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LIPID PROFILE AND SERUM URIC ACID LEVELS IN PATIENTS OF MYOCARDIAL INFARCTION: A CASE CONTROL STUDY

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ABSTRACT

Background: Hyperuricemia has been shown to increase the risk of CHD related-events such as MI independently of other CHD risk factors, and is linked to higher mortality rates of CHD. A recent meta-analysis have shown that each 1 mg/dL increase in serum uric acid, there is a 12% increase in mortality. Several studies have showed that deranged lipid metabolism is one of the major factors in the development of this ischemic heart disease.

Material and Method: The study was carried out on 77 patients of myocardial infarction, and 72 controls. In the study we have investigated changes in lipid profile and serum uric acid in people suffering from myocardial infarction and compare these values with those of normal subjects. Healthy volunteers of 40-60 years who were not suffering from hypertension were selected randomly as controls from the hospital and college.

Results: Study included 72 persons in control group while 77 patients of MI. Total Cholesterol, triglycerides, VLDL and uric acid was significantly elevated in hypertensive group (P<0.0001), while LDL and HDL levels were not significantly different between the two groups. Statistical analysis for males showed no difference between cases and controls for Cholesterol, TG, LDL, VLDL and uric acid (p>0.05). Among female population this difference of elevated parameters in MI group was significant for all the parameters.

Conclusion: Significantly raised cholesterol, triglyceride, VLDL and Serum uric acid levels are associated with myocardial infarction while HDL and LDL levels show no statistical association.

KEYWORDS : Myocardial infarction, Lipid profile, uric acid

INTRODUCTION

Myocardial infarction (MI) characterized by insufficient blood supply to a part of the heart, and it is mostly due to the formation of occlusive thrombus or embolism at the site of rupture or erosion of an atheromatous plague in coronary artery results in myocytic ischemia and cell death.[1]

Lipoproteins are complex functional proteins that transport various lipids and proteins in plasma.[2] Various types of lipoproteins have been classified by their physical and chemical properties, like their flotation characteristics during ultracentrifugation.[3] The lipids are mainly free and esterified cholesterol, triglycerides, and phospholipids. The lipoprotein core is formed by hydrophobic triglyceride and cholesteryl esters, covered with a unilamellar surface of amphipathic phospholipids and scanty amounts of free cholesterol and proteins. Apo B100 is responsible for the secretion of hepatic-derived VLDL, IDL, and LDL, while Apo B48 is a truncated form of Apo B100 which is responsible for secretion of chylomicrons from the small intestine.[4] Uric acid is a heterocyclic organic compound and final metabolic product of purine metabolism in humans which is excreted in urine (Ming, 2012). Uric acid produced from xanthine by the enzyme xanthine oxidase.[5]

Hyperuricemia has been shown to increase the risk of CHD related-events such as MI independently of other CHD risk factors, and is linked to higher mortality rates of CHD. A recent meta-analysis have shown that each 1 mg/dL increase in serum uric acid, there is a 12% increase in mortality.[6] The presence of hyperuricemia significantly increases the risk of CHD in women, but not in men.[7]

Several studies have showed that deranged lipid metabolism is one of the major factors in the development of this ischemic heart disease. There are significantly higher total cholesterol (TC) and triglyceride (TG) levels and lower high-density lipoprotein cholesterol (HDL) levels in AMI patients.[8] Although in one other study, levels serum low-density lipoprotein cholesterol (LDL) levels and the ratio of LDL to HDL were comparable in two groups; however, serum HDL levels were significantly decreased in AMI group.[9]

MATERIAL AND METHODS

The present work was conducted in the department of medical biochemistry of Gandhi Medical College, Bhopal in co-ordination with department of cardiology and department of Biochemistry, Netaji Shubhash Chandra Bose Medical College Jabablpur.

The study was carried out on 77 subjects of myocardial infarction patients, and 72 controls. In the study we have investigated changes in biochemical parameters in patients suffering from myocardial infarction and compare these values with that of normal subjects. Biochemical parameters in included are:-

1. LIPID PROFILE
2. URIC ACID

Healthy volunteers of 40-60 years and and were not suffering from any cardiovascular disease or having risk factors for lipid profile or uric acid changes were selected randomly as controls from the hospital and college. All the cases were taken from cardiology ward of hamidia hospital.

Those patients who were below the age of 20 years of older than 60 years, patients with chronic kidney disease, Gout and those on vitamin A supplements were excluded from the study.

Taking all aseptic and universal precaution 5 ml of blood sample was collected from all cases and controls in plain and sterile vials. Blood was allowed to clot and the serum was separated by centrifugation and different parameters were estimated. Hemolysed samples obtained was analysed for lipid profile and uric acid. Estimation of serum cholesterol was done by enzymatic method end point CHOD-POD method (cholesterol oxidase and per-oxidase method). Serum triglyceride is estimated by enzymatic GPO-POD method (glycerol-3-PO4 oxidase and peroxidase). VLDL-C was calculated as one fifth of triglyceride. Estimation of low density lipoprotein cholesterol was calculated using freidwalds formula [LDL-C = TC (HDL-C +VLDL–C)]. Estimation of uric acid was done by "Uricase / pod , end point assay.

RESULTS AND OBSERVATION

Study included 72 persons in control group while 77 patients of MI in cases group to be compared for lipid profile and serum uric acid.

Table 1: Distribution of control and cases according to age group

S. No.	Age Group (N=149)	No. of control (n=72)	No. of cases (n=77)
1	18-25	28 (38.89 %)	07 (9.09 %)
2	26-35	26 (36.11 %)	15 (19.48 %)

3	36-45	08 (11.11 %)	19 (24.68 %)
4	46-55	06 (8.33 %)	17 (22.08 %)
5	56 & above	04 (5.56 %)	19 (24.68 %)

Most of the patients in control groups were in young to middle age group with 28 (38.89 %) and 26 (36.11 %) patients in 18-25 years and 26-35 years of age groups respectively. Patients in the cases group was mostly in middle to old age where 36 (46.76 %) patients belong to more than 46 years of age.

Table 2: Comparison of all parameter in cases and control for MI

Parameter	Control (n=72)	Cases (n=75)	t-test (p value)
Chol	176.48±14.1	248.25±41.6	<0.0001
TG	152.48±117.2	230.27±69.2	<0.0001
HDL	49.00±8.80	79.78±335.2	>0.005
LDL	121.00±103.2	143.43±32.6	>0.005
VLDL	31.06±7.07	51.44±16.8	<0.0001
Uric Acid	5.31±1.54	16.30±6.8	<0.0001

The above table, comparison of all parameter is shown between hypertensive cases to non-hypertensive controls. Total Cholesterol, triglyceride, VLDL and uric acid was significantly elevated in hypertensive group (P<0.0001), while LDL and HDL levels were not significantly different between the two groups.

Statistical analysis for males showed no difference between cases and controls for Cholesterol, TG, LDL, VLDL and uric acid (p>0.05). Among female population this difference of elevated parameters in MI group was significant for all the parameters. There was no statistical difference seen between male and females of with in the case group and in the control group.

In the control group CHOL was highest in 26-35 years age group, TG & HDL in 18-25 years age group, LDL & VLDL in 56 years and above age group and uric acid was highest for 46-55 years age group.

In the case group CHOL and TG was highest in >56 years age group, HDL and VLDL in 18-25 years age group, LDL in 36-45 years and above age group and uric acid was highest for 26-35 years age group.

DISCUSSION

Our study showed significant derangement in lipid profile in the form of increase CHOL, TG and VLDL. Some studies showed correlations between the occurrence of AMI and abnormality of lipid profiles.[10] Some other studies similar to ours, showed increase in serum triglycerides during AMI.[11,12] Similarly study of *Salahuddin et al.* revealed significantly high levels of triglycerides and low levels of HDL-cholesterol in Acute MI patients.[12] While *Ryder et al.*[13] reported that there is no significant alteration in triglyceride levels. On the contrary study of *Bitla et al*[14] showed no significant change in lipid profile in MI. In our present study, serum triglyceride levels showed significant increase in AMI patients when compared with control subjects. The cause of elevated triglyceride levels may be genetic basis or/and nutritional habits.[15,16] Triglyceride levels may change due to inherited abnormality of very low density lipoprotein. It may be happen due to increased flowing of fatty acids and impaired elimination of VLDL from the plasma.[17]

We found significantly increased serum total cholesterol levels in acute myocardial infarction. Our study is in agreement and in contrast to that by other researchers who found either an increase[18] or a decrease[20] or normal cholesterol[10] in the acute myocardial infarction. This shows that serum cholesterol level reveal no differences between persons with and without AMI. Elevated serum cholesterol has depended on elevated consumptions of fat and genetic basis.[15,21] LDL carries the most of cholesterol in the plasma and increasing of LDL depend on increasing of total cholesterol level[22] Rise of HDL concentration in our findings is in agreement with the other studies that shown either a increase[20] or no change[23] and in agreement with the other study.[12] Several studies have supported that the ratios of LDL-cholesterol /HDL-cholesterol and total cholesterol /HDL-cholesterol show the atherosclerotic injury of the wall of the vessels.[24]

We found slight increase in LDL cholesterol and HDL-cholesterol levels in acute myocardial infarction which was not significant. LDL cell surface receptors cleaned LDL-Ch from the circulation. These receptors may change as a result of coronary heart disease, thus uptake of LDL-Ch is decreased.[25]

Several mechanisms could cause the uric acid metabolic pathway to be a cardiovascular risk factor. Uric acid may stimulate vascular smooth cell proliferation, and reduce vascular nitric oxide production.[26] Our finding that uric acid increases risk of coronary heart disease is in line with previous studies on the association between uric acid and coronary heart disease.[27,28] Some of these studies found the association only in women.[29] In our study, too, associations seemed to be significantly stronger in women than in men.

There are several suggested mediators of the deleterious effect of uric acid on cardiovascular health such as dysfunctional vascular endothelium; adhesiveness of platelets and granulocytes, subsequent release of cytokines and their effect on atherosclerotic plaques, and oxidative stress.[30] A recent study found that hyperuricemia was significantly associated with poor outcomes in cardiac disease patients such as MI patients.[31]

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