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Lipid Profile in Myocardial Infarction – Controversies Revisited

Medical Science

KEYWORDS : Acute Myocardial Infarction, Lipid profile, Dyslipidemia

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ABSTRACT

INTRODUCTION : Myocardial Infarction(MI) is the most common form of heart disease and the single most important cause of death in both developed and developing countries. Risk factors for MI have been extensively studied and various studies are being conducted. MI is due to irreversible necrosis of the heart muscle secondary to prolonged ischemia.

AIM IF THE STUDY : The objective of this study was to evaluate the changes in different fractions of lipid profile in Indian subjects with Acute Myocardial Infarction. During tissue necrosis, acute phasic changes occur that alter the lipid profile levels.

MATERIALS AND METHODS : We compared various lipid parameters like Total Cholesterol (TC), Triglycerides (TG), Low Density Lipoprotein (LDL), High Density Lipoprotein (HDL) taken within 24 hours of admission in 25 Acute Myocardial Infarction patients with 25 healthy control subjects.

RESULTS : Concentration of TC, TG and LDL were found to be significantly increased in AMI patients with a mean value 299mgs/dl, 208mgs/dl and 220 mgs/dl respectively while it was within normal limits with a mean concentration of 184mgs/dl,133mgs/dl and 120mgs/dl in the control group. Concentration of HDL was not significantly altered in MI (37mgs/dl)when compared with control(37mgs/dl).

CONCLUSION : Identification of dyslipidemia and early treatment following myocardial infarction provide potential benefits and thus reduces the morbidity and mortality of Coronary Heart Disease (CHD). Hence it is recommended that lipid profile should be assessed within 24 hours after infarction as an exact knowledge regarding baseline serum lipids can be used to guide selection of lipid lowering medications.

Introduction

Myocardial Infarction is one of the leading cause for morbidity and mortality worldwide. The pathogenesis of AMI is multifactorial and studies have proved that impaired lipid metabolism is one of the crucial factors in the development of this disease(1). Epidemiological studies have shown that there are high levels of TC and LDL and low levels of HDL in CHD patients(2). It is a well-known fact that early treatment of hyperlipidemia following AMI provides potential benefits. During tissue necrosis, as seen in MI, alteration in lipid profile is encountered. Opinion is divided regarding the changes that occur in lipid parameters following MI. Most workers have reported a reduction in TC,HDL and LDL while others have reported no changes in TC or HDL after AMI.(3,4,5,6).

The oxidative modification of LDL occurs through the release of proinflammatory and oxidative signals and is considered to be a strong risk factor for atherosclerosis and hence MI. TG levels is an important and independent risk factor for MI(7). Increased serum levels of HDL fractions are associated with decreased risk of developing atherosclerotic heart disease. HDLparticles are antiatherogenic and participate in reverse cholesterol transport. They have anti-apoptotic, anti-inflammatory and anti-thrombotic properties. Primary prevention strategies should focus on treatment modalities that increase HDL concentration (8).

Higher levels of TC and LDL and low levels of HDL during the first 24 hours of AMI was found to have a strong negative prognostic value (9).

In recent years intensive lowering of LDL using statins and HDL targeted therapy have been established as an effective therapy to lower cardiovascular risks(8).

Materials and Methods

This study was carried out in 25 patients aged between 40-70 years who were admitted to Coronary Care Unit, Government General Hospital, Chennai, within 24 hours of development of infarction. None of these patients had any liver, kidney or thyroid dysfunction. Control group included 25 age and gender matched subjects without any comorbid disease and medications. Diagnosis of AMI was established by clinical examination, ECG and by estimating cardiac enzymes. Venous blood was collected from all the subjects after overnight fasting. Serum lipid profile were estimated in both the groups using standard enzymatic methods. Ethical measures were taken and the subjects were informed about the procedure in detail and a written consent from the patients was also taken before starting the study.

Estimation of lipid profile

Serum Total Cholesterol-CHOD-POD (Cholesterol oxidase peroxidase) method .
Serum Triglycerides-GPO (Glycerol phosphate oxidase) method .
Serum HDL cholesterol –Precipitation method .
Serum LDL cholesterol using Friedwald formula.

To find out the changes in lipid parameters in AMI patients, the mean values obtained for the biochemical parameters for AMI were compared with that of the control group. The statistical significance of the variation in the level of the parameters is arrived at from the p value which is obtained from student's t test. (Table I)

RESULTS

Scrutinizing the results obtained in the above condition taken up for study, the reference range for the different fractions of lipid profile obtained in the study is found to be valid and acceptable. The mean concentration of Total Cholesterol, Triglycerides and LDL were 299.0±19.14, 208.5±16.35 and 220.4±19.34 respectively in AMI patients against 184.33±24.96, 133.35±20.01 and 120.45 ±23.36 respectively in control group. The difference between the mean concentration of the above parameters between the two groups was found to be statistically highly significant ($p<0.001$). HDL concentration in AMI patients (37.10 ± 5.69) did not show any statistical significance against control group (37.25 ± 4.93).

DISCUSSION

In our patients with MI there was increase in the concentration of TC,TG and LDL. The increased cholesterol may be due to profound up regulation of cholesterol synthesis as acute phase response. Studies have shown decrease in TC after AMI which can be explained as due to increased activity of LDL receptor and increased cholesterol catabolism. It may be also due to increase in cellular uptake for tissue repair and hormonal synthesis (10).

TG level was increased in our study .The probable explanation is, after MI adrenergic mediated lipolysis of the adipocytes occurs resulting in increased mobilization of free fatty acids and impaired removal of VLDL from the plasma resulting in increased TG. Few studies have reported a fall in TG after MI(11) but other studies have found no significant alteration (12).

LDL was found to be increased in our subjects. A significant decrease in LDL has been reported following MI by many authors in their studies.(13)

There is no significant alteration in HDL concentration in our study while some studies have shown that there is decrease in HDLconcentration during infarction. Decrease in HDL may be secondary to inflammation due to increased activity of endothelial lipase and phospolipase A2.

CONCLUSION

From these reports its understood that phasic changes do occur in patients following MI and therefore there is a strong recommendation for detection of hyperlipidemia in patients with acute MI. The changes in lipid parameters may be related to dietary modifications or due to metabolic changes during acute crisis. Despite these, changes in lipid parameters at hospital level provides valuable information, especially if levels are assessed within 24 hours of presentation of the symptoms. While dietary and lifestyle modification seems appropriate for those who meet the NCEP(National Cholesterol Education Programme) criteria pharmacological intervention can be started immediately. For those with border line elevation repeat value should be obtained several weeks after discharge.

Table 1.Comparision of the levels of lipid profile in control and AMI

		Serum Triglyceride (mg/dl)	Serum Cholesterol (mg/dl)	Serum HDL (mg/dl)	SerumLDL (mg/dl)
Control	Mean	133.35	184.325	37.25	120.45
	±SD	20.077	24.955	4.929	23.355
AMI	Mean	208.5	299.0	37.10	220.4
	±SD	16.35	19.24	5.69	19.64
P value		<0.001	<0.001	>0.05	< 0.001
Significance		Highly Significant	Highly Significant	Non Significant	Highly Significant

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