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Comparative study of lipid profile, Cpk-Mb and microalbuminuria in patients with myocardial infarction in Rohilkhand region

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Abstract
Background: Myocardial Infarction is the "impairment of heart function due to inadequate blood flow to the heart compared to its need caused by obstructive changes in the coronary circulation in the heart". The present study was undertaken to measure the level of microalbumin, lipid profile and CPK-MB parameters in acute myocardial infarction patients, to know the relationship between MA, lipid profile and CPK-MB parameters and to compare their levels with those of healthy controls.
Aim: 1) To determine and compare the level of microalbuminuria and lipid profile indiabetic and non-diabetic AMI patients and healthy controls.
2) To determine the serum levels of CPK-MB in patients with AMI.
3) To correlate the levels of microalbuminuria with degree of dyslipidemia and cardiac biomarkers in patients with AMI.
Material and Method: This was a prospective case control study. Hundred diagnosed cases of AMI ranging from 35 to 65 years were included in the study. The cases were further sub divided in two groups of 50 each. One group had 50 AMI patients with diabetes mellitus and another had 50 AMI without DM. Hundred age and gender matched apparently healthy controls were taken.
Result: 200 patients were taken out of which 100 were cases of acute MI and 100 controls. Elevated lipid profile, CPK MB increases the risk of MI and there is also increase in microalbuminuria in DM patients.
Conclusion: Elevated lipid profile, CPK MB increases the risk of MI and there is also increase in microalbuminuria in DM patients.

Keywords: acute myocardial infarction, diabetes mellitus, microalbuminuria, CPK MB, lipid profile

Introduction
Microalbuminuria is considered a risk marker of CHD in diabetics and non-diabetics. Patients with microalbuminuria and concomitant diabetes have higher rate of mortality due to chronic heart disease. Lipid profile are routinely measured for risk assessment in preventing CAD because high level of Total Cholesterol (TC), Low Density Lipoproteins (LDL), Triglycerides (TG) and low level of High Density Lipoproteins(HDL) cause deposition of lipid in arteries thus causing atherosclerosis. The oxidation of LDL cholesterol is considered as the most important risk factor for CAD, which plays a central role in atherogenesis. The gold standard for diagnosis of MI has been an elevated level of creatinine phosphokinase myocardial band (CPK-MB), the cardiac specific isoenzymes of CPK. The present study was undertaken to measure the level of microalbumin, lipid profile and CPK-MB parameters in acute myocardial infarction patients, to know the relationship between MA, lipid profile and CPK-MB parameters and to compare their levels with those of healthy controls.

Discussion
Coronary Artery Disease which frequently manifests as Myocardial Infarction continues to exert an enormous toll in western society and also in developing countries like India. Despite progress in its measures in prevention, detection and treatment, it continues to be the leading cause of death. Research over last decade proved the role of inflammation in pathophysiology of atherosclerosis. Inflammation is an important contributor to atherosclerosis, both accelerating the process and precipitating acute plaque rupture.Markers such as TROP-T, CPK-MB are used to diagnose AMI.

Cases were diagnosed based on the diagnostic criteria of AMI i.e symptoms of AMI, changes in ECG and elevated TROP- T levels.

Once a case was diagnosed as AMI. Serum and urine sample were collected. This was considered as 0 hour sample. Following this, the serum samples were collected at 12, 24, 48 and 72 hours from the time of admission. Similarly urine samples were collected at 0, 24, 48, 72 hours.

The cases were divided 2 groups based on presence of DM along with AMI. One group of cases included AMI with DM another AMI without DM. Both these groups were compared with normal healthy controls. O Metwalli [1] in his study involving 58 patients and 30 controls found significant increase in TG levels. They suggested increased TG level may be due to genetic/nutritional /inherited/ acquired abnormalities of lipoprotein especially VLDL which causes alteration in TG levels. We also observed that the serum TG levels was higher in diabetic AMI patients when compared with non diabetic AMI, which was significantly higher (p= 0.021).

In our study, we found significant decrease in HDL levels in both diabetic and non diabetic AMI cases as compared to controls. Results were in accordance with Framingham Study [2], Palanisamy [3] and Suman Sharma [4]. Palanisamyand Suman Sharma found significant decrease in HDL levels along with increase in TC, TG and LDL in patients with acute MI.

The diagnostic criteria for AMI includes classical symptoms of ischemia, changes in ECG and detection of cardiac biomarkers. CPK-MB is a very popular cardiac marker used to diagnose AMI. CPK is an isoenzyme consisting of three isoenzymes. CK-MB is mainly found in the cardiac muscle, where more than 20% ofthe total CK activity is present as CK-MB, and nearly 80% as CK-MM.

Ellis *et al* [5] reported that increases in plasma levels usually occur between 6 to10 hours after the onset of infarction (in the absence of thrombolysis), peaks at 24 hours and return to normal by 36 to 72 hours.

Diabetic patients with AMI showed elevated levels of CPK-MB in comparison to non diabetic AMI. Microalbuminuria is considered to be a strong and independent indicator for Cardiovascular risk. Various studied have shown that patients with CAD had increased excretion of microalbumin in their urine. This is mainly attributed to the associated hypertension in these patients.

The results of our study indicate that there is highly significant microalbuminuria in non diabetic and diabetic acute myocardial infarction patients. The level of significance (p<0.001), of microalbuminuria in our study, was comparable to that observed, in other international studies [6, 7].

Results

The study included a total of 200 subjects out of which 100 were cases of acute myocardial infarction and another 100 were apparently healthy controls. The cases were further sub divided into two groups of 50 each. One group of cases had AMI with diabetes mellitus and another group had AMI without diabetes mellitus.

Table 1: A table showing the distribution of males and females in cases and controls

	Cases		Controls	Total
	AMI with DM	AMI without DM		
Male	44	42	86	172
Female	06	08	14	28
Total	50	50	100	200

Table 2: Level of significance (p value) of the mean of various parameter among cases and controls

	Controls vs AMI with DM	Controls vs AMI without DM	AMI with DM vs AMI without DM
Random Plasma Glucose (mg/dL)	< 0.001	Not significant	0.001
Total Cholesterol (mg/dL)	< 0.001	< 0.001	Not Significant
SerumTriglyceride (mg/dL)	< 0.001	< 0.001	0.021
HDL-C (mg/dL)	< 0.001	< 0.001	< 0.001
LDL-C (mg/dL)	< 0.001	< 0.001	Not significant

Table 3: CPK-MB levels at various intervals with respect to time of admission in patients with acute myocardial infarction with diabetes mellitus and acute myocardial infarction without diabetes mellitus

CPK-MB	Cases		P Value
	AMI with DM (n = 50)	AMI without DM (n = 50)	
At 0 hrs (IU/L)	78.08 ± 68.27	96.32 ± 49.66	Not Significant
At 12 hrs (IU/L)	104.12 ± 63.16	117.42 ± 66.28	Not Significant
At 24 hrs (IU/L)	97.94 ± 57.07	124.94 ± 83.48	Not Significant
At 48 hrs (IU/L)	59.18 ± 31.99	62.36 ± 32.97	Not Significant
At 72 hrs (IU/L)	24.40 ± 16.14	23.54 ± 17.23	Not Significant

Table 4: Urinary micro albumin levels in cases and control at the time of admission

	Cases		Controls (n = 100)
	AMI with DM (n = 50)	AMI without DM (n = 50)	
Urinary microalbumin levels at time of admission (mg/L)	96.64 ± 61.23*	96.12 ± 75.85*	11.50 ± 4.08

* p value < 0.001 when compared with controls

Table 5: Urinary microalbumin levels at various intervals with respect to time of admission in patients with acute myocardial infarction with diabetes mellitus and acute myocardial infarction without diabetes mellitus

Urinary Microalbumin Levels	Cases		P Value
	AMI with DM (n = 50)	AMI without DM (n = 50)	
At 0 hrs (mg/L)	96.64 ± 61.23	96.12 ± 75.85	Not Significant
At 24 hrs (mg/L)	88.48 ± 54.47	78.62 ± 64.06	Not Significant
At 48 hrs (mg/L)	73.24 ± 43.28	54.92 ± 55.66	0.05
At 72 hrs (mg/L)	52.16 ± 35.06	31.46 ± 35.81	0.004

Conclusion

Our present study suggests that estimation of lipid profile and CK-MB inpatients with MI will contribute significantly to the risk assessment, prophylaxis andmanagement of Myocardial Infarction. Addition of microalbuminuria to the aboveprofile can improve the ability to detect absolute coronary risk.

In our study we found a significantly high triglyceride indiabetic with AMI patients when compared with the control group and non- diabeticwith AMI patients. We also found significantly low HDL level in non-diabetic with AMI when compared with control group and diabetic with AMI patients.

It is well known and proved by many authors in their studies that CK-MB is elevated in Acute Myocardial Infarction patients. In our study we also found a significantly high CK-MB in cases of both group as compared with the control group.But when CK-MB was compared between both the groups of cases (diabetic as well asnon-diabetic AMI patients) their was no significant changes seen in CK-MB value.

In our study we found a significantly high microalbuminuria in diabetic as well as in nondiabetic, non-hypertensive acute myocardial infarction patients. In the absence of any renal insufficiency microalbuminuria is a non-specific yet highly sensitive marker of myocardial infarction.

However, more studies are required with a large sample size to ascertain whether microalbuminuria can predict in-hospital mortality and its pathophysiology in this clinical setting.

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