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Case Report

Ayurveda for Duchenne Muscular Dystrophy

Relevance of Ayurveda Management in Duchenne Muscular Dystrophy to Augment the Quality of Life- A Pediatric Case Report

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Abstract: Muscular dystrophy is a myopathy that stakes clinical characteristics of progressive muscular feebleness. Duchenne muscular dystrophy (DMD) is the most common X-linked disorder of muscular dystrophy in children, present in early childhood, and characterized by proximal muscle weakness and calf hypertrophy in affected boys. This case aims to make Ayurveda pediatricians aware of planning a protocol for DMD. The objective is to provide instant relief in motor function to some extent and to augment the quality of life. An 11 years old boy was presented with an in ability to stand, gross muscle weakness, difficulty in movements of lower limbs, and particularly in all vigorous physical activities. He also had decreased strength, stamina, and progressive debility with positive Gower's sign, which suggested DMD. Confirmed economic treatment options are unavailable to prevent progressive illness and mortality. The family members were counselled and assured of improving their quality of life. He was started with multiple pre-panchakarma procedures of alternate Rukshan and Brihan to pacify internal Ayurveda medicines with the support of occupational therapy and physiotherapy in different sittings for three months. His CPK -creatinine kinase was 8335 U/L (NV= 55 to 170 U/L) which became4741U/L post-treatment. He could stand with support for 15 minutes on his own with ease in body movements due to a reduction in contractures & increase in the range of motions measured by the goniometer, as this genetic disease has no medicinal cure. However, by adopting a multi-dimensional integrative approach, family support, and willpower, the quality of life can be augmented to much extent. Ayurveda plays a crucial role in the drastic improvement because of panchkarma shaman procedures, medicines, Yog, physiotherapy, traction, and counselling at regular intervals for lifelong.

Keywords: Myopathy, DMD, Duchenne's Muscular dystrophy, X-linked recessive, Creatine kinase, Gower sign, Ayurved interventions

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Various types of muscular dystrophies are affected due to genetic involvement, such as x-linked recessive, AR-autosomal recessive, AD- autosomal dominant. In the x-linked recessive variety, there are again 3 subtypes such as Duchenne's (DMD), Becker's (BMD), and Emeri Dreifuss (EDMD). In these types, Duchenne's dystrophy has very less lifespan. Former 2 lacks dystrophin protein in muscle, but Becker's dystrophy is slowly progressive and may have an altered amount of dystrophin. EDMD is rare but may be fatal due to cardiomyopathy. These are generally expressed in males. Dystrophies are divided into three categories based on how they are inherited: X-linked, autosomal, and mitochondrial inheritance. Table no. 1 shows the total number of types and their type of genetic inheritance, prevalence, and age of onset in short[1]. The various forms of muscular dystrophy are as follows.

Table 1: Type wise genetic inheritance pattern with the age of onset depiction

Emery-Dreifuss Muscular Dystrophy	X-linked recessive defect in nuclear protein emerin at the Xq27-28 position. Defect in inner laminar protein on 1 chromosome	Prevalence (General Population): 0.22 to 0.39 per 1 lakh population	The age of onset is 10 years onwards,
Limb-Girdle (Erb) Muscular Dystrophy	caused by myotilin gene deletion. X linked dominant	Prevalence (General Population): 0.48 to 1.63 per 1 lakh.	Variable age of onset as per the involvement of muscles. The recessive form is earlier, quick progress, while the dominant is later & slower
Facioscapulohumeral (FSHD) Muscular Dystrophy	due to a mutation in the D4Z4 region in FSHD1, autosomal dominant	Prevalence (General Population): 0.29-3.95 per 100,000.	10-30 years is the age of onset, X linked dominant
Myotonic Muscular Dystrophy: most common in adults	, results from the impaired expression of the Dystrophia Myotonica Protein Kinase (DMPK).	Prevalence (General Population): 8.26 per 100,000.	10-15 years is the age of onset, X linked dominant
Oculopharyngeal Muscular Dystrophy	Caused by an autosomal dominant GCG trinucleotide repeat resulting in deficient mRNA transfer from the nucleus.	Prevalence (General Population): 0.13 per 100,000.	30-40 years is the age of onset, X-linked dominant
Congenital Muscular Dystrophy	Caused by a mutation of the sarcolemmal protein Merosin gene	Prevalence (General Population): 0.99 per 100,000.	Autosomal recessive and present since birth.
Becker Muscular Dystrophy	Caused by a mutation of the muscle protein dystrophin gene	Prevalence (General Population): 4.78 per 100,000.	10-20 years is the age of onset, X-linked recessive
Duchenne Muscular Dystrophy, most common in children	, is a dystrophin gene mutation located on the X chromosome's small arm (p) at the Xp21 position.	Prevalence (General Population): 4.78 per 100,000.	3-5 years is the age of onset, X-linked recessive

This case aims to make pediatricians aware of the good results of Ayurveda management in DMD. It must be started integrating Ayurveda modalities by Ayurveda experts with modern medicines like steroids. The objective is to provide instant relief in motor function to some extent and to augment the quality of life by multiple interventions at regular intervals, including panchakarma procedures, yog, sattvavajay chikitsa (counselling, positivity, etc.), and Occupational and physiotherapy.

1.1. Duchenne Muscular Dystrophy-(DMD)

Females are affected if the X chromosome carrying the normal allele is lost or inactivated. The gene responsible for DMD maps to the short arm of the X chromosome at band Xp21. It is an atypical inherited musculoskeletal disorder that shows clinical characteristics of progressive muscular weakness due to the absence of dystrophin protein in the muscle, which later replaces fibrosis and fat deposition. It is an X-linked recessive disorder occurring 1 in every 3500 live male births and was named after a French neurologist Guillaume Benjamin Amand Duchenne, in 1860[2-4]. As we receive good results in this case, it is aimed to make aware Ayurveda pediatricians plan a protocol of DMD, and the objective is to provide instant relief in motor function to some extent and to augment the quality of life.Early features are Difficulty climbing stairs- places hands on the next step to lift himself and uses the support of railing or wall while later Waddling gait with compensatory lumbar lordosis occurs. It is the most common and severe form of muscular dystrophy, beginning at 3–5 years of age and characterized by proximal muscle weakness and calf hypertrophy in affected boys. DMD has a very high transmutation rate with distinguishing and persistent signs and symptoms[5]. The list of other clinical features is loss of coordination of muscles, developing contractures, progressive scoliosis, impaired gait, cardiomyopathy, poor lung function, and cognizance. The diagnosis is mainly based on history, typical clinical features, and creatinine phosphokinase, which increases in thousands from normal less than five hundred as the maximum range[56]. An early intervention like physiotherapy with Shaman panchakarma procedures such as Kwath dhara, patrapottali, pindaswedan, nadiswedan, upanah, or *Utsadan* and matrabasti is not started. In that case, the patient has to be bound with a wheelchair at or before 8-10 years of age[67]. Oral use of steroids can temporarily halt the progress of the disease but have side effects. Such patients have a very short lifespan of around twenty years as heart muscles get involved gradually and stop working 4,5. Confirmed economic treatment options are unavailable to prevent their progressive illness and mortality. As this is a genetic disease hence comes in the category of Aadibalapravritta vyadhi which is yapya to

asadhya -unable to cure grade of badprognosis[8]. Although temporary but giving results by Ayurvedic management in such diseases then, it is the power of Ayurveda to improve the quality of life with no adverse effects.

1.2. Patient Information

A11 Years old male child was clinically diagnosed with a case of Duchenne muscular dystrophy with the following chief complaints of inability to stand and walk for 1 month. It was associated with multiple joint pain, muscle weakness, difficulty standing, walking even with support, and frequent falling. As per history, his parents did not have a consanguineous marriage; one brother and two sisters were healthy, and no other family members were affected by a similar ailment. In history, he had typhoid at the age of six, and his mother was suffering from swine flu in 2nd trimester of pregnancy and managed by modern medicines.

1.3. Ethical Statement

Written informed consent was obtained before commencing the study, management, and before publication from the parents of the child patient for both sittings.

1.4. Clinical findings

His Clinical Presentation results showed the muscle weakness was mainly in the 'proximal' muscles, meaning those near the body's trunk, around the hips, and the shoulders. There were positive tripod, Gowers, and valley signs. He has been suffering from frequent falls and difficulty rising from a lying or sitting position. Before 2 months, he had a waddling gait and used to walk slowly on his toes. He was losing his balance and stopped standing also. At a younger age, he had trouble running and jumping[5,6]. Currently, he has large calf muscles. He feels muscle pain and stiffness with mild shoulder and hip girdle muscle contractures. Also, he needed to develop more interest in academics. Here is the sitting-wise depiction of pre-post anthropometry in table no. 2.

Table No. 2 Depicts sitting-wise parameters of anthropometry

Anthropometric parameters	1st sitting	2nd sitting after 2 month's gap
Weight in Kg	31	32 kg
Height in cm	116	122
Head Circumference	51	51
Mid arm circumference	19	19.2
Chest Circumference	59 cm	60 cm

Here are the images showing the dependency of a wheelchair despite orthotic support Images 1 and 2 show the Gower's sign positive, taking support from the floor to stand from a sitting posture.

Fig 1: Showing Pre-treatment condition as unable to stand and depicting the dependency on a wheelchair for daily activities

Table 3: Shows the nidanpanchak of DMD with modalities to break the etiology

Nidan Panchak/factors responsible for disease occurrence	Samprapti Ghataka	Samprapti Vighā tana3
Dosha	Vatadhiktridosha	Basti
Dushya	Rakta, mamsa, asthi, sandhi, snayu, kandara	Abhyanga, Swedana
Srotas	Rasa-Rakta and majjavaha	Srotoshodhak & Medhya drugs
Agni	Mandya	Deepan-pachan drugs
Srotodusti	Sanga(obstruction)	Srotoshodhan by basti
Udhabhavsthan	pakwashaya(being a Vatavyadhi)	Basti
Vyaktasthana	sarvang(adhoang)	Sarvang Snehan and sewdan
Roga	DMD	Vatvyadhi Chikitsa(Snehan, Swedan, Basti, Snehapan)
Sadhya/asadhyatva	Kruchhasadhya	Long-term treatment protocol

Fig 2: Patient uses Gower's maneuver sign which confirms the diagnosis of DMD

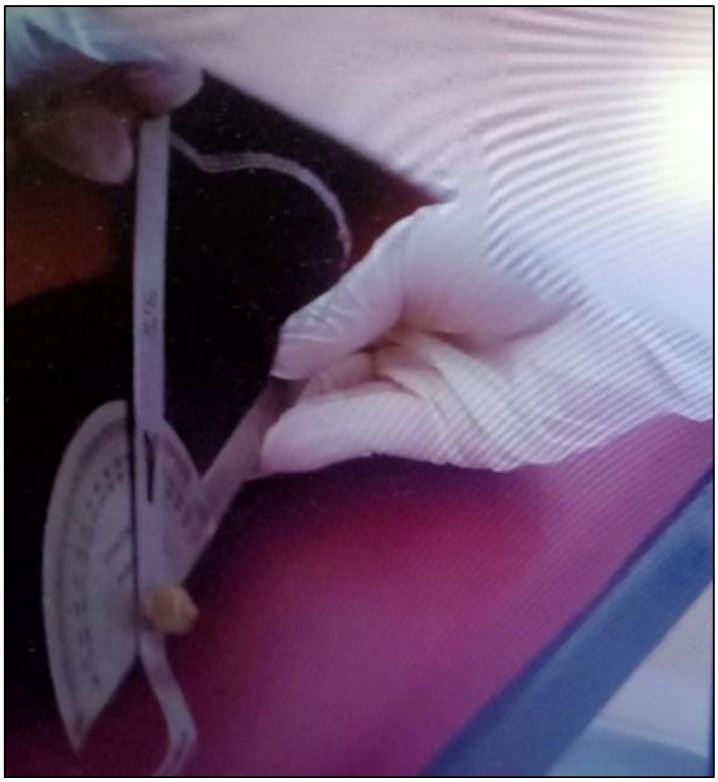
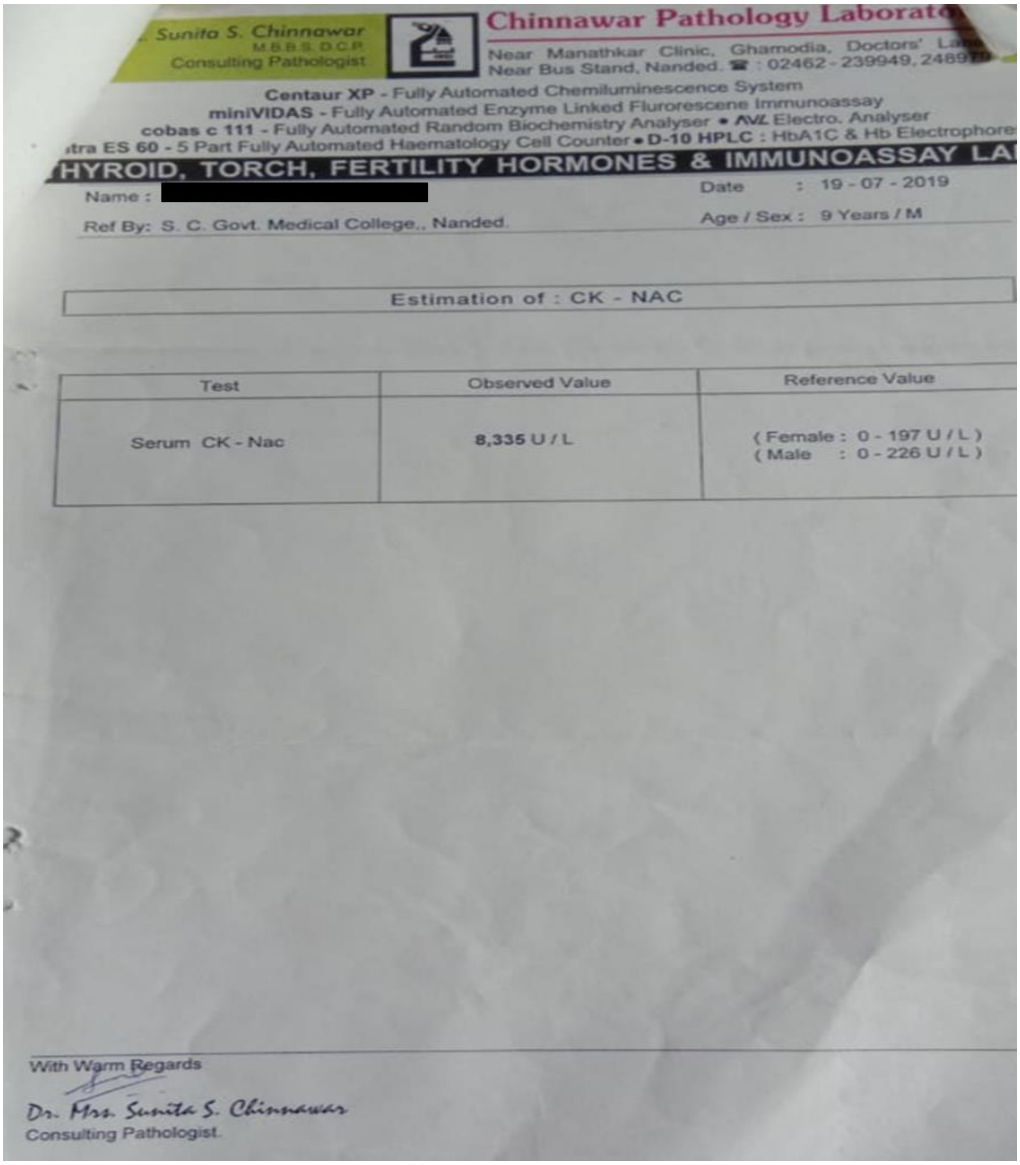


Fig 3: Measurement of angle by Goniometer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Centaur XP - Fully Automated Chemiluminescence System
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cobas c 111 - Fully Automated Random Biochemistry Analyser • AVL Electro. Analyser
tra ES 60 - 5 Part Fully Automated Haematology Cell Counter • D-10 HPLC : HbA1C & Hb Electrophore

HYROID, TORCH, FERTILITY HORMONES & IMMUNOASSAY LA

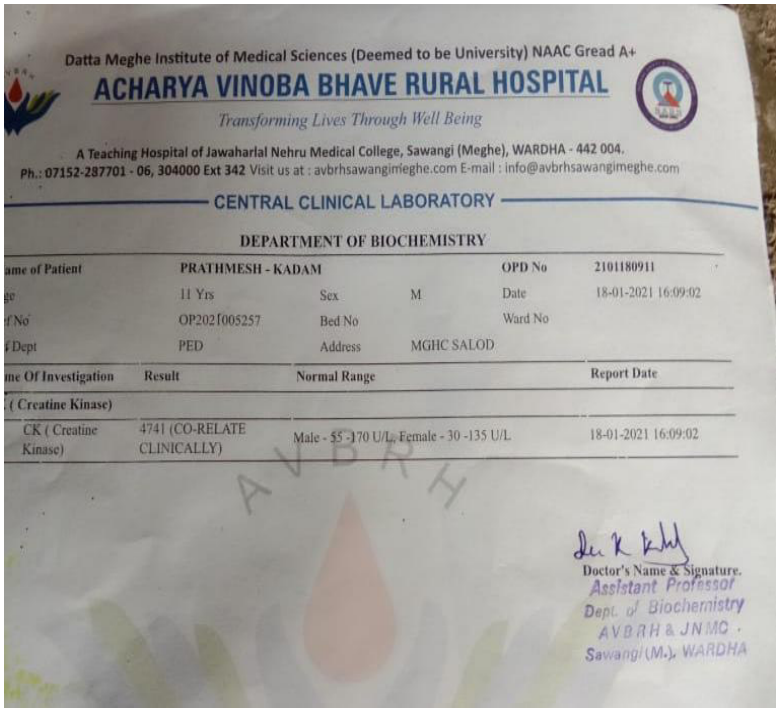
Name : [redacted]
Date : 19 - 07 - 2019
Ref By: S. C. Govt. Medical College., Nanded.
Age / Sex : 9 Years / M

Estimation of : CK - NAC

Test	Observed Value	Reference Value
Serum CK - Nac	8,335 U / L	(Female : 0 - 197 U / L) (Male : 0 - 226 U / L)

With Warm Regards
Dr. Mrs. Sunita S. Chinnawar
Consulting Pathologist.

Fig 4: Receipt from the central clinical laboratory of Acharya Vinoba Bhave rural hospital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CENTRAL CLINICAL LABORATORY

DEPARTMENT OF BIOCHEMISTRY

ame of Patient	PRATHMESH - KADAM			OPD No	2101180911
ge	11 Yrs	Sex	M	Date	18-01-2021 16:09:02
f No	OP202f005257	Bed No		Ward No	
f Dept	PED	Address	MGHC SALOD		

me Of Investigation	Result	Normal Range	Report Date
(Creatine Kinase)			
CK (Creatine Kinase)	4741 (CO-RELATE CLINICALLY)	Male - 55 -170 U/L, Female - 30 -135 U/L	18-01-2021 16:09:02

AVBRH

Doctor's Name & Signature.
Assistant Professor
Dept. of Biochemistry
AVBRH & JNMC
Sawangi (M.), WARDHA

Fig 5: Clinical laboratory report from Acharya Vinoba Bhave rural hospital.

Fig: 6 Show the ability to stand post-treatment

1.5. Diagnostic Assessment

Diagnosing DMD is very easy as it has typical characteristic features that start at an early age group in boys. Generally, boys suffer from delayed milestones and, soon after play or exertion, feel fatigued, and there is a history of recurrent falls due to muscular weakness,[4,5]. The tripod and Gower's sign are positive, clinically supporting the floor and mid-thigh when asked to stand from sitting. Creatinine phosphokinase starts rising in thousands from 150 to 499 U/L normal value. He has kyphosis and scoliosis, together with short stature. There was no bowing of legs[56]. Images 4, 5, and 6 show the difference of CPK (creatinine phosphokinase) value by 4694 during pre-post treatment, a typical diagnostic investigation raised in thousands of numbers. Table no. 4 shows the pre-post-treatment CPK value and neurological assessment.

Table 4: Showing investigations &neurological examination[9] status atthe pre-post treatment.

Sr no.	Investigations	Pre-treatment	Value observed post t/t after 6 months
1	CPK (NV=55-170 U/L)	8335 U/L	4741U/L
2	Power of B/L lower limbs	1/5	4/5
3	Reflexes	Absent in both lower limbs	Sluggishly appeared
4	RoM	Lower limbs RoM-very less with difficulty	RoM increased that he was able to stand and supportive walk
5	Bulk	Calf muscles were hypertrophied, bulky, and hard in consistency	Although weight is increased, calf muscle bulk was reduced by 1 cm in each leg and softened due to pre-panchakarma procedures after post-treatment
6	Tone	hypertonia	slightly improved
7	Motor function	The standing grade of the CDC was 2	Standing grade -5

1.6. Therapeutic interventions

Bahya Snehana was given to the patient through the Bala Ashwagandha taila and Nadi swedana by Dashmoola kwatha was given to the patient. As depicted here, different procedures can be categorized under Vatahar, strotoshodhak, and Brihan functions. These modalities are directly helpful in breaking the etiology by their opposite action, as shown in table no. 3.

- *Vatahara –Utsadan, Snehana, Nadi swedana, Patrapottali swedan, Basti*
- Strotoshodhak- Upanaha, Patra pottali swedan, Niruhabasti, Kwathadhara
- *Brimhana- SSPS, Majja basti*
- *Sarvanga utsadan* with *Dashamoola Taila & Triphala churna* for first 3 days *Upanaha* with *Vacha, Rasna, Ashwagandha, Wheat Powder, Erandapatra* for 10 days.
- *Pindaswedana with Shashtishali rice* for 10days.
- *Majjabasti* with bone marrow of goat with milk (40 ml), *Bala, Rasna, Ashwagandha* with 10ml *of Panchtikta ghrit* dose of 50 ml.
- Physiotherapy, occupational therapy
- Patrapottali Sweda (Nirgundi, Nimba, Arka, Bilva patra, etc., vatahar herbs)
- Internal administration of medicines is shown in Table 5 for 1st to 2nd sitting as per duration.

Table 5: Showing brief information on shaman aushadhis-medical management

No	Medicine	Dose	Anupan, timings & duration
1	*Ashwagandha Churna*	3gm BD	Luke warm water, post-meal x15 days
2	Dhatupachak vati	1 BD	Luke warm water, post-meal x15 days
3	Tab. Ampachak vati	1Tab BD	Luke warm water, post-meal, for 5 days
4	Brahmi Ghrita	10 ml BD	After 5 days of Ampachak vati, with lukewarm water, x15 days

5	Ksheerbala tail-101 Avarta drop	8 drops BD	With Brahmi Ghrita, empty stomach for 15 days
6	Shankha Vati	1Tab.BD	Luke warm water x15 days
7	Kanchnar Guggulu	1Tab.BD	Luke warm water
8	Maharasnadi Kwath	5ml BD	Luke warm water

The details of panchakarma procedures and said medicinal management are shown in Table No. 5, which helped remove contractures and boost motor function. Images 7 and 8 depict the ability to stand independently without support post-treatment compared to the dependency shown in image 1.

Table 6: Depicting the procedural information

No	Name of the procedure & duration	Medicine used with the brief method	Mechanism of Action
1	Utsadan x 15 days	Bala-Ashwagandhadi tail plus Vacha powder in 5: 1 ratio	Improve micro-circulation &&vasodilatation[10]
2	Kwathdhara x 7 days	Dashamool Nirgudi kwath	Relaxes muscles, remove pain[1110]
3	Pindasweda in the morning for the next 7 days of kwathdhara	Shali/rice bolus with Bala & milk	Relieve pain and provide nourishment[7]
4	Upanah in the evening for 7 days	Medicated warm external application	Decreases spasticity[12]
5	Patra pottali swedan, for next 7 days after Upanah	Roasted Vatahar medicinal bolus	Pain and swelling reduction
6	Kalabasti for 30 days	Alternate matra and niruhabasti	Best Vatahar[13]

After the 1st setting of 1 month, the patient could stand with support for 10 minutes. Occupational and physiotherapy were also going on. There were so many interventions that were started for a total of one-month duration as the patient came from a far distance, and owing to the genetic origin of the disease's nature with a bad prognosis, a holistic approach is mandatory to get a cumulative effect. There was a gap of 2 months as the patient's parents couldn't bring him after 1 month due to personal family issues. In 2nd sitting, Kumarkalyan Rasa1/2 tab +Abhrakbhasma 200 mg+Trikatu 300mg+Triphala Churna 500mg +Ashwagandha Churna 1 gm+Guduchi satva 200mg combination was given for 10 days, twice a day. After that, Panchtikta ghrit 10 ml and Ksheerbala 101 Avartan oil 10 drops were given on an empty stomach for 1 month, including Ashwagandha and Kanchnar Guggulu with Maharasnadi Kwath. The patient was admitted for 22 days.

Table 6: Showing the panchakarma procedural details of 2nd sitting from 28.11.2020 to 17.12.2020

No	Name of the procedure & duration	Medicine used	Mechanism of Action
1	Abhyanga x 7 days	Warm Dhanvantar tail	Best Vatashamak[14]
2	Nadiswedan in the morning x 7 days		Pacifies Vata
3	Nasya for the next 10 days	Brahmi taila	Boost brain function, [15]
4	Shirodhara for10 days after Nasya in morning	Brahmi taila	Augment brain circulation and promote relaxation, promote academics & relieve stress[16]
5	Upanah for 10 days' in the evening	Medicated herbal warm external application as a layer on both lower limbs for at least 6 hours	It reduces contractures
6	Rajayapan basti x 10 days	Mamsarasa, Goat marrow, milk and panchtikta ghrit matra basti	Brihan (anabolic) effect as DMD is a wasting disease of weak muscles[17]

In the 2nd session of treatment, the patient was able to walk with the support of a wheelchair; spasticity was reduced, and can able to stand for up to 5 min without support. The same medicinal treatment was followed at home, and procedures were also suggested with a gap of 4 weeks. Still, again due to covid situation, the patient could not come to get admitted for procedures. In Occupational therapy, supportive braces were attached with footwear to make him stand. Physiotherapy was given to the patient, and he trained to continue at hometo strengthen his weak muscles.

1.7. Follow-up and outcomes

The child was brought for follow-up after approximately 3 months of 1st sitting. The outcomes shown during this treatment improve the symptoms of DMD also appreciated by the patient. After 1st sitting, the patient reported mild relief in generalized weakness, and walking was mildly improved. The patient could still not sit in a squatting position, and his slippers slipped out of his foot. After the 2nd sitting, the patient appreciated relief in calf muscle pain and tightness. Mild improvement was also seen in the sitting squatting position. Walking was improved, and the patient could walk without falling. There was no slipping of slippers by the end of the second sitting. The patient has reported improvement in standing from a sitting position. Power in both the upper and lower limbs was improved to 4/5 (Elevation against moderate resistance) after 2nd sitting.

2. DISCUSSION

In *Ayurveda,* this pathogenesis occurs due to the Bheejabagahaavyava Dusti, which leads to Vata Prakopa and takes sthana samshraya in Mamsa and medo Dhatu vitiates and depletes them. Acharya Charaka has mentioned the close relation of Mamsa and Medo Dhatu to Dhatukshayaj vata pathogenesis, which degrades and causes the mamsa dusti. This Ansha-ansha kalpana of the Dhatus signifies the involvement of the Dhatvagni Mandhya causes Kshaya. This Agnimandya caused at the level of the dhatus leads to the formation of ama. Madhavkara explained Srotodusti as a type of Ama itself while Srotorodha, a subtype of srotodusti, produces hypertrophy in the particular region; it also manifests as first parkopa then depletion, i.e., due to vata[18].Initially, counseling was done, and Achar Rasayana (Code of Conduct) was explained to his parents for better parenting and motivation[19]. The treatment principles are deepan, pachan, srotoshodhak, and balya through external and internal interventions. There was agnimandya in this case; hence Amapachak vati was started as there was a need to boost not only *jatharagni* but also *dhatvagnis*. Ashwagandha powder and Dhatupachak vati play an important role in augmenting strength to mansagni with amapachan of mansadhatu to remove the obstruction (sanga)[20.] As a result, contractures, which were present before treatment and after 3 months, were relieved. Also, there was an enhancement in motor function and daily living activities. Informed consent was taken before examination and treatment before admission, and every procedural commencement.[9] was more efficacious than plain Abhyanga and included in both the sittings.Sarvanga Dhara by Kwath reduces rigidity and improves muscle strength due to the Kapha-vatahar action. Pindaswedan is good for diseases associated with wasting. In a few studies, pindasweda was result-oriented due to its simultaneous triple action of the vatahar mechanism by pre-procedure abhyanga, swedan, and lepan with nutrition[21]. Upanah is helpful in the reduction of spasticity and contractures due to its ushna, teekshna Kapha-vatahar action[11] Karmabasti is beneficial in vatashaman, balya, and strotoshodhak[67]. Rajayapan basti was given to boost motor function and bruhan action. As mamsa added in basti is accommodating depleted mamsa with the principle of augmentation if a supplement of the same substance is provided as per Charaka[17]. Shirodhara and Nasya were given to reduce the academic and general stress and behavioral issues due to the disease condition and provided calmness and relaxation to the mind[22]. Also aimed to maintain the brain function due to vasodilatation in future depletion of dystrophin in brain tissue, which may lead to a low intellect and poor memory[23] Yamak Snehapan of Brahmi ghrit and Ksheerbala tail drops with Suvarnakalpa like Kumar kalyanrasa, Abhrak bhasma is supportive of providing quick results in motor function owing to its sarvadhatu pushtikar-anabolic, nootropic, antioxidant-immunomodulator and strotoshodhak properties[24-27]. For the sake of multi-interventions, the child could stand, and the supportive walk started again with a drastic reduction of creatinine phosphokinase as depicted in pre-post investigations images.

3. CONCLUSION

The present study was commenced to augment the functional and physical competencies, curtailing the disability to postpone the further progression of the disease, maintain motor function for a longer time, and improve the quality of daily living activities. This patient's overall effect was nearly 30-35 %, mainly because of multiple interventions like abhyanga, pindaswedan, patrapottalisweda, kalabasti, rajyapan basti, yog counseling, etc. As this disorder is incurable, this percentage of improvement also helps the patient live life easily. Ayurvedic management postpones the contractures like deformity and maintains motor function to some extent. In a nutshell, integration of Ayurved, Yog, physiotherapy, and counselling (Sattvavajay chikitsa) is mandatory to improve the quality of life at regular intervals for lifelong.

4. AUTHOR'S CONTRIBUTION STATEMENT

Case treatment and writing-Dr Renu Rathi & Bharat Rathi, all other authors, helped to design the article.

5. CONFLICT OF INTEREST

Conflict of interest declared none.

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