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Brief Communication

Lipid Profile levels on the second day of Acute Myocardial Infarction; is it the right time for estimation?

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ABSTRACT: The main objective of this study is to note the changes that occur in the lipid profile levels following an acute myocardial ischemic attack and also correlate the changes in the lipid profile levels to the ischemic markers (Cardiac troponin-I and AST). The study included two groups; the first group consisted of 50 patients who were admitted to the hospital with Acute Myocardial Infarction (AMI). The second group patients were normal healthy controls. Serum levels of Cardiac troponin-I (cTnI) and AST were assessed immediately and 12hrs fasting blood drawn 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 decrease and the Very Low Density Lipoprotein (VLDL) and triglycerides (TG) had shown significant increase in cases compared to normal healthy individuals. Total Cholesterol (TC) and Low Density Lipoprotein (LDL) in cases had shown decrease compare to controls but not significant statistically. The cTnI showed a significant negative correlation with decrease 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 clinical risk should be reliably assessed within 24 hours and the lipid parameters assessed after 24 hours are invalid for risk assessment for patients with AMI.

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

INTRODUCTION

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

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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 to such an extent that supply of oxygen to the myocardium does not meet 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 reactions like vasodilatation, leukocyte infiltration and

chemostasis occur and monocytes and macrophages activation and cytokines are released. So acute phasic changes occur that alter the lipid profile levels post MI. Therefore the validity of plasma lipids measured beyond 24 hours from the onset of MI has been questioned by many studies[3].
There are several 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 get damaged. The troponins are released from the damaged myocardial cells so that cardiac troponins (cTns) appear in the blood as early as 3-4 hours of an acute episode and remain elevated for 4-14 days. When myocardial cells are damaged the cytosolic troponins reach the blood stream quickly resulting in a rapid peak of serum troponin levels during the first few hours. The cTnI, a sensitive and specific marker 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.

METHODOLOGY

The study was carried out on 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 consisted of two groups. Group-I (n=50), consisting of patients with ischemic attack and symptoms of chest pain were taken as cases. and Group-II (n=50), consisting of normal healthy individuals were taken as controls. Basic information like age, weight, risk factors, lifestyle etc., was recorded from the individuals according to a questionnaire. Blood samples were collected from the patients after obtaining written consent.
Patients with end stage renal disease, sepsis, pericarditis, pulmonary embolism etc were excluded from the study. Patients with cardiac ischemia with ECG showing ST segment elevation were included in the study. After the patient was admitted to the CCU, blood samples were drawn and centrifuged and serum was separated and used for estimation of cTnI and AST. 12 hours fasting blood samples were drawn from the patients to estimate lipid parameters like TC, HDL and TG. All the samples drawn on second day of post MI were used for the study.

The cTnI was estimated quantitatively using Advia Centaur CP a fully automated hormone analyser (Chemiluminescence technique) and the AST and lipid parameters 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 values limits of ±2SD was used for the samples estimation. All the results were tabulated and statistical analysis was 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

Age and sex distribution of the study subjects of both groups are presented in **Table 1**.

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

It is evident from **Table 2** that cTnI showed a highly significant increase (p <0.001) in cases compared to controls, as it is a good marker of AMI, similarly AST also showed a highly significant increase (p <0.001) in AMI patients. The TC and LDL levels showed slight decrease in cases compared to controls but this was not statistically significant (p >0.05). HDL showed significant decrease (p <0.001) and the VLDL and TG showed a significant increase (p <0.001) in cases compared to controls.
Correlation study (**Table 3**) 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= -0.287, 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.

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

Parameters	Cases (n=50) Mean ±SD	Controls (n=50) Mean ±SD	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

Table 3: Pearson correlation (r) values for MDA and Vitamin-C with different parameters

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

*(**p<0.001; *p<0.05)*

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 the contrary, atherosclerosis seems to be a chronic inflammatory condition that is converted to an acute clinical event by induction of plaque rupture which in turn leads to thrombosis. Hence inflammation occupies a very important central position in all phases of atherosclerosis, although inflammation must smoulder for decades before resulting in a clinical event like AMI[8, 9].

The lipid abnormality is one important risk factor for Ischemic heart disease. There are a number of risk factors which influence the formation of plaques due to excess cholesterol. The plaques that are deposited on the walls of the blood vessels reduce blood flow to the heart muscle and cause ischemia[10]. Due to the ischemia the myocardial cells are damaged. The troponins are released from the damaged myocardial cells and the cardiac troponins (cTns) reach the blood stream quickly resulting in a rapid peak of serum troponin levels in the first few hours[6, 7]. The cTnI is more specific for myocardial injury compared to cTnT[6, 7]. The cTnI had shown highly significant rise in our study with about a 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 markers for AMI. But the specificity is poor with a lot of false positive elevations (Skeletal injury, Liver damage, pulmonary embolism etc.). Since it offers no traditional benefits for the diagnosis of AMI, it is no longer used as a routine test[7]. However the AST levels showed a significant rise in cases compared to controls with about 10-fold rise. 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, and pulmonary embolism.

Decreased HDL and rise in TC and LDL is the main cause for atherosclerosis[11]. However there are several risk factors that enhance atherosclerosis, so lipid parameters should be checked for 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 of TC and LDL with cTnI and AST (**Table 3**), which indicates that the decrease in cholesterol is indirectly proportional to the ischemic attack. HDL showed a significant decrease 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 is similar to the studies of Nigam et al[4] and Wattansuwan et al[3].

The TC and LDL showed slight decrease in cases compared to healthy controls but not significantly. Statistically the decrease was not significant, but we suggest that it was a decrease because all the patients hospitalized with AMI previously had at least one risk factor which provokes lipid profile levels (high TC, LDL, TG and low HDL levels). 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, and when cases were compared to normal healthy controls it was noted as not statistically significant,. But clinically when we note the changes in the same patients with AMI before and after MI, we can note the statistical decrease in TC and LDL levels. That is the limitation of our study. However studies of Swedarsen et al[12] have showed significant decrease in the levels of TC from the day-1 of MI to day-2 in the same patients.

However, there have been many studies in the past few decades which show 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[3,4,11]. The changes in the lipid parameters following AMI are due to acute phase response. AMI, like any other tissue injury, initiates various local and systemic reactions. The cytokines act on systemic targets, including the 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 after 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]. Kenneth et al state that acute phase response induces marked changes in lipid metabolism like increased plasma triglyceride levels and decreased HDL levels[14]. Hollanders et al state that increased catecholamine secretion after MI may be implicated in the serum lipid alteration[15].

REFERENCES

1. Zorio E, Murado J, Arizo D, et al. Haemorheological parameters in young patients with acute myocardial infarction. *Clin Hemorheol Microcir*. 2008;39(1-4):33-41.
2. Kumar A, Sivakanesan R, Nagtilak S. Serum paraoxonase activity in normolipidaemic patients with acute myocardial infarction. *J Clin Diagn Res*. 2008;2:1052-6.
3. Wattanasuwan N, Khan IA, Gowda RM, et al. Effect of acute myocardial infarction on cholesterol ratios. *Chest*. 2001 Oct;120(4):1196-9.
4. Nigam PK, Narain VS, Hasan M. Serum lipid profile in patients with acute myocardial infarction. *Indian J Clin Biochem*. 2004;19:67-70.
5. Barter P. Is high-density lipoprotein the protector of the cardiovascular system? *Eur Heart J Suppl*. 2004;6:19-22.
6. Nigam PK. Biochemical markers of myocardial injury. *Indian J Clin Biochem*. 2007;22:10-17.
7. Chan HC, Woo KS. New diagnostic markers for myocardial infarction. *JHK Cardiol*. 1997 Oct;5:140-5.
8. Patil N, Chavan V, Karnik ND. Antioxidant status in patients with acute myocardial infarction. *Indian J Clin Biochem*. 2007;22:45-51.
9. Libby P. Vascular biology of atherosclerosis: overview and state of art. *Am J Cardiol*. 2003 Feb;91(3A):3A-6A.
10. Beegam KAS, Sasikala K, Meenakshi N, et al. Ischemic Heart Disease - A haematological, biochemical and cytogenetic study. *Int J Hum Genet*. 2003;3(3):159-63.
11. De Silva LD, Kumar A, Sathian B. The significance of lipid profile and positive troponin in predicting cardiac event. *Kathmandu Univ Med J (KUMJ)*. 2009 Jul-Sep;7(27):263-7.
12. Sewdarsen M, Vythilingum S, Jialal I, et al. Plasma lipids can be reliably assessed within 24 hours after acute myocardial infarction. *Postgrad Med J*. 1988 May;64(751):352-6.
13. Ryder RE, Hayes TM, Mulligan IP, et al. How soon after myocardial infarction should plasma lipid values be assessed? *Br Med J (Clin Res Ed)*. 1984 Dec;289(6459):1651-3.
14. Feingold KR, Grunfeld C. The acute phase response inhibits reverse cholesterol transport. *J Lipid Res*. 2010 Apr;51(4):682-4.
15. Hollanders FD, Shafar J, Burton P. Serum lipid changes following the completed stroke syndrome. *Postgrad Med J*. 1975 Jun;51(596):386-9.