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LIPID PROFILE LEVELS ON THE SECOND DAY OF ACUTE MYOCARDIAL INFARCTION.

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ABSTRACT

The lipid profile levels were done in clinical side because of its strong association for assessing the risk for coronary artery diseases. The main objective of the study is to note the changes that occur in the lipid profile levels following an acute ischemic attack and also correlate the changes in the lipid profile levels to the ischemic markers (Cardiac Troponin-I and Aspartate transaminase).The study includes two groups; the first group consists of 50 patients who were admitted to the hospital with AMI. The second group patients are normal healthy controls. Serum levels of Cardiac troponin-I (cTnI) and Aspartate transaminase (AST) were assessed immediately and 12hrs fasting blood drawn on next day for assessing the lipid profile levels. The patients with AMI had shown significant rise in cTnI and AST and the lipid parameters like High density lipoprotein (HDL) had shown significant fall and the Very Low Density Lipoprotein (VLDL) and triglycerides (TG) had shown significant rise in cases compared to normal healthy individuals. TC and LDL in cases had shown fall compare to controls but not significant statistically. The cTnI showed a strong negative correlation with fall in the TC and HDL and LDL. The AST showed significant negative correlation with TC and LDL only. So routine diagnosis of lipid parameter for assessing the risk in clinical side should be reliably assessed within 24 hours and the lipid parameters assessed after 24 hours is invalid for the risk assessment for the patients with AMI.

KEY WORDS; **Acute Myocardial Infarction, Cardiac Troponin-I, Coronary Artery Diseases, Ischemic Heart Diseases, Lipid Profile**

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INTRODUCTION

Coronary artery disease (CAD) is one of the leading causes for the developing of the ischemic heart diseases. However CAD remains the most common cause of death despite significant advancements in the prevention and treatment. Several risk factors (smoking, hypertension, diabetes, obesity etc.) play a main role for developing the CAD[1, 2]. Due to the high levels of TC, LDL and TG and low levels HDL are the cause for deposition of lipid in arteries and causing atherosclerosis. So, lipid profile levels are routinely assessed for the risk assessment for preventing CAD[3].

The oxidation of LDL cholesterol is considered as the most important risk factor for CAD, which plays a central role in atherogenesis[4]. Macrophages uptake of modified LDL results in the formation of foam cells, the hallmark cells of atherosclerosis. The foam cells and macrophages release growth factors and metalloproteinases that lead to cell proliferation, extracellular matrix degeneration, atherosclerotic plaque instability and plaque rupture, the cause for myocardial infarction[5]. Myocardial ischemia results from the reduction of coronary flow to myocardial tissue in such an extent that supply of oxygen to the myocardium doesn't meet the oxygen demand. When this ischemia is prolonged and irreversible then myocardial cell death and necrosis occurs[6]. Due to the tissue injury various local and systemic reaction like vasodilatation, leukocyte infiltration and chemotasis occur and monocytes and macrophages activation and cytokines are released. So acute phasic changes occurs that alter the lipid profile levels in post MI. So, validity of plasma lipids measured beyond 24 hours from the onset of MI has been questioned by many studies[3].

However there are several diagnostic markers for diagnosing AMI, but early and accurate diagnosis is essential in the management of AMI. Sensitive and specific laboratory markers are very important in this situation. Due to the ischemia the myocardial cells gets damaged. The troponins are released from the damaged myocardial cells so that the cardiac troponins (cTns) appear in the blood as early as 3-4 hours of acute episode and remain elevated for 4-14 days. When the myocardial cells are damaged the cytosolic troponins reaches the blood stream quickly resulting in a rapid peak of serum troponin levels in during the first few hours. The cTnI, one of the sensitive and specific markers of myocardial injury can be used for diagnosing AMI easily and accurately[6, 7]. The levels of cTnI levels in plasma provoke degree of infarct size of the myocardial damage due to ischemia. The study is to evaluate the changes in lipid profile levels during AMI and correlating the changes in lipid parameters to the cTnI levels and finally to note the post ischemic effect of AMI on the lipid parameters.

MATERIALS AND METHODS

The study was carried out with the patients admitted with symptoms of cardiac ischemia and chest pain to Coronary Care Unit (CCU), NRI Heart Research Centre at NRI General Hospital. The study consists of two groups. The first group (n=50) consist of patients with ischemic attack and symptoms of chest pain were taken as cases. Whereas the second group (n=50) consists of normal healthy individuals taken as controls. Basic information like Age, Weight, Risk factors, life style etc., was taken from the individuals by consent forms.

The patients with End stage renal diseases, Sepsis, Pericarditis, Pulmonary embolism were excluded from the study. Patients satisfying all the inclusion are included in the study. The blood samples were drawn after the patient admitted to the CCU and centrifuged and serum was separated and used for estimation of cTnI and AST. Fasting blood samples were drawn from the same patients after 12 hrs fasting for the estimation of lipid parameters like TC, HDL and TG.

The cTnI is estimated quantitatively using Advia Centaur CP a fully automated hormone analyser (Chemiluminescence technique) and the AST and lipid parameters (TC, HDL and TG) were estimated quantitatively using Dade Behring Dimension a fully automated chemistry analyser (Bayers technology). The LDL and VLDL values were derived indirectly using Friedwalds equation. Prior to the estimation of samples all the autoanalyzers were calibrated and the controls value limits of ±2SD is used for the

samples estimation. All the results were tabulated and statistics were done using SPSS Software 17.0 and Pearson correlation was used for correlating different parameters. The p-value of <0.05 was considered to be statistically significant.

RESULTS

Table 1
Mean age, number of males and females of cases and controls.

Parameters	Cases Mean ±SD (n=50)		Controls Mean ±SD (n=50)	
Age yrs	54.76±13.8		51.42±13.85	
Number of males and females	Male	41	Male	30
	Female	09	Female	20

All the samples drawn on second day of post MI were used for the study. It is evident from the table 2 that cTnI showed a highly significant increase (p <0.001) in cases compared to controls, as it is the good marker of AMI, similarly AST also showed a highly significant increase (p <0.001) in AMI patients. The TC and LDL levels had shown slight decrease in cases compared to controls but not significant statistically (p< 0.05). Whereas HDL showed significant decrease (p <0.001) and the VLDL and TG showed a significant increase (p <0.001) in cases compared to controls .

Table 2
Mean±SD and p value of different parameters of controls and cases

Parameters	Cases Mean ±SD (n=50)	Controls Mean ±SD (n=50)	P value
CTnI ng/ml	53.95±15.2	0.037±0.25	<0.001
AST U/L	290.4±122.5	25.5±6.21	< 0.001
TC mg/dl	157.9±38.42	166.2±25.8	0.205
HDL mg/dl	35.38±10.20	43.08±6.35	< 0.001
LDL mg/dl	98.6±33.45	100.28±24.47	0.776
VLDL mg/dl	24.39±10.21	20.29±6.20	0.009
TG mg/dl	121.86±50.9	99.80±26.9	0.007

Correlation study (table 3) has revealed significant positive correlation between cTnI and AST (r= 0.542, p <0.001) and cTnI significant negative correlation with TC, HDL and LDL (r= -0.638, p < 0.001; r= -0.283, p 0.043; r= -0.570, p < 0.001). Similarly the AST also showed significant negative correlation with the TC and LDL (r= -0.498, p <0.001; r= -0.483, p <0.001) only. The correlation of TC and AST to cTnI had shown graphically in graph 1 and 2.

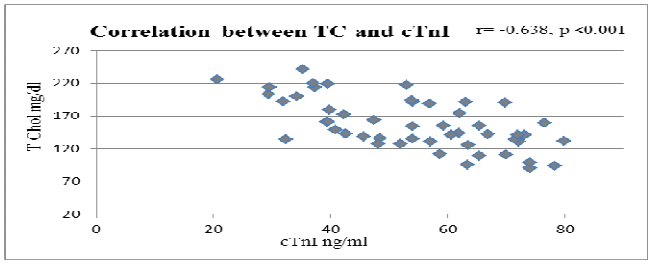
Table 3
Pearson correlation (r) values for Troponin I and AST with Total Cholesterol, HDL, LDL, VLDL and Triglycerides (**p<0.001, *p<0.05)

Parameters	AST U/L	TC mg/dl	HDL mg/dl	LDL mg/dl	VLDL mg/dl	TG mg/dl
cTnI ng/ml	0.542**	-0.638**	-0.287*	-0.570**	-0.180	-0.181
AST U/L	1	-0.498**	-0.215	-0.483**	-0.045	-0.044

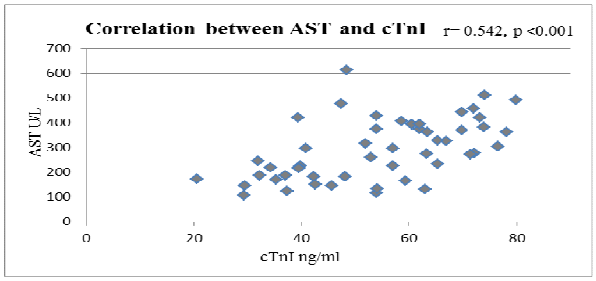
DISCUSSION

The root cause of AMI is mainly atherosclerosis. Contrary to earlier belief, research in the last two decades has shown that atherosclerosis is neither a degenerative disease nor inevitable due to ageing. On contrary atherosclerosis seems to be a chronically inflammatory condition that is converted to an acute clinical event by induction of plaque rupture which in turn leads to thrombosis. Hence inflammatory occupies a very important central position in all phases of atherosclerosis, although inflammation must smoulder for decades before resulting it into a clinical event like AMI[8, 9].

Graph 1
Correlation between Total cholesterol to Cardiac troponin-I



Graph 2
Correlation between Aspartate transaminase and Cardiac troponin-I



The lipid abnormality is one important risk factor for IHD. There are number of risk factors which influence the formation of plaques due to excess cholesterol. The plaque that are deposited on the walls of the blood vessels will narrow the blood flow to the heart muscle and causes ischemia[10]. Due to the ischemia the myocardial cells gets damaged. The troponins are released from the damaged myocardial cells so that the cardiac troponins (cTns) reaches the blood stream quickly resulting in a rapid peak of serum troponin levels in during the first few hours[6, 7]. The cTnI is more specific for myocardial injury compared to cTnT[6, 7]. The cTnI had showed highly significant rise in our study with about thousand fold rise in the serum of patients with AMI compared to controls. The AST is another traditional cardiac enzyme which has been used as one of the marker for AMI. But the specificity is poor with lot of false positive elevation (Skeletal injury, Liver damage, pulmonary embolism etc.). Since it offers no traditional benefits for the diagnosis of AMI, it is no longer used as routine test[7]. However the AST levels showed a significant rise in cases compared to controls with about 10 fold rises. The correlation study showed significant positive correlation between AST and cTnI ($r = 0.542$, $p < 0.001$). The false positive rise of AST in the patients was avoided by excluding the patients with skeletal injury, liver damage, pulmonary embolism etc.

Timely routine diagnosis of lipid parameters should be checked for assessing the risk for atherosclerosis for the treatment. The decreased HDL and rise in LDL is the main cause for atherosclerosis[11].

However there are several risk factors that enhance atherosclerosis. So lipid

parameters should be checked for the risk assessment and its management. But the diagnosis of lipid parameters in AMI patients in our study assessed on the second day has showed a decrease in serum levels of TC, HDL and LDL and rise in VLDL and TG levels. The TC and LDL are decreased in the cases compared to controls, but not significantly (table 2). But the correlation study has showed significant negative correlation TC and LDL with cTnI and AST (table 3), which indicates that the decrease in cholesterol is indirectly proportional to the ischemic attack, Where the cTnI and AST are the markers of Ischemic attack. Whereas HDL showed a significant decrease in the in cases compared to controls and correlation study also showed significant negative correlation with cTnI only. The decrease in HDL levels is indirectly proportional to the cTnI which indicates the post ischemic attack effect.

Thus our study noted a significant alteration in serum HDL, VLDL and TG after AMI. However, we did not find significant changes in serum TC and LDL levels in our study, which goes similar to the studies of P.K Nigam et al[4] and Narrapol Wattansuwan et al[3]. The TC and LDL showed slight decrease in cases compared to healthy controls but not significantly. Statistically it has proven that the decrease was not significant, but clinically we suggest it was a decrease; because all the patients hospitalized with AMI were previously having at least one risk factor which provoked lipid profile levels , with high TC, LDL, TG and low HDL levels compared to controls. Due to the ischemic attack the TC and LDL has decreased on the second day of AMI in cases and was parallelised with the values of controls. When the decrease in the TC and LDL in cases was compared to normal healthy controls it has noted as not significant, statically. But clinically when we note the changes in the same patients with AMI before and after to MI we can note the statistically decrease in TC and LDL levels. That is the limitation of our study. However studies of M.Swedarsen[12] had showed significant decrease in the levels of TC from the day-1 of MI to day-2 in the same patients.

However there are many studies in the past few decades which shows, that AMI results in a significant decrease in TC, HDL and LDL. So the acceptable time for the measurement of plasma lipids after an AMI is within 24 hours after the onset of symptoms, and the plasma lipid levels measured beyond 24 hours are mostly considered to be invalid[3, 4, and 11]. The changes in the lipid parameters following AMI are due to acute phase response. AMI likely any other tissue injury, initiates various local and systemic reactions. The cytokines act on the systemic targets, including liver, to generate changes in the concentration of various heterogeneous plasma proteins that are known collectively as acute phase reactants, including lipoproteins and C-reactive proteins. By day 4 to 5 of post MI there is a significant decrease in the serum concentration of apoprotein A-I and apoprotein B, reflecting the maximum decrease in serum TC, HDL, and LDL[3]. Studies of F.D Hollanders states, increased catecholamine secretion after MI may be implicated in the serum lipid alteration[14].

CONCLUSION

In view of accumulating evidence that treatment of hypercholesterolemia may reduce mortality and morbidity after myocardial infarction, and it appears advantageous to screen and counsel hyperlipidaemia patients as soon as possible after an AMI at a time when patients and their families are most impressionable. Though due to acute phase response followed by AMI, the lipids are reliably assessed within 24 hours after AMI and lipids assessed from second day of AMI are not reliable due to the changes occurring by acute phase response. So we conclude that lipid parameters to assess within the first day of AMI and lipids assessed after first day is invalid for the risk assessment. Most of the authors also suggested to assess the lipid parameters on 1st day of AMI and not to assess from second day to third month of post MI[1, 3, 12.]

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