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Original Research Article

Follow up of serum ferritin levels in myocardial infarction in Indian males

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ABSTRACT

Introduction: National health & nutritional examination survey, 1988-1994, first time reported a significant positive association in iron storage & heart disease risk. Due to the scarcity of this type of study in India, we did this study to find out the relation of serum ferritin with MI

Aim: The current study attempts to evaluation association of ferritin levels at the onset of AMI & at 6^{th} day of follow up of subjects.

Materials and Methods: A total of 39 male patients who were confirmed of having MI on the basis of lipid profile, CKMB, SGOT, SGPT, Trop I, ECG & Chest X Ray were included in the study. Serum ferritin level was estimated in study group on day 1 & day 6 of MI attack while only once in control. Serum ferritin was estimated by enzyme linked fluorescent assay on Mini Vidas auto analyzer from Biomerieux.

Results: Mean serum ferritin levels at Day-1 was not significantly elevated than controls [58.51 (±18.71) ng/ml in day-1 versus 64.09 (±19.42)]. In contrast serum ferritin levels at Day-6 were significantly elevated than controls [226.68 (±18.71) ng/ml at day-6 versus versus ferritin levels in controls ($p < 0.001$)].

Conclusion: In conclusion, our data supports that elevated serum ferritin levels are associated with pathogenesis of AMI.

1. Introduction

It has been gauge that 12.2% of passing happen from ischemic coronary illness that has been a main source of death in intense myocardial localized necrosis (AMI).[1] It has been assessed that there could associate with 30 million patients experiencing computer aided design in India.[2] Information from the USA propose around 25% of passing's in the USA are related with coronary illness each year.[3] Risk factors for AMI incorporate hypertension, smoking, diabetes, absence of activity, corpulence, high blood cholesterol, horrible eating routine & unnecessary liquor intake.[4,5]

Iron is engaged with pathogenesis of atherosclerosis & coronary conduit sickness as proposed by Sullivan et al, as iron to be cardiovascular gamble factor.[6] A Finnish report likewise reasoned that iron admission was related with myocardial dead tissue (MI).[7] Another review proposed that age & sex-related expansions in iron stores have been connected to the pathogenesis of a few normal sicknesses, including atherosclerosis.[8,9] Similarly, though bringing down iron levels by phlebotomy was proposed to be athero-protective.[10]

Ferritin is pervasive intracellular protein that stores iron & delivery it in controlled design. The presence of redox-dynamic iron, as well as high articulation levels of H-and L-ferritin in human atherosclerotic sores offered roundabout help for the iron hypothesis.[11,12] L-ferritin levels are expanded in coronary courses from patients with coronary vein illness (computer aided design), demonstrating that iron aggregates in atherosclerotic plaques.[13] As per

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these epidemiological examinations, high foundational iron levels, checked by serum ferritin levels or transferrin immersion, decidedly connected with expanded hazard of myocardial infarction.[14,15]

However many examinations have been directed in regards to relationship of ferritin & myocardial injury, a couple of studies have been led in India. Moreover, apparently no review has been directed in Indian subcontinent that has followed up ferritin levels in Intense Myocardial Dead tissue.

2. Materials and Methods

The study was conducted in Department of Biochemistry, Subharti Medical College, and Meerut on patients who came to attend OPD/emergency with complaints of chest pain between 2014 – 2015. The diagnosis of AMI was confirmed only after complete physical examination, X-Ray, cardiac markers & other routine biochemistry tests/urine microscopy. The study group consisted of 39 male patients of diagnosed AMI & 40 age matched male controls. Blood sample was collected on Day-1 (onset of AMI) & Day-6 of AMI while one sample was collected for control group. Any subject with suspected or diagnosed case of concurrent medical illness as chronic alcoholism, liver disease, neoplastic disease, diabetes mellitus or hemochromatosis were excluded from current study.

The study was approved by Institutional Ethical Committee & informed consent was taken for both cases & controls. The study group was subjected to structured questionnaire (demographic, medical & lifestyle). After the interview, a 5 ml blood sample was collected from each subject in plain vial. The sample was refrigerated & analysis for serum ferritin was performed on the same day. Serum Ferritin was analyzed by Enzyme Linked Fluorescent Assay on Minividas Autoanalyzer from Biomerieux.

Statistical analysis was done using SPSS & Microsoft excels software. In all tests of significance two sided p-values have been reported.

3. Results

Mean serum ferritin levels at Day-1 was not significantly elevated than controls [58.51 (±18.71) ng/ml in day-1 versus 64.09 (±19.42)]. In contrast serum ferritin levels at Day-6 were significantly elevated than controls [226.68 (±18.71) ng/ml at day-6 versus versus ferritin levels in controls ($p < 0.001$)]. When ferritin levels at day-1 & day-6 were compared for means, day-6 levels were significantly elevated than controls ($p < 0.001$). Mean of net increase in ferritin levels was 168 ± 97.75 & when it compared with levels at day-6, it was found that day-6 levels were significantly increased with p value of < 0.05.

4. Discussion

In the present settled case control concentrate on the accomplice comprised of 39 patients that created AMI (day-1) & they were followed up till 6th day (day-6) including 40 controls that were not followed up. Apparently it is the first such concentrate in quite a while. Serum levels of ferritin on day-1 were not fundamentally changed when contrasted with controls (p=0.19). Though S. Ferritin levels on day-6 were expanded fundamentally in cases than controls ($p < 0.001$) [Table 1 /Figure 1]. The discoveries in present review have been steady with before concentrates as Bharathi et al., & Shipra et al., viewed serum levels of ferritin as essentially higher in cases when contrasted with controls.[16,17] Comparably companion concentrate on Finnish men likewise proved high grouping of serum ferritin to be emphatically connected with the rate of MI.[18]

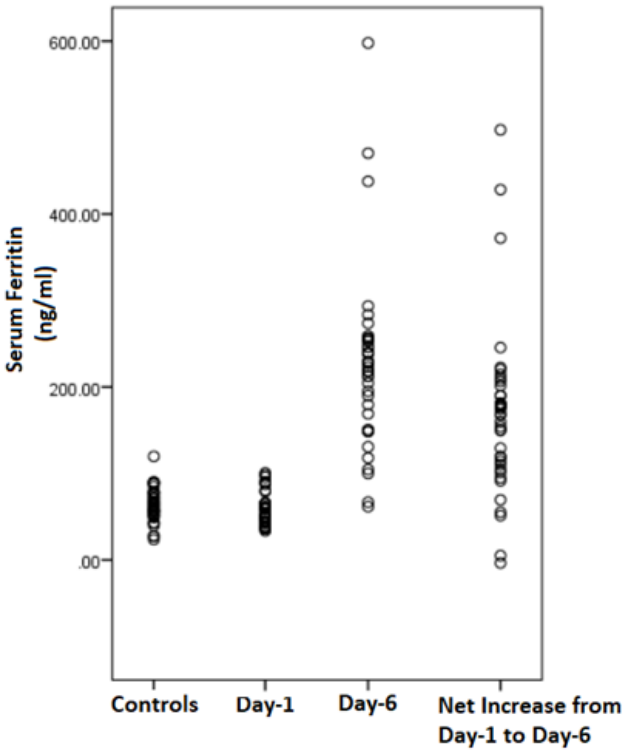

Figure 1: Representation of levels of S. Ferritin in controls, day-1, Day-6 & net increase

Ferritin levels at day-6 were fundamentally expanded that levels at day-1 ($p< 0.001$). Expansion in ferritin levels from day-1 to day-6 was determined by deducting levels at day-6 by day-1 (Net expansion in Ferritin levels). It was seen that when net expansion in ferritin levels was contrasted & day-6, it was genuinely critical on day-6, however not exceptionally huge ($p< 0.05$) [Table 1 /Figure 1]. These finding are predictable with that of Moroz C et al., where it was seen that there was slow expansion in ferritin levels post MI.[19]

Table 1: Comparison of variables in AMI at day-1, day-6 and control

Variable	Case	Control	p value
N	39	40	•
Age	59.15 ± 12.63	59.78 ± 12.63	0.81
Ferritin Levels Day-1 in ng/ml (Mean ± SD)	58.51 ± 18.71	64.09 ± 19.42	0.19
Ferritin Levels Day-6 in ng/ml (Mean ± SD)	226.68 ± 101.28	64.09 ± 19.42	<0.001
Ferritin Levels Day-1 Vs Day-6			< 0.001
Net Increase in Ferritin Levels	168 ± 97.75	p value when compared with Day-6 = 0.01	

* N: Number, Net increase in ferritin level = Levels at day-6 minus levels at day-1

Iron is a redox dynamic metal & strong impetus. The exchange of electrons between the ferrous & ferric states adds to the arrangement of ROS through the Fenton reaction.[20]

Bull LDL is taken up by high-proclivity LDL receptors on macrophages, ultimately driving their advancement into froth cells. Froth cells upregulate proteolytic proteins, for example, grid metalloproteases (MMPs) & corrupt extracellular network structure, prompting plaque crack & MI.[21] Ferroptosis is an iron-subordinate type of corruption & portrayed by collection of lipid peroxides.[22]

Ferritin articulation is incited simultaneously with movement of injury. Union of apoferritin happens inside macrophages & endothelial cells that could be a defensive system against harming impacts of free iron.[23,24] Improved ferritin creation in endothelial cells makes them more impervious to oxidative injury that elevates ibuprofen to forestall endothelial harm in cardiovascular disease.[25]

5. Conclusion

In conclusion, our data supports that elevated serum ferritin levels are associated with pathogenesis of AMI, though it could not be concluded that it is causal association or outcome of the disease due to limited sample size & inclusion of more variables associated with pathogenesis of atherosclerosis.

6. Conflict of Interest

None.

7. Source of Funding

None.

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