 50(3):117-128.
14. HarpreetRanu. Pulmonary Function Tests. Ulster Med J 2011; 80(2):84-90.
15. Bharaththiyagarajan. longitudinal association of Body Mass Index with lung function. Cardia study. April 2008; 9:31.
16. Pradeep Prajapat. A prospective study of pulmonary function test in obese patients. International Journal of Advances in Medicine: Int J Adv Med. 2016 Feb; 3(1):73-76.

SP-12

A Rare Case of Disproportionate Anemia Due to Plasma Cell Leukaemia Presenting as Chronic Kidney Disease

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Abstract: Plasma cell leukaemia (PCL) is a leukaemia that affects plasma cells and has a bad prognosis. Only a few examples of direct renal involvement in PCL have been recorded in the English literature. Anemia of chronic renal disease is mostly coupled with deficiency of erythropoietin. Anemia is related with other irregularities like low platelet count. When anemia along with thrombocytopenia is present, secondary renal disorders other than hematological abnormalities have to be considered. In disease like Multiple myeloma, renal manifestations were common. Hematological abnormalities also need to be detected without being missed. Cortico-medullary differentiation is lost, both kidneys are of normal size and dimensions; Fibrotic changes with tractional brochiectasis with multiple nodules were seen in right lung. No lytic lesions were seen in X -ray of bone. Serum calcium level was decreased, serum phosphate was within normal range and Serum lactate dehydrogenase was increased. RFT however was deranged and urine routine shows albuminuria, 24-hour urine output is 6.8 gm/24 hour. Monoclonal gammopathy is seen in the patient. Beta-2 microglobulin levels were 36 times more than normal. The aspiration bone marrow was done and it revealed polynucleated plasma cells which was more than half of the cell population. In this scenario we present a case of chronic renal disease having severe anaemia along with other bleeding disorders which later was found to be plasma cell cancer.

Keywords: Anemia, Plasma Cell Leukemia, Erythropoietin

INTRODUCTION

Anemia in chronic renal disease has been studied in various circumstances. As kidney function declines anaemia becomes more predominant. It is more prevalent in chronic renal diseases. Association between the severity of anaemia and varying levels of kidney function have been reported in several studies.[1-5] Anemias in patients with renal diseases are almost always linked to deficiency of erythropoietin. Hence it is necessary to calculate the haemoglobin level and correct the iron deficiency. Any other blood disorders and renal disorders must be interpreted and diagnosed properly when there is severe anemia and low platelet count which can lead to other bleeding disorders.[6-9]

CASE REPORT

Presenting Complaints

A 40 year female patient came with symptoms of fever and cough with expectoration for16 weeks, vaginal bleeding for 4 weeks, bleeding from gums, burning sensation during voiding for 8 days. There were no other complaints like anasarca, puffiness of the face or swelling on both feet. She was hospitalized 12 weeks prior to a hospital for similar symptoms. Her blood reports showed severe anemia, TLC and differential white blood cell count were normal.

Past History
No significant past history, no history of any surgeries in the past.
Family History
Nil significant

Peripheral smear revealed iron deficiency type of anemia and also showed low platelet count. Other investigations showed increased level of urea and creatinine, urine routine analysis showed albuminuria and urine output was normal. She was reckoned as case of chronic kidney disease stage IV with sputum negative pulmonary tuberculosis. For 6 months she got treatment for tuberculosis and she also received treatment for renal impairment. She also had complaints of abnormal uterine bleeding past 12 months. Before one year she had normal menstrual cycle. She was poorly built and poorly nourished on general examination. Her blood pressure, pulse rate were normal. She showed signs of severe anemia. Systemic examination of respiratory system showed signs and symptoms of right upper lobe pulmonary fibrosis. Other systems examinations were normal. On investigation severe anemia, leucopenia, thrombocytopenia were seen. Iron deficiency type of anemia is revealed in peripheral smear and also showed presence of plasmacytoid dendritic cells. Ultra sonogram of abdomen revealed bilateral medical renal disease showed bilateral renal parenchymal disease: Cortico-medullary differentiation is lost; both kidneys are of normal size and dimensions; Fibrotic changes with tractional brochiectasis with multiple nodules were seen in right lung. No lytic lesions were seen in X -ray of bone. Serum calcium level was decreased, serum phosphate was within normal range and Serum lactate dehydrogenase was increased. RFT however was deranged and urine routine shows albuminuria, 24 hour urine output is 6.8 gm/24 hour. Monoclonal gammopathy is seen in the patient. Beta-2 microglobulin levels were 36 times more than normal. The aspiration bone marrow was done and it revealed polynucleated plasma cells which was more than half of the cell

population. And further investigation revealed normal mature cell, a few binucleate and multinucleated plasma cells and is suggestive of PCL.

Prognosis

Early diagnosis and management give good prognosis.

DISCUSSION

This subject was found to be a case of chronic renal disease with iron deficiency anaemia with bleeding disorders and low platelet count. The low haemoglobin level and bleeding disorders were not associated to her chronic renal disease. Microcytic hypochromic anaemia due to deficiency of erythropoietin and abnormal platelets coupled with chronic renal disease was unable to explain her anaemia and bleeding disorders.[5] Unproportionate chronic renal disease and anaemia warranted further investigations. Plasma cells are reactive in peripheral smear. Study of bone marrow aspiration and study of smear are done. Plasma cell leukaemia (PCL) is plasma cell disorder and accounts for about5% of plasma cell disorders. More than 25% plasma cell in blood with an APC count >2 x 10^9/L is diagnostic of plasma cell leukemia. Plasma cell myeloma and late onset helps to differentiate primary disorder from secondary disorders.[10-13] There is overlap between leukemia and plasma cell myeloma, plasma cells express CD20 antigen in PCL and they often lack CD56 antigen which is anchoring protein that helps in attachment of plasma cells to bone marrow. PCL of secondary type expresses another protein CD28. Plasma cells in plasma cell leukemia express high proliferative rates and more complex karyotypes than other myeloma. PRAD1/cyclin D1 in PCL plays an vital role in cell cycle control .In plasma cell myeloma kidney involvement involves deposits in mesangium and sometimes in basement membrane.[14,15]

CONCLUSION

The response rate for PCL of secondary type is comparatively low but recovery rate can be increased with addition of thalidomide to the therapy. The median survival rate is around 8-20 months. With transplantation of bone marrow rate of survival is around 37 months.

CONFLICT OF INTEREST

Conflict of interest declared none.

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REFERENCES

1. Korbet SM: Comparison of hemodialysis and peritoneal dialysis in the management of anemia related to chronic renal disease. Semin Nephrol 1989;9(suppl 1):9-15.
2. Eschbach JJ, Funk D, Adamson J, et al: Erythropoiesis in patients with renal failure undergoing chronic dialysis. N Engl J Med 1967;276:653.688
3. Rambach WA, Kurtides E, Alt HL, Del Greco F: Azotemic anemia and the effect of hemodialysis. Trans Am Soc Artif Intern Organs 1963;9:57 61.
4. Solomon A, Weiss DT, Kattine AA: Nephrotoxic potential of Bence Jones proteins. N Engl J Med 1991;324:1845.
5. Kyle RA, Maldonado JE, Bayrd ED. Plasma cell leukemia. Report on 17 cases. Arch Internal medicine 1974;133:813-818.
6. Brown KA: Nonmalignant disorders of lymphocytes. Clin Lab Sci 1997;10:329.
7. Boles JM, Dutel JL, Briere J, Mialon P, Robasckiewicz M, Garre M: Acute renal failure caused by extreme hyperphosphatemia after chemotherapy of an acute lymphoblastic leukemia. Cancer 1984;53: 2425–2429.
8. Conger JD: Acute uric acid nephropathy. Med Clin North Am 1990;74: 859–871.
9. Obrador GT, Price B, O'Meara Y, Salant DJ: Acute renal failure due to lymphomatous infiltration of the kidneys. J Am Soc Nephrol 1997;8:1348–1354.
10. U.S. Renal Data System. USRDS 2003 Annual Data Report: Atlas of End-Stage Renal Disease in the United States, National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases, Bethesda, 2003.
11. Kapoor M, Chan GZ: Malignancy and renal disease. Crit Care Clin 2001;17: 571–598.
12. Johnson RJ, Kivlighn SD, Kim YG, Suga S, Fogo AB: Reappraisal of the pathogenesis and consequences of hyperuricemia in hypertension, cardiovascular disease, and renal disease. Am J Kidney Dis 1999;33: 225–234.
13. Ronco C, Bellomo R, Inguaggiato P, Bonello M, Bordoni V, Salvatori G, D'Intini V, Ratanarat R: Rasburicase therapy in acute hyperuricemic renal dysfunction. Contrib Nephrol 2004;144: 158–165.
14. Eagen JW: Glomerulopathies of neoplasia. Kidney Int 1977;11: 297–303.
15. Smith DM, Weisenburger DD, Bierman P, Kessinger A, Vaughan WP, Armitage JO: Acute renal failure associated with autologous bone marrow transplantation. Bone Marrow Transplant 1987;2: 195–201.

SP-13

A Case of False Positive Prenatal Down's Syndrome Screening

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Abstract: Down syndrome is a chromosomal disorder that has a huge impact in physical and mental status of the child that can be screened and diagnosed prenatally. Incidence of down 's syndrome is 1:500 with the false positive prevalence of 5% in its screening test. With the widespread application of screening tests for down's syndrome in first and second trimesters, a greater number of women are identified as high risk for downs syndrome necessitating invasive diagnostic modalities. Yet the inherent caveat in screening tests is high false positive rate and false negative rate. Here we present a case of false positive screening test reports done in both first and second trimester. A 34 year / primi/ conceived by ovulation induction/ NT scan done on 13weeks+5days revealed absent nasal bone and NT = 4.2 mm, Quadruple test was done and the pregnancy was identified as high risk for downs syndrome (1:187 high risk). Level 2 scan was done which showed small echogenic intracardiac focus (EIF) in Left ventricle of the feta heart. Patient Counselled for amniocentesis at 14 weeks but was not willing for amniocentesis and karyotyping study. shecontinued the pregnancy and delivered alive term healthy baby with no features of downs syndrome. This emphasises the importance of interpretation of the screening tests and counselling the patients and their family for the same.

Keywords: Down syndrome, Chromosomal disorder, Trimester.

INTRODUCTION

Screening test by definition is a test done on large group of people to identify people at risk of harbouring a particular condition. Down syndrome is a chromosomal disorder that has a huge impact in physical and mental status of the child, that can be screened and diagnosed prenatally. To avoid subjecting all pregnant women to invasive chorionic villous sampling or amniocentesis to diagnose downs syndrome, anon invasive screening test like NT scan with double marker in 11 to 14 weeks and quadruple test in 15 to 18 weeks can be done to detect pregnancies at risk of having down's syndrome.[1] Yet, the false positive rateof down's syndrome screening is 5% meaning 5 out of 100 women found to be high risk in the screening test are actually having a healthy baby. Here we present a case of a false positive down's syndrome screening tests done both in first and second trimesters.[2]

CASE REPORT

A 34 year old female / married since 5 Years / primi / socioeconomic class 4 / k/c/o PCOS. she conceived by ovulation induction drug on the second cycle. Her husband's age was 37 years. she came to our hospital at 14weeks+4days for 2nd opinion. She had her regular ANC check-up. All her investigations at booking were normal. Dating scan was done and it was corresponding. NT scan was done on 13weeks+5days revealed "Absent nasal bone and NT = 4.2 mm (high suspicion for down's syndrome) double marker test was not done. In our hospital Quadruple test was done and the pregnancy was identified as high risk for downs syndrome (1:187). Patient was explained about risk of downs syndrome and counselled for amniocentesis @ 14 weeks but patient was not willing for amniocentesis and karyotyping study.Level 2 scan was done which showed absent nasal bone with small echogenic intra-cardiac focus (EIF) in Left ventricle of the fetal heart (high risk of trisomy 21). Length of Femur fall on the 5thpercentile.Pt decided to continue the pregnancy opting out amniocentesis. She continued the pregnancy and delivered alive term healthy male baby of birth weight 2.8 kg by labour naturale. Baby was examined for any feature of Down's syndrome by paediatrician. No feature was found. The baby was followed up. Baby attained age appropriate milestones. USG abdomen, ECHO and Audiogram was done for the baby and it was found to be normal .Baby's mental status was also normal.

DISCUSSION

Down's Syndrome

Down's syndrome is a genetic condition that causes mild to serious physical and developmental problem. Its prevalence is approximately 1:500 recognizedpregnancy. It is also known as trisomy 21 , that is extra chromosome of 21 leading to range of issues that affect both mentally and physically. It was first described by J.L.H.Down in 1860. In 1959 Lejeune demonstrated the cause of down's syndrome.

Down's Syndrome Screening

- 1st trimester screening (11 WEEKS – 13WEEKS+6DAYS)

NT scan (nuchal thickness >3 mm)
PAPP-A
Beta HCG

Age of the mother

- 2nd trimester screening (15 WEEKS – 18 WEEKS)

Triple test : b HCG + AFP + unconjugated estriol
Quadruple test: b HCG + AFP + unconjugated estriol + inhibin A
Soft markers are :(a) Nuchal skinfold thickness
(b) Echogenic foci of heart
(c) mild renal pelvis dilatation
(d)echogenic bowel
(e) clinodactyly
(f) sandal-gap
(g) short femur or humerus
(h) borderlineventriculomegaly
(i) aberrant right subclavian artery

NT Scan

It was discovered by Nicolaides and co-workers in 1992. It is a measurement of fluid in the neck region. It is done between 11-13 weeks.It can be increased in chromosomal abnormalities, congenital heart defects, structural abnormalities like exomphalos, congenital diaphragmatic hernia.[3]

Criteria For NT Measurement

- The crown rump length should be 4.5-8.5cm.
- Mid-sagittal view of head and upper thorax, occupying the whole screen.
- The head must be in neutral position in line with the spine.
- The neck skin should be differentiated from the amnion which is shown by fetal movements.
- The widest part of NT should be measured.
- The callipers for the measurement should be placed on the inner borders of the white lines(skin and skull).
- The NT measurement should be repeated and the maximum reading that meets the above criteria should be used.

Table 1: Interpretation of the screening test

Unconjugated Estriol	Papp-A	Bhcg	Afp	Inhibin A
Decreased	Decreased	Increased	Decreased	Increased

Beta-HCG

In normal pregnancy, peaks at 15 weeks. Followed by, there is a rapid decline until 17 weeks. Then gradually fall between 17 and 22 weeks gestation. In trisomy 21 pregnancy, beta-HCG is increased.

Alpha-fetoprotein

AFP is produced by the liver and GIT in the fetus and excreted in the urine. Serum AFP levels are reduced by around 25% in cases of trisomy 21.

Unconjugated-estriol

It is a product dehydroepiandrosterone sulphate which is produced by fetal adrenal glands. Concentration is decreased by 25% in trisomy21.

Inhibin A

It is produced by placenta to inhibit the release of FSH. In normal pregnancy its level is elevated only in 1st trimester. But in trisomy 21its levels are elevate in 2nd trimester also.

Table 2 : Rate of interpretation

SCREENING METHOD	DETECTION RATE %	FALSE POSITIVE RATE %
Maternal age	30%	5%
Double test	58%	5%
Triple test	69%	5%
Quadruple test	85%	5%
Combined test	85-90%	5%
Intehrated test	85%	1.2%
NT alone	77%	4.5%

KARYOTYPING is the diagnostic test to identify downs during antenatal period. It is done by chorionic villous sampling (between 11-14 weeks) or Amniocentesis (after 16 weeks) which are invasive method.[4] CFF-DNA (cell free foetal DNA

sequencing) is a Non-invasive prenatal diagnostic test for downs syndrome done from 10 weeks. The detection rate is 100% with false positive rate of <1%. The result is given as low risk and high risk. The false positive rate for downs syndrome screening is 5%. Though amniocentesis remains the diagnosis of choice, the invasive nature and cost factor remains hinders to avail the test.The first and second trimesters screening done for this patient was positive for Down syndrome.[5] Even though she was counselled to do amniocentesis in mid trimester, she was willing to continue her pregnancy and she delivered a healthy alive term baby with no anomalies or decreased mental status. 5% prevalence of false positive screening reports should always be held in mind and the patient should be counselled regarding the same.

CONCLUSION

Thus from the above case scenario , we came to know that for every diagnostic test there will be false positive rate which should be discussed with the patient. This emphasises the importance of interpretation of the screening tests, and its benefits, risk & limitations for counselling the patients and their family for the informed decision in prenatal screening for downs syndrome.

CONFLICT OF INTEREST

Conflict of interest declared none.

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REFERENCES

1. Lin AE, Santoro S, High FA, Goldenberg P, Gutmark- Little I. Congenital heart defects associated with aneuploidy syndromes: New insights into familiar associations. In American Journal of Medical Genetics Part C: Seminars in Medical Genetics 2020; 184: 53-63.
2. Bermudez BE, DE Oliveira CM, DE Lima Cat MN, Magdalena NI, Celli A. Gastrointestinal disorders in Down syndrome. American Journal of Medical Genetics Part A. 2019 Aug;179(8):1426-31.
3. Nicham R, Weitzdörfer R, Hauser E, Freidl M, Schubert M, Wurst E, Lubec G, Seidl R. Spectrum of cognitive, behavioural and emotional problems in children and young adults with Down syndrome. In Advances in Down Syndrome Research 2003; 173-191.
4. Saida S. Predispositions to leukemia in down syndrome and other hereditary disorders. Current treatment options in oncology. 2017 Jul;18(7):1-3.
5. Wiseman FK, Al-Janabi T, Hardy J, Karmiloff-Smith A, Nizetic D, Tybulewicz VL, Fisher EM, Strydom A. A genetic cause of Alzheimer disease: mechanistic insights from Down syndrome. Nature Reviews Neuroscience. 2015 Sep;16(9):564-74.
6. B. D. Rink and M. E. Norton, "Screening for fetal aneuploidy," *Seminars in Perinatology*, vol. 40, no. 1, pp. 35–43, 2016.View at: P
7. N. J. Wald, H. S. Cuckle, J. W. Densem et al., "Maternal serum screening for Down's syndrome in early pregnancy," *BMJ*, vol. 297, no. 6653, pp. 883–887, 1988.View at: Publisher Site | Google Scholar
8. L. Hui, E. E. Muggli, and J. L. Halliday, "Population-based trends in prenatal screening and diagnosis for aneuploidy: a retrospective analysis of 38 years of state-wide data," *BJOG : An International Journal of Obstetrics and Gynaecology*, vol. 123, no. 1, pp. 90–97, 2016.View at: Publisher Site | Google Scholar
9. A. Gagnon and R. D. Wilson, "Obstetrical complications associated with abnormal maternal serum markers analytes," *Journal of Obstetrics and Gynaecology Canada*, vol. 30, no. 10, pp. 918–932, 2008.View at: Publisher Site | Google Scholar
10. J. A. Canick and A. R. Mac Rae, "Second trimester serum markers," *Seminars in Perinatology*, vol. 29, no. 4, pp. 203–208, 2005.V
11. K. Duric, S. Skrablin, J. Lesin, D. Kalafatic, I. Kuvacic, and E. Suchanek, "Second trimester total human chorionic gonadotropin, alpha-fetoprotein and unconjugated estriol in predicting pregnancy complications other than fetal aneuploidy," *European Journal of Obstetrics, Gynecology, and Reproductive Biology*, vol. 110, no. 1, pp. 12–15, 2003.View at: Publisher Site | Google Scholar
12. T. Settiyanan, C. Wanapirak, S. Sirichotiyakul et al., "Association between isolated abnormal levels of maternal serum unconjugated estriol in the second trimester and adverse pregnancy outcomes," *The Journal of Maternal-Fetal& Neonatal Medicine*, vol. 29, no. 13, pp. 2093–2097, 2016.View at: Publisher Site | Google Scholar
13. Y. Yaron, M. Cherry, R. L. Kramer et al., "Second-trimester maternal serum marker screening: maternal serum alpha-fetoprotein, beta-human chorionic gonadotropin, estriol, and their various combinations as predictors of pregnancy outcome," *American Journal of Obstetrics and Gynecology*, vol. 181, no. 4, pp. 968–974, 1999.View at: Publisher Site | Google Scholar

14. W. Signoi, "A cohort study of the association between maternal serum inhibin-A and adverse pregnancy outcomes: a population-based study," *BMC Pregnancy and Childbirth*, vol. 19, no. 1, pp. 124–130, 2019.View at: Publisher Site | Google Scholar
15. R. J. Baer, R. J. Currier, M. E. Norton et al., "Outcomes of pregnancies with more than one positive prenatal screening result in the first or second trimester," *P*
16. Epstein CJ. Down syndrome (Trisomy 21). In: Scriver CR, Beaudet AL, Sly WS, Valle D, eds. The metabolic and molecular bases of inherited disease. New York: McGraw-Hill, 1995:749–94
17. L. Say, D. Chou, A. Gemmill et al., "Global causes of maternal deaths: a WHO systematic analysis," *Lancet*, vol. 2, no. 6, pp. E323–E333, 2014.View at: Publisher Site | Google Scholar
18. World Health Organization, "Preterm birth," 2018, https://www.who.int/news-room/fact-sheets/detail/preterm-birth.View at: Google Scholar
19. M. A. Rodger, M. Carrier, G. le Gal et al., "Meta-analysis of low-molecular-weight heparin to prevent recurrent placenta-mediated pregnancy complications," *Blood*, vol. 123, no. 6, pp. 822–828, 2014.View at: Publisher Site | Google Scholar
20. M. Borenstein, L. V. Hedges, J. P. T. Higgins, and H. Rothstein, *Introduction to Meta-Analysis*, Wiley, USA, 2009. G. H. Guyatt, A. D. Oxman, G. E. Vist, and GRADE Working Group, "GRADE: an emerging consensus on rating quality of evidence and strength of recommendations," *BMJ*, vol. 336, no. 7650, pp. 924–926, 2008.View at: Publisher Site | Google Scholar
21. S. Chapman, C. Brumfield, K. Wenstrom, and M. DuBard, "Pregnancy outcomes following false-positive multiple marker screening tests," *American Journal of Perinatology*, vol. 14, no. 8, pp. 475–478, 1997.View at: Publisher Site | Google Scholar
22. T. Hsieh, T. Hung, J. Hsu, W. Shau, C. Su, and F. Hsieh, "Prediction of adverse perinatal outcome by maternal serum screening for down syndrome in an Asian population," *Obstetrics and Gynecology*, vol. 89, no. 6, pp. 937–940, 1997.View at: Publisher Site | Google Scholar
23. R. Ogle, E. Jauniaux, G. S. Pahal, E. Dell, A. Sheldrake, and C. Rodeck, "Serum screening for Down syndrome and adverse pregnancy outcomes: a case-controlled st

SP-14

Demystification of Myths on Covid-19 Vaccination - A Questionnaire Based Study

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Abstract: The study aims at demystifying the prevalent myths on COVID-19 vaccination by communicating at an individual level through an online questionnaire. The present questionnaire-based study included 100 people above 18 years and their knowledge on covid -19 vaccines were assessed and enlightened through the same online questionnaire. Covid-19 has created havoc in the entire world. Vaccination is the only solution to have a halt to this pandemic. People who took part in this study got their most common myths on covid-19 vaccines debunked.

Keywords: COVID-19, Vaccine hesitancy, Myths on Covid- 19 Vaccines.

INTRODUCTION

Vaccination is the most effective method for preventing infectious diseases. It is better to prevent rather than to cure a disease.[1]Covid – 19 pandemics is one public health crisis in history which has generated more words, more images and more sound across media than any single subject in the past. The COVID-19 Pandemic has impacted almost every corner of our life. Fortunately, COVID-19 vaccination is now paving a way for us to get out of this pandemic.[2] Herd immunity should be developed against covid-19 to restore the society to its normal status and this is possible only through vaccination. Vaccination is actually pulling the death rates down. Despite this, low vaccination rates are being reported which is mainly due to vaccine hesitancy.[3] The World Health Organisation defines vaccine hesitancy as 'delay in acceptance or refusal of safe vaccines despite availability of vaccine services'.[4]The WHO recognises Vaccine hesitancy as the world's top threat to public health safety, particularly in low middle-income countries.[5]Vaccine hesitancy is mainly due to the false beliefs that people have on COVID-19 vaccines. Myths have a cultural influence and can have a varying degree of impact over the society.[6] Therefore, prompt steps should be taken to debunk the myths in due time so that there will be an upsurge in vaccine acceptance. Anushka Ashok, a behavioural scientist has rightly said – "The problem of getting the whole world vaccinated is an unprecedented task."

MATERIALS AND METHODS

Lack of confidence in vaccine for COVId-19 poses direct and indirect threats to health and could derail efforts to end the current pandemic.[7]Vaccine acceptance and hesitancy depend upon the beliefs in vaccination, its safety, the trust placed in the system that delivers the vaccines, health issues, fear of side effects and lack of a healthcare workers recommendation.[8]Vaccine hesitancy is also due to the misinformation and conspiracy theories which are spread online. Vaccine refusal is also frequently related to philosophical beliefs and moral faiths regarding health and immunity making "natural" superior over "artificial" medicines.[9]Hence public awareness on the effectiveness of Covid- 19 vaccines and awareness on the threats of vaccine refusals should be created to end this pandemic.

STUDY DESIGN - Questionnaire based study
SAMPLE SIZE - 100people
STUDY SETTING – Online questionnaire
INCLUSION CRITERIA - People above 18 years
EXCLUSION CRITERIA – People below 18 years
PROCEDURE – – An online questionnaire which consists of the most common myths on Covid – 19 vaccines was circulated among 100 people through WhatsApp and they were asked to give their opinion regarding these myths and facts for those myths were added in the same questionnaire for them to demystify their myths.
EXPECTED OUTCOME- People who refused to take the COVID-19 vaccines should get vaccinated after filling this questionnaire.

RESULTS

On answering the questionnaire, people were enlightened with the facts on COVID - 19 vaccine.

Table 1: Most common myths and facts of Covid-19 vaccines discussed in the questionnaire

MYTHS	FACTS
1) The COVID-19 vaccine was developed rapidly and hence it is regarded unsafe for use.	The approved covid- 19 vaccines are certified as safe and effective. None of the testing steps were skipped. The clinical trials and safety reviews took about the same amount of time as any other vaccines.
2) The COVID-19 vaccine causes	The vaccine developers state that some people experience pain at the site of injection, body

serious side effects such as allergic reaction.	aches, headaches and low grade fever lasting for a day or two.These signs indicate that the vaccine is working. If any of these symptoms lastsfor more than two days, a doctor must be consulted. Rarely people can develop severe allergic reactions to the ingredients used in the vaccine. Hence people with a history of anaphylaxis — to the ingredients of the vaccine should not be vaccinated.
3) The COVID-19 vaccine affects the fertility of women.	The vaccine tells the body to make copies of the spike protein present on the coronavirus's surface. This "teaches" the body's immune system to fight against the virus that has that specific spike protein on it. A fake report surfaced on social media stating that the spike protein on this coronavirus was the same as another spike protein called syncitin-1 which is involved in the growth and attachment of the placenta during pregnancy. The report said that getting COVID-19 vaccine would cause a woman's body to fight against syncytin-1 and affect her fertility. The two spike proteins are entirely different and distinct, and getting the COVID-19 vaccine will not cause infertility. Covid- 19 vaccines do not cause any harm to the women's reproductive organs and hence does not affect her fertility.
4) The COVID-19 vaccine alters the DNA.	The vaccine contains messenger RNA (mRNA), which instructs the human body to create the "spike protein" present on the new corona virus surface. When the immune system recognises this protein, it builds an immune response by creating antibodies against it. The mRNA never enters the nucleus of the cell were our DNA is present. The body gets rid of the mRNA once it has finished using the instructions.
5) It is possible to develop vaccines for HIV and other diseases as we have developed vaccine for covid – 19.	Developing vaccines for certain diseases is difficult. For example, the HIV virus can hide itself from the human immune system, and this makes it difficult to develop vaccine for it. But now they have started the process of vaccine preparation for HIV.
6) Once vaccinated you will test positive for COVID-19.	Viral tests used to diagnose COVID-19 check samples from the respiratory system for the presence of the live covid- 19 virus. Since there is no live virus in the vaccines, the vaccines will not affect your test result. But it is possible to get infected with the virus before the vaccine has had time to fully protect your body. If your body develops an immune response to vaccination, which is the goal, you may test positive on some antibody tests. Positive Antibody tests indicate you had a previous infection but that does not indicate a current infection.
7) Once vaccinated you no longer need to wear a mask.	Wearing masks, frequent handwashing and social distancing must be followed until a sufficient number of people become immune to the virus.
8) If already recovered from COVID-19, no need of vaccine.	Though you have got COVID-19 infection in the past, you can still be benefitedby getting vaccinated. Natural immunity against covid-19 varies from person to person. Evidences state that natural immunity may not last for a long period of time. So, it is better to get vaccinated.
9) The mRNA technology used to develop Covid- 19 vaccine is new.	The mRNA technology has been in development for decades. Vaccine makers developed the technology in order to respond quickly to a new pandemic illness like COVID-19.
10) One dose of Covid- 19 vaccine will protect you against COVID-19 disease.	The human body needs two doses of the Covid-19 vaccine -- the first "prime dose" as well as the second "booster dose" to produce enough antibodies against the coronavirus disease.
11) People of certain blood groups will have less severe COVID- 19 infection, so they need not get vaccinated.	Studies tell us not to believe that people belonging to a certain blood group will have less severe COVID-19 disease.
12) COVID-19 vaccines contains microchip in it.	Neither of the Covid- 19 vaccines contain any metal - based ingredients nor it delivers any microchip into the human body.
13)Everyone should wait until a more effective vaccine is developed.	All COVID-19 vaccines are proven efficient. So, get the vaccine available in your locality.
14) The participants enrolled for the clinical trials were less.	Thousands of participants were enrolled for the clinical trials. The participants were followed for two months after taking their second dose which is common with vaccine trials.
15) You will not get COVID-19 disease once your vaccinated.	Vaccination will prevent you from getting Covid- 19 infection but it is possible to get infected with the coronavirus (SARS-CoV-2).
16) Can we delay the routine vaccinations until the Covid- 19 pandemic is over?	Routine vaccination should not be delayed because of the COVID-19 pandemic as it is an essential preventive care service for all ages.
17) Can pregnant women and	Pregnant women and lactating mothers can also get vaccinated for COVID-19. Pregnant

lactating mothers get vaccinated for Covid- 19?	women can consult their doctor before taking the vaccine.
18) Can you donate blood after COVID-19 vaccination?	The NEGVAC in its recommendations said that an individual can donate blood after 14 days of receiving COVID-19 vaccine.
19) Can we get vaccinated for Covid-19 during menstruation?	You can take your COVID-19 vaccine during menstruation.
20) Do COVID-19 vaccines cause heart attacks?	Dr Maulik Patel, a consultant physician in Divine life hospital Adipur, Kutch states that "there is no link between heart attacks with COVID vaccines. No other major vaccine-associated adverse events like heart attacks were identified in post-vaccine surveillance."

Table 2: Details of the vaccination status of the participants of the study

Only 1st Dose of vaccine taken	67% of the participants
Both doses of vaccine taken	23% of the participants
Not vaccinated	10% of the participants
Reasons given by the participants of the study for not getting vaccinated	Fear of the side effects of vaccination Previous COVID infection Unavailability of vaccines

Out of 100 participants, 67% of them received only their first dose of covid vaccine, 23% of them received both doses of vaccine and 10% of them are not vaccinated. According to our study the reasons for not getting vaccinated are the fear of side effects of vaccination, past Covid infection and unavailability of vaccines.

DISCUSSION

The Covid-19 outbreak has shattered our lives. The current coronavirus disease 2019 (COVID19) pandemic is one of the international crises, and researchers are working together to develop a safe and effective COVID-19 vaccine.[10]Vaccine hesitancy plays a vital role in preventing the restoration of the society. The refusal to vaccinate for disapproval of the COVID19 vaccine offered in the country's vaccination program could be a reason for people's refusal to vaccinate and could threaten herd immunity.[11]As research evidence on various aspects of COVID 19 is accelerating, we must not recognize the potential facts about this disease and believe in facts that have no genuine evidence or are not claimed by the international health authorities.[12]The refusal to vaccinate for disapproval of the COVID19 vaccine offered in the country's vaccination program could be a reason for people's refusal to vaccinate and could threaten herd immunity.[13]Urge partnership of researchers and local health workers to coordinate culturally appropriate community vaccination education/promotion programs.[14]Myths on vaccination that prevails among the common people pulls down the vaccination rates and elevates the death rates despite the availability of vaccines.[15] Based on our study, insufficient availability of vaccines in some areas is also one main reason for low vaccination rates next to the myths. Therefore, the process of debunking the myths has to be intensified and fastened to decrease the spread of Covid infection. More and more awareness program is the needs of the hour.Those who have taken both doses of the vaccine have less symptoms and hardly require hospitalization.

CONCLUSION

The arrival of vaccines for the Covid -19 infection is hopefully like the light at the end of the tunnel. So, it is crucial to enrich our knowledge on covid-19 vaccines and make the right decision regarding vaccination and should join hands to put an end to this pandemic. As budding physicians medical students can play their role in spreading the word and gaining success in vaccinating the society and save them from trauma.

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There are no conflicts of interest.

REFERENCES

1. Biswas N, Mustapha T, Khubchandani J, Price JH. The nature and extent of COVID-19 vaccination hesitancy in healthcare workers. Journal of community health. 2021 Dec;46(6):1244-51.
2. Archila PA, Danies G, Molina J, Truscott de Mejía AM, Restrepo S. Towards Covid-19 literacy. Science & Education. 2021 Aug;30(4):785-808.

3. Arshad MS, Hussain I, Mahmood T, Hayat K, Majeed A, Imran I, Saeed H, Iqbal MO, Uzair M, Ashraf W, Usman A. A National Survey to Assess the COVID-19 Vaccine-Related Conspiracy Beliefs, Acceptability, Preference, and willingness to pay among the general population of Pakistan. Vaccines. 2021 Jul;9(7):720.
4. Mac Donald NE, SAGE Working Group on Vaccine Hesitancy. Vaccine Hesitancy: Definition, scope and determinants. Vaccine 2015; 33:4161-4.
5. Ullah I, Khan KS, Tahir MJ, Ahmed A, Harapan H. Myths and conspiracy theories on vaccines and COVID-19: potential effect on global vaccine refusals. Vacunas. 2021 May 1;22(2):93-7.
6. Lockyer B, Islam S, Rahman A, Dickerson J, Pickett K, Sheldon T, Wright J, McEachan R, Sheard L, Bradford Institute for Health Research Covid- 19 Scientific Advisory Group. Understanding COVID- 19 misinformation and vaccine hesitancy in context: Findings from a qualitative study involving citizens in Bradford, UK. Health Expectations. 2021 Aug;24(4):1158-67.
7. Rutten LJ, Zhu X, Leppin AL, Ridgeway JL, Swift MD, Griffin JM, St Sauver JL, Virk A, Jacobson RM. Evidence-based strategies for clinical organizations to address COVID-19 vaccine hesitancy. InMayo Clinic Proceedings 2021 Mar 1 (Vol. 96, No. 3, pp. 699-707). Elsevier.
8. maryam Fatima S, Tariq S. Infodemic as subsequent defiance to manage pandemic: Assessing the reasons behind COVID-19 vaccine refusal. BioSight. 2021 Dec 31;2(2):22-9.
9. Razai MS, Chaudhry UA, Doerholt K, Bauld L, Majeed A. Covid-19 vaccination hesitancy. bmj. 2021 May 20;373.
10. Ullah I, Khan KS, Tahir MJ, Ahmed A, Harapan H. Myths and conspiracy theories on vaccines and COVID-19: Potential effect on global vaccine refusals. Vacunas. 2021 May 1;22(2):93-7.
11. Nichter M. Vaccinations in the Third World: a consideration of community demand. Social science & medicine. 1995 Sep 1;41(5):617-32.
12. Ullah I, Khan KS, Tahir MJ, Ahmed A, Harapan H. Myths and conspiracy theories on vaccines and COVID-19: Potential effect on global vaccine refusals. Vacunas. 2021 May 1;22(2):93-7.
13. Kumar L, Naik Z, Panwar A, Sridhar M, Keluskar V. Knowledge, Attitude, and Practice among the Healthcare Professionals regarding the myths on COVID-19 vaccination-Demystified. medRxiv. 2021 Jan 1.
14. Wong LP, Alias H, Danaee M, Ahmed J, Lachyan A, Cai CZ, Lin Y, Hu Z, Tan SY, Lu Y, Cai G. COVID-19 vaccination intention and vaccine characteristics influencing vaccination acceptance: a global survey of 17 countries. Infectious diseases of poverty. 2021 Dec;10(1):1-4.
15. Streefland PH. Public doubts about vaccination safety and resistance against vaccination. Health policy. 2001 Mar 1;55(3):159-72.

SP-15

Case Report – Ectodermal Dysplasia

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Abstract: Ectodermal dysplasia is a rare genetic disorder which can be of various types. Hypohidrotic Ectodermal Dysplasia manifests as a triad of partial or complete absence of sweat glands, anomalous dentition and hypotrichosis.[3]Classic facial features include frontal bossing, prominent supraorbital ridge, small chin, low, depressed nasal bridge, and prominent lips. It is the most common with a frequency of 1 per 17000 live births. Mortality in the first three years of life is as high as13%due to complications of hyperthermia, failure to thrive, and respiratory infections. Otherwise, life expectancy is normal. To enable early diagnosis and prompt management of inherited disorders like ectodermal dysplasia. This is a case report of a 13-month-old child who presented with incomplete hair growth and hair loss over scalp and eyebrows and on examination, Vellus hair and multiple papules on scalp were seen along with findings like frontal bossing, hypertelorism, depressed nasal bridges were noted which was diagnosed as ectodermal dysplasia. General abnormalities seen in ectodermal dysplasia include a wide range of clinical findings like small primary teeth, defective enamel, atopic dermatitis, xerosis, abnormal quantity, structure and quality of hair, thin slow growing hair often involving scalp, eyebrows and eyelashes, brittle nails, hypo hidrosis /hyperhidrosis of palms and soles and other systemic symptoms like wheezing. Management of hypohidrotic ED is a challenge due to heat intolerance and their susceptibility to pulmonary infections. The parents were counselled about the various aspects that can be encountered in this disorder. Early dental evaluation and treatment is important and helps with language development, mastication, and cosmesis. Asthma and recurrent respiratory infections should be treated appropriately and referral to a pulmonologist may be warranted.

Keywords: Hypohidrotic ectodermal dysplasia, hypotrichosis, vellus hair, Hari Loss, papulse.

INTRODUCTION

Ectodermal dysplasia is a heterogenous group of disorders characterized by a constellation of findings involving two or more of: teeth, skin and its appendages like hair, nails and eccrine and sebaceous glands.[1] The estimated incidence is 3.5 in 10,000 individuals. It affects the epidermis, in which it is responsible for development of keratinocytes and causes abberations in the hair, sebaceous glands, eccrine and apocrine glands, nails, teeth and the ear. The pharyngeal and laryngeal mucosa may be so, atrophic that it results in dysphonia and hoarseness of voice.[2]General abnormalities seen in ectodermal dysplasia include a wide range of clinical findings like small primary teeth, defective enamel, atopicdermatitis, xerosis, abnormal quantity, structure and quality of hair, thin slow growing hair often involving scalp, eyebrows and eyelashes, brittle nails, hypo hidrosis /hyperhidrosis of palms and soles and other systemic symptoms like wheezing.[3] Ectodermal dysplasia can be of various types, such as, Hypo hidrotic ED, Hidrotic ED, Wiktop tooth and nail syndrome, EEC, AECand RHS, Adult.[4]All the above mentioned variants are of Autosomal Dominant inheritance except for the hypo hidrotic ED which can be of XLR, AD, ARinheritance,each with some characteristic findings of their own[5]. Hypohidrotic ED manifests as a triad of partial or complete absence of sweat glands, anomalous dentition and hypotrichosis.[3] It is the most common with a frequency of 1 per 17000 live births.[6]

CASE REPORT

Presenting complaints

A 13-month-old male child presented to OPD with the complaints of loss of hair over scalp and eyebrows for 1 month.

History

Mother gave history of incomplete growth of hair after which there was hair loss. Mother also gave a history of inability to tolerate heat, history of dryness and warmth of skin.

Natal and post natal history

There was no history of consanguinity, still birth or IUD. No history of collodian baby. It was a full term normal vaginal delivery with a birth weight of 3.3 kg and the postnatal period was uneventful except for the history of presence of scaling at birth.

Family history

There was no similar complaints running in the family.

Observations

On examination, the child weighs 11kg and has a height of 74 cm with height for age falling between 25th and 50th percentile and weight for age falling between 75th and 90th percentile. Head to toe: Vellus hair and multiple papules on scalp were seen[Table/Fig-1] along with findings like frontal bossing, hypertelorism, depressed nasal bridges were noted[Table/Fig-2]Dentition could not be assessed. Nails were found to be normal.

Fig-1: Showing multiple papules and vellus hair

Fig-2: Showing frontal bossing and depressed nasal bridge

Special tests: Dermoscopy findings include multiple white dots, vellus hair and few scales.
Diagnosis: The diagnosis is mostly based on history and clinical examination. Additional tests like sequence analysisand molecular genetic testing can be carried out.
Prognosis: Genetic transmission was explained and prognosis for this particular type of ectodermal dysplasia is good
Treatment: Counselling was given thoroughly and the cosmetology treatments explained to the parents if need arise in the future.

DISCUSSION

We hereby report a case of 13 month old male child hair loss and dryness of skin examination was found to have multiple papules on the scalp along with vellus hair. Among the various types of ectodermal dysplasia, this case was clinically diagnosed as hypohidrotic ectodermal dysplasia where there is absence of sweat glands, anomalous dentition and hypotrichosis. There are only a few reported cases of hyperhidrosis ectodermal dysplasia presenting as loss of hair and dryness of skin. Children with congenital or craniofacial defect are unique, and oral problems must be evaluated individually to provide the most ideal treatment. Ninety-five percent of hypohidrotic ectodermal dysplasia cases are inherited as an X-linked recessive disorder.[7]Therefore, the disease is only fully manifest in affected males and female carriers are more mildly affected. All daughters born to an affected male will be a carrier. None of the sons of an affected male will be affected. Each child of a carrier female has a 50% possibility of inheriting the mutation.[8] Unusual facial features exacerbate the social challenges of meeting new people. [9]Lowered self-esteem, speech defects, decreased academic performance and social isolation may result from merely looking different from one's peers.This results in significant improvement in esthetics, masticatory and phonetic function.[10] There can be complications like inability to perspire causing hyperthermia, which may lead to febrile seizures and neurologic damage. Decreased secretions can also lead to xerostomia, xerophthalmia, thick nasal secretions, excessive cerumen, hoarse voice, respiratory infections, and dysphagia.[11] Feeding issues, weight deficits and failure to thrive can be seen in affected infants and children. An increased incidence of atopy is also noted in affected individuals, including eczema, wheezing, asthma, food allergy and abnormal immunoglobulin production.[12] Mortality in the first three years of life is as high as 13% due to complications of hyperthermia, failure to thrive, and respiratory infections. Otherwise, life expectancy is normal.[13] Hence it is important that such cases have to be studied more and in detail where in counselling and prevention of secondary infections is of utmost importance.

CONCLUSION

Management of hypohidrotic ED is a challenge due to heat intolerance and their susceptibility to pulmonary infections. The parents were counselled about the various aspects that can be encountered in this disorder.Education of the patient and family regarding the condition is necessary. Patients should be instructed regarding the signs of overheating. They should be encouraged to modify their activities when necessary and to be aware of their body's limitations when at risk for hyperthermia.The importance of recognizing and promptly treating hyperthermia, failure to thrive and respiratory infections in affected infants and toddlers should be stressed as these issues can lead to mortality. Multidisciplinary care is important in this condition and follow-up with multiple specialists may be required.[14] Symptomatic treatment is warranted for the skin findings and co-morbid diseases. Dental referral is warranted in all cases. Other specialty consultations are warranted based on individual symptoms.Atopic dermatitis can be appropriately managed with topical steroids, topical immunomodulators and antihistamines by a dermatologist. Potential increased risk of melanoma has been reported, so an annual full body skin examination is also advised. Early dental evaluation and treatment is important and helps with language development, mastication, and cosmesis. Dentures in childhood and orthodontia or dental implants are options for treatment in older individuals.Dry eyes and thick nasal secretions can be treated with saline sprays. Consultation with ophthalmology and otolaryngology may be warranted.Hearing and speech evaluations are recommended in affected children.[15] Asthma and recurrent respiratory infections should be treated appropriately and referral to a pulmonologist may be warranted.Xerostomia can be treated with saliva substitutes or sialagogues.Weight deficits can be managed by high caloric diets. Gastroenterology or nutrition consultation may be indicated in cases of failure to thrive.[16] Genetics evaluation can be helpful in coordination of care, genetic counseling and molecular testing.Recently, implant borne total telescopic dentures have been described as a possible treatment strategy. But high- cost difficulties in placement and high failure rate make their use questionable. [17]

CONFLICT OF INTEREST

Conflict of interest declared none.

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REFERENCES

1. Behrman RE, Vaughan III VC. Nelson textbook of pediatrics. WB Saunders company; 1983.
2. Trzeciak WH, Koczorowski R. Molecular basis of hypohidrotic ectodermal dysplasia: an update. Journal of applied genetics. 2016 Feb;57(1):51-61.
3. Bhakta P, Barthunia B, Nigam H, Pawar P. Ectodermal dysplasia-A rare case report. Journal of family medicine and primary care. 2019 Sep;8(9):3054..
4. Wright JT., Grange DK., Ritcher MK. Hypohidrotic ectodermal dysplasia. Gene Reviews. NCBI BookShelf. *Updated.* 2009
5. Finn SB. *Clinical pedodontics.* 3rd ed. Philadelphia: Saunders; 1967.
6. Oliver RD, Fye WN, Halin JA, Steiner JF. Prosthetic management in anhidrotic ectodermal dysplasia: report of case. *J Dent Children.* 1975;43:375–378.
7. Tarjan I, (Department of Pediatric Dentistry and Orthodontics, Semmelweis University, Budapest, Hungary. tarjan@fok.usn.hu), Gabris K, Rozsa N. Early prosthetic treatment of patients with ectodermal dysplasia: A clinical report. *J Prosthet Dent.* 2005 May;93(5):419–424.
8. Lexner MO, (Department of Pediatric Dentistry and Clinical Genetics, University of Copenhagen, Copenhagen, N, Denmark. mol@odont.ku.dk), Bardow A, Juncker I, Jensen LG, Almer L, Kreiborg S, Hertz JM. X-linked hypohidrotic ectodermal dysplasia. Genetic and dental findings in 67 Danish patients from 19 families. *Clin Genet.* 2008 Sep;74(3):252–259.
9. Franchi L, (Department of Orthodontics, University of Florence), Branchi R, Tollaro I. Craniofacial changes following early prosthetic treatment in a case of hypohidrotic ectodermal dysplasia with complete anodontia. *ASDC J Dent Child.* 1998 Mar-Apr;65(2):116–121.
10. Pabst HF, Groth O, McCoy EE. Hypohidrotic ectodermal dysplasia with hypothyroidism. *J Pediatr.* 1981 Feb;98(2):223–227.

11. Gopinath VK, (Department of Pedodontics, Meenakshi Ammal Dental College, Chennai. gopinathvk@yahoo.com), Manoj KM, Mahesh K. Hypohidrotic ectodermal dysplasia: a case report. *J Indian Soc Pedod Prev Dent.* 1999 Sep;17(3):90–92.
12. Hall KR. *Pediatric orofacial medicine and pathology.* 4th ed. London: Chapman and Hall; 1994. p. 163 p.
13. Itthagarun A, (Department of Children's Dentistry and Orthodontics, Faculty of Dentistry, University of Hong Kong), King NM. Ectodermal dysplasia: A review and case report. *Quintessence Int.* 1997 Sep;28(9):595–602.
14. Gorlin RJ., Pindborg JJ., Cohen MM. *Syndromes of head and neck.* 20th ed. New York: Mc Graw Hill; 1976. pp. p. 379–385.
15. Hickey AJ, (Maine Medical Center, USA. ajhfam@maine.rr.com), Salter M. Prosthodontic and psychological factors in treating patients with congenital and craniofacial defects. *J Prosthet Dent.* 2006 May;95(5):392–396.
16. Kramer FJ, (Department of Oral and Maxillofacial Surgery, Georg-August-University of Goettingen, Goettingen Germany. franz-josef.kramer@med.uni-goettingen.de), Baethge C, Tschernitschek H. Implants in children with ectodermal dysplasia: a case report and literature review. *Clin Oral Implants Res.* 2007 Feb;18(1):140–146.
17. Suprabha BS, (Department of Pedodontics and Preventive Dentistry, College of Dental Surgery, Mangalore). Hereditary ectodermal dysplasia: A case report. *J Indian Soc Pedod Prev Dent.* 2002 Mar;20(1):37–40.

SP-16

Baseline Electrolytes Abnormality in Covid-19 Viral Pneumonia

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Abstract: Electrolyte imbalance is not uncommon in COVID-19 patients. Many studies have been done to show various electrolyte imbalance like hyponatremia, hypokalemia and hypocalcemia that can occur, but no studies have been done to show the association of baseline serum electrolytes abnormality and outcome in COVID patients. To study the association of baseline electrolyte abnormalities and unfavorable outcome in COVID-19 viral pneumonia. Retrospective observational study involving 198 individuals of >18 years of age, admitted in COVID-19 ward and ICU of Sree Balaji Medical College and Hospital. Baseline serum sodium, serum potassium and serum chloride of such patients were assessed before starting treatment. Patients were divided into two group, group-1 includes patients with no electrolyte abnormality (NE) and group-2 includes patients with electrolyte abnormality (E). Association of baseline electrolytes abnormality on requirement of oxygen support, non-invasive or invasive mechanical ventilation, disease severity, duration of hospital stay, other markers of severity and treatment outcome were evaluated and statistically analyzed between the groups. Group-2 had statistically significant elevation in LDH with p value 0.0003 and requirement of NIV or mechanical ventilation with p value 0.027 and had a greater number of elder populations. Most of the patients have at-least one electrolyte abnormality at presentation, most common being hyponatremia. Patients with electrolyte abnormality more often required ICU and assisted MV, and longer duration of hospital admission and higher mortality rate. Hyponatremia is one of an separate risk factor for death in COVID-19 patients. Baseline serum electrolyte imbalance is related to unfavorable prognosis in COVID and early assessment of serum electrolytes will be helpful in evaluating the risk of severe COVID.

Keywords: Covid-19, Electrolyte Imbalance, Hyponatremia, Severe Covid, Viral Pneumonia.

INTRODUCTION

Corona virus disease of 2019 is caused by severe acute respiratory syndrome coronavirus 2. The novel SARS-COV-2 disease pandemic was first recorded in China[1] and is still not under control worldwide. The most common initial symptoms are cough, fatigue, headache, fatigue, myalgias, and diarrhoea. Approximately after 1 week of onset of symptoms severe illness presentation occurs.[1] Various manifestations have been seen in COVID-19, can involve multiple systems like respiratory, cardiac, nervous, renal, and gastrointestinal and coagulation system. Respiratory system remains as the primary target. Prognosis and severity of presentation also depends on demographic, clinical and laboratory parameters. Severe and unfavorable outcomes were associated with clinical features and laboratory signs of inflammation, including high grade fevers, elevated levels of serum ferritin, C- reactive proteins and interleukin-6, coagulation parameters abnormality like raised D-dimer levels, abnormal liver and renal function test. Most of the hospitalized patients had altered renal function during the disease course, most often presents with acute kidney injury, hematuria, proteinuria, and electrolyte imbalance.[2] Meta-analysis has showed the association of low levels of serum sodium, serum potassium and serum calcium and severe covid disease[3], but no studies have been done on association of baseline serum electrolyte with survival and disease severity. This study, the initial serum electrolyte level of covid positive cases before starting any treatment is taken into consideration and its association with the disease severity, duration of hospital stay and outcome are noted.

METHOD

Around 198 individuals above 18 years of age who are hospitalized based on AIIMS criteria during the months of April to June of 2021, with RTPCR positive or having signs of COVID viral pneumonia in CT chest are retrospectively included in the study and patient who were >18 years and discharged against medical advice were excluded from the study. Details on patient's age, sex, co-morbidities, COVID-19 RTPCR test results, CT severity score, disease severity based on baseline serum electrolytes such as sodium, potassium and chloride and other biochemical parameters like LDH, D-dimer, serum ferritin, CRP and blood total count, requirement of oxygen support, non-invasive ventilation, mechanical ventilation, ICU admission, duration of hospital stay and outcome were collected retrospectively from hospital medical records. Written informed consent was waived off as all the data was collected from the hospital medical records, and patient's identify is not disclosed. Serum electrolytes, LDH, total counts and CRP levels were classified based on the laboratory reference range - normal, high or low. Serum ferritin and D-dimer levels were classified based on its relation to unfavorable prognosis, 1000mg/L for D-dimer and 300mg/ml for serum ferritin. Based on serum electrolyte levels, patients were grouped into group-1 and group-2. Primary end points were,duration of hospitalization, requirement of mechanical ventilation, ICU admission and patient's final outcome. Both the groups were then compared based on the primary end points.

Limitation

The small sample size is a limitation in this study.

Statistical Outcome

Unpaired sample t-test- used to find the significant difference between the bivariate samples in independent groups and for categorical data Chi-Square test was used. Probability value .05 - significant level.
NE- NORMAL ELECTROLYTE LEVEL – GROUP 1
E- ELECTROLYTE IMBALANCE- GROUP-2

Table I: OXYGEN SUPPORT

			Groups		Total
			NE	E	
O2 SUPPORT	no	Count	17	67	84
		%	44.7%	41.9%	42.4%
	yes	Count	21	93	114
		%	55.3%	58.1%	57.6%
Total		Count	38	160	198
		%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	p-value	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.103[a]	1	.748		
Continuity Correction[b]	.019	1	.890		
Likelihood Ratio	.103	1	.749		
Fisher's Exact Test				.855	.443
N of Valid Cases	198				

Table II: VENTILATOR / NIV SUPPORT

			Groups		Total
			NE	E	
VENTILATOR/NIV	No	Count	35	120	155
		%	92.1%	75.0%	78.3%
	Yes	Count	3	40	43
		%	7.9%	25.0%	21.7%
Total		Count	38	160	198
		%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymptotic Significance (2-sided)	p-value	Exact Sig. (1-sided)
Pearson Chi-Square	5.285[a]	1	.022		
Continuity Correction[b]	4.327	1	.038		
Likelihood Ratio	6.291	1	.012		
Fisher's Exact Test				.027	.014
N of Valid Cases	198				

Table III: ICU ADMISSION

			Groups		Total
			NE	E	
ICU ADMISSION	No	Count	32	124	156
		%	84.2%	77.5%	78.8%
	Yes	Count	6	36	42
		%	15.8%	22.5%	21.2%
Total		Count	38	160	198
		%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	p-value	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.827[a]	1	.363		
Continuity Correction[b]	.475	1	.491		
Likelihood Ratio	.874	1	.350		
Fisher's Exact Test				.508	.250
N of Valid Cases	198				

Table IV: OUTCOME

			Groups		Total
			NE	E	
Outcome	Alive	Count	33	121	154
		%	86.8%	75.6%	77.8%
	Dead	Count	5	39	44
		%	13.2%	24.4%	22.2%
Total		Count	38	160	198
		%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	p-value	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	2.235[a]	1	.135		
Continuity Correction[b]	1.634	1	.201		
Likelihood Ratio	2.454	1	.117		
Fisher's Exact Test				.192	.097
Linear-by-Linear Association	2.224	1	.136		
N of Valid Cases	198				

Table V: LDH

			Groups		Total
			NE	E	
LDH	Low	Count	0	7	7
		%	0.0%	5.0%	4.2%
	Normal	Count	18	38	56
		%	66.7%	27.0%	33.3%
	Elevated	Count	9	96	105
		%	33.3%	68.1%	62.5%
Total		Count	27	141	168
		%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	p-value
Pearson Chi-Square	16.443[a]	2	.0003
Likelihood Ratio	16.370	2	.000
Linear-by-Linear Association	6.124	1	.013
N of Valid Cases	168		

Table VI: HOSPITAL STAY DURATION

			Groups		Total
			NE	E	
Hospital stay	<= 10 days	Count	29	115	144
		%	76.3%	71.9%	72.7%
	> 10 days	Count	9	45	54
		%	23.7%	28.1%	27.3%
Total		Count	38	160	198
		%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	p-value	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.305[a]	1	.581		
Continuity Correction[b]	.122	1	.726		
Likelihood Ratio	.312	1	.576		
Fisher's Exact Test				.687	.370
Linear-by-Linear Association	.304	1	.582		
N of Valid Cases	198				

The comparison of ventilator/ NIV requirement with Group by Pearson's Chi-square test, which shows statistical significance between the O2 support with Group , $\chi^2 = 5.285$ and $p = 0.027 < 0.05$

The comparison O2 support with Group by Pearson's Chi-square test, which shows no statistical significance between the ICU admission, outcome and duration of hospital stay.

The comparison LDH with Group by Pearson's Chi-square test, which shows high statistical significance between the O2 support with Group , $\chi^2 = 16.443$ and $p = 0.0003 < 0.05$

Table VII: T TEST FOR OTHER VARIABLES

Independent Samples Test

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	p-value	Mean Difference	Std. Error Difference	Lower	Upper
AGE	Equal variances assumed	1.721	.191	2.185	196	.030	5.2276	2.3925	.5093	9.9460
CT score	Equal variances assumed	1.313	.254	-.399	111	.691	-.5238	1.3141	-3.1278	2.0802
RR	Equal variances assumed	3.549	.061	1.197	195	.233	.7713	.6446	-.5000	2.0425
ESR	Equal variances assumed	.715	.399	.172	173	.864	.7159	4.1624	-7.4998	8.9316
CRP	Equal variances assumed	1.252	.265	.769	163	.443	8.33386	10.83055	-13.05242	29.72014
LDH	Equal variances not assumed	4.240	.041	2.545	45.053	.014	65.9858	25.9239	13.7742	118.1974
TOTAL COUNT	Equal variances assumed	.861	.355	.577	183	.564	910.5867	1577.5074	-246.168	4023.0275
FERRITIN	Equal variances assumed	1.413	.236	1.147	169	.253	341.22238	297.54865	-246.16	928.61331
D-DIMER	Equal variances assumed	3.256	.073	1.324	175	.187	555.19814	419.25789	-246.168	1382.65075
DAYS OF REQUIRING O2	Equal variances assumed	.003	.957	.894	116	.373	1.2339	1.3808	-1.5009	3.9688

The comparison of age with Groups by Unpaired t-test, shows statistically significant difference, t -value = 2.185 and p = 0.030 < 0.05.

The comparison of LDH with Groups by Unpaired t-test , shows statistically significant difference,t -value = 2.545 and p = 0.014 < 0.05.

The comparison of CRP, D-dimer, ferritin, ESR and TC with groups by Unpaired t-test, showed no statistical significance.

RESULT

Around 199 patients admitted during the period of April to July were taken retrospectively by random convenient sampling. Out of 198, 160 had atleast one electrolyte imbalance. Hyponatremia was the most common electrolyte imbalance seen. On comparing both the groups, group-2 (with electrolyte imbalance) had statistically significant elevation in LDH with p value 0.0003, requirement of NIV or mechanical ventilation were significantly more with p value 0.027 and hada greater number of elder populations. All though other variables of severity, Oxygen requirement, duration of hospital stay, ICU admission, mortality rate was not statistically significant but was numerically higher in group 2. Based on the statistical data, patients belonging to group-2has prognosed to severe disease. Hence baseline serum electrolyte can be taken as a predictor of severity, especially hyponatremia.

DISCUSSION

Globally, as of 7 June 2021, there have been 172,956,039 confirmed cases of COVID-19, including 3,726,466 deaths.[4] In India, from 3 January 2020 to 7 June 2021, there have been 28,909,975 confirmed cases of COVID-19 with 349,186 deaths, whereas in Tamil Nadu 2,274,704 confirmed with 27765 deaths has been reported to WHO. The most common initial symptoms are cough, headache, fatigue, myalgias, fever, and diarrhea. Approximately 1 week after the onset of symptoms severe illness

presentation occur. The majority of severe Covid-19 patients have low lymphocytes and thromboembolic complications, and also disorders affecting the central or peripheral nervous system, acute injurycardiac/kidney/liver, along with cardiac arrhythmias, rhabdomyolysis, coagulopathy, and shock.[5] These multiple organ failures may be linked with clinical and laboratory signs of inflammation, such as high temperature, low platelets, elevated levels of serum ferritin, C- reactive proteins and interleukin-6. Renal impairment in covid-19 is one of the common manifestations. Virus enters the cell by directly binding to ACE2 receptors.[6] and serine protease family which is abundant in kidney cells such as podocytes and tubule epithelial cells.[7] Filtration process of absorption and secretion is mainly by podocytes and straight cells of proximal tubular, hence cytopathic damage to these cells can cause electrolyte imbalance. Fluid imbalance occurs due to fever or decreased fluid intake in patients can also be a cause for renal impairment in COVID-19[8], which can lead to electrolyte imbalance. Corona virus can also affect the GI tract epithelial cells which is mediated by spike protein on the viral coating initiated by the cellular transmembrane serine protease 2and can cause GI symptoms[9], fluid imbalance and difficulty in absorption of nutrients. Few drugs which were previously prescribed for treating covid pneumonia like chloroquine and hydroxychloroquine[10] and drugs that can inhibit RAS system (reduces aldosterone production) can also cause electrolyte imbalance. Most common electrolyte disturbance is hyponatremia which is also associated with increased severity and mortality. Hypernatremia is also seen in some patients and is associated with increased duration of hospital stay and increased risk of death. Hypokalemia is also a complication of COVID, which can exacerbate acute respiratory distress syndrome and also can cause myocardial injuries.[11] So, both sodium and potassium are considered as a significant indicator of severity in COVID-19 patients. Electrolyte imbalance can lead to many complications so it is important to diagnose and treat electrolyte imbalance early. Most fatal complication of hyponatremia is acute cerebral edema[12]. Seizure, mood disorders, rhabdomyolysis and coma are some other complications of hyponatremia. Rapid correction of sodium levels can also cause demyelinating osmotic syndrome. Hypokalemia if not treated can cause cardiovascular dysfunction and neurohormonal activation. Imbalance in chloride levels is associated with increased risk of AKI, morbidity and mortality.[13] Electrolyte imbalance can also be associated with hypovolemia or hypervolemia, if not treated can cause ischemic injury of vital organs, multiple organ failure and severe disease.[14.15]

CONCLUSION

Hyponatremia is an independent factor related to death in COVID patients. Baseline serum electrolyte imbalance is related to unfavorable prognosis in COVID-19 and early assessment of serum electrolytes will be helpful in evaluating the risk of severe COVID.

CONFLICT OF INTEREST

Conflict of interest declared none.

Funding for the Study

None

Author Contributions

Dr PON DIVYA BHARATHI and Dr MANIMEKALAI are the co-first authors. They are responsible for the integrity of the data and had full access to all data in the study.

Study concept and Designing: Dr VINATHA

Acquisition, analysis and interpretation of data: Dr PON DIVYA BHARATHI

Critical revision of manuscript: Dr VINATHA

Supervision: Dr MANIMEKALAI

REFERENCES

1. 1.Rothan HA, Byrareddy SN. The epidemiology and pathogenesis of coronavirus disease (COVID-19) outbreak. J Autoimmune 2020;109:102433
2. Pei G, Zhang Z, Peng J, Liu L, Zhang C, Yu C, et al. Renal involvement and early prognosis in patients with COVID-19 pneumonia. J Am Soc Nephrol 2020. epub ahead of print.
3. Lippi G, South AM, Henry BM. Electrolyte imbalances in patients withsevere coronavirus disease 2019 (COVID-19). Ann Clin Biochemis 2020. 4563220922255.
4. WHO Coronavirus (COVID-19) Dashboard | WHO Coronavirus (COVID-19) Dashboard With Vaccination Data
5. Guo T, Fan Y, Chen M, et al. Cardiovascular implications of fatal outcomes of patients with coronavirus disease 2019 (COVID-19). JAMA Cardiol 2020;5:811-8.
6. P. Zhou, X.-L. Yang, X.-G. Wang et al., "A pneumonia outbreak associated with a new coronavirus of probable bat origin," Nature, vol. 579, no. 7798, pp. 270–273, 2020.
7. 7.W. Lv, M. Wu, Y. Ren et al., "Coronavirus disease 2019: coronaviruses and kidney injury," The Journal of Urology, vol. 204, no. 5, pp. 918–925, 2020.

8. R. Valizadeh, A. Baradaran, A. Mirzazadeh, and L. V. K. S.Bhaskar, "Coronavirus-nephropathy; renal involvement in COVID-19," Journal of Renal Injury Prevention, vol. 9, no. 2, article e18, 2020.
9. R. H. Hunt, J. E. East, A. Lanas et al., "COVID-19 and Gastrointestinal Disease: Implications for the Gastroenterologist," Digestive Diseases, vol. 39, pp. 119–139, 2020.
10. M. A. Chary, A. F. Barbuto, S. Izadmehr, B. D. Hayes, and M. M. Burns, "COVID-19: therapeutics and their toxicities," Journal of Medical Toxicology, vol. 16, no. 3, pp. 284–294, 2020.
11. G. Lippi, A. M. South, and B. M. Henry, "Electrolyte imbalances in patients with severe coronavirus disease 2019 (COVID-19)," Annals of Clinical Biochemistry, vol. 57, no. 3, pp. 262–265, 2020.
12. J. W. Lee, "Fluid and electrolyte disturbances in critically ill patients", Electrolyte & Blood Pressure, vol. 8, no. 2, pp. 72–81, 2010.
13. R. K. Thekkeveedu, S. Ramarao, N. Dankhara, and P. Alur, "Hypochloremia secondary to diuretics in preterm infants: should clinicians pay close attention?" Global Pediatric Health, vol. 8, p. 2333794X2199101, 2021
14. S. Taghavi and R. Askari, "Hypovolemic shock," in StatPearls, StatPearls Publishing, Treasure Island (FL), 2021.
15. M. Kopač, "Evaluation of hypervolemia in children," Journal of Pediatric Intensive Care, vol. 10, no. 1, 2021.

SP-17

Honey and Fenugreek as Synergistic Anti-Inflammatory Agents

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Abstract: Natural extracts have a good medicinal value and have been researched extensively for their therapeutic use in humans. Two such natural extracts with great medicinal values are Honey and Fenugreek. Honey has a number **o**f medicinal values. It was used as conventional medicine to treat back pain, skin diseases, arthritis and rheumatism. The phenolic and flavanoid components in honey are found to have oxidative property to combat oxidative stress. Fenugreek seeds are used as a spice, in colic, flatulence, dysentery, diarrhea, diabetes, and lipid disorders in India . Several preclinical and clinical research have outlined the pharmaceutical uses of fenugreek as antidiabetic, antihyperlipidemic, antiobesity, anticancer, antiinflammatory, antioxidant, antifungal, antibacterial, galactogogue and for miscellaneous pharmacological effects, including improving women's health. Combination of honey and fenugreek were used in this study to find the synergistic anti-inflammatory property. Venous blood (2 ml) was used to find the anti-inflammatory property. HRBC (Haemoglobin RBC) solution was obtained by standard technique using Alsever solution. Diclofenac was used as standard for both test and control. Both the test and control were tested with increasing concentration of Diclofenac. The solutions were incubated, centrifuged and the absorbance of the supernant was read at 560nm.The absorbance value was found and percentage protection(percentage of HRBC hemolysis) was obtained by using formula Percentage of Protection (%) = 100- [(OD of sample/OD of Control) X 100]. The percentage protection at 100 µg,200 µg,300 µg,400 µg,500 µg were 41.6%,47.9%,52.0%,56.2% and 58.3% respectively. From the loss of membrane stabilization property of the hemoglobin RBC, the anti-inflammatory property of the synergistic combination was proven.

Keywords: Anti-inflammatory, Fenugreek, Enzymes, hemoglobin RBC, Alsever solution, Percentage protection.

INTRODUCTION

Honey has domestic as well as medicinal utility, in practice since long for its various properties including, antioxidant property that has recently come to limelight. Honey is a multifaceted mixture of enzymes, peptides and amines. Its active ingredients contain biologically active substances such as, caffeic acid, phenethyl ester and flavonoid glycones.[1,2] Honey was used as conventional medicine to treat back pain, skin diseases, arthritis and rheumatism. Honey seems to have good antioxidants properties with a number of preventative effects towards diseases such as, inflammatory disorders and aging, to mention a few.[3] Among the compounds with biological activity that are present in honey, the compounds that display antiinflamatory capacity, such as phenolic acids and flavonoids, have received special attention from research groups, due to their role in the prevention of diseases associated with oxidative stress.[4] Fenugreek seeds are used as a spice, in colic, flatulence, dysentery, diarrhea, diabetes, and lipid disorders in India.[5] Extensive preclinical and clinical research have outlined the pharmaceutical uses of fenugreek as antidiabetic, antihyperlipidemic, antiobesity, anticancer, antiinflammatory, antioxidant, antifungal, antibacterial, galactogogue and for miscellaneous pharmacological effects, including improving women's health. The flavanoids and polyphenols display the anti-inflammatory activity.[6] Several animal experiments have also shown anti-inflammatory activity of honey and fenugreek.[7] This study aimed to look for the anti-inflammatory activity of the combination of both honey and fenugreek.

MATERIALS AND METHODS

2 ml of venous blood was collected for studing the anti-inflammatory activity. The collected blood (1ml) was mixed with equal volume of Alsever solution (2 % dextrose, 0.8 % sodium citrate, 0.5 % citric acid and 0.42 % NaCl) and centrifuged at 3,000 rpm for 5 min. Supernant discarded and the lower layer collected. RBC's were washed with isosaline (0.85% NaCl) (centrifuge 3000 rpm for 5 min) and 1 ml of RBC's were collected and 9ml of isosaline was added. The obtained solution is HRBC (haemoglobin RBC). Various concentrations of samples were taken in test tubes and made upto 1 ml using distilled water and to each tube, 1 ml of phosphate buffer, 2 ml hypo saline (0.36% NaCl) and 0.5 ml of HRBC suspension were added. Incubated at 37°C for 30 minutes, centrifuged at 3,000 rpm for 20 minutes. Absorbance of supernatant was read at 560 nm. Diclofenac (1mg/ml) was used as standard and a control was prepared without sample. The percentage (%) of HRBC protection (Hemolysis)(Fig:3)was calculated using the following formula,

Percentage of Protection (%) = 100- [(OD of sample/OD of Control) X 100].

Sample preparation

5 g of fenugreek was soaked in 10 ml ethanol ,boiled and filtered.1ml filtrate was added to equal volume of honey. 0.2 ml of this solution was added 1.8 ml of buffer. Different dilutions were used for the assay.(Fig:2)
Stock 100+100 µg (honey + fenugreek) . Blank- 0.48

RESULTS

The percentage protection was calculated from the above formula.It was found that increasing concentration of the sample caused increase in the anti-inflammatory activity proven by increase in hemolysis of blood thus esthablishing the loss of membrane stabilization of RBC due to increased concentration of the sample .The percentage protection at 100 µg,200 µg,300 µg,400 µg,500 µg were 41.6%,47.9%,52.0%,56.2% and 58.3% respectively(Table 1).

Table 1: Percentage protection of increasing concentration of the synergistic combination

Concentration (µg)	100	200	300	400	500
Sample	0.28	0.25	0.23	0.21	0.20
% of protection	41.6	47.9	52.0	56.2	58.3

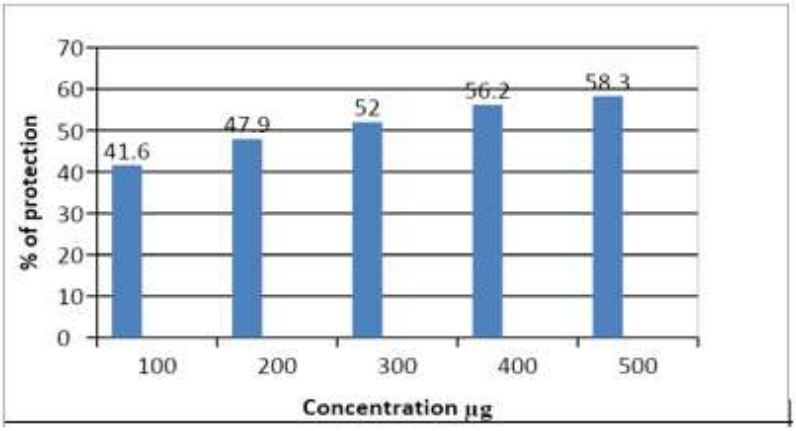

Fig 1: Bar chart showing increasing concentration of synergistic combination on X axis versus % protection on Y axis

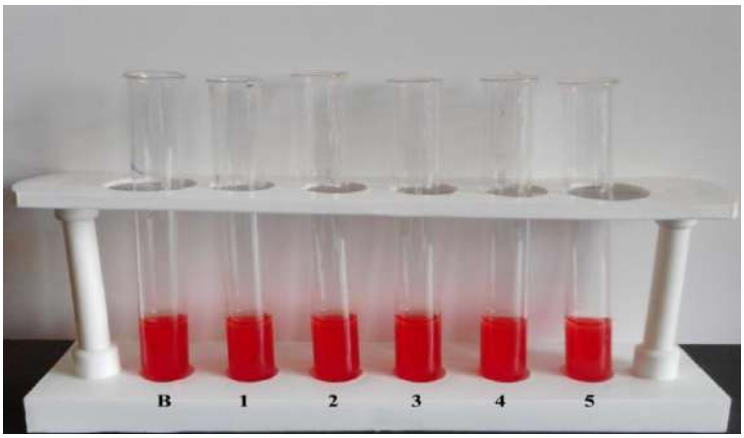

Fig 2: Testtubes showing the HRBC SOLUTION with testtube B for sample versus other testtubes for control.

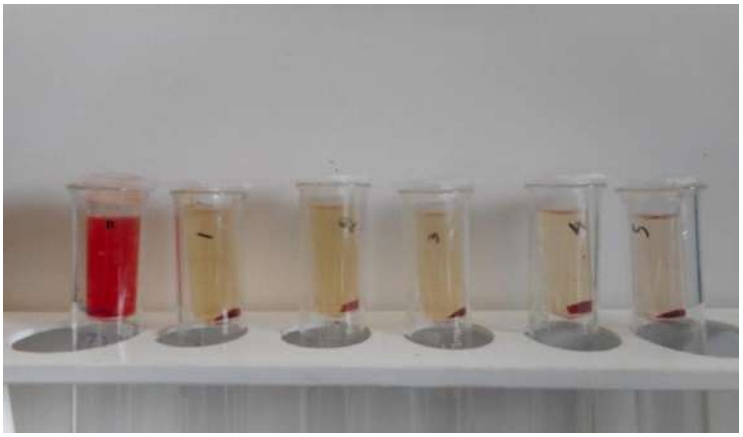

Fig 3: Hemolysis pattern observed in the subsequent testtubes proving the anti-inflammatory activity of the synergistic combination.

DISCUSSION

The phenolic and flavonoids compounds in honey reduces COX2 and iNOS.[8] The study by Eyarefe et al.[9] revealed that both natural honey and amikacin enhanced wound healing in non-diabetic rat patients.Another study in rabbits using Pistacialentiscus fatty oil (PLFO), and honey mixture also showed antiinflammaotry activity.[10] Another study prove that honey is an effective dressing agent instead of conventional dressings, in treating patients of diabetic foot ulcer.[11]Another study also proved the healing property of honey in Vernal Keratoconjuctivitis.[12] Another study on Fenugreek roved that Inhibition of inflammatory swelling was 45% and 62% in the lower and higher dose groups, respectively, compared with 100% in untreated animals.[13] Ethanol extract, mucilage, and flavonoids of fenugreek seeds were found to have anti-inflammatory, anti-arthritic, and anti-oxidant activities.[14,15] The *in vivo* effect of methanolic extract using cream based system, and found to reduce the inflammation. As per the recent studies the Mrna expression of inflammatory markers were reduced. This study was thus initiated to determine the combined anti-inflammatory effect of Honey and Fenugreek and has derived positive results.

CONCLUSION

Thus the above studies have proved that Honey and fenugreek when combined together have synergistic anti-inflammatory effect. Further animal studies and clinical trials are required to confirm the therapeutic application of the combinations.

CONFLICT OF INTEREST

Conflict of interest declared none.

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Ethical statement: None

REFERENCES

1. Saad F, Gleason DM, Murray R, Tchekmedyian S, Venner P, Lacombe L, Chin JL, Vinholes JJ, Goas JA, Chen B. A randomized, placebo-controlled trial of zoledronic acid in patients with hormone-refractory metastatic prostate carcinoma. Journal of the National Cancer Institute. 2002 Oct 2; 94(19):1458-68
2. Zakaria NH, Ahmad NZ, Hashim SN, Adnan LH, Halim M, Shariff M, Mohamad N, Mat KC, Bakar NH. Analgesic effect of honey bioactive compounds and its role in reducing morphine tolerance. Journal of Applied Pharmaceutical Science. Vol. 2015 Nov 27; 5(11):146-50.
3. Samarghandian S, Afshari JT, Davoodi S. Honey induces apoptosis in renal cell carcinoma. Pharmacognosy Magazine. 2011 Jan; 7(25):46
4. Ruiz-Ruiz JC, Matus-Basto AJ, Acereto-Escoffié P, Segura-Campos MR. Antioxidant and anti-inflammatory activities of phenolic compounds isolated from Meliponabeecheii honey. Food and Agricultural Immunology. 2017 Nov 2;28(6):1424-37.
5. Kirtikar KR, Basu BD. Indian Medicinal Plants. New Delhi (India): Bishen Singh Mahendra Pal Singh; 1980. p. 700
6. Aher RR, Belge SA, Kadam SR, Kharade SS, Misal AV, Yeole PT. Therapeutic importance of fenugreek (Trigonellafoenum-graecum L.): a review. J Plant Sci Res. 2016;3(1):149.
7. Pundarikakshudu K, Shah DH, Panchal AH, Bhavsar GC. Anti-inflammatory activity of fenugreek (Trigonellafoenum-graecum Linn) seed petroleum ether extract. Indian journal of pharmacology. 2016 Jul;48(4):441.
8. Samarghandian S, Farkhondeh T, Samini F. Honey and health: A review of recent clinical research. Pharmacognosy research. 2017 Apr;9(2):121.
9. Eyarefe O.D., Ologunagba F.M., Emikpe B.O.: Wound healing potential of natural honey in diabetic and non-diabetic wistar rats. Afr J Biomed Res 2014, 17, 15–21.
10. Nisbet H.O., Nisbet C., Yarim M., Guler A., Ozak A.: Effects of three types of honey on cutaneous wound healing. Wounds 2010, 22, 275–283.

11. Imran M, Hussain MB, Baig M. A randomized, controlled clinical trial of honey-impregnated dressing for treating diabetic foot ulcer. J Coll Physicians Surg Pak. 2015 Oct 1;25(10):721-5.

12. Imran, M., Hussain, M.B. and Baig, M., 2015. A randomized, controlled clinical trial of honey-impregnated dressing for treating diabetic foot ulcer. *J Coll Physicians Surg Pak*, *25*(10), pp.721-725.

13. Snehlata HS, Payal DR. Fenugreek (Trigonellafoenum-graecum L.): an overview. Int J Curr Pharm Rev Res. 2012 Jan 1;2(4):169-87.

14. Suresh P, Kavita CH, Babu SM, Reddy VP, Latha AK. Effect of ethanol extract of *Trigonellafoenum-graecum* (Fenugreek) seeds on Freund's adjuvant induced arthritis in albino rats. Inflammation. 2012;35:1314–21.

15. Sindhu G, Ratheesh M, Shyni GL, Nambisan B, Helen A. Anti-inflammatory and antioxidative effects of mucilage of *Trigonellafoenumgraecum* (Fenugreek) on adjuvant induced arthritic rats. IntImmunopharmacol. 2012;12:205–11.

SP-18

A Human Cadaveric Study Comparing Four Suture Techniques [Baseball, Kessler, Bunnel, Un has Noval Technique] On 2-0 Prolene For Load Failure And Gap Formation

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Abstract: The objective of this study was to assess the biomechanical stability of three types of chondral flap repair techniques as well as a hydrogel scaffold implantation on the acetabular articular surface using a physiological human cadaveric model. Aim of this study was to test the most commonly used a human cadaveric study comparing four suture techniques Baseball, Kessler, Bunnel, Unhas Noval technique on 2-0 prolene for load failure and gap formation. After the clearance from local ethics committee, fresh human cadaveric tendons were sutured, twelve human cadavers (24knees) were selected for striping hamstring tendon. These findings may be helpful for the future clinical treatment of rupture of tendons and in vivo clinical application studies.

Keywords: Human Cadaver, Suture Techniques**,** Load Failure and Gap Formation

INTRODUCTION

Rupture of tendons and ligaments are common in orthopaedic surgery. However, there is no absolute data on most suitable suture technique. Still There is difficulty in overcoming restoration of the hand function, tendon gliding and primary end to end repair within sheath of tendon.[1-3] Early controlled rehabilitation has improved the outcomes after flexor tendon repair, reducing the adhesion formation, increasing repair strength and improvement in functional result. Ligament and tendon injuries are common, particularly between athletes, and they can cause significant pain and loss of mobility. In the Sutter Health network, orthopedic surgeons provide a range of treatments to repair damage to the knee, ankle, shoulder, elbow, hand and wrist. [4-6] In this study we have done human cadaveric study comparing four suture techniques [baseball, kessler, bunnel, unhasnoval technique] on 2-0 prolene for load failure and gap formation.

MATERIALS AND METHODS

After the clearance from local ethics committee, twelve human cadavers (24knees) were selected for striping hamstring tendon. Tendons which showed signs of degeneration was discarded. Each experiment required 7cm of harvested tendon.5 trials of each suture technique on 2-0 prolene was tested for load failure [tendon pullout/ knot opening / suture / rupture] and gap formation [force required to produce initial gap formation and 2mm gap at repair site] was evaluated using universal extensometer.

Fig: 1 UNIVERSAL EXTENSOMETER

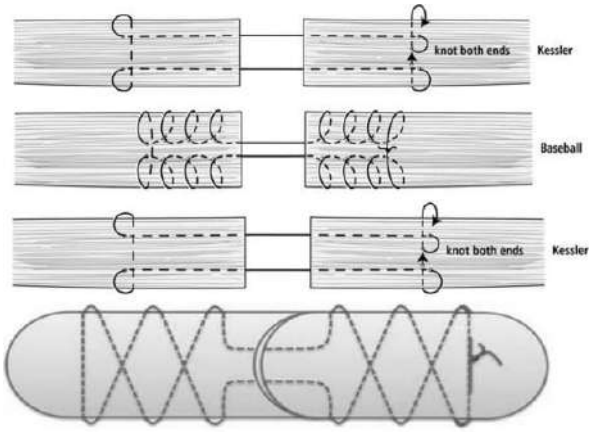

Fig: 2 BUNNEL SUTURE TECHNIQUE

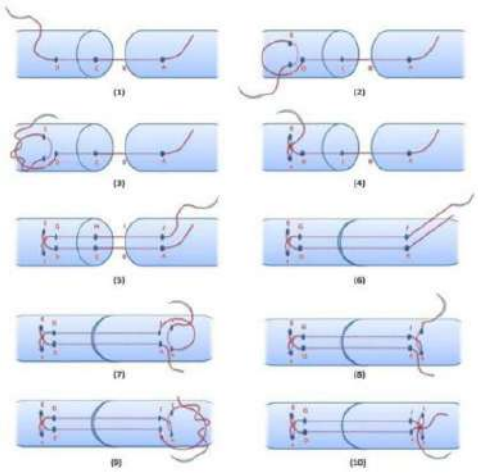

Fig:3 UNHAS SUTURE TECHNIQUE

STATISTICAL ANALYSIS

Statistical analysis was performed, after values presented in mean and standard deviation. ANOVA Test-parametric data. Kruskal wallis test -non-parametric data. *P* value of <0.01 (two-tailed) was considered to be statistically significant.

RESULTS

Mean load failure is 30 +/- 1.5811 for unhas, 16.4+/- 1.5166 for kessler, 25.2 +/- 1.3038 for Bunnel, 31.8+/- 1.6432 for Baseball on 2-0 prolene, which had significant p-value ($p<0.05$).The mean 2mm gap formation was 18+/- 0.8944 for unhas, 12 +/- 1.4142 for kessler15.4 +/- 1.402 for Bunnel, 19.2+/- 0.4772 for baseball on 2-0 prolene, which had p-value significant at ($p<0.05$).The initial gap formation did not have significant p-value ($p<0.05$) .The mean load attained by baseball and unhas technique were significantly higher.

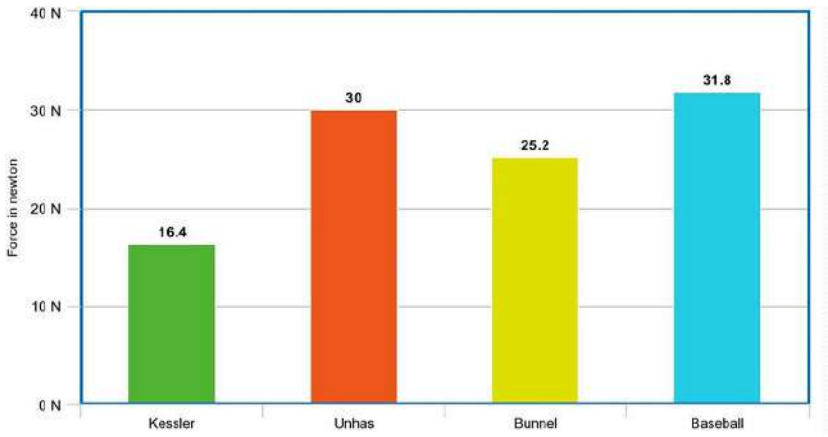

Fig: 4 Mean load failure F-ratio is 103.15217 and the P-value is <.00001.
Significant result is at $p < 0.05$.

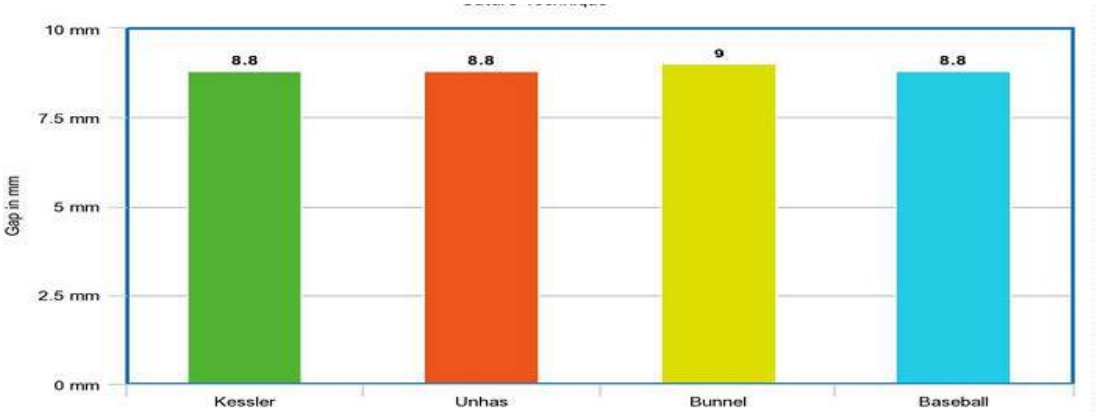

Fig: 5 F-ratio - 0.33333.P-value is <.8014. The result was not Significant result was not obtained at P< 0.05.

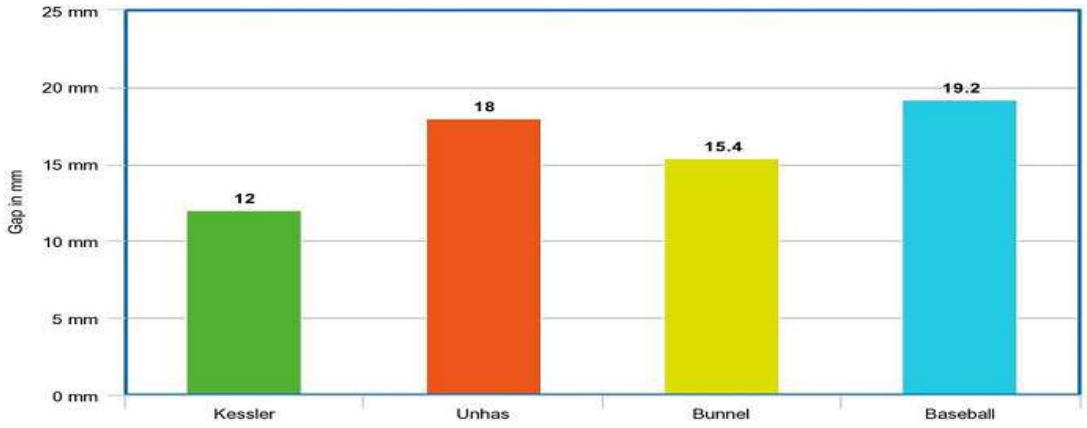

Fig: 6 F-ratio - 49.78295. P-value is <.00001. Significant result to be obtained at p < 0.05.

DISCUSSION

The technique which include repair strength, gapping resistance, main- taining glide, reducing tendon damage, minimizing adhesion formation, and also provide easiness which can be performed with conventional suture material and less operating time are ideal suture. Baseball and unhas suture technique were able to withstand signicantly higher maximum failure loads than the Kessler suture and bunnel.[7]Ultimate failure testing represents the occurence of a high load event which may happen unintentionally and lead to suture failure.The gap resistance and ultimate strength of repaired tendon are the primary parameters that define themechanical properties of a surgical repair.[8]Maintaining a certain baseline tension on the core suture during surgery greatly benefits gap resistance. Peripheral suture could also significantly increase the strength of tendon repairs.Locking loops in which the type of suture used to hold the tendon on either side in Un has suture technique is superior compared with Bunnell suture technique which uses grasping loops.[9] Locking loops provide better grasp of tendon fibers and prevent suture pullout. As grasping loops do not tighten around tendon fibers it would be expected that grasping repairs would fail by suture pullout with repetitive load.[10] There have been a few case reports in the literature about the isolated distal semitendinosus tendon rupture.[11, 12] Recently, Cooper and Conway reported the retrospective case series with the results of treatment in professional athletes.[13] However, this is the first report of isolated distal semitendinosus tendon rupture developed in non-athlete ordinary office-working man with injury developed during daily activity. Because of the rarity, there is paucity of evidence over the best method of managing the injury; whether surgical or non-surgical. Tendon forces up to 9 N were present during passive mobilization of the fingers. Tendon forces up to 35 N were present during active unresisted finger motion.There were limitations to this study. Cyclic loading was not conducted and linier load to failure may not mimic physiological conditions.

CONCLUSION

Ruptures repaired using 2-0 prolene by baseball and un has technique were able to withstand comparatively higher maximum failure loads than the Kessler suture and Bunnel suture techniques. Certain baseline tension is maintained on the core suture and it greatly benefits gap resistance during surgery. Peripheral suture could also significantly increase the strength of tendon repairs. These findings may be helpful for the future clinical treatment of rupture of tendons and in vivo clinical application studies .

CONFLICT OF INTEREST

Conflict of interest declared none.

REFERENCES

1. Tanaka T, Amadia PC, Zhao CF, et al. Biomechanical properties of locking versus grasping suture. Summer Bioengineering Conference, Minnesota Orthopaedic Biomechanics Laboratory; Rochester, Minnesota, 2003;635–636.
2. Viinikainen A, Goransson H, Ryhanen J. Primary flexor tendon repair techniques. Scand J Surg. 2008; 97:333–340.
3. Cannon DL. Flexor and extensor tendon injuries. In: Canale ST, Beaty JH, eds. Campbell's Operative Orthopaedics, 12th ed. Philadelphia: Elsevier Mosby; 2013:3249.
4. Ryan JD. Principles and Techniques of Tendon Repair. Georgia: The Podiatry Institute; 2010. Available at: www.podiatryinstitute.com/pdfs/ Update_2010/2010_52.pdf. Accessed January 15, 2016.
5. Sebastin SJ, Ho A, Karjalainen T, et al. History and evolution of the kessler repair. J Hand Surg. 2013;38A:552–561
6. Latendresse K, Dona E, Scougall PJ, Schreuder FB, Puchert E, Walsh WR (2005) Cyclic testing of pullout sutures and micro-mitek suture anchors in Xexor digitorum profundus tendon distal Wxation. J Hand Surg Am 30(3):471–478.
7. Santoni BG, McGilvray KC, Lyons AS, Bansal M, Turner AS, Macgillivray JD, Coleman SH, Puttlitz CM (2010) Biomechanical analysis of an ovine rotator cuV repair via porous patch augmentation in a chronic rupture model. Am J Sports Med 38(4):679–686.
8. Cooper DE, Conway JE (2010) Distal semitendinosus ruptures in elite-level athletes: low success rates of nonoperative treatment. Am J Sports Med 38(6):1174–1178.
9. Schilders E, Bismil Q, Sidhom S, Robinson P, Barwick T, Talbot C (2006) Partial rupture of the distal semitendinosus tendon treated by tenotomy: a previously undescribed entity. Knee 13(1):45– 47.
10. Sekhon JS, Anderson K (2007) Rupture of the distal semitendinosus tendon: a report of two cases in professional athletes. J Knee Surg 20(2):147–150.
11. Cooper DE, Conway JE. Distal semitendinosus ruptures in elite-level athletes: low success rates of nonoperative treatment. Am J Sports Med. 2010. 38:1174–1178.
12. Sekhon JS, Anderson K. Rupture of the distal semitendinosus tendon: a report of two cases in professional athletes. J Knee Surg. 2007. 20:147–150.
13. Schilders E, Bismil Q, Sidhom S, Robinson P, Barwick T, Talbot C. Partial rupture of the distal semitendinosus tendon treated by tenotomy: a previously undescribed entity. Knee. 2006. 13:45–47.

SP-19

Immediate Effect of Sheethali Pranayama on Short Term Heart Rate Variability (Hrv) in Healthy Subjects.

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Abstract: Sheetali pranayama is also known as cooling breath pranayama which is used to control and cool the mind, body and emotions. Heart rate variability is a non invasive procedure which is widely used to measure the time interval between heart rate variations. The present study is aimed to evaluate the immediate effect of sheetali pranyama practice on short term heart rate variability in healthy subjects. Apparently (n=60) healthy volunteers including both the genders were recruited and divided into two groups, pranayama group (n=30) and control group (n=30). The findings of this study conclude that sheetali pranayama (5 min) practice improves the cardiovascular parameters through parasympathetic dominance among the healthy volunteers. It can be advised as adjuvant therapy for the individuals with exacerbated by stress.

Key Words: Yoga, sheetali pranayama, Heart rate

INTRODUCTION

In day to day life, to maintain healthy lifestyle yoga plays a vital role which mainly includes pranayama.[1] Yoga is considered as major factor to maintain the wellbeing of the person mentally and physically and acts as an effective stress buster.[2] Yoga plays an significant role in reducing stress, reducing sympathetic activity, increasing parasympathetic activity, decreasing blood pressure, and improving sense of well-being.[3] Nowadays yoga has become more popular practice around the world and people practicing has grown by over 50% in the last four years.[4,5] The word yoga means fusion of an individual responsiveness with the universal responsiveness in a state of super consciousness known as Samadhi.[3] There are pranayamas such as Bhramari, Kapalbathi, Nadisudhi, sheetkari and Sheetali pranayama practice regulates the normal healthy lifestyle of an individuals. These pranayamas can prevent and reduce the risk of diabetes, obesity and hypertension.[6,7] Pranayama practice on regular basis can provide positive result on respiratory, cardiovascular, functions and also improves autonomic function on parasympathetic dominance.[2] This can decreases the strain and effect of stress among the individuals by maintaining the mental and physical health.[4] Sheetali pranyama is a very simple pranayama were everyone can practice it easily.[8] Sheetali pranayama is a cooling breath pranayama which can control an individuals mind, body and emotions.[9] The word sheetali is a Sanskrit word implies "cool or rigid". Practicing sheetali pranayama on regular basis among hypertensive patients causes drastic changes in their blood pressure and heart rate after 20 minutes of practice.[10] The present study is aimed to evaluate the immediate effect of sheetali pranayama on short term variability in healthy subjects.

METHODS

Study participants

This study is a randomized control study was performed among 60 healthy volunteers of both the gender with age group between 17-35 years of age (based on the previous study, the sample size was calculated). Healthy volunteers have been equally divided into two groups, 30 control group and 30 pranayama group. Ethical committee approval has been obtained from the institutional ethical committee of the institution. The study procedure was explained thoroughly and informed consent was obtained from the participants.

Selection criteria

Volunteers were selected on the basis of inclusion and exclusion criteria. Age group between 17-35 years who volunteers to participate in the study has been included in this study. Participants with recent surgery, respiratory illness, endocrine abnormalities, athletes and regular yoga practitioner have been excluded from this study. Measurement of weight was obtained by the volunteers without footwear and light clothing; height was measured in a standing position.

Intervention details

After elaborating the pranayama practice, the pranayama intervention for the pranayama group volunteers have been explained by the trained yoga and neuropathy doctor based on the standard procedure. For performing sheetali pranayama the volunteers were asked to breathe in with closed eyes through their folded tongue in the form of tube after that slow exhalation through both the nostrils have been followed. Volunteers have been instructed to perform 10 cycles followed by resting period of 2 minutes. The participants were asked to do minimum of 20 rounds within the period of 5 minutes.

Outcome measurement

The body mass index was measured by weight in kilograms divided by height in meter squares (kg/m^2). Heart rate variability recording was carried out in the morning after 2 hours of light breakfast. Before the test starts the participants were instructed to void urine. After a supine rest on the couch for 15 minutes, lead ECG II was obtained with analog to digital convertor (A-D). R-R interval raw data was acquired from simple analog – digital (A-D) converter we stored separately and HRV was done using kubios software.[11]

Statistical analysis

Data expressed as mean (median) and SD. Normality of data was tested using Kolmogrov-Smirnov test. A p value of > 0.05 indicated normal Gaussian distribution. As the data sets of HRV were skewed and not normally distributed, Wilcoxon signed rank test and Mann Whitney U test was performed using R statistical software version 3.1.1.

RESULTS

Table:1 - Study participants of pranayama and control group baseline characteristic.

Variables	Pranayama Group n-30	Control Group n-30
Age(yrs)	23.27±5.93	19.30±5.83
Gender(M/F)	19/11	17/13
Height(cm)	161.7±14.12	159.00±7.92
Weight(kg)	63.65±12.53	67.11±7.95
BMI(kg/m^2)	24.43±4.07	23.19±6.18

Table 1 shows the antropology of both the pranayama group and control group. They were no significantly difference in age, height, weight and BMI, so both groups were ideal for comparison. There were 30 each in both the control and pranayama group. The mean age distribution was 23.27±5.93 in pranayama group and 19.30± 5.83 in the control group. There were 19 males and 11 females in pranayama group and 17 males and 13 females in control group. Mean Height distribution is 161.7 ±14.12 in pranayama group and 159.00 ± 7.92 in control. Mean weight distribution is 63.65±12.53 in pranayama group and 67.11±7 .95 in control. Mean BMI is 24.43±4.07 in pranayama group and 23.19±6.18 in control.

Table: 2. Comparison of Frequency domains of Short term HRV parameters in between pranayama and control group

Variables	Control group (n-30)		P value	Pranayama Group (n-29)		P value
	Before	After		Before	After	
RR interval	789.90 (792.23)[617.89-823.98]	799.28 (802.10)[672.81-845.29]	0.78	779.20 (732.82)[657.10-810.87]	727.90 (731.88)[679.20-830.23]	0.12
SDNN (ms)	52.14(54.02) [48.14-64.12]	56.10(55.18) [44.45-65.02]	0.52	54.24(55.19) [48.22-89.02]	57.48 (52.80) [58.16-78.08]	0.78
RMSSD (ms)	32.28(3.40) [28.18-42.13]	30.56(30.27) [26.08-38.16]	0.72	37.24(32.68) [26.12- 59.05]	34.82 (38.30) [34.16-68.18]	0.40
pNN 50%	16.19(17.80) [10.29-32.31]	12.19(11.42) [9.18-28.54]	0.59	17.34(15.18) [34.22-51.97]	24.44 (13.02) [17.07-39.92]	0.05

HR, heart rate; RR, R R interval; RMSSD, square root of the mean squared differences between adjacent normal RR intervals; SDNN, standard deviation of all normal RR intervals;), pNN50 the proportion derived by dividing NN50 by the total number of NN intervals,

Table 2 shows Comparison of Frequency domains of Short term HRV parameters in between pranayama and control group. RR interval decreased from 779.20 msec 727.90 msec after pranayama practice. The SDNN values were 54.24 before pranayama and increased to 57.48 after pranayama (p=0.78), RMSSD values were 37.24 before pranayama and decreased to 34.82 after pranayama(p=0. 40). pNN50% values were 17. 34 before the pranayama and decreased to 14. 44 after the pranayama and was found to be significant(p=0. 05).

Table: 3. Comparison of Time domains of Short term HRV parameters in between

pranayama and control group						
Variables	Control group (n-29)		P value	Pranayama Group (n-30)		P value
	Before	After		Before	After	
L.F (n.u)	56.12(55.37) [48.09-66.13]	58.22(56.90) [52.30-68.10]	0.92	59.8(68.12) [52.16-59.33]	52.14 (51.92) [34.37-58.09]	0.23
H.F (n.u)	46.90(46.02) [41.99-58.80]	45.45(46.90) [40.52-59.29]	0.42	42.18(43.94) [38.92-54.32]	49.08 (49.82) [38.81-65.12]	0.09
LF/HF Ratio	2.52(2.54) [1.06-1.47]	2.46(2.47) [0.96-1.49]	0.68	1.6(1.51) [0.98-1.90]	2.79 (2.80) [0.74-2.89]	0.02

LF- low frequency; HF-high frequency; LF/HF ratio, ms- milliseconds; nu- normalized units.# P<0.05, ## P<0.01.

Table 3 shows comparison of Short term HRV parameters in between pranayama and control group. On comparing the value of LF, HF&LF/ HF RATIO among the pranayama group the LF/ HF ratio was 1.6 before the pranayama and increased to 2.79 after the pranayama and was found to be significant(p=0.02). The LF values were 59. 8 before pranayama and decreased to 52.14 after pranayama, HF values were 42 .18 before pranayama nad increased to 49.08 after pranayama. No significant changes were noted in the LF and HF values (LF: p=0 .23 ; HF: p=0 .09). Table 1 showed the baseline characteristics of the pranayama group and control group participants. They were no significantly difference in age, height, weight and BMI, so both groups were ideal for comparison. In time domain parameter of HRV, pnn50% showed significant increase in pranayama group after sheetali pranayama (24.44 vs 12.19) compared to control group participants (Table: 2). LF/HF ratio also showed significant improvement compared to control group participants immediately after sheetali pranayama practice (Table: 3). Other parameters not showed any significant changes among the pranayama group after the intervention.

DISCUSSION

In this present study we have found that immediately after the practice of sheetali pranayama showed the parasympathetic dominance among participants. In the previous study done by Rohini.et.al, [8] found that immediately after sheetali pranyama HR and BP decreases. After the 3 months of sheetali pranayama practice among hypertension patients they found that significant improvement in the heart rate variability. In another study, it was observed that when blood pressure and heart rate were measured immediately after practice sheetali and sheetkari Pranayama, which is identical to Sheetali Pranayama alone, they both decreased significantly.[12] Sheetali (cooling) pranayama alone is framed for the present analysis, which is very easy to perform without any alteration of nasal manipulations or quick breathing techniques . This pranayama practise would cause a chill in the throat, and it might stimulate the vagal nerve and alter blood pressure management by inducing transient increases in cardiac parasympathetic activity (Baroreceptor reflex).[13] The baroreflex mechanism, which involves parasympathetic activation and sympathetic inhibition, is thought to be a short-term blood pressure regulator. Also, yoga-based practices have been found to balance the activity of the autonomic nervous system (ANS) and GABA systems in part through stimulation of the vagus nerves, the main peripheral pathway of the ANS.[14,15] The current study's findings could be attributable to the above-mentioned process, and the current findings point to parasympathetic dominance. A limitation of our study would be the small sample size, healthy subjects, and single session of pranayama practice.

CONCLUSION

Previous study shows that the sheetkari and sheetali pranayama improves the cardiovascular parameters among high blood pressure individuals and also can bring about the immediate effect on blood pressure by daily practice.[12] The findings of this study conclude that sheetali pranayama (5min) practice improves the cardiovascular parameters through parasympathetic dominance immediately after the practice of sheetali pranayama among the healthy volunteers. It can be practiced routinely for the reduction of stress on large number of individuals with a long distance follow up is required to substantiate the findings.

CONFLICT OF INTEREST

Conflict of interest declared none.

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REFERENCES

1. Saraswati SS, Hiti JK. Asana pranayama mudra bandha. Bihar, India: Yoga Publications Trust; 1996.
2. Kuppusamy M, Kamaldeen D, Pitani R, Amaldas J, Shanmugam P. Effects of Bhramari Pranayama on health–a systematic review. J. Tradit. Complement. Med. 2018;8:11-6.
3. Kanojia S, Sharma VK, Gandhi A, Kapoor R, Kukreja A, Subramanian SK. Effect of yoga on autonomic functions and psychological status during both phases of menstrual cycle in young healthy females. J Clin Diagn Res. 2013 Oct;7(10):2133-9. doi: 10.7860/JCDR/2013/6912.3451. Epub 2013 Sep 13. PMID: 24298457; PMCID: PMC3843391.
4. Dillette AK, Douglas AC, Andrzejewski C. Yoga tourism–a catalyst for transformation?. Annals of Leisure Research. 2019 Jan 1;22(1):22-41.
5. Bhalla N. Online Meditation, Yoga, and Rising Interestin Eastern Philosophy. Religion Online: How Digital Technology Is Changing the Way We Worship and Pray [2 volumes]. 2019 Mar 7:214.
6. Kuppusamy M, Kamaldeen D, Pitani R, Amaldas J. Immediate effects of Bhramari pranayama on resting cardiovascular parameters in healthy adolescents. Journal of clinical and diagnostic research: JCDR. 2016 May;10(5):CC17.
7. Lalitha S, Maheshkumar K, Shobana R, Deepika C. Immediate effect of Kapalbhathi pranayama on short term heart rate variability (HRV) in healthy volunteers. Journal of Complementary and Integrative Medicine. 2021 Mar 1;18(1):155-8
8. Rohini P, Roopa S, Padmavathi R and Maheshkumar K, on Immediate effects of the practise of Sheethali pranayama on heart rate and blood pressure parameters in healthy volunteers
9. Thanalakshmi J, Maheshkumar K, Kannan R, Sundareswaran L, Venugopal V, Poonguzhali S. Effect of Sheetali pranayama on cardiac autonomic function among patients with primary hypertension and romized controlled trial. Compl Ther Clin Pract 2020:101138.
10. Jagadeesan T, Choudhary AK, Loganathan S, Rajendran K, Allu AR, Kuppusamy M. Yoga practice (Sheetali Pranayama) on cognition in patients with hypertension: A randomized controlled study. Integrative medicine research. 2021 Sep; 10(3).
11. Maheshkumar K, Dilara K, Maruthy KN, Sundareswaren L. Validation of PC-based sound card with biopac for digitalization of ECG recording in short-term HRV analysis. North American journal of medical sciences. 2016 Jul;8(7):307.
12. The immediate effect of Sheethali and Sheethkari Pranayama on blood pressure and cardiovascular changes among hypertensive patients. Kumar N, Thanalakshmi J, Kannan R, Maheshkumar K, Allu AR, Vijayalakshmi B.. International Journal of Research in Pharmaceutical Sciences. 2018 Oct 31;9(4):1249-52
13. Barrett KE, Barman SM, Boitano S, Brooks H. Ganong's review of medical physiology, 23rd ed. NY: McGraw-Hill Medical; 2009.
14. Shetty P. Effects of Sheetali and Sheetkari Pranayamas on Blood Pressure and Autonomic Function in Hypertensive Patients. Integrative Medicine: A Clinician's Journal. 2017;16:32.
15. Streeter CC, Gerbarg PL, Saper RB, Ciraula DA, Brown RP. Effects of yoga on the autonomic nervous system, gamma-aminobutyric-acid, and allostasis in epilepsy, depression, and post-traumatic stress disorder. Med Hypothesis. 2012;78:571–9.

SP-20

A Case Report on Recurrent Ipsilateral Ectopic Pregnancy

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Abstract: Almost 1% of all pregnancies are ectopic, most common site of implantation is in the fallopian tube. Most ectopic Pregnancies continue to grow and lead to rupture of the tube. Ampulla is most common site (80%) in the fallopian tube and 12% in isthmic portion. Early Rupture in the first few weeks is the outcome of isthmic pregnancy as isthumus being the narrowest and usually there is tubal rupture in the first few weeks. It is suspected when if the women who are pregnant complaints during the first trimester: lower abdominal pain, amenorrhea and vaginal bleeding. An increase in Beta hCG level above the 2000 mIU/ml with an empty uterus on a transvaginal ultrasound is needed for confirming the diagnosis of ectopic pregnancy. In this case study, we report on a 26-year-old woman, G2 P0A1 previous ectopic treated with Right salpingotomy who presented to OBG OPD at Sree Balaji Medical College and Hospital, with a history of 6weeks of amenorrhea. Patient had no complaint. Abdominal ultra sound revealed no intra uterine pregnancy and there was an adnexal mass on the right side of the uterus (40 mm lucent destiny) without any free fluid in pelvic cavity. TVS confirmed the diagnosis of Ectopic pregnancy and laparoscopic surgery was performed under general anesthesia. Right Salpingectomy was done.

Keywords: Amenorrhea, Ectopic pregnancy, Isthmus, Salpingotomy.

INTRODUCTION

Pregnancy is called ectopic when implantation of blastocyst occurs outside the cavity of uterus. Extra uterine pregnancy account for 1.3% to 2.4% of all pregnancies.[1-5] Over 98% cases implants in the fallopian tube and the remaining implant on the myometrium, the cervix, the ovary, and abdomen. Ectopic pregnancy can present as pelvic pain or abdominal pain amenorrhea may or may not have bleeding from the vagina in the first trimester.[2,6-9] Diagnosis often requires serial human chorionic gonadotropin levels and a transvaginal ultrasound. The treatment strategy is depending on the patient condition.[10] Treatment varies from expectant management, to surgical management.

CASE PRESENTATION

26-year-old woman, G2 P0 previous ectopic conceived by natural conception treated with salpingotomy who presented to Sree Balaji Medical College and Hospital, OBG department.

Chief complaints

Complaints of 6weeks of amenorrhea. At 7 weeks gestation Transvaginal ultrasonography confirmed the absence of pregnancy in the uterine cavity and presence of a right tubal pregnancy (a fetus and it's heartbeat were detected).

Tests

The patient's serum betahCG level of 4,000 international units/mL.

Previous Medical history

She had history of one right tubal pregnancy which had been treated surgicallybysalpingotomy3 years ago. During previous Salpingotomy procedures, left tube which was normal its patency checked and confirmation of patency checked by chromotubation using methylene blue. All of the pregnancies were conceived by spontaneous conception.

Observation

on admission her vital sign was normal and no abdominal pain or vaginal bleeding was present.. We explained about surgical options available and complications to the patient, whether to perform salpingotomy, or salpingectomy and she opted for salpingectomy.

Treatment

Procedure done

Laparoscopic surgery was performed under general anesthesia. There was a mass about 40×40 mm in ampulla of right tube. Salpingectomy was performed and the intra and post-operative period was uneventful and patient was discharged on post-operative day one . After 7 days, she was reviewed and found to be well.

DISCUSSION

Ectopic pregnancy is a complication of first-trimester. It is alife-threatening condition and is regarded as a major cause of pregnancy related deaths, because it is responsible for 9% to 13%of maternal mortality all.[11-13] The most common site of ectopic pregnancy is fallopian tubes. Many risk factors are correlated with ectopic pregnancy such as previous ectopic pregnancy, adhesions due to previous pelvic surgeries, tubal damage, smoking habits, history of infertility, elderly woman, and in vitro fertilization treatment.[14] Most of the women with ectopic pregnancies does not have identifiable risk factors. The ideal management for recurrent ectopic pregnancy is unclear. The risk of recurrent ectopic pregnancy found to be fourfold higher in cases involving previous medical or surgical management, and the risk of ectopic pregnancies does not have significant difference between medically and surgically managed cases.[5] In cases of recurrent ectopic pregnancy, the surgical management should be completely discussed with patient and their family, and proper informed consent should be obtained. In the present case, salpingotomy was performed during the first ectopic pregnancy, which might be the reason for second ectopic pregnancy.

CONCLUSION

Clinicians should be aware that one ectopic pregnancy is a risk factor for recurrent ectopics. Early diagnosis and management of recurrent ipsilateral ectopic pregnancy is recommended for decreasing morbidity and mortality.

CONFLICT OF INTEREST

Conflict of interest declared none.

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REFERENCES

1. Varma R, Gupta J. Tubal ectopic pregnancy. ClinEvid 2009; 1406: 30-33.
2. Shaw JL, Dey SK, Critchley HO, Horne AW. Current knowledge of the aetiology of human tubal ectopic pregnancy. Human reproduction update. 2010 Jul 1;16(4):432-44.
3. Cunningham FG, JW W. Williams obstetrics. 22nd edn McGraw-Hill Professional. New York. 2005.
4. Milingos DS, Black M, Bain C. Three surgically managed ipsilateral spontaneous ectopic pregnancies. Obstetrics & Gynecology. 2008 Aug 1;112(2):458-9.
5. Abraham C, Seethappan V. Spontaneous live recurrent ectopic pregnancy after ipsilateral partial salpingectomy leading to tubal rupture. International journal of surgery case reports. 2015 Jan 1;7:75-8.
6. Tanaka T, Hayashi H, Kutsuzawa T, Fujimoto S, Ichinoe K: Treatment of interstitial ectopic pregnancy with methotrexate: report of a successful case. Fertil Steril. 1982, 37:851-2. 10.1016/s0015-0282(16)46349-1.
7. Barash JH, Buchanan EM, Hillson C: Diagnosis and management of ectopic pregnancy. Am Fam Physician. 2014, 90:34-40.
8. Hendriks E, Rosenberg R, Prine L: Ectopic pregnancy: diagnosis and management. Am Fam Physician. 2020, 101:599-606
9. Mol BW, Hajenius PJ, Engelsbel S, Ankum WM, Van der Veen F, Hemrika DJ, Bossuyt PM: Serum human chorionic gonadotropin measurement in the diagnosis of ectopic pregnancy when transvaginal sonography is inconclusive. Fertil Steril. 1998, 70:972-81. 10.1016/s0015-0282(98)00278-7.
10. Skjeldestad FE, Hadgu A, Eriksson N: Epidemiology of repeat ectopic pregnancy: a population-based prospective cohort study. Obstet Gynecol. 1998, 91:129-35. 10.1016/s0029-7844(97)00603-0.
11. Simpson JW, Alford CD, Miller AC: Interstitial pregnancy following homolateral salpingectomy. A report of 6 new cases and review of the literature. Am J Obstet Gynecol. 1961, 82:1173-9. 10.1016/s0002-9378(16)36210-x
12. Kumari P, Lal P: Ectopic pregnancy in tubal remnant. J Case Rep. 2015, 5:24-5. 10.17659/01.2015.0007.
13. Stovall TG, Ling FW: Ectopic pregnancy. Diagnostic and therapeutic algorithms minimizing surgical intervention. J Reprod Med. 1993, 38:807-12.
14. Lau S, Tulandi T: Conservative medical and surgical management of interstitial ectopic pregnancy. Fertil Steril. 1999, 72:207-15. 10.1016/s0015-0282(99)00242-3

SP-21

Tuberculosis of Larynx - A Diagnostic Dilemma

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Abstract: Tuberculosis is a chronic granulomatous infection of larynx caused by Mycobacterium Tuberculosis. It usually occurs secondary to Pulmonary Tuberculosis or by lympho-hematogenous spread from Extra Pulmonary sites. It is commonly misdiagnosed for malignancy of larynx leading to lot of morbidity. Risk of infection is more in Refugees, Immigrants and patients with decreased immunity due to other co morbidities. Though there is marked parallelism in patients with laryngeal and pulmonary tuberculosis patients present with either laryngeal or pulmonary symptoms depending on the site involved or its severity. The so-called local immunity being suppressed by local harmful factors plays a vital role in deciding the susceptibility of the sites to infection. Here, we discuss a case of Tuberculosis of larynx wrongly diagnosed and investigated for malignancy of larynx as the symptoms of Pulmonary Tuberculosis was completely overshadowed by laryngeal symptoms.

Keywords: Tuberculosis, Larynx, Pulmonary Foci.

INTRODUCTION

Tuberculosis infections are traced back to skeletal remnants during excavation under Heidelberg in early Stone Age (5000 BC) and were also known as dry disease or sorrow consumptive. In 1882 an outstanding German Bacteriologist Robert Koch discovered Mycobacterium Tuberculi. Anti tubercular protective vaccination BCG was discovered by K.Geren a French Scientist. In 1907 the skin prick test for diagnosis was started by a Pediatrician Dr.K.Pirke. But till the discovery of a powerful antibiotic Streptomycin in 1943, there was no effective antidote for tubercular infection. After the introduction of anti tuberculosis agents, preventive programs and better socioeconomic conditions, Tuberculosis incidence decreased dramatically up until the 1980s [1]. In subsequent years, however, the epidemic spread of HIV, illicit drug use and the emergence of multi-drug-resistant mycobacterium have resulted in a resurgence of Tuberculosis. In 1993, it became the leading cause of death from a single infectious agent. Increased numbers of migration and travelling to and from less-developed countries also contributed to the worldwide spread of Tuberculosis.[1] Over the past 5 yrs there is a 52% increase in newly diagnosed cases involving Respiratory organs and 2.6 times increase in death rate. Literature data indicate that laryngeal tuberculosis represents generally less than 2% of Extra pulmonary cases.[2,3] *The clinical pattern of laryngeal tuberculosis has also changed in comparison to the past. Earlier, it affected posterior larynx in most patients, since it directly spreads along the airway.*[4] The correct incidence of Laryngeal Tuberculosis for patients with pulmonary Tuberculosis is difficult to be determined because systematic Otorhinolaryngologic evaluation is not usually conducted.[5,6]

CASE REPORT

Presenting Complaints

A 47 yr old male presented to OPD with complaints of change in voice and intermittent low grade fever for the 6 months and Difficulty in breathing for 15 days. Since the patient was working abroad he did not reveal his history fully for the fear of losing the job but once he developed Difficulty in breathing he came back to India. On proper history taking patient had history of cough with expectoration, intermittent low grade fever with diurnal variations, loss of weight and loss of appetite for the past 6 months.

Medical History

Patient had undergone treatment for fever and voice change with antibiotics and antipyretics for 3 months with no significant improvement. The patient was referred by a General Physician to Medical Oncologist for treatment of Malignancy. He was referred by Medical Oncologist to Otorhinolaryngologist to do biopsy for tissue diagnosis as the clinical diagnosis was Malignancy of larynx.

Family History

Patient had no significant family history of Tuberculosis. There was no history of exposure in working environment to his knowledge

Observation

On systematic history taking and Clinical Examination we made some observations that there is a Infective Primary Foci in the lung causing secondary changes in the Larynx. We decided to proceed with Radiological and Endoscopic evaluation to confirm our Diagnosis

Investigations

CT chest showed multiple cavitatory lesions very typical of Pulmonary Tuberculosis. His Video laryngoscopy showed typical Moth eaten appearances of both vocal cords with inter arytenoid ulcerations (Figure 1). His sputum and Bronco-Alveolar Wash were sent for Acid Fast Bacilli. Flexible bronchoscopy was done and small bits of tissue were taken from false cords for Histopathological examination. Bronco-Alveolar Wash and Sputum were positive for Tuberculosis and Biopsy revealed a Granulomatous lesion.

Diagnosis

Laryngeal Tuberculosis with Primary Pulmonary Tuberculosis was the final diagnosis

Treatment

Patient came under category I of NRTCP and needed 6 months regimen.We planned to start on Isoniacid, Ethambutol, Pyrizinamide and Rifampicin regimen, dose depending on his weight, age and severity of the disease. The patient was put on 600mg of Rifampicin, 300mg of Isoniacid, 800mg of Ethambutol and 1800 mg of Pyrizinamide for initial phase of 2 months.Then he was put on control phase with Rifampicin, Isoniacid and Ethambutol.The patient was on regular follow up and was observed meticulously for side effects and adverse reactions.

Prognosis

The patient had very good Prognosis. He started feeling very comfortable by 2nd week with no difficulty in breathing. His appetite improved and started gaining weight by 2 months. His voice became near normal at the end of 6 months. Once the patient finished his full course of 6 months, sputum was sent for AFB and CT chest was done. CT chest showed minimal residual cavity with good aeration of the apex.Sputum was negative and Video laryngoscooy showed near normal larynx (Figure 2). Patient is on regular follow up till date.

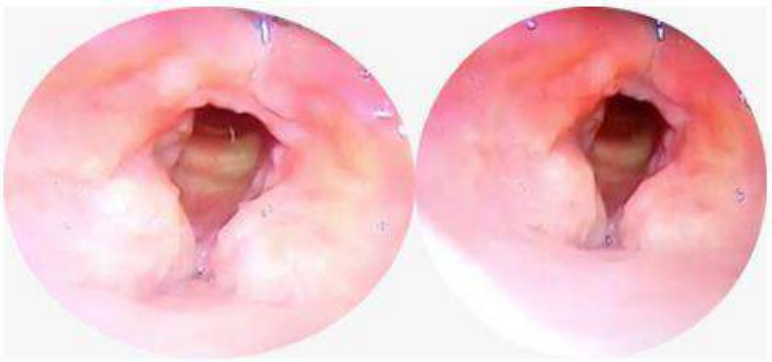

Figure 1: Video laryngoscopy of the patient showing typical nibbled appearance of vocal cords with inter arytenoid granulations

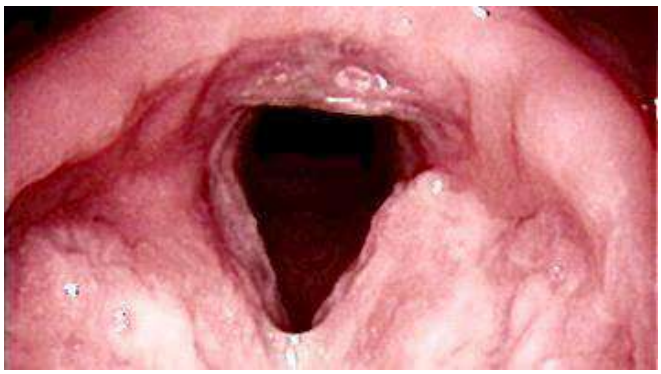

Figure 2: Video laryngoscopy of the patient after the initial phase of regimine showing near normal vocal cords with minimal congestion

DISCUSSION

In the 1920s, in England, Laryngeal Tuberculosis was considered to be the most common disease of the larynx.[7] The incidence and clinical course of Tuberculosis is directly dependent on the duration and form of disease. According to Reudi, Tuberculosis

of larynx is seen in 10% of patients with early Pulmonary Tuberculosis, 30% of patient with long term illness and in 70% of patients who died of pulmonary tuberculosis in their autopsy. Laryngeal involvement is more in exudative open active form of Patients than productive form. In some cases laryngeal infection will be the main presenting symptom of reactivation of dormant inactive foci in lungs. The incidence of Tuberculosis infection is more in larynx than in nose, ear and pharynx which is explained by the bactericidal activity of secretions and the defense mechanism of the lining mucous membrane. Mycobacterium Tuberculi is an immobile Acid Fast Bacilli. They are aerobic and facultative anaerobes with resistance to various environmental factors. According to the majority of studies, Laryngeal Tuberculosis occurs mostly by a direct bacilli spread from a bronchial site through bronchial secretion to the larynx (bronchogenic theory).[8,9] The important source of infection is sputum from patients with Respiratory Tuberculosis which gets dried up in dust and spreads in atmosphere - KochsKornet theory. Infections can also spread by lympho-hematogenous route and from infected cattle via unpasteurized milk (bovine form). Three stages of spread are involved in Tuberculosis of larynx-formation of infiltrates, ulcer and cartilage damage.The infected sputum on reaching the larynx causes superficial maceration, loosening and sloughing of epithelium. The organism penetrates through the damaged epithelium into the closed lymphatic spaces of sub epithelial layers causing superficial infiltrates and tubercular process. This then forms a tuberculoma, followed by ulceration. As the disease progresses cartilage gets involved due to secondary infection. Hematogenous spread is seen in patients with miliary Tuberculosis. It is also observed that the lesion in larynx is in the same side as the Primary foci in lung. This is explained by two theories. One is the lymphogenous spread from affected tubercular lymph node on the same side. Another explanation is the delivery of organism on the same side due to ciliary activity. In hematogenous spread laryngeal involvement occurs randomly throughout the larynx without site or side predilection. Laryngeal Tuberculosis can manifest as edema, hyperemia or ulcerative lesions, but can also present as a nodule, an exophytic mass or obliteration of an anatomical structure.[10] The glosso-epiglottic folds adjacent to the epiglottis present a higher potential for the accumulation of the secretions from the lower airways, which can remain for a long time contributing to the development of infection and illness.[1] On the other hand, by classical acceptance, the posterior part of the larynx is more frequently affected due to the accumulation of infected secretions in patients who spend more time lying in bed.[11] However, this injury pattern is rarely seen nowadays, recent studies showing that the anterior half of the larynx is affected twice more often than the posterior half. Histopathologically, tuberculosis of larynx is classified into chronic infiltrative form, miliary form and lupus form. In chronic infiltrative form there are sub epithelial infiltrates that spread along the mucosa and transform to diffuse form. Then it undergoes caseous decay forming ulcers surrounded by granulomatous formation containing tuberculous nodules. There is also proliferation of connective tissue causing thickening of the mucous membrane. The miliary form which is less common is characterized by diffusely disseminated small nodular infiltrates covering the entire surface of mucous membrane. In lupus form the infiltrates are encapsulated and symmetrically located [laryngitis circumscribed]. Adjacent to the nodular infiltrates are ulcers with superficial cicatricial changes surrounded by dense connective tissue. This type is most commonly seen in epiglottis and is completely destroyed. The most common symptom is change in voice associated with pain. As the disease progresses patient may have odynophagia due to involvement of cartilage. Odynophagia occurs in case of the involvement of the epiglottis, aryepiglottic folds and arytenoid regions. These laryngeal sites have stronger movements during deglutition, therefore a higher potential of generating painful stimuli.[12] Detailed history usually leads us to underlying pulmonary foci like cough, fever with diurnal variations, loss of appetite and weight. On examination hyperemia of the posterior 1/3 of vocal cords and arytenoids is the earliest sign. Turban epiglottis is due to pseudo edema of epiglottis, Moth eaten or mouse nibbled appearance of vocal cords due to ulceration of the free margins, mamillary bodies in the inter arytenoid region due to superficial ragged ulcers are also seen. The lesions of the larynx are variable and may appear as ulcerative, granulomatous, polypoid or non-specific inflammatory.[13] The important differential diagnosis include Sarcoidosis, Scleroderma, Malignancy, Syphilis, Wegener's and Lupus. It is not uncommon that Tuberculosis located in other organs mimics other diseases, thus delaying the diagnosis or even worse, lead to wrong therapeutic decisions. Diagnosis depends on Radiological assessment of Lungs and sputum for Acid Fast Bacilli. In some cases Gene X pert helps to clinch the diagnosis. Biopsy of the false or true vocal cords can help in cases where these measures have failed. Treatment is based on the organs involved, severity, age and weight of the patient. The laryngeal framework is restored to normal in about 18 weeks by anti tuberculosis medication. The fibrotic healing of the tuberculosis lesions may lead to long-term compromise of voice.[14,15] Surgery may be required in case of complications like posterior glottic stenosis, vocal cord fixity when crico arytenoid joint is affected and paralysis when recurrent laryngeal nerve is involved. They are kept under regular follow up and advised strict sanitary measures to prevent spread of the infection. The aim of treating tuberculosis is to reduce the case fatality and morbidity by ensuring relapse free cure.it also aims in preventing drug resistance and to break the chain of transmission. The RNTCP [REVISED NATIONAL TUBERCULOSIS CONTROL PROGRAM] divides the patient into 3 category. They are newly diagnosed case, relapse and failure cases and sputum negative cases. The patients were put on 2 phase during the regimen. The first initial phase focuses in quick killing of bacilli and reducing the high bacterial load. It is for 2 months. The 2nd control phase is for 4 months and it kills persistent bacilli and prevents relapse. RNTCP is now renamed as national tuberculosis elimination programme [NTEP] in January 2020 focussing on eradicating tuberculosis by 2025.

CONCLUSION

Tuberculosis of larynx is an often missed diagnosis. It is completely curable with no residual morbidity. The prognosis depends on the severity of the disease, the organs involved, immune status and associated co morbid conditions. Systemic symptoms have become rare. Proper history taking, systematic clinical evaluation supported with radiological and microbiological investigations should help us in early diagnosis and effective management. While we talk on early diagnosis, preventive measures play a major role in controlling the spread of disease. Social awareness, hygienic habits and counseling help in keeping the spread of tuberculosis under control.

CONFLICT OF INTEREST

Conflict of interest declared none.

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REFERENCES

1. Rizzo PB, Da Mosto MC, Clari M, Scotton PG, Vaglia A, Marchiori C: Laryngeal tuberculosis: an often forgotten diagnosis. Int | Infect Dis. 2003, 7: 129-131. 10.1016/S1201-9712(03)90008-7.
2. Williams RG, Douglas-Jones T: Mycobacterium marches back. J Laryngol Otol. 1995, 109: 5-13. 10.1017/S0022215100129123.
3. Nishiike S, Irifune M, Doi K, Sawada T, Kubo T (2002) Laryngeal tuberculosis: a report of 15 cases. Ann OtolRhinolLaryngol 111: 916-918.
4. Nalini B, Vinayak S. Tuberculosis in ear, nose, and throat practice: its presentation and diagnosis. American Journal of otolaryngology. 2006 Jan 1;27(1):39-45.
5. Reis JG, Reis CS, Costa DC, Lucena MM, Schubach AD, Oliveira RD, Rolla VC, Conceição-Silva F, Valete-Rosalino CM. Factors associated with clinical and topographical features of laryngeal tuberculosis. PloS one. 2016 Apr 14;11(4):e0153450.
6. Topak M, Oysu C, Yelken K, Sahin-Yilmaz A, Kulekci M. Laryngeal involvement in patients with active pulmonary tuberculosis. European archives of oto-rhino-laryngology. 2008 Mar;265(3):327-30.
7. Thomson S. The Mitchell Lecture On Tuberculosis Of The Larynx: Its Significance To The Physician: Deliverd before the Royal College of Physicians of London, November 6th, 1924. British medical journal. 1924 Nov 8;2(3332):841.
8. Bailey CM, Windle- Taylor PC. Tuberculous laryngitis: a series of 37 patients. The Laryngoscope. 1981 Jan;91(1):93-100.
9. Lim JY, Kim KM, Choi EC, Kim YH, Kim HS, Choi HS. Current clinical propensity of laryngeal tuberculosis: review of 60 cases. European Archives of Oto-Rhino-Laryngology and Head & Neck. 2006 Sep;263(9):838-42.
10. Yencha MW, Linfesty R, Blackmon A: Laryngeal tuberculosis. Am J Otolaryngol.2000, 21: 122-126.10.1016/S0196-0709(00)85010-3.
11. Kandiloros DC, Nikolopoulos TP, Ferekidis EA, Tsangaroulakis A, Yiotakis JE, Davilis D, Adamopoulos GK. Laryngeal tuberculosis at the end of the 20th century.The Journal of Laryngology & Otology. 1997 Jul;111(7):619-21.
12. Chen J, Yang ZG, Shao H, Xiao JH, Deng W, Wen LY, Tang SS. Differentiation of tuberculosis from lymphomas in neck lymph nodes with multidetector-row computed tomography. The International journal of tuberculosis and lung disease. 2012 Dec 1;16(12):1686-91.
13. Shin JE, Yuhl Nam S, JooYoo S, Yoon Kim S. Changing trends in clinical manifestations of laryngeal tuberculosis. The Laryngoscope. 2000 Nov;110(11):1950-3.
14. Akkara SA, Singhania A, Akkara AG, Shah A, Adalja M, Chauhan N. A study of manifestations of extrapulmonary tuberculosis in the ENT region.Indian Journal of Otolaryngology and Head & Neck Surgery. 2014 Jan 1;66(1):46-50.
15. Harney M, Hone S, Timon C, Donnelly M: Laryngeal tuberculosis: an important diagnosis. J Laryngol Otol. 2000, 114: 878-880.

SP-22

Left Lateral Rectus Palsy: A Case Report

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Abstract: Abducens nerve has the longest intracranial course. Hence commonly affected due to increased intracranial pressure. Sixth cranial nerve palsy due to raised intracranial pressure, termed as false localizing sign, presents as squint and diplopia. Common cause of increased intracranial pressure is hydrocephalus infection and intracranial tumors. Early diagnosis and management based on multimodality approach for early recovery. Here we discuss a case report of a 11 years old boy who came to our hospital with alleged history of trauma over the occipital region by a marble stone while playing at his residence, following injury there was no history of convulsions, loss of consciousness. After 5 days he developed giddiness followed by vomiting and double vision. CT and MRI imaging was normal, diplopia charting revealed weakness of left lateral rectus muscle. Child was treated with oral steroids and multivitamins. On follow-up he recovered completely. If any child who presented with isolated sixth nerve palsy (abducens nerve), thorough clinical history should be taken. Multimodality approach involving pediatrician, neurologist and ophthalmologist is mandatory. Early diagnosis and management prevents further complications.

Keywords: Abducens Nerve, Diplopia, Intracranial pressure, Squint, Extraocular movements (EOM)

INTRODUCTION

Among 12 cranial nerves abducens nerve, the sixth nerve has the longest intracranial course. Sixth nerve palsy, a disorder that affects eye movement. Hence commonly affected due to increased intracranial pressure. Common cause of increased intracranial pressure is hydrocephalus infection and intracranial tumours.[1] Other causes are trauma, vascular malformation, meningitis, Gradenigo syndrome. There are six syndromes associated with lesion of sixth cranial nerve they are as follows Brainstem Syndrome, elevated intracranial pressure syndrome, Petrous apexsyndrome, Cavernous sinus syndrome, Orbital syndrome of sixth nerve, Isolated sixth nerve palsy syndrome[2,3].Abducens nerve supplies ipsilateral lateral rectus muscle and pathology of this nerve results in abduction palsy.

CASE REPORT

Presenting complaints

11 years old boy came to our hospital with complaints of double vision with tilting of head towards left side to avoid double vision since morning. His informant is mother and reliability is good.

Medical history

No relevant medical history.

Past History

He had alleged history of trauma over the occipital region due to hit by a marble stone while playing at his residence before 5 days, after few minutes of trauma he had one episode of vomiting, containing food particles which is non-projectile, non-bile stained. There was no history of convulsions, loss of consciousness, headache, bleeding from the injured site.

FIGURE 1: Showing loss of abduction, levo depression and levo

elevation of left eye

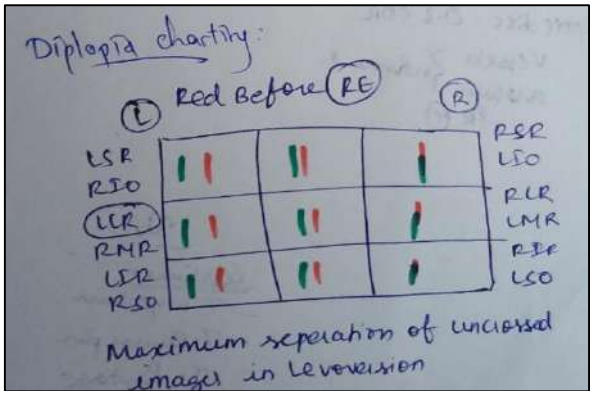

FIGURE 2: Showing Diplopia charting

Family History

No relevant family history of head trauma, no history of seizure or neurological disorders in family

Observation

CNS examination was done, were in cranial nerve examination of his Extraocular muscles (shown in fig 1) adduction, levodepression and levoelevation was restricted in his left eye but right eye EOM movements are normal.Rest of the cranial nerves, motor, sensory, cerebellar, spine and cranial examination was normal.

Special test

CT Brain was taken, which appears to be normal. Patient was suspected to be a case of left lateral rectus palsy so advised to take MRI brain along with orbit.

Investigation

Complete blood count and ESR appears to be normal, Radiologist, Neurologist and ophthalmologist opinions was obtained. His MRI Report were normal, diplopia charting (shown in fig 2),revealed weakness of left lateral rectus muscle.

Treatment

This Child was treated with oral steroids prednisolone at 2mg/kg for two weeks, Tablet Renerveplus for 2 weeks, left eye shielding, multivitamins. On follow-up he recovered completely. After one-month spontaneous resolution was observed.

Prognosis

Early diagnosis and management give good prognosis.

DISCUSSION

Course of sixth cranial nerve - It leaves the brainstem at pontomedullary junction in an upward and outward pathway. It ascends via the subarachnoid space and penetrates the dura mater along petrous part of the temporal bone. Above the petrous part of the sphenoid bone, it makes an angle of 120° & it reaches the cavernous sinus and runs along the internal carotid artery. Then enters the orbit via the superior orbital fissure and gives nerve supply to the lateral rectus muscle[3].As it has the longest course injuries are more prone to occur. The causes are head injuries, neoplasms like meningioma, Acoustic neuroma, and nasopharyngeal carcinoma. Other causes are idiopathic, congenital, hydrocephalus, infections like otitis media, and others. Nearly 12% to 42% of acquired 6th nerve palsies are due to trauma.[4,15] The incidence of 3rd, 4th and 6th cranial nerve palsies in a pediatric population was reported to be 7.6 per 100 000 population.[10] Pediatric abducens nerve palsy is very rare. Assessment of extraocular muscle done by guiding the child to look in nine different directions by following the finger. The child will look at up, down, right, left, up and right, up and left, down and right, down and left. Robertson described tumours of the posterior fossa to be responsible for 39% of sixth nerve palsy in a cohort of 133 children.[5,11]Isolated sixth nerve palsies occurs followed by viral infections and followed by immunization of live attenuated vaccines.[12] The prognosis of 6th nerve palsy is good. Failure to improve suggests more serious intracranial pathology.[13] Sixth nerve palsy was first diagnosed by Knox et al[6] that occurs in children rarely after respiratory tract infection. He reported 12 children with a sixth nerve palsy. Out of which 3 patients had otitis media complicated with Gardeningo syndrome. Our case, given the fact that the patient could attend frequent ophthalmic follow-up visits. Treatment in Pediatric age groups includes alternate patching which is used to prevent amblyopia in the affected eye. Prism therapy is temporary press on Prism on the lens of affected eye. If the Prism therapy fails then strabismus

surgery is intervened. Botulism injection on the affected eye prevents nasal deviation and contracture.[7,8,9] The majority of cases were self-limiting and do not require medical management.

CONCLUSION

If a child presents with isolated sixth nerve palsy, thorough clinical history should be taken. It is essential to rule out any underlying pathology like demyelinating disorders, malignancy or infection affecting CNS. Multimodality approach involving pediatrician, neurologist and ophthalmologist is mandatory.

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CONFLICT OF INTEREST

Conflict of interest declared none.

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REFERENCES

1. Abbott MB, Vlasses CH. Nelson textbook of pediatrics. Jama. 2011 Dec 7;306(21):2387-8.
2. Rucker CW. The causes of paralysis of the third, forth, and sixth cranial nerves. Am J Ophthalmol. 1966;61:1293–1298.
3. Kodsi SR, Younge BR. Acquired oculomotor, trochlear, and abducent cranial nerve palsies in pediatric patients. American journal of ophthalmology. 1992 Nov 1;114(5):568-74.
4. Lee MS, Galetta SL, Volpe NJ, Liu GT. Sixth nerve palsies in children. Pediatric neurology. 1999 Jan 1;20(1):49-52.
5. Shumway CL, Motlagh M, Wade M. Anatomy, head and neck, eye extraocular muscles. StatPearls [Internet]. 2021 Aug 11.
6. Knox DL, Clark DB, Schuster FF. Benign VI nerve palsies in children. Pediatrics. 1967 Oct 1;40(4):560-4.
7. Rowe FJ, Hanna K, Evans JR, Noonan CP, Garcia- Finana M, Dodridge CS, Howard C, Jarvis KA, MacDiarmid SL, Maan T, North L. Interventions for eye movement disorders due to acquired brain injury. Cochrane Database of Systematic Reviews. 2018(3).
8. Sabermoghadam A, EtezadRazavi M, Sharifi M, Kiarudi MY, Ghafarian S. A modified vertical muscle transposition for the treatment of large-angle esotropia due to sixth nerve palsy. Strabismus. 2018 Jul 3;26(3):145-9.
9. Leiba H, Wirth GM, Amstutz C, Landau K. Long-term results of vertical rectus muscle transposition and botulinum toxin for sixth nerve palsy. Journal of American Association for Pediatric Ophthalmology and Strabismus. 2010 Dec 1;14(6):498-501.
10. Holmes JM, Mutyala S, Maus TL, Grill R, Hodge DO, Gray DT . Pediatric third, fourth, and sixth nerve palsies: a population-based study. Am J Ophthalmol 1999; 127: 388–392.
11. Gupta K, Orisme W, Harreld JH, Qaddoumi I, Dalton JD, Punchihewa C, Collins-Underwood R, Robertson T, Tatevossian RG, Ellison DW. Posterior fossa and spinal gangliogliomas form two distinct clinicopathologic and molecular subgroups. Acta neuropathologica communications. 2014 Dec;2(1):1-9.
12. Cheng D. R., Crawford N. W., Hayman M., Buckley C., Buttery J. P. Recurrent 6 th nerve palsy in a child following different live attenuated vaccines: Case report. BMC Infectious Diseases. 2012;12, article no. 105 doi: 10.1186/1471-2334-12-105.
13. Galetta S. L., Smith J. L. Chronic isolated sixth nerve palsies. JAMA Neurology. 1989;46(1):79–82.
14. Elder C, Hainline C, Galetta SL, Balcer LJ, Rucker JC. Isolated abducens nerve palsy: update on evaluation and diagnosis. Current neurology and neuroscience reports. 2016 Aug;16(8):1-7.
15. Mahoney NR, Liu GT. Benign recurrent sixth (abducens) nerve palsies in children. Archives of disease in childhood. 2009 May 1;94(5):394-6.

SP-23

Management of an Ovarian Teratomain Pregnancy-A Case Report

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Abstract: Ovarian teratoma also called as a dermoid cyst of the ovary is usually benign in nature and typically contains a diversity of tissues including hair, teeth, bone thyroid etc. In the pre-ultrasonography era, the incidence of ovarian neoplasmswith pregnancy is about 1in 2000. With routine ultrasonography and an increase in early detection, the incidence has increased to 2.2%. The majority are benign tumors. Its seen more during pregnancy owing to the frequent antenatal ultrasounds. Mostly ovarian cysts do resolve spontaneously only the cysts that persists, during pregnancy their management remains controversial and varies. The size may vary from small to very large tumor. Small tumors are asymptomatic, but large tumors present with mass or abdominal pain. They can undergo torsion or rupture and occasionally obstruct labour. Malignancy has to be excluded before conservative management is decided upon. Most benign tumors are asymptomatic and regress spontaneously. When the ovarian tumor is persistent, the enlarging uterus pushes it into the abdomen after 12-14 weeks. They may occasionally be wedged posteriorly and obstruct labor. Rarely, malpresentation occur if there is torsion, it usually occurs in the second trimester or in the puerperium, when the tumors are freely mobile in the abdomen.

Keywords: Teratoma, pregnancy, caesarean, preterm

INTRODUCTION

Mature cystic teratoma (dermoid cyst) is one of the most common benign ovarian neoplasms discovered during pregnancy (24–40%) [1]Higher prevalence is seen during reproductive period and its picked up more during frequent visits made in the antenatal period. Ovarian malignancy is rare in pregnancy.[1] There is increased chance of impaction leading to retention of urine, mechanical distress in presence of large tumour, malpresentations and non engagement of head during delivery. In labour there is higher incidence of obstruction and if the growth is impacted in the pelvis.[2] There will be exaggeration of signs and symptoms in an antenatal woman as compared to anon-pregnant woman. Rupture is rare, but once it has occurred it can cause complications such as chemical or granulomatous peritonitis mimicking advanced ovarian malignancy.[3-6] Teratoma in pregnancy often incidentally diagnosed, rarely do they present with pain abdomen. This case shows us a regular follow up of an ovarian teratoma and its management in pregnancy. Most benign tumors are diagnosed on routine ultrasonography in the first or second trimester. When masses are larger than 5-6cm or persistent after 18 weeks, the risk of complications is higher. When there is suspicion of malignancy or a complication such as torsion, hemorrhage or rupture, immediate surgery is indicated irrespective of gestational age. Persistent cysts with benign features and <8cm in size may be followed up or aspirated under ultrasound guidance. Dermoid cysts can be left alone and be removed postnatally. Complex masses, large tumors > 8 cm, and tumors that continue to increase in size should be removed laparoscopically or by laparotomy.

CASEREPORT

Presenting complaints

A 26 year old woman a primigravida came on her first antenatal visit at 7weeks. The patient presented to us now at 34 weeks with complaints Ofpreterm premature rupture of membranes associated with complaints of lower abdomen pain for the last 3 days, pain radiating to the back.

Menstrual history

Regular menstrual cycles 3-4/28 days cycle, normal flow, dysmenorrhea++

Marital history

Married for 11 months

Obstetric history

Primigravida Booked and immunized with us Patient on follow up and regular Antenatal visits.The patient was monitored regularly to visualize the size of the cysts and to rule out torsion the size gradually increased.

Past history

No significant past history, no history of any surgeries in the past.

Family history

Nil significant

On examination

Abdominal examination showed abdomen distended from the period of gestation Fetal heart rate present along with diffuse lower abdomen pain. Per vaginal examination showed active leaking along with cervix 25% effaced and Os admits 1 finger.

Radiological investigation

Ultrasound done showed bilateral adnexal cyst of size 8.4x8.1 cm and 8.29x9.5 cm in Right and left sides respectively done as booking visit.

Special tests

Tumor markers done AFP- 0.30, CEA- 0.56, Beta HCG- 4826.7, CA- 125 – 8.8.

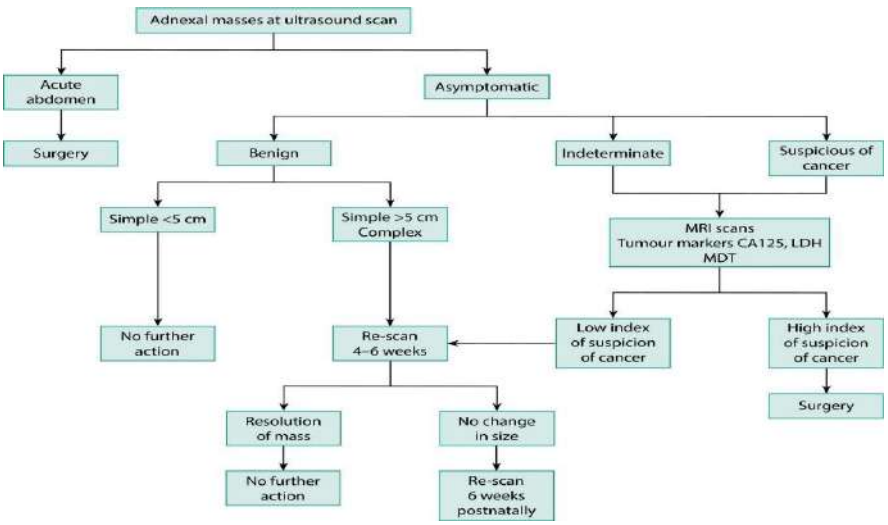
Management

An emergency caesarean section was performed following which a 9x8cm mass on her left ovary and 9x10cm mass on her right ovary was seen intra-operatively. Proceeded with the surgery where the bilateral cysts were removed, the mass appeared tan with punctate focal haemorrhage. Histopathology report confirmed the diagnosis of mature cystic teratoma. Her postoperative course was uncomplicated.

DISCUSSION

Germ cell tumor are the most frequent ovarian tumors in young women aged 20-40 years, accounting for more than 50% of ovarian tumors with a peak incidence in the early 20s.[7-9] Mature dermoid cyst is the most common form of benign germ cell tumor which contains fully differentiated tissue types derived from all three embryonic germ cell layers (mesenchymal, epithelial and stroma). Hair, teeth, fat, skin, muscle, cartilage, bone and endocrine tissue are frequently present. Upto 10% of dermoid cysts are bilateral. The risk of malignant transformation is rare (<2%), usually occur over 40 years of age.[10-13] Abdominal palpation reveals the cystic swelling felt distinctly from the gravid uterus. The patient is examined in trend elenburg position to feel the groove between the gravid uterus and the ovarian tumour vaginally. Ultrasonography iscost effective tool in describing ovarian tumor. MRI is recommended for more precise information about the tumour. A vigilant expectancy for vaginal delivery is followed if the tumour is well above the presenting part.Caeserean section should be performed if the tumoris impacted in pelvis resulting in obstruction and removal of the tumor can also be performed in same sitting.[14,15]As pregnancy advances, ovarian mass may get displaced from its position, ovarian commonly encountered during 8 to10 weeksofpregnancyasthe tumour is out of the pelvis and also following early puerperium due to lax abdomen wall. Intra-cystic haemorrhage inside ovarian cyst is due to increased vascularity. Infection occurs following miscarriage and delivery. Physiological event of thrombosis initiate sepsis. Patient may be asymptomatic or may present with urinary retention due to impacted tumor. Large cyst causes mechanical distress and acute abdomen is adverse effect of the tumour.

FLOWCHART TO MANAGEMENT OF TERATOMA IN PREGNANCY



CONCLUSION

In our case we regularly monitored the patient to watch for the size of the cysts and to rule out torsion fetal growth restriction intra-cystic hemorrhage rupture infection and pre-term deliveries. The ideal time of elective operation will be around14 to18 weeks of gestation as the risk of abortion is less and pedicle can be accessed easily. Surgery can be with held until delivery if the patient is beyond 36 weeks of gestation and tumor can be removed as early in peurperium as possible. In case of any complications tumor must be removed irrespective of the period of gestation.However early prophylactic careful intercession ideally with laparoscopy ought to be considered for ovarian masses between 5 cm and 10 cm, masses under 5 cm can be noticed. Finally, masses more prominent than 10 cm ought to be prophylactically resected. Doctor should rehearse their best judgment while overseeing ovarian masses in pregnancy. Albeit only one case is deficient to reach out any critical inferences or give proposals, it features the requirement for additional exploration with respect to ovarian twist from mature teratomas in pregnancy.

CONFLICT OF INTEREST

Conflict of interest declared none.

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FUNDING SOURCE: None

ETHICAL STATEMENT: None

REFRENCES

1. Comerci JT, Licciardi F, Bergh PA, Gregori C, Breen JL: Mature cystic teratoma: a clinicopathologic evaluation of 517 cases and review of the literature. Obstet Gynecol.1994, 84:22-28.
2. Peterson WF, Prevost EC, Edmunds FT, Hundley JM, Morris FK: Benign cystic teratomas of the ovary; a clinico-statistical study of 1,007 cases with a review of the literature. Am J Obstet Gynecol.1955, 70:368-382.
3. Stuart GC, Smith JP: Ruptured benign cystic teratomas mimicking gynecologic malignancy. Gynecol Oncol.1983,16:139-143.
4. Suprasert P, Khunamornpong S, Siriaunkgul S, Phongnarisorn C, Siriaree S: Ruptured mature cystic teratomas mimicking advanced stage ovarian cancer: a report of 2 cases study. J Med AssocThai. 2004, 87 (12):20041522-1525.
5. Phupong V, Sueblinvong T, Triratanachat S: Ovarian teratoma with diffused peritoneal reactions mimicking advanced ovarian malignancy. Arch Gynecol Obstet. 2004, 270:189-191.
6. Pepe F, Lo Monaco S, Rapisarda F, Raciti G, Genovese C, Pepe P: An unusual case of multiple and bilateral ovarian dermoid cysts. Case report. G di Chir. 2014, 35:75-7. 10.11138/gchir/2014.35.3.075
7. Hasson J, Tsafrir Z, Azem F, et al.: Comparison of adnexal torsion between pregnant and nonpregnant women. Am J Obstet Gynecol. 2010, 202:536.e1-6. 10.1016/j.ajog.2009.11.028
8. Tan KH, Chen KC, Wang TL, Chong CF, Chen CC: Ovarian cystic teratoma torsion in pregnancy. Emerg Med J. 2010, 27:879-80. 10.1136/emj.2008.063883
9. Hasiakos D, Papakonstantinou K, Kontoravdis A, Gogas L, Aravantinos L, Vitoratos N: Adnexal torsion during pregnancy: report of four cases and review of the literature. J ObstetGynaecol Res. 2008, 34:683-7. 10.1111/j.1447-0756.2008.00907.x
10. Smorgick N, Pansky M, Feingold M, Herman A, Halperin R, Maymon R: The clinical characteristics and sonographic findings of maternal ovarian torsion in pregnancy. FertilSteril. 2009, 92:1983-7. 10.1016/j.fertnstert.2008.09.028
11. Walid MS, Boddy MG: Bilateral dermoid cysts of the ovary in a pregnant woman: case report and review of the literature. Arch Gynecol Obstet. 2009, 279:105-8. 10.1007/s00404-008-0695-3
12. Peña JE, Ufberg D, Cooney N, Denis AL: Usefulness of Doppler sonography in the diagnosis of ovarian torsion. FertilSteril. 2000, 73:1047-50. 10.1016/S0015-0282(00)00487-8
13. Caspi B, Levi R, Appelman Z, Rabinerson D, Goldman G, Hagay Z: Conservative management of ovarian cystic teratoma during pregnancy and labor. Am J Obstet Gynecol. 2000, 182:503-5. 10.1067/mob.2000.103768
14. Kolluru V, Gurumurthy R, Vellanki V, Gururaj D: Torsion of ovarian cyst during pregnancy: a case report. Cases J. 2009, 2:9405. 10.1186/1757-1626-2-9405
15. Yakasai IA, Bappa LA: Diagnosis and management of adnexal masses in pregnancy. J Surg Tech Case Rep. 2012, 4:79-85. 10.4103/2006-8808.110249
16. Hess LW, Peaceman A, O'Brien WF, Winkel CA, Cruikshank DP, Morrison JC: Adnexal mass occurring with intrauterine pregnancy: report of fifty-four patients requiring laparotomy for definitive management. Am J Obstet Gynecol. 1988, 158:1029-34. 10.1016/0002-9378(88)90212-8

SP-24

Management of Post Traumatic Nasal Deformities

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Abstract: Nasal deformity following trauma and the cosmetic and functional problems after injury is a very common reason for patients seeking septorhinoplasty. Nasal bone fractures occur frequently because the nasal bone is located at the forefront of the face. The commonest cause of injury was sporting activities. Nasal septal fractures have been associated with nasal bone fractures in 42% to 96% of cases.Nasal bone and septal fractures have an impact not only on cosmetic appearance but also on nasal airway function as well. Although many methods and algorithms have been proposed for management of specific posttraumatic nasal deformities, such as twisted, deviated, saddle, or short nose, these algorithms usually focus on a specific deformity in isolation from the remainder of the nose. Their management is further complicated by the presence of fractured or significantly deformed septal cartilage. The final deformity will not only depend on the age at which it occurred but also the mechanism, severity, and direction of the original trauma. As such, their expectations differ from patients who want to undergo rhinoplasty only for cosmetic reasons. Posttraumatic nasal deformity correction requires attention to numerous defects and anatomical scenarios.

Keywords: Nasal deformity, Septorhinoplasty, nasal complex deformity.

INTRODUCTION

Among the aesthetic surgical procedures performed in the world, rhinoplasty is the oldest performed in the history. It was initially developed in ancient Greece and India (Sushruta Samhita). Sushruta is considered as the father of rhinoplasty surgery in the world.[1] The first documented evidence of the available records regarding rhinoplasty comes from Edwin smith papyrus. This surgery has varied indications ranging from functional obstructions in the nasal cavity to pure cosmetic reasons and these include asymmetry of the alar cartilages or upper lateral cartilages, nasal tip with lack of support, rotation or projection, over projected nose, nasal tip asymmetry or deformities, crooked nose, saddle nose, cleft lip nose or other congenital anomalies, septal perforations and revision rhinoplasty.[2-4] Septorhinoplasty has been considered to be one of the most difficult aesthetic surgical procedures.[5-7] There are multiple variables contributing to the difficulty of this operation including skin thickness, interplay between bone, cartilage, mucosa and patient's expectations.[8,9] The saddle-nose deformity and crooked-nose deformity are two problems which are very challenging to treat in post-traumatic nose patients (Figure 1).[10] Their surgical treatment involves modification of the septum, and may include reconstruction of the L-shaped dorsalcaudal strut. Knowledge about the three dimensional anatomy, and also about procedures currently in use for stabilising this structure, is necessary to the operating surgeon to maintain the functional integrity and the structural integrity of the nose.[11]

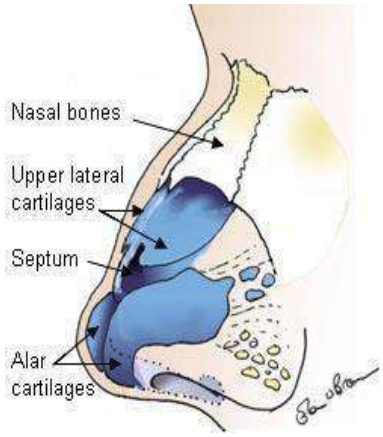

Fig 1: Bones and Cartilages That Form Nose

CASE HISTORY

- We treated 20 cases of post-traumatic nasal deformity. Patients presented to the ENT OPD with nasal obstruction and nasal complex deformity following trauma (Figures 2 and 3).
- The commonest cause of injury was sports related injuries, road traffic accidents and assault. The commonest preoperative objective findings included airflow obstruction and septal deformity.

Fig 2: Case 1 Preop and Post op

Fig 3: Case 2 Preop and Post op

- Diagnostic Nasal Endoscopy (DNE) and Computed Tomography scan of the nose and paranasal sinuses (PNS) were done to get a better understanding of the anatomy and extent of the defect (Figure 4).
- All patients underwent Septorhinoplasty under general anaesthesia, where we established structural and functional correction.

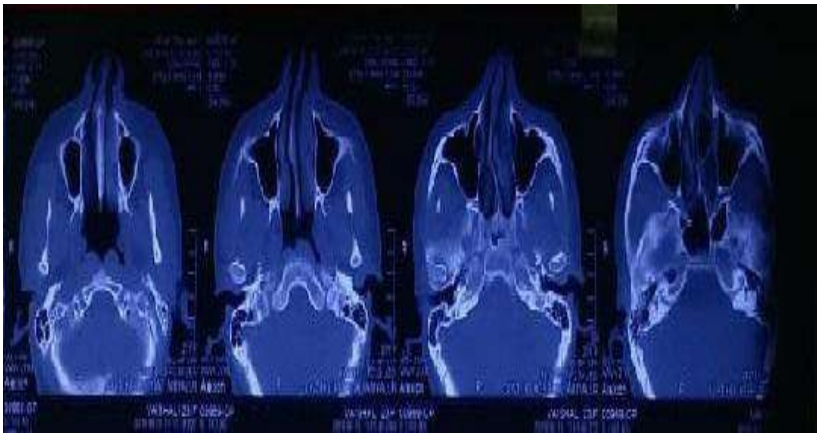

Fig 4: Ct Paranasal Sinus Showing Deviation of the Septum

DISCUSSION

Keen examination of the numerous defects and evaluation of the various anatomical scenarios are required to correct the post-traumatic deformities of the nose.[5]The saddle-nose deformity and crooked-nose deformity are two problems which are very challenging to treat in post-traumatic nose patients. Their surgical treatment involves modification of the septum, and may include reconstruction of the L-shaped dorsalcaudal strut.[12]

Preoperative Evaluation

To decide on the best surgical technique to be utilised to treat a patient, the surgeon has to thoroughly examine the patient through keen inspection and palpation to identify the anatomical sites which need correction.[13] The width and the symmetry of the nose should be thoroughly evaluated. Parallel dorsal lines of the frontal nasal contour should be best done by examining the patient from above the head. Proper lighting placed over the head region is necessary to identify the minute irregularities.

Septorhinoplasty

When a rhinoplasty is performed for deviation of the external nose, most of which are post traumatic, it is very essential to correct the septal deviation to get the desired results.[14] Depending on the type of approach it can be divided into:

1. External approach – Here incision is made on the outside skin, hence called "External or open rhinoplasty". This provides the surgeon the best chance for symmetric reconstruction (Figure 5).
2. Internal approach – Here there is no incision on the outside of the skin, hence called "Internal or closed rhinoplasty".
3. The principle of septorhinoplasty includes the following steps:

1. Septoplasty
2. Tip correction which may include remodeling, projection or rotation.
3. Removal of the hump
4. Narrowed of the nose with osteotomies
5. Final correction of the deformities

Fig 5: Case 3 Preop and Postop Images

Steps of Septorhinoplasty (External Approach)

- Incision: A transcollumellar incision in a form of inverted V to minimize the resultant scar which usually is invisible post operatively along the margin of the lower lateral cartilage (Figure 6).[9]

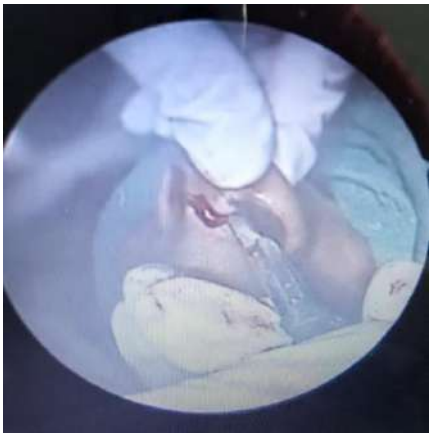

Fig 6: Inverted V Incision

- A skin flap is elevated with the help of sharp scissors to expose the nasal tip and lower lateral cartilage.
- For correction of the septal deformity, fibrous tissue between the two lower lateral cartilages is separated to expose the caudal end of the septum. The perichondrial incision is given at the caudal end to elevate the mucoperichondrial flap.
- To correct the deviated nose, elevation of the septal flaps is done on both the sides and the septal cartilage is separated from the lower and upper lateral cartilages. If necessary to correct the dorsal deformity incision and excision of a portion of upper lateral cartilages may be necessary. Hump can be directly visualized and should be corrected before doing other procedures including septal surgery.
- Osteotomies are very important to correct the bony deviation. A median oblique osteotomy followed by a lateral osteotomy is done till the level of the frontomaxillary suture line at the root of the nose. Both the lateral osteotomies are joined together in the midline. Thus the cartilage and bony framework can be mobilized easily and can be brought to the midline to correct the deformity.
- Sometimes a hump is in fact a pseudohump which is due to depressed tip as a result of previous septal procedure due to excessive resection of inferior strip of cartilage or gross subluxation or fractured nasal septum cartilage.
- The hump removal is done by reduction rhinoplasty.

- When the surgery is complete, a splint is applied to help the nose maintain its new shape. Nasal packs or soft plastic splints also may be placed in nostrils to stabilize the septum.

Complications

General complications

1. Surgical site infection
2. Bleeding
3. Pain
4. Blood clots

Specific complications

1. Swelling
2. Bruising
3. Redness
4. Bleeding
5. Scarring
6. Abscess
7. Haematoma
8. Nerve injury
9. Cosmesis
10. Rejection of graft
11. Obstruction of nose
12. Toxic shock syndrome
13. Reduction in sensation of smell

However these complications were not encountered.[15]

CONCLUSION

Nasal fractures are the most common fractures in adults. Among patients who suffer midface trauma, nasaldeviation/deformity and obstruction/disturbance in nasal airflow are also seen. Postoperatively, patients considered the nasal appearance and airway to be satisfactory or significantly improved. An otorhinolaryngology surgeon provides functional and structural correction of the nose, whereas a plastic surgeon may be able to provide only cosmetic correction. Hence it is concluded that this type of surgery is effective in improving the appearance and function of the traumatized nose and postoperatively, these patients will not complain of nasal obstruction or a nasal-complex deformity.

CONFLICT OF INTEREST

Conflict of interest declared none.

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ETHICAL STATEMENT: None

REFERENCES

1. Hopkins C. Patient reported outcome measures in rhinology. Rhinology. 2009 Mar 1;47(1):10-7.
2. Buckland JR, Thomas S, Harries PG. Can the Sino- nasal Outcome Test (SNOT- 22) be used as a reliable outcome measure for successful septal surgery?. Clinical Otolaryngology & Allied Sciences. 2003 Feb;28(1):43-7.
3. Gray LP. Deviated nasal septum incidence and etiology. Annals of Otology, Rhinology & Laryngology. 1978 May;87(3_suppl2):3-20.
4. Baumann I, Baumann H. A new classification of septal deviations. Rhinology. 2007 Sep 1;45(3):220-3.
5. Becker SS, Dobratz EJ, Stowell N, Barker D, Park SS. Revision septoplasty: review of sources of persistent nasal obstruction. American journal of rhinology. 2008 Jul;22(4):440-4.
6. Singh A, Patel N, Kenyon G, Donaldson G. Is there objective evidence that septal surgery improves nasal airflow?. The Journal of Laryngology & Otology. 2006 Nov;120(11):916-20.
7. Pirila T, Tikanto J. Unilateral and bilateral effects of nasal septum surgery demonstrated with acoustic rhinometry, rhinomanometry, and subjective assessment. American journal of rhinology. 2001 Mar;15(2):127-34.
8. Naito K, Cole P, Chaban R, Oprysk D. Nasal resistance, sensation of obstruction, and rhinoscopic findings compared. American Journal of Rhinology. 1988 Mar;2(2):65-9.
9. Courtiss EH, Goldwyn RM. The effects of nasal surgery on airflow. Plastic and reconstructive surgery. 1983 Jul 1;72(1):9-21.
10. Sipila J, Suonpaa J. A prospective study using rhinomanometry and patient clinical satisfaction to determine if objective measurements of nasal airway resistance can improve the quality of septoplasty. European archives of oto-rhino-laryngology. 1997 Aug;254(8):387-90.

11. Cheng, LH., Lee, JC., Wang, HW. *et al.* Twisted nose: a new simple classification and surgical algorithm in Asians. *Eur Arch Otorhinolaryngol* 269, 551–556 (2012).
12. Higuera S, LeeEI, ColeP, HollierLHJr, StalS. Nasal trauma and the deviated nose. *PlastReconstr Surg*. 2007;120(7 Suppl 2):64S-75S.
13. Chang CS, BergeronL , ChenPK . Diced cartilage rhinoplasty technique for cleft lip patients. *Cleft Palate Craniofac J*. 2011;48(6):663-669.
14. Ma JG, WangKM, Zhao XH, Cai L, Li X. Diced costal cartilage for augmentation rhinoplasty. *Chin Med J (Engl)*. 2015;128(19):267-2681.
15. EnozM. Asymmetric hump resection and unilateral low to high osteotomy: surgical technique for treatment of a deviated nose. *J Otolaryngol Head Neck Surg*. 2008;373):E93-E95.

SP-25

Outcome of Neck of Femur Fracture

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Abstract: The mechanisms of injury leading to neck of femur fractures, and the risk patient populations. Gives the diagnostic approach for assessment of a patient presenting with femoral neck fractures, including any indication in imaging and differentials. Outline process of treatment options for neck of femur fractures. Hip fractures are joint damages. Femoral neck fractures are a definite type of intra capsular hip fracture. Hip fractures are common injury, especially seen in elderly in the emergency settings. It is also seen in young people who perform in gaming and high-energy trauma. Diagnosis and management are required immediately to prevent complications. Neck of femur fractures are a specific type of hip fracture. The neck of femur connects the femoral shaft with femoral head. The hip joint is the articulation of the head of femur with the acetabulum. This location makes the femoral neck to fracture. The blood supply of the femoral head is to be considered in displaced fracture as it runs along the neck of femur. Neck of femur fractures are associated with low energy falls in the elderly and high energy falls in the adult population. In younger patients sustaining a neck of femur fracture, the cause is usually high-energy trauma such as a substantial height or road traffic accidents. Risk factors for neck of femur fractures include females, decreased mobility, and bone density decreased. The femoral neck links the femoral shaft with the femoral head. This study evaluation, outcome of femoral neck fractures and reviews the role of the inter professional team in evaluating, diagnosing, and managing the condition. Neck of femur fracture treated with modular bipolar has significantly shown good results.

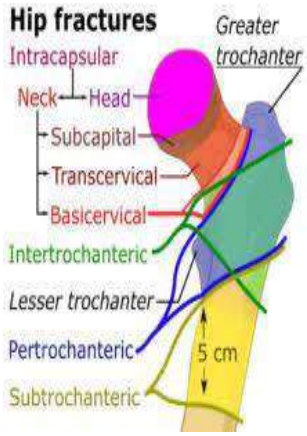
Keywords: Neck of Femur, Fracture, bipolar hemiarthroplasty.

INTRODUCTION

Neck of femur fracture is mostly seen in old age people due to low trauma at home like slip and fall and sustained injury to the hip. Neck of femur fractures are a specific type of hip fracture.[1-3] The neck of femur connects the femoral shaft with femoral head. The hip joint is the articulation of the head of femur with the acetabulum. This location makes the femoral neck to fracture. The blood supply of the femoral head is to be considered in displaced fracture as it runs along the neck of femur.[4-6] Neck of femur fractures is associated with low energy falls in the elderly and high energy falls in the adult population. In younger patients sustaining a neck of femur fracture, the cause is usually high-energy trauma such as a substantial height or road traffic accidents. Risk factors for neck of femur fractures include females, decreased mobility, and bone density decreased.[7] 60% of all neck of femur fracture is been seen in age group between 50-80years and is due to high or low trauma. In developing modalities neck of femur fractures are treated well with bipolar or modular bipolar hemiarthroplasty or total hip arthroplasty.[8-10] In some areas like farmers who wants to squat and work Girdle stone arthroplasty is also been done for the neck of femur fracture.[11]

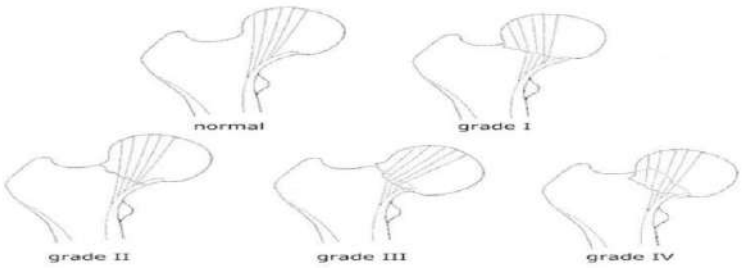
METHODS

There are 10 patients with an age between 60 to 70 years. Fractures are classified based on Anatomical, Garden and Pauwels classification. Out of these 3 cases where of Anatomical subcapital, Gardens – Type 2 and Pauwels – Type 1. Other cases had different types which is based on classifications below based on Anatomical may be subcapital, transcervical or basicervical.



ANATOMICAL CLASSIFICATION- SUBCAPITAL, TRANSCERVICAL AND BASICERVICAL.

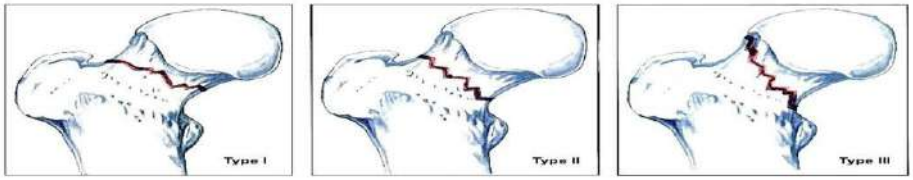
1. Garden Classification (based on AP radiographs and does not consider lateral or sagittal plane alignment)	
Type I	Incomplete, valgus impacted
Type II	Complete fracture. nondisplaced
Type III	Complete, displaced < 50%
Type IV	Complete, displaced
Posterior roll-off and/or angulation of femoral head leads to increased reoperation rates	



The Garden Classification

- Type I: Incomplete fracture - valgus impacted-non displaced
- Type II: Complete fracture - nondisplaced
- Type III: Complete fracture - partial displaced
- Type IV: Complete fracture - fully displaced

3. Pauwels Classification (based on vertical orientation of fracture line)	
Type I	< 30 deg from horizontal
Type II	30 to 50 deg from horizontal
Type III	> 50 deg from horizontal (most unstable with highest risk of nonunion and AVN)



- Type I less than 30 degrees
- Type II 30 to 50 degrees
- Type III greater than 50 degrees

Treatment

The treatment for most of the Neck of Femur fracture is to give the patient painless mobilization and make the patient to walk. Young patients with Neck of femur fractures will require treatment emergency operation. Vertically oriented fractures such a Pauwel type3 fractures are common in younger and high-energy road traffic trauma patients. A DHS is biomechanically more stable for these fracture patterns. With displaced fractures in younger patients, the goal is to achieve anatomic reduction through emergent open-reduction internal fixation with modular bipolar hemiarthroplasty has shown good and satisfactory results. Non-displaced fractures of neck of femur are treated with percutaneous cannulated screws or a DHS. However, there is a higher rate of (AVN) with the use of a DHS compared to cannulated screws.The most preferred treatment according to age based on old age is total hip replacement. Modular Bipolar Hemiarthroplasty is also now done which provides early range of movements and prevents protusioacetabuli also. Whatever may be the treatment the patient is mobilized following a series of process after the surgery.

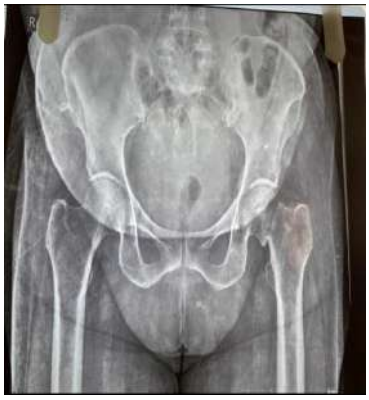


Fig 1: AP radiograph of pelvis with both hips pre operative xray shows left neck of femur fracture.

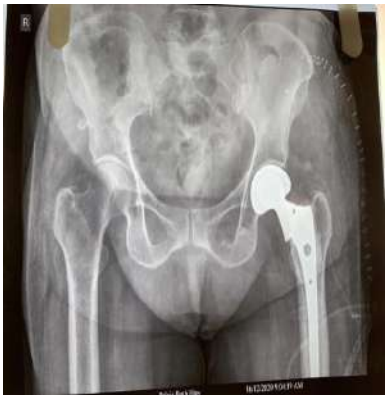
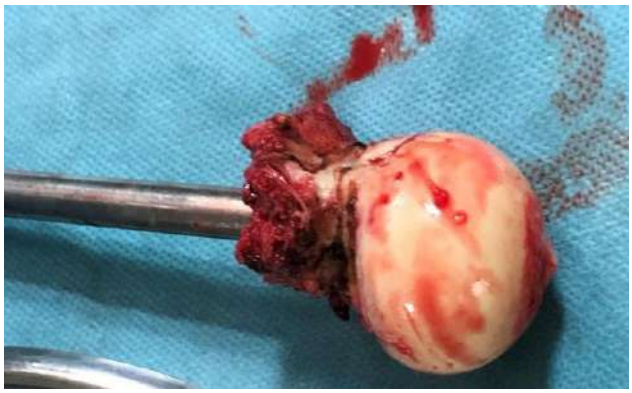
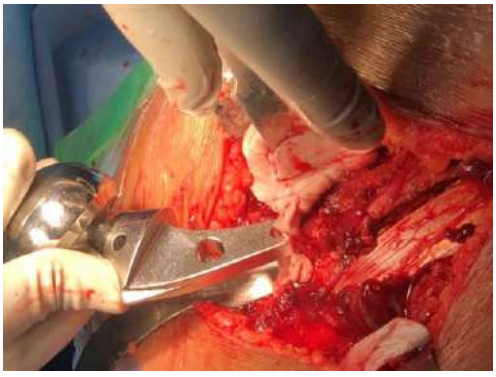


Fig: 2: AP radiograph of the same patient follow up with modular bipolar hemiarthroplasty done for the patient.

INTRA OP IMAGES



This is the intra oppic of the femoral head removed.



This is the intra operative image of the implant placement modular bipolar into the femur.

DISCUSSION

Treatment of Neck of Femur fracture is very important or else patient can't mobilize and patient won't be able to walk. This study is done in such a way that all the patients treated well with the treatment of choice and patient where symptomatically improved and all patients x-ray showed signs of union. The hip joint is the articulation of the head of femur with the acetabulum.[12] This location makes the femoral neck to fracture. The blood supply of the femoral head is to be considered in displaced fracture as it runs along the neck of femur. Neck of femur fractures is associated with low energy falls in the elderly and high energy falls in the adult population. In younger patients sustaining a neck of femur fracture, the cause is usually high-energy trauma such as a substantial height or road traffic accidents. Risk factors for neck of femur fractures include females, decreased mobility, and bone density decreased. After this patient mobilized well and started doing their routine activities.[13] Bone density at various sites for prediction of hip fractures.

CONCLUSION

Femoral neck fractures in adults are uncommon. Neck of femur fractures are associated with low energy falls in the elderly and high energy falls in the adult population. In younger patients sustaining a neck of femur fracture, the cause is usually high-energy trauma such as a substantial height or road traffic accidents. Risk factors for neck of femur fractures include females, decreased mobility, and bone density decreased. They usually occur as a result of high-energy trauma and patients often have associated injuries. However, there are numerous issues under the physician's control that can minimize and prevent these complications. The key features in giving femoral neck fractures should include early diagnosis, early surgery, anatomic reduction, capsular decompression and stable internal fixation. However neck of femur fracture fixation using modular bipolar hemiarthroplasty is the best modality of choice and patient improved well.

CONFLICT OF INTEREST

Conflict of interest declared none.

REFERENCES

1. Crist BD, Eastman J, Lee MA, Ferguson TA, Finkemeier CG. Femoral Neck Fractures in Young Patients. Instr Course Lect. 2018 Feb 15;67:37-49.
2. Azar FM, Canale ST, Beaty JH. Campbell's Operative Orthopaedics, E-Book. Elsevier Health Sciences; 2020 Dec 23.
3. Cummings SR, Black DM, Nevitt MC, Browner W, Cauley J, Ensrud K, Genant HK, Palermo L, Scott J, Vogt TM. Bone density at various sites for prediction of hip fractures. The Study of Osteoporotic Fractures Research Group. Lancet. 1993 Jan 09;341(8837):72-5.
4. Protzman RR, Burkhalter WE. Femoral-neck fractures in young adults. J Bone Joint Surg Am. 1976 Jul;58(5):689-90
5. Johnell O, Kanis JA. An estimate of the worldwide prevalence and disability associated with osteoporotic fractures. Osteoporos Int. 2006 Dec;17(12):1726-33.
6. Lakstein D, Hendel D, Haimovich Y, Feldbrin Z. Changes in the pattern of fractures of the hip in patients 60 years of age and older between 2001 and 2010: A radiological review. Bone Joint J. 2013 Sep;95-B(9):1250-
7. Koval KJ, Zuckerman JD. Hip Fractures: I. Overview and Evaluation and Treatment of Femoral-Neck Fractures. J Am Acad Orthop Surg. 1994 May;2(3):141-149.
8. Barney J, Piuzzi NS, Akhondi H. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Jul 6, 2021. Femoral Head Avascular Necrosis.
9. Li M, Cole PA. Anatomical considerations in adult femoral neck fractures: how anatomy influences the treatment issues? Injury. 2015 Mar;46(3):453-8.
10. Dedrick DK, Mackenzie JR, Burney RE. Complications of femoral neck fracture in young adults. J Trauma. 1986 Oct;26(10):932-7
11. Kazley JM, Banerjee S, Abousayed MM, Rosenbaum AJ. Classifications in Brief: Garden Classification of Femoral Neck Fractures. Clin Orthop Relat Res. 2018 Feb;476(2):441-445.
12. Bhandari M, Devereaux PJ, Swiontkowski MF, Tornetta P, Obremskey W, Koval KJ, Nork S, Sprague S, Schemitsch EH, Guyatt GH. Internal fixation compared with arthroplasty for displaced fractures of the femoral neck. A meta-analysis. J Bone Joint Surg Am. 2003 Sep;85(9):1673-81.
13. Fixation using Alternative Implants for the Treatment of Hip fractures (FAITH) Investigators. Fracture fixation in the operative management of hip fractures (FAITH): an international, multicentre, randomised controlled trial. Lancet. 2017 Apr 15;389(10078):1519-1527.

SP-26

Radiological Based Study of Inclination of The Acetabular Cup After Total Hip Replacement and its Correlation With the Functional Outcome

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Abstract : To correlate the Inclination of the acetabular cup after Total hip replacement and its correlation. A prospective study of 46 adults hips without acetabular defects, treated with first time Total hip arthroplasty between july 2018 to November 2020. We aimed to find the inclination of the acetabular cup using a CT-scan post-operatively and to correlate it with the functional outcome. The harris hip score was used to evaluate the functional outcome of patients. 90% of our subjects had no limb length discrepancies. A majority of 35 cases had a range-of-motion in the 211-300^0 category. The others were in the range of 161-210^0.None of our patients had any flexion contracture. However, 97.8% (45 patients) of our study population had excellent functional outcomes. The CT scan gives an accurate measurement of the acetabular implant position and shows a good outcome of our free hand intraoperative acetabular implant positioning.

Keywords: Acetabular cup , Inclination , total hip replacement , harris hip score , CT scan.

INTRODUCTION

Total hip arthroplasty (THA) is a procedure where both, the femoral head and acetabulum of the diseased hip joint is replaced by prosthetic components. It can be done for severe hip osteoarthritis (OA), inflammatory arthritis, avascular necrosis of femoral head, acute femoral neck fracture in the active elderly, failed hip surgeries and hip dysplasia. Various studies suggest that factors such as the acetabular implant's version, inclination, depth, the femoral implant's version, the neck shaft angle, surgical approach and patient factors affect the outcome of the surgery. Post operatively the patient may have complications such as pain, deformity, increased dislocation rates, decreased range of movements, increased wear of the implants. Among the various factors that affect the outcome, the acetabular cup position, in both the sagittal and coronal plane, is quite principal. The normal acetabulum abduction angle ranges between 48-67^0. The recommended inclination after total hip arthroplasties is 30-50^0. Intra-operatively the cup is tried to be placed within the recommended ranges of inclination. A post-operative assessment of the cup position will also judge the intra-operative free-hand positioning of the component.

MATERIALS AND METHODS

Operatively, the inclination is calculated by the angle between the inserter handle (acetabular axis) and the floor. Radiographically, acetabular inclination is the angle between the longitudinal axis and acetabular axis through an anteroposterior view.[1] Measurements through a CT scan have a higher reliability and accuracy compared to other methods of measurements. We measured the inclination on a coronal section of the CT scan. Toevaluate the outcomes of Total hip arthroplasty, various functional scores are available of which the Charnley modification and the Harris Hip Score(HHS) are most commonly used.[2] The HHS is a clinician-based scoring tool. It doesn't require other special training or time. It has a high reliability and validity, being tested against the SF-36 and the WOMAC scoring systems. It includes pain, deformity, function and range of movement of the patient. The score outcome is graded as poor if <70, fair if 70-79, good if 80-89 and excellent if 90-100[3]. The initial study population included 62 patients. Of these patients, 13 patients did not comply with our inclusion criteria and were hence excluded. From the remaining, 1 patient failed to come for further follow up, 2 patients passed away due to unrelated causes.[3] Hence, the final study population was 46hips.We included all adult patients after physeal closure where first time total hip replacement has been done. Patients with acetabular defects, congenital or acquired were excluded. All the cases were performed by the same primary surgeon, using the posterior approach to the hip. The patients were called over for regular follow-up visits up for a minimum of 6 months, up to 2 years. CT scans were taken when the patients came for follow-up. The CT scan DICOM file was saved and used to assess the acetabular cup placement angle. During follow-ups, clinical assessment of the patients for their functional outcome was done based on the HHS[4]. The data was collected for all parameters used by the HHS during the clinical examination at the last follow-up date of each patient. The final HHS was calculated by adding the individual score. The CT scan of the hip was taken and taking coronal cuts, inclination of the acetabular component was calculated for each patient. Acceptable values were taken as 30-55^0.To determines the inclination; we first defined the coronal plane, referencing it from the posterior columns on an axial section. The plane was translated to the acetabular cup axis. The inclination was determined by measuring the angle between the apex of the cup apex and a line through the inferior part of the tear drops.[5] The patients were scored on each follow-up visit. The final score taken on the last clinical visit was used. Each patient was interviewed based on the parameters of the questionnaire. The data was compiled accordingly and summed up to get the numerical value of the hhs. This was later graded into excellent, good, fair and poor.

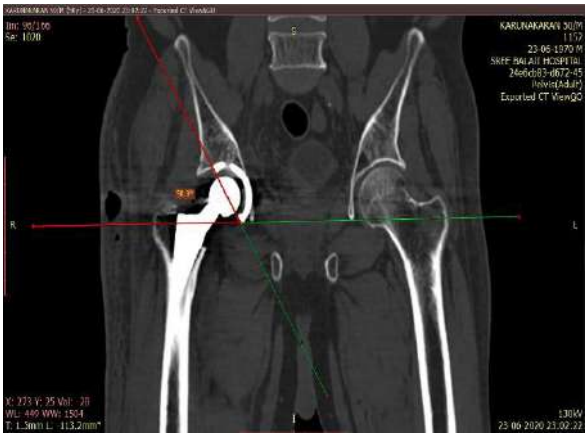

Fig. I

RESULTS AND ANALYSIS

Table .I Limb length discrepancies among 46 patients.	
Limb Length discrepancies	No. Of Cases
Nil	41
<3.2 cm	5
>3.2 cm	0

90% of our subjects had no limb length discrepancies. None of our other patients had any significant discrepancies. None of our patients had any flexion contractures, fixed abduction or fixed internal rotation. A majority of 35 cases had a range-of-motion in the 211-300^0 category. The others were in the range of 161-210^0.None of our patients had any flexion contracture.

Table 2 : Harris hip score among 46 patients.	
Harris Hip Score	No. Of Cases
Excellent	45
Good	I
Fair	0
Poor	0

Harris hip score are used for the assessment of the results of hip surgery and is intended to evaluate various hip disabilities. There are ten items covering four domains. The domains are pain, function, absence of deformity and range of motion. The HHS is a measure of dysfunction so the higher the score, the better the outcome for the individual. Almost all our patients had an excellent score on the Harris Hip ScoreGrading system. Only one patient graded as good.

Table 3 : Inclination among 46 patients.	
Inclination	No. Of Cases
20.1-25	I
25.1-30	I
30.1-35	9
35.1-40	8
40.1-45	13
45.1-50	3
50.1-55	7
55.1-60	2
60.1-65	2

Inclination is the angle between the longitudinal axis of the patient and the acetabular axis as projected onto the coronal plane. Acetabular inclination was calculated by drawing a line tangential to the face of the acetabular cup on the AP scout image and calculating the angle relative to a line drawn between the ischialtuberosities. The majority of the cup were placed between 30-45^0 of inclination. Only 4 cases were in an increased abduction angle.

DISCUSSION

Determining the optimal placement angle for the acetabular component in THA can be challenging. The placement depends on the version, inclination, depth and height. The aim is to achieve stability and normal range of movement. When the implants range of movement matches the native hip movement, impingement will be absent. Version of the cup is the implant orientation in the sagittal plane and inclination is the orientation in the coronal plane. A more horizontally inclined cup decreases the range

of movements, whereas a more vertically inclined cup leads to higher edge loading causing increased wear of the implants. Harris et al recommended an inclination of 30°. Harkesssuggested an inclination of 45. McCollum et al determined the safest range for cup placement was 30°-50° of abduction.[5]Lewinnek et al recommended the safe zone as between an inclination of 40°(±10°).[6] In our study the average acetabular cup inclination was found to be 41.9^0. D DD'lima et al concluded from their study that the hip had maximum stability and range of movements when the acetabular abduction angle was between 45^0-55^0.[7] Pedersen showed that a placement of less than 40° abduction and less than 10° anteversion achieves optimal range-of-motion.[8] Following our study, 4 patients had excessive inclination, the range of movements were decreased on the operated hip. Biedermann et al showed that slight variations in cup positioning can increase rates of dislocation.[9] Ali Khan et al showed that in patients who suffered dislocations, the cup position was either too anteverted(>$15.^0$) or too vertical(>50^0).[10] One of our cases in whom the cup had a higher inclination, dislocated his hip after a low impact fall. Once relocated, no further episodes of dislocation were seen. To avoid impingement, it has been recommended to keep the acetabular cup inclined at 40°-50°. However, McCarthy et al concluded that impingement occurrence varied in individuals and certain activities reduces the safe zone.[11-13] Although hip dislocations may occur sans impingement, it is the most often cause. An optimal acetabular component position is crucial in providing an impingement free range of motion, preventing dislocation and providing stability Although controversial, from a clinical view point we consider the safe zone as between 30-55° of inclination. Many of these studies don't take into account the femoral anteversion, neck-shaft relation, restoration of hip biomechanics, surgical approaches among other variables. These have been calculated in theoretical mathematical models alone.[14-16] We evaluated all our patients using the HHS. The HHS is an easy clinician-based tool to assess the pain, deformity, function and activity levels of a patient after THA. It has a high validity and reliability. The hip score is graded as poor (<70), fair (70-79), good (80-89), excellent (90-100). The scoring system requires no special training and can be completed quickly using minimal equipment. Almost all of our patients had an excellent functional outcome. None had a poor outcome. Intra-operatively, the acetabular component was placed using a pure manual free hand technique.[17] The cups were aimed to be placed at an angle within the suggested range of cup inclination. 90% of the cups were in the acceptable ranges of inclination. Our study was limited by the number of cases and the duration of study. A longer and larger study could potentially reveal other complications of THA, leading to a better understanding of the effect of the acetabular inclination on the clinical and functional outcome. Using more than one scoring system may help to get a better assessment of the functional outcome.

CONCLUSION

There were 46 patients who complied with our study. Each patient underwent unilateral THA for the first time and did not have any acetabular defects. Most of our patients underwent THA for Osteoarthritis of the hip.97.8% of our patients had an excellent functional outcome; the rest was a good outcome when graded by the HHS. About 90% of the acetabular implants were between $30\text{-}55^0$ of inclination. Among the rest, 1 case had a single episode of dislocation, which was relocated under general anaesthesia in our hospital. No further episodes of dislocation occurred. Few cases had a reduced range of movement. However, the functional outcomes of all these cases were excellent. The CT scan gives an accurate measurement of the acetabular implant position and shows a good outcome of our free hand intraoperative acetabular implant positioning. Perhaps better accuracy may be achieved using a computer-navigated system.

CONFLICT OF INTEREST

Conflict of interest declared none.

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REFERENCES

1. Learmonth ID, Young C, Rorabeck C. The operation of the century: Total hip arthroplasty. Lancet. 2007;390:1508-19.
2. Banaszkiewicz PA. Traumatic arthritis of the hip after dislocation and acetabular fractures: treatment by mold arthroplasty: an end-result study using a new method of result evaluation. InClassic Papers in Orthopaedics 2014 (pp. 13-17). Springer, London.
3. Harkess WJ. Arthroplasty of the hip: dislocation and subluxation. Campbell's operative orthopaedics. Eighth ed. St. Louis: Mosby. 1992;54.
4. McCollum DE, Gray WJ. Dislocation after total hip arthroplasty. Causes and prevention. Clinical orthopaedics and related research. 1990 Dec 1(261):159-70.
5. Lewinnek GE, Lewis JL, Tarr RI, Compere CL, Zimmerman JR. Dislocations after total hip-replacement arthroplasties. J Bone Joint Surg Am. 1978 Mar 1;60(2):217-20.
6. D'lima DD, Urquhart AG, Buehler KO, Walker RH, COLWELL CW. The effect of the orientation of the acetabular and femoral components on the range of motion of the hip at different head-neck ratios. JBJS. 2000 Mar 1;82(3):315-21.
7. Pedersen DR, Callaghan JJ, Brown TD. Activity-dependence of the "safe zone" for impingement versus dislocation avoidance. Medical engineering & physics. 2005 May 1;27(4):323-8.
8. Biedermann R, Tonin A, Krismer M, Rachbauer F, Eibl G, Stöckl B. Reducing the risk of dislocation after total hip arthroplasty: the effect of orientation of the acetabular component. The Journal of bone and joint surgery. British volume. 2005 Jun;87(6):762-9.

9. Murray DW. The definition and measurement of acetabular orientation. The Journal of bone and joint surgery. British volume.1993 Mar;75(2):228-32.
10. Ali Khan MA, Brakenbury PH, Reynolds IS. Dislocation following total hip replacement. The Journal of bone and joint surgery. British volume. 1981 May;63(2):214-8.
11. McCarthy TF, Alipit V, Nevelos J, Elmallah RK, Mont MA. Acetabular cup anteversion and inclination in hip range of motion to impingement. The Journal of arthroplasty. 2016 Sep 1;31(9):264-8
12. Patil S, Bergula A, Chen PC, Colwell CW, D'Lima DD. Polyethylene wear and acetabular component Orientation. J bone joint surg [Am].2003; 85-A:56-63.
13. Little NJ, Bush CA, Gallagher JA, Rorabeck CH, Bourne RB. Acetabular polyethylene wearaand acetabular inclination and femoral offset. Clin Orthop Relat Res.2009;467:2895-2900.
14. Bhaskar D, Rajpura A, Board T. Current concepts in acetabular positioning in total hip arthroplasty. Indian J Orthop. 2017; 51(4):386-96.
15. Scheerlinck T. Cup positioning in total hip arthroplasty. Acta Orthop Belg. 2014; 80(3):336-47.
16. Brooks P. Dislocation following total hip replacement: causes and cures. Bone joint J. 2013.
17. Beverland DE, ONeill CKJ, Rutherford M, et al. placement of the acetabular component. The bone and joint journal. 2016; 98-B.

SP-27

Acute Mercury Poisoning Presenting As Interstitial Pneumonitis

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Abstract: Mercury poisoning can result from inhalation of mercury vapors or ingestion of mercury salts. This case study shows how important suspicion of mercury poisoning is, its complex presentations and importance of timely intervention and management. We report a case of a19 year old female student who presentedwith chiefcomplaints of cough, breathlessness, anxiety, palpitations for 10 days. Chest radiograph revealed high density scattered radio- opacity over both lung fields. A Computed Tomography scan of thorax with abdomen was suggestive of high density opacity along entire broncho-vascular bundle with similar opacity over liver, pancreas, bowel and kidney. Her skeletal survey showed similar high density opacity in relation to elbow joint. Arterial Blood Gas analysis showed metabolic acidosis. Serum and Urinary mercury levels were more than 250 micrograms. Patient was treated with D- penicillamine, corticosteroids and kept under ICU monitoring. Follow up chest x-ray showed significant improvement. Cause of mercury poisoning was found to be homicidal. Conclusion – Mercury poisoning has myriad presentations so, high clinical suspicion is required. Any high density opacity radiologically should raise suspicion of heavy metal poisoning. Source of poisoning must be identified if not case must be medico-legalized to rule out homicide.

Keywords: Mercury, Poisoning, Pneumonitis.

INTRODUCTION

Mercury is the only metal that is liquid at room temperature and is highly soluble in blood and plasma.[1,2] Mercury exists in 3 forms: (1) elemental mercury, (2) inorganic -mercury vapor and mercurous or mercuric salts , and (3) organic – methyl mercury.[3] Inhalation of elemental mercury vapor can result in quick absorption through mucous membranes and lungs and gets rapidly oxidized to other forms.[4] Methyl mercury is easily absorbed through the gut and deposits in many tissues, but does not cross the blood-brain barrier as efficiently as elemental mercury; however, on entering the brain it is progressively demethylated to elemental mercury.[5] The target organ for inhaled mercury vapor is primarily the brain.[5] Mercurous and mercuric salts primarily damage the gut lining and kidney, while methyl mercury is widely distributed throughout the body.[6,7] Two forms of poisoning exists acute and chronic of whichacute form is mostly inhalational and manifestsas pulmonary edema and ARDS.[5]Large acute exposures to elemental mercury vapor induce severe pneumonitis which can be fatal.[8]Mercury can be transformed by bacteria into methylmercury which then bioaccumulates in aquatic life and ingested by humans.[8]Factors which determine health effects and their severity in mercury poisoning includes type of mercury, dose, age or developmental stage of the person exposed (fetus is most susceptible),duration of exposure and route of exposure (inhalation, ingestion or dermal contact).[7] High amount of ingested mercury manifest as vomiting with mucus and blood, abdominal pain, painful passage of blood and mucus in stool.[8,9] Renal involvement is also common which presents with oliguria, albuminuria and hematuria.[10] Central nervous system toxicity causes ataxia, speech impairment, visual field constriction, sensory disturbance, deafness, blindness, tremors, involuntary movements, mental retardation and coma. Here we present a case of a young female presenting with mercury poisoning.

CASE REPORT

A 19 year old female presented with cough and breathlessnesssince5 days.She was treated by a private practitioner with antibiotics but showed no improvement. She was referred to our hospital for further evaluation and management. Patient had no history of hemoptysis, chest pain, loss of weight, loss of appetite or orthopnea. Her past history was not significant, her personal history shows she is an on smoker, has no exposure to smoke, dust, fumes and takes mixed diet, her bowel and bladder habits are normal. On examination, she was pale. Her vitals were normal except she had tachycardia.On Auscultation,normal vesicular breath sounds with bilateral fixed monophonic rhonchi. Chest x-ray showed multiple high density scattered opacities involving both lung fields with similar opacities over abdomen (Figures 1,2). CT thorax with abdomen showed multiple scattered dense radio opacities in both lung fields as well as both kidneys, visualized bowel loops, liver parenchyma, muscular plane and in relation to spine Figures (3,4,5,6,7). Plain X- ray abdomen standing shows whole pelvic and abdomen spilling of high density opacities Figure (8). X- ray of elbow joint shows similar opacity Figures (9,10). So, high density radiopacity scattered all over the body with ABG suggestive of metabolic acidosis with no other signs of infection prompting a suspicion of heavy metal poisoning with mercury as prime culprit.Serum mercury was more than 250 micrograms and Urine mercury was more than 250 micrograms. A diagnosis of acute mercury poisoning was confirmed. Patient was treated by chelation therapy with D – penicillamine 250 mg TDS for 3 days. Patient was closely monitored in Intensive Respiratory Care Unit to watch for signs of ARDS.Injection Methylprednisolone was started simultaneously.Patient gradually improved and her chest x-ray after 15 days showed significant improvement.Patient was discharged giving symptomatic coverage and follow-up was done after 2 weeks and repeat X – Ray showed significant improvement Figure (11). Source of mercury poisoning was found to be homicidal and was considered a medicolegal case.

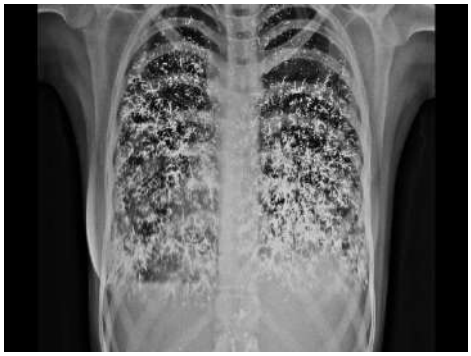

Fig 1 – shows chest x- ray multiple high density scattered opacities involving both lung fields and abdomen.

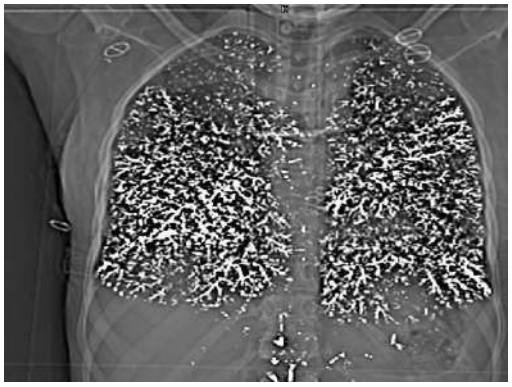

Fig 2 - shows chest x- ray multiple high density scattered opacities involving both lung fields and abdomen.

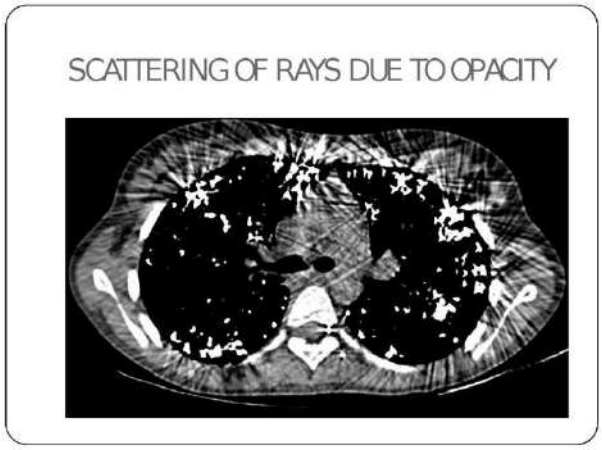

Figure 3 – shows CT thorax with multiple scattered dense radio opacities in both lung fields.

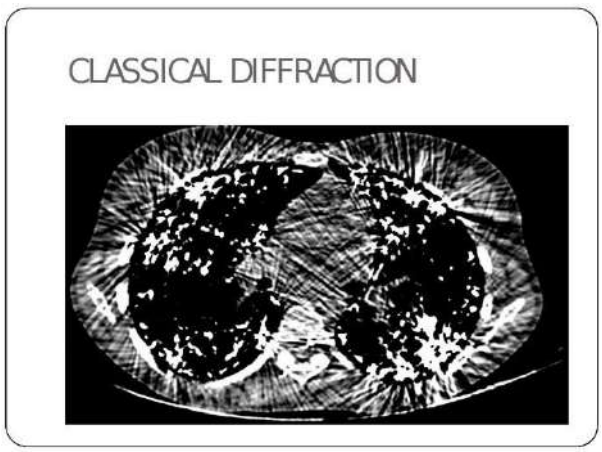

Figure 4- shows classical diffraction in CT thorax.

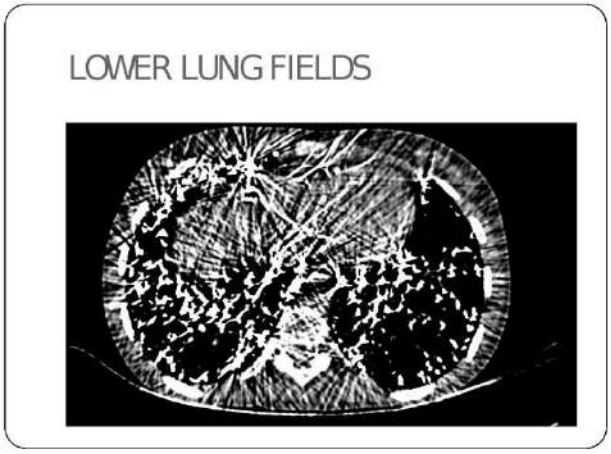

Figure 5- shows multiple dense radio opacities in bilateral lung fields.

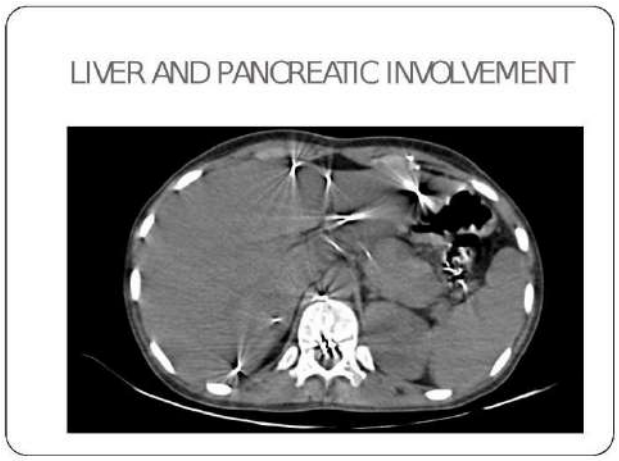

Figure 6- shows CT abdomen with dense radio opacities in liver and pancreas.

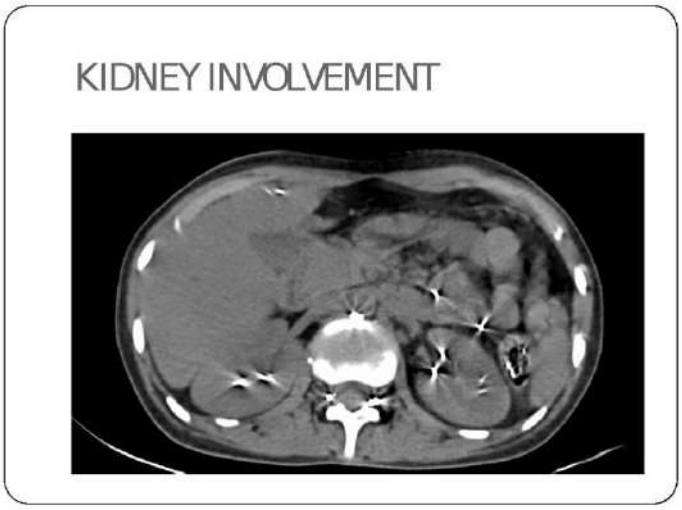

Figure 7 – shows a CT abdomen with dense radio opacities in both kidneys.

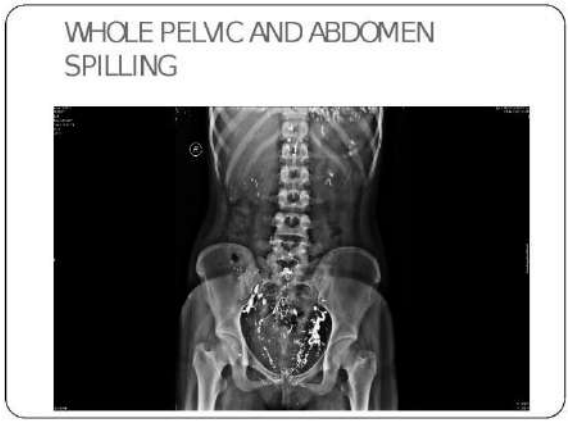

Figure 8 – shows involvement of whole pelvic and abdomen.

Figure 9 – X Ray arm and forearm showing radio opacities.

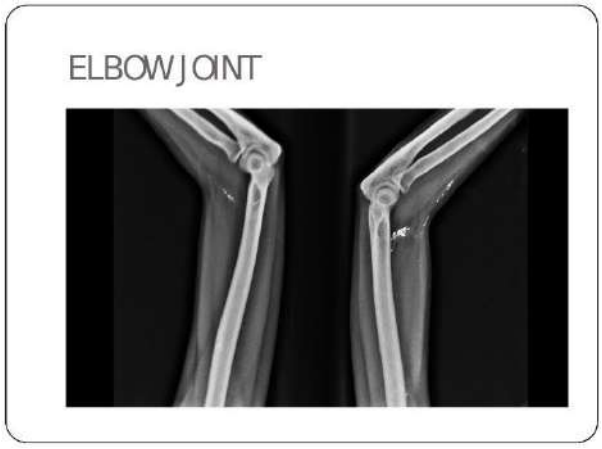

Figure 10 – shows X Ray elbow joint showing dense radio opacities.

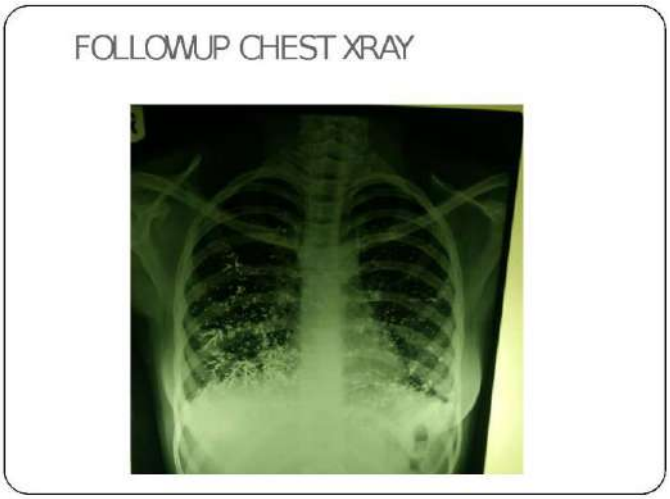

Figure 11 – Chest X Ray on follow up shows reduction in opacities and significant improvement.

DISCUSSION

The first documented outbreak of acute methyl mercury poisoning by consumption of contaminated fish occurred in Minamata, Japan, in 1953 and named Minamata disease. Here we seea case of mercury poisoning of homicidal nature causing interstitial pneumonitis.[11] Mercury is a highly toxic heavy metal as it gets readily accumulated in aquatic organisms, the most toxic form being methyl mercury which is a neurotoxin.[12] Exposure to mercury can occur via ingestion of contaminated food(fish), dentalprocedures (amalgams),occupational exposure (e.g. mining) and use of mercury based instruments like thermometers, sphygmomanometer).[13] Elemental mercury vapor has rapid absorption across the alveolar membrane and transported via blood to brain and nervous system.[13] Mercury is converted to mercuric ions rapidly which then gets excreted in the urine and feces. Half-life of mercury is about 60 days.[14] CNS involvement causes tremors, impaired gait and rigidity suggesting basal ganglia and cerebellar involvement. A memory defect suggests temporal lobe involvement.[15] Pulmonary manifestations include interstitial pneumonitis, bilateral infiltrates, non cardiogenic pulmonary edema and acute respiratory distress.[16] Urinary excretion of 300mg/L suggests mercury poisoning and in this case was more than 250 micrograms. 1) Initial phase (few days post exposure) is manifested as a flu-like illness with fever, myalgia, chills, dry mouth and headache. Toxic pneumonitis can present with respiratory failure which can complicate the case. 2) Intermediate phase (symptoms presenting 2 weeks post exposure) can cause severe multiorgan symptoms (central nervous system, respiratory tract, gastrointestinal and renal system). Mercury vapor inhalation is a direct airway irritant, a cellular poison and death can result due to respiratory failure. 3) Late phase involves resolution of other symptoms and persistence of nervous system symptoms[17] Penicillamine is an effective chelating agent for mercury poisoning and has added benefit of oral administration and more potent than Dimercaptopropanol (BAL).[18] D-penicillamine is also an oral compound which is useful in less severe mercury poisoning cases. In our case, patient was initially treated with antibiotics but her symptoms persisted. Chelation therapy with D-penicillamine for 3 days and simultaneous treatment with methylprednisolone improved the patient's condition.

CONCLUSION

Mercury poisoning has various clinical presentations and requires a high index of suspicion for diagnosis. Close monitoring for signs of ARDS is important .This case report shows timely intervention and management in an Intensive care setup improved patient's outcome.

REFERENCES

1. Pillay VV. Modern medical toxicology. Jaypeebrothers medical publishers (P) Ltd; 2013.
2. City, Minamata. "Minamata disease: the history and lessons." (2000).
3. Bakir, F., Damluji, S. F., Amin-Zaki, L., Murtadha, M., Khalidi, A., Al-Rawi, N. Y., ... & Doherty, R. A. (1973). Methylmercury poisoning in Iraq. Science, 181(4096), 230-241.
4. Skerfving, S. B., &Copplestone, J. F. (1976). Poisoning caused by the consumption of organomercury-dressed seed in Iraq. Bulletin of the World Health Organization, 54(1), 101.
5. Berlin, M. "Handbook of the Toxicology of Metals, V. 2." (1985): 376-405.
6. Textbook of Forensic Medicine and Toxicology, Nageshkumar G Rao;466- 467
7. Fishman's Pulmonary Diseases and Disorders,Michael A. Grippi, MD,JackA.Elias, MD,Jay A. Fishman, MD,Robert M. Kotloff, MD,Allan I. Pack, MBChB, PhD,Robert M. Senior, MD; 1377
8. https://www.who.int/news-room/fact-sheets/detail/mercury-and-health
9. Biswas G. Review of forensic medicine and toxicology. JP Medical Ltd; 2012 Jul 20.
10. Gochfeld, Michael. "Cases of mercury exposure, bioavailability, and absorption." Ecotoxicology and environmental safety 56.1 (2003): 174-179.

Shigeo Ekino, Mari Susa, Tadashi Ninomiya, Keiko Imamura, Toshinori Kitamura,Minamata disease revisited: An update on the acute and chronic manifestations of methyl mercury poisoning,Journal of the Neurological Sciences,Volume 262, Issues 1–2,2007,Pages 131-144.

11. Rafati-Rahimzadeh M, Rafati-Rahimzadeh M, Kazemi S, Moghadamnia AA. Current approaches of the management of mercury poisoning: need of the hour. DARU Journal of Pharmaceutical Sciences. 2014 Dec;22(1):1-0.
12. Ellenhorn MJ, Barceloun DG. Medical Toxicologydiagnosis and treatment of human poisoning.
13. Rowens, Bradley, et al. "Respiratory failure and death following acute inhalation of mercury vapor: a clinical and histologic perspective." Chest 99.1 (1991): 185-190.
14. Haddad, Lester M., Michael W. Shannon, and James F. Winchester, eds. Clinical management of poisoning and drug overdose. Philadelphia: Saunders, 1998.
15. Kanluen, S., and C. A. Gottlieb. "A clinical pathologic study of four adult cases of acute mercury inhalation toxicity." Archives of pathology & laboratory medicine 115.1 (1991): 56-60.
16. Jaeger, A., et al. "Accidental acute mercury vapor poisoning." Veterinary and human toxicology 21 (1979): 62-63.
17. Lien, D. C., et al. "Accidental inhalation of mercury vapour: respiratory and toxicologic consequences." Canadian Medical Association Journal 129.6 (1983): 591.

SP-28

The Use of Dienogest in The Treatment of Endometriotic Cyst: A Case Study and overview of Literature

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Abstract: The choice of treatment in endometriotic cyst is based on the clinician's discretion and on the preferred outcomes. The clinical decision on treatment is made based on the severity of the disease, age of the patient and the specific social requirement of the patient. The following case study describes a case of endometriotic cyst of the ovary that was presented in the gynaecology department of a tertiary care center and how it was managed. A36-year old female P1L1 presented on 31st of March, 2021 with chief complaints of right side lower abdominal pain and vomiting for four days. Pre-treatment ultrasound showed a large Solid/Cystic Right Tubo-Ovarian Mass which was further verified by MRI as endometriotic cyst. Patient was started on T.Dienogest 2mg/ day for two months and reviewed. After two months, ultrasound showed seedling fibroid with no evidence of endometriotic cyst. T.Dienogest 2 mg/day for two months produced significant results with a resolution of cystic lesions on ultrasound imaging. Considering the literature and the findings from this case study, it should be noted that all medical modalities must be tried before opting for surgery, especially when it is not associated with infertility especially in perimenopausal age group as with the onset of menopause there will be decrease in estrogen production. Dienogest promotes antiproliferative, immunologic and antiangiogenic effects on endometrial tissue thereby treating the main important side effect of new onset triple dysmenorrhoea

Key words: Endometriotic Cyst, Medical Management, Dienogest, Case Study, India

INTRODUCTION

The presence of endometrium-like tissue outside the uterus is defined as endometriosis.[1,2] It is estimated to affect around 10% of the women of reproductive age.[3] The origin of endometriosis is debatable though the common idea is that it is due to the backflow of menstrual bleeding.[4] Ovary is the most commonly affected organ while fallopian tubes, rectum, pelvic serosa, retroperitoneal structures and sometimes even lungs can get affected. [5,6] In the ovary, the typical presentation is in the form of a cyst containing old blood.[7] This is called as endometriotic cyst or chocolate cyst. This presentation is seen in around 17%-44% of the women who report endometriosis.[8] The major clinical impacts of endometriosis are pain, infertility and the potential for neoplastic transformation. The common presentation is 5 D's endometriosis-associated Deep seated pelvic pain, Dysmenorrhea ,Dyspaerunia, Dysuria and Dyschezia If inadequately treated, it progresses to Infertility. Neoplastic transformations have been reported recently.[9,10] Neoplasms are commonly seen in the elderly women who have crossed the child bearing age and in the post-menopausal age. Diagnosis of chocolate cyst of the ovary is challenging mainly during the initial days of presentation. However, MRI can be used to detect endometriotic cyst that has a characteristic appearance. Once the diagnosis is established, treatment consists of medical management, regular follow-up and surgical intervention if required. The choice of treatment is based on the clinician's discretion and on the preferred outcomes. For instance, medical management is effective in pain management with less impact on improvement in fertility.[11-13] The varying presentation along with the different age groups necessitates the need for specific treatment strategies case-by-case keeping in mind the preferred primary, secondary outcomes of the management.[14] The clinical decision on treatment is made based on the severity of the disease, age of the patient and the specific social requirement of the patient. Considering the low prevalence and lack of uniform reporting of the endometriotic cyst, individual case study approach is suitable to report cases of chocolate cysts of the ovary. The following section describes a case of endometriotic cyst of the ovary that was presented in the gynaecology department of a tertiary care center.

Post-treatment ultrasound findings

Findings 1

a) Uterus appears stretched and elongated measuring 9.8*5*3.8cm adherent to the anterior abdominal wall.
b) Endometrial thickness is 6mm
c) Multiple seedling fibroid largest measuring 1.2*0.7 cm is noted in the posterior wall of the uterus.

Impression
Seedling fibroid with no evidence of endometriotic cyst.
FINDINGS 2
MRI Findings
Presentation

Uterus appears stretched and elongated measures 10.6*5.1*4.1 cm. A few seedling fibroids noted in the uterus largest measuring 6.2*5.6 mm in the posterior myometrium. Multiple cysts in the elongated cervix largest measuring 7.0*6.5 mm. Endometrial

thickness measures 5 mm Right ovary not seen separately. Instead a large multiloculated T1 and T2 hyperintense cystic lesion with T2 hypointense components within showing a GRE blooming at some places measuring 12.3*9.8*8 cm noted in the right adnexa and POD. Part of the lesion appears hyperintense on T1 and shows T2 shading sign. The lesion appears to cause mass effect over uterus pushing the uterus anteriorly. Left ovary measures 3.1*2.8*2.1 cm. A follicle and tiny haemorrhagic cyst seen in left ovary

Findings

Uterus appears stretched and elongated- utero-parietal adhesion. Few seedling fibroids in the uterus. A large multiloculated T1 and T2 hyperintense cystic lesion with T2 hypointense components within showing a GRE blooming at some places, noted in right adnexa and POD. Part of the lesion appears hyperintense on T1 and shows T2 shading sign. Right ovary not seen separately – endometriotic cyst to be considered. Suggested HPE correlation

DISCUSSION

Ovarian endometriotic cysts are treated either medically or surgically though sometimes observation is also a treatment of choice. Medical management is through the administration of progestogens, androgens, oral contraceptive pills and GnRH agonists (Gonadotropin-releasing hormones). The treatment choice depends on the symptoms of the patient and the desired outcomes. In the presented case, the treatment mainly was targeted against endometriosis associated pain. The FINDINGS 1 and FINDINGS 2 clearly depicts the treatment efficacy with T.dienogest,since there was only a seedling fibroid in the USG report further imaging modalities were deffered. Dienogest is an oral progestin that has been approved for use in monotherapy for endometriosis in many countries. The scientific reason for using Dienogest in endometriosis is derived from the fact that it combines the advantages of progesterone derivatives and 19-nor progestin derivatives. Further, it has a high bioavailability and is completely absorbed. The shorter half-life of 10 hours ensures that there is no risk in repeated dosing as the single dose is completely excreted in urine within 24 hours. Preclinical studies show that Dienogest can moderately inhibit gonadotropin secretion thereby reducing the endogenous production of estradiol. Continuous administration of the drug leads to a hypoestrogenic, hypergestagenic local endocrine environment that causes decidualisation of endometrial tissue followed by atrophy of the endometriotic lesions.[15] The pelvic pain is not only due to ovarian chocolate cyst. The presence of concomitant inflammation or adhesions or extra ovarian endometriosis may augment and aggravate the pain.[16] For medical management, GnRH agonist is the effective first line of treatment.[17] Oral contraceptives, Danazol and Gestrinone have shown proven efficacy in treating chocolate cysts.[18] Studies show that Dienogest (a selective progestin) as an effective medication.[19] A recent systematic review showed that using Dienogest 2 mg/day gave better results than placebo in the reduction of pelvic pain and in the reduction of endometriotic lesions. Extended therapy also produced significant results with less serious side effects.[20]

CONCLUSION

In the present study, Dienogest produced similar effects in the patient. T.Dienogest 2 mg/day for three months alone produced significant results with a resolution of cystic lesions on ultrasound imaging. Considering the literature and the findings from this case study, it should be noted that medical modalities must be tried before opting for surgery, especially when it is not associated with Infertility. Medical management should especially be tried in perimenopausal age group as with the onset of menopause there will be fall in estrogen production. In general women are more prone to surgeries like caesarean section and sterilisation , hence all efforts must be made to detect the disease clinically or through minimally invasive procedures. Dienogest promotes antiproliferative, immunologic and antiangiogenic effects on endometrial tissue thereby treating the main important side effect of new onset triple dysmenorrhoea associated with endometriosis

REFERENCES

1. Giudice, L. C. (2010). Endometriosis. New England Journal of Medicine, 362(25), 2389-2398.
2. Farquhar, C. M. (2000). Endometriosis. Bmj, 320(7247), 1449-1452.
3. Eskenazi, B., & Warner, M. L. (1997). Epidemiology of endometriosis. Obstetrics and gynecology clinics of North America, 24(2), 235-258.
4. Okeke, T. C., Ikeako, L. C., & Ezenyeaku, C. C. T. (2011). Endometriosis. Nigerian Journal of Medicine, 20(2), 198-206.
5. Jubanyik, K. J., &Comite, F. (1997). Extrapelvic endometriosis. Obstetrics and gynecology clinics of North America, 24(2), 411-440.
6. Guerriero, S., Conway, F., Pascual, M. A., Graupera, B., Ajossa, S., Neri, M., ... &Alcazar, J. L. (2020). Ultrasonography and atypical sites of endometriosis. Diagnostics, 10(6), 345.
7. Martin, D. C., & BERRY, J. D. (1990). Histology of chocolate cysts. Journal of Gynecologic Surgery, 6(1), 43-46.
8. Christensen, J. T., Boldsen, J. L., & Westergaard, J. G. (2002). Functional ovarian cysts in premenopausal and gynecologically healthy women. Contraception, 66(3), 153-157.
9. Borgfeldt, C., &Andolf, E. (2004). Cancer risk after hospital discharge diagnosis of benign ovarian cysts and endometriosis. Acta obstetricia et gynecologica Scandinavica, 83(4), 395-400.
10. Greenlee, R. T., Kessel, B., Williams, C. R., Riley, T. L., Ragard, L. R., Hartge, P., ... & Reding, D. J. (2010). Prevalence, incidence, and natural history of simple ovarian cysts among women> 55 years old in a large cancer screening trial. American journal of obstetrics and gynecology, 202(4), 373-e1.
11. Adamson, G. D. (1990). Diagnosis and clinical presentation of endometriosis. American Journal of Obstetrics &Gynecology, 162(2), 568-569.

12. Vercellini, P., Viganò, P., Somigliana, E., & Fedele, L. (2014). Endometriosis: pathogenesis and treatment. Nature Reviews Endocrinology, 10(5), 261-275.
13. Olive, D. L., & Pritts, E. A. (2001). Treatment of endometriosis. New England Journal of Medicine, 345(4), 266-275.
14. Mandai, M., Suzuki, A., Matsumura, N., Baba, T., Yamaguchi, K., Hamanishi, J., ... & Konishi, I. (2012). Clinical management of ovarian endometriotic cyst (chocolate cyst): diagnosis, medical treatment, and minimally invasive surgery. Current Obstetrics and Gynecology Reports, 1(1), 16-24.
15. Schindler, A. E. (2011). Dienogest in long-term treatment of endometriosis. International journal of women's health, 3, 175.
16. Fauconnier, A., Fritel, X., &Chapron, C. (2009). Endometriosis and pelvic pain: epidemiological evidence of the relationship and implications. Gynecologie, obstetrique&fertilite, 37(1), 57-69.
17. Oezkan, S., & Arici, A. (2009). Advances in treatment options of endometriosis. Gynecologic and obstetric investigation, 67(2), 81-91.
18. Vercellini, P., Crosignani, P., Somigliana, E., Vigano, P., Frattaruolo, M. P., & Fedele, L. (2011). 'Waiting for Godot': a commonsense approach to the medical treatment of endometriosis. Human Reproduction, 26(1), 3-13.
19. Strowitzki, T., Marr, J., Gerlinger, C., Faustmann, T., & Seitz, C. (2010). Dienogest is as effective as leuprolide acetate in treating the painful symptoms of endometriosis: a 24-week, randomized, multicentre, open-label trial. Human Reproduction, 25(3), 633-641.
20. de Paula Andres, M., Lopes, L. A., Baracat, E. C., &Podgaec, S. (2015). Dienogest in the treatment of endometriosis: systematic review. Archives of gynecology and obstetrics, 292(3), 523-529.

SP-29

Ovarian Hyperstimulation Syndrome-A Case Report

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Abstract: Ovarian Hyperstimulation Syndrome (OHSS) is an uncommon, iatrogenic complication of ovarian stimulation by ART (assisted reproductive technology) and other infertility procedures. Following gonadotropin therapy, OHSS occurs usually several days after assisted ovulation or oocyte retrieval. There is an increase in the prevalence of OHSS following extensive use of assisted reproduction technology. This is a case report of Ovarian Hyperstimulation Syndrome diagnosed and managed promptly on time. We conclude that though a self resolving condition, it can sometimes become life threatening, with its myriad clinical presentation hence physicians and gynaecologists and infertility specialists should take care in prevention and early diagnosis and treatment of OHSS.

Keywords: Ultrasound guided paracentesis, albumin therapy, cabergoline

INTRODUCTION

In treating infertility, ovulation induction plays a crucial role. However in few patients, Ovarian stimulation rarely results in an iatrogenic complication because of the exaggerated response known as the ovarian hyper stimulation syndrome.[1-3] There is excessive response to medications like clomiphene citrate, gonadotropins due to which large number of follicles develop. Also as human chorionic gonadotropins causes the ovary to undergo extensive luteinization, large amounts of estrogen and progesterone and also local cytokines are being released.[4-6] The vascular endothelial growth factor (VEGF) is important to induce vascular hyper permeability, therby there is local capillary leak. This could lead to a lethal condition that happens due to accumulation of excessive exudate combined with intravascular volume depletion and subsequent hemoconcentration. OHSS is divided into mild, moderate and severe.[7-9] In mild OHSS, ovaries are enlarged upto 12cm and they can also present with bloating, nausea, vomiting and swelling of the abdomen. The size of the ovary is a marker of the degree of OHSS. When OHSS is severe, patients can present with shortness of breath, abdominal pain, dehydration .Also it is divided into early OHSS and late OHSS.[10] Early OHSS develops before pregnancy and late OHSS is seen in early weeks of pregnancy. Rarely deaths are reported due to this condition. Early diagnosis and immediate management prevents complications of OHSS. Hence ultrasound examination, blood tests are routinely done. Also studies have shown that cabergoline can also be used which might reduce the fluid accumulation. If patients don't improve with outpatient care, we need to admit the patient and monitor them closely.[11-13]

CASE REPORT

A 32 year old nulligravida with PCOD, gives history of undergoing embryo transfer 2 days before. As part of IVF protocol, she was administered gonadotropin releasing hormone. She was subsequently given human chorionic gonadotropin after confirming the presence of multiple follicles on USG, followed by oocyte aspiration under sedation.

Chief Presenting Complaints

She came to casualty with complaints of breathlessness, severe abdominal pain and abdominal distension associated with vomiting

Menstrual history

Regular cycles, 3/26-28; Not associated with pain and clots

Marital history

Married for 2 years; Non consanguineous marriage

Past history

Ovulation induction with letrozole followed by intrauterine insemination done 3 cycles in the past . In view of reduced AMH , she preferred to undergo Intracytoplasmic sperm injection(ICSI).

Medical history

No chronic medical illness

Family history

Not significant

Observation

At the time of admission, she was conscious, well oriented, with pulse rate 110/min, blood pressure 130/80mmHg and oxygen saturation 92% in room air. On examination, abdomen was severely distended with ascites. No evidence of palpable mass in the abdomen. Patient was transferred to ICU for close monitoring. She was conscious, oriented and was hemodynamically stable.

Diagnosis

In view of recent embryo transfer and the presenting complaints , diagnosis of OHSS was made.

Treatment

However, she required oxygen supplementation and CPAP support for 2 days for respiratory distress and diuretic therapy view of reduced urine output. ultrasound examination of her chest and abdomen revealed bilateral mild pleural effusion and bilateral bulky ovaries with moderate ascites. She underwent ultrasound guided paracentesis after which she felt comfortable. She improved with fluid restriction, prophylactic broad spectrum antibiotics, albumin infusion and low molecular weight heparin. Pleural effusion resolved and she was discharged in stable condition. In recent trend , with increase in ART procedures, this case alarms me to use ovarian stimulating agents with much more caution as not every individual react the same way to the standard treatment protocols. I understood that I need to individualise and treat them , keeping in mind the risk factors.

DISCUSSION

OHSS is usually a complication of ART following exogenous gonadotropins therapy. But rarely cases are also reported with natural conception. Risk factors that can predispose to OHSS are younger age, PCOD, multiple follicles, retrieval of more than 20 follicles, high doses of exogenous gonadotropins, raised serum estradiol (E2) levels, conception and previous history of OHSS. This patient is a known case of PCOD and had multiple follicles on USG.[13-15] Recognizing the risk factors and individualizing the management regimen are the key factor in prevention of OHSS. Monitoring ovarian response to gonadotropins by USG and serum E2 levels is considered as the gold standard in predicting the patients at risk of OHSS.[5] Also when there is a sign of impending OHSS or risk of developing OHSS following measures to be followed:

1. Cycle cancellation
2. Coasting (withholding the Follicular Stimulating Hormone injections) and monitoring the follicular development and E2 level
3. Withhold the HCG injections used for ovulation trigger
4. Reduce the dose of the HCG trigger from 10,000 IU to 5,000 IU
5. Tab. Cabergoline 0.5mg daily (post oocyte retrieval whenever indicated)
6. Use progesterone and avoid HCG for leuteal phase support.
7. Parenteral administration of prophylactic 25% albumin (20-50g) during oocyte retrieval in high risk cases such as markedly elevated estradiol levels or previous history of OHSS.[6]

The distinguishing feature of OHSS is increase in capillary permeability and fluid accumulation in the third space. Abdominal discomfort and distention due to ascites is the first indication of the fluid shift. Massive extravascular exudation can result in tense ascites, pleural effusion, pericardial effusion, oliguria, electrolyte imbalance and hypovolemic shock.[7,16,17] It can even lead to life threatening complication like ARDS, renal failure, thromboembolic events and death. Usually OHSS resolves spontaneously with supportive and symptomatic management. Fluids should be administered judiciously to maintain adequate urine output and reverse hemoconcentration. Central venous pressure monitoring is needed in patients with pulmonary edema and renal impairment. 20% albumin is an effective plasma expander when crystalloids fail to correct hemodynamic instability and achieve satisfactory urine output. Also, fresh frozen plasma may be used as an alternative. Diuretics should be used carefully as these patients may have intravascular volume depletion. Low dose dopamine has also been used in attempt to improve urine output when other measures fail.[18] Ultrasound (USG) guided paracentesis is done in patients with tense ascites that results in respiratory distress, oliguria and abdominal pain.[19] Infertile women with PCOS pose a threat during ovarian stimulation because they tend to exhibit increased response to gonadotropins, clomiphene citrate, ovarian drilling, insulin sensitizing agents or ART (assisted reproductive technology). This leads to increased risk of OHSS (ovarian hyperstimulation syndrome) and multiple gestations. Recent application of GnRH antagonist protocol, blastocyst transfer and oocyte / embryo vitrification results in reduction or elimination of OHSS associated with ovarian hyperstimulation for IVF management.[10,20]

CONCLUSION

Infertility affects 10-15% of the couples in industrialized countries. With increasing rates of infertility in the population, the assisted reproductive techniques have also increased dramatically. With increased use of ART, OHSS may occur more frequently in future. It is important to categorise women based on their risks of developing OHSS and individualizing the treatment to curtail their chances of developing the syndrome. There is no perfect strategy that completely eliminates the risks of OHSS. Hence prevention is better than cure. Primary prevention includes Targeted unifollicular ovulation, reducing the gonadotropin dose, avoiding adjunct GnRH agonist utilization , reducing the gonadotropin duration, utilizing adjuvant metformin

therapy, utilizing aromatase inhibitors for ovarian stimulation, individualizing IVF treatment regimes, avoiding Hcg for luteal phase support. Early diagnosis, closed monitoring and supportive therapy may help in preventing further complications.

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CONFLICT OF INTEREST

Conflict of interest declared none.

REFERENCES

1. McClure N, Healy DL, Rogers PA, Sullivan J, Robertson DM, Haning Jr RV, Connolly DT. Vascular endothelial growth factor as capillary permeability agent in ovarian hyperstimulation syndrome. The Lancet. 1994 Jul 23;344(8917):235-6.
2. Shaker AG, Zosmer A, Dean N, Bekir JS, Jacobs HS, Tan SL. Comparison of intravenous albumin and transfer of fresh embryos with cryopreservation of all embryos for subsequent transfer in prevention of ovarian hyperstimulation syndrome. Fertility and Sterility. 1996 May 1;65(5):992-6.
3. Isik AZ, Vicdan K. Combined approach as an effective method in the prevention of severe ovarian hyperstimulation syndrome. European Journal of Obstetrics & Gynecology and Reproductive Biology. 2001 Aug 1;97(2):208-12.
4. Forman RG, Frydman R, Egan D, Ross C, Barlow DH. Severe ovarian hyperstimulation syndrome using agonists of gonadotropin-releasing hormone for in vitro fertilization: a European series and a proposal for prevention. Fertility and sterility. 1990 Mar 1;53(3):502-9.
5. Carizza C, Abdelmassih V, Abdelmassih S, Ravizzini P, Salgueiro L, Salgueiro PT, Jine LT, Nagy P, Abdelmassih R. Cabergoline reduces the early onset of ovarian hyperstimulation syndrome: a prospective randomized study. Reproductive biomedicine online. 2008 Jan 1;17(6):751-5.
6. Várnagy Á, Bódis J, Mánfai Z, Wilhelm F, Busznyák C, Koppán M. Low-dose aspirin therapy to prevent ovarian hyperstimulation syndrome. Fertility and sterility. 2010 May 1;93(7):2281-4.
7. Elchalal U, Schenker JG. The pathophysiology of ovarian hyperstimulation syndrome-views and ideas. Hum Reprod. 1997;12:1129–37.
8. Lee TH, Liu CH, Huang CC, Wu YL, Shih YT, Ho HN, et al. Serum anti-mullerian hormone and estradiol levels as predictors of ovarian hyperstimulation syndrome in assisted reproduction technology cycles. Hum Reprod. 2008;23:160–7
9. Delvigne A, Rozenberg S. Epidemiology and prevention of ovarian hyperstimulation syndrome (OHSS): a review. Human reproduction update. 2002 Nov 1;8(6):559-77.
10. Nastri CO, Teixeira DM, Moroni RM, Leitão VM, Martins WP. Ovarian hyperstimulation syndrome: pathophysiology, staging, prediction and prevention. Ultrasound in Obstetrics & Gynecology. 2015 Apr;45(4):377-93.
11. 1. Borenstein R, Elhalah U, Lunenfeld B, Schwartz ZS. Severe ovarian hyperstimulation syndrome: A reevaluated therapeutic approach. *Fertil Steril.* 1989;51(5)
12. 2. Rosen GF, Lew MW. Severe ovarian hyperstimulation in a spontaneous singleton pregnancy. *Am J Obstet Gynecol.* 1991;165(5pt1):1312–131
13. 3. Zalel Y, Katz Z, Caspi B, Ben-Hur H, Dgani R, Insler V. Spontaneous ovarian hyperstimulation syndrome concomitant with spontaneous pregnancy in a woman with polycystic ovary disease. *Am J Obstet Gynecol.* 1992;167(1):122–124.
14. Olatunbosun OA, Gilliland B, Brydon LA, Chizen DR, Pierson RA. Spontaneous ovarian hyperstimulation syndrome in four consecutive pregnancies. *Clin Exp Obstet Gynecol.* 1996;23(3):127–132.
15. Schnorr JA, Jr, Miller H, Davis JR, Hatch K, Seeds J. Hyperreactio luteinalis associated with pregnancy: A case report and review of the literature. *Am J Perinatol.* 1996;13(2):95–97.

16. Schenker JG, Polishuk WZ. The role of prostaglandins in ovarian hyperstimulation syndrome. *Eur J Obstet Gynecol Reprod Biol.* 1976;6(2):47–52
17. Pride SM, Yuen BH, Moon SY. Clinical, endocrinologic, and intraovarian prostaglandin F response to H1 receptor blockade in the ovarian hyperstimulation syndrome: Studies in the rabbit model. *Am J Obstet Gynecol.* 1984;148(5):670–674.
18. Dourron NE, Williams DB. Prevention and treatment of ovarian hyperstimulation syndrome. *Semin Reprod Endocrinol.* 1996;14(4):355–365.
19. Clement PB. Tumor-like lesions of the ovary associated with pregnancy. *Int J Gynecol Pathol.* 1993;12(2):108–115.
20. Wajda KJ, Lucas JG, Marsh WL., Jr Hyperreactio luteinalis. *Arch Pathol Lab Med.* 1989;113(8):921–925.

SP-30

Ovarian Ectopic Pregnancy In A Iucd User, A Case Report

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Abstract: Background: The rarest form of non tubal ectopic pregnancy is OVARIAN PREGNANCY. The most end result of ovarian pregnancy is the rupture before the first trimester ends. One of the cause for ovarian pregnancy is the use of intrauterine device (IUD). This report shows a case of ovarian ectopic pregnancy in a IUCD user. Mrs. X, 30yrs, G3P2L2 with two previous cesarean sections with H/O Cu-T insertion as a contraceptive. The patient presented with signs and symptoms of ruptured ectopic pregnancy. Patient was taken up for emergency laparotomy. Intraoperatively ruptured ovarian ectopic pregnancy was identified for which oophorectomy was done .Histopathological examination confirmed it to be an ovarian ectopic pregnancy. IUCD as a contraceptive prevents intra uterine implantation of about 99%. When pregnancy (implant) occurs during IUCD usage ,it is high likely to end up in ectopic implantation. The use of IUD is found to be the most common risk factors for ovarian ectopic pregnancy as discussed in this case report.

Key Words: Ovarian pregnancy, intra uterine devices, laparoscopy, laparotomy, histopathology

INTRODUCTION

The rare variant of ectopic implantation is Ovarian ectopic pregnancy . It ends up with rupture even before the end of the first trimester. The rate of occurrence of ovarian ectopic pregnancy with natural contraception varies the rate of incidence of IUCD users. Ovarian pregnancy is common with one in every ten ectopic pregnancy.[1] While the risk factors are similar to those of tubal pregnancy, the use of an IUD seems disproportionately associated with it. Although the ovary can accommodate the growing pregnancy more readily than its fallopian tube counterpart, rupture at a young age is usually the result.[2] Advance ovarian pregnancies are exceptional. Approximately 75% terminate in first trimester and are often misdiagnosed as corpus luteum haemorrhage.[3] The Spielberg criteria (1878)- a) fallopian tube as the affected site must be intact b) the foetal sac must occupy the position of the ovary c) the ovary must be connected to the uterus by ovarian ligament and d) ovarian tissue must be located in the sac wall, are essential for confirmation of early ovarian pregnancy. In advance pregnancies last criterion i.e. detection of ovarian tissue in the wall of sac may not be satisfied as parenchyma is compressed laminated and distended by developing foetus.[4] USG plays an vital role in the diagnosis of ectopic pregnancy.

Diagnosis

Findings are almost similar to that of tubal pregnancy or a bleeding corpus luteum. Observations of severe bleeding are rare (1/3 of all cases). During surgery, the ovarian pregnancies are typically regarded as "corpus luteum cysts" or "bleeding corpus luteums". Transvaginal sonography has always shown an accurate diagnosis of unruptured ovarian pregnancy during the evaluation of ectopic pregnancy.

Management

The line of management is surgical for ovarian pregnancies .Small lesion with early bleeding has been managed by ovarian wedge resection or cystectomy. Oophorectomy or ovariectomy is performed for larger lesions . Laparoscopic management can also be used to resect or used to perform laser ablation for the same. Methotrexate is the final and a successful management to treat unruptured ovarian ectopic pregnancy .

CASE REPORT

Mrs X, 30yrs, G3P2L2, previous with two previous cesarean sections , using IUCD as a contraceptive presented with hypogastric pain with H/O 2months of amenorrhoea . β hCG was 800 IU .
No c/o bleeding or spotting PV
No other complaints .
PAST MEDICAL HISTORY:
Menarche at 12yrs. Regular menstrual cycle , normal flow .
H/O previous 2 LSCS.
H/O copper T in situ 2yrs back
Not a known case of DM/ HTN/TB/BA/THYROID DISORDER/ EPILEPSY.
NO H/O blood transfusion -no post transfusion reaction
No H/O drug allergy

FAMILY HISTORY :

Nil significant.

O/E:
Afebrile , dull looking
Pallor +
Hydration - good .
S/E:
CVS/RS – NAD
P/A- Tense, hypogastric tenderness +
P/S- cervix & vagina – healthy
No abnormal discharge.
P/V- cervix – downwards , uterus – anteverted , bulky
Cervical motion tenderness +, fornices free
INVESTIGATION:

- Complete blood count
- Urine routine
- Blood grouping & typing
- Urine pregnancy test
- RFT with Serum electrolytes
- Bleeding time & clotting time
- COVID RT-PCR

SPECIAL INVESTIGATION

USG Whole Abdomen
PROGNOSIS , FIGURES & TABLES- Not applicable

DIAGNOSIS & TREATMENT

USG showed
Uterus of normal size with Cu-t insitu with no gestational sac.
Right adenexal mass of 1x2cm with peripheral vascularity with free fluid in peritoneal cavity.
Imp- Right tubal ectopic pregnancy
Then diagnosed as a case of Ruptured ectopic and taken up for EMERGENCY LAPAROTOMY.
The intra op findings are
Uterus- normal
Right ovary enlarged with evidence of ectopic gestation , bleeding +
Right tube- normal with no evidence of rupture .
Left tubes and ovary – normal
Hemoperitoneum + (1000ml) in the cavity .
As there is a possibility of tubal ectopic with secondary attachment to ovary / primary ovarian ectopic pregnancy was considered. Hence, proceeded with RIGHT SALPHINGO OOPHORECTOMY and sent for HPE . Cu-T was removed by pulling the thread.
HPE findings showed primary ovarian ectopic pregnancy .

DISCUSSION

The embryo that gets implanted and develops anywhere outside the uterine cavity is named as ECTOPIC PREGNANCY. The various sites where the ectopic pregnancy can occur are fallopian tubes, ovaries or peritoneal cavity. Hertig estimated that ovarian pregnancy occurs one in 25 000 to 40 000 pregnancies. It is characterized by a poor clinical symptoms and a difficult ultrasound diagnosis. The surgical criteria remain hard to prove.[5-7] However, Ovarian ectopic pregnancy accounts for about 3% of ectopic pregnancies.[8-10] Its physiopathology is not well known, it would seem to be secondary to a reflux of the fertilized oocyte towards the ovary.[2,11] Cases of ovarian ectopic pregnancy after in vitro fertilization maybe due to the reflux theory.[3,12] Pregnancy is preferentially implanted on the scar of the original follicular ostium, rich in fibrin and new capillaries.[4] This theory corresponds to the intra follicular and juxta follicular forms. More rarely, this implantation will be done at a distance from the corpus luteum or even on the contralateral ovary, corresponding then to the juxta cortical and interstitial forms whose pathophysiology remains obscure. More rarely, ovarian ectopic pregnancy can be bilateral or part of a heterotopic pregnancy.[5] Choi *et al* reported endometriosis and previous abdominal surgeries as the most common risk factors in ovarian pregnancy but in this case report the patient had no features of endometriosis or history of abdominal surgery. Recently ovarian ectopic pregnancy has been reported after tubal ligation.[13] Rupture in the first trimester is the common presentation in an ovarian ectopic pregnancy, but the pregnancy may advance to full term. The increased use of intra uterine contraceptive devices (IUDs) is believed to be the major cause of ovarian pregnancy. It is estimated that the use of the IUCD reduces the rate of implantation in the uterus by 99.5%, the tubal implantation by 95%, and there is no prevention of implantation in the ovaries with the IUCD.[14] Various observational studies have shown 59-90% use of IUCD in women with primary ovarian pregnancy. The copper ions induce inflammation in the endometrial lining. By tubal mobility the ions reach the tubal epithelium. Hence

inflammation occurs in tubal epithelium also. This prevents pregnancy in uterus and tubes. Hence when pregnancy occurs with IUCD in situ, the ovaries are most commonly affected. No specific USG criteria have been approved for diagnosing ovarian ectopic pregnancy preoperatively. Echogenic mass in ovary can suggest ovarian ectopic pregnancy. Other findings that can be observed are echogenic outer ring with internal anechoic area, fetal heart pulsation in colour Doppler.[5] Differential diagnoses in USG are corpus luteal cyst, tubal ectopic pregnancy attached to ovary, ovarian germ cell tumour. High index of suspicion is necessary to diagnosis ovarian ectopic pregnancy pre operatively. Diagnosis is usually confirmed intra operatively and with the help of Histopathological examination. The treatment of choice for ovarian ectopic pregnancy is surgical management either laparoscopy / laparotomy. In our case, since the patient was hemodynamically unstable with ruptured ectopic pregnancy, Laparotomy was preferred. Even in cases of unruptured ovarian ectopic pregnancy. Studies have shown that methotrexate is not as effective as in tubal pregnancy. Some studies have shown that methotrexate is effective only in 40% of ovarian pregnancy.

CONCLUSION

Primary ovarian pregnancy is a rare form of ectopic pregnancy. The diagnosis is generally made at the time of surgery & confirmed by HPE. In the surgical management, wedge resection, oophorectomy is the possible options. Acute abdomen with hemoperitoneum in a woman of reproductive age group should always raise the suspicion of ectopic pregnancy in the treating physician. While tubal pregnancy is the most common type of ectopic pregnancy, no. Tubal ectopic pregnancy should also be considered especially when the woman is on intra uterine contraceptive device as the contraceptive. Preoperative diagnosis by ultrasound is difficult. Intraoperative findings may mimic ruptured corpus luteal cyst. Though wedge resection is a possible surgical option, in our patient since there was heavy bleeding, oopherectomy was done. Histopathological examination would always confirm the diagnosis of primary ovarian pregnancy.

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CONFLICT OF INTEREST

Conflict of interest declared none.

REFERENCES

1. Patel Y, Wanyonyi SZ, Rana FS. Laparoscopic management of an ovarian entopic pregnancy: Case report. East African medical journal. 2008;85(4):201-4.
2. Hertig AT. Discussion of Gerin-Lojoie L. Ovarian pregnancy. Am J Obstet and Gynecol. 1951;62:920.
3. Grimes HG, Nosal RA, Gallagher JC. Ovarian pregnancy: a series of 24 cases. Obstetrics and gynecology. 1983 Feb 1;61(2):174-80.
4. Bouyer J, Coste J, Fernandez H, Pouly JL, Job-Spira N. Sites of ectopic pregnancy: a 10 year population-based study of 1800 cases. Human reproduction. 2002 Dec 1;17(12):3224-30.
5. Hertig AT. Discussion of Gerin-Lojoie L. Ovarian pregnancy. Am J Obstet and Gynecol. 1951;62:920.
6. Hallatt JG. Primary ovarian pregnancy: a report of twenty-five cases. *Am J Obstet Gynecol.* 1982;143:55–60.
7. Studzinski Z, Branicka D, Filipczak A, Olinski K. Prolonged ovarian pregnancy: A case report. *Ginekol Pol.* 1999;70:33–35.
8. Agdi M, Tulandi T. Surgical treatment of ectopic pregnancy. Best Pract Res Clin Obstet Gynaecol. 2009 Aug;23(4):519-27.
9. Sergent F, Mauger-Tinlot F, Gravier A, Versycke E, Marpeau L. Ovarian pregnancies: reassessment of diagnostic criteria. J Gynecol Obstet Biol Reprod (Paris). 2002 Dec;31(8):741-6.
10. Kraemer B, Elizabeth K, Ersin G, Juhasz-Boess I, Erich-Franz S, Diethelm W *et al* . Ovarian ectopic pregnancy: diagnosis, treatment, correlation to carnegie stage 16 and review based on a clinical case. Fertil and Sterile. 2009;92:392.
11. Molinaro TA, Barnhart KT. Ectopic pregnancies in unusual locations. Semin Reprod Med. 2007;25:123-30.
12. Comstock C, Huston K, Lee W. The ultrasonographic appearance of ovarian ectopic pregnancies. Obstet Gynecol. 2005 Jan;105(1):42-5.
13. Choi HJ, Im KS, Jung HJ, Lim KT, Mok JE, Kwon YS. Clinical analysis of ovarian pregnancy: a report of 49 cases. *Eur J Obstet Gynecol Reprod Biol.* 2011;158:87–89.
14. Vahnu C, Rajlaxmi M, Vandana R. Primary Ovarian Pregnancy after Interval Tubal Ligation: A Case Report. *J Fam Reprod Health.* 2013;7:187–188.

SP-31

Juvenile Lupus Erythematosiswith Sjogren Syndrome - Overlap Syndrome-A Rare Presentation Of Connective Tissue Disorder

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Abstract: Overlap syndrome is an inflammatory rheumatic condition in which the patient presents with clinical symptoms showing several different immune disorders. In this case study we concluded that Our case is overlap syndrome because it meets both the SLICC criteria and the criteria for primary Sjogren's syndrome. They share similar clinical features, etiology, and similar immunological findings that confuse the final diagnosis. Immediate diagnosis can prevent harmful consequences. 13 year old girl presented with complaints of fever for 5days, child had swelling and pain in B/L parotid region for 1 week at the time of presentation, she had history of recurrent parotid swelling for the past 1 year with significant weight loss. On examination child was anaemic weighing 24kg, undernourished. B/L cervical significant lymphadenopathy was present, Oral cavity examination showed oral candidiasis, Rt parotid duct opening inflamed, no discharge noted. Head and neck examination showed bilateral parotid gland enlargement. Vitals were within normal limits. By the work up local causes, infective causes, immunodeficiency, structural causes of recurrent parotitis was ruled out. Hence was further evaluated for the rheumatological causes of recurrent parotitis. ANA was done which showed high positivity, hence Immunoblot and ANA profile was evaluated.ANA profile showed values suggestive of connective tissue disorder with points towards both lupus and sjogren syndrome. Biopsy was taken to check for involvement of renal parenchyma.

Keywords: Sjogren syndrome, Systemic lupus erythematous, Overlap syndrome.

1. INTRODUCTION

The term overlap syndrome is typically applied to patients who have two or more distinctly recognizable rheumatic diseases and the presence of specific autoantibodies. The patient's characteristics may range from the presence of a single clinical or laboratory finding, such as a positive ANA to the presence of a number of clinical or serological features specific to both SLE and Sjogren syndrome.[1,] There may be evolution of the disease into a clinically recognizable rheumatic disease or with features of both, as overlap syndrome. Sjogren Syndrome (SS) is defined as a chronic autoimmune disease characterised by inflammation of the exocrine glands. The principal inflammatory targets are the salivary and lacrimal exocrine glands, resulting in dryness of the mucosal surfaces of the mouth and eyes.[2] However, there can be more extensive exocrinopathy involving skin, the respiratory tract, and urogenital tracts. Extra-glandular or systemic features can also be part of the disorder.

2. CASE PRESENTATION

13-year-old girl presented with Complaints of fever for 5days, child had swelling and pain in B/L parotid region for 1 week at the time of presentation, she had history of recurrent parotid swelling for the past 1 year, which was episodic, 10 episodes till now, sudden onset, spontaneous resolution occurs after a month when on anti-inflammatory drugs. These episodes were associated with fever intermittently. There was significant weight loss over 1 year., non-scarring alopecia, occasional arthralgia and myalgia on exertion with no joint swelling, appetite was reduced. There were no bowel and bladder disturbances. There was an h/o episodic erythematous rash on sun exposed areas.

Past history

Frequent hospitalization for same illness from 2018. She was developmentally normal, child withunderweight for age. Child was immunized according to National Immunization Schedule.

Examination

On examination child was anaemic, weighing 24kg, undernourished. B/L cervical significant lymphadenopathy was present, Oral cavity examination showed oral candidiasis, Rt parotid duct opening inflamed, no discharge noted. Head and neck examination showed bilateral parotid gland enlargement. Vitals were within normal limits.

3. DIAGNOSIS

Laboratory test showed TLC-13030, neutrophilia with lymphopenia, Hb-10.5, RBC-3.65, PT-2.62 lakh, Urine was Pale yellow, slightly turbid, PH-8, protein-1+, sugar- absent, RBC-NIL, Cast & crystals-absent, pus cells-3-4, epi-2-3. Peripheral smear revealed Normochromic RBC, Serum Amylase was elevated-503 IU/L CRP-9.6mg/dl, elevated ESR. LFT- Albumin-3.8g , Globulin-5.3gm, A/G ratio-0.7, GGT-16 IU/L, Retroviral serology-negative, RFT-Normal, Blood culture-negative, 24hrs urine revealed significant proteinuria, Schimers test- positive, other ophthalmology examination was normal. Ultrasonogram of salivary gland did not show findings suggestive of sialactesis or sialolithiasis. Direct Coombs test was negative to rule of hemolytic

anaemia and Mantoux was negative.By these work up local causes, infective causes, immunodeficiency, structural causes of recurrent parotitis was ruled out. Hence was further evaluated for the rheumatological causes of recurrent parotitis in view of

3.1 Female child
3.2 Recurrent fever
3.3 Raised ESR & CRP
3.4 Persistent lymphopenia
3.5 Raised globulin
3.6 Proteinuria
3.7 Mother h/o DLE.

Hence ANA was done which showed high positivity, hence Immunoblot and ANA profile was evaluated.ANA profile showed values suggestive of connective tissue disorder with points towards both lupus and sjogren syndrome. Biopsy taken to check for involvement of renal parenchyma.

Table 1: Rheumatological work up

ds DNA	0.04	Negative
C3	111mg/dl	Normal
C4	15.50 mg/dl	Normal
Cardiolipin AB IgG	1.29GPL/ml	Negative
Cardiolipin AB IgM	1.45MPL/ml	Negative
ANA	1:100(3+)	Positive

Table 2:Immunoblot

Mi2	Negative	SSB	Strong positive
RNP	Negative	ds DNA	Negative
Sm	Borderline	nucleosomes	Negative
SSA native	Strong positive	Histones	Negative
Ro 52	Strong positive	ribosomes,AMA-M2, OFS-70	Negative

Renal biopsy showed Minimal mesangial lupus nephritis which is very minimal.

DISCUSSION

Sjogren syndrome and SLE share same etiopathogenic links which is why presentation of them are alike and evolution is very unpredictable[3]. They share genetic, epigenetic, hormonal factors which is why its more common in females, environmental exposure vulnerable on sun exposure, basically they target the cell by activation of B-cell and T-cell, which in turn activates cytokines which destroy the organ.[4] The loss of immune tolerance, increased antigenic load, excess T cell help, defective B cell suppression, and the shifting of T helper 1 (Th1) to Th2 immune responses leads to B cell hyperactivity and the production of pathogenic autoantibodies.[5] Finally, certain environmental factors are probably required to trigger the disease.Primary Sjogren syndrome is autoimmune condition affecting exocrine gland which causes keratoconjuctivitis sicca, parotitis in absence of other autoimmune condition, whereas secondary sjogren is occur in presence of other autoimmune condition.[6] Below are features comparing primary and secondary sjogren syndrome.

Table 3 : Features of primary SS and sSS-SLE

Features	pSS	sSS-SLE
Age[6,86,87]	↑	↓
Female prevalence[11,86,87]	= or ↑	= or ↓
Frequencies		
Eye dryness[6,8,73,86]	= or ↑	= or ↓
Dry mouth[6,8,86]	= or ↑	= or ↓
Parotiditis[24,86]	↑	↓
Arthritis[86]	↓	↑
Kidney involvement[8,24,86]	↓	↑
Anti-Ro (SSA)/anti-La (SSB)[6,86]	↑	↓

Features in relation to our child is 13yrs,female, dry eye, recurrent parotitis, kidney involvement, anti Ro/anti La positive. Child did not have arthritis. Fulfilling criteria were recurrent parotitis, systemic manifestations, anti-SSA, anti-SSB, high ANA, elevated serum amylase leucopenia and high ESR.

Requirements: ≥ 4 criteria (at least 1 clinical and 1 laboratory criteria)
OR biopsy-proven lupus nephritis with positive ANA or Anti-DNA

Clinical Criteria	Immunologic Criteria
1. Acute Cutaneous Lupus*	1. ANA
2. Chronic Cutaneous Lupus*	2. Anti-DNA
3. Oral or nasal ulcers *	3. Anti-Sm
4. Non-scarring alopecia	4. Antiphospholipid Ab *
5. Arthritis *	5. Low complement (C3, C4, CH50)
6. Serositis *	6. Direct Coombs' test (do not count in the presence of hemolytic anemia)
7. Renal *	
8. Neurologic *	
9. Hemolytic anemia	
10. Leukopenia *	
11. Thrombocytopenia (<100,000/mm^3)	

†SLICC: Systemic Lupus International Collaborating Clinics

4. TREATMENT

The girl was treated with steroids and hydroxychloroquine and lubricant drops for dry eyes. She showed total remission and her renal parameters improved. Child is currently under follow up.

5. CONCLUSION

Primary sjogren is a rare condition to present atyoung age. Secondary sjogren is also rare in adolescence. Our case satisfies both SLICC criteria and criteria for primary Sjogren syndrome, hence it is a Overlap syndrome. They share similar clinical features, etiopathogenity, similar immunological findings confounding the ultimate diagnosis, we can prevent adverse outcomes on prompt diagnosis. Sjogren syndrome is treated supportively with lubricant eye drops. All SD patients with xerostomia should be given fluoride for caries prophylaxis. Disease-modifying anti-rheumatic drugs can be used to treat inflammatory musculoskeletal pain starting with hydroxychloroquine as first-line therapy. Hydroxychloroquine at 10mg/kg/day. Self-care measures and exercises will reduce pain and fatigue. Biological therapy like rituximab is best used in patients with serious organ manifestations who fail more conservative treatments. Follow up is must, vaccination for pneumococcal disease& counselling is very much essential.

6. CONFLICT OF INTEREST

Conflict of interest declared none.

7. ACKNOWLEDGEMENT

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8. FUNDING SOURCE

None

9. REFERENCES

1. Cassidy JT, Petty RE, Laxer RM, Lindsley CB. Textbook of pediatric rheumatology E-Book. Elsevier Health Sciences; 2010 Oct 15.
2. Robert M, kliegmann S, Bonita F, ST G, Joseph W. Nelson textbook of pediatrics: first south Asia edition. Elsevier India; 2015.
3. Yao Q, Altman RD, Wang X. Systemic lupus erythematosus with Sjogren syndrome compared to systemic lupus erythematosus alone: a meta-analysis. J Clin Rheumatol. 2012;18(1):28–32.
4. Alani H, Henty JR, Thompson NL, Jury E, Ciurtin C. Systematic review and meta-analysis of the epidemiology of polyautoimmunity in Sjögren's syndrome (secondary Sjögren's syndrome) focusing on autoimmune rheumatic diseases. Scandinavian journal of rheumatology. 2018 Mar 4;47(2):141-54.

5. Novljan MP, Rozman B, Jerše M, Rotar Ž, Vidmar G, Kveder T, Tomšič M. Comparison of the different classification criteria sets for primary Sjögren's syndrome. Scandinavian journal of rheumatology. 2006 Jan 1;35(6):463-7.
6. Venables PJ. Management of patients presenting with Sjogren's syndrome. Best Pract Res Clin Rheumatol. 2006 Aug;20(4):791-807.

SP-32

Cutaneous Peripheral T-Cell Lymphoma, Unspecified – A Case Report

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Abstract: Primary Cutaneous Peripheral T-Cell Lymphoma, unspecified is a rare, fatal dermatologic disease with features similar to many common inflammatory skin conditions, making the diagnosis very difficult and this is diagnosis of exclusion. Regulatory T cells protect against autoimmune reactions to self antigens and assist in the resolution of cutaneous inflammation. CTCLs result from errors in the production of T-lymphocytes or transformation of T-lymphocytes into malignant cells The types of cutaneous T cell lymphoma include CD4 and CD30 lymphoproliferative diseases including mycosis fungoides and sezary syndrome. Here we report a case of 59 year old female, presented with painful raised lesion over right leg for 6 months. Initially started as a plaque and gradually progressed to a nodule within two months. O/E: Multiple tender erythematous nodules with areas of pigmentations were present in right leg. Punch biopsy was performed and initially diagnosed as fungal infection and confirmed as cutaneous T cell lymphoma in second biopsy using immunohistochemistry with positive CD3 and CD45. Due to the rarity of PTL-NOS and lack of literature, the knowledge about evidence-based treatments and survival is less. Recently monoclonal antibodies have been developed against the cytoplasmic granules of lymphocytes. Further research is needed to identify the underlying mechanisms of CTCL development and course as well as to better tailor treatment strategies to individual patients.

Keywords: Cutaneous, T cell Lymphoma, unspecified, CD, Mycosis fungoides and Sezary syndrome, Positive, monoclonal antibodies.

1. INTRODUCTION

There are two main types of lymphocytes: B-lymphocytes, which may produce specific antibodies to "neutralize" certain invading microorganisms, and T-lymphocytes, which may directly destroy microorganisms or assist in the activities of other lymphocytes. Regulatory T cells protect against autoimmune reactions to self antigens and assist in the resolution of cutaneous inflammation CTCLs result from errors in the production of T-lymphocytes or transformation of T-lymphocytes into malignant cells.[1,2] Primary cutaneous T cell lymphomas are heterogeneous group of lymphoproliferative disorders, characterized by monoclonal expansion of malignant T cells.[3] The most common form of cutaneous T cell lymphoma is mycosis fungoides and sezary syndrome. Primary cutaneous peripheral T cell lymphoma, Unspecified, is an aggressive dermatologic malignancy which constitutes around 10% of all cutaneous T cell lymphoma and commonly affects middle age to elderly individuals and they frequently present with nodules or tumors.[4] CTCL subtypes demonstrate a variety of clinical, histological, and molecular features, and can follow an indolent or a very aggressive course.

2. CASE REPORT

2.1 Presenting Complaint

59 year old female, presented with painful raised lesion over right leg for 6 months. Initially started as a plaque and gradually progressed to a nodule within two months.

2.2 Medical History & Family History: Not Suitable

2.3 Observation

O/E: Multiple tender erythematous nodules with areas of pigmentations were present in right leg (Fig 1). The provisional diagnosis was keloid blastomycosis

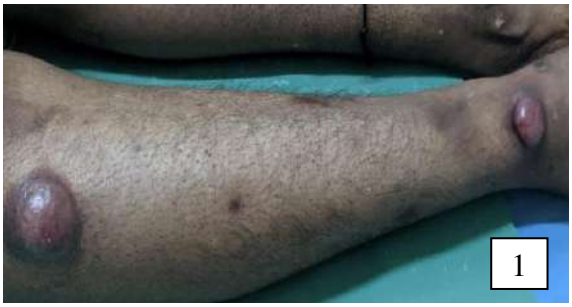


Fig 1: Multiple tender erythematous nodules with areas of pigmentations were present in right leg

3. SPECIAL TEST

First, punch biopsy and excision biopsy was done from the nodules from knee and near medial malleolus respectively. The histopathological picture was inconclusive from both the biopsies. Special stains were done which was negative for fungus since the provisional diagnosis was given as keloid blastomycosis and reported as inflammatory pathology. Second punch biopsy performed after twenty days, squamous epithelium with the dermis showing diffuse sheets of round cells with dense nucleias seen in (Figure 2), medium to large cells with haloed nucleias seen in (Figure 3) and few round cells with convoluted nuclei in the dermis. Few giant nuclei, mitotic figures and epidermotrophismas seen in (Figure 4) are also seen Immunohistochemistry showed positivity for CD3 (T cell marker) & CD45 (Leucocyte common antigen) as seen in (Figure 5 & 6) and negative for CD30, CD56as seen in (Figure 7 & 8), CD20 and Pan CK, thus confirms diagnosis as primary cutaneous peripheral T cell lymphoma, Unspecified by ruling out Merkel cell carcinoma using pan CK and diagnosed as Cutaneous T cell lymphoma.

Diagnosis: Primary Cutaneous Peripheral T-Cell Lymphoma, unspecified

Treatment: Patient was referred to other center for further management.

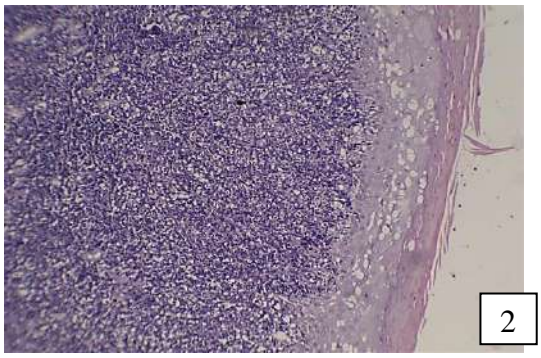


Fig 2: Shows dense round cells in dermis (4x)

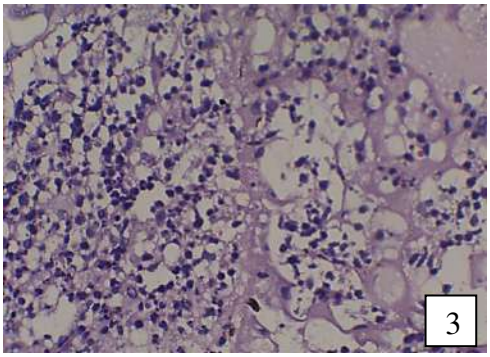
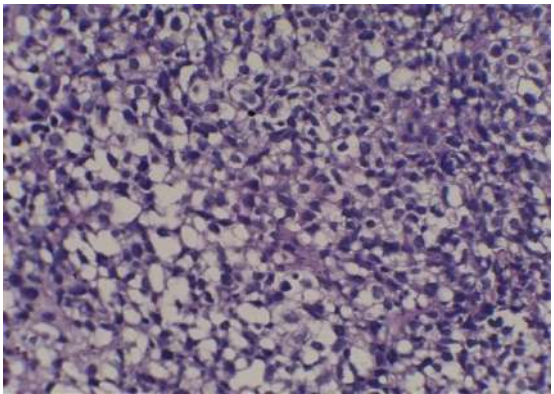


Fig 3: shows Epidermotrophism (40x)



4

Fig 4: Round cells with haloed nuclei(40x)

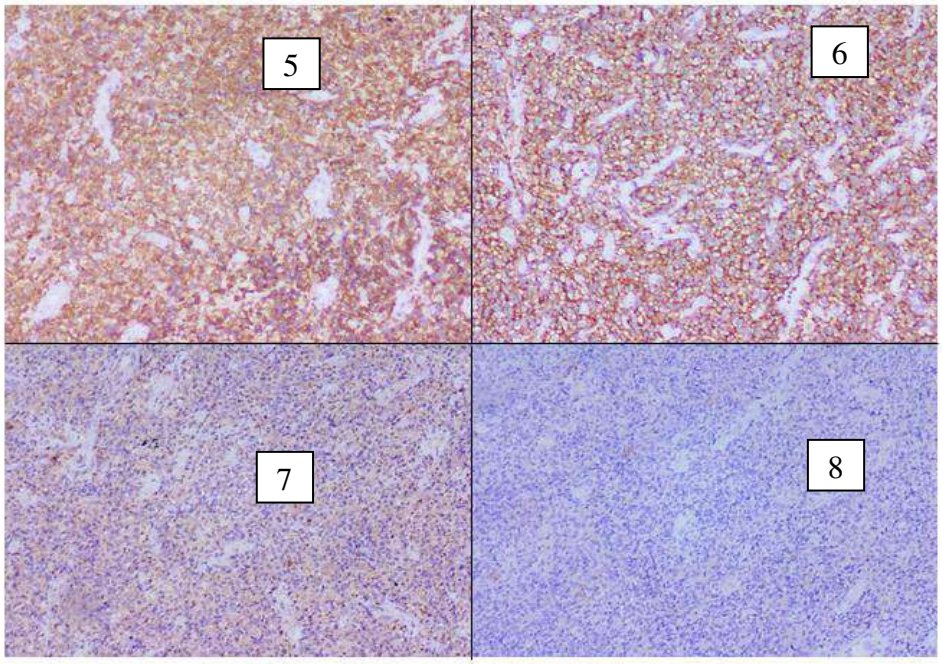


Fig 5: Shows positivity for CD3, Fig 6: Shows positivity for CD 45, Fig 7: Shows negative for CD 20, Fig 8: Shows negative for CD 56

4. DISCUSSION

In 1806, Alibert initially described mycosis fungoides (MF) as the infiltration of skin by lymphocytes. In 1974, Edelson used the term "cutaneous T cell lymphomas" (CTCLs) for MF and its leukemic variant, Sézary syndrome (SS), which are the major types of CTCL.[5] CTCL represent approximately 75–80% of all primary cutaneous lymphomas, whereas primary cutaneous B cell lymphomas account for approximately 20–25%.[6] Primary cutaneous T cell lymphoma are rare type of Non-Hodgkin's lymphoma constituting only 7%. Primary Cutaneous Peripheral T-Cell Lymphoma, unspecified is a rare aggressive form of extra nodal Non-Hodgkin's lymphoma with features similar to many common inflammatory skin conditions, making the diagnosis very difficult. Peripheral T-cell lymphoma (PTCL) refers to the thosetumours deriving from post-thymic (or mature) T-cells, not the site of origin. It accounts for approximately 10% of all cutaneous T cell lymphoma.[7] Patients are middle aged to elderly individuals with male to female ratio of 2.5:1. Patients presents with generalized nodules or tumors.The current edition of the World Health Organization (WHO) includes 12 CTCL subtypes with discrete diagnosable clinical, histologic and phenotypic features.[8] Histological features of cutaneous T cell lymphoma, unspecified has diffuse or nodular infiltrates with variable numbers of medium sized to large pleomorphic or immunoblast like T cell are typical.[9] PTL, unspecified, in the WHO classification represent a heterogeneous group which includes all T-cell neoplasms that do not fit into any of the better defined subtypes of T-cell lymphoma/leukemia.[10]The WHO-EORTC has developed set of criteria to diagnose PTCL from others. The criterion is based on exclusion of three entities: Primary Cutaneous CD4-Positive Small/Medium T-Cell Lymphoma (CD4+ SMTL), Primary Cutaneous CD8-Positive Aggressive Epidermotropic T-Cell Lymphoma (CD8+ AECTCL), and Primary Cutaneous Gamma/Delta T-Cell Lymphoma (CGD-TCL).[11] For the designation of Primary cutaneous peripheral T cell lymphoma, Unspecified, the larger cell component should constitute at least 30% of neoplastic infiltrates and epidermotrophism is generally mild or absent, in our case the biopsy showed more than 30% of larger cell component. The distinction from a systemic or primary cutaneous peripheral T-cell lymphoma, unspecified is more difficult in cases predominant with small and medium sized pleomorphic T cells. There is no difference in the survival of patients presenting with solitary or multifocal skin lesions, both develop widespread disease rapidly. The prognosis is usually poor due to rapid dissemination of the cutaneous tumors[12] and systemic involvement contributing to the five-year survival rate of less than 20%.Due to the rarity of PTL-NOS and lack of literature, the knowledge about evidence-based treatments and survival is less. It has been studied that age greater than sixty, Eastern Cooperative Oncology Group (ECOG) performance status of equal to or greater than two, lactate dehydrogenase levels at normal values or above, and involvement of the bone marrow are independent predictors of decreased survival. In a study bysavage et alhas explained about the prognostic significance of CXCR3 and CCR4 (Th2) expression was found to be associated with a poor outcome and remained significant after adjustment for other clinical factors, including the International prognostic index.[13,14] Majority of studies showed poor outcome even after standard chemotherapy.Recently, monoclonal antibodies against components of the cytotoxic granules present in the cytoplasm of cytotoxic lymphocytes have become available.[12] Recent studies suggest that more intensive regimens are also not effective in these PTLs, unspecified.[6] Patients with CTCL have reduced quality of life and a lack of effective treatment options. Further research is needed to better identify the underlying mechanisms of CTCL development and course as well as to better tailor treatment strategies to individual patients.[15]

5. CONCLUSION

CTCL should be suspected in patients with patches, plaques, erythroderma, or papules that persist or multiply despite conservative treatment. In its early stages, it mimics inflammatory skin conditions making the diagnosis difficult. So, high index of suspicion is mandatory in early diagnosis of these cases and confirmation with histopathology and immunophenotyping. There are many new therapies that are currently being investigated in clinical trials, and the DAB_{389}IL-2 fusion protein was recently approved for the treatment of refractory MF/SS.

6. CONFLICT OF INTEREST

Conflict of interest declared none.

7. ACKNOWLEDGEMENT

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8. FUNDING SOURCE: None

9. REFERENCES

1. Singh S, Bhatnagar S, Sarin A, Borde P, Bisht N. A case of cutaneous T-cell lymphoma (CD− 8+) with multiple lesions treated with definitive radiotherapy. Iraqi Journal of Hematology. 2018 Jul 1;7(2):93.Elder DE. Lever's histopathology of the skin. Lippincott Williams & Wilkins; 2014 Sep 9.
2. Clark RA. Skin-resident T cells: the ups and downs of on site immunity. Journal of Investigative Dermatology. 2010 Feb 1;130(2):362-70.
3. Aderhold K, Carpenter L, Brown K, Donato A. Primary cutaneous peripheral T-cell lymphoma not otherwise specified: a rapidly progressive variant of cutaneous T-cell lymphoma. Case reports in oncological medicine. 2015 Oct;2015.
4. Grogg KL, Jung S, Erickson LA, McClure RF, Dogan A. Primary cutaneous CD4-positive small/medium-sized pleomorphic T-cell lymphoma: a clonal T-cell lymphoproliferative disorder with indolent behavior. Modern Pathology. 2008 Jun;21(6):708-15.
5. Rodd AL, Ververis K, Karagiannis TC. Current and emerging therapeutics for cutaneous T-cell lymphoma: histone deacetylase inhibitors. Lymphoma. 2012 Aug 16;2012.
6. Willemze R. Cutaneous T-cell lymphoma: epidemiology, etiology, and classification. Leukemia & lymphoma. 2003 Nov 1;44(sup3):S49-54.
7. Bekkenk MW, Vermeer MH, Jansen PM, van Marion AM, Canninga-van Dijk MR, Kluin PM, Geerts ML, Meijer CJ, Willemze R. Peripheral T-cell lymphomas unspecified presenting in the skin: analysis of prognostic factors in a group of 82 patients. Blood. 2003 Sep 15;102(6):2213-9.
8. Geller S, Myskowski PL, Pulitzer M, Horwitz SM, Moskowitz AJ. Cutaneous T-cell lymphoma (CTCL), rare subtypes: five case presentations and review of the literature. Chinese clinical oncology. 2018 Nov 21;8(1):5-.
9. Willemze R, JLM Meijer C. Classification of cutaneous T-cell lymphoma: from Alibert to WHO-EORTC. Journal of cutaneous pathology. 2006 Feb;33:18-26.
10. Willemze R, Jaffe ES, Burg G, Cerroni L, Berti E, Swerdlow SH, Ralfkiaer E, Chimenti S, Diaz-Perez JL, Duncan LM, Grange F. WHO-EORTC classification for cutaneous lymphomas. Blood. 2005 May 15;105(10):3768-85.Gallamini A, Stelitano C, Calvi R, Bellei M, Mattei D, Vitolo U, Morabito F, Martelli M, Brusamolino E, Iannitto E, Zaja F. Peripheral T-cell lymphoma unspecified (PTCL-U): a new prognostic model from a retrospective multicentric clinical study. Blood. 2004 Apr 1;103(7):2474-9.
11. Savage KJ. Aggressive peripheral T-cell lymphomas (specified and unspecified types). ASH Education Program Book. 2005;2005(1):267-77.

12. Kummer JA, Vermeer MH, Dukers D, Meijer CJ, Willemze R. Most primary cutaneous CD30-positive lymphoproliferative disorders have a CD4-positive cytotoxic T-cell phenotype. Journal of investigative dermatology. 1997 Nov 1;109(5):636-40.
13. Bekkenk MW, Vermeer MH, Jansen PM, van Marion AM, Canninga-van Dijk MR, Kluin PM, Geerts ML, Meijer CJ, Willemze R. Peripheral T-cell lymphomas unspecified presenting in the skin: analysis of prognostic factors in a group of 82 patients. Blood. 2003 Sep 15;102(6):2213-9.
14. Dummer R, Vermeer MH, Scarisbrick JJ, Kim YH, Stonesifer C, Tensen CP, Geskin LJ, Quaglino P, Ramelyte E. Cutaneous T cell lymphoma. Nature Reviews Disease Primers. 2021 Aug 26;7(1):1-22.
15. Siegel RS, Pandolfino T, Guitart J, Rosen S, Kuzel TM. Primary cutaneous T-cell lymphoma: review and current concepts. Journal of Clinical Oncology. 2000 Aug 15;18(15):2908-25.

SP-33

Case Report of Pelvic Tuberculosis

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Abstract: Pelvic tuberculosis in females is a chronic disease with low grade symptoms. The commonly affected part in genital tuberculosis in females are the fallopian tubes, followed by endometrial involvement, thus causing infertility among affected females. Tuberculosis continues to be a major health problem throughout the world affecting about 9.4 million people annually with about two million deaths. Over 95 % of new TB cases and deaths occur in developing countries with India and China together accounting for 40 % of the world's TB burden. Co-infection with human immunodeficiency virus (HIV), more liberal immigration from high risk to low risk areas due to globalization has been responsible for increased incidence all over the world. Multidrug resistant (MDR) and extreme-drug resistant TB (XDR), usually caused by poor case management, are a cause of serious concern. The female genital tuberculosis is of higher incidence in developing countries. Early diagnosis and prompt treatment of genital TB can be helpful, especially in nulligravida females. This article says the clinical presentation, diagnosis with investigations and laparoscopy and the management for genital tuberculosis. Here is a case report on pelvic tuberculosis, where a woman presents with menstrual irregularities with lower abdominal pain, evaluated and treated for the same.

Keywords: Pelvic, menstrual irregularities, Mycobacterium.

1. INTRODUCTION

Tuberculosis (TB) is a contagious disease, caused by Mycobacterium tuberculosis. TB is the most common infectious disease-causing death worldwide after human immunodeficiency virus. Countries in Asia like India, Pakistan the prevalence of tuberculosis is quite high.[1, 2]. TB cases worldwide there were an estimated 1.4 million TB deaths in a year. The most common form is pulmonary TB, but it can also affect other parts of body.[3,4] Genital TB in females is an important disease, where the infected women face problems like menstrual irregularities, pregnancy loss, morbidity, short- and long-term sequelae.[5,6] Symptoms of GTB are usually non-specific, for this the prevalence of GTB is less than expected. In this form of infection, fallopian tubes (95-100%), endometrium (50-60%) and ovaries are the most affected areas.[7,8]. In 50% of genital tuberculosis cases menstrual function can remain normal. Among the menstrual abnormalities, oligomenorrhea and amenorrhea are commonly seen, in 19% cases menorrhagia can also present. Dysmenorrhea is very rare. Tuberculosis bacilli reach the female genital tract mainly by hematogenous spread. The fallopian tubes, ovaries and endometrium are affected mostly.[9, 10] Diagnosis of genital tuberculosis can be made by several tests like tuberculin tests, culture, histopathology, hysterosalpingogram and nucleic amplification. The mainstay of treatment is multidrug treatment.

2. CASE REPORT

A 37 years old parous woman presented with lower abdominal pain and menstrual irregularities for 6 months consulted outside, since the symptoms were persistent came to OBG OPD at Sree Balaji Medical College and Hospital for further treatment.

2.1 Presenting Complaints

Patient gave a history of weight loss of 4 to 5 kgs.
c/o abdominal pain on and off in the past 2 months
no c/o menstrual irregularities, burning micturition, bladder disturbances.

2.2 Menstrual History

Age of menarche: 14 yrs, initially regular cycles,
3/30 days cycle, not associated with clots/pain.
Now in the past 6 months patient had one month of amenorrhea followed by heavy menstrual bleeding, associated with pain.

2.3 Marital History

Married since 15 yrs, non-consanguineous marriage
Medical history:
Not a known case of diabetes, hypertension, bronchial asthma, tuberculosis, epilepsy, heart disease.
No history of any previous surgery except incision and drainage; no history of any previous blood transfusion.

2.4 Family History

No history of any medical disorder in family.

2.5 Examination

On clinical examination Patient was anemic and abdominal examination revealed tenderness over right and left lumbar region and in paraumbilical region.

2.6 Investigations

All basic investigations were done and ultrasound whole abdomen report showed left mild proximal hydrouretronephrosis, uterus and bilateral ovaries were normal.

2.7 Special Investigations

CECT Abdomen showed bilateral hydrosalphinx, 1. Tubular cystic lesion – 3.5x2.1 cm in right adnexa and 2. Small elongated cystic lesion – 3.0x2.0 cm in left adnexa. ESR – 60, CA125 was 50 mg/dL. Patient was advised for Mantoux test suspecting Tuberculosis in view of bilateral hydrosalphinx. Mantoux test came positive (14 mm), pulmonologist advise taken for ruling out primary foci in lungs; CT Chest done showed subpleural fibrosis, minimal septal and subpleural thickening noted in bilateral lung fields with no active primary foci.

2.8 Procedure

Patient was counselled for laparoscopy for evaluation of pelvic organ tuberculosis and was proceeded for the same. Laparoscopically abdomen and pelvis was approached, over visceral and parietal peritoneum a small white lesions were noted, omental sampling was taken and sent for histopathological study, inview of more adhesions abdomen and pelvis were not studied completely. The histopathological study of omentum showed granulomatous inflammatory necrosis. Thus diagnosed to have abdominal and pelvic tuberculosis and patient was referred to pulmonologist and started on Anti tubercular treatment without any further delay.

2.9 Treatment

Patient was followed up after starting antitubercular treatment, she was improving symptomatically. Ultrasound pelvis done after 3 months on starting ATT revealed decrease in size of bilateral hydrosalphinx (cystic lesion of 2.4x1.3 cm in right adnexa and cystic lesion of 2x1.5 cm in left adnexa), uterine endometrium normal and bilateral ovaries normal.

3. DISCUSSION

Genital tuberculosis cases maybe symptomatic or asymptomatic. The symptoms of tuberculosis can overlap with other pelvic diseases.[11,12]. Mostly young women are diagnosed to have genital TB during the workup of infertility. Female GTB is usually a secondary complication of pulmonary or extrapulmonary TB forms located other than the genital tract.[13, 14]. The patients suffering from genital tuberculosis are usually asymptomatic and may go undiscovered. Worldwide genital tuberculosis is found to cause 5-10% of women infertility.[15] Extapulmonary tuberculosis is more common HIV positive patients. Increasing prevalence in HIV infection with tuberculosis is a major factor in the tuberculosis epidemic particularly in Africa and Asia.

4. CONCLUSION

In our case, since the patient presented with symptoms, patient was evaluated clinically with investigations and laparoscopy, by HPE report can conclude pelvic tuberculosis. Thus, the correlation of clinical symptoms and confirmation by diagnostic laparoscopy will be helpful for prompt diagnosis and treatment of female pelvic tuberculosis.

5. CONFLICT OF INTEREST

Conflict of interest declared none.

6. ACKNOWLEDGEMENT

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7. FUNDING SOURCE: None

8. REFERENCES

1. World Health Organization. Global tuberculosis report 2013. World Health Organization; 2013.
2. Akbulut S, Arikanoglu Z, Basbug M. Tubercular tubo-ovarian cystic mass mimicking acute appendicitis: a case report. Journal of medical case reports. 2011 Dec;5(1):1-4.
3. Ghosh K, Chowdhury JR. Tuberculosis and female reproductive health. Journal of postgraduate medicine. 2011 Oct 1;57(4):307.

4. Thangappah RB, Paramasivan CN, Narayanan S. Evaluating PCR, culture & histopathology in the diagnosis of female genital tuberculosis. The Indian journal of medical research. 2011 Jul;134(1):40.
5. Sachan R, Patel ML, Gupta P, Verma AK. Genital tuberculosis with variable presentation: a series of three cases. Case Reports. 2012 Aug 27;2012.
6. Eftekhar M, Pourmasumi S, Sabeti P, Aflatoonian A, Sheikhha MH. Mycobacterium tuberculosis infection in women with unexplained infertility. International Journal of Reproductive BioMedicine. 2015 Dec;13(12):749.
7. Jindal UN. An algorithmic approach to female genital tuberculosis causing infertility. *Int J Tuberc Lung Dis.* 2006; 10:1045–1050.
8. Parikh FR, Nadkarni SG, Kamat SA, et al. Genital tuberculosis—a major pelvic factor causing infertility in Indian women. *Fertil Steril.* 1997;67:497–500.
9. Gupta N, Sharma JB, Mittal S, et al. Genital tuberculosis in Indian infertility patients. *Int J Gynecol Obstet.* 2007;97:135–138.
10. Singh N, Sumana G, Mittal S. Genital tuberculosis: a leading cause for infertility in women seeking assisted conception in North India. *Arch Gynecol Obstet.* 2008;278:325–327.
11. Neonakis IK, Gitti Z, Krambovitis E, Spandidos DA. Molecular diagnostic tools in mycobacteriology. *J Microbiol Methods.* 2008;75:1–11.
12. Duggal S, Duggal N, Hans C, Mahajan RK. Female genital TB and HIV co-infection. *Indian J Med Microbiol.* 2009;27:361–363.
13. Sharma JB, Pushparaj M, Gupta N, et al. Genital tuberculosis: an important cause of Asherman's syndrome in India. *Arch Gynecol Obstet.* 2008;277:37–41
14. Sharma JB, Malhotra M, Pundir P, et al. Cervical tuberculosis masquerading as cervical carcinoma: a rare case. *J Obstet Gynaecol Ind.* 2001;51:184.
15. Sharma JB, Sharma K, Sarin U. Tuberculosis: a rare cause of rectovaginal fistula in a young girl. *J Obstet Gynaecol Ind.* 2001;51:176.

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Beta Thalassemia Major Of Late Diagnosis In Pregnancy: An Atypical Observation And Successful Pregnancy Outcome

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Abstract: B-thalassemia is the most common single gene disorder in India. B-thalassemia is an autosomal recessive disease characterized by defective hemoglobin synthesis. It is an inherited defect of beta-globin chain of hemoglobin. B-thalassemia is having it geographical prevalence in Mediterranean, Asiana, Middle eastern countries. The clinical manifestations of B-thalassemia are seen between 6months to 2 years of life. Pregnancy with thalassemia is high risk for both mother and the fetus, and a successful outcome is achieved with the help of continuous pre-conception, antenatal, postpartum management by obstetrician and haemato-oncologist experts. Here we present a case report of B-thalassemia major discovered late in pregnancy.

Keywords: Beta Thalassemia, B globin Gene HbA2, NST- Nonstress test, hypo fertility, spontaneous conception

1. INTRODUCTION

Hemoglobinopathies are among the most prevalent inherited diseases: about 7% of the world's population is a carrier, and 300,000–500,000 children are born each year with a severe haemoglobin abnormality.[1,2] Thalassemia major, thalassemia intermedia, and thalassemia minor are the three main types. Point mutations or, more rarely, deletions in the beta globin gene on chromosome 11 cause diminished (beta+) or absence (beta0) synthesis of the beta chains of haemoglobin, resulting in beta-thalassemias (Hb). Individuals with thalassemia major are usually diagnosed within the first two years of life. Infants who are affected do not thrive and become paler.[3,4] Feeding difficulties, diarrhoea, irritability, recurring fevers, and increasing abdominal enlargement caused by Spleen and liver enlargement are possible side effects and require regular RBC transfusions to stay alive.[5,6] A multidisciplinary therapeutic team approach to thalassemia management is ideal, and it should ideally be done at a holistic thalassemia care centre with a team of paediatric haematologists, paediatricians, transfusion medicine specialists, endocrinologists, psychologists, and social workers, as well as the support of a well-equipped blood bank. In our patient presents a rare scenario of Beta thalasemia diagnosed in pregnancy.[7,8]. As existing treatments improve, particularly in the control of iron deposits and the survival rate increases, the number of pregnancies among thalassemic women will continue to rise.[9,10] For women suffering from such an illness, pregnancy is now a viable option. Despite the possibility of multiple difficulties, careful supervision by both expert obstetricians and haematologists can lead to a successful pregnancy which is rare.[11]

2. CASE REPORT

2.1 Presenting Complaint

A 20-year-old woman married for 9 months Primigravida from chrompet with socioeconomic class V 1st visit to SBMCH OPD at 23 weeks and 2days, she is booked and immunized outside. She came here for routine antenatal checkup and wanted to follow up delivery at SBMCH. She had a spontaneous conception and pregnancy confirmed by dating scan. No further scans were done.

2.2 Medical History

Patient gives no significant past medical or surgical history. Patient has been on irregular follow up and this is her second antenatal visit to SBMCH.

2.3 Family Hisotry

No history of death of infants in the family needing frequent blood transfusions.

2.4 Special Tests And Investigations

On Reviewing her reports, at 7 weeks, her baseline HB- 8.1g/dl, for which patient was advised iron rich diet, was counseled for further evaluation, patient did not go for any follow up visit. At SBMCH, we sent all baseline investigations, revealed hb- 8.3 g/dl, Pcv-24.9 %, MCV- 25. Peripheral smear revealed, predominantly microcytic hypochromic RBC's, with anisopoiklocytosis, showing elongated cells, pencil shaped cells. Medicine opinion obtained and advised parenteral iron injections and Vit b12 injections. ECG and maternal echo done -normal. cardiology opinion obtained, suggested stable cardiac status. Her hb deficit - 1282.84 mg, 7 doses of injection iron sucrose infusion was given, and 2 doses of Inj. Vitb12 1000mcg weekly IM given.patient was screened with 2hr -75g of OGCT for GDM value - 112mg /dl After 3 weeks, patient reviewed at 26weeks with hb report of 6.3g/dl, MCV- 23fl, MCHC- 29 g/dl, serum ferritin- 13 ng/dl.

2.5 Diagnosis

In view of refractory anemia, after ruling out other causes of anemia, hematologist opinion was obtained in view of suspected thalassemia, HB electrophoresis was done showed, HbA2 – (C to c) – 4.1% and HbA2-95.9%, suggestive of B-thalassemia Major. Husband was screened for Thalassemia showed no evidence of thalassemia.

2.6 Treatment Plan

3unit of PRBC transfusion was done and repeat Hb -9.4 g/dl. Patient was advised to stop oral iron. USG whole abdomen done revealed mild hepatosplenomegaly. Patient was followed up with weekly antenatal visits. 1 course of antenatal steroids were covered at 28weeks. USG growth scan was done at 30weeks showed appropriate for growth.

2.7 Prognosis

At 36weeks, patient came with complaints of decreased fetal movements since last night, NST showed reduced beat to beat variability and 2 decelerations upto 90 bpm. Patient taken up Emergency LSCS in view of fetal distress. An alive male baby cried after birth with Apgar score -8/10, 9/10. Baby breast fed within 1 hour of birth. Postnatally 1 course of IV antibiotics covered. Patient discharged with Baby on fourth post op day.

3. DISCUSSION

The diagnosis of Beta thalassemia is often made in childhood, its diagnosis in adults is Rare and exceptional. The diagnosis of Beta thalassemia major often made before 2 years of life with severe anemia needing blood transfusions and iron chelation therapy lifelong.[1] Early revelation is classic between 6 and 24 months. The late discovery in our patient who was previously asymptomatic remains Exceptional. Spontaneous conception is rare in beta thalassemia major and reveals uniqueness in our case report.[12]. Beta thalassemia results from inheritance of two defective B globin genes. There are more than 200 thalassemic mutations reported. Heterozygous carriers of Beta thalassemia are asymptomatic and only diagnosed with altered lab investigations.[13] The globin chain synthesis reduction leads to unbalanced Beta/Alpha globin chain productions where the Alpha chains in abundance form erythrocyte inclusions and lead to hemolysis due to ineffective erythropoiesis.[4]. The evolution towards this major form has been reported in studies attributing to acquired paternal uniparental isodisomy of 11p15.[14] chromosomal region. Pregnancy results in physiological hemodilution and increased nutritional requirements for fetus thus aggravating pre - existing anemia to transfusion dependent anemia. the classical morphological features of Beta thalassemia are not reported in our patient.[5] In beta thalassemia major classically severe form hypochromic microcytic anemia which is also an exclusion in our patient.[15]. Because thalassemia major necessitates frequent blood transfusions, there is iron overload, which leads to iron accumulation in the brain and pituitary, resulting in reproductive axis failure and delayed puberty, sexual development, and infertility. Many patients with -thalassemia major have been able to live through adolescence thanks to advancements in treatment. Although assisted reproductive procedures and breakthroughs in treating iron overload have increased the number of successful pregnancies in such people, iron overload-related hypogonadism might decrease fertility[11]. Spontaneous pregnancy in Beta -thalassemia major is rare in this patient with no past treatment of the pathology.[7] As hypo fertility is a common feature of Beta thalassemia which occurs due to iron deposits in hypothalamus-pituitary axis. Studies like singer et al in 2011 suggested that ovarian reserve is preserved in majority of thalassemia major patients implicating the possibility of spontaneous pregnancy.[8] Pregnancy complications in Beta thalassemia major are abortions; gestational hypertension, fetal growth restriction, thrombosis, and postpartum hemorrhage were not observed in our patient.[9]

4. CONCLUSION

This is an atypical case of symptomatic Beta-thalassemia major with transfusion need discovered in pregnancy. Because thalassemia major necessitates frequent blood transfusions, there is iron overload, which leads to iron accumulation in the brain and pituitary, resulting in reproductive axis failure and delayed puberty, sexual development, and infertility. Many patients with -thalassemia major have been able to live through adolescence thanks to advancements in treatment. Although assisted reproductive procedures and breakthroughs in treating iron overload have increased the number of successful pregnancies in such people, iron overload-related hypogonadism might decrease fertility. Women with thalasemia major have a higher risk of heart failure, alloimmunization, viral infections, thrombosis, osteoporosis, new endocrinopathies, diabetes mellitus, hypoparathyroidism, and hypothyroidism. If Hb Barts hydrops is suspected in the antenatal stage, maternal problems include early onset severe preeclampsia, delivery of a highly hydropic foetus and placenta, and postpartum haemorrhage. However, with the advent use of iron chelation therapy the rate of pregnancy in thalassemia major is possible and increasing. with appropriate maternal and fetal monitoring in antenatal period will reduce maternal and neonatal morbidity and mortality. The challenge remains the early diagnosis of non-transfusion dependent forms in at risk families and pregnancy planning to reduce maternal and fetal morbidity and mortality.

5. CONFLICT OF INTEREST

Conflict of interest declared none.

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8. REFERENCES

1. Aessopos A, Karabatsos F, Farmakis D, et al. Pregnancy in patients with well-treated β-thalassemia: outcome for mothers and newborn infants. Am J Obstet Gynecol. 1999; 180:360–365. Daskalakis GJ, Papageorgiou IS, Antsaklis AJ, Michalas SK. Pregnancy and homozygous beta thalassaemia major. Br J ObstetGynaecol. 1998; 105:1028–1032.
2. Kumar RM, Rizk DE, Khuranna A. Beta-thalassemia major and successful pregnancy. J Reprod Med. 1997; 74:127–131.
3. Tampakoudis P, Tsatalas C, Mamopoulos M. Transfusion-dependent homozygous β-thalassaemia major: successful pregnancy in five cases. Eur J ObstetGynecolReprod Biol. 1997; 74:127–131.
4. Origa R, Piga A, Quarta G. Pregnancy and β-thalassemia: an Italian multicenter experience. Haematologica. 2010; 95:376–381.
5. Avila WS, Rossi EG, Ramires JA. Pregnancy in patients with heart disease: experience with 1,000 cases. ClinCardiol. 2003; 26:135–142.
6. Voskaridou E, Balassopoulou A, Boutou E. Pregnancy in β-thalassemia intermedia: 20-year experience of a Greek thalassemia center. Eur J Haematol. 2014; 93:492–499.
7. De Sanctis V, Soliman AT, Elsedfy H. Growth and endocrine disorders in thalassemia: the international network on endocrine complications in thalassemia (I-CET) position statement and guidelines. Indian J EndocrinolMetab. 2013; 17:8–18.
8. De Sanctis V. Growth and puberty in thalassaemia. Horm Res. 2002; 58:72–79.
9. Galanello R, Origa R. Beta-thalassemia. Orphanet J Rare Dis. 2010; 5: 11. Published online 2010 May 21. doi: 10.1186/1750-1172-5-11.
10. Nadkarni A, Caraskar AC, Krishnamoorty R, Lu CY, Ghosh K, Colah R. Molecular pathogenesis and clinical variability of β thalassemia syndrome among Indians. Am J Hematol 2001; 68: 75–80.
11. Yadav P, Kumari S, Kumar V. Successful Outcome of Pregnancy in β-thalassemia Major Individual. Int J Sci Stud 2017;5(1):252-254
12. MARTIN, KATHLEEN. "Successful pregnancy in β- thalassaemia major." Journal of Paediatrics and Child Health 19.3 (1983): 182-183.
13. Saxena R, Banerjee T, Aniyery RB. Thalassemia and its management in pregnancy. world J anemia 2017 ;1(1): 5-17.
14. Nassar AH, Naja M, Cesaretti C, Eprassi B, Cappellini MD, Taher A. Pregnancy outcome in patients with beta-thalassemia intermedia at two tertiary care centers, in Beirut and Milan. Haematologica 2010; 93 (10): 1586–1587
15. Guide lines for the clinical management of thalassemia. 2nd ed. Nicosia Cyprus: Thalassemia International Federation; 2009. World Bank 2006, reported joint WHO – March of time meeting 2006-Quoted;

SP-35

A Rare Case Of Placenta Accreta In A Primigravida

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Abstract: A placenta accreta is an abnormally adherent placenta, which is abnormally attached to myometrium. With every rise in caesarean sections rate, its incidence increases with it. Among its consequences, hemorrhage during birth is the first and most serious complication, which is lethal and risky to the mother, followed by uterine rupture and invasion into surrounding organs such as the bladder and rectum. Placenta accreta in an unscarred uterus that occurs very rarely. An interesting case of placenta accreta in an unscarred uterus was accidently found during an emergency caesarean operation, as we report. At 39 weeks, a 25-year-old female primigravida, unbooked patient, with no known risk factors and no previous documented reports presented to Sree Balaji medical college. She had no history of scarred uterus.

Keywords: Placenta Accreta, Adherent Placenta, Uterine Rupture, Unscarred Uterus.

1. INTRODUCTION

Morbidly adherent placenta (MAP) is one of the risk factors for maternal death, accounting for 15% of all cases presenting with obstetrical haemorrhage and requiring blood transfusion. MAP is generally encountered in high-risk women with placenta previa, Elevated AFP levels in the second trimester and free Beta-hCG levels more than 2.5 multiples of the median, two or more caesarean deliveries and previous scarred uterus, uterine curettage previously performed, maternal age of 35 years, High gravidity and multiparity.[1,2] Placental accrete vera accounts for 75–80 percent of all placental accreta. The other two forms of MAP are less prevalent and more severe.[3,4] Placenta accreta is associated with complications such as Local organ damage and damage to neurovascular structures in the retroperitoneum and lateral pelvic walls from placental implantation and removal, post-operative bleeding requiring repeated surgery, amniotic fluid embolism, and coagulation complications due to massive bleeding and transfusion of blood/blood products are all associated with placenta accrete, which can lead to infection, multiorgan failure, and maternal death.[5] Due to a deficiency in the decidua basalis layer, the placental trophoblast invades beyond the Nita Buch's layer of the endometrium, resulting in placenta accreta. When compared to individuals who had an unscarred uterus, those who had a previous caesarean surgery had a higher incidence of placenta previa (1.31 percent) (0.75 percent).[6] The primary cause of morbidity in this syndrome is considerable blood loss during delivery. Pregnancies are also thought to be complicated by placenta accreta, which leads to an increased risk of invasion into bladder and rectum and uterine rupture. In unscarred uterus, placenta accreta occurs quite rarely.[7] we describe a case of placenta accreta detected during an emergency caesarean operation in an unscarred uterus.

2. CASE SCENARIO

2.1 Presenting Complaint

A 25-year-old primigravida admitted to the labour ward for delivery with complaints of leaking p/v for 2 hours.

2.2 Medical History

She had no previous medical or surgical history, and no previous medical records.

2.3 Family History

She had no significant family history and no history of peripartum hysterectomy in the family.

2.4 Observation

On examination, fundal height corresponds to term size, uterine contractions are present (2'/15-20"/10), FHS +, per speculum examination confirmed leaking present. per vaginal examination revealed cervix is soft anterior cervix, 50% effaced, 2cm dilated, presentation- cephalic, vertex at brim, pelvis adequate, membrane present.

2.5 Special Test

The obstetric ultrasound revealed a single intrauterine live pregnancy at 38 weeks and 4 days, with a normal and regular fetal heart rate, a cephalic presentation, fetal parameters appropriate for gestational age, estimated fetal weight- 3120g, amniotic fluid decreased in relation to membrane rupture, and placental location –fundal. CTG revealed persistent fetal bradycardia during labour, necessitating an emergency caesarean section.

2.6 Diagnosis

The manual removal of the placenta was tried, however the delivery of the placenta failed. Because the plane of separation between the uterine wall and the placenta could not be recognized, the diagnosis of placenta accreta was made.

2.7 Treatment Plan

We chose a conservative approach, removing the placenta with vascular closure of bilateral uterine arteries and confirming hemostasis.

2.8 Prognosis

Because of the patient's continued blood loss (about 1000ml), 1-unit PRBC was transfused intraoperatively. Post-operatively, patient was kept in high dependency unit for monitoring and transferred to ward after 24hrs. Patient discharged after 10 days of hospital stay with her baby.

3. DISCUSSION

Placenta accreta diagnosis has been rising in recent times. Due to damage or congenital deficiency of the decidua basalis and increasing incidence of uterine surgeries, the likelihood of placenta previa and accreta is increasing. With One previous caesarean delivery, the risk of placenta previa is twofold in subsequent pregnancies, increasing the incidence from 0.38 percent to 0.63 percent. When the placenta is previa, placenta accreta becomes more common; its occurrence is estimated to be around 10%.[8] The cause of placenta accreta is unknown, however risk factors exist. A quantitative or qualitative deficit in the decidua basalis provides a region vulnerable to uncontrolled trophoblast invasion, resulting in placenta accreta. Prior caesarean delivery, uterine instrumentation, intrauterine scarring, placenta previa, smoking, maternal age over 35, grand multiparity, recurrent miscarriage, and Myomectomy for fibroid uterus in infertile individuals are all risk factors for placenta accreta.[9,10] The placenta accreta is usually caused by a combination of causes. Placenta accreta occurs when placental villi invade the surface of the myometrium; Placenta increta occurs when placental villi extend into the myometrium; and Placenta percreta occurs when placental villi penetrate through the myometrium to the uterine serosa and may invade nearby organs such as the bladder. In contrast to numerous instances, this woman had an unscarred uterus and no known risk factors for placenta accrete.[11,12] Placenta accreta is a silent condition that occurs during pregnancy. Ultrasound, which has become the predominant modality of screening for women at risk of placenta accreta, and magnetic resonance imaging are used to confirm the diagnosis. When it comes to spotting placenta accreta, ultrasound is the first line of defense. The presence of placental gaps, the lack of a hypoechoic border between the placenta and myometrium, and a disruption of the hyperechoic zone at the interface of the uterine serosa and the bladder are all typical ultrasonography indicators. MRI is not yet recommended as a first-line screening method. When utilized as a second line examination after ultrasound suspicion of placenta accreta, it was found to have a sensitivity and specificity of 88 percent and 100 percent, respectively, in a recent study. An aberrant bulging of the lower segment, the heterogenicity of the placental signal intensity in T2, and black intraplacental bands in T2 are among the diagnostic criteria indicated. The diagnosis is occasionally made at the moment of delivery, as in our case, when the placenta was difficult to remove. Some differential diagnoses, such as a preserved imprisoned placenta, choriocarcinoma, and others, can be problematic during delivery.[13] The "gold standard" for management is a caesarean section at 34 weeks of pregnancy with hysterectomy, which includes performing hysterectomy after the birth of the child without an attempt at artificial delivery when the diagnosis of placenta accreta has been made or after an attempt at artificial delivery when the diagnosis of placenta accreta has been made intraoperatively. Although this strategy may lower maternal morbidity, it also causes people to lose their fertility. In the absence of hemorrhage, conservative treatment with the placenta left can be explored In the case of mild bleeding, uterine arterial ligation with uterine padding (in the case of a caesarean section) or arterial embolization (in the case of a vaginal delivery) can be performed, but in the event of failure or severe hemorrhage, a hysterectomy must be performed right away. The treatment has been conservative.[14] In carefully selected instances with hemodynamic stability, normal coagulation status, and a goal to preserve fertility, conservative therapy may be attempted. Prior counselling on the hazards associated with conservative management is required. Methotrexate administration, uterine artery ligation, internal iliac artery ligation, uterine artery embolization, and radiofrequency ablation are just a few of the choices accessible. The importance of close monitoring, frequent tests, the risk of postpartum bleeding, infection, treatment failure, and, as a result, the likelihood of hysterectomy, cannot be overstated.[11] To keep the uterus adequately constricted and limit the risk of postpartum haemorrhage, continued uterotonic assistance is often recommended. Methotrexate is a folate antagonist that is effective against dividing trophoblasts because it targets quickly dividing cells.[12] Several authors have successfully described it (leaving all or part of placenta in place). Conservative treatment failures have also been the topic of case studies reporting the incidence of subsequent bleeding after caesarean section, which can be a life-threatening situation for the patient. A retrospective analysis of 50 instances with placenta accreta found that 26 of them responded well to conservative therapy (placenta left in place during caesarean section).[14] Only five individuals (19%) required a secondary hysterectomy. This treatment approach appears to be appealing to people who still want to get pregnant. Conservative treatment was successfully carried out in this situation.[3]

4. CONCLUSION

Placenta accreta is a condition that can cause major hemorrhage problems during pregnancy and after delivery. The diagnosis is occasionally made at the moment of delivery, as in our case, when the placenta is difficult to detach. This should motivate us to ask more questions of our patients and to search the ultrasound criteria for placenta accreta in them more thoroughly. As a result, the ultrasound report for these individuals should specifically state it. The mortality and morbidity associated with this

condition can be controlled with adequate care from a multidisciplinary team. In order to refer such patients to appropriate therapy, antenatal diagnosis is required.The occurrence of a morbidly adherent placenta in a patient with no preexisting risk factors is extremely unusual. In these circumstances, fertility preservation is a top priority, hence being vigilant is more important.

5. CONFLICT OF INTEREST

Conflict of interest declared none.

6. ACKNOWLEDGEMENT

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8. REFERENCES

1. Giuri M. Amazon eröffnet Forschungszentrum. Lebensmittel Zeitung. 2021;73(34):29-29.
2. Fox H. Placenta Accreta, 1945–1969. Obstetrical & Gynecological Survey. 1972;27(7):475-490.
3. Fujisaki M, Furukawa S, Maki Y, Oohashi M, Doi K, Sameshima H. Maternal morbidity in women with placenta previa managed with prediction of morbidly adherent placenta by ultrasonography. J Pregnancy 2017; 2017:8318751.
4. Hung TH, Shau WY, Hsieh CC, Chiu TH, Hsu JJ, Hsieh TT. Risk factors for placenta accrete. Obstet Gynecol 1999; 93:545-50.
5. Wu S, Kocherginsky M, Hibbard JU. Abnormal placentation twentyyear analysis. Am J Obstet Gynecol 2005; 192:1458-61.
6. Jacques SM, Qureshi F, Trent VS, Ramirez NC. Placenta accreta mild cases diagnosed by placental examination. Int J Gynecol Pathol 1996; 15:28-33.
7. Chandraharan E, Rao S, Belli AM, Arulkumaran S. The triple-P procedure as a conservative surgical alternative to peripartum hysterectomy for placenta percreta. Int J Gynaecol Obstet 2012; 117:191-
8. Konijeti R, Rajfer J, Askari A. Placenta percreta and the urologist. Reviews in Urology. 2009;11(3):173-176.
9. Attieh E, AbboudJ, ChalhoubS, Riachi M. Placenta percreta et rupture utérine: à propos de deux cas. J Gynécol Obstét Biol Reprod.1993;22(6):649-652.
10. Timor-Tritsch. Early placenta accreta and cesarean section scar pregnancy: a review. Am J Obstet Gynecol 2012
11. Kuhite H, Mirji S, Shingatgeri S, Shinde G. A Rare Case of Morbidly Adherent Placenta in a Primigravida. J South Asian Feder Obst Gynae 2018;10(Suppl 1):351-354.
12. Cahill, Alison G. "Placenta accreta spectrum." American journal of obstetrics and gynecology 219.6 (2018): B2-B16.
13. Esakoff, T. F., et al. "Diagnosis and morbidity of placenta accreta." Ultrasound in Obstetrics & Gynecology 37.3 (2011): 324-327.
14. Sentilhes, Loil. "Fertility and pregnancy outcomes following conservative treatment for placenta accreta." Human reproduction 25.11 (2010): 2803-2810.

SP-36

Remembering The Pioneers In Epidemiology And Their Contributions To Public Health

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Abstract: This article describes the developments in the field of Epidemiology from the ancient times. Eminent people in different time periods had made greater contributions in improvement of scientific knowledge and evidence-based medicine. The evolution of epidemiology was too slow in the early ages. In the last few centuries, medicine and epidemiology have shown tremendous advancements in treatment of diseases, preventive measures and health promotion. The pioneers in Epidemiology whose works need to be highly appreciated are Hippocrates, John Graunt, James Lind, Edward Jenner, Ignaz Semmelweis and John Snow. The contribution of each deserves special mention as they changed the way diseases are looked at and modified the thinking of humans regarding disease causation and prevention.

Keywords: Public health, advancement, innovators.

1. INTRODUCTION

Epidemiology is the foundation for Preventive Medicine both in the traditional and modern periods. Although the origin of Epidemiology dates back to ancient days, it made a very sluggish process till the early 20th Century. The evolution of epidemiology was rapid in the last few decades and it has made a great contribution to the medical knowledge.[1] Epidemiology was initially thought of as a branch of medical science which treats epidemics. Later it extended to cover all the diseases, both infectious and non-infectious.[2] Brian McMahon in the year 1960 broadened the concept of epidemiology as study of distribution and determinants of disease frequency in man. In the present era, the widely accepted definition of epidemiology was given by John M. Last which states "The study of the distribution and determinants of health-related states or events in specified populations, and the application of this study to control of health problems" .[2,3]. The field of epidemiology has played a major role in identifying the etiopathogenesis of communicable and non-communicable diseases, their progress and control measures. The evolution on theories of disease causation and the discovery of micro-organisms established the epidemiology of Communicable Diseases. This lead to the development of preventive measures including vaccination.[1] The Pioneers in the 16th to 19th Century such as John Snow, James Lind and Ignaz Semmeilweis studied various diseases and their causative factors and suggested preventive measures to bring down the disease transmission. The concept of disease causation started with a theory of "pandora box" and evolved over a period of time with discovery of bacteria.[2]

2. EARLY CONCEPTS OF DISEASE - THE PANDORA BOX

In the ancient period, men relied on hunting for their living. They lived in groups and moved constantly, hence the chances of overcrowding; water contamination and waste accumulation were very rare. The primitive people believed that the diseases occurred due to natural spirits. According to Greek mythology, the illnesses spread into the world when Pandora opened the box in which Zeus has force locked all the diseases, sorrows and crimes that affected the humans.[4] For a long time, people believed that the occurrence of diseases is because of curse from the God. With agricultural revolution, the food supply to man became more stabilized and the population started expanding. People started staying in places for a long time and the migration slowly reduced. With domestication of animals for the purpose of agriculture, the chances of disease transmission increased.[5]

3. PIONEERS IN EPIDEMIOLOGY

3.1 *Hippocrates And His Corpus*

For many centuries, explanations for diseases were based on religion, superstition and myths. The first significant contribution in the field of epidemiology was the "*Hippocratic Corpus*". Hippocrates (460-377 BC) stressed the fact that diseases occur due to imbalance of man with environment, imbalances in diet, personal behaviors in his book "*On airs, waters and places*". Hippocrates initiated the rational thinking for understanding health and disease among men. He also coined the terminologies "epidemic" and "endemic". [4,6] Despite the contributions of the Hippocratic Corpus, the medical and scientific progress was arrested for several centuries. The population increased rapidly, cities and towns became thickly populated, but there was little attention to environment cleanliness, waste disposal and proper sanitation. All these factors promoted theoccurrence of endemic diseases and periodic epidemics.[1]

3.2 John Graunt And Health Statistics

In the 16th century, in London, the parish clerks initiated recording data on deaths. In 1662 John Graunt (1620-1674), a founding member of the Royal Society of London, analysed and summarized the data from these "*Bills of Mortality*" and made a publication entitled "*Natural and Political Observations Mentioned in a Following Index, and Made Upon the Bills of Mortality*."[7]
John Graunt analyzed the data extensively and recorded a number of observations regarding common causes of death (Figure 1), gender and seasonal variation in death rates. He also noted that some diseases had relatively constant death rates, while others had varying death rates. Graunt also estimated population size and rates of population growth, and he was the first to construct a "life table" in order to address the issue of survival from the time of birth. He provided statistical evidences for many theories on disease that existed those days, and also disproved some widespread theories on them[8].

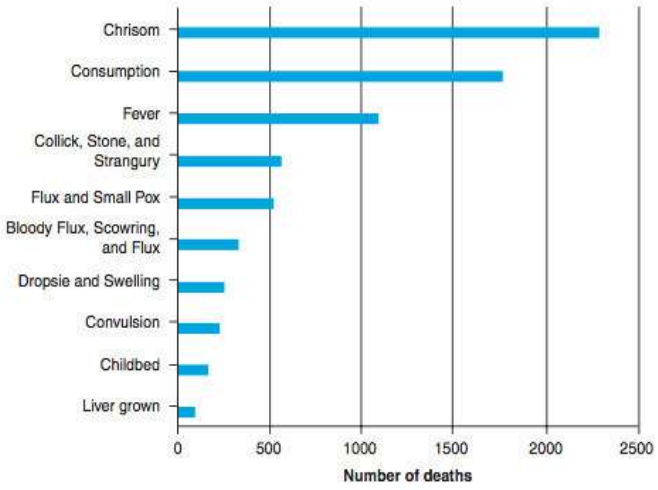

Figure 1: Yearly mortality bill showing the common causes of death [7]

[Chrisom - death of child within one month of baptism; Consumption - Tuberculosis; Strangury - slow and painful discharge of urine; Bloody flux - dysentery; Scowring - Diarrhea; Flux - excessive discharge from the body; Dropsie - edema; Liver grown - enlarged liver] Figure 1 shows the causes of deaths as summarized in the Yearly mortality bill by John Graunt in the 16th century. His publication is one of the hallmark achievements in the field of epidemiology.

3.3 James Lind And Scurvy

A million British seamen died of scurvy in the 18th century when Britain was entangled in a war against France and Spain. James Lind (1716-1794), a Scottish surgeon joined the British Navy as an Apprentice Doctor and later he became the surgeon in-charge of a 50-gun ship involved in watching the English Channel. Nearly after eight weeks in the sea, the sailors developed symptoms of scurvy. James Lind's idea was that scurvy can be controlled by giving acidic foods. He divided the sick men into six pairs and they were provided with six different supplements along with the food - cider, diluted sulfuric acid, vinegar, sea water, purgative mixture and oranges and lemon.[9] This trial was said to be the "*first controlled clinical trial*" in the history of Epidemiology. The pair which took the oranges and lemon showed improvement. This trial happened in a situation where apart from the intervention; other characteristics of the participants remained similar. Later after few years, the British navy made supply of citrus fruits mandatory for the sailors, thereby bringing down the mortality.[9,10]

3.4 Edward Jenner And Small Pox

Though small pox has been eradicated, it took a great toll in the 18th century where lakhs of people died due to the disease. People who survived the disease developed immunity thereby preventing recurrence. The measure which was practiced to prevent small pox infection was "Variolation" in which healthy individuals are injected with materials which was taken from persons affected with smallpox. Due to this procedure, some individuals developed smallpox disease, and few of them died. Edward Jenner (1749-1823) was interested in developing a much safer and good approach to prevent smallpox.[10,11] In the year 1768, Jenner heard a dairy maid talking "I can't get smallpox as I have already had cowpox". After this, Jenner did an observational analysis and noted that women who worked as dairy maids developed a milder form of disease called cowpox. These women were immune to smallpox during the outbreaks. This convinced Jenner that cowpox protects people against smallpox and he tested his hypothesis by administering cowpox material to a child. Weeks later, he inoculated smallpox material into the same child and the child didn't develop the disease. This invention later saved millions of lives of humans and also guided the health community in the path of smallpox eradication. In the year 1980, the World Health Organisation (WHO) declared eradication of smallpox and this is recognised as a great achievement in the field of Preventive Medicine.[11]

3.5 Ignaz Semmelweis And Puerperal Fever

Occurrence of fever in the postpartum women which was known as "childbed fever" those days was an important cause of mortality among women soon after delivery. Ignaz Semmelweis (1818-1865) was the in-charge of the First Obstetrical Clinic in General Hospital, Vienna in July 1846. The cause of childbed fever was not clearly known and many factors like atmospheric

toxins, solar and magnetic influences, impure air were thought of as the reasons behind the fever. There were two obstetric clinics, the first Clinic was staffed by physicians and medical students and the second Clinic by midwives.[12] Semmelweis made a detailed analysis of six years 'death records and found that the death rates of women in the first clinic was much higher than in the second clinic (Table 1). What Semmelweis did was a classical "case control study" and he identified the risk factor as the physicians' improper hand washing practices after doing the autopsies and attending the women in labor clinic. So he made a strong recommendation towards proper hand washing before entering into the obstetric clinic. This practice showed a decline in the death rates in the first clinic which was a strong evidence for his causal hypothesis. For his contribution to prevention of puerperal fever, Semmelweis is known as *"The Savior of Mothers and an Early Pioneer of Antiseptic"*. [12]

Table 1: Mortality rates of the first and second obstetric clinics at Vienna General Hospital between 1841 and 1846 [13]

Year	Mortality rate in First clinic (%)	Mortality rate in Second clinic (%)
1841	7.8	3.5
1842	15.8	7.6
1843	9.0	6.0
1844	8.2	2.3
1845	6.9	2.0
1846	11.4	2.8
1847-48(post intervention)	2.2	2.0

Table 1 shows the comparison of mortality rates in the first and second Obstetric Clinics at Vienna General Hospital during 1841 to 1846. The mortality rates were higher in the first clinic before intervention. After handwashing practices were implemented, the mortality rates drastically reduced.

3.6 John Snow And Cholera

Cholera became a major threat to mankind during 1800s and epidemics happened frequently in England and mortality rates were high. John Snow (1813-1858), a Physician in London, spent several decades studying cholera in a systematic way. People believed that cholera spread through miasmas or through person-person contact. In the year 1854, an epidemic of cholera happened in the Broad street, London. Around 600 people living in and around broad street died of cholera in the month of September. Snow hypothesized that the disease is transmitted through contaminated water. Water supplies to the houses were through Southwark and Vauxhall company and the intake were from highly polluted part of the Thames River. Lambeth company shifted its water intake upstream in the Thames to a less polluted part of the river. Based on Snow's hypothesis, mortality rate of cholera will be lower in people getting water from Lambeth than in people getting water from other companies. He went house to house, counting all deaths from cholera in each house and determined which company supplied water to each house. The analysis showed the death rates were higher among the houses supplied with the polluted water (Table 2). He learnt that majority of victims lived near and obtained water from the broad street pump. He drew a map showing the location of the water pumps, and the location of the victims which were clearly clustered around the pump. This type of map, which marks the location of disease cases, is now referred to as a "**spot map**".[14, 15] Snow based on his observations made a recommendation to remove the handle of broad street water pump and after its implementation, the outbreak rapidly subsided. Snow's way of thinking and handling this cholera epidemic needs appreciation. He proposed a new hypothesis on how the disease was transmitted. He tested hypothesis in a systematic manner and made comparisons between groups of people supplied by water companies. On detailed field work, he provided evidence for a positive association between drinking water from broad street pump and development of cholera. He was also able to suggest and intervention to prevent the spread of disease. This stresses the importance of evidence based practice in preventive medicine.[16]

Table 2: Comparison of mortality rates by source of water supply during cholera outbreak in the year 1854 [14]

Water Company	Death rate per 10,000 houses
Southwark & Vauxhall	315
Lambeth	38
Remaining areas	56

Table 2 shows the difference in mortality rates during a major cholera outbreak in the 19th century. Death rates were significantly higher among houses with water supply from Southwark & Vauxhall Company.

4. CONCLUSION

Pioneers in the area of epidemiology introduced different concepts of disease. For a long time, the predominant interest in epidemiology was the area of infectious diseases. Studies done by the pioneers provided the impetus to the growth of epidemiology and tremendous improvement in epidemiologic methods. Their contributions to epidemiology serve as the foundation of preventive medicine and are worth revisiting. Epidemiology is the cornerstone of Public Health and pioneers such as John Snow, James Lind and John Graunt have laid the foundation for strong epidemiological research and implementation in the community to bring down the prevalence of diseases. The principles of epidemiology were initially restricted to

Communicable diseases research. Later on, the field expanded to include non-communicable diseases, accidents and genetic diseases. In addition to these, the world is also under constant threat of emerging and re-emerging infections such as the ongoing corona virus pandemic which devastated many countries. Public health specialists of the modern era need to keep in mind the epidemiological approaches taught by the pioneers in Epidemiology and apply them in practice for betterment of the population's health.

5. CONFLICT OF INTEREST

Conflict of interest declared none.

6. ACKNOWLEDGEMENT

Authors thank the management of Sree Balaji Medical College and Hospital for their support.

7. FUNDING SOURCE

None

8. REFERENCES

1. Pearce N. Traditional epidemiology, modern epidemiology and public health. Am J Public Health. 1996; 86:678-83
2. Cardus D. Towards a medicine based on the concept of health. Preventive Medicine. 1973;2(3):309-312.
3. Manning Feinleib, A Dictionary of Epidemiology, Fourth Edition - Edited by John M. Last, Robert A. Spasoff, and Susan S. Harris, Am J Epidemiology 2001; 154:93–94
4. Feezer LW. Theories concerning the causation of disease. Am J Public Health 1921; 11:908-912
5. Pearce-Duvet JM. The origin of human pathogens: evaluating the role of agriculture and domestic animals in the evolution of human disease. Biol Rev CambPhilos Soc. 2006; 81:369-82
6. Van der Eijk, Philip, editor. "Water, Health and Disease in The Hippocratic Treatise Airs, Waters, Places." Greek Medicine from Hippocrates to Galen: Selected Papers, by Jacques Jouanna and Neil Allies, Brill, Leiden; Boston, 2012, pp. 155–172
7. Jones HW. John Graunt and his bills of mortality. Bull Med Libr Assoc. 1945; 33:3-4
8. Natural and Political Observations Made Upon the Bills of Mortality. John Graunt. American Journal of Sociology. 1939;45(2):297-297.
9. Bartholomew M. James Lind and scurvy: A revaluation, J Marit Res 2002:4; 1-14
10. RajwirBhalwar. History of Public Health. In: Textbook of Public Health and Community Medicine; 1st edn. Pune; 2009. p8-16
11. Riedel S. Edward Jenner and the history of smallpox and vaccination. Proc (BaylUniv Med Cent). 2005; 18:21–25
12. Nasser Pouyan, Semmelweis, the Savior of Mothers and an Early Pioneer of Antiseptic, Am J Med Sci 2014;4: 256-261
13. Kadar N. Rediscovering Ignaz Philipp Semmelweis (1818-1865). Am J Obstet Gynecol. 2019;220(1):26-39
14. Koo D, Thacker S. In snow's footsteps: Commentary on shoe-leather and applied epidemiology. Am J epidemiol. 2010; 172:737-9
15. Abrizah A, Thelwall M. Can the impact of non-Western academic books be measured? An investigation of Google Books and Google Scholar for Malaysia. Journal of the Association for Information Science and Technology. 2014;65(12):2498-2508.
16. Gan'czak M. John Snow and Cholera–The Bicentenary of Birth. Przegl Epidemiol. 2004; 68:89-92.

SP-37

A Case Report Of Enchondroma Presenting Radiologically As A Destructive Lesion

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Abstract: Enchondroma is the most common benign bone tumor of the hand & it is intramedullary and cartilaginous tumor. It originates from the cartilage and it is commonly located in the metaphysis of the proximal phalanx. Furthermore, enchondromas along with pathological fractures occur because the disease process has weakened the bone. Often, these injuries result from minor trauma, which might cause a fracture in healthy bone. Recent study shows that simple curettage with or without bone grafting is an effective treatment for most patients with simple solitary enchondromas. This is a case report of Enchondroma of proximal phalanx of third digit of left hand in a skeletally immature patient with no history of trauma, presented with a large swelling & pain with short duration. Clinically patient had warmth and tenderness. X-Ray showed cortical breach with periosteal reaction; MRI revealed destructive eccentric lytic lesion. Patient was planned for excision biopsy intra-operatively patient had pathologic fracture and soft tissue mass extending from the cortical breach was found. Which was sent for histopathological examination & culture. Intra-operatively malignancy was considered to be a possibility but HPE eventually turned out to be enchondroma. The pathological fractures along with enchondroma have no significant role on the treatment outcomes compared to those with simple non-fractured enchondromas. Here, after three months post-op follow up fracture shows ossification.

Keywords: Enchondroma, proximal phalanx, simple curettage, eccentric lytic lesion, pathological fracture.

1. INTRODUCTION

Background**:** Enchondroma are common tumors involving the phalanx of the hand this are mostly discovered incidental and do not warrant treatment however enchondroma in rare instances are associated with complications like pain, neurovascular complications, mechanical limitation of joint movement and rarely malignant transformation fracture.[1] aim [of] [t]he pathological fractures along with enchondroma have no significant role on the treatment outcomes compared to those with simple non-fractured enchondromas.

2. CASE REPORT

Patients presenting complaints: 12 years old adolescent male presented with complaints of pain in the proximal phalanx of middle finger of left hand. Associated with pain, which is insidious in onset, dull aching, gradually progressive, not radiating.
Previous history: History of swelling present which gradually increasing in size for past one week.
Family history: Nil
Observation: Firm to hard swelling of size 1x2cm along the palmar aspect and 1x1cm over the dorsal aspect of proximal phalanx.

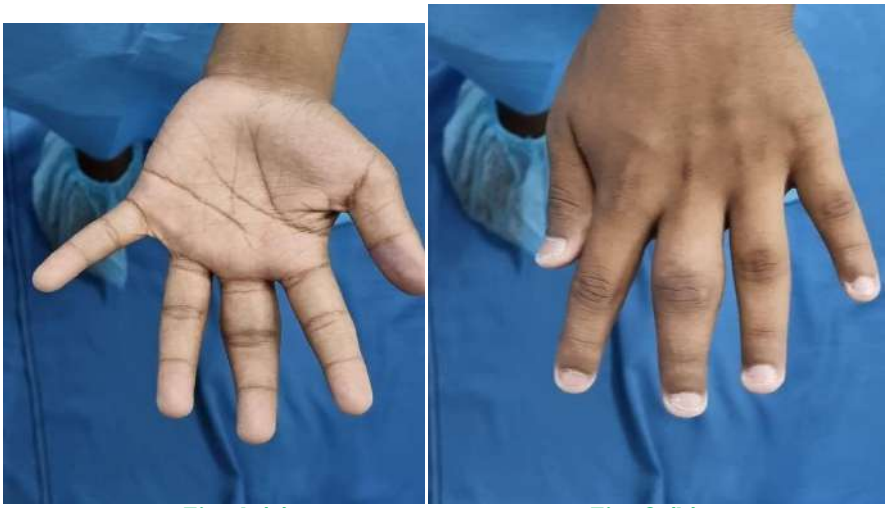

Fig -1 (a) Fig -2 (b)

FIG 1: 1 (a) Firm hard swelling of size 1x2 cm along the palmar aspect and 1 (b) 1x1 cm swelling over the dorsal aspect of proximal phalanx.

On radiographic Investigations

X-Ray shows A Translucent lesion with strippled calcification is seen in the left hand of proximal phalanges of third finger. MRI shows Destructive eccentric lytic lesion seen in the ventral aspect of distal end of proximal phalanx of middle finger with overlying cortical break & associated soft tissue component.

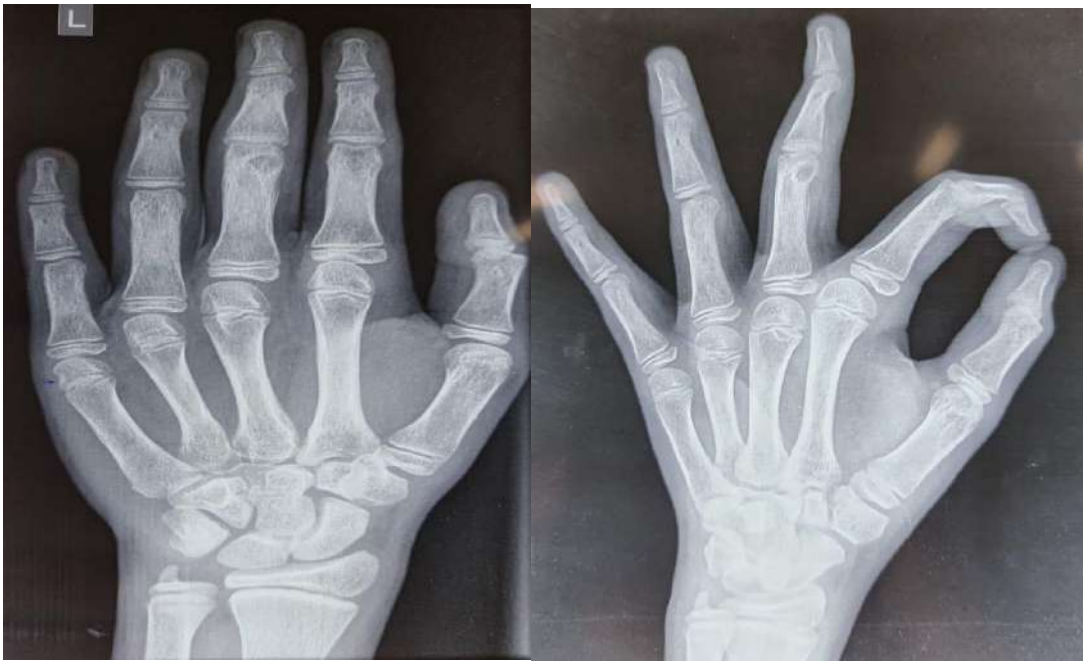

Fig (2): Translucent lesion with strippled calcification

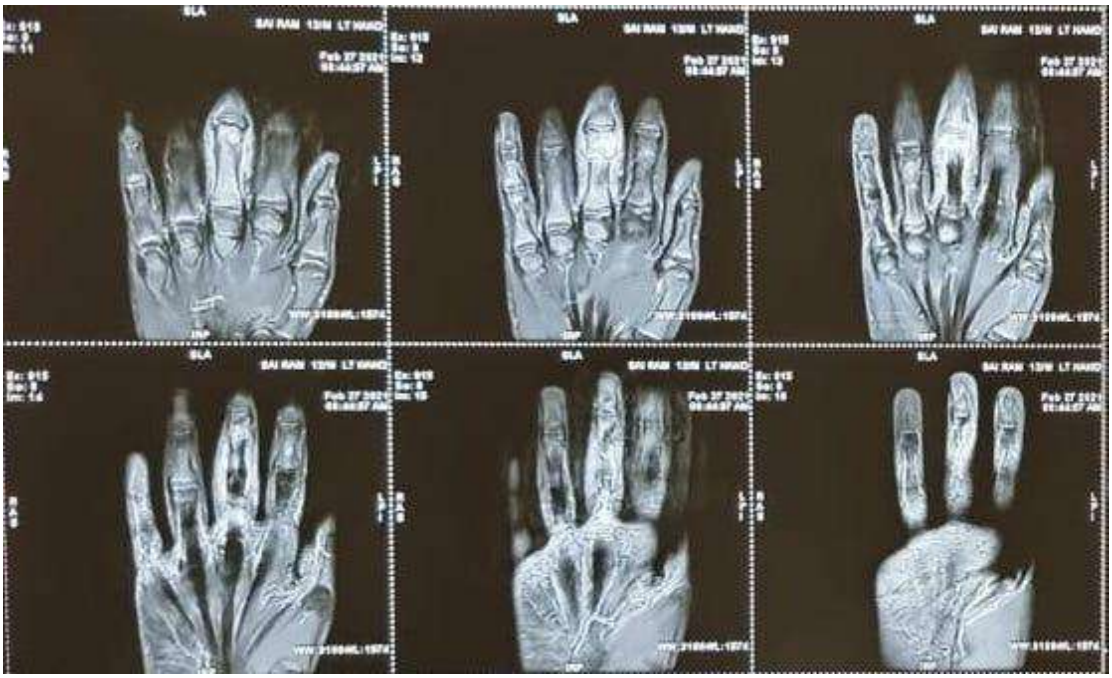

Fig (3): Destructive eccentric lytic lesion in ventral aspect of distal end of proximal phalanx of middle finger with cortical break.

Diagnosis:
Since the clinical picture and MRI images are consistent with diagnosed of Enchondroma.

Treatment:
Surgical excision was planned and performed under general anesthesia, through medial longitudinal incision, skin and subcutaneous tissue were retracted the tumour is exposed it is lobulated white translucent in appearance, the specimen was sent for histopathology examination, curettage of surrounding bone was done and the skin incision was closed with 3.0 ethilion.

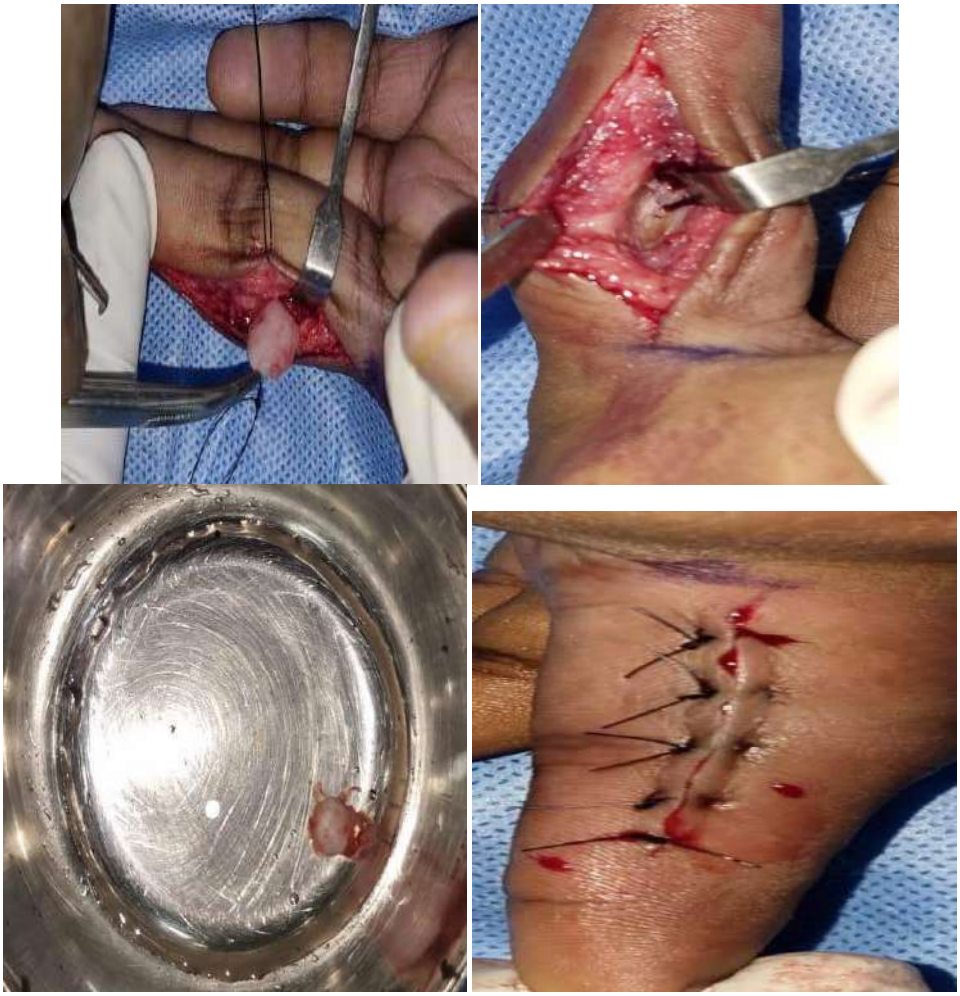

Fig 4 – Tumour, which is lobulated white translucent in appearance

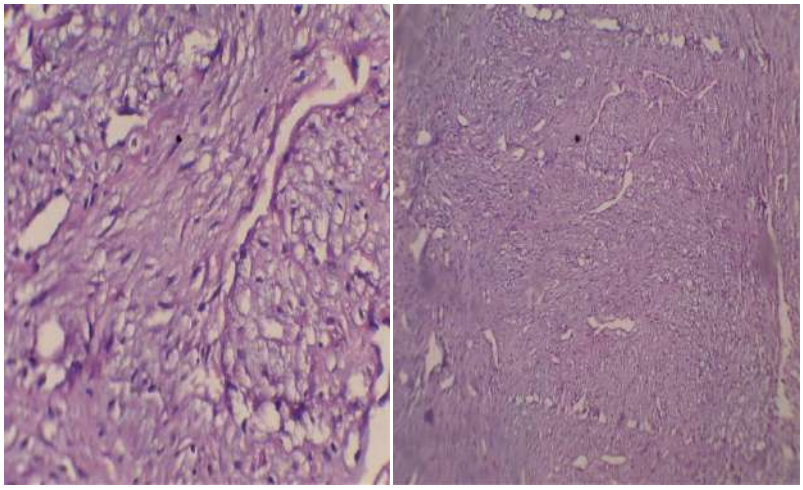

Fig (5): Microscopic appearance of the tumour shows hypocellular and avascular hyaline cartilage interspersed with chondrocytes.

3. DISCUSSION

Enchondromas are mineralized or unmineralized hyaline cartilage tumours occurring in the medulla of long bones it is considered a development disorder due to failure of endochondral ossification. It commonly occurs in the same bones of the hand and it is a most common primary tumor in the hand it involves all ages and both sex[2]. Pathological fractures occur because the bone has been weakened the disease process. This lesions are solitary in nature multiple enchondroma's is associated with Ollier's disease and Mafucci syndrome. Solitary enchondroma's are mostly asymptomatic but rarely can cause pain, deformity, mechanical obstruction to motion.[3] The most fearing complication of enchondroma is malignant transformation into chondrosarcoma although it is considered less than 1%.[4] Differential diagnosis includes bone infarcted, simple bone cyst, fibrous dysplasia, enchondroma is mostly diagnosed by clinical and radiological findings and Biospy is rarely needed to confirm the

diagnosis. Small lesions causing symptoms can be surgical excised and bone grafting can be done.[5-7] In our case the lesion involving the proximal phalanges of middle of left hand in a 12year old young adolescent boy along with pathological fracture is surgical excised and diagnosis is confirmed with histo pathological examination. The pathological fracture associated with enchondroma has no significant rol on treatment outcomes. In post op follow up fracture site showed ossification.[8,9]

Fig (6): Post-op x-ray & postoperative follow up clinical pictures.

4. CONCLUSION

In this case we describe the Enchondroma arising from proximal phalanx of third digit of left hand associated with pathological fracture by confirmation of magnetic resonance imaging, excision biopsy and further confirmed by histopathological examination. The curettage with or without bone grafting is an effective and safe treatment for most patients with Simple solitary enchondromas. The presence of a pathological fracture does not change the future outcome Compared with lesions after fracture union. Early surgical intervention is needed for better results and no significant increased risks for patients with Pathological fractures caused by enchondroma.

5. CONFLICT OF INTEREST

Conflict of interest declared none.

6. ACKNOWLEDGEMENT

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7. FUNDING SOURCE

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8. REFERENCES

1. Bachoura A, Rice IS, Lubahn AR. The surgical management of hand enchondroma without post curettage void augmentation: authors experience & a systematic review. Hand (N Y) 2015; 10:461–71.
2. Jacobson ME, Ruff ME. Solitary enchondroma of the phalanx. J Hand Surg Am 2011; 36:1845–7.

3. Shimizu K, Kotoura Y, Nishijima N. Enchondroma of the distal phalanx of the hand. J Bone Joint Surg Am 1997; 79:898–900.
4. Gaasbeek RD, Rijnberg WJ, van Loon CJ. No local recurrence of enchondroma after curettage and plaster filling. Arch Orthop Trauma Surg. 2005; 125:425.
5. Hasselgren G, Forssblad P, Tornvall A. Bone grafting unnecessary in thetreatment of enchondromas in the hand. J Hand Surg Am 1991; 16:139–42.
6. Yalcinkaya M, Akman YE, Bagatur AE. Recurrent metacarpal enchondroma treated with strut allograft: 14-year follow-up. Orthope- dics 2015;38: e647–50.
7. Sassoon AA, Fitz-Gibbon PD, Harmsen WS, et al. Enchondromas of the hand: factors affecting recurrence, healing, motion, and malignant transformation. J Hand Surg Am 2012; 37:1229–34.
8. Figl M, Leixnering M. Retrospective review of outcome after surgicaltreatment of enchondromas in the hand. Arch Orthop Trauma Surg. 2009; 129:729–34.
9. Haase SC. Treatment of pathologic fractures. Hand Clin 2013;29: 579–84.
10. Pianta TJ, Baldwin PS, Obopilwe E, Mazzocca AD, Rodner CM, Silverstein EA: A biomechanical analysis of treatment options for enchondromas of the hand. 2013;8(1):86-91.
11. Unni KK, Inwards CY: Chondroma in Dahlin's Bone tumors, ed 6. Philadelphia PA, Wolters Kluwer/Lippincott Williams & Wilkins, 2010, pp 22-40.
12. Simon MJ, Pogoda P, Hovelborn F. Incidence, histopathologic analysis and distribution of tumors of the hand. BMC Musculoskelet Disord 2014; 15:182.24885007.
13. Gaulke R: The distribution of solitary enchondromata at the hand. J Hand Surg Br 2002;27(5):444-445.12367543.
14. Sassoon AA, Fitz-Gibbon PD, Harmsen WS, Moran SL: Enchondromas of the hand: Factors affecting recurrence, healing, motion, and malignant transformation. J Hand Surg Am 2012;37(6):1229-1234.
15. Milgram JW: The origins of osteochondromas and enchondromas: A histopathologic study. Clin Orthop Relat Res 1983;(174):264-284.
16. Bachoura A, Rice IS, Lubahn AR, Lubahn JD: The surgical management of hand enchondroma without postcurettage void augmentation: Authors experience and a systematic review. Hand (N Y) 2015;10(3):461-471.

SP-38

A Case Series Of Paediatric Distal Tibia Epiphyseal Injuries Managed By Reducing The Fracture By Closed Technique And Fixing Internally With Kirschner Wire With The Help Of C-Arm Guidance.

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Abstract: The main purpose of the study is to prospectively analyse the functional outcome of distal tibia epiphyseal injury managed by reducing the fracture by closed technique and fixing internally with Kirschner wire with the help of C-arm Guidance. A prospective analysis of 22 children (13 boys and 9 girls, with mean age 13yrs) with salter harris classification fracture managed by reducing the fracture by closed technique and fixing internally with Kirschner wire with the help of C-arm Guidance. Between july 2019 and july 2021 was performed. The results were studied based on range of motion (rom) and visual analog score (vas). The mean rom and vas at the interval of 3 weeks, 5 weeks, 9 weeks, 6 months, shows excellent improvement. The mean vas score is 8.8 and range of motion is excellent. In the Present study, 22 patients with paediatric distal tibial epiphyseal injuries were managed by reducing the fracture by closed technique and fixing internally with Kirschner wire with the help of C-arm Guidance allows for early rehabilitation of the patient and has excellent functional outcome with less incidence of complications. Hence, we strongly recommend considering it in the treatment of such fractures.

Keywords: Kirschner Wire, Epiphyseal Injury, Distal Tibia, Prospective Study.

1. INTRODUCTION

One of the Major fractures in children are distal tibia epiphyseal fracture while playing with their friends. About 70% of distal tibia epiphyseal fracture happens in the school days. According to the current day scenario, football accounts for distal epiphyseal fracture in children. Physeal Injuries are more common in boys. Ankle is stable in children mostly due the ligaments around the talofibular joint. Ligaments around the talofibular joint is responsible for the paediatric distal tibia epiphyseal injury[1]. Paediatric fracture in distal tibia epiphysis is responsible for instability in children. It may lead to disturbance in the day-to-day activities. And may disturb the schooling of the children. Complication of the paediatric epiphyseal injury of distal tibia fracture is difficulty in walking and difficulty in baring weight. The most common system they follow to classify the fracture is salter-harris fracture classification. Tat classifies the paediatric distal tibia epiphyseal fracture into 4 types 1st 2 types are not very risky. It can be managed by common methods.[2] Other 2 types are little tricky to manage. This paediatric followup study is to analyse the effect of Kirschner wire in the management of paediatric distal tibia epiphyseal fracture in paediatric age group.[3,4] The main purpose of this study is to prospectively analyse the functional outcome of distal tibia epiphyseal injury managed by reducing the fracture by closed technique and fixing internally with Kirschner wire with the help of C-arm Guidance.

2. MATERIALS AND METHODS

This study was done at our institution between july 2019 and july 2021. 22 children (13 boys and 9 girls) with mean age of 13yrs (range 6-14yrs) with salter harris fracture classification type 2 is included in this present study. All children were managed by reducing the fracture by closed technique and fixing internally with Kirschner wire with the help of C-arm Guidance. Pop application is done to the children before surgery All investigations were done.

3. INCLUSION CRITERIA

Distal tibia epiphyseal fracture which is displaced
Distal tibia epiphyseal fracture along with associated unstable fracture
Distal tibia epiphyseal Injury in Mentally Ill Children.
Distal tibia epiphyseal Injury in brain death patients.

4. EXCLUSION CRITERIA

Distal tibia epiphyseal fracture which is found to be Undisplaced
Distal tibia epiphyseal Fractures that can be treated by POP APPLICATION.
Distal tibia epiphyseal Fractures for a Children Age less than three years and
Distal tibia epiphyseal Fractures for a Children Age more than fifteen years
Compound Distal tibia epiphyseal fractures
Distal tibia epiphyseal Fractures that require open reduction
Nonuninon Distal Tibia Epiphyseal fractures.

5. RESULTS

The treatment method was decided after classifying the distal tibia epiphyseal fracture based on Salter Harris classification. The patients were taken for surgery as early as possible time depending on their co morbidities and skin condition (Fig 1&2). Preoperative X-Ray of the ankle was taken (FIG 3) which helps in diagnosing the epiphyseal fractures and plan for management. Most of the distal epiphyseal fractures were treated within 48-72 hrs. All surgeries were done under C-Arm Guidance. Fractures were managed by reducing the fracture by closed technique and fixing internally with Kirschner wire with the help of C-arm Guidance. Under General Anaesthesia, Patient in Supine position. The limb that is found to be fractured is well prepared and draped. Fracture site was noted with the help of C-arm, Fracture is reduced using closed technique and internally it is fixed with k-wire of proper size. Post operative X-Ray was taken to confirm the reduction and position of K-wires (FIG 4). Further follow up X-Ray was taken at 6 weeks and K wires removed (FIG 5). Gait training exercises and ankle mobilisation was done a week following k wire removal, which regains the normal range of movements (FIG 6). This Study Outcome was based on range of motion (rom) and visual analog score (vas). Follow-up of the patient both clinical and radiological, was made at 3weeks, 5weeks, 9 weeks, 6months, and 1year. The mean ROM and VAS at the interval of 3weeks, 5weeks, 9 weeks, 6months, 1 year shows excellent improvement. The mean vas score is 8.8 and range of motion is excellent. The results show the efficacy of the fracture managed by reducing the fracture by closed technique and fixing internally with Kirschner wire with the help of C-arm Guidance in the treatment for paediatric distal tibia epiphyseal injury.

Case: 4yr/female:

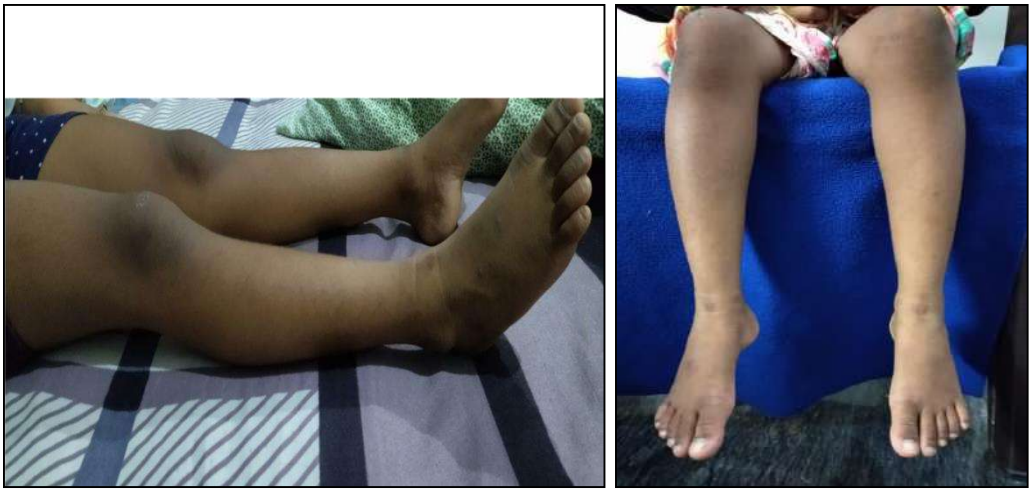

Fig 1 & 2: This Clinical Picture Shows Diffuse Swelling Over the Right Ankle with Restriction of Movements. Diffuse Swelling over the right ankle region indicative of any traumatic condition which is acute condition.

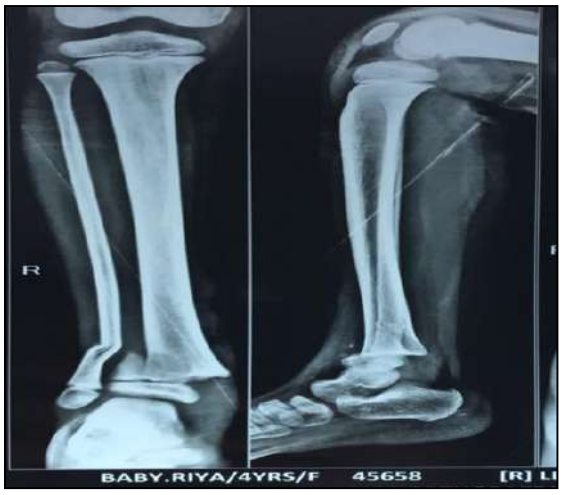

FIG 3: Xray Rt Ankle Shows break in the cortices Of the Rt Tibia & fibula- Distal Part and It Confirms Paediatric Displaced Distal Tibia Epiphyseal Injury.

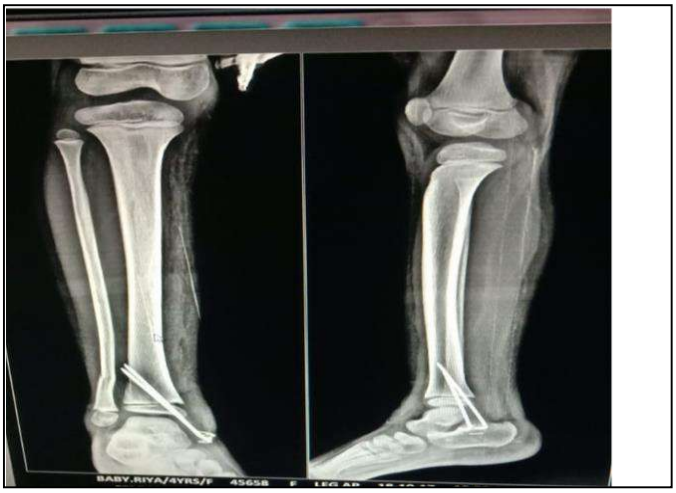

FIG 4: Post operative Xray Right Ankle Shows Fixation Made at Rt Tibia Epiphyseal Injury by Kirschner wire Fixation and reduction was found to be very much satisfactory.

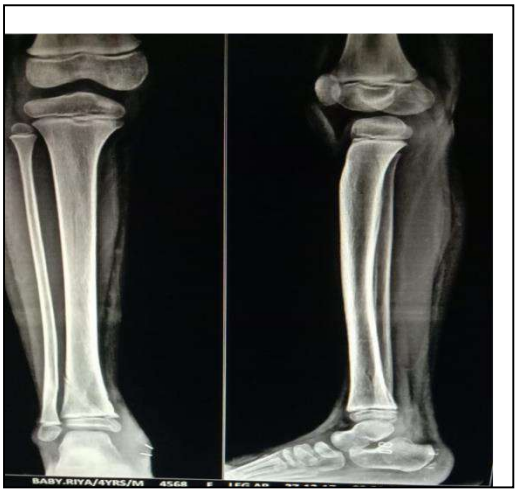

FIG 5: Xray Right ankle shows union of epiphyseal fracture after the removal of k-wire after 6 weeks of treatment

FIG 6: patient is now able to walk and bare weight and able to plantar flex, Dorsiflex, inversion and eversion after 8 weeks of treatment. Movements of the ankle joint was completely recovered.

6. DISCUSSION

In this present study, 22 children with paediatric distal tibial epiphyseal injuries were managed by reducing the fracture by closed technique and fixing internally with Kirschner wire with the help of C-arm Guidance. The fixation of the epiphyseal injuries using kirschner wire allows for early rehabilitation of the patient and has excellent functional outcome with less incidence of

complications.[5-9]. The results show the efficacy of the fracture managed by reducing the fracture by closed technique and fixing internally with Kirschner wire with the help of C-arm Guidance in the treatment for paediatric distal tibia epiphyseal injury. Follow up period of 22 children were good. We strongly recommend to consider this management for paediatric epiphyseal injury of distal tibia.[10-13]

7. CONCLUSION

From the above Study, we conclude that Closed Kirschner wire fixation is the best effective treatment in the paediatric distal tibia epiphyseal injuries. Fixation of paediatric distal epiphyseal injury by closed reduction technique with Kirschner wire is the best treatment than any other method of treatment.

8. ACKNOWLEDGEMENT

We thank all the patients who were the part of the study. We also thank the management of Sree Balaji Medical College and Hospital for their support

9. AUTHOR CONTRIBUTIONS

Ganesh MT contributed towards treatment protocol and follow up. Akilan contributed in preparing the case series and collecting clinical and radiological images. Vasanth Kumar contributed editing, drifting case report.

10. INFORMED CONSENT

Written and oral informed consent were obtained from the participant in the study.

11. ETHICAL COMMITTEE APPROVAL

Proper ethical committee approval was taken for the study.

12. FUNDING SOURCE

None

13. CONFLICT OF INTEREST

Conflict of interest declared none.

14. REFERENCES

1. Mann DC, Rajmaira S. Distribution of physeal and nonphyseal fractures in 2650 long-bone fractures in children aged 0–16 years. J PediatrOrthop 1990; 10:713–716.
2. Mizuta T, Benson WM, Foster BK, Morris LL Statistical analysis of the incidence of physeal injuries. J PediatrOrthop 1987; 7:518–523.
3. Peterson CA, Peterson HA. Analysis of the incidence of injuries to the epiphyseal growth plate. J Trauma 1972; 12:275–281.
4. Epiphyseal Growth Plate Fractures. Journal of Pediatric Orthopaedics. 2007;27(7):848.
5. WH, Craig C, Banks HH. Epiphyseal injuries. PediatrClin North Am 1974; 21:407–422.
6. Tachdjian M. Paediatric Orthopaedics and Fractures. JAMA: The Journal of the American Medical Association. 1981;245(2):184.
7. Aitken AP. Fractures of the epiphyses. ClinOrthopRelat Res 1965; 41:19–23.
8. Barmada A, Gaynor T, Mubarak SJ. Premature physeal closure following distal tibia physeal fractures: a new radiographic predictor. J PediatrOrthop 2003; 23:733–739.
9. Nenopoulos SP, Papavasiliou VA, Papavasiliou AV. Outcome of physeal and epiphyseal injuries of the distal tibia with intra-articular involvement. J PediatrOrthop 2005; 25:518–522.
10. Rohmiller MT, Gaynor TP, Pawelek J, Mubarak SJ. Salter-Harris I and II fractures of the distal tibia: does mechanism of injury relate to premature physeal closure? J PediatrOrthop 2006; 26:322–328.

11. Cass JR, Peterson HA. Salter-Harris Type-IV injuries of the distal tibial epiphyseal growth plate, with emphasis on those involving the medial malleolus. J Bone Joint Surg Am 1983; 65:1059–1070.
12. Dugan G, Herndon WA, McGuire R. Distal tibialphyseal injuries in children: a different treatment concept. J Orthop Trauma 1987; 1:63–67.
13. Karrholm J, Hansson LI, Svensson K. Prediction of growth pattern after ankle fractures in children. J PediatrOrthop 1983; 3:319–325.

SP-39

Case Report of Recurrent Bartholin Abscess

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Abstract: The most common reported abscess in gynaecological outpatient clinics worldwide is bartholin's gland abscess; it has also been reported that Bartholin's gland abscess is three times more common in occurrences than Bartholin's gland cyst. It is more common in women who are at risk of acquiring sexually transmitted infections; however, other sources of infection should be investigated to rule out other possible causes of disease. Here we are discussing a case report of a patient presenting with recurrent Bartholin abscess, initially was treated by incision and drainage and later marsupialisation was also done for Bartholin cyst. Further treatment for primary infertility was also started by ruling out causes of infertility in the patient and her partner. Bartholin cysts/abscesses are predominantly found in women of child-bearing age. The incidence of Bartholin cysts in most often noted at the onset of puberty and increases with age until menopause. The Bartholin gland obstruction may also occur after trauma to the area, episiotomy or childbirth, it may also occur without an identifiable cause. Bartholin's cyst/abscess affect up to 3 in 100 women and can be treated with antibiotics or with a small procedure. Symptomatic Bartholin cysts and abscesses account for 2 percent of all gynaecologic visits per year.

Keyword: Bartholin gland, Bartholin cyst, Bartholin abscess, marsupialisation.

INTRODUCTION

Bartholin glands are two pea size glands that are located next to the vaginal entrance (one on the right and one on the left) and they cannot normally be seen or felt. They normally secrete a small amount of fluid through a duct which keeps the entrance of the vagina moist. If the duct becomes blocked this can form a fluid-filled swelling (cyst). If the cyst becomes infected the swelling may become filled with pus (then called an abscess).Bartholin glands are essential glands of female reproductive system, secretes mucus to ensure vaginal and vulval lubrication. Bartholin glands are prone to infections and abscess formation which leads to vestibular pain and dyspareunia. Bartholin cyst occurs due to the blockage of ductal outlet. Infection of this cyst likely to result in bartholin gland abscess which was found to be 3 times more common than cyst. Recurrent Bartholin's gland abscess among women of reproductive age is commonly associated with the risk of being in contact with the sexually transmitted poly-microbial infection.[1,2]

CASE REPORT

32-Year-old female married in the last 4 years nulli gravida, home maker by occupation came to the gynaecology OPD at Sree Balaji Medical Hospital, Chennai

Presenting Complaints

Complaints of pain and swelling in the left labia majora for 2 weeks which initially started as a small swelling, then increased in size, and became painful which was pricking type. It was associated with fever and inability to walk properly and was accompanied with painful micturition.

Menstrual History

Age of menarche – 14 yrs, regular monthly cycles, 3/30 days cycle, not associated with clots or abdominal pain.

Marital History

Married in the past 4 years, Non-consanguineous marriage

Past History

She reported similar complaints twice within 1 year for which incision and drainage was done outside.

Medical History

Not a known case of diabetes, hypertension, bronchial asthma, tuberculosis, epilepsy, heart disease.
No history of any previous surgery except incision and drainage; no history of any previous blood transfusion.

Family History

No history of any medical disorder in family

Investigation

CBC, thyroid function test, blood grouping and typing, serology were done. Basic investigations were normal except HBs Ag which was found to be positive.

Examination

On local examination, there was tender flauctuant mass of size 4x3 cm involving the left labia majora and minora. Cervix and vagina healthy. No abnormal discharges. Per vaginal examination showed cervix pointing downwards, uterus anteverted normal size, fornices free.

Procedure

After preliminary investigations, Incision and drainage was done under anaesthesia under adequate antibiotic coverage. Post operatively patient was normal. Since patient had history of recurrent bartholin abscess, patient was counselled for marsupialisation before it gets infected. Since she is a case of primary infertility, patient and patient's partner were investigated and patient's husband was diagnosed as Klinefelter's syndrome. The patient came with complaint of swelling in labia within 2 months. On local examination there was a bartholin cyst of size 3x3 cm following which marsupialisation was done under anaesthesia. Post-operative period was uneventful and there was no recurrence for next 6 months.

DISCUSSION

Bartholin glands are also called as greater vestibular glands and are located in posterior end of vestibular bulb the duct is 2cms, which opens into vestibule. The bartholin gland is homologous to penile urethra and part of skin of urethra.[3] the bartholin gland cyst is due to infection, thick mucus or swelling which causes the bartholin gland duct blockage. The microorganisms causing recurrent Bartholin's gland abscess are poly microbial and often commensal microorganisms that are not sexually transmitted.[4] Bartholin gland infection can also be caused by sexually transmitted infections. Reason of recurrence probably was suggested to be the previous treatment by incision and drainage rather than treatment by marsupialization-type incision and followed by unknown prolonged course of oral antibiotics.[5,6] A Bartholin's abscess usually requires treatment because it can be painful. Antibiotics should be started and small procedure should be performed to drain the pus. Main aim is to drain the pus and create an opening or duct to prevent blockade in the future.

Treatment available for Bartholin's gland infection:

A. Conservative management: sitz bath
B. Surgical management:
1. Bartholin gland balloon (word catheter)
2. Bartholin abscess: Incision and drainage
3. Bartholin cyst: Marsupialization or removal of gland
Based on the clinical presentation, we have to decide the mode of treatment.[6]

CONCLUSION

Since for bartholin abscess for this patient,incision and drainage was done thrice, the patient was advised for marsupialisation at earlier period,she reported to us at earlier stage for which marsupialisation was done, and on follow up for size months showed no recurrence. Bartholin's cyst or abscesses usually appear suddenly for no apparent reason so there is usually no way to prevent them. However, some bartholin's abscesses are caused by sexually transmitted infections so using barrier contraceptives, can help to prevent in some cases.

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FUNDING SOURCE

None

CONFLICT OF INTEREST

Conflict of interest declared none.

REFERENCES

1. Omole F, Simmons BJ, Hacker Y. Management of Bartholin's duct cyst and gland abscess. American family physician. 2003 Jul 1;68(1):135-40.

2. Krissi H, Shmuely A, Aviram A, From A, Edward R, Peled Y. Acute Bartholin's abscess: microbial spectrum, patient characteristics, clinical manifestation, and surgical outcomes. European Journal of Clinical Microbiology & Infectious Diseases. 2016 Mar 1;35(3):443-6.
3. Begum S, Roy S, Yusuf A. Anaerobic bacteria: infection and management. IOSR Journal of Dental Medical Sciences. 2015;14(12):69-72.
4. Hoosen AA, Nteta C, Moodley J, Sturm AW. Sexually transmitted diseases including HIV infection in women with Bartholin's gland abscesses. Sexually Transmitted Infections. 1995 Jun 1;71(3):155-7.
5. John CO, Enyinda CE, Okonya O. Bartholin's Cyst and Abscesses in a Tertiary Health Facility in Port Harcourt, South-South Nigeria. Journal of Medical and Biological Science Research. 2015 Oct;1(8):107-1.
6. Heim LJ. Evaluation and differential diagnosis of dyspareunia. American family physician. 2001 Apr 15;63(8):1535.
7. Lee WA, Wittler M. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Feb 25, 2021. Bartholin Gland Cyst.
8. Pundir J, Auld BJ. A review of the management of diseases of the Bartholin's gland. J Obstet Gynaecol. 2008 Feb;28(2):161-5.
9. Yuk JS, Kim YJ, Hur JY, Shin JH. Incidence of Bartholin duct cysts and abscesses in the Republic of Korea. Int J Gynaecol Obstet. 2013 Jul;122(1):62-4.
10. Marzano DA, Haefner HK. The bartholin gland cyst: past, present, and future. J Low Genit Tract Dis. 2004 Jul;8(3):195-204.
11. Visco AG, Del Priore G. Postmenopausal bartholin gland enlargement: a hospital-based cancer risk assessment. Obstet Gynecol. 1996 Feb;87(2):286-90.
12. Kroese JA, van der Velde M, Morssink LP, Zafarmand MH, Geomini P, van Kesteren P, Radder CM, van der Voet LF, Roovers J, Graziosi G, van Baal WM, van Bavel J, Catshoek R, Klinkert ER, Huirne J, Clark TJ, Mol B, Reesink-Peters N. Word catheter and marsupialisation in women with a cyst or abscess of the Bartholin gland (WoMan-trial): a randomised clinical trial. BJOG. 2017 Jan;124(2):243-249
13. Reif P, Ulrich D, Bjelic-Radisic V, Häusler M, Schnedl-Lamprecht E, Tamussino K. Management of Bartholin's cyst and abscess using the Word catheter: implementation, recurrence rates and costs. Eur J Obstet Gynecol Reprod Biol. 2015 Jul; 190:81-4.
14. Ozdegirmenci O, Kayikcioglu F, Haberal A. Prospective Randomized Study of Marsupialization versus Silver Nitrate Application in the Management of Bartholin Gland Cysts and Abscesses. J Minim Invasive Gynecol. 2009 Mar-Apr;16(2):149-52.
15. Fambrini M, Penna C, Pieralli A, Fallani MG, Andersson KL, Lozza V, Scarselli G, Marchionni M. Carbon-dioxide laser vaporization of the Bartholin gland cyst: a retrospective analysis on 200 cases. J Minim Invasive Gynecol. 2008 May-Jun;15(3):327-31.

SP-40

Lacrimal SAC Rhinosporidiosis: A Case Report

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Abstract: Rhinosporidiosis is a chronic granulomatous disease that mostly affects the mucous membrane and is caused by the aquatic protistan parasite Rhinosporidium seeberi. The nose is the most prevalent site of involvement, accounting for 83.3 percent of cases, with ocular involvement accounting for 11.2 percent and other sites such as the larynx, trachea, and bronchus accounting for 5.5 percent of cases. The participation of the lacrimal drainage system in several oculosporidiosis case series ranged from 14.3 percent to 59.6 percent. In a review of 31 studies, isolated lacrimal sac involvement in rhinosporidiosis was detected in 45.8% (72 out of 157) instances of the lacrimal drainage system. To treat rhinosporidiosis of the lacrimal sac, a variety of surgical methods have been utilised, including dacryocystorhinostomy, Dacryocystectomy, lateral rhinotomy, and local lesion excision, with success rates ranging from 28.5 percent to 92.3 percent. Because a uniform surgical procedure was performed in all instances of a certain series, regardless of the level of disease, there was a considerable range in the success rate. We provide a case study of an isolated lacrimal sac rhinosporidiosis in a 36-year-old male from Southern India. In this situation, the manner of presentation and administration, as well as a review of literature, are briefly reviewed.

Keywords: Rhinosporidiosis, lacrimal sac, Rhinosporidiumseeberi.

INTRODUCTION

Rhinosporidiosis is a chronic granulomatous disease caused by the bacterium Rhinosporidium seeberi, which is most commonly found in tropical places such as southern India and Sri Lanka.[1]The condition most commonly affects the nose and nasopharynx, and patients usually come with a painless mass and a history of nasal haemorrhage.[2] A polypoidal tumour in the nasal cavity is a common symptom.[3] Despite the fact that nasal blockage and epistaxis are typical clinical symptoms, epiphora as a single symptom might be difficult to diagnose.[4] We present a case of isolated lacrimal sac rhinosporidiosis masquerading as chronic dacryocystitis that was successfully controlled with endoscopic excision.[5] For the past two years, a 36-year-old male arrived at the ENT outpatient department with complaints of diffuse infraorbital edema on the left side and occasional wetness from the left eyes. In his village, the patient had a history of swimming in ponds. Cattle frequently visited the pond.

CASE REPORT

Rhinosporidiosis is a chronic granulomatous disease caused by the bacterium Rhinosporidium seeberi, which is most commonly found in tropical places such as southern India and Sri Lanka. For the past two years, a 36-year-old male arrived at the ENT outpatient department with complaints of diffuse infraorbital edema on the left side and occasional wetness from the left eyes. In his village, the patient had a history of swimming in ponds. Cattle frequently visited the pond. In examination, there was a 2x3 cm diffuse oval nontender swelling on the medial infraorbital region that was mushy in nature. The borders were clearly defined. Pinchable skin covered the swelling. On aspiration of the edema, there was a lot of fluid. The extraocular motions and vision were both within normal norms. The fluid regurgitated during the lacrimal syringing test, suggesting partial patency of the left nasolacrimal duct. Dermoid cyst, Fibroma, Lipoma, Neuroma, and Dacrocystocoel were the differential diagnoses based on clinical examination. A diagnostic nasal endoscopy revealed no abnormalities during the investigation. Along the inferior aspect of the left orbit, CT PNS revealed an isodense lesion with slight enhancement within the preseptal compartment. The results of FNAC were inconclusive. Under general anesthesia, the plan was to execute a total excision of the lesion. Without spilling the spores, a mass of numerous pink vascularized white studs was extracted as a whole. A biopsy was collected and sent to a pathologist for histological analysis. Numerous sporocysts in various stages of maturation were found under stratified squamous epithelium of the lacrimal sac in a histological section of the biopsy. Lacrimal sac rhinosporidiosis with medial supraorbital disease was established by histopathology. After surgery, the patient was given 100 mg of diaminodiphenyl sulphone (dapsone) orally every day for three months. During follow-up, there has been no evidence of recurrence.

DISCUSSION

Rhinosporidiosis is caused by the bacterium Rhinosporidium seeberi, which was first identified in 1900 by an Argentinian physician named Guillermo Seeber.[6] It is native to the Indian subcontinent, Sri Lanka, Bangladesh, and Nepal. It has, however, been reported from a wide range of geographical places around the world.[7] Infection is more common in the first four decades, and men are 2.5 times more likely to contract it.[8] About 15% of cases involve the eye and ocular adnexa, with the conjunctiva and lacrimal sac being the most typically affected.[9] Bathing in stagnant pond water is thought to be a significant risk factor for infection.[10] Inhalation of polluted field dust has also been suggested as a source of infection.[11] The creature is thought to thrive in a humid, warm, tropical climate, with stagnant pond water providing the best conditions for survival.[12] Complete local excision of the granulomatous lesion with appropriate cauterization is the preferred treatment for rhinosporidiosis at any affected site.[13] Spillage and seeding of spores on adjacent normal tissues must be avoided to prevent recurrence.[14] Because the lacrimal sac is a relatively isolated organ in comparison to the other related places in the body, it is easier to remove completely than most

other tissues.[15] It's hardly surprising, then, that full excision of the diseased lacrimal sac is the most widely advised treatment technique for lacrimal sac rhinosporidiosis in the literature. [16]

Theories of Spread Of Disease:

- Demellow's theory of direct transmission
- Autoinoculation theory of Karunarathnae (responsible for satellite lesions)
- Haematogenous spread - to distant sites
- Lymphatic spread - causing lymphadenitis (rarity)

Fig. 1. The mass before surgery. This patient presented with a medial lower lid swelling

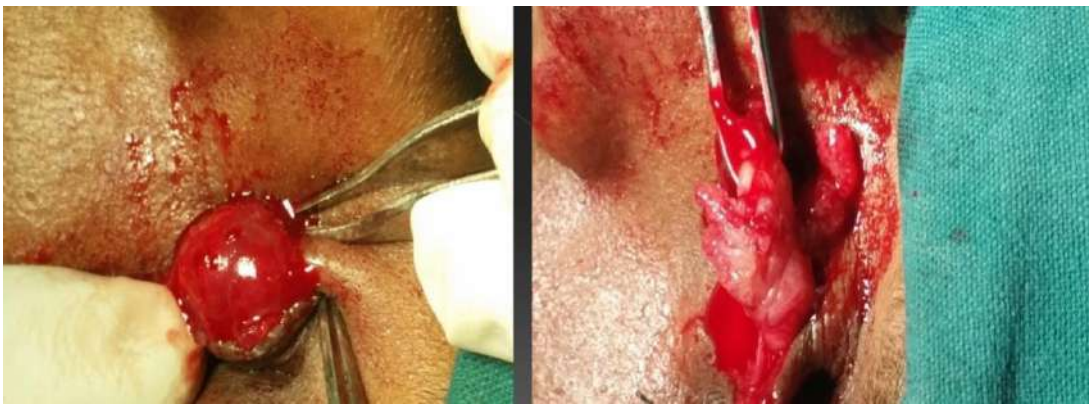

Fig. 2. Intraoperative findings: A sac diverticulum and an intrasac granuloma were discovered during surgery. On the surface of the granuloma, there were many sporangia that looked like sago grains.

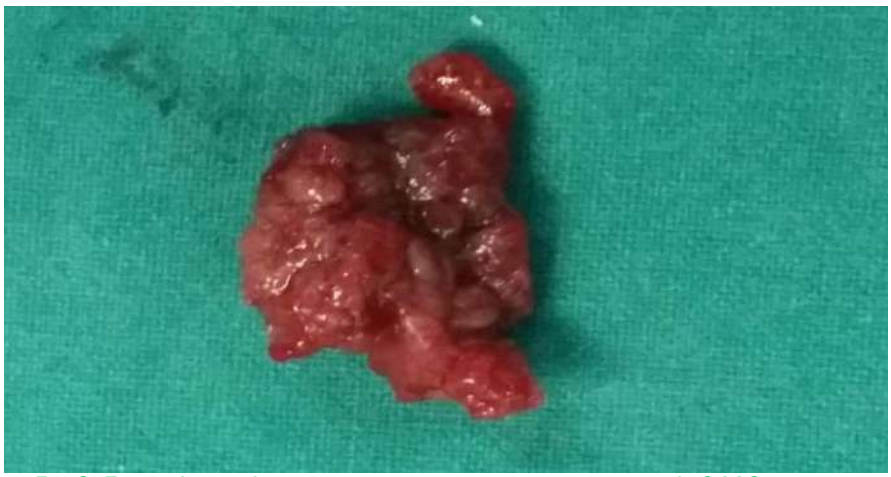

Fig. 3. Excised granulomatous tissue, measuring appropriately 2 X 3 cm

CONCLUSION

In conclusion Rhinosporidiosis affects the nose and nasopharynx but it rarely affects the lacrimal sac, skin and tonsils. This case of lacrimal sac rhinosporidiosis of medial supraorbital pathology is rare. Total surgical external excision appears to be a viable option isolated lacrimal sac rhinosporidiosis, based on the results of this case report. Combined with adequate precautions can

result in great long-term outcomes with no infection recurrence. In other studies, based on their previous experience, the authors do not now utilise routine dapsone prophylaxis in their own practise for these instances. Hence dapsone prophylaxis was not used in this case.

REFERENCES

1. Kumari R, Laxmisha C, Thappa DM. Disseminated cutaneous rhinosporidiosis. Dermatol Online J. 2005;11(1):19.
2. Thappa DM, Venkatesan S, Sirka CS, Jaisankar TJ, Gopalkrishnan, Ratnakar C. Gopalkrishnan, Ratnakar C. Disseminated cutaneous rhinosporidiosis. J Dermatol. 1998;25(8):527-32.
3. Gonazalez G, Viada J, Escalona A, Na'quira N. Nasal rhinosporidiosis - four cases relate literature review. Int Arch Otorhinolaryngol. 2007;11:428-9
4. Karthikeyan P, Vijayasundaram S, Pulimoottil DT. A retrospective epidemiological study of rhinosporidiosis in a Rural Tertiary Care Centre in Pondicherry. J Clin Diagn Res. 2016;10(5):MC04-08.
5. Morelli L, Polce M, Piscioli F, et al. Human nasal rhinosporidiosis: an Italian case report. Diagn Pathol. 2006;1(1):25.
6. Girish N, Prathima. Rhinosporidiosis of lacrimal sac: a case report. Int Clin Pathol J. 2017;4(4):85-6.
7. Bhagat, A., Singla, K., Singh, K. and Heer, P., 2019. Ocular rhinosporidiosis. *QJM: An International Journal of Medicine*, 113(7), pp.497-498.
8. Tiwari R. Rhinosporidiosis: A Riddled Disease of Man and Animals. Advances in Animal and Veterinary Sciences. 2015;3(2s):54-63.
9. Aroor R, Gowda M, Bhat V, Bhandary S. Novel Approach to Rhinosporidiosis. An International Journal of Otorhinolaryngology Clinics. 2014;6(2):55-57.
10. Kameswaran S, Lakshmanan M. In: ENT disorders in a tropical environment. Kameswaran S, Kameswaran M, editors. (MERF Publications, Chennai), 1999; p. 19–34.
11. Sah BP, Chettri ST, Si S, Kandel DR, Ir D. Lacrimal sac rhinosporidiosis: an unusual case report. Am J Med Case Rep. 2014;2(4):84-6.
12. Watve JK, Mane RS, Mohite AA, Patil BC. Lacrimal sac Rhinosporidiosis. Indian J Otolaryngol Head Neck Surg. 2006;58(4):399-400.
13. Nerurkar NK, Bradoo RA, Joshi AA, Shah J, Tandon S. Lacrimal sac rhinosporidiosis: a case report. Am J Otolaryngol. 2004;25(6):423-5.
14. Thakur SKD, Sah SP, Badhu BP. Oculosporidiosis in Eastern Nepal: a report of five cases. Southeast Asian J Trop Med Public Health. 2002;33(2):362-4.
15. Belliveau MJ, Strube YNJ, Dexter DF, Kratky V. Bloody tears from lacrimal sac rhinosporidiosis. Can J Ophthalmol. 2012;47(5):e23-4.
16. Mithal C, Agarwal P, Mithal N. Ocular and adnexal rhinosporidiosis: the clinical profile and treatment outcomes in a tertiary eye care centre. Nepal J Ophthalmol. 2012;4(1):45-8.

SP-41

Study Between Microscopy, PCR and Rapid Diagnostic Tests for The Identification of Malarial Parasite - A Review

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Abstract: Aim & objective: To review the articles that Saponin hemolysis detection of malarial parasite in thick smear preparation is superior to Rapid diagnostic tests and PCR Background: Malaria detection has been done using thick and thin smears so far. But using Saponin hemolysis, thick smears can be done for malaria better microscopic detection. This technique also proven to be highly accurate than nonmorphological methods of detection including rapid diagnostic tests and PCR. The present study is a review article showed that when comparision between morphological and nonmorphological methods of detection of malaria is considered, microscopy thick smear by saponin hemolysis proven to be giving the best results. Various review and non-review articles were studied which were on malaria detection by microscopy and rapid detection methods and PCR. A brief review of those results was carefully studied and compared the results of microscopy with other methods. Conclusion-Microscopy, if performed by skilled personnel along with appropriate techniques, is much reliable as compared to the first response (RDT) in areas where parasite density is lower. A quick diagnostic test can be helpful in areas with large numbers of parasites as an alternative to routine smears.

Keywords: Malarial parasite, PCR, Saponin haemolysis, Rapid diagnostic test

INTRODUCTION

Thick film examination for malaria often is a challenge to detect malaria parasite because of many obscuring factors. Detecting malarial parasite on a quick detection method would be helpful in various fields like blood banks, hematology. There are various studies proved that saponin hemolysis release the pigments of malarial parasite and made it easy for detection. But this procedure has not gained widespread acceptance possibly due to the unawareness of the procedure, insufficient training. The present review article mainly emphasis on utilizing saponin hemolysis with centrifugation based on various studies. This review emphasized on the advantage of saponin hemolysis over other diagnostic tests[1]. There are four well-established species of the malaria parasite that infect humans, namely Plasmodium falciparum, P. vivax, P. ovale, and P. malariae. P. falciparum, the deadliest species, accounts for 99.7% of infections in Sub-Saharan Africa. P. vivax is the most common in the Americas and accounts for 75% of infections [2]. For Asia and Oceania, the number of P. falciparum and P. vivax infections are relatively equivalent. P. ovale and P. malaria are widely dispersed, but have low incidence [3]. An additional species, P. knowlesi, is a simian malaria parasite that is usually found in long-tailed and pig-tailed macaques. A systematic review on literature was conducted utilizing the PubMed online database & Google Scholar. Various review articles and non-review articles were screened by terminologies such as, 'malaria diagnostic tests', and 'diagnostic modalities of malaria'.According to Augustine U. Orjih Preethi Cherian Suad AlFadhli from Kuwait states that Modified Saponin utilization method can improve the detection of malaria and overcome the challenge[1]. With saponin hemolysis, detection of plasmodium in infected RBC'S increased from 0.7–2% to 65–97% after saponin hemolysis as proved by already published reports. However, those patients blood contained only 1-15 parasites per one field and showed 20–600 parasites when seen under the microscope after hemolysis by saponin is done . Saponin hemolysis helped to identify parasites in large volumes that can be identified in small volumes of blood and can be smeared. This concentration method helped us to detect malarial parasites in a short time under microscopic evaluation. In the current study, this method was compared to PCR for tests.Among other methods for malaria detection apart from microscopy, the methods are based on antigen capture. consume less time and don't require high skilled experience; however these are not highly sensitive or specific. Tests based on immunofluorescent antibody detection require a microscope which is not cost effective, and other techniques based on serology requiring reagents, may not be readily available in all countries. Immunological tests give a clue to history of previous exposure to malaria but not about past or current infection. Polymerase chain reaction (PCR) is an advanced research method in molecular analysis which is widely used for detection. of correct species. However, the instruments and reagents that are required are not cost effective and are not done in less time than microscopic screening of slides.As a procedure, Saponin Hemolysis powder (Sigma) is diluted with saline at pH 7.4, to obtain a concentration of 0.015% solution. It was rearranged daily to test the smear and stored in the refrigerator when not in use. Samples were placed at 1600 rpm, using a benchtop centrifuge. The steps included the following. First, 1 ml of the experimental sample was transferred to a sterile plastic 15ml centrifuge tube with a cap and centrifuged for 10 minutes after which the plasma was extracted. After the centrifugation series, the supernatant was quickly transferred to another centrifuge tube. The pellet from first centrifugation is mixed with 2-5 ml of saponin solution and frozen for about 5-15 minutes. The supernatant obtained following this was then re-incubated within 5 min and thus received pellet A. The supernatant along with pellet A was transferred to a tube containing the first supernatant followed by 30 min centrifugation thus receiving pellet B.Thin smear preparation of patients blood and pellet A are prepared on glass slides and air-dried at regular temperature. Thick smears of the patient's blood coated before and after saponin hemolysis are prepared in a 2 cm wide area containing 10 microliter of blood or pellet B in glass slides and dried at room temperature. They were fixed in methanol for 30 minutes before being stained with 10% Giemsa stain.

Current Malaria Diagnostic Options

Microscopy

Screening of thin and thick smears that are considered "gold standard" and is used to detect the malaria virus in the blood and thus direct appropriate treatment. One drop of blood is then taken from the patient with a finger stick or venipuncture. If venipuncture blood drop is used for blood collection, then it is spread on a slide immediately after collection to prevent prolonged exposure to anticoagulants in a collection tube that alters the morphology of the parasite. Thick smears are adjusted to place one to two drops of blood on a slide in one circle. Red blood cells are then lysed and various blood cells such as trophozoites, gametocytes and schizonts. Thin smears are used to check the morphology for identifying animal species and are repaired with a single layer of cells. The sensitivity and specificity of this method are 95% and 98%, respectively when polymerase chain reaction (PCR) is used for comparison. Of the 154 patients, 80 (51.9%) had a fever of ≥37.5 ° C. 106 patients (68.8%) were showing positivity in the first response®, 132 (85.7%) were screened by microscopy, and 121 (78.6%) were screened by PCR. Sensitivity, specificity, PPV, and NPV initial response compared to the smear evaluation method were 82.2%, 100.0%, 100.0%, and 34.3%, respectively, and compared with PCR, were 75.4%, 75.0 %, 95.3%, and 31.2%, respectively. Sensitivity, specificity, PPV, and NPV from smear evaluation compared to PCR were 92.3%, 50.0%, 90.91%, and 54.5%, respectively. There was a significant difference between RDT and smear evaluation procedure($P \leq 0.05$) according to AfomaMbanefo and the Nirbhay Kumar study [4].

Comparison of First Response® [Rapid Diagnostic Test] By Microscopy As "Gold Standard"

Of the 154 registered patients, 106 (68.8%) were diagnosed with HIV in both First Response® and microscopy while 25 (16.2%) patients were diagnosed without First Response® and microscopy According to Batwala et al. in 2010, which showed that RDT sensitivity was significantly higher than other strategies and was significantly better for <5-year-old children, i.e., 97.7% [95% confidence interval (CI): 88-99.9] compared to those ≥ 5 years, i.e. , 83.7% (95% CI: 69.3-93.2)[4]. Zero samples were obtained by First Response®-positive but microscope-negative while 23 (14.9%) first Response®-negative but with microscope-positive. Analysis data included sensitivity, specificity, PPV, and NPV were 82.17%, 100.00%, 100.00%, and 34.29% respectively. Significance tests (chi-square tests) showed that these were statistically significant when First Response® was compared with micrscopy method ($P \leq 0.05$).

Comparison of First Response® with polymerase chain reaction as "gold standard"

Of the 101 patients (65.6%) tested positive for both First Response® and PCR while 15 (9.7%) samples were found to be free of both First Response® and PCR. Samples were First Response® -positive but PCR-negative was 5 (3.2%) while those were First Response® -negative but PCR-positive were 33 (21.4%)[4]. Sensitive analysis, specificity, PPV, and NPV included were 75.37%, 75.00%, 95.28%, and 31.25%, respectively. Significance tests (chi-square test) did not show significant statistical differences when First Response® was compared with the PCR method ($P \leq 0.05$.

Comparison of microscopy and polymerase chain reaction as "gold standard"

Of the 154 patients, 120 (77.9%) were diagnosed with P. falciparum in both microscope and PCR methods and 12 (7.8%) were found to be free of both diagnostic methods. The samples tested positive for microscopy but without PCR were 12 (7.8%) while 10 (6.5%) were non-microscopy, and PCR-positive samples[4]. The sensitivity analysis, specificity, PPV, and NPV were 92.31%, 50.00%, 90.91%, and 54.55%, respectively. Significance tests (chi-square tests) showed no significant differences between microscopy and PCR methods ($P \leq 0.05$).

Stratification of parasitic density in thick blood smear and its association with rapid diagnostic testing, polymerase chain reactions

Of the 20 patients who showed no microscope 2 and 6 have received RDT and PCR, respectively. The parasite range of 101-1,000 had the highest positivity (75) and in 75, 72 and 73 patients had RDT and PCR, respectively. In terms of study area, patients from Nsukka District Hospital contributed 73.3%, 25.3%, and 0% of patients with a parasite count of 1-100, 101-1000, and> 1000 respectively.

DISCUSSION

According to a study by AfomaMbanefo and Nirbhay Kumar, the discovery of microscopy when considered as gold standard, PPV and First Response® specifications was very high. Specification (100%) and PPV (100%) showed higher percentages [1]. Harani et al. in 2006 reported a similar specification of 98.3% of P. falciparum uses an RDT kit but a predictable PPV of 78.0% has been identified. Sensitivity and NPV of 82.17% and 34.29% were lower than reported elsewhere.[2] When PCR was used as a gold standard, First Response® (RDT) showed lower sensitivity (75.37%) and clarity (75.00%) compared to microscopy but had a higher PPV of 95.28% and a lower NPV of 31.25%, similar to the findings compared to a microscope. On the other hand, microscopy showed higher sensitivity (92.3%), PPV (90.91%), and NPV (54.55%) with initial response but with a low specificity of 50.00%. The study also stated that First Response® consists of 101 (65.6%), 5 (3.2%), 15 (9.7%), and 33 (21.4%) samples true, false, negative, true, and false samples, respectively. 120 (77.9%), 12 (7.8%), 12 (7.8%), and 10 (6.5%), samples, respectively, by microscopy.[3] First Response® had more false false samples, i.e., 33 (21.4%) compared with microscopy, i.e., 12 (7.8%) but less

false, i.e., 5 (3.2%) than microscopy, i.e. , 12 (7.8%). . This proved that First Response® was more likely to detect falciparum infection in sick patients while microscopy may detect falciparum infection in patients without Plasmodium infection. This will lead to improper treatment which may lead to drug resistance. With an actual positive of 120 (77.9%), microscopy had a sensitivity of 92.3% and a PPV of 90.91%. On the other hand, RDT with 33 false negative (21.4%) and 5 false positives (3.2%) had low NPV of 31.25% and high PPV of 95.28%.[5] When microscopy was used as the gold standard, the specificity and PPV of First Response® were very high. The specificity (100%) and PPV (100%) was higher than reported elsewhere.[6,7] Harani *et al.* in 2006 reported a similar specificity of 98.3% for *P. falciparum* using an RDT kit but a lower predictive PPV of 78.0%.[8] According to Batwala et al. in 2010, which showed that RDT sensitivity was significantly higher than other strategies and was significantly better for <5-year-old children, i.e., 97.7% [95% confidence interval (CI): 88-99.9] compared to those ≥ 5 years, i.e., 83.7% (95% CI: 69.3-93.2)[9]. As RDTs might not be very sensitive in detecting malaria, especially in areas with varying transmission intensities as proven by some studies[12,13] and both RDT and microscopy have their limitations in detecting malaria[12,14,15] , there is a need to be more accurate and sensitive methods which can detect such as PCR in assessing the accuracies of these diagnostic methods inspite most of the studies used microscopy as the gold standard.[16,17,18] Few studies used Nested PCR as the gold standard instead of microscopy as suggested by Andrade *et al.* in 2010[11] and Batwala *et al.* in 2010.[9] Andrade *et al.* in 2010 showed that Nested PCR was the gold standard for diagnosis of malaria both symptomatic and asymptomatic in the Brazilian Amazon because it stated that major cases presented with major specificity while microscopy showed a low performance of 65.1% for correct diagnosis.[19] The study also revealed that the conversion time was 20 min, 45 min, and 1,440 min for First Response®, microscopy, and PCR, respectively. A typical malaria diagnosis, a PCR method with a transition time of 1,440 min. Structures specific to certain types such as RBC'S enlargement, the presence of Schüffner dots and Maurer tearing are easily identified by the unchanging RBC'S. Saponin binds to a membrane containing cholesterol and is a way of resisting saponin by P. falciparum -infected erythrocytes. Because the gametocytes of P. falciparum remains morphologically stable, a large amount of blood can be smearee to detect parasites in saponin hemolysis which could be possibly missed in thick and thin smears.[20]With only the gametocyte shape in thick blood smears one can differentiate the different malarial species and thus can be used as a direct marker. These infested erythrocytes are more pronounced in the periphery containing two chromatin dots in P. vivax, although very rarely. Also, a concentration of prepared saponin can be made in white blood cells such as neutrophils and monocytes that contain phagocytosed P. vivax parasites. The disadvantages of saponin hemolysis include: large amounts of blood are needed to obtain the best results, so they are not suitable for infants and young children. Preparing time can take longer than regular thick & thin smears as it requires centrifugation instrument which may not be readily available in poorly equipped laboratories.[21,22]

CONCLUSION

This technique of saponin hemolysis which is described in our study was initially developed for the purpose of eliminating the uninfected RBC's from the in-vitro Plasmodium falciparum cultures that was to be used for research purposes. Now this study proves that this can be used as a detecting technique for malaria. The present review study also indicates that the microscopy methods are higly sensitive and more preferable to PCR and RDT which require skills,time consuming and expensive. Also, saponin hemolysis method could be useful in malarial detection as the parasitic index increased after hemolysis by saponin technique. Microscopy, if performed by skilled personnel along with appropriate techniques, is much reliable as compared to the first response (RDT) in areas where parasite density is lower. Rapid diagnostic tests would be useful in areas with higher parasite density as an alternative to smear microscopy.

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CONFLICT OF INTEREST

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REFERENCES

1. Augustine U. Orjih Preethi Cherian Suad AlFadhli Department of Medical Laboratory Sciences, Faculty of Allied Health Sciences, Kuwait University, Kuwait, Med Princ Pract 2008;17:458–463
2. WHO. World Malaria Report 2019. 2019. Available online: https://www.who.int/publications-detail/worldmalaria-report-2019 (accessed on 16 January 2020).
3. Ashley, E.A.; Phyo, A.P.; Woodrow, C.J. Malaria. Lancet 2018, 391, 21–27. [CrossRef]
4. Ayogu EE, Ukwe CV, Nna EO. Assessing the reliability of microscopy and rapid diagnostic tests in malaria diagnosis in areas with varying parasite density among older children and adult patients in Nigeria. Journal of postgraduate medicine. 2016 Jul;62(3):150.
5. Hopkins H, Bebell L, Kambale W, Dokomajilar C, Rosenthal PJ, Dorsey G. Rapid diagnostic tests for malaria at sites of varying transmission intensity in Uganda. J Infect Dis. 2008; 197:510–8.

6. McMorrow ML, Masanja MI, Abdulla SM, Kahigwa E, Kachur SP. Challenges in routine implementation and quality control of rapid diagnostic tests for malaria--Rufiji District, Tanzania. Am J Trop Med Hyg. 2008; 79:385–90
7. Batwala V, Magnussen P, Nuwaha F. Are rapid diagnostic tests more accurate in diagnosis of Plasmodium falciparum malaria compared to microscopy at rural health centres? Maler J. 2010; 9:349.
8. Mbanefo A, Kumar N. Evaluation of malaria diagnostic methods as a key for successful control and elimination programs. Tropical Medicine and Infectious Disease. 2020 Jun;5(2):102.
9. Andrade BB, Reis-Filho A, Barros AM, Souza-Neto SM, Nogueira LL, Fukutani KF, et al. Towards a precise test for malaria diagnosis in the Brazilian Amazon: Comparison among field microscopy, a rapid diagnostic test, nested PCR, and a computational expert system based on artificial neural networks. Malar J. 2010; 9:117.
10. Hopkins H, Bebell L, Kambale W, Dokomajilar C, Rosenthal PJ, Dorsey G. Rapid diagnostic tests for malaria at sites of varying transmission intensity in Uganda. J Infect Dis. 2008; 197:510–8.
11. Wongsrichanalai C, Barcus MJ, Muth S, Sutamihardja A, Wernsdorfer WH. A review of malaria diagnostic tools: Microscopy and rapid diagnostic test (RDT) Am J Trop Med Hyg. 2007;77(Suppl):119–27.
12. Valéa I, Tinto H, Nikiema M, Yamuah L, Rouamba N, Drabo M, et al. Performance of OptiMAL-IT compared to microscopy, for malaria detection in Burkina Faso. Trop Med Int Health. 2009; 14:338–40.
13. Guthmann JP, Ruiz A, Priotto G, Kiguli J, Bonte L, Legros D. Validity, reliability and ease of use in the field of five rapid tests for the diagnosis of Plasmodium falciparum malaria in Uganda. Trans R Soc Trop Med Hyg. 2002; 96:254–7.
14. McMorrow ML, Masanja MI, Abdulla SM, Kahigwa E, Kachur SP. Challenges in routine implementation and quality control of rapid diagnostic tests for malaria--Rufiji District, Tanzania. Am J Trop Med Hyg. 2008; 79:385–90.
15. Harani MS, Beg MA, Khaleeq L, Adil SN, Kakepoto GN, Khurshid M. Role of ICT malaria immunochromatographic test for rapid diagnosis of malaria. J Pak Med Assoc. 2006; 56:167–71.
16. Tham JM, Lee SH, Tan TM, Ting RC, Kara UA: Detection and species determination by PCR: comparison with microscopy and with Parasight-F and ICT malaria Pf tests in a clinical environment. J Clin Microbiol 1999; 37:1269–1273.
17. Mason DP, Kawamoto F, Lin K, Laoboonchai A, Wongsrichanalai C: A comparison of two rapid field immunochromatographic tests to expert microscopy in the diagnosis of malaria. Acta Trop 2002; 82:51–59.
18. Cordray, M.S.; Richards-Kortum, R.R. Emerging nucleic acid-based tests for point-of-care detection of malaria. Am. J. Trop. Med. Hyg. 2012, 87, 223–230.
19. Bell DR, Wilson DW, Martin LB. False-positive results of a plasmodium falciparum histidine-rich protein 2-detecting malaria rapid diagnostic test due to high sensitivity in a community with fluctuating low parasite density. Am J Trop Med Hyg. 2005; 73:199–203.
20. Mens P, Spieker N, Omar S, Heijnen M, Schallig H, Kager PA. Is molecular biology the best alternative for diagnosis of malaria to microscopy. A comparison between microscopy, antigen detection and molecular tests in rural Kenya and urban Tanzania. Trop Med Int Health. 2007; 12:238–44
21. Bartoloni A, Sabatinelli G, Benucci M: Performance of two rapid tests for Plasmodium falciparum malaria in patients with rheumatoid factors. N Engl J Med 1998; 338:1075.
22. Cropley IM, Lockwood DNJ, Mack D, Pasvol G, Davidson RN: Rapid diagnosis of falciparum malaria by using Para Sight F test in travelers returning to the United Kingdom: prospective study. BMJ 2000; 321:484–485.

SP-42

A Rare Case of Norwegian Scabies in an Immunocompromised Infant

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Abstract: BACKGROUND: Scabies is caused by Sarcopetes scabei var hominis. It is an itchy rash caused due to infestation of eight mites called Sarcoptes scabiei which is small and has eight legs, burrows into skin. Patients usually presents with itchy lesions which gets worse at night Scabies spreads due to prolonged skin contact which is most common in school going children. Crusted scabies, also known as Norwegian scabies, occurs in elderly and immunocompromised individuals and will be very contagious. AIM: Our aim of this case report is to diagnose crusted scabies as early as possible to avoid deadly complication in immunocompromised individuals, early intervention and management will save the individual. CASE REPORT: Here we report a 10 months old girl who came to our hospital with pruritic Crusted Scabies, how they are diagnosed and treated will be discussed below.It is important to diagnose crusted scabies as early as possible as it may cause superadded infection and all family members should be treated and personal hygiene should be encouraged.

Key Words: Cursed scabies, Norwegian Scabies, Sarcopetes scabie var hominis, Immunocompromised infant, Permethrin

INTRODUCTION

Scabies is known as seven-year itch is caused by infestation of female mite sarcoptes scabiei var hominis which leads to burrowing and releases toxic substances leading to intense itching[1].The primary lesion is a burrow, which is a thread like serpentine line with a minute papule at the end. Secondary lesions are pustules, exzematous lesions and nodules. Lesions are usually seen in webs of hamds, ulnar aspect, elbow, axilla, genitalia, gluteal region[11].Crusted scabies known as Norwegian scabies is most severe and proliferation of mites will be rapid is characterized by crusted, nodules, hyperkeratotic papules and plaques. Itching may be present. It occurs in immunocompetent individuals like HIV infection and in those who have illness like leukemia, diabetes. It is rarely reported in healthy infants[2].

CASE REPORT

A 10 months old baby girl, came with complaints of severe itching and rash all over the body(fig-1) for the past 4 months. The skin is crusted and excoriation was present all over the body. At 7 months of age they went to a nearby hospital where she was misdiagnosed with secondary bacterial infection and was treated with topical mometasone cream for 2 weeks, oral antibiotics for 1week. Despite treatment, there was no improvement. As there was no improvement, the infant was referred to our hospital for further management. Birth history-was born as the first child who was born at 37 weeks, NVD, cried immediately and had no nicu admission. Post-natal history was uneventful. Normal developmental milestones attained appropriate for age. Immunized according to national immunization schedule according for age. Family history reveals that prior to child's onset of lesions both parents had multiple itchy papulo-erythematousrash over web spaces of fingers, but they did not have crusted lesions. They were diagnosed to have scabies and treated with permethrin.

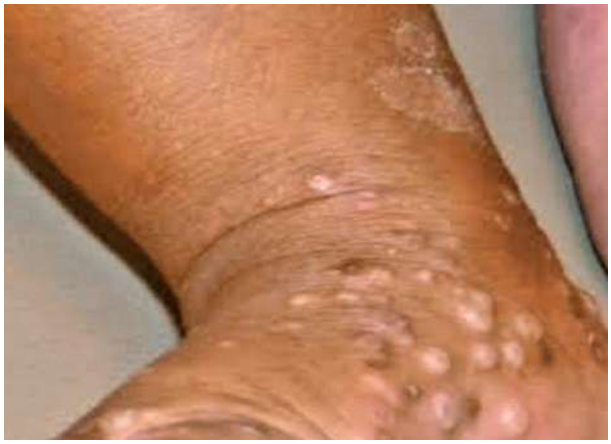

FIG-1 showing crusted papular lesion over the ankle

On general examination, the infant weight was 8.5kg, temperature 36.5°c, heart rate 86/ min and RR 26/min, multiple crusted, scaly, excoriated, erythematous patches and plaques were seen all over the body. A microscopy of skin scrapings shows scabies mites and eggs. All blood investigations were normal. Serology shows non-reactive to HIV, HBs Ag negative. Then the child was

treated with topical permethrin 5% over the entire body during night for 6 weeks. At the end there was complete resolution of the lesions and the infant recovered completely.

DISCUSSION

Crusted scabies is rarely reported in the paediatric population. It was first diagnosed in Norway by Danielssen and Boeck where millions of mites are found in patients with leprosy[3]. They are characterized erythroderma, hyperkeratosis, and crusting of the skin.[8] The Lesions will be seen over the soles, palms, and extensor surface of elbow.[4,5] When the crust is removed the skin will be smooth, red[8]. This type of scabies occurs in immunocompromised individuals and those who were on immunosuppressive drugs, malignancy, systemic illness, congenital disorders, in case of malnutrition, physical disability, graft versus host disease.[4,6,7]
In scabies, face is usually spared except in infants in whom face, scalp, palms and soles were involved. Secondary infections are common and are often treated with antibiotics. Scabicides are 5% permethrin, crotamiton 10%, benzyl benzoate 25%, ivermectin are used.[11,12,15]Baysaletalin 2004 reported a case who was 4½ month old with crusted scabies with 4-month h/o itchy erythematous, scaly, excoriated papules over trunk and hyperkeratotic papules over palms and soles. Gualdi et al in 2009 reported a case who was 3-month-old with atopic dermatitis from birth[1] treated with local application of betamethasone and gentamicin, but as the symptoms worsened she was started on Oralbetamethasone at a dose of 0.5 mg/day. In Microscopy examination of lesions, it revealed numerous scabies mites[14]. As the norwegian scabies is more contagious early diagnosis and management is important. In this case report it was misdiagnosed asatopic dermatitis associated with secondary bacterial infection. The differentiating feature is that atopic dermatitis presents with vesiculation, exudation, xerosis, scaling and sometimes lichenification.[10]The term milk crust refers to representation of yellow color crusts over the scalp that resembles Scalded Milk.[10]The nose is not involved and known as Head LightSign.[11]So the differential diagnosis for norwegian scabies is atopic dermatitis, seborrheic dermatitis, drug eruption, insect bites, contact dermatitis, ichthyosis vulgaris, Langerhans cell histiocytosis, cutaneous lymphoma, psoriasis.[13] The presence of scabies mite on skin scraping under microscopy is the differentiating feature.

CONCLUSION

Norwegian scabies should be diagnosed and treated early to prevent the complications. So, parents should be advised on personal hygiene and all members in the family should be treated. Adequate diet and close monitoring should be done.

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CONFLICT OF INTEREST

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REFERENCES

1. Gualdi, L. Bigi, G. Galdo, and G. Pellacani, "Neonatal Norwegian scabies: three cooperating causes," Journal of Dermatological Case Reports, vol. 3, no. 2, pp. 34–37, 2009
2. Banerji, "Scabies," Paediatrics& Child Health, vol. 20, no. 7, pp. 395–398, 2015.France, 1848.
3. D. G. Danielssen and W. Boeck, Treatment of Leprosy or Greek Elephantiasis, JB Balliere, Paris, France, 1848.
4. M. Gupta, M. Khalequzzaman, S. Jodele, J. Mortensen, M. A. Mueller, and A. Gupta, "Crusted scabies in a 15-year-old boy with congenitalneutropenia and myelodysplastic syndrome," Journal of Clinical Oncology, vol. 32, no. 12, pp. e49–e51, 2014.
5. A. Ferreira, A. Esteves, Y. Mahia, A. Rosmaninho, and A. Silva, "An itchy problem: a clinical case of crusted scabies," European Journal of Case Reports in Internal Medicine, vol. 4, no. 5, Article ID 000591, 2017.
6. F. C. d. R. Lima, A. M. M. Cerqueira, M. B. S. Guimarães, C. B. d. S. Padilha, F. H. Craide, and M. Bombardelli, "Crusted scabies due to indiscriminate use of glucocorticoid therapy in infant," AnaisBrasileiros de Dermatologia, vol. 92, no. 3, pp. 383–385, 2017.
7. Torrelo and A. Zambrano, "Crusted scabies in a girl with epidermolysisbullosa simplex," British Journal of Dermatology, vol. 142, p. 197-198, 2000.
8. V. Baysal, M. Yildirim, C. Turkman, B. Aridogan, and G. Aydin, "Crusted scabies in a healthy infant," Journal of the European Academy of Dermatology andVenereology, vol. 18, p. 188–190, 2004.
9. W. Rose, G. Rajendran, and J. Peter, "Crusted scabies," Indian Pediatrics, vol. 51, no. 8, p. 680.
10. K. C. Leung, K. L. E. Hon, and W. L. M. Robson, "Atopic dermatitis," Advances in Pediatrics, vol. 54, p 241–273
11. Balai M, Khare AK, Gupta LK, Mittal A, Kuldeep CM. Pattern of pediatric dermatoses in a tertiary care centre of South West Rajasthan. Indian Journal of Dermatology. 2012 Jul;57(4):275.
12. Johnston G, Sladden M. Scabies: diagnosis and treatment. Bmj. 2005 Sep 15;331(7517):619-22.
13. Mallongi A, Puspitasari A, Ikhtiar M, Arsunan AA. Analysis of Risk on the Incidence of Scabies Personal Hygiene in Boarding School Darul Arqam Gombara Makassar. Indian Journal of Public Health Research & Development. 2018 Apr 1;9(4).

14. Chouela E, Abeldaño A, Pellerano G, Hernández MI. Diagnosis and treatment of scabies. American journal of clinical dermatology. 2002 Feb;3(1):9-18.
15. Karthikeyan K. Treatment of scabies: newer perspectives. Postgraduate medical journal. 2005 Jan 1;81(951):7-11.

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Bilateral Congenital Glaucoma – A Rare Case Report

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Abstract: Glaucoma presenting in children is a blinding condition yet the lack of clinical data and a epidemiologic data is a huge burden to the society. Glaucoma in children presents with signs and symptoms that can be easily identified by a paediatrician or by the parents whereas in adults it is mostly asymptomatic and difficult to identify. It is recommended to examine the patient under anaesthesia to arrive at the diagnosis and which helps is making the plan of treatment. The primary mode of treatment is Surgery while medical management has a limited role in the treatment of glaucoma. Although Goniotomy or trabeculotomy ab externo is done in for congenital glaucoma, Primary combined trabeculotomy–trabeculectomy offers better quality of life. The new Classification system of paediatric glaucoma is given by the childhood glaucoma research network (CGRN). Early diagnosis and timely therapeutic intervention leads and proper refraction correction leads to a better outcome and decreased morbidity. In Children with decreased vision, Treatment of residual vision and visual rehabilitation should be done with a follow-up for lifetime.

KEYWORDS: Congenital glaucoma, trabeculotomy, trabeculectomy and combined trabeculotomy– trabeculectomy

CASE REPORT

One day old newborn male baby, delivered via emergency LSCS. It was a full term delivery with birth weight of 2.330kg in Sree Balaji medical college and hospital on 24th September 2019.The delivery was uneventful and the general condition of both mother and baby were stable. Ophthalmologists were called to the NICU for an opinion regarding the bilateral bluish discoloration of the baby's eyes. On examination of the baby there was bilateral bluish discoloration of the cornea, along with increased corneal diameter. Due to the diffused edema, details of structures posterior to cornea could not be perceived. Under local anesthesia corneal diameter and IOP were measured.

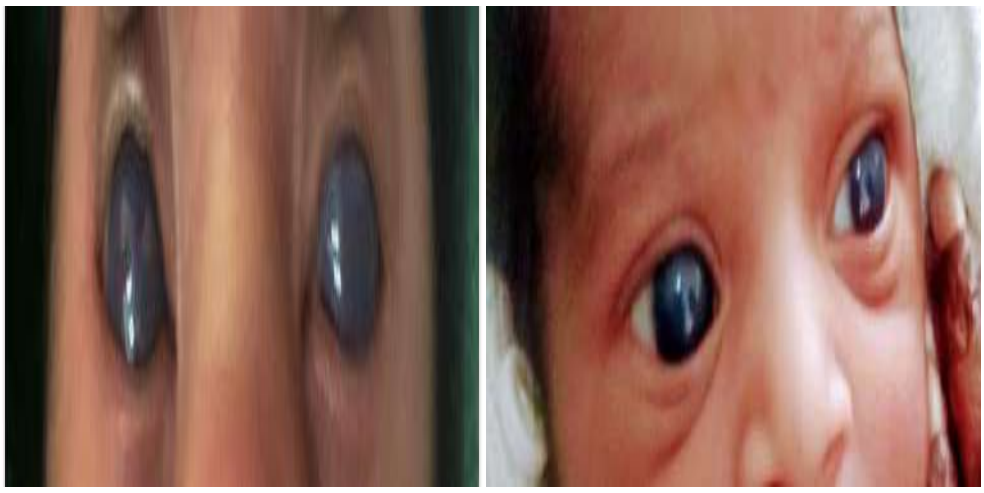


Fig 1 and 2: bilateral bluish discoloration of the cornea

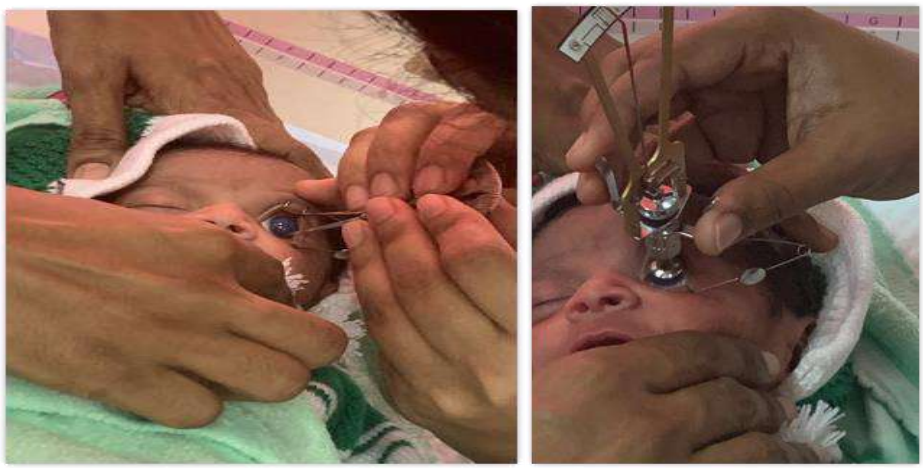
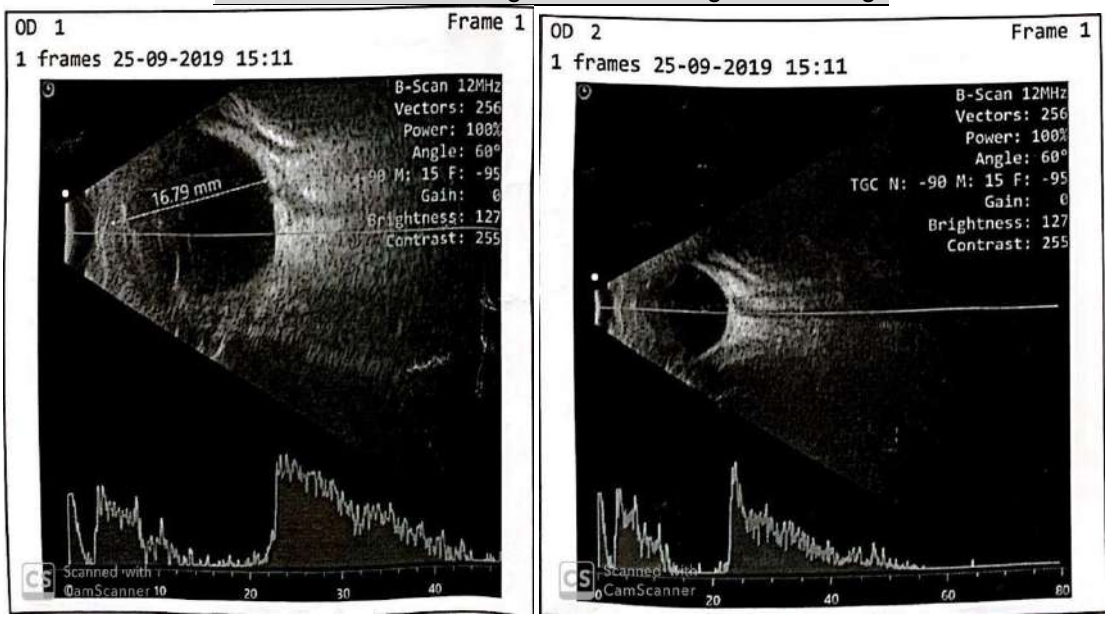


Fig 3 and 4: measurement of IOP and corneal diameter under LA

Table 1: Measurement of IOP and corneal diameter			
		Right eye	Left eye
Corneal diameter	Horizontal	12mm	12mm
	Vertical	11mm	11mm
IOP	5.5gm	40.0 mmHg	41.5 mmHg
	7.5gm	36.0 mmHg	35.8 mmHg



5: (USG) – Bscan was done to rule out posterior segment pathology

MANAGEMENT OF THE BABY

The baby was started on a topical beta blocker eye drops and antibiotics. Opinion was sorted with the glaucoma specialist with for the treatment of congenital glaucoma. After examination and evaluation of the baby under anesthesia trabeculotomy was performed first in the right eye and then in the left eye at a later point. The baby is on regular follow up with the ophthalmologist and the paediatrician.

Fig 8: post trabeculotomy of the right eye

Fig 9: post trabeculotomy of both eyes

FAMILY HISTORY

On taking detailed Family history, the grandmother revealed she had a twin delivery and both her daughters (which include the baby's mother) had similar complaints at birth.They underwent a certain surgical procedure back then which was around 40 days post birth, that on ocular examination appears to be peripheral iridotomy. Most probably by Scheie's procedure. On examination of the mother, there was a very apparent alternating divergent squint (exotropia), in right eye there is an updrawn pupil, whereas in left eye, a peripheral iridotomy scar is seen in the 7'o clock position. Bedside visual acuity both eyes were >5/60.

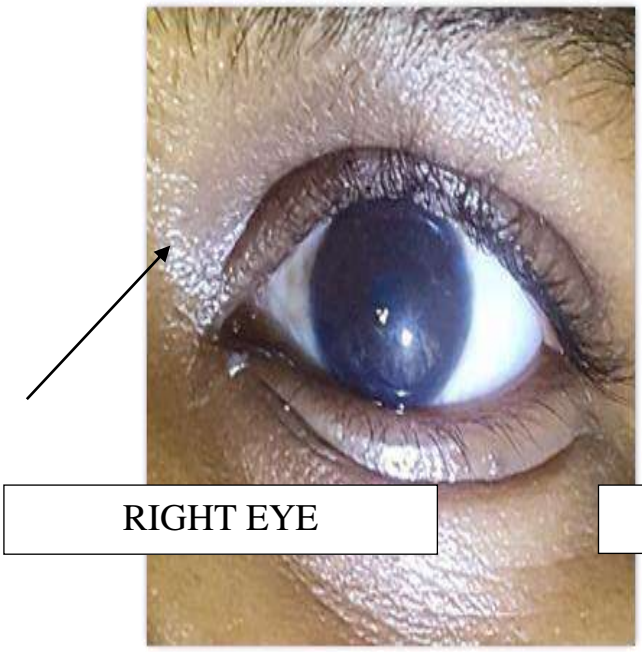


Fig 6: right eye of the mother showing updrawn pupil

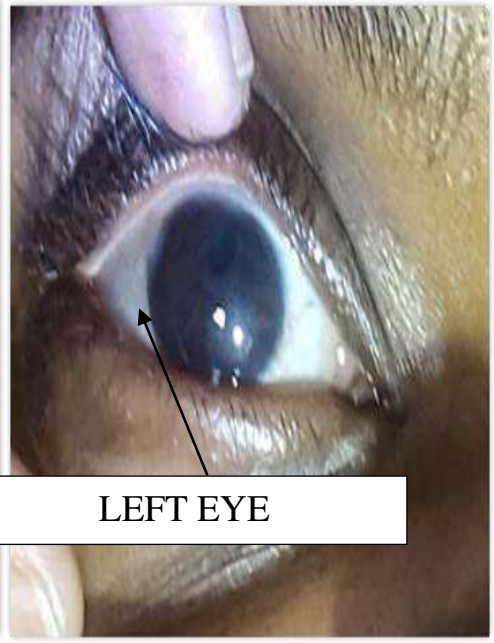


Fig7: left eye of the mother showing a peripheral iridotomy scar

DISCUSSION

Primary Congenital Glaucoma

Congenital glaucoma accounts for 38% of all childhood glaucoma's.1 It is Most common form of paediatric glaucoma. In Andhra Pradesh, India the incidence was 1 in 3300 showed a large scale study .3 Congenital glaucoma (Primary) is rare, it has an incidence of 1: 10000 population. It can be classified as

- Congenital Glaucoma -True : (40%) in which Intra Ocular Pressure is raised during the intrauterine life;
- Glaucoma - Infantile : (55%) presents before 3 years of age
- Glaucoma - Juvenile, it is the least common, usually presents between 3 to 16 years

Improper development of the AC angle leads to impaired outflow of aqueous fluid leading to development of Primary congenital glaucoma. The prognosis depends of factors like severity and age of onset of diagnosis. The Classical triad of congenital glaucoma includes epiphora, photophobia, and blepharospasm. 65% patients are male with bilateral involvement (70%) and 80 % of them are diagnosed within the first year of birth. Descemet breaks, oedema of the cornea and optic neuropathy, and eventually buphthalmos - large eye, and amblyopia - lazy eye leads to visual impairment. Due to raised IOP before 3 years of age, stretching of the eye leads to buphthalmosia. The increased visualization of uvea leads to blue appearance of sclera. Curvilinear healed breaks in descemet membrane are called hab striae, complications include myopia and lens subluxation.We have a very small interval or a window period where these children can be successfully treated. If not, the disease can be potentially blinding.The current standard of care involves Medical Therapy (prior to angle surgery) as first line therapy usually in the form of Beta-blocker + pilocarpine. Medical therapy after incisional surgery includes prostaglandin analogue/ beta-blocker as first line and Carbonic anhydrase inhibitor as second. There is increase risk of central depression in infants who are medicated with Alpha-agonist, hence it is contraindicated. Systemic agents are usually needed only temporarily prior to surgery.Medical therapy helps to clear the cornea to aid examination and surgical intervention. Definitive treatment is surgical intervention. The first two and the most relevant two surgical procedures devised for PCG are goniotomy and trabeculotomy. Goniotomy is the procedure that channels a route for the drainage of aqueous through Schlemm's canal by incision made in the trabecular meshwork under direct visualization.It has a success rate of 80 %.[8, 9] In India, goniotomy is technically impossible because most patients present with clouding .[3]Trabeculotomy involves an ab external approach to create a direct communication disrupting the tissue between Schlemm's canal and the anterior chamber. It can be done in an eye with corneal opalescence. It has a Success rate of 87% to 92% for the cases when the surgery is done before 1 year of age.10 Traeculectomy may be useful after angle surgery. Primary trabeculectomy is not the recommended first-line procedure in congenital glaucoma because it has high incidence of complications and lower success rate in normalizing intraocular pressure. 50% of them fail in the initial five – ten years.12, 13 Surgeons have advocated added trabeculectomy whenever there is incomplete canalization in cases of trabeculotomy with equivocal results.14Usually amenable to surgical treatment, good results are possible if the disease is diagnosed and appropriately treated in time. Ninety eight percent of the PCG require surgical treatment.6 Addition of antifibrotics further increases the success rates as proven in many Indian studies.14,15 Glaucoma drainage devices GDDs to be used in paediatric glaucoma was first introduced by Netland and Walton.20 In a study by Beck et el21 comparing trabeculectomy with GDDs, 20.8% success was noted in trabeculectomy group (mean follow-up 11.5 months) while 71.7% success was noted in the aqueous shunt devices group (mean follow-up 31.5 months). Refractory paediatric glaucoma remains a difficult condition to tackle. Glaucoma drainage devices have proven to be more predictable and safer procedure when compared to the conventional angle

surgery. Regular follow up and IOP monitoring, corneal diameter and other parameters is required long term, Aggressive treatment of Amblyopia and refractive error is required.

Genetics of Childhood Glaucoma

The most cases of primary congenital glaucoma are sporadic.10% - 40% are familial and there is an association with consanguineous marriages. Autosomal recessive pattern of inheritance is seen in familial cases with a variable expression and penetrance of 40% - 100%.16Three loci for PCG have been found GLC3A (2p21), GLC3B (1p36), and GLC3C (14q24.3). Two candidate genes: CYPB1 and LTBP2 have also been implicated.17.18 the majority of congenital glaucoma map to GLC3A locus on chromosome 2 (2p21). Mutations in the CYP1B1 are noticed in different ethnic groups and have been a part the pathogenesis. Various distinct mutations were identified in the coding region of CYP1B1 in patients of PCG-affected families, of which many mutations are novel in the Indian population.19 Families linked to these loci display autosomal recessive inheritance pattern. Genetic counseling is to be advices to the at-risk families and this will help in the prevention of PCG-related blindness.

CONCLUSION

The paediatric eye has its own inherent difficulties. This combined with the failure to report the symptoms, makes these children the most challenging scenarios that the ophthalmologist may encounter. Prompt diagnosis and intervention at the right time leads to the a good outcome and a life with less morbidity.

CONFLICT OF INTEREST

Conflict of interest declared none.

REFERENCES

1. Taylor RH, Ainsworth JR, Evans AR, Levin AV. The epidemiology of pediatric glaucoma: the Toronto experience. Journal of American Association for Pediatric Ophthalmology and Strabismus. 1999 Oct 1;3(5):308-15.
2. Fung DS, Roensch MA, Kooner KS, Cavanagh HD, Whitson JT. Epidemiology and characteristics of childhood glaucoma: results from the Dallas Glaucoma Registry. Clinical Ophthalmology (Auckland, NZ). 2013;7:1739..
3. Dandona L, Williams JD, Williams BC, Rao GN. Population-based assessment of childhood blindness in southern India. Archives of ophthalmology. 1998 Apr 1;116(4):545-6.
4. Sood D, Rathore A, Sood I, Singh G, Sood NN. Long-term outcome of combined trabeculotomy–trabeculectomy by a single surgeon in patients with primary congenital glaucoma. Eye. 2018 Feb;32(2):426-32.
5. Gupta PC, Singh RR, Pandav SS, Ram J. Rhexis fixation of intraocular lens in a case of buphthalmos with cataract. Nepalese Journal of Ophthalmology. 2016 Dec 12;8(1):71-3.
6. Mukkamala L, Fechtner R, Holland B, Khouri AS. Characteristics of children with primary congenital glaucoma receiving trabeculotomy and goniotomy. Journal of Pediatric Ophthalmology & Strabismus. 2015 Nov 1;52(6):377-82.
7. Møller PM. Goniotomy and congenital glaucoma. Acta ophthalmologica. 1977 Jun;55(3):436-42.
8. Broughton WL, Parks MM. An analysis of treatment of congenital glaucoma by goniotomy. American journal of ophthalmology. 1981 May 1;91(5):566-72.
9. Allen L, Burian HM. Trabeculotomy ab externo: a new glaucoma operation: technique and results of experimental surgery. American journal of ophthalmology. 1962 Jan 1;53(1):19-26.
10. Harms H, Dannheim R. Epicritical consideration of 300 cases of trabeculotomy'ab externo'. Transactions of the ophthalmological societies of the United Kingdom. 1970 Jan 1;89:491-9.
11. Grover DS, Smith O, Fellman RL, Godfrey DG, Butler MR, De Oca IM, Feuer WJ. Gonioscopy assisted transluminal trabeculotomy: an ab interno circumferential trabeculotomy for the treatment of primary congenital glaucoma and juvenile open angle glaucoma. British Journal of Ophthalmology. 2015 Aug 1;99(8):1092-6.
12. Fulcher T, Chan J, Lanigan B, Bowell R, O'Keefe M. Long-term follow up of primary trabeculectomy for infantile glaucoma. British Journal of Ophthalmology. 1996 Jun 1;80(6):499-502.
13. Cadera W, Pachtman MA, Cantor LB, Ellis FD, Helveston EM. Filtering surgery in childhood glaucoma. Ophthalmic surgery. 1984 Apr 1;15(4):319-22.

14. Mandal AK, Bhatia PG, Bhaskar A, Nutheti R. Long-term surgical and visual outcomes in Indian children with developmental glaucoma operated on within 6 months of birth. Ophthalmology. 2004 Feb 1;111(2):283-90.
15. Mandal AK, Gothwal VK, Nutheti R. Surgical outcome of primary developmental glaucoma: a single surgeon's long-term experience from a tertiary eye care centre in India. Eye. 2007 Jun;21(6):764-74.
16. Sarfarazi M, Stoilov I. Molecular genetics of primary congenital glaucoma. Eye. 2000 May;14(3):422-8.
17. Akarsu AN, Turacli ME, Aktan SG, Barsoum-Homsy M, Chevrette L, Sayli BS, Sarfarazi M. A second locus (GLC3B) for primary congenital glaucoma (Buphthalmos) maps to the 1p36 region. Human molecular genetics. 1996 Aug 1;5(8):1199-203.
18. Stoilov IR, Sarfarazi M. The third genetic locus (GLC3C) for primary congenital glaucoma (PCG) maps to chromosome 14q24. 3. Investigative ophthalmology & visual science. 2002 Dec 1;43(13):3015-.
19. Panicker SG, Reddy AB, Mandal AK, Ahmed N, Nagarajaram HA, Hasnain SE, Balasubramanian D. Identification of novel mutations causing familial primary congenital glaucoma in Indian pedigrees. Investigative ophthalmology & visual science. 2002 May 1;43(5):1358-66.

SP-44

Shaft of Tibia and Fibula Fracture - A Case Report

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Abstract: Tibia and fibula shaft fracture is most common fracture of the paediatric age group which requires appropriate diagnosis and treatment to minimize complications. Paediatric bones are so fragile that may cause to break and there will be intact periosteum. Bone composition and mechanism such as osteoblast the builder of the bone and osteoclast the cutter and remodeling of the bone. Tibia and fibula fracture occur mostly due to trauma to the leg occured due to bending and torsional forces acting like direct or indirect trauma. Diagnosis is always made clinically and radiologically. Boys are more common than girls in this case. The most common causes for this trauma such as pedestrian or road traffic accidents. Tibia is the triangular shaped bone and the most common bone to get injured. It is the most weight bearing part of the lower limb. Once diagnosed the treatment should be started with immobilization of the limb and adequate care should be taken to prevent from complications. Various modalities of treatment are available such as kirschner nail, external fixation, flexible intramedullary nail and plating .in this study we had a patient where he stained trauma road traffic accident and sustained injury to the left leg. The patient came following the trauma first the limb was immobilised to prevent the complications with pop and the patient was posted for Titanium elastic nailing system and protocol according to it was followed. Most paient come with pain, bruises and inability to walk. Complications are uncommon also include deformity, growth arrest, nonunion and compartment syndrome.

Keyword: Tibia, Fibula, TENS, Compartment syndrome.

INTRODUCTION

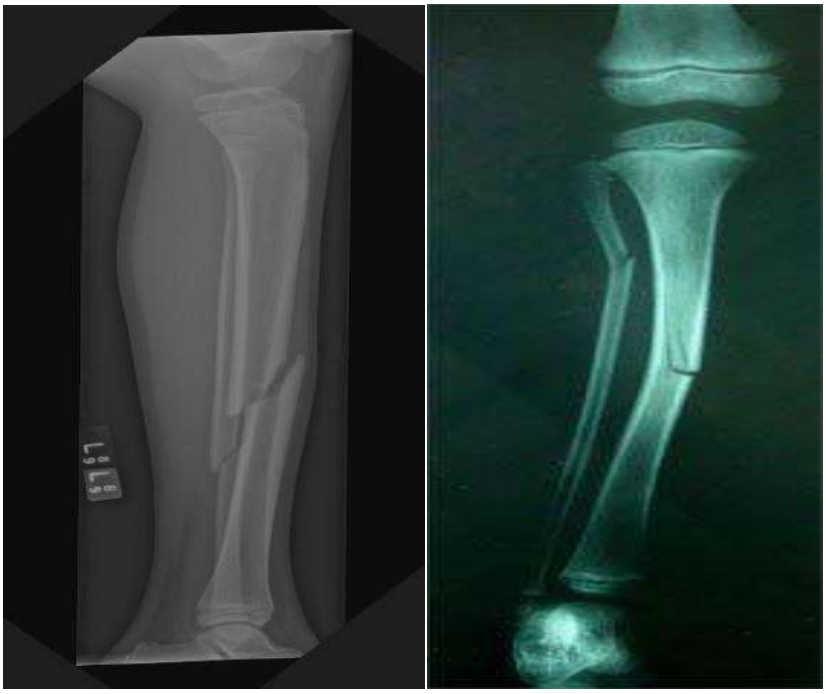
Pediatric Tibial Shaft Fractures are the third most common long bone fracture in children.20% of all the paediatric fractures occurs in tibia. Most of these fractures constitute 30% between the age group of 10 to 20 only.[1,2] Tibia is a triangular shaped bone with apex anteriorly that broadens distally the anteromedial border is subcutaneous. Rarely isolated fibula fracture can also been seen in poly trauma patients.[3] Child abuse is important and taken into account for most fractures. Diagnosis is always made clinically and radiologically. Boys are more common than girls in this case. The most common causes for this trauma such as pedestrian or road traffic accidents. Tibia is the triangular shaped bone and the most common bone to get injured. It is the most weight bearing part of the lower limb. Once diagnosed the treatment should be started with immobilization of the limb and adequate care should be taken to prevent from complications. Various modalities of treatment are available such as kirschner nail, external fixation, flexible intramedullary nail and plating .in this study we had a patient where he stained trauma road traffic accident and sustained injury to the left leg. Stiffness and deformity is encountered in many of these fractures. Primary treatment consist of manipulation and pop application for the fracture, but individualized based on age, modalities, type of fracture and neurovascular injury.

Classification
Paediatric tibial and fibula shaft fracture

Incomplete
Greenstick fracture of the tibia and fibul

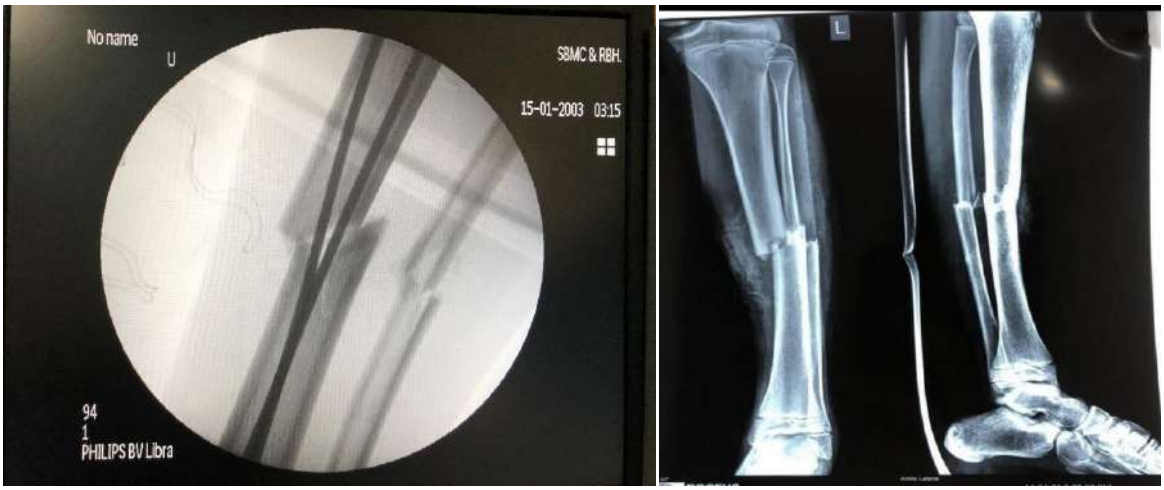
Complete
Complete fracture of the tibia with or without ipsilateral fibula fracture or plastic deformity

Tibial Spiral Fracture
Nondisplaced spiral fracture of the tibia with intact fibula
The figure shown above shows xrays of the tibia and fibula fracture the first picture shows incomplete tibia fracture and the second image shows complete fracture tibia fracture of the mid shaft .

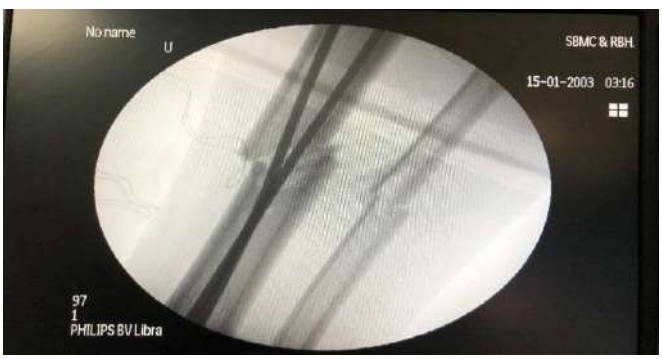


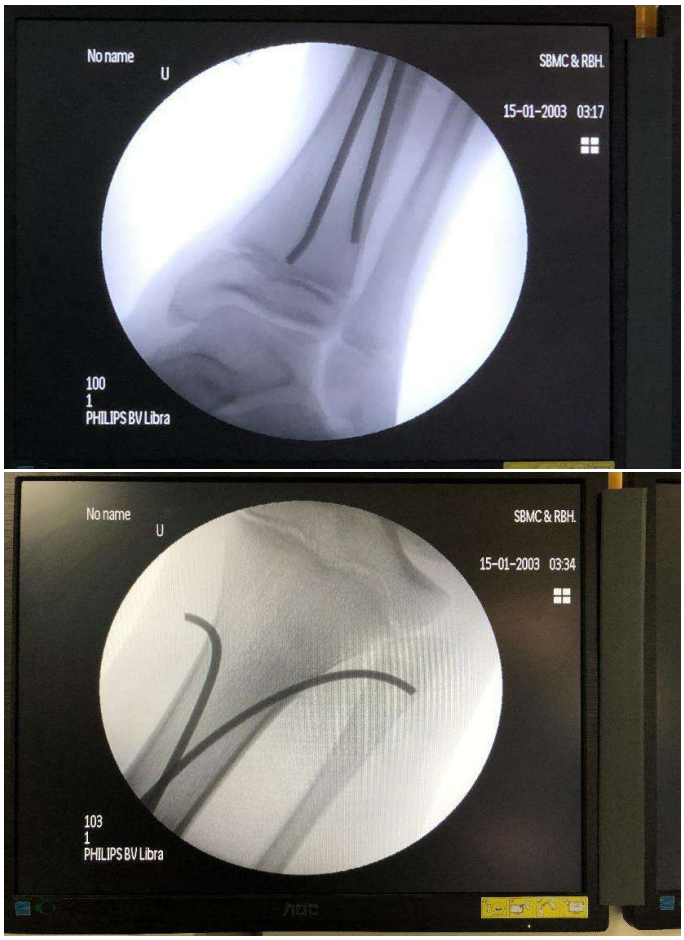
CASE REPORT

A 13 year old boy presented to our opd with history of RTA and sustained injury to the left leg. Pain was unable to bear weight on his leg. The pain was chronic in onset, progressive in nature, initiated with trivial trauma. No history of any comorbidities, no history of previous surgeries. There was obvious deformity present and limb length discrepancy. Dorsalis pedis and posterior tibial pulse felt after reducing the fracture and temporary pop application. He was planned for closed reduction with TENS nail for tibia. Under c-arm guidance 3mm TENS nail was passed from proximal aspect of tibia, 2cm below the metaphyseal area on adjacent sides. Dorsalis pedis and posterior tibial pulse felt after reducing the fracture and TENS nailing for tibia. Patient was said to non weight bearing and was put pop and discharged and asked for serial followup.

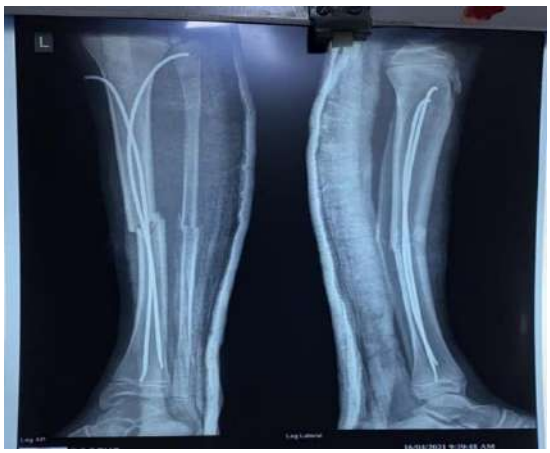


The first xray pic shows the tibi and fibula fracure following the trauma, the second picture shows the intraop image of the tens procedure done for the fracture displacement in this case.





The above images are the intraop images of the TENS nailing procedure proximal part, middle and the distal parts of the tibia in the c-arm images as seen above that was taken during the procedure.



This is the post operative xray of the patient with tibia, fibula fracture where TENS nailing done for tibia.

DISCUSSION

Treatment of shaft of tibia and fibula fracture in paediatric is very important which requires management as individual based on age, size and nature of trauma and associated soft tissue injury. Most of these fractures are treated non-operatively in a cast.[4,5] Compartment syndrome is rare when compared to adult but examined regularly to prevent it Paediatric bones are so fragile that may cause to break and there will be intact periosteum. Bone composition and mechanism such as osteoblast the builder of the bone and osteoclast the cutter and remodeling of the bone. Tibia and fibula fracture occur mostly due to trauma to the leg occured due to bending and torsional forces acting like direct or indirect trauma. Diagnosis is always made clinically and radiologically. Boys are more common than girls in this case. The most common causes for this trauma such as pedestrian or road traffic accidents. This study is done in such a way that all the patients treated well with the treatment of choice and patient where symptomatically improved and all patients x-ray showed signs of union.

CONCLUSION

After closed reduction and TENS nail fixation for tibia, patient improved well and mobilized well. Distal pulse was checked regularly; no signs of deformity seen and patient symptomatically improved hence discharged and asked to follow up regularly. Regular follow up of the patient was done and patient showed good signs of healing. First patient post operatively showed signs of recurvatum but coorection done with regular follow up with pop and patient mobilized well. The patient is in serial follow up and the patient is able to walk and o his regular activities like before. Hereby I would like to say that tibia fracture in paediatric age group with Tens nailing shows good signs of healing and is a good treatment modality of choice.

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None

CONFLICT OF INTEREST

Conflict of interest declared none.

REFERENCES

1. Nimick CJ, Collman DR, Lagaay P. Fixation orientation in anklefractures with syndesmosis injury. J Foot Ankle Surg.2013;52(3):315–8.
2. Sagi HC, Shah AR, Sanders RW. The functional consequenceof syndesmotic joint malreduction at a minimum 2-yearfollow-up. J Orthop Trauma. 2012;26(7):439–43.
3. Moore JA Jr, Shank JR, Morgan SJ, Smith WR. Syndesmosisfixation: a comparison of three and four cortices of screwfixation without hardware removal. Foot Ankle Int.2006;27(8):567–72.
4. Virani, S.R.; Dahapute, A.A.; Bava, S.S.; Muni, S.R. Impact of negative pressure wound therapy on open diaphyseal tibial fractures: A prospective randomized trial. J. Clin. Orthop. Trauma 2016, 7, 256–259.
5. Schepers T. To retain or remove the syndesmotic screw: areview of literature. Arch Orthop Trauma Surg.2011;131(7):879–83.
6. Song P, Pu LLQ. The Soleus Muscle Flap: An Overview of Its Clinical Applications for Lower Extremity Reconstruction. Ann Plast Surg. 2018 Dec;81(6S Suppl 1):S109-S116.
7. Bezstarosti H, Van Lieshout EMM, Voskamp LW, Kortram K, Obremskey W, McNally MA, Metsemakers WJ, Verhofstad MHJ. Insights into treatment and outcome of fracture-related infection: a systematic literature review. Arch Orthop Trauma Surg. 2019 Jan;139(1):61-72.
8. Sivasundaram L, Trivedi NN, Gatta J, Ning AY, Kim CY, Mistovich RJ. Demographics and Risk Factors for Non-Accidental Orthopedic Trauma. Clin Pediatr (Phila). 2019 Jun;58(6):618-626.
9. Stella M, Santolini E, Sanguineti F, Felli L, Vicenti G, Bizzoca D, Santolini F. Aetiology of trauma-related acute compartment syndrome of the leg: A systematic review. Injury. 2019 Jul;50 Suppl 2:S57-S64.
10. Kazley J, Jahangir A. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Aug 14, 2021. Tibia Diaphyseal Fracture.
11. Rounds AD, Burtt KE, Leland HA, Alluri RK, Badash I, Patel KM, Carey JN. Functional outcomes of traumatic lower extremity reconstruction. J Clin Orthop Trauma. 2019 Jan-Feb;10(1):178-181.
12. Bauer J, Orendi I, Ladenhauf HN, Neubauer T. [Bony knee injuries in childhood and adolescence]. Unfallchirurg. 2019 Jan;122(1):6-16.
13. Zhang Z, Swanson WB, Wang YH, Lin W, Wang G. Infection-free rates and Sequelae predict factors in bone transportation for infected tibia: a systematic review and meta-analysis. BMC Musculoskelet Disord. 2018 Dec 13;19(1):442.

SP-45

Spontaneous Conception after Hysterosalpingography in Infertile Woman with Bilateral Tubal Factors

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Abstract: Hysterosalpingography (HSG) has become a commonly performed examination due to recent advances and improvements in, as well as the increasing popularity of, reproductive medicine. HSG plays an important role in the evaluation of abnormalities related to the uterus and fallopian tubes. Uterine abnormalities that can be detected at HSG include congenital anomalies, polyps, leiomyomas, surgical changes, synechiae, and adenomyosis. Tubal abnormalities that can be detected include tubal occlusion, salpingitis isthmica nodosum, polyps, hydrosalpinx, and peritubal adhesions. Hysterosalpingography (HSG) is an outpatient fluoroscopy method that assesses the uterine endometrial cavity and fallopian tube patency. It is controversial whether this procedure enhances fertility. Some studies show a slight increase in fertility lasting about 3 months after a normal HSG. However, most doctors perform HSG only for diagnostic reasons. HSG is considered a very safe procedure. However, there is a set of recognized complications, some serious, which occur less than 1% of the time like infection, iodine allergy, radiation exposure etc but in some cases, HSG may have a therapeutic effect. Nevertheless, HSG remains a valuable tool in the evaluation of the uterus and fallopian tubes. Radiologists should become familiar with HSG technique and the interpretation of HSG images. We present the case of a 30-year-old parous with recurrent pelvic inflammatory disorder with secondary infertility; with post antibiotic hysterosalpingography resolved the issue which unknowingly lead to pregnancy in this woman.

Keywords: Hysterosalpingography, Bilateral Tubal Factors, Conception

INTRODUCTION

15% of reproductive-aged couples around the world suffers with infertility.3.9 to 16.8% is the overall prevalence of primary infertility according to World Health Organisation.[1,2] 1/3rd cases constitute female factors, 1/3rd cases due to male factors and the rest 1/3 remains unexplained factors for infertility.[3,4] In the larger part of infertile females, a tubal factor predominates. The causes for tubal obstruction include tubal endometriosis, chlamydial and tuberculous infections, salpingitis, previous tubal ectopic pregnancy, peritubal adhesions due to previous history of appendicectomy, ovarian, uterine or adnexal operations.[5,6] Other treatable causes such as tubal hindrance due to debris, fine adhesions or indeed unexplained tubal spasm which can be managed with selective fallopian tube catheterization. Proximal tubal blockage is seen in 10–20% of HSG examinations. Hysterosalpingography (HSG) is routinely performed in the mid-follicular phase of a woman's menstrual cycle for cavity and tubal patency assessment as a part of the infertility screening. Hysterosalpingography (HSG) is a minimally invasive radiographic imaging of the uterine cavity and fallopian tubes involving the injection of contrast media with fluoroscopic visualization. It is often used as the first line of assessment in a context of subfertility, commonly performed within the first 5–12 days of the menstrual cycle after the cessation of menstrual flow.

CASE PRESENTATION

A case of a 30-years-old woman presenting with recurrent pelvic inflammatory disease in the past 3 years and was on treatment on and off. She had her last menses on the 15/07/21 with obstetric score of G3P2L2 with 2 full term normal vaginal deliveries with good antenatal and postnatal period. She attended infertility OPD in view of being anxious to conceive with a recurrent history of vaginal infections for which she and her husband was treated with antibiotics.2 months later the patient came with similar complaints and was treated for the same. Hysterosalpingography done post treatment for tubal assessment on 1/07/21 showed a bilateral proximal tubal obstruction. Ultrasound Dating scan dated 1/9/21, showed a 6-week pregnancy and the patient was registered under our antenatal care.

DISCUSSION

Our patient's significant risk factors for tubal occlusion were age, sexually transmitted diseases, and Mycoplasm infections. Indeed, a number of demographic factors have been linked to low rates of conception among women, including advanced reproductive age and the consequent tubal damage caused by other sexually transmitted infections (STIs). Direct injury and subsequent morphological and functional abnormalities of the reproductive organs, including the cervix, endometrium, fallopian tubes, and ovaries, are causative for STI-related infertility, leading to tubal occlusions and ectopic pregnancies.[7,8] It's possible that a spontaneous pregnancy due to a tubal obstruction is fortunate, although there are various possible explanations. Because HSG analysis is operator-dependent, a cornual spasm could have been misinterpreted as a tubal occlusion in our circumstance.[9] Muscle spasms can produce temporary tubal occlusion, preventing contrast from filling a tube that is otherwise patent. Cornual occlusion is distinguished by a sharp or irregular cornual margin, whereas spasm is distinguished by a rounded smooth cornual margin, albeit these differences can be difficult to discern definitely. The tube in spasm may usually be distinguished from one that is permanently clogged by repeating the examination at a later time. Apart from other causes,

salpingitis isthmica nodosa, can be a cause of tubal obstruction in our case. On HSG, several, tiny diverticula extending from the isthmic lumen into the wall are seen, which is frequently referred to as tubal diverticulosis. In 80% of instances, this disease affects both tubes and is commonly accompanied with proximal ampullary dilatation or blockage. Although the disease is strongly linked to infertility and ectopic pregnancy, the tubes may remain patent in certain rare cases, allowing for spontaneous pregnancy. Tubal patency test therapeutic benefits are well explored in the literature, and HSG is no exception.[10] The passage of the contrast medium via the tubes can break small adhesions or flush mucus plugs through the tubes in women with tubal obstruction, leading to tubal repermeabilization and spontaneous pregnancy, as in our instance. The HSG was done on this woman using iodinated water-soluble contrast material. The role of the type of the contrast medium in the occurrence of spontaneous pregnancy after hysterosalpingography has been widely addressed. Water-soluble contrast, on the other hand, is the most commonly used media because it produces better pictures and prevents the other underlying complications. Although HSG being an easily accessible investigation for infertility evaluation it has its own drawbacks like false positives, dye allergies, infections.

CONCLUSION

In conclusion, HSG is a safe, low cost and a well-tolerated procedure for tubal assessment, which should be performed at the end of the infertility investigation protocol. Tubal infertility can be diagnosed with hysterosalpingography (HSG), a low-cost, diagnostic and therapeutic test. The high false-positive rate for proximal tubal occlusion (39 %), probably due to tubal spasm, demonstrates the importance of antiperistaltic agents and delayed imaging. Most cases of pregnancy are found to be spontaneous without tubal or uterine surgery. This concludes therapeutic effect of the HSG procedure, i.e., improved patency of the fallopian tube because of the flushing during the examination which increases the chances of conception without the patient undergoing further surgeries.

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Conflict of interest declared none.

REFERENCES

1. Kilcoyne A, O'Shea A, Gervais D, Lee S. Hysterosalpingography in endometriosis: performance and interpretation. Abdominal Radiology. 2020;45(6):1680-1693.
2. Schankath A, Fasching N, Urech-Ruh C, Hohl M, Kubik-Huch R. Hysterosalpingography in the workup of female infertility: indications, technique and diagnostic findings. Insights into Imaging. 2012;3(5):475-483.
3. Maheux-Lacroix S, Boutin A, Moore L, Bergeron M, Bujold E, Laberge P et al. Hysterosalpingosonography for diagnosing tubal occlusion in subfertile women: a systematic review with meta-analysis. Human Reproduction. 2014;29(5):953-963.
4. Preutthipan S, Linasmita V. A prospective comparative study between hysterosalpingography and hysteroscopy in the detection of intrauterine pathology in patients with infertility. Journal of Obstetrics and Gynaecology Research. 2003;29(1):33-37.
5. Chunyan G, Bin P, Ping Y, Yue Z, Yang X, Hongju T et al. Assessment of the Influence on Spontaneous Pregnancy of Hysterosalpingo-Contrast Sonography. BioMed Research International. 2018; 2018:1-8.
6. Eisenberg R. Obstetrical and gynecologic radiology. In: Eisenberg R, editor. Radiology, an illustrated history. St Louis, Mo: Mosby; 1992. pp. 347–363.
7. Hull M, Glazener C, Kelly NJ, Conway DI, Foster PA, Hinton RA, et al. Population study of causes, treatment, and outcome of infertility. BMJ. 1985; 291:1693–1697.
8. 11. Akande V, Garas A, Cahill D. The effect of diclofenac and paracetamol on pregnancy and implantation rates in infertile women undergoing IVF treatment. Journal of Obstetrics and Gynaecology. 2006;26(8):785-787.
9. Strandell A, Bourne T, Bergh C, et al. The assessment of endometrial pathology and tubal patency: a comparison between the use of Ultrasonography and X-ray hysterosalpingography for the investigation of infertility patients. Ultrasound Obstet. Gynecol. 1999; 14:200–204.
10. Dmowski WP, Lesniewicz R, Rana N, Pepping P, Noursalehi M. Changing trends in the diagnosis of *endometriosis*: a comparative study of women with pelvic *endometriosis* presenting with chronic pelvic pain or infertility. Fertil Steril. 1997; 67:238–243.
11. Eisenberg R. Obstetrical and gynecologic radiology. In: Eisenberg R, editor. Radiology, an illustrated history. St Louis, Mo: Mosby; 1992. pp. 347–363.
12. Hull M, Glazener C, Kelly NJ, Conway DI, Foster PA, Hinton RA, et al. Population study of causes, treatment, and outcome of infertility. BMJ. 1985; 291:1693–1697.

13. Akande VA, Hunt LP, Cahill DJ, Ford WCL, Jenkins JM. A cohort study of the prediction of Chlamydia infection causing subfertility, the value of treatment independent management and prognosis for pregnancy in 1119 women following laparoscopy. Presented at British Congress of Obstetrics and Gynaecology; Birmingham. 2001.
14. Strandell A, Bourne T, Bergh C, et al. The assessment of endometrial pathology and tubal patency: a comparison between the use of Ultrasonography and X-ray hysterosalpingography for the investigation of infertility patients. Ultrasound Obstet. Gynecol. 1999; 14:200–204.
15. Dmowski WP, Lesniewicz R, Rana N, Pepping P, Noursalehi M. Changing trends in the diagnosis of endometriosis: a comparative study of women with pelvic endometriosis presenting with chronic pelvic pain or infertility. Fertil Steril. 1997; 67:238–243.
16. Chatman DL, Ward AB. Endometriosis in adolescents. J Reprod Med. 1982; 27:156–160.
17. Koninckx PR, Ide P, Vandenbroucke W, Brosens IA. New aspects of the pathophysiology of endometriosis and associated infertility. J Reprod Med. 1980;24:257–260.

SP-46

Spontaneous Conception in Mosaic Turner Syndrome- An Atypical Instance

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Abstract: As a result of chromosomal abnormalities of X chromosome, 1 in each 2500 females is born with turner syndrome. Ovarian dysgenesis causing infertility is the most common feature. Patients with 45, X/46, XY mosaicism present with a variety of phenotypes ranging from most commonly mixed gonadal dysgenesis to others such as phenotypic males, genital ambiguity, Turner syndrome, and women with normal female secondary sex characteristics. Turner syndrome presents with bilateral streak gonads, whereas mixed gonadal dysgenesis describes those presenting with an absent or abdominal streak gonad on one side and a normal or dysgenic testis on the other. The phenotype in a 45, X/46, XY mosaic patient likely depends on the distribution of mosaicism percentage in different tissues which has been shown to differ between blood and gonadal tissue. The most common scenario is that a girl has only one X chromosome in all of her cells. However, some girls with Turner syndrome have a full or partial absence of the X chromosome in only some of their cells. When an individual has a different chromosomal content in his/her cells, it is called mosaicism. This case report is with respect to an unconstrained pregnancy in 27-year-old female, primi with mosaic Turner disorder. At 38weeks 4days, came with spontaneous labour pains with a past history noted for mosaic Turner syndrome diagnosed in her teenage years after a symptomatic assessment for short stature, although she reported regular menstrual cycles starting at age 14. She delivered a healthy female weighing 2650gm. 5.6% of Turner Syndrome patients conceive spontaneously. This case uncovers the significance of having an elaborate review about preserving fertility and managing possible pregnancy comorbidities with Turner syndrome patients and in case of family history at the time of diagnosis.

Keywords: Mosaic Turner disorder, Karyotyping, Fertility

INTRODUCTION

One of the most common chromosomal disorders is Turner syndrome and also the 2nd most common cause of chromosomal abnormalities that results in pregnancy losses. 1 in each 2500 female newborn is affected by turner's syndrome.[1-3] Clinically patients present with short stature, webbed neck, gonadal dysgenesis, renal abnormalities and many other features in line. Accelerated follicular atresia is one of the causes of primary amenorrhea, premature ovarian failure and infertility in these patients which majorly affects their lives. [4,5] Several aberrations of X chromosome include deletions, translocations, and duplications. 55% of patients have complete loss of X chromosome, 24.5% have a partial deletion of X chromosome, 20% carry varying degrees of mosaicism, most commonly a 45, X0/ 46XX karyotype.[6,7] Majority of the women who conceive spontaneously belong to a group of mosaic karyotype. Hence for these patients who attained menarche, timely counselling for preservation of fertility and evaluation of the factors contributing for successful pregnancy till term.

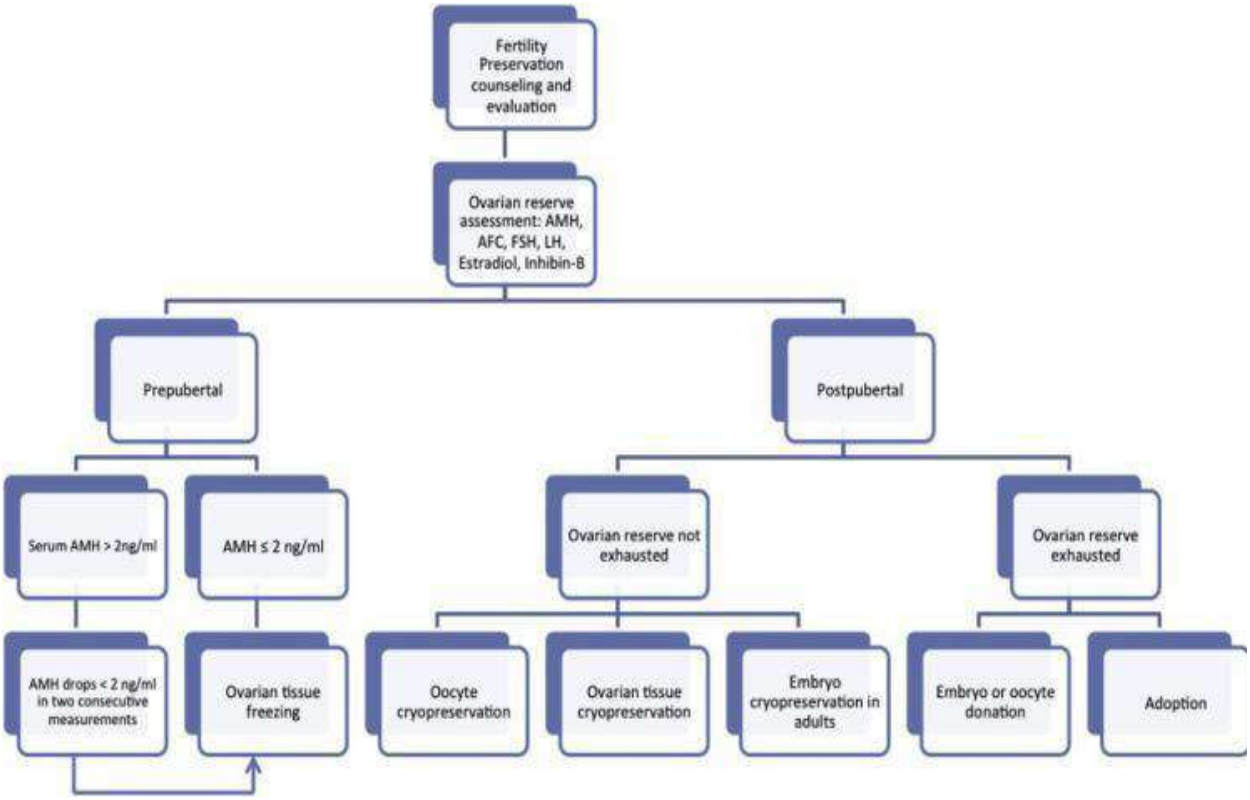
CASE REPORT

A 27year old, Pimi, 38weeks+4days presented to our outpatient clinic with complaints of lower abdominal pain radiating to the back. Patient was worked up for short stature at age 14 when her karyotyping showed the mosaic variant of Turner syndrome. According to the history patient attained menarche at 13years old, had regular menstrual cycles that lasted 5-6 days with mild to moderate flow. The patient came with 38weeks+4days gestational age with spontaneous conception and all the routine investigations were done which were in normal range. 1st trimester screening done which was normal. Anomaly scan showed no fetal anomalies. Growth scan and interval growth scan done were corresponding to gestational age. Since mother is a case of turner syndrome, the patient was advised to do fetal echocardiography so as to rule out cardiac anomalies which showed no cardiac anomalies. Patient underwent a full term normal vaginal delivery and delivered a healthy 2650g male baby with Apgar scores of 9 and 9 at 1 and 5 minutes respectively.

DISCUSSION

Fertility is a major concern for patients with Turner Syndrome especially when it's a mosaic variant.[9,10] Considering the challenges, counselling around the high probability of future infertility with the patients themselves and their guardians, it is obvious that guardians often feel they lack adequate information about fertility related issues with their daughters.[11,12] This feeling is compounded by the social disgrace of infertility, desire for their daughter to have biological children, and their misfortune of not having biological grandchild.[13,14] These challenges make it even more critical for healthcare professionals to accomplice with parents and encourage fertility- related issues pertaining to patients with turner's syndrome. To start with- hormonal and growth hormone therapy should be initiated in the early growing years.[15, 16] Recent recommendation is to start low dose oestrogen therapy or estradiol transdermal patch at 12 years so as to promote secondary sexual characteristics, reproductive organ development and mineralisation and adequate growth of the bone.[17,18] Long term hormonal therapy is a

must in patients with turner syndrome so as to prevent the patient from cardiovascular disease, osteoporosis and other related problems.[19,20] There are different methods of preserving fertility which are as follows:



Ovarian tissue cryopreservation is which hundreds or thousands of follicles and oocytes are retrieved, via a laparoscopy and frozen for later transplantation, most commonly used method in pre-pubertal girls.[21, 22] In turners, mosaic variant with functional ovaries, normal hormonal levels of follicle stimulating hormone, anti-Mullerian hormone and spontaneous puberty is a criterion for successful pregnancy outcome and also are good candidates for autologous in vitro fertilization if need be.[23] Pregnancy in turner's syndrome patients is a high-risk condition indicating high morbidity and mortality, so should be monitored closely and meticulously.

CONCLUSION

Future fertility is an important consideration for patients with Turner syndrome. Accurate and early diagnosis of 45, X/46, XY mosaicism can allow for counseling about reproductive potential and pursuing pregnancy with in vitro fertilization with donor egg and/or gestational surrogacy. Successful pregnancy outcomes have occurred in patients with 45, X/46, XY mosaicism as well as 46, XY gonadal dysgenesis following oocyte donation and in vitro fertilization, although most of the reported cases were delivered by cesarean section. Although this patient's uterus measured only 4.4 × 2.3 × 1.2 cm on ultrasound, there is no contraindication to pregnancy due to uterine size. Uterine size is likely a result of low estrogen status rather than an indication that the uterus is unfit to carry a pregnancy to term. In summary, this case demonstrates that Turner syndrome with low level mosaicism may be missed by conventional karyotype. Some females diagnosed with Swyer syndrome may actually have Turner syndrome with low level mosaicism. Approximately 70-80% of patients diagnosed with Swyer syndrome do not have SRY mutations and Turner syndrome with low level mosaicism may be the actual cause of gonadal dysgenesis in some of these patients. In cases where conventional karyotype results do not closely match the clinical presentation, FISH analysis for low level mosaicism may be informative.

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REFERENCES

1. Practice Committee of the American Society for Reproductive Medicine. Increased maternal cardiovascular mortality associated with pregnancy in women with Turner syndrome. Fertility and sterility. 2012 Feb 1;97(2):282-4.
2. Wasserman D, Asch A. Reproductive medicine and Turner syndrome: ethical issues. Fertility and sterility. 2012 Oct 1;98(4):792-6.
3. Hewitt JK, Jayasinghe Y, Amor DJ, Gillam LH, Warne GL, Grover S, Zacharin MR. Fertility in T urner syndrome. Clinical endocrinology. 2013 Nov;79(5):606-14.
4. Gug C, Rațiu A, Navolan D, Drăgan I, Groza IM, Păpurică M, Vaida MA, Mozoș I, Jurcă MC. Incidence and spectrum of chromosome abnormalities in miscarriage samples: a retrospective study of 330 cases. Cytogenetic and Genome Research. 2019;158(4):171-83.
5. Oktay K, Bedoschi G, Berkowitz K, Bronson R, Kashani B, McGovern P, Pal L, Quinn G, Rubin K. Fertility preservation in women with Turner syndrome: a comprehensive review and practical guidelines. Journal of pediatric and adolescent gynecology. 2016 Oct 1;29(5):409-16.
6. Viuff M, Skakkebæk A, Nielsen MM, Chang S, Gravholt CH. Epigenetics and genomics in Turner syndrome. InAmerican Journal of Medical Genetics Part C: Seminars in Medical Genetics 2019 Mar (Vol. 181, No. 1, pp. 125-132).
7. Reindollar RH. Turner syndrome: contemporary thoughts and reproductive issues. InSeminars in reproductive medicine 2011 Jul (Vol. 29, No. 04, pp. 342-352). © Thieme Medical Publishers.
8. Folsom LJ, JS Fuqua. Reproductive issues in women with turner syndrome. Endocrinol Metab Clin North Am.2015;44: 723-737.
9. Gravholt CH, Andersen NH, Conway GS, Dekkers OM, Geffner ME, Klein KO, Lin AE, Mauras N, Quigley CA, Rubin K, Sandberg DE. Clinical practice guidelines for the care of girls and women with Turner syndrome: proceedings from the 2016 Cincinnati International Turner Syndrome Meeting. European journal of endocrinology. 2017 Sep 1;177(3): G1-70.
10. Telvi L, Lebbar A, Del Pino O, Barbet JP, Chaussain JL. 45, X/46, XY mosaicism: report of 27 cases. Pediatrics. 1999 Aug 1;104(2):304-8.
11. Knudtzon J, Aarskog D. 45, X/46, XY mosaicism. European journal of pediatrics. 1987 May;146(3):266-71.
12. Méndez JP, Ulloa-Aguirre A, Kofman-Alfaro S, Mutchinick O, Fernández-del-Castillo C, Reyes E, Pérez-Palacios G. Mixed gonadal dysgenesis: clinical, cytogenetic, endocrinological, and histopathological findings in 16 patients. American journal of medical genetics. 1993 May 15;46(3):263-7.
13. Chang HJ, Clark RD, Bachman H. The phenotype of 45, X/46, XY mosaicism: an analysis of 92 prenatally diagnosed cases. American Journal of Human Genetics. 1990 Jan;46(1):156.
14. Wolff DJ, Van Dyke DL, Powell CM. Erratum: Laboratory guideline for Turner syndrome (Genetics in Medicine (2010) 12: 1 (52-55)). Genetics in Medicine. 2012 Feb;14(2):281.
15. Schoemaker MJ, Swerdlow AJ, Higgins CD, Wright AF, Jacobs PA. Cancer incidence in women with Turner syndrome in Great Britain: a national cohort study. The lancet oncology. 2008 Mar 1;9(3):239-46.
16. Klein KO, Rosenfield RL, Santen RJ, Gawlik AM, Backeljauw PF, Gravholt CH, Sas TC, Mauras N. Estrogen replacement in Turner syndrome: literature review and practical considerations. The Journal of Clinical Endocrinology & Metabolism. 2018 May;103(5):1790-803.
17. Bardeguez AD, De Ziegler D, Weiss G. Multifetal pregnancy in a gonadal dysgenesis mosaic. Obstetrics and gynecology. 1990 Sep 1;76(3 Pt 2):502-4.
18. Chen MJ, Yang JH, Mao TL, Ho HN, Yang YS. Successful pregnancy in a gonadectomized woman with 46, XY gonadal dysgenesis and gonadoblastoma. Fertility and sterility. 2005 Jul 1;84(1):217-e5.
19. Kan AK, Abdalla HI, Oskarsson T. Two successful pregnancies in a 46, XY patient. Human reproduction (Oxford, England). 1997 Jul 1;12(7):1434-5.
20. Sauer MV, Lobo RA, Paulson RJ. Successful twin pregnancy after embryo donation to a patient with XY gonadal dysgenesis. American Journal of Obstetrics & Gynecology. 1989 Aug 1;161(2):380-1.
21. Sánchez-Moreno I, Canto P, Munguía P, De Leon MB, Cisneros B, Vilchis F, Reyes E, Méndez JP. DNA binding activity studies and computational approach of mutant SRY in patients with 46, XY complete pure gonadal dysgenesis. Molecular and cellular endocrinology. 2009 Feb 27;299(2):212-8.
22. Scherer G, Held M, Erdel M, Meschede D, Horst J, Lesniewicz R, Midro AT. Three novel SRY mutations in XY gonadal dysgenesis and the enigma of XY gonadal dysgenesis cases without SRY mutations. Cytogenetic and Genome Research. 1998;80(1-4):188-92.

SP-47

Stage 3 Idiopathic AVN of Femur Treated by Core Decompression, Bone Grafting and Autologous Bone Marrow Implantation: A Case Report

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Abstract: Avascular necrosis or osteonecrosis is a state resulting from impairment of vascular supply to the femoral head. It usually affects people from the 3rd to 5th decade of life. It ultimately leads to collapse of the head of the femur. Often, we come across various aetiologies that leads to the causation of this condition. A prominent one among various types on the basis of the cause is the idiopathic type. This condition is an indication for about 15% of total hip arthroplasties. Late diagnosis or presentation at later stages usually lands patients in THA. But when preservation of femoral head is considered, other options can be tried. Here we present a case of a 46-year-old male who had come to our OPD with a 5 months history of hip pain. He was diagnosed to be a case of idiopathic AVN of hip (Ficat and Arlet stage III) and was managed by core decompression by three 3.2mm drillings followed by a cancellous bone graft. Patient was followed up till 1 year which showed a radiologically regressed lesion. Management of a late stage of AVN may require a total hip arthroplasty. If total hip arthroplasty is performed on a young patient, a need for revision arthroplasty arises, which is unavoidable owing to increasing life expectancy. There are literatures that shows patients who had stage III osteonecrosis (Steinberg classification; subchondral lucency, without collapse) managed with core decompression having excellent outcomes (no requirement of surgery) for a period up to 10 years without any intervention. So decompression can be considered in patients with late stage of the disease. a core decompression can be attempted for a even late stage of AVN in association with bone marrow implantation and bone grafting which may widen the time gap before a total hip arthroplasty.

Keywords: Avascular necrosis, core decompression, grafts, Ficat & Arlet Staging

INTRODUCTION

Avascular necrosis, otherwise called osteonecrosis, is a condition characterized by impairment of osseous blood circulation. Its incidence differs from regions. On a major scale trauma is the major risk factor for development of AVN.[1,2] Various factors have been identified to be in relation to the development of osteonecrosis like steroid overuse, excess alcohol consumption, Gaucher's disease, haemoglobinopathies and people who are exposed to sudden change in barometric pressure like deep sea divers. The common pathogenesis in the above-mentioned conditions is directed to a variation in the fat content or framework of bone marrow, with a substantial increase in the intraosseous pressure and depletion in vascular supply to the trabeculae of the bone. In the former situation the vascularity to the head of the femur is disturbed when the retinacular vessels going through the surface of the neck of femur are cut off as a result of displacement of head of femur following a fracture, or excessive stretching when the head of the femur is dislocated from the acetabular surface[3]. Avascular necrosis of the head of femur is a condition that severely damages the hip joint in patients who are in the third to fifth decades of life. The main consequence of avascular necrosis is the segmental collapse of the head of femur. Outcome of the existing condition is predicted by the scale and site of the original necrotic lesion in the area where the weight is transmitted. The outcome of treatment of avascular necrosis of the head of femur is significantly more successful if diagnosed at initial stages of the disease in terms of hip preservation. To evaluate patients with avascular necrosis about six different classification systems have been devised. Even though there are various staging systems; there is not a single classification system for establishing the degree and site of the involvement in the head of the femur as well as the acetabulum[4]. The system used here in this case report is Ficat and Arlet staging as it has been widely used. Stages I and II are considered to be initial stages of osteonecrosis whereas stages III and IV are regarded as late stages. Several treatment procedures have been recommended for treating initial stages of osteonecrosis. These procedures include core decompression, vascularised fibular grafts, valgus osteotomy, non-vascularised bone grafts, as well as hip resurfacing. Use of growth factors and bone marrow cell implantation which can be used as alternatives to surgical management is still in trial stages. The most common surgery for treating initial stages of osteonecrosis of the femoral head is core decompression of the hip. The prognosis is totally dependent on early detection and most effective treatment plan, with stage I patients having better clinical outcomes than stage 3 patients. Here we present a case report of late stage avascular necrosis of the hipjoint (Ficat and Arlet Stage III) managed by core decompression of the hip and cancellous iliac crest bone grafting and followed up for 1 year showing radiological features of regression.

CASE REPORT

A 46-year-old man came to our outpatient department with a 5 months history of left hip pain. Pain aggravated on walking or even bearing weight on the affected side. Pain was not relieved by rest or analgesics. There was no history of night cries. The pain was chronic in onset, progressive in nature, initiated by trivial trauma before the onset. He had no history of fever, weight loss or loss of appetite. He had no prior history of any medical comorbidity. No prior history of steroid intake, surgeries or fractures around the hip. He is a non-smoker and had never consumed alcohol in his life. Pain interfered with his daily routine. He experienced difficulty in squatting and sitting cross legged. He had a limb with an antalgic type of gait. There was no obvious

deformity, limb length discrepancy or muscle wasting. Anterior joint line and greater trochanter were not tender. Bitrochanteric compression was pain free. There were no palpable swellings. Thigh segment and leg segment was measured to be 51cms and 42cms respectively on both sides. Mid-thigh circumference and calf circumference was measured to be 53cms and 37cms respectively bilaterally. Flexion was 80degree pain free bilaterally. Extension was possible up to 20-degree pain free. Internal rotation was 10degree on the unaffected side and had a painful 5 degree on the affected side. External rotation, adduction and abduction were 45-degree, 25 degree and 35 degree respectively which were painless. Sectoral sign was positive. He had no signs of any distal neurovascular deficit. X-ray of pelvis and MRI showed signs of avascular necrosis of left hip with degenerative changes Ficat and Arlet stage III, ARCO stage III, MITCHELL stage D. He was planned for Core decompression with cancellous iliac crest bone graft. A 3-4 cm incision was made. Three 3.2mm guide wires were passed; at the centre, antero superior and posterior aspect under C arm guidance. Reaming was done with a 4mm cannulated reamer. 3 ml of bone marrow and a cancellous bone graft was harvested from the left iliac crest. Post operative period was uneventful. Patient was encouraged on non-weight bearing mobilization from postoperative day 2. He was discharged on day 10 of surgery. He was followed up at 4 weeks, 3 months, 6 months and 1 year of surgery which showed improvement in his condition with resolution of symptoms on every follow up with features of radiological regression.

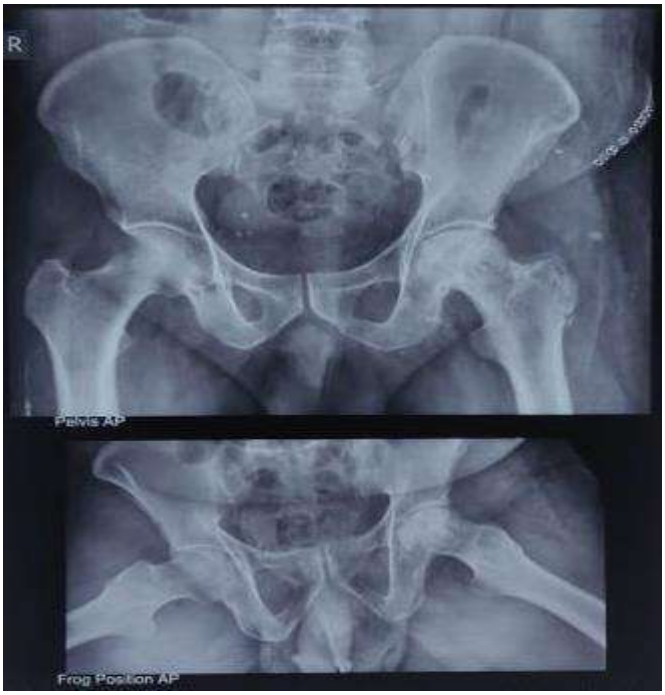

Fig 1- Preop X ray of Pelvis showing left femoral head flattening with moderate depression (2- 4mm). More than 15 percent collapse is noted (Steinberg).

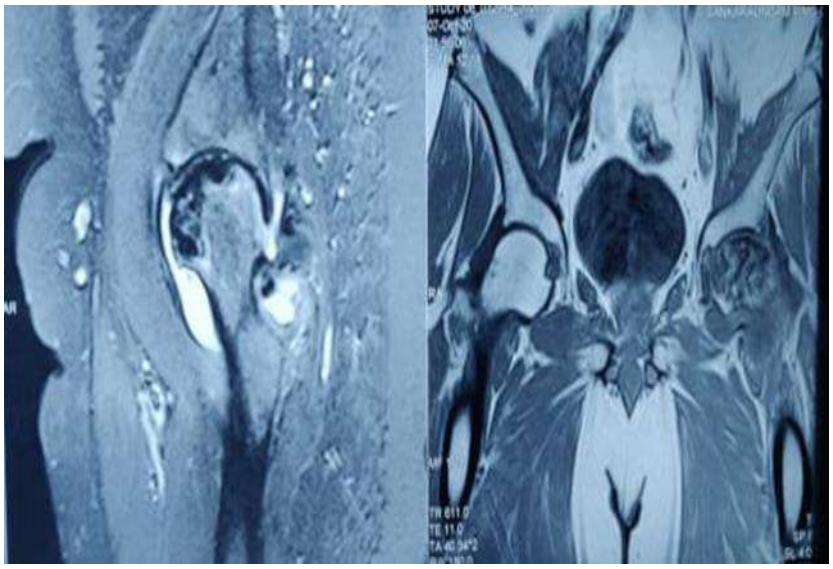

Fig 2- MRI Pelvis showing geographical lesion in the left femoral head.

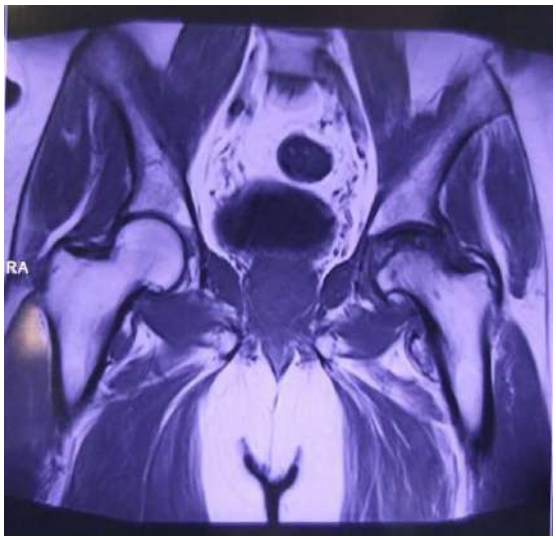

Fig 3- MRI Pelvis showing cortical collapse

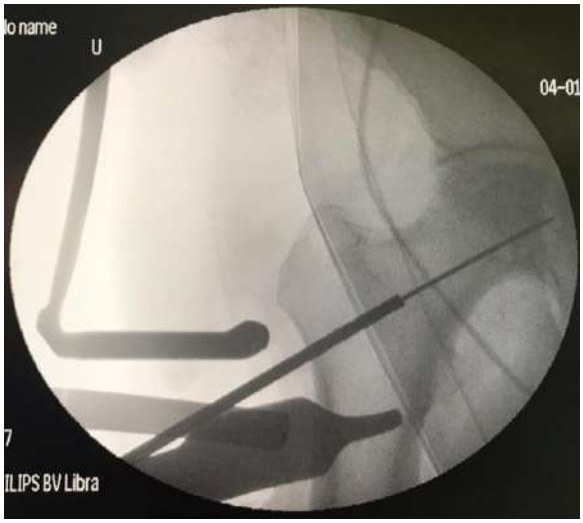

Fig 4- C arm picture showing introduction of 3.2mm guide wire. Three 3.2mm guide wires were passed; at the centre, antero-superior and posterior aspect under C arm guidance.

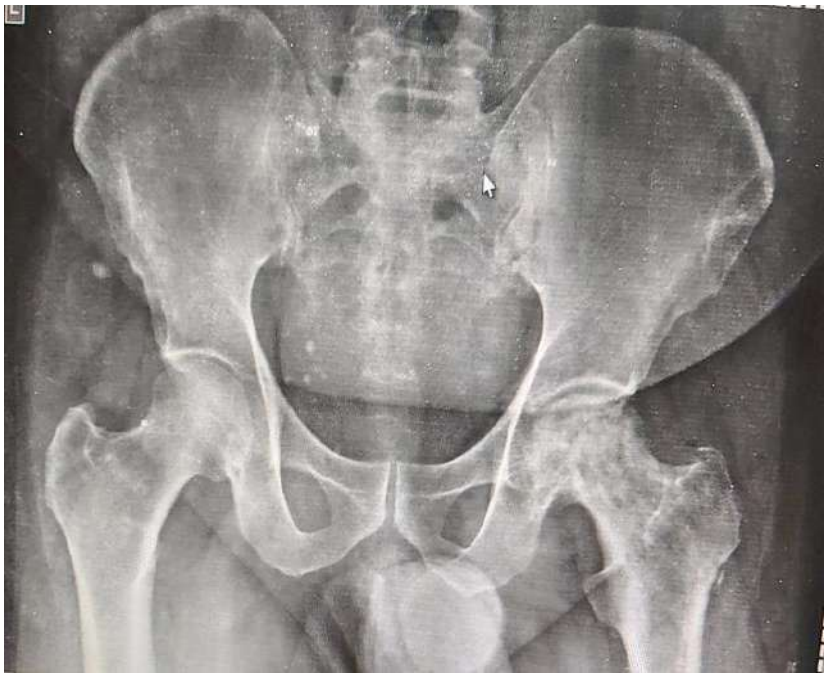

Fig 5- Follow up x ray showing mild resolution of osteonecrotic changes with improvement in sphericity of the femoral head.

DISCUSSION

Avascular necrosis of the femur has multiple aetiologies. In certain cases the exact aetiology is unknown. The patient may end up with degenerative arthritis of the affected hip and a total hip arthroplasty may be the next treatment plan due to hindrance of daily activities by pain. As the appearance of avascular necrosis is more inclined towards an elderly age group but recent figures show a rise in younger population.[1]Management of a late stage of AVN may require a total hip arthroplasty. If total hip arthroplasty is performed on a young patient, a need for revision arthroplasty arises, which is unavoidable owing to increasing life expectancy. So several treatment modalities need to be considered before managing a case of AVN in a younger individual. It projects the importance of diagnosis of the disease in its early stages. Various treatment methods have been tried to hinder the progress of the disease and prevent development of osteoarthritis of hip thereby preventing a total hip arthroplasty. Usually a core decompression is performed for an initial stage of avascular necrosis thereby reducing the intraosseous pressure in the head of femur and enables vascular invasion and revitalize the osteonecrotic tissue.[2] The usual indication for a core decompression is symptomatic presentation with AVN without femoral head collapse[9]. There are literatures that shows patients who had stage III osteonecrosis (Steinberg classification; subchondral lucency, without collapse) managed with core decompression having excellent outcomes (no requirement of surgery) for a period up to 10 years without any intervention[13]. So decompression can be considered in patients with late stage of the disease.[3] Here we have managed a case of a Stage III [Ficat and Arlet] idiopathic avascular necrosis of femur with core decompression and bone grafting [cancellous bone graft harvested from iliac crest]. On postoperative follow ups upto a period of 1 year the patient had shown satisfactory improvement both clinically and radiologically. [11,14,15]Harvesting of bone marrow mononuclear cells (autologous) appears to be an effective treatment without any risk even for a late stage of avascular necrosis of the femoral head.[4] Few literatures have reported cases were bone marrow implantation was performed which shows patients having remarkable reduction in pain and joint symptoms, as well as a decrease in the occurrence of fractural phases.[5]

CONCLUSION

A core decompression is generally considered for an early stage of AVN with no subchondral collapse. But after taking into consideration the necessity of a revision arthroplasty in future for a younger age group; a core decompression can be attempted for a even late stage of AVN in association with bone marrow implantation and bone grafting which may widen the time gap before a total hip arthroplasty or completely rules it out. Symptomatic hips with osteonecrosis but with no collapse are the best candidates for this treatment modality. [7]Successful results (no future surgery) have been obtained in certain patients with Steinberg stage III avascular necrosis (subchondral lucency, absence of collapse). As a result, even more severe condition might well be regarded for core decompression in some selected individuals. [7,15]Patients who received a greater amount of progenitor cells in the hips had greater outcomes. [8,10]Hips that had undergone vascularized fibular grafting performed better than the hips that received core decompression, as determined by enhanced vascularity and decreased osteonecrosis progression as determined by ARCO staging system. [8,12]During the entire postoperative period, the mean Harris Hip Score of the hips that are fibular grafted was better than that of the decompression treated hips, but the differences were minor at first, and the differences were unlikely to be clinically significant; by 18 months after procedure, the differences were most likely clinically significant. [8,9]The mid-term results of vascularized fibular grafts in the patients have been attributed to greater femoral head vascularity and the possibility for bone rejuvenation.

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REFERENCES

1. Agarwal T, Patel PS, Sooknundun M, Mohapatra AR, Joshi HS, Salgia A. Management of stage I and II A/B avascular necrosis of femoral head with core decompression autologous cancellous bone grafting and platelet rich plasma factors. Med J DY Patil Univ 2015;8:713-8
2. D'Ambrosi R, Biancardi E, Massari G, Ragone V, Facchini RM. Survival Analysis after Core Decompression in Association with Platelet-Rich Plasma, Mesenchymal Stem Cells, and Synthetic Bone Graft in Patients with Osteonecrosis of the Femoral Head. Joints. 2018;6(1):16-22.
3. Hernigou P, Poignard A, Zilber S, Rouard H. Cell therapy of hip osteonecrosis with autologous bone marrow grafting. Indian J Orthop. 2009 Jan;43(1):40-5.
4. Gangji V, De Maertelaer V, Hauzeur JP. Autologous bone marrow cell implantation in the treatment of non-traumatic osteonecrosis of the femoral head: Five year follow-up of a prospective controlled study. Bone. 2011 Nov;49(5):1005-9.
5. Sen RK. Management of avascular necrosis of femoral head at pre-collapse stage. Indian J Orthop. 2009 Jan;43(1):6-16.

6. Gasbarra E, Perrone FL, Baldi J, Bilotta V, Moretti A, Tarantino U. Conservative surgery for the treatment of osteonecrosis of the femoral head: current options. Clin Cases Miner Bone Metab. 2015 Jan-Apr;12(Suppl 1):43-50.
7. Hernigou P, Poignard A, Zilber S, Rouard H. Cell therapy of hip osteonecrosis with autologous bone marrow grafting. Indian J Orthop. 2009 Jan;43(1):40-5.
8. Cao L, Guo C, Chen J, Chen Z, Yan Z. Free Vascularized Fibular Grafting Improves Vascularity Compared With Core Decompression in Femoral Head Osteonecrosis: A Randomized Clinical Trial. Clin Orthop Relat Res. 2017 Sep;475(9):2230-2240.
9. Martinot P, Dartus J, Justo A, Riouach H, Cremer P, Flouzat-Lachaniette CH, Hernigou P, Kerboull L, Chiron P; French Society of Orthopaedic Surgery and Traumatology (SoFCOT). Does augmented core decompression decrease the rate of collapse and improve survival of femoral head avascular necrosis? Case-control study comparing 184 augmented core decompressions to 79 standard core decompressions with a minimum 2 years' follow-up. Orthop Traumatol Surg Res. 2020 Dec;106(8):1561-1568.
10. Lakshminarayana S, Dhammi IK, Jain AK, Bhayana H, Kumar S, Anshuman R. Outcomes of Core Decompression with or without Nonvascularized Fibular Grafting in Avascular Necrosis of Femoral Head: Short Term Followup study. Indian J Orthop. 2019 May-Jun;53(3):420-425.
11. Pawar N, Vaish A, Vaishya R. Core decompression and bone marrow aspirate concentrate injection for Avascular Necrosis (AVN) of the femoral head: A scoping review. J Clin Orthop Trauma. 2021 Nov 11;24:101691.
12. Shah SN, Kapoor CS, Jhaveri MR, Golwala PP, Patel S. Analysis of outcome of avascular necrosis of femoral head treated by core decompression and bone grafting. J Clin Orthop Trauma. 2015 Sep;6(3):160-6. doi: 10.1016/j.jcot.2015.03.008.
13. Hernandez A, Nuñez JH, Sallent A, Gargallo-Margarit A, Gallardo-Calero I, Barro V. Core Decompression Combined with Implantation of Autologous Bone Marrow Concentrate with Tricalcium Phosphate Does Not Prevent Radiographic Progression in Early Stage Osteonecrosis of the Hip. Clin Orthop Surg. 2020 Jun;12(2):151-157.
14. Jindal K, Aggarwal S, Kumar P, Rathod P. Core decompression with bone marrow aspirate concentrate in post collapse avascular necrosis of hip: A systematic review and meta-analysis. J Clin Orthop Trauma. 2021 Feb 17;17:78-87.
15. Talathi NS, Kamath AF. Autologous stem cell implantation with core decompression for avascular necrosis of the femoral head. J Clin Orthop Trauma. 2018 Oct-Dec;9(4):349-352.

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Status of Malnutrition In India: From National Nutrition Policy (1993) To National Nutrition Mission (2018) - A Review

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Abstract: Malnutrition is an age-old problem primarily seen in developing countries like India. Though lots of initiatives have been taken by the government, the country still has high rates of malnutrition. The populations predominantly affected are the pregnant women, lactating women and children under five years of age. Our Country has made a drastic progress in the field of nutrition through various innovative programmes and schemes to bring down the prevalence of malnutrition. Malnutrition is the root cause of mortality due to many diseases. Malnutrition and infections form a vicious cycle and are the leading causes of morbidity and mortality in India. Addressing Malnutrition has always been the primary goal in evolution of health care from "health for all" goal in 1970s through "Millennium Development Goals" in 2000 to "Sustainable Development Goals" in 2015. India has progressed relatively well in improving the health of the Community post-independence. Still, malnutrition remains a critical public health threat to the country. Almost 50% of the under-five children are undernourished. The prevalence of anemia among pregnant and lactating mothers is alarmingly high. This in turn leads to loss of economic productivity and thereby retards the growth of the nation. This article discusses the national programmes and policies related to the field of nutrition and the present nutritional status of population in India. The National Nutrition policy was introduced in the year 1993 and the government implemented interventions covering multiple sectors such as health, nutrition, poverty reduction and community participation. The National Nutrition Mission was launched in the year 2018 with specific emphasis on intersectoral coordination. The progress of the nation from national nutrition policy to national nutrition mission is strong but still needs more interventions to improve the health status of the population.

Key words: Undernutrition, poverty, initiatives, India.

INTRODUCTION

India has been fighting malnutrition for Centuries and the present "Sustainable Development Goals' (SDG) for the year 2030 aims for 'no poverty' and 'zero hunger' as the first two goals. India as a signatory to SDGs is working towards ending poverty in all its forms and ending hunger through food security, sustainable agriculture and better nutrition.[1] The Country has progressed a lot in bringing down the prevalence of malnutrition. Still the rates of under nutrition especially in the vulnerable groups are high and there are inter-state and intra-state variations too.[2] This article discusses the progress in the field of nutrition after the introduction of National Nutrition Policy in the year 1993.

National Nutrition Policy 1993

Nearly 45 years after Independence, India announced the first ever policy on Nutrition in the year 1993. The importance of this policy was that nutrition was recognized as a multi sectoral issue, and many direct and indirect interventions were planned. The policy highlighted the significance of Intersectoral coordination which often lacks at various levels.[3] The vision was to create "Malnutrition free India" and the primary goals of the National Nutrition Policy were

1. Reduction in the incidence of moderate and severe malnutrition and stunted growth among children.[3]
2. Reduction in the incidence of low birth weight to less than 10%. Elimination of blindness due to Vitamin A deficiency.[3]
3. Reduction in the iron deficiency anemia among pregnant women to 25%. Universal iodization of salt (USI) for reduction of iodine deficiency disorders to below the endemic level.[3]
4. Special emphasis to geriatric nutrition. Annual production of 250 million tonnes of food grain Improving household food security through poverty alleviation programsPromoting appropriate diets and healthy lifestyle.[3]

The interventions were classified broadly as direct (short term) and indirect (long term). Direct interventions were nutrition interventions for the vulnerable groups - pregnant and lactating mothers and young children, emphasis on growth monitoring, special services for adolescent girls through the Integrated Child Development Services (ICDS) scheme, fortification of foods, creating awareness on low cost nutritious foods, and control of hidden hunger, otherwise called the "Micronutrient deficiencies".[3]

Figure 1 - Four primary interventions for the vulnerable groups[2,3]

Figure 1 summarizes the four primary interventions for the betterment of the vulnerable groups. If these interventions are well implemented across the Country, we can improve the nutritional status of women and children and thereby bring down the associated morbidity and mortality. Long term interventions are the indirect policy instruments which require greater political commitment and sustained intersectoral coordination. The various indirect interventions planned as per the policy are as follows.[3] - Food security, Improve purchasing power of poor, Land reforms, Improvement in dietary pattern, Health and Family welfare, Basic health and nutrition knowledge, Prevention of food adulteration, Nutrition surveillance, Monitoring, Research, Communication through established media, Minimum wage administration, Community participation, Improving literacy and Empowerment of women.[3] After the National Nutrition policy, numerous changes have been made in the Community Nutrition programmes during the 11th and 12th five year plans.[4] Table -1 lists various community nutrition programmes implemented to improve the nutritional status of the population[5-11]. National Institution for Transforming India (NITI) has now replaced the planning commission and serves as the think-tank for newer strategies in the field of Health and Family Welfare[12]. Latest innovations in the field of nutrition are the National Nutrition strategy which was published in 2017 and National Nutrition Mission which was launched in 2018 by the Honorable Prime Minister Mr. Narendra Modi.[13,14]

Table 1- Various Nutrition Programmes implemented in India [5-11]

Community Nutrition Programmes	Other Programmes with significant nutrition component
National Vitamin A prophylaxis programme	Dr.Muthulakshmi Reddy Maternity Benefit Scheme
Nutritional anemia – Anemia Mukt Bharat	Akshaya Patra
Iodine Deficiency Disorder control programme	NikshayPoshanYojana
Integrated Child Development Services scheme	Annapurna scheme
Mid-day meal scheme Mid-day meal programme	Antyodayayojana

Table 1 highlights the nutrition programmes implemented at the community level. The programmes cover the nutritional problems of public health importance such as vitamin A deficiency, anemia and malnutrition.

National Nutrition Strategy 2017

The National Nutrition Strategy published in the year 2017 envisions creating "Malnutrition free India" (Kuposhan Mukt Bharat). Life cycle approach is focused on this strategy to ensure that every child, adolescent girl and woman attains optimal nutritional status. The prime age for prevention of under nutrition is first three years of life (first 1000 days of human life).[13]

Figure 2 - Nutrition interventions under National Nutrition Strategy 2017 [13]

Figure 2 shows a summary of interventions planned under the National Nutrition Strategy implementing the life cycle approach. The strategy covers all stages of life starting from fetal stage, infancy, adolescence, pregnancy and the reproductive period.

National Nutrition Mission 2018 (POSHAN ABHIYAAN)

The Government of India has launched the "National Nutrition Mission" or "Poshan Abhiyaan" in March 2018. The primary target of Poshan abhiyaan is to bring down under nutrition and low birth weight by 2% every year and its named as "*Mission 25*

by 2022", i.e, to bring down stunting from 38.4% (National Family Health Survey-4 data) to 25% by the year 2022. The other aims are to reduce anemia in children, adolescent girls and pregnant women by 3% every year.[14]Similar to National Nutrition policy, this Mission strives to bring a multi-ministerial convergence to attain "Malnutrition free India" by 2022 (Table 2). The districts will be covered through this mission in three phases (Table 3). This mission is launched not as a programme but as "Jan Andolan" meaning "people's movement" stressing the significance of community involvement at all stages.[14]

Table 2 - Partners in PoshanAbhiyaan and various health programmes[14]

Ministry	Health programmes
Ministry of Women and Child Development	a. PoshanAbhiyaan b. Integrated Child Development Services (ICDS) scheme c. BetiBachao, BetiPadhao (BBBP) d. Pradhan MantriMatruVandanaYojana (PMMVY)
Ministry of Rural development	a. Deendayal Antyodaya Yojana - National Rural Livelihood Mission (DAY-NRLM) b. Mahatma Gandhi National Rural Employment Guarantee Scheme (MGNREGS)
Ministry of Drinking Water and Sanitation	a. Swachh Bharat Mission (SBM) b. Safe Drinking water (SDW) c. Water, Sanitation and Hygiene (WASH)
Ministry of Human Resource development	a. Mid-day Meal scheme b. Unified District Information System for Education (UDISE)
Ministry of Health and Family Welfare	a. Mother's Absolute Affection (MAA) programme b. Rashtriya Kishor Swasthya Karyakram (RKSK) c. Anemia Mukt Bharat (AMB) d. Intensified Diarrhea Control Fortnight (IDCF) e. Home Based NewBorn Care (HBNC) f. Home Based Care For Young Child (HBYC) g. Rashtriya Bal Swasthya Karyakram (RBSK) h. National Health Mission (NHM)

Table 2 summarizes national health programmes implemented under various ministries. The synchronization of health ministry with other related sectors confirms the significance of intersectoral coordination to alleviate malnutrition.

Table 3 - Coverage of PoshanAbhiyaan in a phased manner [14]

Year	No.of.districts covered
2017-18	315
2018-19	235
2019-20	Remaining districts
Total budget	**Rs.9046.17 crores**

Table 3 shows the manner in which the districts were covered by National Nutrition Mission over a period of 3 years across the country. Districts with high malnutrition rates were given the first preference.

Present status - The Global Hunger Index for India

The Global Hunger Index (GHI) is a tool designed to measure and analyse hunger and its trend at global, regional and national levels. GHI score is calculated using four indicators – under nourishment, under five children stunting, fewer than five children wasting and under five child mortality. The score ranges from 0 to 100, where 100 is the worst stage and 0 indicates no hunger. Table 4 shows the GHI severity scale and lists out some countries for each scale.[15] In the year 2018 GHI rating, India has ranked 103rd out of total 119 countries. The score for the year 2018 is 31.1 which indicated a serious form of hunger existing in the population. The GHI score for India from 2000 to 2018 is shown in figure 3.[16]

Table 4 - Global Hunger Index severity scale [15]

GHI Score	Severity scale	Countries
Upto 9.9	Low	Argentina, Uruguay, China
10.0-19.9	Moderate	Mauritius, Thailand, Malaysia
20.0-34.9	Serious	***India***, Nepal, Phillipines
35.0-49.9	Alarming	Sierra Leone, Zambia, Madagascar
50 and above	Extremely alarming	Central African Republic

Table 4 shows the grading of severity according to global hunger index score. India falls under 'serious' severity scale.

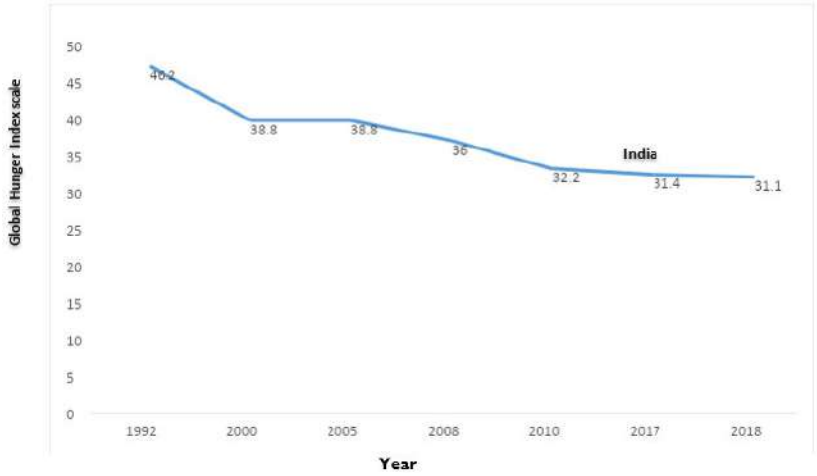

Figure 3 - Trend of GHI score for India from the year 2000 to 2018 [16]

Figure 3 shows the trend in global hunger index for India from the year 1992 to 2018. The score has shown a decline during this period which could be attributed to nutrition programmes implemented in the country. With implementation of various strategies in the field of nutrition and health, India has brought down the GHI score from 46.2 in 1992 to 31.1 in 2018. Still, the score is high and India is among the 45 Countries that are in the category of "serious hunger". [15] With the establishment of National Nutrition Mission, we can expect a decrease in the score in the coming years. Though India has progressed well in bringing down the rates of mother and child under nutrition, the improvement is not uniform. Certain states like Kerala, Tamilnadu have better health and nutrition indicators, whereas states like Uttarakhand, Uttar Pradesh, Jharkhand, Madhya Pradesh, Bihar have higher rates of child malnutrition. Madhya Pradesh (60%) has the highest number of undernourished children in the Country followed by Jharkhand (56.5%) and Bihar (55.9%). Even with the large public distribution system providing population food grains at much subsidized price and with the world's largest "Integrated Child Development Services" (ICDS) scheme, child under nutrition is still a threatening public health problem in India. This is mainly because of economic inequality, improper utilization of funds, poor implementation of the nutrition programmes, lack of intersectoral coordination and overall ignorance about the significance of nutrition, lack of awareness among the people especially in the rural areas regarding the government schemes available and non-accountability of the health workers.

CONCLUSION

With the National Nutrition Mission launched in the year 2018, India has introduced newer strategies to tackle malnutrition with community participation and intersectoral coordination. If implemented properly, these initiatives can bring down the rates of under nutrition in women and children. Global Hunger Index score need to be reduced to less than ten, and thereby this will help the country to achieve the Sustainable Development Goals (SDG) 1 and 2 - no poverty and zero hunger by 2030. With close to ten years for the SDG 2030 agenda, the government has to accelerate the implementation and monitoring of nutrition programmes especially in districts where the prevalence of under nutrition is high.

CONFLICT OF INTEREST

Conflict of interest declared none.

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REFERENCES

1. United Nations. Sustainable Development Goals. 2021 May 27.
2. Narayan J, John D, Ramadas N. Malnutrition in India: status and government initiatives. J Public Health Policy. 2019;40(1):126–41.
3. National Nutrition Policy. Government of India. Department of Women and Child Development. Ministry of Human Resource Development; 1993.
4. Planning Commission, Government of India: Five Year Plans. cited 2021 May 27.
5. Kapil U, Chaturvedi S, Nayar D. National nutrition supplementation programmes. Indian Pediatr. 1992;29(12):1601–13.
6. Akshaya Patran (TAPF): NGO in India, Feeds Mid-Day Meals to Children. 2021 Jun 27.
7. Anemia Mukt Bharat. cited 2021 Jun 27.
8. Beneficiaries Entitled for 10 Kg. Foodgrains per Month under Annapurna Scheme. 2021 Jun 2.
9. Mission Antyodaya.2021 Jun 27.

10. Nikshay Poshan Yojana: Central TB Division. 2021 Jun 27.
11. Chhabra K. Tamil Nadu Amma Maternity Nutrition Kit Scheme for Pregnant Women. [Sarkari Yojana. 2021 15. 12.
12. National Institution for Transforming India, Government of India. 2021 Jun 7.
13. Nourishing India - National Nutrition Strategy | NITI Aayog, (National Institution for Transforming India), Government of India. 2021 Jun 27.
14. Poshan Abhiyaan - Ministry of Women and Child Development, Government of India. [2021 Jun 27.
15. Global Hunger Index Results - Global, Regional, and National Trends [Internet]. Global Hunger Index - A Peer-Reviewed Publication. 2021 Jun 27.
16. National Family Health Survey (NFHS-4), 2015-16: India. Mumbai: IIPS. 2021 Jun 27.

SP-49

A Case Report of Thyroid Dyshormonogenesis

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Abstract: Primary congenital hypothyroidism is one of the most common neonatal endocrine disorders. The most common causes are thyroid dysgenesis, which defines a spectrum of developmental abnormalities of the thyroid gland. Thyroid dyshormonogenesis, defined as a defective molecular pathway that results in the failure of the hormone production by a thyroid gland that is structurally intact. A delay in the treatment of neonatal hypothyroidism will result in a profound neuro developmental delay. Hence, simple screening of thyroid profile after birth should be done in all the babies. We hereby, report a case of a 15-day old neonate who is incidentally diagnosed with congenital hypothyroidism due to thyroid dyshormonogenesis based on elevated TSH level in the newborn screening test. Thyroid dyshormonogenesis has a good prognosis if treated early and appropriately.

Keywords: congenital hypothyroidism, thyroid dyshormonogenesis

INTRODUCTION

Congenital hypothyroidism can be divided into primary and central. Primary congenital hypothyroidism is due to a defect that is affecting the thyroid gland itself while central congenital hypothyroidism is due to impaired thyroid-stimulating hormone (TSH). Central congenital hypothyroidism causes pituitary or hypothalamic pathology. Primary CH is the most common neonatal endocrine disorder. The most common cause is thyroid dysgenesis (TD).[1] It is a spectrum of thyroid developmental abnormalities. The second important cause is thyroid dyshormonogenesis, which is a failure in the production of hormone by a structurally normal gland. Central CH is usually rare which usually occurs in isolation, or in association with other pituitary hormone deficits. In central CH there is failure of TSH production whereby subnormal thyroid hormone levels. In thyroid screening we should measure both Free T4 and TSH to avoid missing central CH. A delay in the treatment of neonatal hypothyroidism can result in profound neurodevelopmental delay which can be averted by prompt diagnosis and simple eltroxin therapy.[2]

CASE REPORT

Presenting complaint

10 months old boy diagnosed at birth with congenital hypothyroidiam came for regular follow up.

Medical history

This boy baby is first born to a non-consanguineous marriage. The baby was delivered at term via vaginal delivery with birth weight of 2.8 Kg .Baby cried immediately after birth. Antenatal History was insignificant. Postnatally baby was feeding well, stools changed to yellow on day 4 of life and there was no persistent jaundice.

Previous History – not suitable for the case

Family history

There was no family history of thyroid disorders.

Observation

At 15 days of life, baby had adequate weight gain. A small umbilical hernia was noted. At present, child is 12 months old. He is developmentally normal. There is no umbilical hernia (Figure 1).

Figure 1 – This picture depicts there is no umbilical hernia

Investigations

Routine thyroid screening after 72 hours of life showed borderline TSH -9.26 µIU/ml. At day 15 of life repeat TSH value was very high - 99.60 µIU/ml and 127.37 µIU/ml done in two different occasions. In view of persistent high TSH further workup was done. Ultrasonography of neck showed mild thyromegaly with both lobes and isthmus slightly enlarged.

Special investigations

Tc-99m pertechnetate scan showed a diffusely enlarged thyroid gland (figure 2)

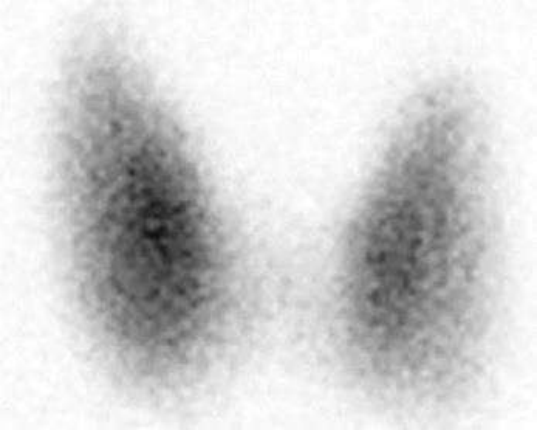

Figure 2 *technetium* pertechnetate scan shows diffuse enlargement of thyroid gland

Diagnosis

The above findings are consistent with primary hypothyroidism due to dyshormonogenesis.

Prognosis

Congenital hypothyroidism due to thyroid dyshormonogenesis has good prognosis. Eltroxin therapy can be stopped at 3 years of age and child should be reevaluated.

Treatment

The baby was started on eltroxin therapy initially at the dose of15mcg/kg/day. Subsequently TSH levels normalised. At present, child is 12 months old and on eltroxin at the dose of 10 mcg/kg/day.

DISCUSSION

Hypothyroidism is one of the most common endocrine disorder with a variable clinical presentation. In neonate it manifests as persistent jaundice, constipation with coarse facies. There may be small umbilical hernia like in our case. Screening programs for early recognition and diagnosis of congenital hypothyroidism have virtually eradicated the incidence of intellectual disability and impaired somatic growth that is caused by deficiency of thyroid hormone. This was possible by the early diagnosis and management. CH is one of the most common preventable causes of intellectual disability[3]. A failure in early diagnosis and treatment can lead to severe intellectual disability and physical morbidities. The consequences of CH occur because the presentation and the clinical features are pretty subtle and can be easily missed or might not be evident in the early stages of life. And infants with CH usually appear normal at the time of birth in-spite of deficiency of thyroid hormones.[4,5,6,7] Thyroid dyshormonogenesis is a rare condition. It accounts for 15% Of CH. Genetic testing has been playing an expanding role in the diagnosis of thyroid disorders. These studies have helped in identifying several candidate genes responsible for these etiologies of CH[8]. Due to financial constraints genetic testing was not done in our case. A serum level of TSH >10mIU/L after 72 hours of life in term neonate or Cord blood TSH level of >20 mIU/L is considered a positive screening test[9,10]. All neonates should undergo mandatory thyroid screening to rule out CH irrespective of etiology and risk factors. Similar study had been conducted at Chandigarh, India where significance of neonatal thyroid screening has been emphasised.[11] If thyroid screening is positive it should be evaluated immediately with confirmatory testing and examination.[8,12] The tests include total thyroid with USG neck and technetium scan to identify the etiology. In our case there was no significant family history or any clinical clues. Routine thyroid screening test helped to diagnose the child with CH[13,14]. Thyroxine therapy is very safe though there had been very few case reports of late-onset circulatory dysfunction in extremely premature infants.[15]

CONCLUSION

Congenital hypothyroidism is usually not diagnosed early due to subtle clinical presentation. A regular neonatal thyroid screening irrespective of maternal history can help to diagnose such cases and prompt treatment can be started at an early stage. It is a preventable cause of intellectual disability. Parental counselling regarding the prognosis of the disease and need for regular follow up should be insisted. If diagnosed early and treated thyroid dyshormonogenesis has a good prognosis.

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CONFLICT OF INTEREST

Conflict of interest declared none.

Patient consent: obtained

REFERENCES

1. Chai J, Yang XL, Guo MZ, Liu L, Liu SG, Yan SL, Ge YL. DUOX2 mutations in children with congenital hypothyroidism. Zhongguo Dang dai er ke za zhi= Chinese Journal of Contemporary Pediatrics. 2015 Jan 1;17(1):40-4.
2. Khurram IM, Choudhry KS, Muhammad K, Islam N. Clinical presentation of hypothyroidism: a case control analysis. J Ayub Med Coll Abbottabad. 2003;15(1):45-9.
3. Rama Devi AR. Newborn screening in India, experience from pilot initiative (ICMR Multicenter Project). Abstract presented at 8th Asia Pacific Regional Meeting of the International Society for Neonatal Screening. New Delhi, Sep
4. Raza H, Riaz S, Jamal M, Shirazi H, Gul S. Congenital Hypothyroidism Newborn Screening-The PIMS Experience. Ann Pak Inst Med Sci. 2013;9(3):198-200..
5. Harris KB, Pass KA. Increase in congenital hypothyroidism in New York State and in the United States. Molecular genetics and metabolism. 2007 Jul 1;91(3):268-77.
6. van Trotsenburg, P.; Stoupa, A.; Léger, J.; Rohrer, T.; Peters, C.; Fugazzola, L.; Cassio, A.; Heinrichs, C.; Beauloye, V.; Pohlenz, J.; et al. Congenital hypothyroidism: A 2020–2021 consensus guidelines update—An ENDO-European Reference Network Initiative endorsed by the European Society for Pediatric Endocrinology and the European Society for Endocrinology. *Thyroid* 2021, *31*, 387–419.
7. Itonaga, T.; Higuchi, S.; Shimura, K.; Nagasaki, K.; Satoh, M.; Takubo, N.; Takahashi, I.; Sawada, H.; Hasegawa, Y. Levothyroxine dosage as predictor of permanent and transient congenital hypothyroidism: A multicenter retrospective study in Japan. *Horm. Res. Paediatr.* 2019, *92*, 45–51
8. Sparling DP, Fabian K, Harik L, Jobanputra V, Anyane-Yeboa K, Oberfield SE, Fennoy I. Congenital hypothyroidism and thyroid dyshormonogenesis: a case report of siblings with a newly identified mutation in thyroperoxidase. Journal of Pediatric Endocrinology and Metabolism. 2016 May 1;29(5):627-31.
9. Higuchi, S.; Hasegawa, Y. Levothyroxine dosages less than 2.4 μg/kg/day at 1 year and 1.3 μg/kg/day at 3 years of age may predict transient congenital hypothyroidism. *Clin. Pediatr. Endocrinol.* 2019, *28*, 127–133
10. Yamamura, H.; Kokumai, T.; Furuya, A.; Suzuki, S.; Tanahashi, Y.; Azuma, H. Increase in doses of levothyroxine at the age of 3 years and above is useful for distinguishing transient and permanent congenital hypothyroidism. *Clin. Pediatr. Endocrinol.* 2020, *29*, 143–149

11. Kaur G, Srivastav J, Jain S, et al. Preliminary report on neonatal screening for congenital hypothyroidism, congenital adrenal hyperplasia and glucose-6-phosphate dehydrogenase deficiency: a Chandigarh experience. Indian J Pediatr. 2010;77:969–73.
12. Kimura S, Kotani T, McBride OW, Umeki K, Hirai K, Nakayama T, Ohtaki S. Human thyroid peroxidase: complete cDNA and protein sequence, chromosome mapping, and identification of two alternately spliced mRNAs. Proceedings of the National Academy of Sciences. 1987 Aug 1;84(16):5555-9.
13. Kishore KR, Ranieri E, Fletcher J. Newborn screening for congenital hypothyroidism in India - is overdue. J Neonatal Biol. 2014;3:129.
14. Corbetta C, Weber G, Cortinovis F, et al. A 7-year experience with low blood TSH cutoff levels for neonatal screening reveals an unsuspected frequency of congenital hypothyroidism (CH). Clin Endocrinol. 2009;71:739–45.
15. Yagasaki, H.; Kobayashi, K.; Nemoto, A.; Naito, A.; Sugita, K.; Ohyama, K. Late-onset circulatory dysfunction after thyroid hormone treatment in an extremely low birth weight infant. *J. Pediatr. Endocrinol. Metab.* 2010, *23*, 153–158.

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SP-50

Tricompartmental Osteoarthritis Treated Surgically with Total Knee Arthroplasty - A Case Report.

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Abstract: The most effective treatment for end stage osteoarthritis knee is total knee replacement.In developing countries like India, that too without much instrumentation and technologies like lift and elevator results in development of osteoarthritis in many people.In this case 65-year-old female came with complaint of pain in bilateral knee (left knee pain more than right knee). Pain present for the past five years but aggravated for the past three months. Examination revealed varus deformity of both knees (left more than right) with bilateral medial and lateral joint line tenderness. Movement of bilateral knees is painful and restricted. Plain radiograph of bilateral knee standing showed absolute reduction in joint space (left more than right). Patient was operated with left sided total knee replacement. Right sided total knee replacement is done after 6 months. Postoperative period uneventful without any complications. Patient is able to walk without any pain.

Keywords: Total Knee arthroplasty, Knee replacement, Osteoarthritis, Tricompartmental.

INTRODUCTION

In patients with degenerative arthritis and rheumatoid arthritis the effective surgical intervention to relieve pain and to get functional recovery is by total knee replacement.Increase in old age population in society lead to increase in prevalence of arthritis. It is chronic degenerative disorder due to multiple cause characterized by morphological and biochemical change in synovial membrane and capsule, sclerosis of subchondral region, bone margin hypertrophy and decreased articular cartilage.[1,2]Pathological feature of late OA knee progress from softening to ulcer formation to patchy disintegration of cartilage along with synovial inflammation. Symptoms mainly include pain after prolonged standing and stiffness following inactivity. Osteoarthritis can be primary or secondary. In primary OA knee etiology is unknown mostly related to aging.Secondary OA is due to another disease.[3,4]Osteoarthritis is the second most rheumatological problem with prevalence of twenty two percent to thirty nine percent in India.[5] Women are more frequently affected than male and the number of cases increase with aging of patient.Among women above the age of 65 years, forty five percent have symptoms whereas seventy percent patient have radiological evidence.[6]It is the tenth cause for nonfatal burden.Hence complete relief of pain and back to previous condition is required for most of the patients with OA knee. In this case we have done TKR for patients with tricompartmental osteoarthritis.Postoperative period uneventful. Patient relieved of pain following surgery.[7]

METHODS

A sixty-four-year-old female came with her son to Sree Balaji Medical College and Hospital Orthopaedics OPD with chief complaints of pain in bilateral knee (left knee more than right knee). Pain present for the past five but aggravated for the past three months. Pain aggravated on prolonged standing and walking and relieved on taking analgesics. Patient has difficulty in climbing up and down stairs but she is able to walk with walker support. She is not able to sit with crossed leg and squatting. Patient had a history of slip and fall five years back and sustained both bone fractures to the right leg for which she went for native treatment, splinting was done 7 times over a period of twelve month. Examination revealed varus deformity in the left knee with bilateral knee medial and lateral joint line tenderness. Crepitus present in both knees, patellar tap is negative. ROM is painful and restricted (left more than right). All special tests (for ligament and meniscus) are found to be normal. Patient was scheduled for elective TKR for the left knee. Elective TKR for right knee was done 6 months after.

RESULT

Bilateral knee has been operated with the same procedure at interval of six months.All required preoperative investigations are done and anaesthetic fitness obtained elective TKR under spinal anaesthesia (Figure 1). Under aseptic precaution, under epidural and spinal anaesthesia, patient in supine position, parts painted and draped with betadine. Knee joint is flexed to ninety degree, a ten centimetre midline skin incision made extending from two finger space above the superior pole of patella to tibial tuberosity. Incision is deepened to subcutaneous tissue. Retinaculum is incised with standard parapatellar retinacular approach, extending proximally to length of quadriceps tendon. Incision was continued along the medial side of patella up to three to four centimetre onto the anteromedial surface of tibia. Medial side of the knee joint is exposed by subperiosteally elevating anteromedial capsule and deep medial collateral ligament off the tibia to the posteromedial corner of the knee. Lateral side of synovium found to be hypertrophied, biopsy taken and sent for histopathological examination. Patella is everted after extending the knee joint. Knee is flexed again to remove ACL and anterior horns of medial and lateral menisci.Distal femoral cut done in five-degree valgus, eleven-millimetre cut was done. Box cut was made.Tibial preparation was made with a cut of eight-millimetre and three-degree slope. Implant with femoral component of size sixty millimetre and Tibial component of seventy-one-centimeter size inserted with cement. Polyethylene of 12mm size is inserted between the tibial and femoral components. Patella resurfacing was done and retained to it's normal position. Movements checked by flexion and extension.

Valgus and Varus test performed and found to be normal. Fourteen size drain kept and wound closed in layers. Sterile dressing done.Postoperative period uneventful without any complications.Drain removed after forty eight hours following surgery. Intravenous antibiotic given for two days followed by oral antibiotics given till suture removal. Walker mobilization was done from second postop day (Figure 2).

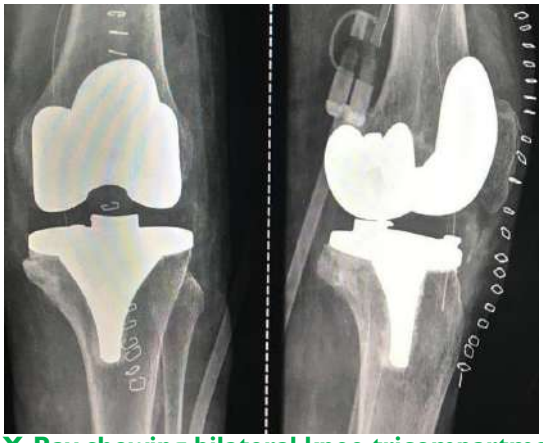

Figure 1: Pre-operative X-Ray showing bilateral knee tricompartmental osteoarthritis with right leg malunited proximal both bone fracture.

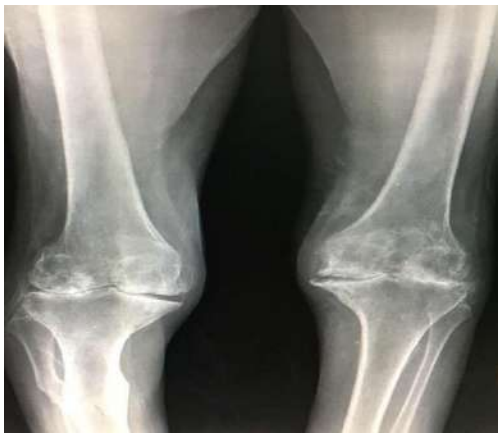

Figure 2:Postoperative X-Ray of TKR.

DISCUSSION

Total knee arthroplasty is more effective than providing continuous non-surgical treatment of end stage osteoarthritis with analgesics and anti-inflammatory drugs. Osteoarthritis develops in the old age group. The most important factors that hip replacement must address are pain relief, the ability to return to work, and an increase in the patient's level of activity with particularly emphasis on walking capacity.[8]At this age people have associated comorbid conditions like diabetes and hypertension.[9] Treating these patients with continuous analgesics and anti-inflammatory drugs lead to development of complications like organ failure. In developing countries like India, without much sources and technology, most patients seeking government hospitals are treated conservatively with analgesics along with wax baths to relieve pain. The most common sites for the pain are the groin and anterior and lateral thigh, the buttock, and the knee.[10] The examination of the arthritic hip may show an antalgic or trendelenburg gait, although this sign is unreliable.[11]Development of osteoarthritis can be prevented by early diagnosis and starting on quadriceps strengthening exercise and avoiding stress factors. But the only available modality for end stage osteoarthritis is total knee replacement. In our case, the age of the patient is sixty-four years which is the most common age for development of osteoarthritis.It mostly affects females which is in line with our case. She came with end stage tricompartmental osteoarthritis, hence the only option available is knee arthroplasty.[12] Total number of knee arthroplasty done in developed country is more compared to developing countries like India due to lack of availability of sources. After bilateral total knee replacement, she was completely relieved of pain and able to bear weight and walk.[13]Hence from this case we conclude that total knee arthroplasty is an effective treatment modality for end stage osteoarthritis.

CONCLUSION

TKA has been recognized as a successful treatment for knee arthritis that delivers relatively high satisfaction compared to the other non-surgical treatments for OA knee.We have successfully treated tricompartmental osteoarthritis bilateral knee joint with total knee arthroplasty of both knee in a sixty-five-year-old female. She was completely relieved of pain and able to bear weight and walk after surgery. Hence, we conclude that total knee arthroplasty is the best procedure to get relieved of pain rather than going with conservative treatment in end stage osteoarthritis.

CONFLICT OF INTEREST

Conflict of interest declared none.

ACKNOWLEDGEMENT: Author thank the management of Sree Balaji Medical College and Hospital for their support

FUNDING SOURCE: None

REFERENCES

1. Carr AJ, Robertsson O, Graves S, Price AJ, Arden NK, Judge A, Beard DJ. Knee replacement//lancet. 2012;379(9823):1331-40.
2. Vogt JC, Saarbach C. LCS mobile-bearing total knee replacement. A 10-year's follow-up study. Orthopaedics & Traumatology: Surgery & Research. 2009 May 1;95(3):177-82.
3. Juni P, Reichenbach S, Dieppe P. Osteoarthritis: rational approach to treating the individual. Best practice & research Clinical rheumatology. 2006 Aug 1;20(4):721-40.
4. Hamel MB, Toth M, Legedza A, Rosen MP. Joint replacement surgery in elderly patients with severe osteoarthritis of the hip or knee: decision making, postoperative recovery, and clinical outcomes. Archives of internal medicine. 2008 Jul 14;168(13):1430-40.
5. Mikkelsen W. M., Dodge H. J., Duff I. F., Kato H. Estimates of the prevalence of rheumatic diseases in the population of Tecumseh, Michigan, 1959-60. Journal of Chronic Diseases. 1967;20(6):351–369.
6. Silman AJ, Hochberg MC. Epidemiology of the rheumatic diseases. Oxford University Press; 2001.
7. Akinpelu AO, Alonge TO, Adekanla BA, Odole AC. Prevalence and pattern of symptomatic knee osteoarthritis in Nigeria: A community-based study. Internet Journal of Allied Health Sciences and Practice. 2009;7(3):10.
8. Johnston R. C., Fitzgerald R. H., Jr., Harris W. H., Poss R., Muller M. E., Sledge C. B. Clinical and radiographic evaluation of total hip replacement. A standard system of terminology for reporting results. The Journal of Bone and Joint Surgery. American. 1990;72(2):161–168.
9. Mathers CD, Stein C, Ma Fat D, Rao C, Inoue M, Tomijima N, Bernard C, Lopez AD, Murray CJ. Global Burden of Disease 2000: Version 2 methods and results. Geneva: World Health Organization. 2002 Oct.
10. Bulstrode C. Oxford Textbook of Trauma and Orthopaedics. Oxford, UK: Oxford University Press; 2002.
11. Cibere J., Thorne A., Bellamy N., et al. Reliability of the hip examination in osteoarthritis: effect of standardization. Arthritis Care and Research. 2008;59(3):373–381.
12. Solomon, L., Beighton, P. & Lawrence JS. Rheumatic disorders in the South African Negro-Part II. Osteo-Arthrosis. South African Medical Journal. 1975 Oct 1;49(42):1737-40.
13. Davis MA, Ettinger WH, Neuhaus JM, Hauck WW. Sex differences in osteoarthritis of the knee: the role of obesity. American journal of epidemiology. 1988 May 1;127(5):1019-30.

SP-51

Twin Pregnancy in Bicornuate Uterus- A Dilemma in Management?

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Abstract: The uterine malformation is found to be around 3-5% in the general population. Most of them go unnoticed. Out of which 15% - 25% are associated with higher risk of complications like preterm delivery, miscarriages, foetal malpresentations, premature rupture of membranes and fertility problems. A 25 year old lady G2P2L1, known to have a bicornuate uterus, came with complaints of bleeding per vaginum for 1 day preceded by 7 weeks of amenorrhea. It was associated with severe lower abdominal pain and increasing in intensity and frequency. Examination revealed active bleeding through os, uterus bulky and internal os closed. Ultrasonogram of the pelvis showed two different gestational sacs in each horn of the bicornuate uterus. A gestational sac was visualised in the right horn but the foetal pole and yolk sac could not be visualised. It showed signs of sac separation and the sac in the left horn showed a live foetus of 6 weeks gestation. Blighted ovum was diagnosed one week later in the right horn. The patient opted to continue her pregnancy. The patient had spontaneous onset of labour pain with good uterine contractions and foetal monitoring was done. Later, she was taken up for an emergency caesarean section in view of non-progress of labour. Uterine abnormalities are usually associated with complications such as foetal malpresentations, preterm labour and sometimes perinatal mortality. Most of these anomalies may not be suspected until the complications happen like abortion. Even though successful pregnancy can occur, they are still at further risk of certain complications.

Keywords: Bicornuate uterus, Ultrasonogram, Vaginum

INTRODUCTION

Congenital abnormalities of the female genital tract are due to abnormal formation and fusion of the paramesonephric and Mullerian ducts during organogenesis.[1] The uterine malformation is found to be around 3-5% in the general population.[2] Most of them go unnoticed. Out of which 15% - 25% are associated with higher risk of complications like preterm delivery, miscarriages, foetal malpresentations, premature rupture of membranes and fertility problems.[3] The fallopian tube, uterus, cervix and upper two-thirds of vagina comprise the Mullerian duct; failure of Mullerian duct fusion can result in didelphys or bicornuate uterus.[4] Most of these congenital anomalies remain asymptomatic; often recognised during pregnancy, recurrent miscarriage, fertility or incidental findings. Early diagnosis and close follow-up can improve reproductive outcomes.[5]Adams et al., in a more recent case report published in 2019, advocated for the continued publication of case reports involving uterine malformations and twin pregnancy in order to provide a larger body of data for what is a rare presentation.[6]

CASE REPORT

Presenting complaints

A 25year old lady G2P2L1, known as a bicornuate uterus, came with complaints of bleeding per vaginum for 1 day preceded by 7 weeks of amenorrhea. It was associated with severe lower abdominal pain and increasing in intensity and frequency.

Medical history

She had previously normal vaginal term delivery; growth restricted baby weighing 2kg.

Previous and current family history

She had no history of similar complaints in the immediate family. In the current pregnancy she came with bleeding per vaginum for 1 day, per speculum examination revealed active bleeding through os, uterus bulky and internal os closed.

Observation : Not relevant

Diagnosis

Ultrasonogram of the pelvis showed two different gestational sacs in each horn of the bicornuate uterus. A gestational sac was visualised in the right horn but the foetal pole and yolk sac could not be visualised. It showed signs of sac separation and the sac in the left horn showed a live foetus of 6 weeks gestation. Dilatation and evacuation was deferred to protect the healthy foetus. The abortion resolved spontaneously. One week later, the ultrasound was repeated again. It showed live fetus of 6 weeks gestation in the left horn of uterus and right horn showed irregular gestational sac, with no evidence of fetal pole and yolk sac following which blighted ovum was diagnosed.

Special tests and investigation

Routine Blood investigations were done; bleeding time, clotting time and serum fibrinogen done and found to be normal. She was counselled about the risks and consequences regarding the pregnancy outcomes.

Treatment

The patient opted to continue her pregnancy and was on regular follow-up as advised. Meanwhile she was on regular progesterone 200mg per oral for occasional abdominal pain. In the third trimester interval growth scan was done, it showed live foetuses of 36 weeks gestation in breech presentation. The patient had spontaneous onset of labour pain with good uterine contractions and foetal monitoring was done. Later, she was taken up for emergency caesarean section in view of non-progress of labour. Intraoperative findings were bicornuate uterus and fetal head in the left horn and breech in the lower segment. Right horn was found to be empty. The patient had delivered live male baby of 2400 grams and Apgar score 9 and 10 for one min and five minute respectively.

Prognosis

More than half of women with uterine malformations will remain asymptomatic.15% - 25% are associated with higher risk of complications like preterm delivery, miscarriages, foetal malpresentations, premature rupture of membranes and fertility problems[3]

Figures

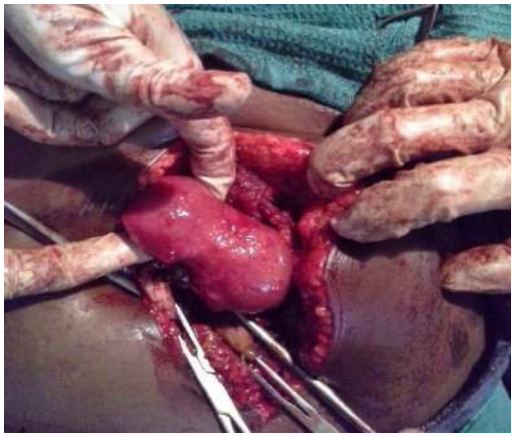

Figure 1: bicornuate uterus anterior view showing the right horn empty.

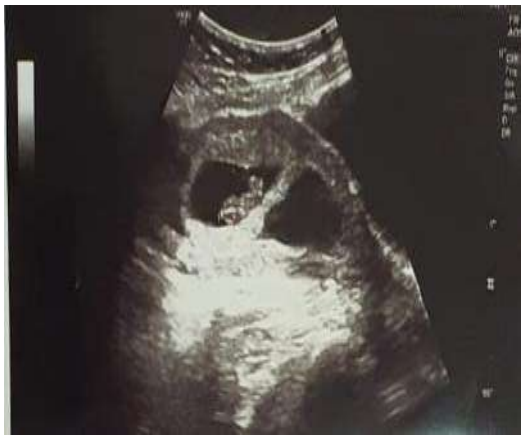

Figure 2: bicornuate uterus with live foetus in one sac and blighted ovum in the other sac at 6 weeks of gestation.

DISCUSSION

Complete bicornuate uteri have two uterine cavities without any connection. These malformations can be associated with increased risks of preterm delivery, malpresentation, spontaneous abortion, placental abruption, intrauterine growth restriction, operative delivery and preterm delivery. The reason behind this complication can be due to reduced muscle mass, cervical insufficiency and abnormal blood flow.[7] In this case Dilation and evacuation was not done at 6 weeks in view of live foetus in the left horn. There are specific guidelines on management of delivery in a malformed uterus or follow-up because the incidence is very low. Abdominal or vaginal route was selected for delivery though one has to be cautious of vaginal delivery. Risks associated with vaginal delivery include uterine rupture, malpresentation and shoulder dystocia. The earliest article published by Duraisamy et al and Bhagwat SA et al, since then the incidence of congenital malformations has been

increasing in India and still requires awareness to determine the ture prevalence.[8,9] Pregnancies with Mullerian anomalies can be asymptomatic or present with various obstetric complications. Sometimes the occurrence of these obstetric complications gives us diagnostic clue.[10] In our case blighted ovum and malpresentation of the fetus could be attributed to the malformed uterus. Ultrasound plays an important role in diagnosing anomalies of the reproductive system. It has sensitivity of 23% in diagnosing anomalies.[11] In our case the diagnosis of bicornuate uterus was not made before the first pregnancy, probably due to small size of horns or difficulty in imaging though it was correctly diagnosed in the second pregnancy.[12] Even though other effective method of evaluation is hysterosalpingography, the non-invasive imaging (transvaginal 3D ultrasound) is the preferred choice (ultrasound). MRI is also helpful in evaluating the uterine contour in reproductive women.[13]The study by Fox et al. on the use of cervical cerclage in twin pregnancies with abnormal uterus found no benefit. Depending on the circumstances, the risk-benefit ratio of progesterone and cerclage can be considered.[14]Prenatal diagnosis of uterine anomalies should be diagnosed to avoid obstetric complications and the management needs to individualise because of the possible risk and its rarity.There are no specific guidelines for the mode of delivery in a bicornuate uterus. However, because of the associated contractility abnormalities in labour and cervical dystocia, the incidence of caesarean section is significantly higher in cases of uterine malformations. There has also been a rare report of uterine rupture during induction of labour in women with uterine malformations.[15]

CONCLUSION

Uterine abnormalities are usually associated with complications such as foetal malpresentations, preterm labour and sometimes perinatal mortality. Most of these anomalies may not be suspected until the complications happen like abortion. Even though successful pregnancy can occur, they are still at further risk of certain complications. It is always necessary to raise awareness among patients regarding the possible outcomes.The case also emphasises the risk of missing a diagnosis of bicornuate uteri prenatally in settings where access to high-quality obstetric ultrasound and other diagnostic modalities is not always guaranteed. To avoid missing this rare occurrence, ultra-sonographers working in rural areas should receive additional training in order to recognise it.Guidelines to help healthcare workers detect these congenital malformations, so that favourable outcomes for mother and fetus needs to be established.

CONFLICT OF INTEREST

Conflict of interest declared none.

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FUNDING SOURCE: None

REFERENCES

1. Elias HE, Amisi JA. Twin pregnancy in a bicornuate uterus in rural Kenya: A case report for accidental discovery and successful delivery. African Journal of Primary Health Care and Family Medicine. 2020 Jan 1;12(1):1-4.
2. Society TA. The American Fertility Society classifications of adnexal adhesions, distal tubal occlusion, tubal occlusion secondary to tubal ligation, tubal pregnancies, Müllerian anomalies and intrauterine adhesions. Fertility and sterility. 1988 Jun 1;49(6):944-55.
3. Cruceyra M, Iglesias C, De La Calle M, Sancha M, Magallón SL, González A. Successful delivery of a twin pregnancy in a bicornuate uterus (uterus bicornisunicollis) by bilateral Caesarean section. Journal of Obstetrics and Gynaecology Canada. 2011 Feb 1;33(2):142-4.
4. Raga F, Bauset C, Remohi J, Bonilla-Musoles F, Simón C, Pellicer A. Reproductive impact of congenital Müllerian anomalies. Human Reproduction (Oxford, England). 1997 Oct 1;12(10):2277-81.
5. Acien P. Incidence of Müllerian defects in fertile and infertile women. Human reproduction (Oxford, England). 1997 Jul 1;12(7):1372-6.
6. Adams K, Minuzzo L. Successful delivery of spontaneously conceived twins in a single horn of a bicornuate uterus: A case report. Case Rep Womens Health. 2019 Feb 27;21:e00103. doi: 10.1016/j.crwh.2019.e00103. PMID: 30886828; PMCID: PMC6401982.
7. Airoldi J, Berghella V, Sehdev H, Ludmir J. Transvaginal ultrasonography of the cervix to predict preterm birth in women with uterine anomalies. Obstetrics &Gynecology. 2005 Sep 1;106(3):553-6.
8. Duraisamy AE. Twin Pregnancy in a Bicornuate Uterus. *Ind Med Gaz*. 1926;61(10):497-498.
9. BHAGWAT SA. Twin pregnancy in bicornuate uterus. J Indian Med Assoc. 1953 May;22(8):330-1. PMID: 13069735.
10. Rock JA, Breech LL. Surgery for anomalies of mullerian ducts. In: Rock JA, Jones HW III, editors. Telinde's Operative Gynaecology. 10th ed. Philadelphia: Lippincott Williams and Wilkins; 2011.
11. Golan A, Langer R, Wexler S, Segev E, Niv D, David MP. Cervical cerclage--its role in the pregnant anomalous uterus. International journal of fertility. 1990 May 1;35(3):164-70.
12. Ravasia DJ, Brain PH, Pollard JK. Incidence of uterine rupture among women with mu ̈llerian duct anomalies who attempt vaginal birth after cesareandelivery. American journal of obstetrics and gynecology. 1999 Oct 1;181(4):877-81.
13. Petrozza JC, Gray MR, Davis AJ, Reindollar RH. Congenital absence of the uterus and vagina is not commonly transmitted as a dominant genetic trait: outcomes of surrogate pregnancies. Fertility and sterility. 1997 Feb 1;67(2):387-9.

14. Fox NS, Roman AS, Saltzman DH, Klauser CK, Rebarber A. Twin pregnancy in patients with a uterine anomaly. J MaternFetal Neonatal Med. 2014 Mar;27(4):360-4. doi: 10.3109/14767058.2013.819331. Epub 2013 Jul 26. PMID: 23805982.
15. Mazhari F, De A, Tripathi R. Successful vaginal delivery of spontaneously conceived twins in each horn of a bicornuate uterus with previous caesarean: a case report. International Journal of Reproduction, Contraception, Obstetrics and Gynecology. 2019 Nov 1;8(11):4581-5.

SP-52

Vaginal Varicose Veins: A Case Report

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Abstract: Varicose veins are common in pregnancy and usually affect the lower extremities. The reasons are increased venous pressure, obstruction to venous drainage of lower extremities and hormonal factors. Vaginal or Valvular varicosities may occur in pregnancy and may rupture leading to risk of hemorrhage during childbirth. A patient, female, 34 years old G2P1L1 with gestational age 23 Weeks and 5 days was sent to OBG OPD for evaluation of large swelling in the vagina. She came with complaints of having discomfort while walking for two weeks. She had similar complaints in her previous pregnancy at 27 weeks and 2 days and was diagnosed to be a case of vaginal varicosities. She was delivered by cesarean section and the varicosities disappeared soon after delivery. There was no history of cancer, varicose veins, or liver disease in the family.If a woman comes with a complaint of large vaginal swelling during pregnancy, the differential diagnosis would beutero vaginal prolapse, infective conditions like Bartholin's cyst, congenital causes like Gartner vaginal wallcyst, vaginal varicosities and cancerous growth. The mass was diagnosed to be a large vaginal varicosity. In view of high risk of hemorrhage during the delivery, a repeat cesarean section was recommended. Close observation during the postpartum period was done. Spontaneous resolution is a common result which was seen in our patient.

Keywords: Vaginal varicose, Gestational, Patient

INTRODUCTION

Varicose veins are unusually swollen veins. Pregnancy is associated with dilated veins due to hormonal influences and increased blood volume. This is further exaggerated by obstruction to venous drainage by the enlarging uterus.[1] Varicosities can occur anywhere in the pelvic and genital region. Vaginal varicosities are less common than vulvar varicosities.[2] Genital varicosities usually develop in the second trimester and resolve spontaneously after delivery.[3] The venous drainage of the vulva enters the pelvic cavity through the saphenous hiatus, pudendal canal, obturator canal and inguinal canal. The venous blood drains mainly to the three pathways: (1) the internal iliac vein, (2) the femoral vein, and (3) the ovarian vein. Moreover, there are anastomoses and communicating veins between the three pathways. The internal pudendal and obturator veins are tributaries of the internal iliac vein. The internal pudendal vein traverses the pudendal canal along the lateral wall of the is chiorectal fossa. The obturator vein enters the pelvis through the obturator canal. The internal pudendal and obturator veins are likely to be compressed by the enlarged gravid uterus, and the mechanical obstruction of the first pathway may be the most probable cause of vulval varicosities in pregnancy.[4] The risk factors forgenital varicosities include increasing age, occupations involving standing for long periods and genetics. Portal hypertension and Klippel-Trenaunay syndrome are the non-pregnancy related causes of genital varicosities. Klippel-Trenuanysyndrome is a congenital vascular disorder that often presents withvulvalrugosities.[5] Vaginal varicosities can also present in Pelvic Congestion Syndrome which sometimes presents in pregnancy with signs of pelvic pain, painful menstrual cycles, pain during micturation, dyspareunia, vulvar and perivulvar varicosities.[6] The vaginal varicosities may rupture due to trauma during the second stage of labour leading to vaginal hematoma. There may be hemorrhage which may have feto-maternal effect.[7] Treatment is generally conservative, firm pressure might be enough.[8] However, there may be an increased need for cesarean deliveryin order to reduce the chance of hemorrhage.[9] In this case, we present large vaginal varicosities in a 34 year old G2P1L1 at 23 weeks and 5days of gestation for which hemorrhage was a concern.

CASE REPORT

Presenting complaint

A female patient, aged 34 years, with an obstetric score of G2P1L1 at 23 week and 5 days of gestation came to the hospital OPD.She complained of swelling in the vagina. The patient had severe discomfort due to the mass, which made walking difficult.

Obstetrichistory

The patient had a past history of vaginal varicosities in her previous pregnancy which was diagnosed at 27 weeks and 2 days. She underwent cesarean delivery in view of risk of hemorrhage. A boy baby, weighing 3.4 kg was delivered. Her intrapartum and postpartum period was uneventful. After delivery, varicosities resolved spontaneously.

Menstrualhistory

Patient attained menarcheat 12 yearsof ageand has regular menstrual cycles with a mild to moderate flow for 3-5 days. There is no past history of gynecologic abnormality. Pap smear tests were negative.

Medical history

She has no past history of liver diseaseor coagulopathies. The patient had no past relevant surgical history.

Family history

She gives no history of malignancy or cancer in her family.

Examination

On examination, bluish black swellings were seen arising from the anterior and lateral vaginal walls. Thesemasses filled the vagina and protruded beyond the hymenal ring. On palpation, the masses were compressible and bag of worms feel was present. Clinical diagnosis of vaginal varicosities was made.

Investigation

Transvaginal ultrasound with colour Doppler was done which showed varicosities arising from the vaginal walls. Doppler ultrasound of the lower extremities did not reveal any abnormality.

Diagnosis

Diagnosis of Vaginal Varicosities was made from the clinical examination and Doppler scan.

Treatment

Since the patient has a past history of previous cesarean delivery and also has high risk of hemorrhage if the patient underwent vaginal delivery, the patient was advised to continue prenatal care and advised for a repeat cesarean section at 38 weeks. A girl baby weighing 3 kg was delivered. The patient had complete spontaneous resolution of her vaginal varicosities when examined during her 6 week postpartum.

DISCUSSION

Vaginal varicose it is are rare in pregnancy. Only few cases have been reported and many of these patients had portal hypertension.[10] However, our patient didn't have portal hypertension. Most of these patients are asymptomatic and varicosities are an incidental finding. Some patients come with complaints of mass or swelling like our patient. If present, pain, pruritis, dyspareunia and discomfort are the common presenting symptoms.[11] Some patients may present with vagina bleeding due to spontaneous or traumatic rupture of varicose veins.[12] Usually, they are treated conservatively by vulvular compression with a pelvic supporter, support hose, elevation of lower limbs, minimal activity involving sitting and standing, and exercise.[13-15] Cesarean section is necessary only for obstetric indications. Usually during vaginal delivery, the veins get compressed due to baby's head and chance for hemorrhage is less. However, if the varices are large and chance for trauma is high, cesarean section has been done in some cases.[15]

CONCLUSION

The varicosities resolve spontaneously by 6 weeks postpartum. The possibility of other anatomical and pathological conditions like leg varices and arteriovenous malformations likeKlippel-Trenuany syndrome should be kept in mind.

CONFLICT OF INTEREST

Conflict of interest declared none.

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REFERENCES

1. Choudhary V, Bellad MB. Varicosities of vulva during pregnancy-a rare case report. Journal of Dental and Medical Sciences. 2017;16(1):75-6.
2. Kim HJ, Lee GH. A case of vaginal varix during pregnancy. Korean Journal of Obstetrics & Gynecology. 2012 Jan 1;55(1):29-32.
3. Furuta N, Kondoh E, Yamada S, Kawasaki K, Ueda A, Mogami H, Konishi I. Vaginal delivery in the presence of huge vulvar varicosities: a case report with MRI evaluation. European Journal of Obstetrics & Gynecology and Reproductive Biology. 2013 Apr 1;167(2):127-31.

4. Jindal S, Dedhia A, Tambe S, Jerajani H. Vulvovaginal varicosities: An uncommon sight in a dermatology clinic. Indian J Dermatol. 2014 Mar 1;59(2):210.
5. Sergey G Gavrilov. Vulvar varicosities: diagnosis, treatment, and prevention. Int J Womens Health. 2017;9:463-475
6. HadizaMoutariSoule et al. PAMJ-CM- 3(157); 04 Aug 2020
7. Mark Sueyoshi, Steven Clevenger, Elaine Hart, "Large Vaginal Varicosities in the setting of Pregnancy without known Hepatic or Vascular Risks:A Case Report and the Review of Literature" Case reports in Obstetrics and Gynecology, vol 2018, Article ID 2394695, 4 pages, 2018
8. R. Tanaka, Y. Fujita, K. IshibashiHiasa et al., "Successful Management of Pregnancy Complicated by Klippel-Trenaunay Syndrome Using MR Angiography-Based Evaluation," *Case Reports in Obstetrics and Gynecology*, vol. 2011, 4 pages, 201
9. S. S. ADLER, "Extensive varix of vulva and vagina in full term pregnancy; delivery by cesarean section," *American Journal of Obstetrics & Gynecology*, vol. 51, pp. 272–274, 1946.
10. D. Bell, P. B. Kane, S. Liang, C. Conway, and C. Tornos, "Vulvar varices: an uncommon entity in surgical pathology," *International Journal of Gynecological Pathology*, vol. 26, no. 1, pp. 99–101, 2007.
11. S. R. Watermeyer, N. Davies, and R. Goodwin, "The Klippel-Trenaunay syndrome in pregnancy," *BJOG: An International Journal of Obstetrics &Gynaecology*, vol. 109, no. 11, pp. 1301-1302, 2002
12. F. Cunningham, K. J. Leveno, S. L. Bloom, C. Y. Spong, J. S. Dashe, and B. L. Hoffman, *Williams Obstetrics*, McGraw-Hill, New York city, NY, USA, Twenty-Fourth edition, 2013.
13. N. Kikuchi, S. Ohira, R. Asaka, A. Takatsu, H. Kobara, H. Ando et al., "A case of vaginal varices that caused massive bleeding after vaginal delivery," *The Shinshu Medical Journal*, vol. 64, no. 1, pp. 35–39, 2016 (Japanese).
14. A. H. Scultetus, J. L. Villavicencio, D. L. Gillespie, T. C. Kao, and N. M. Rich, "The pelvic venous syndromes: analysis of our experience with 57 patients," *Journal of Vascular Surgery*, vol. 36, no. 5, pp. 881–888, 2002
15. G. Orlando, P. Goffette, A. Geubel, and J. Lerut, "Vaginal bleeding complicating portal hypertension: A particular entity—Report of two cases and review of the literature," *Transplant International*, vol. 18, no. 12, pp. 1382–1385, 2005.

SP-53

A Case Report on Scurvy And Its Association of Vitamin C In Diet

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Abstract: According to past literature scurvy has been reported in huge numbers secondary to deficit of Vitamin C in diet. Here we discuss a seven-year-old girl child who presented with complaints of rash, following which she developed ache in bilateral lower limbs. Investigations showed deficiency of vitamin C, hence the child was admitted for treatment feeding program. The assessment concluded that the child had problems with eating and lacked essential vitamins in the diet. Child was found to have only specific 15 types of foods. Throughout the treatment course, she had acquired knowledge about 32 new foods which she started to eat. During follow-up after 6 months the child gained significant weight and eventually her BMI improved.Children who are treated appropriately with supplementation and dietary changes have shown significant improvement and complete recovery. Children with special needs are more at risk for long-term problems with feeding or eating, healthcare providers may provide additional attention to these children to determine the need for referral to providers to address feeding or eating problems.

Keywords: Vitamin C, Scurvy, Children

INTRODUCTION

Deficiency of Vitamin C is known as Scurvy. Ascorbic acid is the other name for Vitamin C present in citrus fruits and vegetables. It is uncommon to see scurvy in young children; a study conducted in a paediatric hospital identified only 32 children with scurry over a period of five years among these only 4 presented due to diet deficit. 3 children were found to have co-existing conditions like autism and 1 of them had intellectual disability[1-3].

CASE REPORT

Presenting complaint

In this case report we will be discussing about a girl who is 7 years old, who presented with complaints of a rash sensitive to sunlight which was reoccurring for the last 3 years.

Previous history

3 weeks back patient presented with rash sensitive to sunlight. On detailed history taking she had reduced appetite and hence gastroenterology opinion was obtained for the same. Examination of the skin on the trunk and extremities showed fine scaling. Examination of skin on lower right leg showed papules which were small in size, pink in colour. With the above said presentations we arrived at a clinical diagnosis of ichthyosis vulgaris and the plan of treatment was sort. After 2 weeks child presented to the casualty with complaints of pain in both knee joints. On local examination of both knee joints it was non tender and when asked to walk she did not complain of any difficulty. Imaging studies of the knee and leg did not reveal any significant abnormalities. She was then diagnosed with tenosynovitis and discharged.

Current history

After a week's time she again presented to the casualty with complaints of pain over the right knee and ankle. When asked to walk she had pain over the left calcaneus. This presentation was indicative of Migratory arthralgia.

Birth history

Prenatal, natal and postnatal period were uneventful. Child was developmentally normal.

Diet history

At 12 months of age, breastfeeding was stopped and she was started on milk & snacks (cereals). For the next 4 years she was on snacks, chocolate pudding, vanilla ice cream, chocolate, and ripe banana, her diet did not include any vegetables, meats or other fruits. No new food was introduced to her. During the time of diagnosis her BMI when plotted was at the 1st percentile. Since her diet was deficit of major macro and micronutrients her growth was much below the expected level.

Investigations

Vitamin C level was tested and the levels were < 7 umol/L (23 to 114 umol/L is the normal reference range). Pre albumin, ferritin and iron saturation were all reduced than the normal reference range. Other vitamins were also tested like vitamin A and vitamin D which were within the normal limits.

Diagnosis

Diagnosis was based on history,clinical findings and vitamin C levels significantly lower than normal values.

Prognosis

In most cases there was full improvement in the condition of the child with resolution of signs and symptoms completely after the finishingthe treatment fully and with dietary modifications.

Treatment

Ascorbic acid therapy was started and further advised to undergo a feeding program.She was started with the feeding program and asked to eat 32 new foods which included all food groups thereby balancing her diet.

RESULT

After a year of her feeding program she grew by 8cms and weight showed an increase of 9kgs, her BMI increased to 85th percentile. During this year of intensive treatment she was provided a meal whenever she was brought to the out patient department to assess the child's ability to consume different types of food.

DISCUSSION

The use of ascorbic acid for treatment of scurvy due to dietary deficit has been explained in earlier studies. In most scenarios there was full resolution of signs and symptoms after the completion of treatment. In certain circumstances there were multiple vitamin deficiencies hence vitamin C was alone not enough to match the required nutritional deficit[4]. Not only vitamin supplementation is useful to meet the deficit, the diet of the child plays the major role in correcting the nutritional deficiencies. In the above mentioned case the diet of the child played a major role to meet the deficit. In another study it was found that four children who had presented with haematological complications were diagnosed with scurvy due to dietary deficit of Vitamin C. It is common to see other vitamin deficiencies coexisting with Vitamin C deficiency more commonly Vitamin A, Vitamin B1, Vitamin B when there is an improper diet intake[5]. With extensive review of literature of case reports and studies it is clear that short term correction of deficiencies shows resolution, whereas long term follow up outcomes were difficult. Selective food eaters are prone to multiple vitamin deficiencies than those who eat a complete balanced nutritious diet. In the existing literature, reports of contact with pediatric providers prior to diagnosis of the vitamin C deficiency were noted in numerous studies[6-9]. There are no indications that children diagnosed with vitamin deficiencies are not receiving regular healthcare. Based upon our experience with the current case and the children referred to our organization's feeding program, we suggest the extent of some children's diet limitations are not always clear to healthcare providers[10,11]. One large population-based study found 46% of parents identified their children as picky eaters at some point during childhood and picky remitted in two-thirds of cases within 3 years[12]. Picky eating does persist in some children, with one study showing picky eating as a stable trait through age 11[13]. As providers hear about picky eating often and it usually resolves, it may be difficult to differentiate the transient picky eating commonly seen from the selective eating that could result in nutrient deficiencies. While the role of dietary limitations on the development of nutritional deficiencies, namely vitamin C, was the focus of this case study and literature review, it is worth mentioning the child in this case study demonstrated a significant increase in her body mass index, increasing from the 1st to 85th percentile in 1 year. Certainly, some of this growth can be attributed to the increased number of calorie-dense foods he learned to eat, we also hypothesize the increased total variety of foods, including fruits and vegetables, also helped with weight gain. It is known that eating a food or limited foods over time results in monotony, or a decreased desire to eat this food or foods[14]. Increasing diet diversity can decrease the effects of monotony and lead to increased weight gain, especially if the diet contains some foods high in energy density[15,16]. Thus, increasing a child's diet variety can not only prevent nutritional deficiencies, it can support adequate intake.

CONCLUSION

While the child in our case study was a young girl with typical development, review of the clinical cases of vitamin C deficiency showed children with special needs, especially children with autism spectrum disorders, were over-represented. This is consistent with the broader literature on childhood feeding problems which shows feeding problems occur at a higher prevalence in children with special needs. As children with special needs are more at risk for long-term problems with feeding or eating, healthcare providers may provide additional attention to these children to determine the need for referral to providers to address feeding or eating problems.

CONFLICT OF INTEREST

Conflict of interest declared none.

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REFERENCES

- Solanki M, Baweja DK, Patil SS, Shivaprakash PK. Ascorbic acid deficiency: A case report. J Dent Child. 2011;78(2):115–9.
- Harknett KM, Hussain SK, Rogers MK, Patel NC. Scurvy mimicking osteomyelitis: case report and review of the literature. Clin Pediatr. 2014; 53(10):995–9.
- Golriz F, Donnelly LF, Devaraj S, Krishnamurthy R. Modern American scurvy—experience with vitamin C deficiency at a large children's hospital. PediatrRadiol. 2017;47(2):214–20.
- Paul C, Williams KE, Riegel K, Gibbons B. Combining repeated taste exposure and escape prevention: an intervention for the treatment of extreme foods selectivity. Appetite. 2007;49(3):708–11
- Willmott NS, Bryan RAE. Scurvy in child with epilepsy on a ketogenic diet with oral complications. Eur Arch Paediatr Dent. 2008;9(3):148 –52.
- Kapadia M, Beck M, Comito M, Dandekar S. Malignancy vs scurvy; similar manifestation different pathophysiology-case series. Pediatr Blood Cancer. 2016;63:S86.
- O 'Hara C. Scurvy related to the use of a homemade tube feeding formula. Infant Child AdolescNutr. 2015;7:381 –4.
- Ali SR, Hamilton R, Callaghan M, Brown A, Gibson L. G429 (P) a 13 year old with fussy-eating induced blindness. Arch Dis Child. 2014;99:A179 –80.
- Baird JS, Ravindranath TM. Vitamin B deficiencies in a critically ill autistic child with a restricted diet. Nutr Clin Pract. 2015;30(1):100 –3.
- Keown K, Bothwell J, Jain S. Nutritional implications of selective eating in a child with autism spectrum disorder. BMJ Case Rep. 2014. https://doi.org/10. 1136/bcr-2013.202581.
- Williams KE, Hendy HM, Field DG, Belousov Y, Riegel K, Harclerode W. Implications of avoidant/restrictive food intake disorder (ARFID) on children with feeding problems. Child Health Care. 2015;44(4):307 –21.
- Cardona Cano S, Tiemeier H, Van Hoeken D, Tharner A, Jaddoe VWV, Hofman A, Verhulst FC, Hoek HW. Trajectories of picky eating during childhood: a general population study. Int J Eat Disord. 2015;48:570 –9
- Mascola AJ, Bryson SW, Agras WS. Picky eating during childhood: a longitudinal study to age 11 years. Eat Behav. 2010;11(4):253 –7.
- Raynor HA. Can limiting diet variety assist with reducing energy intake and weight loss? Phys Behav. 2012;106(3):356 –61.
- McCrory MA, Fuss PJ, McCallum JE, Yao M, Vinken AG, Hays NP, Roberts SP. Dietary variety within food groups: associations with energy intake and body fatness in men and women. Am J Clin Nutr. 1999;69(3):440 –7.
- Field DG, Garland M, Williams KE. Correlates of specific childhood feeding problems. J Paediatr Child Health. 2003;39:299 –304