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Original Research

Serum ferritin level in patients with acute myocardial infarction

¹Dr Lal Babu Prasad, ²Dr Bhautikkumar Bhanabhai Patel

^{1,2}Assistant Professor, Department of General Medicine, Venkateshwara Institute of Medical Sciences, Gajraula, Uttar Pradesh, India

ABSTRACT:

Background: AMI is one of the most common causes of morbidity and mortality in the industrialised world, and is becoming increasingly common in India as well. The present study was conducted to assess serum ferritin level in patients with acute myocardial infarction. **Materials & Methods:** of 80 patients of coronary heart disease of both genders (Group I) and age and sex-matched controls were selected (Group II). Serum ferritin was measured by enzyme-linked immunosorbent assay (ELISA) test. **Results:** The mean hemoglobin in group I was 12.1 gm% and in group II was 12.9 gm%, Ferritin level in group I was 280.4 µg/l and in group II was 98.2 µg/l, cholesterol level was 190.2 mg/dl and in group II was 86.4 mg/dl, HDL was 38.5 mg/dl in group I and 44.1 mg/dl in group II, LDL level was 116.4 mg/dl in group I and 86.2 mg/dl in group II, TG was 164.2 mg/dl in group I and 132.4 mg/dl in group II and VLDL was 33.5 mg/dl in group I and 27.2 mg/dl in group II. The risk factors for AMI was diabetes in 63 and 12, hypertension in 65 and 15, smoking in 52 and 6, BMI >25 kg/m2 in 48 and 14, cholesterol >200 mg/dl in 52 and 20, HDL < 35 mg/dl in 50 and 12, LDL >150 mg/dl in 68 and 5, TG >160 mg/dl in 72 and 3 and VLDL >40 mg/dl in 61 and 18 in group I and II respectively. The difference was significant (P< 0.05). **Conclusion:** Measurement of serum ferritin may be used as a complementary tool for confirming the diagnosis of AMI. **Key words:** Acute myocardial infarction, Enzyme-linked immunosorbent assay, Serum ferritin

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Corresponding author: Dr Bhautikkumar Bhanabhai Patel, Assistant Professor, Department of General Medicine, Venkateshwara Institute of Medical Sciences, Gajraula, Uttar Pradesh, India

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INTRODUCTION

AMI is one of the most common causes of morbidity and mortality in the industrialised world, and is becoming increasingly common in India as well. The mortality rate in AMI is approximately 30% within first month, of which 50% are attributed to sudden cardiac death, and affects people in their most productive period of life.¹ The WHO Region for South-East Asia has set a target of reducing premature mortality from cardiovascular disease and other noncommunicable diseases (NCDs) by 25% by 2025 and has developed a regional action plan to achieve this target.² Atherosclerosis is the primary cause of heart disease and stroke. Deaths arising as a complication of atherosclerosis claim the lives of millions of people each year in the Western world, and are also rapidly rising in developing countries.³

Ferritin, an iron storage protein, is widely distributed in eukaryotic cells. Multiple molecular types of ferritin have been isolated from different tissues such as liver, spleen, heart and placenta.⁴ These types behave differently on isoelectric focusing. Thus, ferritin derived from adult heart and placenta shows more acid iso-ferritins on isoelectric focusing than liver and spleen ferritins. The concentration of serum ferritin in healthy individuals is positively correlated with the body iron stores.⁵ High concentrations of serum ferritin have been reported in patients with iron overload, severe liver diseases, and malignant diseases. In acute inflammation, the serum ferritin level increases concomitantly with a decrease in serum iron.⁶ The present study was conducted to assess serum ferritin level in patients with acute myocardial infarction.

MATERIALS & METHODS

The present study consisted of 80 patients of coronary heart disease of both genders (Group I). All were included in the study with their written consent. The diagnosis of AMI was based on any two of the following criteria: Typical history of severe chest pain radiating to the neck or arms for < 12 hours duration 1 ECG changes of ST elevation > 2 mm in two or more chest leads, or > 1 mm in two or more limb leads 1 Rise in serum cardiac enzymes concentration (troponin T or I), more than twice the upper limit of normal. Presumably, new onset left bundle-branch block. Age and sex-matched controls were selected (Group II).

All the subjects were assessed by clinical examination, ECG, serum creatine kinase-MB (CK-

RESULTS Table I Patient characteristics

Parameters	Group I	Group II	P value
Hemoglobin (gm%)	12.1	12.9	0.13
Ferritin (µg/l)	280.4	98.2	0.02
Cholesterol (mg/dl)	190.2	86.4	0.04
HDL (mg/dl)	38.5	44.1	0.05
LDL (mg/dl)	116.4	86.2	0.01
TG (mg/dl)	164.2	132.4	0.04
VLDL (mg/dl)	33.5	27.2	0.05

Table I, graph I shows that mean hemoglobin in group I was 12.1 gm% and in group II was 12.9 gm%, Ferritin level in group I was 280.4 µg/l and in group II was 98.2 µg/l, cholesterol level was 190.2 mg/dl and in group II was 86.4 mg/dl, HDL was 38.5 mg/dl in group I and 44.1 mg/dl in group II, LDL level was 116.4 mg/dl in group I and 86.2 mg/dl in group II, TG was 164.2 mg/dl in group I and 132.4 mg/dl in group II and VLDL was 33.5 mg/dl in group II. The difference was significant (P< 0.05).



Graph I Patient characteristics

Table II Risk factors for AMI in both genders

Parameters	Group I	Group II	P value
Diabetes	63	12	0.05
Hypertension	65	15	0.04
Smoking	52	6	0.01
BMI >25 kg/m ²	48	14	0.02
Cholesterol >200 mg/dl	52	20	0.03
HDL < 35 mg/dl	50	12	0.03
LDL >150 mg/dl	68	5	0.01
TG >160 mg/dl	72	3	0.001
VLDL >40 mg/dl	61	18	0.04

MB). Height and weight and BMI were recorded. Body mass index > 25 kg/m^2 was considered as a risk factor for AMI. Estimation of lipids was done by enzymatic method using autoanalyzer. Serum ferritin was measured by enzyme-linked immunosorbent assay (ELISA) test. Results were tabulated and subjected to statistical analysis. P value less than 0.05 was considered significant. Table II, graph II shows that risk factors for AMI was diabetes in 63 and 12, hypertension in 65 and 15, smoking in 52 and 6, BMI >25 kg/m2 in 48 and 14, cholesterol >200 mg/dl in 52 and 20, HDL < 35 mg/dl in 50 and 12, LDL >150 mg/dl in 68 and 5, TG >160 mg/dl in 72 and 3 and VLDL >40 mg/dl in 61 and 18 in group I and II respectively. The difference was significant (P<0.05).



Graph II Risk factors for AMI in both genders

DISCUSSION

Iron is a transition metal that can catalyze toxic redox reactions and, it has been suggested to be involved in many harmful biological processes in the human body.^{7,8} Free radicals (FR), especially the hydroxyl radical (-OH), are extremely reactive and initiate tissue damage and lipid peroxidation. Consequently, when excess iron is consumed and transferrin becomes saturated, free iron can be released.^{9,10} The production of FR by free iron has been found in some studies to cause oxidative damage to the coronary arteries, and possibly oxidize LDL, resulting in even more coronary damage.^{11,12} The present study was conducted to assess serum ferritin level in patients with acute myocardial infarction.

In present study, we found that mean hemoglobin in group I was 12.1 gm% and in group II was 12.9 gm%, Ferritin level in group I was 280.4 µg/l and in group II was 98.2 µg/l, cholesterol level was 190.2 mg/dl and in group II was 86.4 mg/dl, HDL was 38.5 mg/dl in group I and 44.1 mg/dl in group II, LDL level was 116.4 mg/dl in group I and 86.2 mg/dl in group II, TG was 164.2 mg/dl in group I and 132.4 mg/dl in group II and VLDL was 33.5 mg/dl in group I and 27.2 mg/dl in group II. Moroz et al¹³ in their study serum ferritin level was determined in 20 patients with acute myocardial infarction (AMI) during the first 10 days post infarction. Starting on the second day, a gradual increase in serum ferritin level was detected, reaching a maximum of four times the initial level on the sixth day after the infarction. In addition, a significant increase in ferritin content was found in the peripheral blood monocytes on the fifth day after the event. The control group comprised six patients suffering from chest pains not due to AMI. In all of them the serum ferritin level was found to be within normal limits. Peripheral blood monocytes derived from healthy individuals incubated with hydrocortisone, showed a significant enhancement of their ferritin content, a finding suggesting that these cells activated by steroids during stress could be a source of the increased serum ferritin level following AMI.

We found that risk factors for AMI was diabetes in 63 and 12, hypertension in 65 and 15, smoking in 52 and 6, BMI >25 kg/m2 in 48 and 14, cholesterol >200 mg/dl in 52 and 20, HDL < 35 mg/dl in 50 and 12, LDL >150 mg/dl in 68 and 5, TG >160 mg/dl in 72 and 3 and VLDL >40 mg/dl in 61 and 18 in group I and II respectively. Sharma et al14 assessed association of serum ferritin with AMI and assessed the relationship of serum ferritin with the established conventional risk factors for AMI. Mean serum ferritin (263.674 \pm 89.029 µg/l) was significantly higher in cases than controls (98.833 \pm 62.682 µg/l). Amongst the patients of AMI, significantly higher level of serum ferritin was found in diabetics (340.63 \pm 90.78 µg/l) than non-diabetics (225.19 \pm 58.30 μ g/l), male elderly (> 60 years of age) smokers $(304.20 \pm 88.60 \ \mu g/l)$ compared to non- smoker $(206.37 \pm 48.88 \ \mu g/l)$, elderly male with high (> 150 mg/dl) LDL cholesterol $(323.48 \pm 86.73 \mu g/l)$ compared to patients with normal (< 150 mg/dl) LDL cholesterol (249.33 \pm 66.12 µg/l). Similarly, young patients of AMI (< 50 years of age) with high (> 160 mg/dl) triglyceride had significantly higher serum ferritin (324.06 \pm 160.68 µg/l) compared to patients

having normal (< 160 mg/dl) triglyceride level (242.46 \pm 70.78 ug/l). Also, male patients of AMI with high (> 40 mg/dl) VLDL cholesterol had significantly higher serum ferritin (326.49 \pm 77.95 µg/l) compared to male patients with normal (< 40 mg/dl) VLDL cholesterol (257.18 \pm 85.46 µg/l).

CONCLUSION

It is concluded that measurement of serum ferritin may be used as a complementary tool for confirming the diagnosis of AMI.

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