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Research Article

Acute Myocardial Infarction

Metabolic Profile of patients with acute Myocardial Infarction

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Background: Acute myocardial infarction (AMI) is a multifactorial disease characterized by profound cardiac metabolic changes. Serum FFA is elevated dramatically during and following AMI. Recently some researchers noted utility of serum FFA as a predictor of complications particularly arrhythmias in patients of AMI. Hence we included estimation of serum FFA along with blood glucose and lipid profile in our diagnosed cases of AMI. The aim of the present study was to characterize demographic features, anthropometric and glucometabolic profile of patients of AMI and compare with controls. **Material and methods:** A total of 50 diagnosed patients of AMI (chest pain, ST elevation, elevated CK-MB values and Troponin T values within 12 hours of onset of pain) were included in the present case control study. Detail clinical, personal history, past medical history, demographic and anthropometric data recorded. To assess metabolic profile, blood glucose, lipid profile and FFA were estimated. **Results:** Cases from AMI group had mean BMI 22.6+/- 2.2 Kg/m2 and waist circumference 83.2+/- 4.6 cm. We observed significant hyperglycemia, hypercholesterolemia, hypertriglyceridemia and raised LDL (mean- 213+/-54 Vs 84+/-13, 220+/-42 Vs 142+/-11, 356+/-102 Vs 112+/-43, and 165 +/- 23 Vs 102+/-13 respectively $p<0.001$) in cases compared to controls. Serum FFA elevated significantly (mean- 3.2mmol/l +/-0.9 Vs 0.8mmol/l+/-0.3, $p<0.001$) with significantly decreased HDL (mean- 31+/-5.6 Vs 54+/-10, $p<0.05$) levels in comparison with control. **Conclusion:** Present study reveals that patients of AMI without previous T2DM, dyslipedemia or other known cardiac risk factors have raised FFA with altered atherogenic lipoprotein pattern.

Keywords: Acute myocardial infarction, Free fatty acids, Lipid profile, Blood glucose

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Introduction

Rising prevalence of AMI due to excess calories intake and sedentary lifestyle has become an important health issue in developing countries. AMI is a multifactorial disease with impaired lipoprotein metabolism playing crucial role in the pathogenesis. It is characterized by profound cardiac metabolic changes. Increased concentration of low density lipoprotein (LDL) and decreased high density lipoprotein are independent risk factors for development of coronary atherosclerotic disease [1]. Routinely serum total cholesterol, triglycerides, LDL and HDL are measured for diagnosis of dyslipedemia.

Circulating free fatty acids (FFA) derived from lipolysis in adipose tissues contribute to insulin resistance and its consequent complications of noncommunicable diseases. Serum FFA is elevated dramatically during and following AMI. But there are few studies in literature reporting relation of FFA in patients of AMI. Recently some researchers noted utility of serum FFA as a predictor of complications particularly arrhythmias in patients of AMI [2, 3]. Hence we included estimation of serum FFA along with blood glucose and lipid profile in our diagnosed cases of AMI.

We attempted to explore biochemical explanations for rising prevalence of AMI. The aim of the present study was to characterize demographic features, anthropometric and glucometabolic profile of patients of AMI and compare with controls.

Material and methods

Present study was conducted in the Department of Biochemistry, Government medical college Aurangabad and participants were enrolled from Intensive Care Unit of the hospital. Study was approved by Institutional Ethical Committee. Informed consents were obtained from the patients and their relatives. A total of 50 diagnosed patients of AMI (chest pain, ST elevation, elevated CK-MB values and Troponin T values within 12 hours of onset of pain) were included in the present case control study. Patients with more than 12 hours of onset of AMI, renal disease, previous cardiovascular disease, thyroid disease, and diabetes mellitus were excluded from the study. Also we excluded the patients receiving lipid lowering agents from the study. 50 healthy, age and sex matched individuals with no cardiovascular diseases were the controls.

Detail clinical history, past medical history and demographic data including personal habits of smoking, alcoholism, family history of CVD in first degree relatives of the participants were recorded on the day of admission. Systolic and diastolic blood pressures were recorded for all subjects. Anthropometric variables were recorded in terms of weight (Kg), Height (Cm), waist and hip circumference (Cm). Body Mass Index (BMI) was calculated as Weight (Kg)/ Height (m2) as a measure of obesity and Waist: Hip ration (W: H) as indicator of visceral adiposity.

After 12 hours fast, 5 ml venous blood samples were collected and after serum separation, various biochemical assays were performed to determine glucometabolic profile. We assayed blood glucose (Glucose Oxidase Peroxidase end point method), serum total cholesterol (Cholesterol Oxidase Peroxidase end point method), triglycerides (Lipase/Glycerokinase/Glycerophosphate oxidase), high density lipoproteins (direct ethodpolyethylene glycolpretreated enzymes), low density lipoproteins (Friedewalds formula) and free fatty acids (kit from Randox diagnostics) on Fully Automated Chemistry Analyzer (Transasia).

The results were statistically processed using nonparametric criteria; differences were considered to be statistically significant if $P < 0.05$. Numerical variables were described as mean and standard deviation.

Results

Table: Demographic, clinical and biochemical features of cases and controls

Variables	Cases (Mean +/- S.D.)	Control (Mean +/- S.D.)
Age (years)	54.6+/- 8.	51.3+/- 5
Sex (M:F)	32 (64%):16 (32%)	34 (68%):16 (32%)
H/O smoking	23 (46%)	8 (16%)*
Family H/o AMI	25 (50%)	5 (10%)*
BMI (Kg/m2)	22.6+/- 2.2	21.4+/-1.4
Waist circumference (cm.)	85.2+/- 4.6	81.7+/- 2.1*
Blood glucose (mg %)	213+/-54	84+/-13*
Total cholesterol (mg %)	220+/-42	142+/-11*
Triglycerides (mg %)	356+/-102	112+/-43*
LDL (mg %)	165 +/- 23	102+/-13*
HDL (mg %)	31+/-5.6	54+/-10*
FFA (mmol/l)	3.2mmol/l +/-0.9	0.8mmol/l+/-0.3*

Fifty diagnosed cases of AMI were included in the present study; 32 (64%) were males and 18 (36%) were females. Mean age of the cases was 54.6+/- 8 years old. Pertinent demographic, clinical and biochemical variables as seen during study are represented in above table.

(*- p< 0.05 statistically significant)

Cases from AMI group had mean BMI 22.6+/- 2.2 Kg/m2 and waist circumference 83.2+/- 4.6 cm. We observed significant hyperglycemia, hypercholesterolemia, hypertriglyceridemia and raised LDL (mean- 213+/-54 Vs 84+/-13, 220+/-42 Vs 142+/-11, 356+/-102 Vs 112+/-43, and 165 +/- 23 Vs 102+/-13 respectively p<0.001) in cases compared to controls. Serum FFA elevated significantly (mean- 3.2mmol/l +/-0.9 Vs 0.8mmol/l+/-0.3, p<0.001) with significantly decreased HDL (mean- 31+/-5.6 Vs 54+/-10, p<0.05) levels in comparison with control. No significant association of biochemical parameters with BMI and waist circumference was observed.

Discussion

AMI has been recognized as an acute metabolic stress condition with atherogenic dyslipedemia. Hence we aimed to characterize metabolic profile of patients with AMI. The study group involved 50 cases of diagnosed AMI and 50 age and sex matched healthy controls. Results of the present study showed significantly different demographic, clinical and biochemical differences in cases and control groups. We evaluated serum FFA levels in addition to routine lipoproteins and blood glucose levels in AMI patients without known major cardiac risk factors. Cases had significant atherogenic dyslipidemia with raised total cholesterol, triglycerides, LDL (mean 220+/-42 Vs 142+/-11, 356+/-102 Vs 112+/-43, and 165 +/- 23 Vs 102+/-13 respectively p<0.001) and hyperglycemia (mean213+/-54 Vs 84+/-13 p< 0.05). FFA levels differ significantly in AMI cases compared to control group (mean- 3.2mmol/l +/-0.9 Vs 0.8mmol/l+/-0.3, p<0.001).

In accordance with previous study reports, our cases of AMI had significantly raised FFA with altered lipoprotein levels and blood glucose. Recent study by Vijay Kumar et al from India demonstrated significantly raised levels of FFA within 24 hours of AMI normalizing at 7 day [2]. Nadhipuram V and associates measured total and specific serum FFA in AMI and observed causal relation between FFA and ischemia modified albumin [3]. Some researchers reported fall in levels of serum, HDL and LDL following AMI with variations in TGs. On the contrary some studies showed no difference in serum TC and HDL values [4-6]. Nigam suggested utility of assessment of lipoproteins within 24 hours of AMI [7]. One study from Iran observed significant rise in FFA in AMI with decreased content of polyunsaturated fatty acids suggesting its protective action in development of coronary atherosclerosis [8].

There is inhibition of B- oxidation of long chain fatty acids in mitochondria during ischemia which results in accumulation of long chain acyl carnitine and acyl coenzyme A [9]. Serum FFA is raised due to compensatory hyperadrenargic state in AMI patients due to pain, anxiety, stress. FFA and glucose are the major sources of energy for heart. Cardiac metabolism is mainly aerobic and most of the ATP is provided by oxidative phosphorylation. Normally heart maintains metabolic homeostasis with a complex cytoplasmic and mitochondrial network regulating ATP production. In well oxygenated heart, 50-70% of energy is derived from FFA and rest from carbohydrates. But during ischemia, this is hampered as decreased supply of oxygen results in decreased oxidation of glucose and FFA [10]. Increased circulating FFA leads to raised consumption of oxygen by myocardium which further worsens ischemic damage [2].

Some researchers also commented that serum FFA is the earliest and more sensitive sign of AMI than the changes of electrocardiography. Results of the prospective Quebec cardiovascular study in men showed strong association of concentration of FFA with the development of ischemic heart diseases [11]. Olga Gruzdeva and colleagues studied relationship between FFA, markers of insulin resistance (IR) and oxidized lipoproteins in AMI and acute left ventricular failure patients [12]. They commented that AMI and its clinical complications are accompanied by a significant increase in the level of FFA, which not only reflect myocardial injury, but also Insulin resistance (IR) remains a confounding factor for oxidized LDL. IR in hepatocytes decreases glycogenesis and stimulates glycogenolysis and gluconeogenesis resulting in hyperglycemic state during AMI which is another important independent predictor of complications and mortality in these patients [13].

Asian Indians are at high risk of cardiovascular diseases even at normal BMI and WC than their counterparts [14]. A number of researchers suggested that FFA play an important role in atherosclerosis manifestation. Monitoring blood FFA levels in acute coronary events can play an important role in diagnostics and has prognostic value for choosing a strategy from a perspective of risk assessment in this category of patients. Clinical observations revealed association of development of arrhythmias with rise in circulating FFA in AMI patients. Certain type of arrhythmias have metabolic basis. Hence FFA estimation has been proposed as a predictor of arrhythmias in AMI [9].

Conclusion

Present study reveals the patients of AMI without previous T2DM, dyslipedemia or other known cardiac risk factors have raised FFA with altered atherogenic lipoprotein pattern. Study provides background to recommend estimation of serum FFA in lipid profile for diagnostic and prognostic purpose.

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