ijlpr 2022; doi 10.22376/ijpbs/lpr.2022.12.5.L49-54

International Journal of Life science and Pharma Research

ISSN 2250-0480

Association of Biochemical Anemic Markers in STEMI and NSTEMI Patients – A Retrospective Study

S. Aishwarya[1], M.Vasanthan[1]*, V.M.Vinodhini[1], Renuka. P[1] and V.E. Dhandapani[2]

[1] *Department of Biochemistry, SRM Medical College Hospital & Research Centre, Faculty of Medicine and Health Sciences, SRM Institute of Science and Technology, SRM Nagar, Kattankulathur, 603203, Kanchipuram, Chennai, TN, India.*

[2]*Department of Cardiology, SRM Medical College Hospital & Research Centre, Faculty of Medicine and Health Sciences, SRM Institute of Science and Technology, SRM Nagar, Kattankulathur, 603203, Kanchipuram, Chennai, TN, India.*

Abstract: Body iron stores and its significant association with the increased risk of cardiac disease was first time reported by the National Health and Nutrition Examination Survey. There are several molecular mechanisms and discrete pathways involved in developing Myocardial Infarction (MI) as a result of increased body iron store. Various studies showed that there lies a significant association between serum iron and serum ferritin with the MI, but to the best of our knowledge studies have not focused on the difference in their levels in STEMI and NSTEMI patients. Hence, the aim of the study is to associate the levels of iron and ferritin with STEMI and NSTEMI patients and the objective of the study is to investigate and associate the iron, ferritin and lipid profile parameters among STEMI and NSTEMI patients. The cross-sectional study recruited 75 individuals who lie in the age group of 25-55 years. The participants were divided into three groups with 25 participants in STEMI, 25 in NSTEMI (based on the ECG findings and cardiologist findings) and 25 healthy participants who are age and sex-matched as controls. The blood sample was obtained from the participants and analysed for the following biochemical parameters: CK - MB, iron, ferritin, and lipid profile. The data was analysed using SPSS software and represented as mean and SD. One-way ANOVA, Pearson's correlation and odds calculation were carried out. For all statistical test p value <0.05 were considered as significant. It is found that serum iron and ferritin were significantly elevated in STEMI individuals compared to NSTEMI and controls. But the lipid profile levels were increased in NSTEMI individuals. It is found that individuals with increased levels of iron and ferritin are at high risk of developing STEMI and NSTEMI compared with controls. Iron and ferritin were significantly elevated in STEMI individuals compared to NSTEMI individuals. In NSTEMI group lipid profile levels were increased and is associated with ferritin levels compared to STEMI group. Independent to the type of MI, individuals with high levels of serum iron and ferritin are at higher risk of developing myocardial infarction.

Keywords: Iron, Ferritin, Myocardial Infarction, STEMI, NSTEMI and Lipid Profile.

***Corresponding Author**

M.Vasanthan , Department of Biochemistry, SRM Medical College Hospital & Research Centre, Faculty of Medicine and Health Sciences, SRM Institute of Science and Technology, SRM Nagar, Kattankulathur, 603203, Kanchipuram, Chennai, TN, India.

Received On 31 May 2022

Revised On 08 July 2022

Accepted On 15 July 2022

Published On 01 September 2022

Funding This research did not receive any specific grant from any funding agencies in the public, commercial or not for profit sectors.

Citation S. Aishwarya, M.Vasanthan , V.M.Vinodhini , Renuka. P and V.E. Dhandapani , Association of Biochemical Anemic Markers in STEMI and NSTEMI Patients – A Retrospective Study.(2022).Int. J. Life Sci. Pharma Res.12(5), L49-54 http://dx.doi.org/10.22376/ijpbs/lpr.2022.12.5.L49-54

This article is under the CC BY- NC-ND Licence (https://creativecommons.org/licenses/by-nc-nd/4.0)

Copyright @ International Journal of Life Science and Pharma Research, available at www.ijlpr.com

1. INTRODUCTION

Body iron stores and its significant association with the increased risk of cardiac disease was first reported by the National Health and Nutrition Examination Survey (NHANES III), in 1988-1994[1]. Iron plays an important role in various physiological processes. Iron in overload is known as a risk factor for the progression of atherosclerosis[2]. Various mechanisms are involved in atherogenesis due to increased iron load. Thereafter, various studies showed that there lies a significant association of serum iron, serum ferritin with the myocardial infarction (MI)[3] . Ferritin is the protein complex in which the iron is stored in our body. Serum ferritin concentration is the measure of iron stored in our body[4]. Therefore, higher ferritin concentration is a unique risk factor for MI[5] . There are several molecular mechanisms and discrete pathways involved in developing MI as a result of increased body iron store. The pathological role of iron in inducing MI is involved by catalyzing free radicals such as H_2O_2 and O_2^-, which may induce various deteriorating processes. Iron in ferrous state, contributes to the following reactions known as the Haber–Weiss and Fenton reactions that catalyze the formation of OH- from O_2- and H_2O_2. From heme and ferritin, iron is liberated by the reactive oxygen species (ROS) during oxidative stress[6]. These reactions are mechanically related in formation of atherosclerosis, by increasing the LDL- C peroxidation, thus increasing the uptake by the macrophages that develop into foam cells, leading to Acute myocardial infarction (AMI)[7]. The Fenton and Haber–Weiss reactions are also relevant in the elucidation of iron's role in cardiac reperfusion injury. In ischemic injury reperfusion of cardiac myocytes results in highly toxic OH- is produced in the presence of intracellular iron, thus supporting lipid peroxidation and release of hydrolytic enzymes, with increased in intracellular lysozyme fragility complicating pre-existing atherosclerosis that results in AMI[8]. Another evidence is that iron oxidatively damages the atherosclerotic plaque by promoting endothelial activation, enhancing adhesion molecule expression. Circulating iron oxidizes LDL-C thus enhances LDL retention in sub-endothelium with macrophage progression to form foam cells leading to sudden AMI[9]. Several causes result in the development of MI in the adult population, which include age, sex, smoking, lack of exercise, hyperlipidemia, diabetes mellitus (DM), obesity, high blood pressure (hypertension), alcohol, high levels of homocysteine, and serum folate[10]. In the majority of cases, MI was found to have a direct link with hyperlipidemia[11] . The most predominant form of hyperlipidemia was found to have increased LDL cholesterol and low HDL cholesterol levels[12] . Recent studies have linked high ferritin concentration with decline in ST-evaluation with accentuated left ventricular ejection fraction (LVEF) decline among MI patients on treatment with percutaneous coronary intervention. Thus, evaluation of ferritin would be a simple investigation procedure to identify high risk patients with AMI during hospital stay[13]. But to the best of our knowledge, no studies have observed that a rise in serum iron, ferritin and lipid profile parameters would increase the risk of STEMI or NSTEMI in the Indian population. Hence, the aim of the study is to associate the levels of iron and ferritin with STEMI and NSTEMI patients and the objective of the study is to investigate and associate the iron, ferritin and lipid profile parameters among STEMI and NSTEMI patients.

2. MATERIALS AND METHODS

This cross-sectional study was conducted from November 2020 to April 2021 recruiting a total of 150 participants between the age group of 25-55 years, the participants were divided into three groups. The STEMI and NSTEMI group included 100 subjects (both the groups had 50 participants each) admitted in CICU (Cardiac Intensive Care Unit), SRM Medical College Hospital & Research Centre, Kattankulathur, Tamil Nadu, India and diagnosed based on ECG findings and cardiologist report. The 50 age and sex matched apparently healthy participants attended the MHC (Master Health Checkup) of SRM Medical College Hospital & Research Centre, Kattankulathur, Tamil Nadu, India were taken in control group. The study protocol was approved by the Institutional ethics committee **(**ECN: 1872(A)/IEC/2019**).** Clinically diagnosed patients with MI of both sex between the age group between 25-55 years were included in the study group. Patients with the clinical condition that affects the serum levels of iron and ferritin such as kidney and liver disease, post-surgery patients, malignancy, malabsorption disease, alcoholics, smokers, pregnancy, inflammatory disease and patients with bleeding disorders were excluded from that study. After getting informed and written consent from the participants, a detailed history and relevant anthropometric measurements like height and weight was recorded for the calculation of BMI. After overnight fasting of 10-12 hours, under aseptic precautions 3 ml of fasting blood sample was collected using the plain vacutainer tubes. After centrifugation at 2500 rpm for 3 minutes, the serum sample was separated and analysed for the following biochemical parameters viz cardiac marker (CK-MB) by UV Kinetic G6P, Total Cholesterol by CHOD-POD, Triglycerides by enzymatic GPO-PGO, HDL-C and LDL-C by Direct Antibody Inhibition and serum Iron by TPTZ Binging method in Beckman Coulter Auto analyser. Serum Ferritin was estimated by Enhanced CLIA VITROS Eci Immunoanalyser Ortho Clinical Diagnostics. VLDL was calculated using the formula triglyceride/ 5, the NON-HDL-C was calculated by total cholesterol – HDL-C in mg/dl and cholesterol ratio was calculated by total cholesterol / HDL-C in mg/dl.

3. STATISTICAL ANALYSIS

The Statistical Package for the Social Sciences (SPSS 22.0) was used for data analysis. Continuous variables were presented as Mean ± SD. Where, categorical variables were expressed as frequencies and percentages. The post hoc test (one-way ANOVA) was used to compare the variance between control, STEMI and NSTEMI groups. Pearson's correlation was used to correlate the analysed biochemical parameters with iron and ferritin among STEMI and NSTEMI participants. To calculate crude odds ratio bivariate logistic regression analysis was used at 95% confidence interval. For all statistical tests, a p value < .05 was taken to indicate a significant difference.

4. RESULTS

Total of 150 individuals were recruited in the study. The participants were divided into 3 groups as STEMI, NSTEMI and controls. The mean age of STEMI and NSTEMI were 43.8 and 43.9 respectively (range 25-55 years). It is found that males are more prone to develop myocardial infarction compared to females, as the number of male individuals with STEMI (80%) and NSTEMI (72%) were higher compared to females. The mean BMI of STEMI and NSTEMI were 23.1 ± 3.5 and 23.2 ± 2.7 respectively with no statistical difference. The participants

were further classified based on their BMI as normal (18-22.9) and overweight (23-24.9) and obese (≥25). It was found that among STEMI individuals, 52% were normal, 16% were overweight and 32% were obese, among NSTEMI individuals 40% were normal, 36% were overweight and 24% were obese. It is seen that individuals with increase in BMI are more likely to develop myocardial infarction. (Table 1)

Table 1. Demographic and anthropometric measurement of STEMI, NSTEMI and Control groups.

Demographic factor	STEMI	NSTEMI	CONTROLS	p value
Age (mean ± SD)	43.8 ± 8.7	43.9 ± 6.8	43.4 ± 9.7	0.9
25 - 40years	12 (24%)	16 (32%)	14 (28%)	
40 - 55 years	38 (76%)	34 (68%)	36 (72%)	
Sex				
Males	40 (80%)	36 (72%)	36 (72%)	
Females	10 (20%)	14 (28%)	14 (28%)	
Anthropometric measurement				
BMI (mean ± SD)	23.1 ± 3.5	23.2 ± 2.7	22.6 ± 2.9	0.7
Normal	26 (52%)	20 (40%)	28 (56%)	
Overweight	8 (16%)	18 (36%)	10 (20 %)	
Obese	16 (32%)	12 (24%)	12 (24 %)	

Statistically significant with p value <0.05*

Comparison of mean levels of biochemical parameters between groups were done. It is found that CK-MB, iron and ferritin were significantly higher in STEMI group compared to NSTEMI group and controls. On the other hand, total cholesterol, LDL-C, non-HDL-C and cholesterol ratio were significantly elevated among NSTEMI individuals compared to STEMI and controls. The HDL-C was significantly lowered in STEMI and NSTEMI individuals compared to controls. Hence, increase in lipid profile levels result in increased risk of developing NSTEMI. (Table. 2)

Table 2. Comparison of Measured Biochemical Parameters between STEMI, NSTEMI and Control groups using one way ANOVA.

Parameters	Stemi	Nstemi	Control	P Value
CK- MB	333.4± 82	166.2 ± 68.7	12.4±4.4	0.0001***
Iron	73 ±17	65.4 ±13.9	48.6 ±10.2	0.0001***
Ferritin	308.4 ±65.8	188.8 ±46.6	28.3 ±38.9	0.0001***
Total cholesterol	190.4 ±29	214.2 ± 24.2	177.8 ± 29.07	0.0001***
Triglyceride	106.8 ±38.1	128.7 ±27.6	111.16 ±35.3	.06
HDL- C	31.9 ±6.6	41.7 ±11	42.7 ±11.5	.001**
LDL-C	139.6 ±17.3	146.9 ±18.3	112.3 ±23.9	.0001***
VLDL-C	21.2 ±7.6	25.5 ±5.5	22.7 ±7.8	0.09
NON-HDL-C	158.5 ± 27.4	172.4 ± 23	135 ±28	0.0001***
Cholesterol ratio	6.1 ±1.1	5.4 ± 1.5	4.4 ±1.3	0.0001***

Values are expressed in mean ± standard deviation. The values are statistically significant based on the p value.* p value < .05, ** p value < .01, *** p value <.001, NS-Not Significant.

The Pearson's correlation coefficient was carried out in iron and ferritin with other biochemical parameters among STEMI and NSTEMI groups. It is found that increase in serum iron is positively associated with increase in cholesterol ratio, and increase in serum ferritin is significantly associated with decreased HDL-C levels among STEMI groups. Among NSTEMI individuals, it is found that increased serum ferritin is significantly positively associated with raised levels of CK-MB, total cholesterol, triglyceride, LDL-C, VLDL-C, Non-HDL-C and cholesterol ratio. (Table 3)

Table 3. Correlation of Iron and Ferritin with Biochemical Parameters among STEMI and NSTEMI groups.

Biochemical Parameters	STEMI group				NSTEMI group			
	Iron		Ferritin		Iron		Ferritin	
	r	p	r	p	r	p	r	p
CK- MB	-0.18	0.30	0.07	0.7	0.1	0.3	**0.8**	**0.0001*** **
Total cholesterol	0.28	0.16	-0.2	0.18	0.002	0.9	**0.6**	**0.0001*** **
Triglyceride	0.13	0.52	-0.1	0.6	0.03	0.8	**0.6**	**0.0001*** **
HDL- C	0.2	0.15	**-0.5**	**0.01****	-0.3	0.09	0.09	0.9
LDL-C	-0.1	0.6	-0.07	0.7	0.2	0.3	**0.6**	**0.0001*** **
VLDL-C	0.3	0.12	-0.1	0.6	0.05	0.7	**0.6**	**0.0001*** **
Non-HDL-C	0.3	0.06	-0.1	0.4	0.1	0.4	**0.6**	**0.0001*** **
Cholesterol ratio	**0.4**	**0.04***	0.2	0.25	0.3	0.14	**0.4**	**0.03***

. r is the correlation coefficient value. The values are statistically significant based on the p value. *p value < .05, ** p value < .01, *** p value <.001, NS-Not Significant.

It is observed that the individuals with the serum iron levels ≥ 51.5 µg/dl and ferritin levels >137ng/dl are at 26 times and 132.2 times respectively are at high risk of developing STEMI. (Table 4) The individuals with the serum iron levels ≥ 51.5 µg/dl and ferritin levels >137ng/dl are at 12.4 times and 132.2 times respectively are at high risk of developing NSTEMI. (Table 5) Hence independent to the type of MI, increased levels of serum iron and ferritin are at higher risk of developing myocardial infarction.

Table 4. Bivariate logistic regression for risk factors with STEMI.

Factors		STEMI	CONTROLS	Odds ratio	p value	95% CL
Age	<40	12	14	1.23	0.7	0.3-4.3
	≥40	38	36			
Sex	Males	40	36	1.5	0.5	0.4-5.7
	Females	10	14			
BMI	18.5-24.9 (normal)	26	28	1.1	0.7	0.3-3.5
	>25 (overweight)	24	22			
IRON	< 51.5 µg/dl	2	26	26	0.003**	3-222.9
	≥ 51.5 µg/dl	48	24			
FERRITIN	≤ 137ng/dl	4	46	132.2	0.0001***	17.1-1020
	>137ng/dl	46	4			

Statistically significant with p value <0.05*

Table 5. Bivariate logistic regression for risk factors with NSTEMI.

Factors		NSTEMI	CONTROLS	Odds ratio	p value	95% CL
Age	<40	16	14	0.8	0.7	0.2-2.7
	≥40	34	36			
Sex	Males	36	36	1	1	0.2-3.4
	Females	14	14			
BMI	18.5-24.9 (normal)	20	28	1.9	0.2	0.6-5.8
	>25 (overweight)	30	22			
IRON	< 51.5 µg/dl	4	26	12.4	0.002	2.4-64.4
	≥ 51.5 µg/dl	46	24			
FERRITIN	≤ 137ng/dl	4	46	132.2	0.0001***	17.1-1020
	>137ng/dl	46	4			

Statistically significant with p value <0.05*

5. DISCUSSION

Recent studies have shown that increase in body iron stores is associated with risk of acute myocardial infarction[3]. Myocardial Infarction (MI) is evidence of sustained ischemia that results in myocardial cell necrosis based on the World Health Organization (WHO) criteria. Based on the ECG findings MI is classified into 3 distinct phases[14]. Unstable Angina (UA)-Patients present with pain in chest with ECG finding shows no ST-elevation, but normal cardiac biomarkers. Non-ST elevation MI (NSTEMI) - Patients present with chest pain, with raise in the biomarkers but no ST-elevation in ECG. ST-elevation MI (STEMI) - Patients presenting with pain in chest with ECG findings showing ST-elevation[15] . This study consists of three groups namely STEMI, NSTEMI and control groups. The STEMI and NSTEMI were diagnosed based on ECG findings. The mean age group of the participants were 43.8 ± 8.7, 43.9 ± 6.8 and 43.4 ± 9.7 in STEMI, NSTEMI and control groups. They were further sub classified based on the age group from 25-40 and 40-55 years. It is found that most of the STEMI and NSTEMI lie between 40-55 years, it is observed that increase in age is associated with risk of

developing myocardial infarction. The study by Mudiyappa Herakall et al, found the mean age was 48.3 ± 14.24 cases and 47.6 ± 8.15 among the case and control group[16] . In this study, it is observed that male patients are more prone to develop myocardial infarction than female, which is in controversy to the study by Maas, et al which shows that females are at higher risk of developing myocardial infarction[17] . It is found that ,most of the patients at the higher BMI are found to develop MI, where a study by Holay et al support this finding that the difference in BMI of patients in the controls and cases group is observed[18] . In this study it is observed that the mean CK-MB, iron and levels of ferritin were found to be significantly elevated in STEMI group when compared with NSTEMI group. A cross-sectional study by Morad Rostami et al showed a significant rise in mean iron and ferritin levels in patients with AMI compared with a control group[19]. The study by Vijaya et al concluded that, raised in the ferritin levels seem to be a strong AMI risk factor and it has been hypothesized that, higher intake or increase in iron stores may promote atherogenesis[20] . The study by Chanchal et al, also suggested raise in the levels of ferritin in serum is associated with development of AMI among the male population in Manipuri, and by measuring their levels in serum can used as a complementary tool for diagnosing and confirming the diagnosis of AMI[21] . A cross-sectional study by Muhammed T.et al showed a significant rise in mean iron and ferritin levels in patients with AMI compared with a control group[22]. Moacir Fernandes de Godoy et al undergone a study with 115 patients with coronary arteriography and concomitant evaluation of serum ferritin , and verified that, women with serum ferritin levels > 80 ng/mL presented with more severe obstructive chronic heart diseases (CHD) than women with lower levels. In men, the serum ferritin level was not a predictor element of the degree of obstruction[23]. However, a study by Birger Wolff et al observed an independent relationship between serum ferritin levels and carotid atherosclerosis among men. This relationship appeared to be strengthened by a synergistic association between ferritin and LDL cholesterol and support to the hypothesis that iron is linked to cardiovascular disease[24]. Whereas, controversy to the previous findings, a study by Wilma Delphine Silvia et al, showed that, increased risk of developing AMI is highly associated with elevated ferritin levels among males[25].

In this study, it is observed that the lipid profile levels were significantly elevated and HDL-C was decreased in NSTEMI groups. A study by Jitender Sharma et al showed that total cholesterol, LDL-C, and triglycerides levels were found to be raised in the sera of AMI patients than the controls. On the contrary, HDL-C levels were significantly reduced in serum levels of the AMI patients as compared to the serum levels of the control subjects[26]. It is observed that increase in serum iron is positively correlated with raised levels of cholesterol ratio, and increase in serum ferritin is significantly correlated with decreased HDL-C levels among STEMI group. Among NSTEMI individuals, it is found that increased serum ferritin is significantly and positively associated with increased levels of CK-MB, total cholesterol, triglyceride, LDL-C, VLDL-C, Non-HDL-C and cholesterol ratio. A study by Silvia WD et al showed that serum ferritin was directly linked with serum cholesterol ($r=0.439$, $p<0.01$) and serum LDL-C ($r=0.381$, $p < 0.01$). Serum ferritin was inversely associated with serum HDL-C ($r=-0.210$, $p < 0.05$)[27] . It is observed that the individuals with the serum iron levels ≥ 51.5 µg/dl and ferritin levels >137ng/dl are at 26, 12.4 times and 132.2 times respectively are at high risk of developing STEMI and NSTEMI. Hence independent to the type of MI, increased levels of serum iron and ferritin are at higher risk of developing myocardial infarction. Iron reduction by iron-chelating therapy or restrictions on the intake of iron-rich foods could reduce atherosclerotic lesion size and increase plaque stability[28].

6. CONCLUSION

The mean levels of iron and ferritin are significantly elevated among STEMI compared to NSTEMI individuals. It is seen that increase in serum iron and ferritin in individuals are more likely to develop STEMI. But it is observed that, the lipid profile levels are significantly elevated and associated with ferritin among the NSTEMI group compared to STEMI group. Independent to the type of MI, individuals with increased levels of serum iron and ferritin are at higher risk of developing myocardial infarction. Hence, regular monitoring of iron and ferritin levels can be done to avoid developing myocardial infarction.

7. AUTHOR CONTRIBUTION STATEMENT

All authors contributed to this article conception and design. The first draft was written by Dr.M. Vasanthan. Preparation, data collection , statistical analysis and final draft was done by S.Aishwarya. The final draft of manuscript was reviewed by Dr.Renuka.P, Dr.V.M.Vinodhini and Dr.V.E. Dhandapani. All the authors reviewed and approved the final version of the manuscript.

8. CONFLICT OF INTEREST

Conflict of interest declared none.

9. REFERENCES

1. Basuli D, Stevens RG, Torti FM, Torti SV. Epidemiological associations between iron and cardiovascular disease and diabetes. Front Pharmacol. 2014;5:117. doi: 10.3389/fphar.2014.00117, PMID 24904420.
2. Ramakrishna G, Rooke TW, Cooper LT. Iron and peripheral arterial disease: revisiting the iron hypothesis in a different light. Vasc Med. 2003;8(3):203-10. doi: 10.1191/1358863x03vm493ra, PMID 14989563.
3. Moradi M, Fariba F, Mohasseli AS. Relation between the serum ferritin level and the risk for acute myocardial infarction. J Res Health Sci. 2015;15(3):147-51. PMID 26411659.
4. Knovich MA, Storey JA, Coffman LG, Torti SV, Torti FM. Ferritin for the clinician. Blood Rev. 2009 May;23(3):95-104. doi: 10.1016/j.blre.2008.08.001, PMID 18835072.
5. Silvestre OM, Gonçalves A, Nadruz W, Claggett B, Couper D, Eckfeldt JH et al., Wilson Nadruz Junior, et al. Ferritin levels and risk of heart failure-the Atherosclerosis Risk in Communities Study. Eur J Heart Fail. 2017 Mar;19(3):340-7. doi: 10.1002/ejhf.701, PMID 27976478.

6. Kehrer JP. The Haber–Weiss reaction and mechanisms of toxicity. Toxicology. 2000 Aug 14;149(1):43-50. doi: 10.1016/s0300-483x(00)00231-6, PMID 10963860.
7. Kathryn Moore, Frederick Sheedy, and Edward Fisher. Macrophages in atherosclerosis: a dynamic balance. Nat Rev Immunol. 2013 Oct; 13(10): 709–721. doi: 10.1038/nri3520. PMCID: PMC4357520
8. Vinchi F, Muckenthaler MU, Da Silva MC, Balla G, Balla J, Jeney V. Atherogenesis and iron: from epidemiology to cellular level. Front Pharmacol. May 05 2014;5:94. doi: 10.3389/fphar.2014.00094, PMID 24847266.
9. Kehrer JP. The Haber–Weiss reaction and mechanisms of toxicity. Toxicology. 2000 Aug 14;149(1):43-50. doi: 10.1016/s0300-483x(00)00231-6, PMID 10963860.
10. Ascherio A, Hunter DJ. Iron and myocardial infarction. Epidemiology. 1994;5(2):135-7. PMID 8172987.
11. Kumar N, Kumar S, Kumar A, Shakoor T, Rizwan A. Lipid profile of patients with acute myocardial infarction (AMI). Cureus. 2019 Mar 18;11(3):e4265. doi: 10.7759/cureus.4265, PMID 31139524.
12. Lusis AJ. Atherosclerosis. Nature. 2000 Sep 14;407(6801):233-41.
13. Brinza C, Floria M, Popa IV, Burlacu A. The prognostic performance of ferritin in patients with acute myocardial infarction: A systematic review. Diagnostics (Basel). 2022 Feb;12(2):476. doi: 10.3390/diagnostics12020476, PMID 35204567.
14. Jacobson C. ECG diagnosis of acute coronary syndrome. AACN Adv Crit Care. 2008;19(1):101-8. doi: 10.1097/01.AACN.0000310757.13480.b8, PMID 18418110.
15. Mechanic OJ, Grossman SA. Acute myocardial infarction. StatPearls. Vol. 27; 2019:ID: NBK459269.
16. Herakall M, Biradar MS. A study of serum ferritin in acute myocardial infarction. Ann Int Med Dent Res. Jun 2018, Vol (4), Issue (5).
17. Maas AHEM, Appelman YEA. Gender differences in coronary heart disease. Neth Heart J. 2010 Dec;18(12):598-602. doi: 10.1007/s12471-010-0841-y, PMID 21301622.
18. Holay MP, Choudhary AA, Suryawanshi SD. Serum ferritin-a novel risk factor in acute myocardial infarction. Indian Heart J. 2012;64(2):173-7. doi: 10.1016/S0019-4832(12)60056-X, PMID 22572495.
19. Rostami M, Aberomand M, Khirollah A, Jorfi M. Evaluation of serum iron and ferritin levels in myocardial infarction patients. j bioinform intelli control. 2013;2(1):79-82. doi: 10.1166/jbic.2013.1029.
20. Vijaya Bhaskar M, Srilekha S, Saranya M, Balu Mahendran K, Madhulatha M. Assessment of ferritin and its association with C – reactive protein and malondialdehyde in acute myocardial infarction. Int J Res Med Sci. 2015 Dec;3(12):3581-5.
21. Chanchal L, Shaini L, Sachin Deba Th, Sangeeta N, Arpita Das VL, Lalrindiki C et al. Serum ferritin level in male with established coronary artery disease. IOSR JDMS. Dec 2014, e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 13, Issue 12 Ver. I:58-62.
22. Muhammed T.Gürgöze, IsabellaKardys, et al. Relation of Iron Status to Prognosis After Acute Coronary Syndrome. The American Journal of Cardiology. 2022, vol. 168: 22-30.
23. Fernandes de Godoy M, Takakura IT, Machado RD, Grassi LV, Nogueira PR. Serum ferritin and obstructive coronary artery disease: angiographic correlation. Arq Bras Cardiol. Apr 2007;88(4).
24. Wolff B, Völzke H, Lüdemann J, Robinson D, Vogelgesang D, Staudt A et al. Association between high serum ferritin levels and carotid atherosclerosis in the study of health in pomerania (SHIP). Stroke. Feb 1 2004;35(2):453-7. doi: 10.1161/01.STR.0000114875.31599.1C, PMID 14726541.
25. Silvia CR, Biswas S, Uthappa S, Shetty P. Wilma Delphine. Ferritin Potent Threat Acute Myocardial Infarction?. JAPI. 2003;51.
26. Sharma J, Mahajan B, Rajput R, Gupta VK. Analysis of iron and lipid profiles in Indian male acute myocardial infarction patients. Scholars Acad J Biosci. 2017;5(5):359-62.
27. Silvia WD, Biswas S, Uthappa S, Shetty P. Ferritin, a potent Threatfor acute myocardial infarction? J Assoc Physicians India. 2003;51:947-50. PMID 14719581.
28. Sharkey-Toppen TP, Tewari AK, Raman SV. Iron and atherosclerosis: nailing down a novel target with magnetic resonance. J Cardiovasc Transl Res. 2014 Jul;7(5):533-42. doi: 10.1007/s12265-014-9551-y, PMID 24590608.