SERUM LIPOPROTEIN (a) AND LIPID PROFILE IN YOUNG SOUTH INDIAN PATIENTS WITH MYOCARDIAL INFARCTION.

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ABSTRACT

Coronary artery disease is now a major health problem in India. In past few decades the battle to reduce the incidence of coronary artery disease has led the researchers to look for various clinical markers, which would help early diagnosis of the diseases. The present study was undertaken to assess the level of lipoprotein (a) [Lp(a)] and few other lipids in selected myocardial infarction (MI) patients below 45 years without having any traditional risk factors but with positive family history. Fasting blood samples were taken from 65 patients and their total cholesterol, LDL cholesterol, VLDL cholesterol, HDL cholesterol, triglycerides and serum Lp(a) were determined. The control group consisted of 50 age matched healthy individuals. The mean Lp(a) level was 58.6 ± 3.20 mg/dl in patients and 19.70 ± 0.18 mg/dl in controls. Thus Lp(a) levels were found significantly higher in patients with MI ($p < 0.05$ for patients versus control) as compared to the controls. There was no significant difference in the levels of total cholesterol (TC), LDL, VLDL, HDL, TGL as compared to controls but there was an increase in TC/HDL cholesterol ratio. The results of this study suggest that high level of Lp(a) and TC/HDL ratio has a distinctive association with MI, independent of other common coronary risk factors. Hence, Lp(a) level in serum emerges to be a promising marker for diagnosis of coronary artery diseases.

INTRODUCTION

Acute myocardial infarction is the most important consequence of coronary artery disease. Although traditional risk factors of MI are helpful in diagnosis, specific clinical markers would be valuable in identifying the persons who are at risk. In the past few decades, much attention has been focused on serum Lp(a) and other lipids mainly because of their strong association with coronary artery disease.

Lp(a) is a complex lipoprotein consisting of a central core of LDL, covalently linked by a single disulphide bond to a polypeptide chain of apolipoprotein (a). The structural gene for apo(a) is located on chromosome six(1). Many studies have shown a direct relationship between Lp(a) and coronary heart disease (2,3). The present study was designed to verify whether there is increase in the concentration

of Lp(a) and other lipids in young South Indian MI patients, without having any traditional risk factors except for positive family history and to compare the results with that of age matched healthy controls.

DESIGN AND METHODS

Population Study

65 young male survivors of MI (< 45 years of age) with no traditional risk factors (diabetes, high cholesterol, hypertension, smoking, abnormal body mass index and non-vegetarians) but with positive family history were selected for the study. None of the selected patients had a record of altered blood pressure, hypercholesterolemia or altered blood glucose levels. The control group comprised of 50 age and sex matched healthy individuals. MI was confirmed with characteristic ECG signs and rise in total creatine-kinase activity (more than 3 times the upper reference value of our laboratory) and altered CK-MB fractions.

Laboratory measurements

Blood was collected from patients within four hours from the onset of the infarction. After centrifugation

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serum was stored at -20°C until measurement. The measurement was done within 7 days after collection of the samples. Lp(a) was measured immunoturbidimetrically on an auto analyzer. (Technicon RA-50, USA).

Serum concentrations of total cholesterol, triglycerides and high density lipoprotein cholesterol were measured by the auto-analyzer. (Cobas-Mira, Switzerland). Low density lipoprotein cholesterol was calculated using the Friedewald formula. $\text{LDL Cholesterol} = \text{Total Cholesterol} - (\text{TGL} / 5 + \text{HDL})$ and the total / HDL cholesterol ratio was also calculated.

Statistical analysis

All values are expressed as the mean $\pm$ standard error. To test the significance, the student's 't' test was used.

RESULTS AND DISCUSSION

Statistically significant differences were observed in the mean of serum Lp(a) concentrations between the myocardial infarction patients (58.6 ± 3.20 mg/dl) (Table:1, Fig:1) and in controls. (19.6 ± 0.10 mg/dl). The mean values of TGL, LDL, and VLDL lies within the reference values for both the patients and the controls. But the mean total / HDL Cholesterol ratio was above 5 in MI patients and in controls it was below 4 (Table: 1, Fig: 2).

When compared with controls, the mean serum Lp(a)

levels were significantly higher among south Indian MI patients with positive family history. The optimum TC/HDL cholesterol ratio is 3 and the average is 4. A TC/HDL ratio > 5 appears to be a strong predictor of coronary artery disease (4,5) 25% of the MI patients have increased TC/HDL (>6) cholesterol ratio and this needs further study extending to a larger sample size.

Lp(a) may induce atherosclerosis in the following manner. The apo(B)-100 which is a component of Lp(a) binds to LDL receptors and acts as atherogenic protein. It also mimicks or blocks plasminogen and competes for binding to fibrinogen, thus acting as a prothrombin agent. Lp(a) inhibits clot lysis and sometimes it binds to proteoglycan thereby promoting increased uptake by macrophages and induces smooth muscle proliferation. (Fig 1). (2,6,7)

Our data suggests that irrespective of other traditional risk factors, increase in Lp(a) is an independent risk factor for myocardial infarction patients with positive family history. Thus elevated levels of Lp(a) may serve as an important criterion to identify the individuals who need to undergo Lp(a) lowering treatment, thereby preventing them from myocardial infarction.

ACKNOWLEDGEMENT

The authors are grateful to the management of Lady Doak College for providing the financial support to conduct this study.

Fig - 1
Lp(a) & HDL levels in the study & control population

Table 1
Lp(a) and lipid levels in the study and control population

Parameters mg/dl	MI patients n = 65	Controls n =50
Lp(a) (mg/dl)	58.6 ± 3.20*	19.6 ± 0.10*
Total cholesterol (mg/dl)	199 ± 2.00	152 ± 1.80
LDL (mg/dl)	130 ± 4.28	84.4 ± 3.00
TGL (mg/dl)	155 ± 5.26	138 ± 4.20
HDL (mg/dl)	32.6 ± 0.30	40.4 ± 0.39
VLDL (mg/dl)	31 ± 0.90	27.6 ± 0.77
TC/HDL	6.0	3.8

* p < 0.05 – statistically significant and all values are expressed as **Mean±S.E.**

Fig - 2
Ratio of total cholesterol & HDL in patients & control

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