Original Article ISSN (O):2395-2822; ISSN (P):2395-2814

A Study of Serum Ferritin in Acute Myocardial Infarction: A Case Control Study in BLDE (Deemed To Be University), Shri BM Patil Medical College, Hospital and Research Centre Vijayapura.

Mudiyappa Herakall[1], MS Biradar[2]

[1]Junior Resident, Department of Medicine, BLDE (Deemed to be University), Sri BM Patil Medical College, Hospital and Research Centre, Vijayapur-586103, Karnataka, India.

[2]Professor, Department of Medicine, BLDE (Deemed to be University), Sri B M Patil Medical College, Hospital and Research Centre, Vijayapur-586103, Karnataka, India.

Received: June 2018
Accepted: June 2018

ABSTRACT

Background: Cardiovascular disease is the leading cause of death worldwide. India has reportedly shown the highest burden of Acute coronary syndromes in the world2. The association of high iron stores and coronary heart disease was first suggested by Sullivan.10 Results of some studies have been in favor of ferritin being a risk factor for acute MI (AMI).11 Serum ferritin was observed to be one of the strongest indicators of the presence and progression of carotid artery disease.28,27 Hence the present study was done at our tertiary care centre to study the levels of serum ferritin in acute myocardial infarction and compare the relationship of serum ferritin with conventional risk factors of acute myocardial infarction like diabetes mellitus, body mass index , hypertension and smoking. Aims: To study the levels of serum ferritin in acute myocardial infarction and compare relationship of serum ferritin with conventional risk factors of acute myocardial infarction like diabetes mellitus, body mass index, hypertension and smoking. **Methods:** Study design: A total of 200 patients (100 cases and equal number of controls) were enrolled in a case control study to assess the levels of serum ferritin in acute myocardial infarction. Study Site: BLDEU'S Shri B. M. Patil Medical college Hospital and Research Centre, Vijayapur. Study population: Patients admitted to BLDE(deemed to be university) Shri B. M. Patil Medical college Hospital and Research Centre, Vijayapur with acute myocardial infarction. Study duration: The study was carried out for a period of October 2015 to January 2018. **Result:** It was observed that significantly higher number of patients in Cases Group had serum ferritin level >300µg/l as compared to Control Group (55% vs. 9%). The mean serum ferritin levels were significantly higher in Cases Group as compared to Control Group (332.5 vs. 153.8 µg/l) ($p<0.05$). The mean serum ferritin levels of males and females patients in Cases Group were significantly higher as compared to Control Group (320.3 vs. 160.1µg/l and 327.7 vs. 137.5 µg/l respectively) as per Student t-test ($p<0.05$). There was no significant difference of mean serum ferritin levels of males and females patients within the group ($p>0.05$). It was observed that significantly more patients in Cases Group (69%) than Control Group (34%) had concentrations above the cut-off of 200 µg/L ($p<0.05$). In multivariate analysis, Diabetes Mellitus (P = 0.001, OR = 7.64, 95% CI 2.37–24.58), HDL (P < 0.001, OR = 0.86; 95% CI 0.79–0.93) and serum ferritin (>200 µg/L) (P < 0.001, OR = 5.72, 95% CI 2.16–15.17), are found to be independently associated with AMI. **Conclusion:** Higher levels of ferritin, seems to be a strong risk factor for AMI. Patients with higher ferritin level can easily be identified during routine haematological analysis along with other risk factor estimation. Regular monitoring of serum ferritin levels may help in reduction of cardiovascular morbidity and mortality.

Keywords: Acute myocardial infarction; Serum ferritin; conventional risk factors of acute myocardial infarction.

INTRODUCTION

Cardiovascular disease is the leading cause of death worldwide. The incidence and prevalence of Coronary artery disease (CAD) has increased tremendously in India during the last two decades and this change is largely attributable to lifestyle changes.[1] India has reportedly shown the highest burden of Acute coronary syndromes in the world.[2]

There are two facets of CAD: Stable CAD and Unstable CAD which includes patients with acute coronary syndrome (Unstable angina, Non-ST elevation myocardial infarction, ST elevation myocardial infarction).[1]

Diagnosis of acute myocardial infarction is based on history of acute chest pain in conjunction with ECG criteria and laboratory findings. ECG is still the most readily available and fastest method for the diagnosis of AMI.[3]

Name & Address of Corresponding Author
Dr. Mudiyappa Herakall,
Room No: B-23, NRI hostel,
Smt. Bangaramma Sajjan Campus,
Dr B M Patil Road (Solapur Road),
Vijayapur-586103, Karnataka, India.

The Electrocardiogram remains a crucial tool in the identification and management of Acute myocardial infarction. A detailed analysis of patterns of ST-segment elevation may influence decisions regarding the use of reperfusion therapy.4The electrocardiogram is also crucial for identifying new conduction abnormalities and arrhythmias that influence both short- and long-term outcome.[4]

Myocardial infarction (MI) is the most common cause of death worldwide. The major risk factors for MI are family history, diabetes mellitus, smoking, hypertension, and lipids.[5] Excess serum ferritin as a risk factor for MI is a relatively newer concept.[6] Ferritin is a large protein shell having molecular weight 450 KDa comprised 24 subunits, covering an iron core containing up to 4000 atoms of iron. Ferritin acts as the soluble storage form of iron in tissue.[7] High serum ferritin may increase the risk of MI in the presence of other risk factors that increase the formation of free radicals, thus accelerating atherogenesis through stimulation of low-density lipoprotein (LDL) oxidation.[8]

A possible association between body iron status and the risk of coronary heart disease was bolstered from a 3-year Finnish study relating increased levels of both serum levels of ferritin and dietary iron to an increased risk of MI.[9] The association of high iron stores and coronary heart disease was first suggested by Sullivan.[10] Results of some studies have been in favor of ferritin being a risk factor for acute MI (AMI).[11]

A harmful biological effect of excessive iron loading in the human body has been recently suggested. In this regard, iron overloading especially in myocardial tissue has been proposed to be a potent risk factor for ischemic heart disease and occurring AMI.[9,10,12,13] The cardiac iron deposition results in a decrease of heart function on a certain genetic background.[14] Iron can also directly injure the myocardium. Iron can be accumulated in cells as hemosiderin, ferritin, and free iron named labile cellular iron that is the most toxic form stimulating the formation of free radicals.[15,16]

Since serum ferritin concentrations are directly proportional to intracellular ferritin concentration, it is considered the best clinical measure of body iron stores.[17] Recently, some evidences have been provided linking the increased incidence of coronary artery disease and elevated level of stored iron concentration.[9]

National Health and Nutrition Examination Survey (NHANES III), first time reported a significant positive association in iron storage and heart disease risk between 1988-1994. Several researchers, thereafter, have found and reported an association between iron overload, serum ferritin (SF) and acute myocardial infarction (MI).[9,10,25-26]

Free iron—a catalyst of the production of free radicals—has been implicated in ischemic myocardial damage and lipid peroxidation. Hypotheses as to how free iron may accelerate the progression of atherosclerosis or contribute to myocardial injury after an ischemic event have been generated from basic research. Direct evidence that high stored iron concentrations or high iron intakes increase the incidence of ischemic heart disease in humans, however, is limited. The strongest supporting evidence stems from a cohort study of eastern Finnish men, in whom high concentrations of serum ferritin and dietary iron were positively associated with the incidence of myocardial infarction.[9] Furthermore, serum ferritin was observed to be one of the strongest indicators of the presence and progression of carotid artery disease.[27,28] Blood donation, which depletes iron stores in the donors, was associated with reduced risk of myocardial infarction18 and cardiovascular disease.[29] However, most subsequent studies investigating whether iron status or dietary iron intake are associated with increased risk of myocardial infarction or ischemic heart disease have not provided consistent results.[19-22,24,30-31]

Hence the present study was done at our tertiary care centre to study the levels of serum ferritin in acute myocardial infarction and Compare the relationship of serum ferritin with conventional risk factors of acute myocardial infarction like diabetes mellitus, body mass index, hypertension and smoking.

MATERIALS AND METHODS

Study design: A total of 200 patients (100 cases and equal number of controls) were enrolled in a case control study to assess the levels of serum ferritin in acute myocardial infarction.

Study Site: BLDEU'S Shri B. M. Patil Medical college Hospital and Research Centre, Vijayapur.

Study population: Patients admitted to BLDEU'S Shri B. M. Patil Medical college Hospital and Research Centre, Vijayapur with acute myocardial infarction.

Study duration: The study was carried out for a period of October 2015 to January 2018.

Inclusion Criteria

The diagnosis of AMI was based on European society of cardiologists/American college of cardiology Definition of AMI.

1. Ischaemic symptoms (sudden onset of chest pain lasting 30 min; often associated with shortness of breath, weakness, nausea, vomitting).
2. Electrocardiogram changes indicative of ischemia (ST segment elevation or depression).
3. Increased cardiac bio-markers (CKMB, Trop T and or Trop I),
4. Presumably new onset bundle-branch block.
5. Age group > 18 years

Exclusion Criteria

1. Patients with high ferritin levels like haemochromatosis, liver disease, tuberculosis,

chronic inflammatory diseases, those on iron therapy.

2. Past history of AMI or CHD
3. Age group < 18 years

All subjects were subjected to detailed history, physical examination and relevant investigations. Cases and controls were investigated for conventional risk factors (BMI, blood sugar, lipid profile). Study subjects were evaluated for serum creatine kinase-MB fraction (CK-MB), Trop-T, serum ferritin along with complete blood counts, renal function , liver function etc.

All patients were interviewed as per the prepared proforma and then complete clinical examination and laboratory investigations was done.

Statistical Analysis

Statistical testing was conducted with the statistical package for the social science system version SPSS 20.0. Continuous variables were presented as Mean ± SD or median (IQR) for non-normally distributed data. Categorical variables were expressed as frequencies and percentages.

The comparison of normally distributed continuous variables between the groups was performed using Student's t test else Mann Whitney U test was used for Non-normal distribution data. Nominal categorical data between the groups were compared using Student t-test, Chi-squared test, Fisher's exact test or as appropriate. Multivariate analysis was done in the end.For all statistical tests, a p value less than 0.05 were taken to indicate a significant difference. Results were graphically represented where deemed necessary. Graphical representation was done in MS Excel 2010.

RESULTS

Table 1: Distribution of patients according to Age

Age (years)	Cases		Control		p Value
	N	%	N	%	
21-30	10	10%	2	2%	>0.05
31-40	12	12%	16	16%	
41-50	42	42%	52	52%	
51-60	14	14%	22	22%	
>60	22	14%	8	8%	
Total	100	100%	100	100%	
Mean ± SD	48.3 ± 14.24		47.6 ± 8.15		

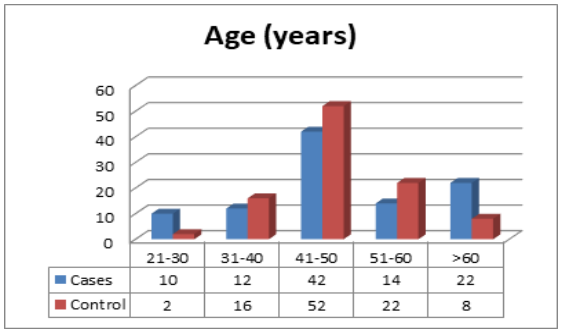

Figure 1: Distribution of patients according to Age

A total of 200 patients (100 cases and equal number of controls) were enrolled in a case control study to assess the levels of serum ferritin in acute myocardial infarction.

Table 2: Distribution of patients according to Sex

Sex	Cases		Control		p Value
	N	%	N	%	
Male	70	70%	72	72%	>0.05
Female	30	30%	28	28%	
Total	100	100%	100	100%	

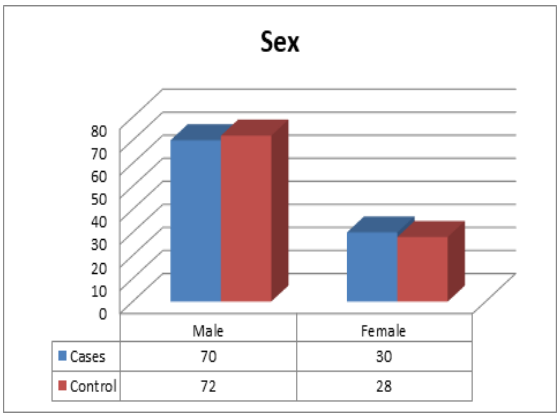

Figure 2: Distribution of patients according to Sex

Table 3. Distribution of patients according to BMI

BMI (kg/m2)	Cases		Control		p Value
	N	%	N	%	
Normal	34	34%	63	63%	<0.05
Overweight	38	38%	20	20%	
Obese	28	28%	17	17%	
Total	100	100%	100	100%	
Mean ± SD	27.1 ± 3.77		24.9 ± 3.73		

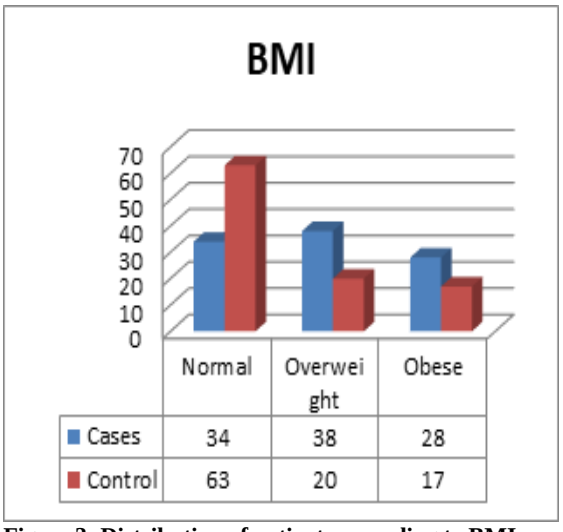

Figure 3: Distribution of patients according to BMI

Table 4: Distribution of patients according to Comorbidities

Comorbidities	Cases		Controls		p Value
	N	%	N	%	
Hypertension	32	32%	28	28%	>0.05
Diabetes Mellitus	27	27%	23	23%	>0.05

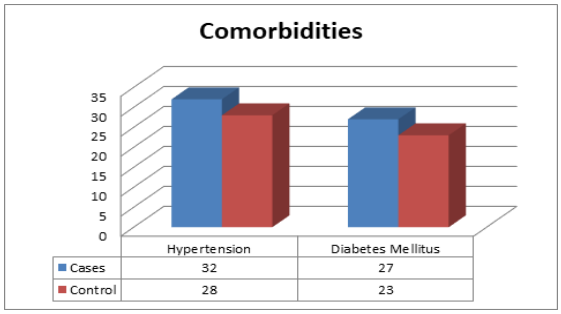

Figure 4: Distribution of patients according to Comorbidities

Table 5: Distribution of patients according to Habits

Habits	Cases		Controls		p Value
	N	%	N	%	
Smoking	38	38%	31	31%	>0.05
Alcohol	25	25%	28	28%	>0.05

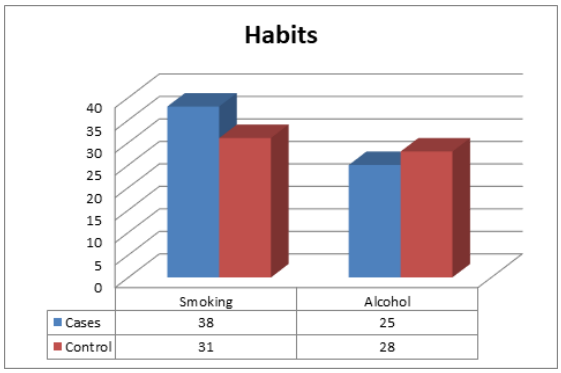

Figure 5: Distribution of patients according to Habits

Table 6: Baseline Parameters of patients

Parameters	Cases		Controls		p Value
	Mean	SD	Mean	SD	
Haemoglobin (g/dl)	13.79	3.35	13.48	3.33	>0.05
ESR (mm/hr)	23.9	12.15	21.3	11.22	>0.05
SBP (mmHg)	123.5	15.17	125.9	18.13	>0.05
DBP (mmHg)	75.6	10.79	77.4	10.96	>0.05

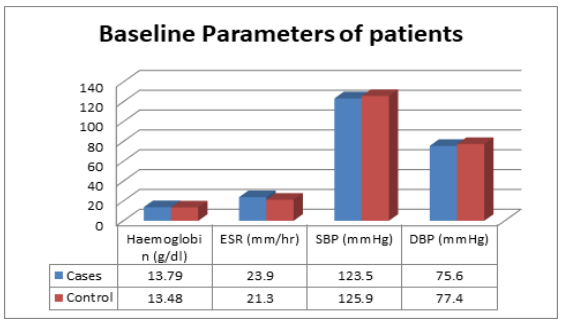

Figure 6: Baseline Parameters of patients

Table 7: Lipid Profile Parameters of patients

Parameters	Cases		Controls		p Value
	Mean	SD	Mean	SD	
Total Cholesterol (mg/dl)	242.8	32.63	189.4	13.01	<0.05
Triglycerides (mg/dl)	189.7	12.46	142.9	24.33	<0.05
LDL(mg/dl)	169.4	16.98	119.2	30.71	<0.05
HDL (mg/dl)	33.6	4.72	44.6	5.96	<0.05

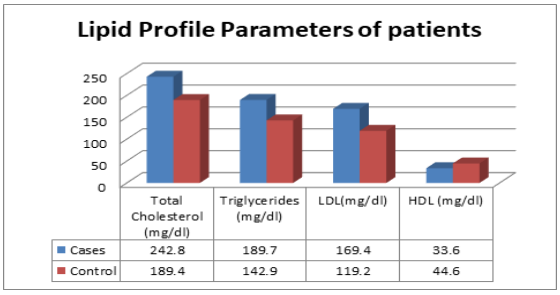

Figure 7: Lipid Profile Parameters of patients

Table 8: Distribution of patients according to Serum Ferritin levels

S. Ferritin (µg/l)	Cases		Controls		p Value
	N	%	N	%	
<100	10	10%	35	35%	<0.05
100-199	21	21%	31	31%	
200-299	14	14%	25	25%	
>300	55	55%	9	9%	
Mean ± SD	332.5	165.5	153.8	90.5	

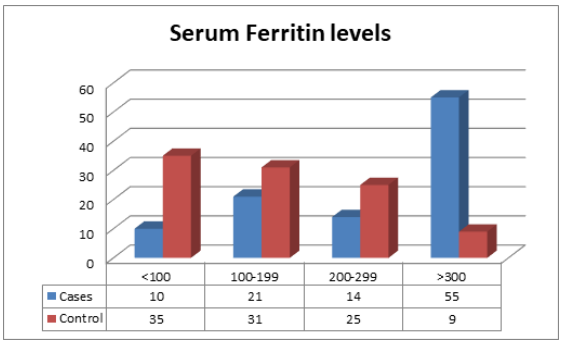

Figure 8: Distribution of patients according to Serum Ferritin levels

Table 9: Association of Serum Ferritin with Sex

Serum Ferritin (µg/l)	Cases		Controls		p Value
	Mean	SD	Mean	SD	
Male	320.3	165.54	160.1	94.76	<0.05
Female	327.7	168.15	137.5	77.75	<0.05
p Value	>0.05		>0.05		

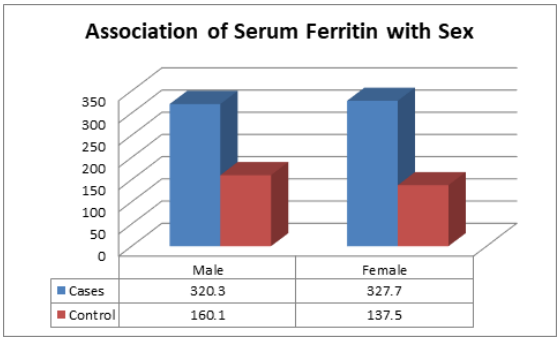

Figure 9: Association of Serum Ferritin with Sex

Table 10: Association of Acute Myocardial Infarction (AMI) with high serum ferritin

Serum Ferritin (µg/l)	Cases		Controls		p Value
	N	%	N	%	
<200µg/l	31	31%	66	66%	<0.05
≥200µg/l	69	69%	34	34%	

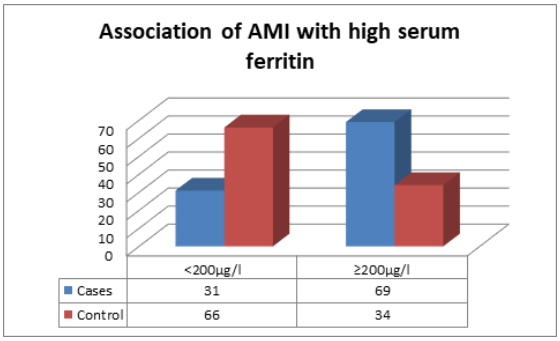

Figure 10: Association of Acute Myocardial Infarction (AMI) with high serum ferritin

Table 11: Multivariate analysis for association of various risk factors with Acute Myocardial Infarction (AMI)

Parameters	OR	95% CI	p Value
Age	1.8	0.31 - 1.39	p>0.05
Sex	1.23	1.00–1.51	p>0.05
BMI	1.27	0.45–2.85	p>0.05
Hypertension	0.81	0.61–1.09	p>0.05
Diabetes Mellitus	1.42	1.26–1.60	p<0.05
Smoking	0.75	0.54–1.03	p>0.05
Alcohol	1.37	1.162-1.235	p>0.05
Comorbidities	0.76	0.681-1.328	p>0.05
Habits	1.24	1.285-2.681	p>0.05
Total Cholesterol	1.61	0.995-1.922	p>0.05
Triglycerides	1.63	1.231-1.782	p>0.05
LDL	0.86	0.742-1.529	p>0.05
HDL	1.12	1.01–1.25	p<0.05
Serum Ferritin	1.70	1.15–2.50	p<0.05

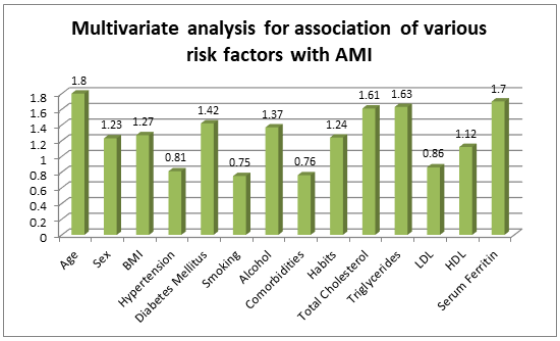

Figure 11: Multivariate analysis for association of various risk factors with Acute Myocardial Infarction (AMI)

DISCUSSION

A total of 200 patients (100 cases and equal number of controls) were enrolled in a case control study to assess the levels of serum ferritin in acute myocardial infarction.

Incidence of acute myocardial infarction is increasing across the globe among all ages and both sexes with increasing morbidity and mortality. In AMI, irreversible tissue injury occurs due to sustained ischemia and recent pivotal studies have shown that the innate immune system is activated sequentially mediating both injury and repair mechanisms.[34,35] The role of ferritin in pathogenesis of coronary artery disease (CAD) has generated considerable interest in recent times.

Epidemiological studies have found a positive relationship between body iron stores and coronary artery diseases.[9,36] Subsequently, evidence of an association of elevated serum ferritin and increased risk of AMI came from various authors,[13,37] which is similar to our findings. However results of some other studies did not show significant correlation between high ferritin and risk of AMI.[24,38] The main possibility that iron over load leads to increased lipid peroxidation and foam cell formation but apart from this the chemical properties of oxidized lipoproteins were found to be chemotactic to blood monocytes, facilitate the entry of lipoproteins by a cytotoxic endothelial injury, and give rise to smooth muscle cell proliferation.[39-41] Native low density lipoprotein in contrasts lacks all these atherogenic potentials.[39,41] One study yielded a strong relation between sonographically assessed carotid atherosclerosis and prominent iron stores in both genders particularly when associated with hypercholesterolemia.[28] Lipid peroxidation therefore may constitute an initiating and crucial step in the development of fatty streaks and plaques. Blood donation has also been reported to be associated with decreased risk of cardiovascular events.[29]

In the present study, majority of the patients in both the groups were in the age group of 41-50 years (42% and 52% respectively). The mean age of the patients in Cases Group was 48.3 ± 14.24 years as compared to 47.6 ± 8.15 years in Control Group. Majority of the patients in both the groups were males and the difference was statistically not significant as per Chi-square test (p>0.05).

Vijaya BM et al,[42] study found the mean age were 45.7±3.8, 46.4±4.1 and 46.2±4.5 in control group and group I and II respectively and the average number of M/F were 82/19.

There was significant difference in BMI of the patients between groups as per Student t-test (p<0.05). This was similar to the study of Bharathi BK et al,[43] study which found significant difference in BMI of patients in controls and cases group.

Hypertension and diabetes mellitus was more prevalent amongst Cases Group as compared to Control Group (32% vs. 28% and 27% vs. 23% respectively) in our study.

The study of Moradi M et al,[44] consisted of 100 consecutive patients with first acute myocardial infarction (AMI), and a control group (n = 50) without history of AMI. There was no significant difference in risk factors including diabetes mellitus, hypertension and current smoking between the groups.

It was observed in our study that the Total Cholesterol, Triglycerides and LDL values were significantly higher and HDL values were significantly lower in Cases Group as Compared to Control Group as per Student t-test (p<0.05). This

correlates to the study of Ishran R et al and Silvia WD et al,[33,46]

In the present study, significantly higher number of patients in Cases Group had serum ferritin level >300µg/l as compared to Control Group (55% vs. 9%). The mean serum ferritin levels were significantly higher in Cases Group as compared to Control Group (332.5 vs. 153.8 µg/l) ($p<0.05$).

Ishran R et al,[33] observed that most (74%) of the myocardial infarction (MI) cases had ferritin level >200 µg/L, while most (74%) of the control subjects had ferritin level below 200 µg/L. Serum ferritin level (>200 µg/L) was significantly associated with AMI ($P<0.01$). Median serum ferritin level of MI cases (211µg/L) was significantly higher than controls (111µg/L).

In our study, the mean serum ferritin levels of males and females patients in Cases Group were significantly higher as compared to Control Group (320.3 vs. 160.1µg/l and 327.7 vs. 137.5 µg/l respectively) as per Student t-test ($p<0.05$). There was no significant difference of mean serum ferritin levels of males and females patients within the group ($p>0.05$).

It was observed in our study that significantly more patients in Cases Group (69%) than Control Group (34%) had concentrations above the cut-off of 200 µg/L ($p<0.05$). In multivariate analysis, Diabetes Mellitus ($P = 0.001$, OR = 7.64, 95% CI 2.37–24.58), HDL ($P < 0.001$, OR = 0.86; 95% CI 0.79–0.93) and serum ferritin (>200 µg/L) ($P < 0.001$, OR = 5.72, 95% CI 2.16–15.17), are found to be independently associated with AMI.

In univariate analysis done by Holay MP et al,[32] Diabetes Mellitus, hypertension, serum cholesterol, high-density lipoprotein (HDL) < 35 and smoking were found to be significantly associated with AMI. In multivariate analysis, high serum ferritin (> 200 µg/L) ($P < 0.001$, OR = 5.72, 95% CI 2.16–15.17), DM ($P = 0.001$, OR = 7.64, 95% CI 2.37–24.58), low HDL (< 35 mg%) ($P < 0.001$, OR = 0.86; 95% CI 0.79–0.93) are found to be independently associated with AMI. When ferritin, cholesterol, BMI and HDL were taken as continuous variables, then also mean serum ferritin ($P = 0.001$) was found to be significantly associated with AMI.

Bharathi BK et al,[43] study reported In univariate analysis, alcohol intake, BMI, DM, hypertension, serum cholesterol, serum triglyceride, high-density lipoprotein (HDL) < 35 and smoking were found to be significantly associated with AMI.

Silvia WD et al,[45] noted that serum ferritin was significantly directly associated with haemoglobin ($r=0.586$, $p < 0.01$), serum cholesterol ($r=0.439$, $p< 0.01$), serum LDL cholesterol ($r=0.381$, $p < 0.01$), serum triglycerides ($r=0.280$, $p < 0.01$) and serum VLDL cholesterol ($r=0.286$, $p < 0.01$). Serum ferritin was significantly inversely correlated with serum HDL cholesterol ($r=-0.210$, $p < 0.05$).

Free Iron, as well as other transition metals, can catalyze free radical formation. For this reason iron is tightly bound to transport and storage proteins to prevent their involvement in free radical formation. It has been hypothesized that increased iron intake or iron stores may promote atherogenesis by increasing free radical formation and oxidative stress.[19] Oxidative stress increases the peroxidation of low-density lipoprotein (LDL) thereby increasing its uptake by macrophages with increased foam cell formation and atherosclerosis.[46,47]

CONCLUSION

Iron is considered as an essential dietary constituent till now, is considered a pro-oxidant. According to "iron hypothesis", iron is believed to be detrimental for the cardiovascular system in promoting atherosclerosis development and progression. Iron, in its catalytically active form, can participate in the generation of reactive oxygen species and induce lipid peroxidation, triggering endothelial activation, smooth muscle cell proliferation and macrophage activation; all of these processes are considered to be proatherogenic.

Higher levels of ferritin, seems to be a strong risk factor for AMI. Patients with higher ferritin level can easily be identified during routine haematological analysis along with other risk factor estimation. Regular monitoring of serum ferritin levels may help in reduction of cardiovascular morbidity and mortality.

Acknowledgements

The authors are indebted to Dr. S L Sajjanar, Assistant Professor, Department of Cardiology for his support and all the staff of the Department of Biochemistry, Shri B M Patil Medical College Hospital & Research Centre, Vijayapura who helped in the laboratory investigation work.

REFERENCES

1. Sharma S, Bhargava B, Bahl VK et al. API Expert Consensus Document on Management of Ischemic Heart Disease. Japi 2006:June;(54)469-80
2. Xavier D, Prem P, Devereaux PJ et al, Treatment and outcomes of acute coronary syndromes in India(CREATE): a prospective analysis of registr y data.The Lancet 2008Apr;9622(371):1435-42
3. Schmitt C, Lehmann G, Schmieder S et al. Diagnosis of Acute Myocardial Infarction in Angiographically documented Occluded Infarct Vessel:Limitations of ST-Segment Elevation in Standard and Extended ECG Leads. Chest 2001;(120):1540-46
4. Zimetbaum PJ, Josephson ME, Use of the Electrocardiogram in Acute Myocardial infarction. New Eng.J Med 2003Mar;(348):933-40
5. Ali SM, Hossian AM, Islam K et al. Normal blood pressure of Bangladeshi adults. Bangladesh Heart J 1997;12:3-7.
6. Boon NA, Fox KA, Bloomfield P. Diseases of the cardiovascular system. In: Haslet C, Chilvers ER, Hunter JA. Boon NA, editors. Davidson's Principles and Practice of

Medicine. 18th ed. New York: Churchill Livingstone; 1999. p. 191-302.
7. Chowdhury BK. Studies on Serum Magnesium and Potassium in Ischemic Heart Disease; Department of Biochemistry, Bangbandhu Sheikh Mujeb Medical University, Dhaka, Bangladesh Thesis; 2001.
8. Ross R. The pathogenesis of atherosclerosis – an update. N Engl J Med 1986;314:488-500.
9. Salonen JT, Nyyssönen K, Korpela H et al. High stored iron levels are associated with excess risk of myocardial infarction in eastern Finnish men. Circulation 1992;86:803-11.
10. Sullivan JL. Iron and the sex difference in heart disease risk. Lancet 1981 13;1:1293-4.
11. Klipstein-Grobusch K, Koster JF, Grobbee DE et al. Serum ferritin and risk of myocardial infarction in the elderly: the Rotterdam Study. Am J Clin Nutr 1999;69:1231-6.
12. Halliwell B, Gutteridge JM. Role of free radicals and catalytic metal ions in human disease: An overview. Methods Enzymol 1990;186:1-85.
13. Salonen JT, Nyyssönen K, Salonen R. Body iron stores and the risk of coronary heart disease. N Engl J Med 1994;331:1159.
14. Walker JM. The heart in thalassaemia. Eur Heart J 2002;23:102-5.
15. Hershko C, Link G, Cabantchik I. Pathophysiology of iron overload. Ann N Y Acad Sci 1998;850:191-201.
16. Westwood MA, Anderson LJ, Maceira AM et al. Normalized left ventricular volumes and function in thalassemia major patients with normal myocardial iron. J Magn Reson Imaging 2007;25:1147-51.
17. Cook JD, Lipschitz DA, Miles LE et al. Serum ferritin as a measure of iron stores in normal subjects. Am J Clin Nutr 1974;27:681-7.
18. Tuomainen TP, Salonen R, Nyysso¨nen K et al. Cohort study of relation between donating blood and risk of myocardial infarction in 2682 men in eastern Finland. BMJ 1997;314:793-4.
19. Baer DM, Tekawa IS, Hurley LB. Iron stores are not associated with acute myocardial infarction. Circulation 1994;89:2915-8.
20. Sempos CT, Looker AC, Gillum RF et al. Body iron stores and the risk of coronary heart disease. N Engl J Med 1994;330:1119-24.
21. Liao Y, Cooper RS, McGee DL. Iron status and coronary heart disease: negative findings from the NHANES I epidemiologic follow-up study. Am J Epidemiol 1994;139:704-12.
22. Mänttäri M, Manninen V, Huttunen JK et al. Serum ferritin and ceruloplasmin as coronary risk factors. Eur Heart J 1994;15:1599-603.
23. Ascherio A, Willett WC, Rimm EB et al. Dietary iron intake and risk of coronary disease among men. Circulation 1994;89:969-74.
24. Reunanen A, Takkunen H, Knekt P et al. Body iron stores, dietary iron intake and coronary heart disease mortality. J Intern Med 1995;238:223-30.
25. Reddy RS, Yousuf S. Emerging epidemic of cardiovascular disease in developing countries. Circulation. 1998;190:596–601.
26. Umesh NK, Monica BK, Christopher TB et al. Prevalence of Conventional Risk Factors in Patients with Coronary Heart Disease. JAMA. 2003;290(7):898–94.
27. Kiechl S, Aichner F, Gerstenbrand F et al. Body iron stores and presence of carotid atherosclerosis: results from the Bruneck Study. Arterioscler Thromb 1994;14:1625–30.
28. Kiechl S, Willeit J, Egger G et al. for the Bruneck Study Group. Body iron stores and the risk of carotid atherosclerosis. Prospective results from the Bruneck Study. Circulation 1997;96:3300–7.
29. Meyers DG, Strickland D, Maloley PA et al. Possible association of a reduction in cardiovascular events with blood donation. Heart 1997;78:188–93.
30. Stampfer MJ, Grodstein F, Rosenberg I et al. A prospective study of plasma ferritin and risk of myocardial infarction in US physicians. Circulation 1993;87:688 (abstr).
31. Corti MC, Guralnik JM, Salive ME et al. Serum iron level, coronary artery disease, and all-cause mortality in older men and women. Am J Cardiol 1997;79:120–7.
32. Holay MP, Choudhary AA, Suryawanshi SD. Serum ferritin—a novel risk factor in acute myocardial infarction. Indian Heart Journal. 2012;64(2):173-177. doi:10.1016/S0019-4832(12)60056-X.
33. Ishran R, Pipliwal PS, Verma MK. Serum Ferritin in Patients with Acute Myocardial Infarction. IOSR Journal of Dental and Medical Sciences (IOSR-JDMS). 2016. Volume 15, Issue 2 Ver. VI, PP 08-11
34. Nahrendorf M, Pittet MJ, Swirski FK. Monocytes: protagonists of infarct inflammation and repair after myocardial infarction. Circulation. 2010;121:2437-45.
35. Hilgendorf I, Gerhardt LM, Tan TC et al. Ly-6C high monocytes depend on Nr4a1 to balance both inflammatory and reparative phases in the infarcted myocardium. CircRes. 2014;114(10):1611-22.
36. Solymoss BC, Marcil M, Gilfix BM. The place of ferritin among risk factors associated with coronary artery disease. Coron Artery Dis 1994; 5:231–5.
37. Delphine W, Silvia CR, Biswas S et al. Ferritin—a potent threat for acute myocardial infarction. J Assoc Physicians (India) 2003; 51:947–50.
38. Gupta R, Rastogi S, Nagar R et al. Dietary and serum iron, body iron stores and coronary heart disease. J Assoc Physicians (India) 2000; 48:489–92.
39. Steinberg D, Parthasarathy S, Carew TE et al. Beyond cholesterol: modifications of low density lipoprotein that increase its atherogenicity. N Engl J Med. 1989; 320:915-924.
40. Boulanger CM, Tanner FC, Bea ML et al. Oxidized low density lipoproteins induce mRNA expression and release of endothelin from human and porcine endothelium. Ore Res. 1992; 70:1191-1197.
41. Quinn MT, Parthasarathy S, Fong LG et al. Oxidatively modified low density lipoproteins: a potential role in recruitment and retention of monocyte/ macrophages during atherogenesis. Proc Natl Acad Sci USA. 1987; 84:2995-2998.
42. Vijaya BM, Srilekha S, Saranya M et al. Assessment of ferritin and its association with C - reactive protein and malondialdehyde in acute myocardial infarction. Int J Res Med Sci 2015;3:3581-5.
43. Bharathi BK and Chandrakar S. Serum Ferritin - A Potential Threat And Risk Factor For Acute Myocardial Infarction. IJPBS. 2013, Volume 3, Issue 1, 7-13.
44. Moradi M, Farnaz Fariba, Ali Sadeghi Mohaseli. Relation between the Serum Ferritin Level and the Risk for Acute Myocardial Infarction. Journal of Research in Health Sciences. 2015;15(3):147-151
45. Silvia WD, Biswas S, Uthappa S et al. Ferritin, a Potent Threat for Acute Myocardial Infarction? JAPI. 2003:51;947-950.
46. Sempos CT, Looker AC, Gillum RF. Iron and Heart disease. The epidemiological data. Nutr Rev 1996;54:73-4.
47. Ester bauer II, Phul GJ II, Jargons G. The role of lipid peroxidation and antioxidants in oxidative modification of LDL. J Free Radical Biological Med 1992;13:341-90.

How to cite this article: Herakall M, Biradar MS. A Study of Serum Ferritin in Acute Myocardial Infarction: A Case Control Study in BLDE (Deemed To Be University), Shri BM Patil Medical College, Hospital and Research Centre Vijayapura. Ann. Int. Med. Den. Res. 2018; 4(5):ME01-ME07.

Source of Support: Nil, **Conflict of Interest:** Nil.