ORIGINAL ARTICLE

ASSOCIATION OF SERUM URIC ACID WITH DYSLIPIDEMIA IN ACUTE MYOCARDIAL INFARCTION PATIENTS

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ABSTRACT

Although the link between hyperuricemia and various diseases like metabolic syndrome, stroke, and coronary artery disease had been recognized, data on the relationship of serum uric acid (SUA) with dyslipidemia in patients with acute myocardial infarction (AMI) are limited. This study was conducted to evaluate the association of serum uric acid with dyslipidemia in patients with acute myocardial infarction (AMI) at Chittagong Medical College Hospital, Chattagram, Bangladesh. This was a hospital based cross-sectional analytical study comprising one hundred AMI patients admitted in coronary care unit (CCU) in Chittagong Medical College Hospital between July 2020 to June 2021. Important variables in this study were serum uric acid, serum fasting total cholesterol, triglycerides, HDL-C, LDL-C, waist circumference, BMI, age and gender. Sixty-two percent AMI patient had hyperuricemia. Increased serum uric acid was significantly associated with increased TG, total cholesterol, LDL level and decreased HDL level in AMI patients. Serum uric acid significantly correlated with components of fasting lipid profiles in AMI patients. The proportion of patients with hyperuricemia was high in AMI patients. Serum uric acid was associated and correlated with components of fasting serum lipid profiles.

Key words: AMI, Hyperuricemia, Lipid profiles

Introduction

Acute myocardial infarction (AMI) is a global health problem in both developed and developing countries and it is the leading cause of mortality and morbidity worldwide. According to the global status on non-communicable disease report 2011, approximately 2.5 million deaths are from cardiovascular diseases in India in 2008, two-thirds due to coronary artery disease[1]. South Asian populations have a high risk and 5-10 years earlier onset for acute myocardial infarction (AMI) in contrast to Western populations[2,3]. AMI in young individuals can cause death, disability and an increased economic burden. Previous studies have found that AMI patients (<40 years) had a high prevalence of smoking, family history, hyperuricemia, dyslipidemia and a relatively high incidence of normal coronary arteries[2,3]. Clinical and epidemiological studies

have proved that serum uric acid (SUA) is an important independent risk factor for developing coronary artery disease and cardiovascular mortality[4,5]. However, the definite role of uric acid in AMI is still the subject of debate because it is always accompanied with other risk factors such as diet, obesity and dyslipidemia[6].

Dyslipidemia is a strong risk factor for atherosclerotic cardiovascular disease (ASCVD). Desirable level of lipid profile is advocated to prevent or control atherosclerotic cardiovascular disease hazards, especially in patients with myocardial infarction. Therefore, to identify the risk factors for predicting dyslipidemia is clinically relevant. A number of studies have indicated the association between serum uric acid (SUA) and dyslipidemia. Previous studies have reported that hyperuricemia is associated with hypertriglyceridemia, low high-density lipoprotein cholesterol (HDL-C) and non-HDL-C[7-11]. A retrospective cohort study has shown that hyperuricemia increases the risk for developing high LDL-C and hypertriglyceridemia[12]. However, limited information is available on the association of serum uric acid with dyslipidemia in AMI not only in Bangladesh but also globally. Henceforth the present study was designed to explore the association of serum uric acid with dyslipidemia in patients with AMI.

Materials and Methods

Study design and study population

This is a cross sectional analytical study was carried out in Department of Biochemistry, Chittagong Medical College, Chattogram. The study population were diagnosed AMI patients admitted in Coronary Care Unit, Department of Cardiology, Chittagong Medical College Hospital, Chattogram.

Sample selections

A total 100 AMI patients were recruited using non-probability purposive sampling from July 2020 to June 2021 (one year). The inclusion criteria for cases were diagnosed patients of AMI in all age groups. People with gout, chronic kidney disease, patients on drugs like salicylates, thiazide diuretics, pyrazinamide were excluded. Exclusion was done by medical history with records and clinical examinations. Then they were requested to report to the department of Biochemistry, Chittagong Medical College at next morning following an overnight (10-12 hours) fasting.

Ethical consideration

Ethical clearance for this study was taken from the Ethical Review Committee of Chittagong Medical College (Memo No: CMC/PG/2020/667) informed written consent were taken from all patients before the interview and the study objectives and procedures were explained to them in their native language (Bengali).

Data collection and variables of the study

Patients; socio-demographics like age, gender were collected using a predesigned data collection form. The participants were interviewed face to face by researcher herself.

Anthropometric measurements, such as weight, height and waist circumference were measured using standardized protocols and calibrated equipments. Blood pressure were measured by auscultatory method using mercury sphygmomanometer in sitting position with calf at the level of the heart after 10 minutes of rest.

After overnight fasting, venous blood was collected using standard phlebotomy techniques and blood samples were analysed using standard protocols in the biochemistry laboratory. Serum uric acid level was measured by autoanalyzer (Dimension EXL 200) in uricase method. Fasting serum lipid profile were measured by enzymatic kinetic method using an auto analyzer, Dimension EXL 200.

Operational definition

AMI is defined as the detection of a rise and/or fall of cardiac troponin values and at least one of the following criteria, symptoms of ischemia, new ischemic ECG changes, development of pathological Q waves, imaging evidence of new loss of viable myocardium or new regional wall motion abnormality in a pattern consistent with an ischemic etiology[13]. Hyperuricemia is defined as the serum uric acid level in male ≥ 416.7 μmol/L (7 mg/dL) and in female ≥ 356.9 μmol/L (6 mg/dL)[14]. Desirable levels of fasting lipid profile[15] is defined as total cholesterol <200 mg/dL (<5.7 mmol/L); triglycerides <150 mg/dL (<1.69 mmol/L); HDL-C >40 mg/dL (>1.03 mmol/L) (male), and >50 mg/dL (>1.29 mmol/L) (female); LDL-C <100 mg/dL (<2.58 mmol/L).

Statistical analysis

Data were processed and analyzed using IBM-SPSS (Statistical Package for Social Science) version 20.0 for Windows. Data were expressed as mean±standard error of means (SEM), frequency and percentages. p value ≤ 0.05 was considered statistically significant. Hypothesis testing was done done by Chi-square ($\chi 2$) test and Pearson's correlation co-efficient.

Results

In this study out of total 100 AMI patients, 67% were male and 33% were female. The average age of patients was 57.73 years (SEM±1.09) ranging from 34-80 years. Mean SBP was 129.30 mm Hg (SEM±1.86) ranging from 100-190 mm Hg. Mean DBP was 82.50 mm Hg (SEM±1.22) ranging from 60-110 mm Hg. Mean BMI was 25.09 kg/m2 and waist circumference was 87.64 cm (Table I). The mean serum uric acid level among patients was 6.83 mg/dL (SEM±0.16) ranging from (3.20 to 10.20) mg/dL. Mean values of triglycerides, total cholesterol, HDL cholesterol and LDL cholesterol were 189.54 (SEM±6.74), 177.41 (SEM±4.33), 40.07 (SEM±0.36) and 104.57 (SEM±3.40) mg/dL respectively in patient group (Table II). Table-III demonstrates that 62% patients had hyperuricemia and 38% cases had normal uric acid levels. Table IV shows that increased serum uric acid level was significantly associated with increased triglycerides, increased total cholesterol, increased LDL cholesterol and decreased HDL cholesterol (p value <0.001). Pearson correlation test shows significant positive correlation of serum uric acid level with total cholesterol (r value+0.417), triglycerides (r value+0.465), LDL cholesterol (r value+0.474) and significant negative convelation with HDL cholesterol (r value - 0.254) (Table V, Fig. 1).

Table I: Baseline characteristics of study population (n=100)

Variables		Frequency (%)	Mean± SEM	Range
Age (years)			57.73±1.09	34–80
Gender	Male	67 (67)		
	Female	33 (33)		
Systolic blood pressure (mm Hg)			129.30±1.86	100–190
Diastolic blood pressure (mm Hg)			82.50±1.22	60–110
Body mass index (kg/m^2)			25.09±0.28	18.60–35.20
Waist circumference (cm)			87.64±0.76	59–120

Table II: Serum uric acid and serum fasting lipid profile values of study population (n=100)

Variables	Mean± SEM	Range
Serum uric acid (mg/dL)	6.83±0.16	3.20–10.20
Serum total cholesterol (mg/dL)	177.41±4.33	100–290
Serum triglyceride (mg/dL)	189.54±6.74	100–411
Serum LDL-C (mg/dL)	104.57±3.40	50–189
Serum HDL-C (mg/dL)	40.07±0.36	30–50

Table III: Distribution of cases according to serum uric acid (n=100)

Uric acid status	Frequency	Percentage
Hyperuricemia	62	62.0
Normal uric acid	38	38.0

Table IV: Association between serum uric acid status and serum fasting lipid profile status among cases (n = 100)

Fasting serum lipid profiles		Serum uric acid		Total (n = 100)	P values
		Hyperuricemia (n = 62)	Normal (n = 38)		
Serum TC (mg/dL)	Increased	30 (48.4)	3 (7.9)	33 (33.0)	< 0.001
	Normal	32 (51.6)	35 (92.1)	67 (67.0)	
Serum TG (mg/dL)	Increased	50 (80.6)	13 (34.2)	63 (63.0)	< 0.001
	Normal	12 (19.4)	25 (65.8)	37 (37.0)	
Serum LDL (mg/dL)	Increased	43 (69.4)	8 (21.1)	51 (51.0)	< 0.001
	Normal	19 (30.6)	30 (78.9)	49 (49.0)	
Serum HDL (mg/dL)	Decreased	49 (79.0)	19 (50.0)	68 (68.0)	< 0.001
	Normal	13 (21.0)	19 (50.0)	32 (32.0)	

Table V: Correlation between serum uric acid and serum fasting lipid profiles among the cases (n =

Variables	Pearson's correlation coefficient (r)	p values
SUA in mg/dL and TC in mg/dL	+ 0.417	< 0.001
SUA in mg/dL and Serum TG in mg/dL	+ 0.465	< 0.001
SUA in mg/dL and Serum LDL in mg/dL	+ 0.474	< 0.001
SUA in mg/dL and Serum HDL in mg/dL	- 0.254	< 0.05

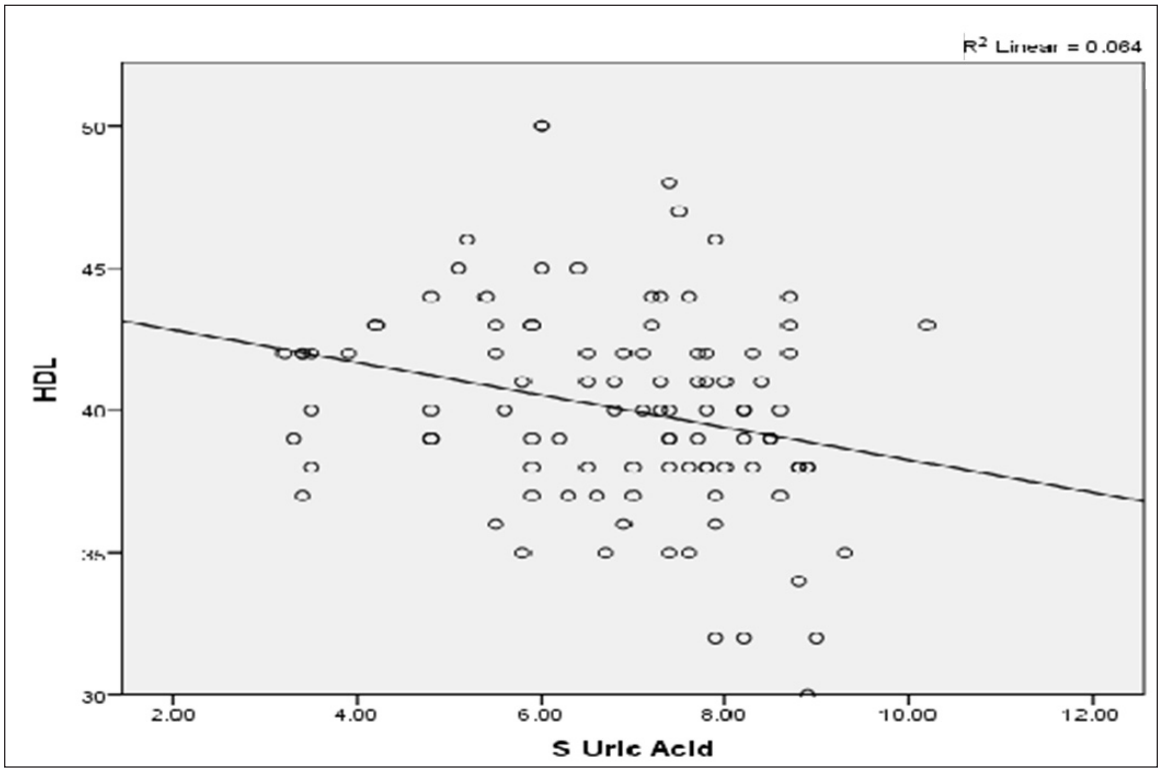

Fig 1. The scatter plot showing negative correlation between serum uric acid and serum HDL-C among the cases ($p < 0.05$)

Discussion

In this study average age of study population was 57.73±1.09 years. In a similar study done by Behera et al mean age was 50.4±13.1 years in STEMI patients[16]. It was reported that the mean age of first acute myocardial infarction was 53.0±11.4 years in South Asian data[17]. In this study the majority of the subjects were males (67% vs. 42% respectively). Harris et al reported 77% were males and 23% were females in 100 cases[18]. In another study, by Gosar et al in Indian population 65% were male[19]. This highlight that acute MI is predominantly observed in male population due to higher stress and social life among males.

In our study, there was significant increase in the serum uric acid levels in patients with AMI. Similar findings were observed in two other studies[20,21].

Increased level of uric acid causes decreased production of nitric acid, endothelial dysfunction, myocardial microvascular disease and local inflammation, oxidative metabolism, platelet adhesiveness and aggregation. High level of uric acid is also associated with xanthine oxidase activity[17,19,22].

Epidemiologic studies have shown that serum uric acid is often associated with dyslipidemia, especially hypertriglyceridemia[23,24]. In our study hyperuricemia was significantly associated with increased level of TG, TC and LDL-C but with decreased level of HDL-C in AMI. In this study we also observed a significant positive correlation between SUA and TG whereas there was a significant negative correlation between SUA and HDL-C ($r = -0.254$, $p < 0.05$) in AMI patients, suggesting a vital role of uric acid in the regulation of dyslipidemia. These results strengthened previous studies that showed a pathogenesis overlap between hyperuricemia and dyslipidemia[25,26]. When establishing the diagnosis of hyperuricemia, clinical suspicion of coexistent dyslipidemia should be required. These pathologies had a close relationship with coronary artery diseases.

Dyslipidemia is a strong risk factor for atherosclerotic cardiovascular disease (ASCVD)[27]. Our study findings strengthen the evidence about the relationship of serum uric acid with dyslipidemia and CAD. Hyperuricemia affects adipocytes contributing insulin resistance and inflammation[28]. In our study abnormal findings of lipid profiles, a reliable indicator of insulin resistance, also showed positive correlation with SUA.

Current study showed strong relationship of hyperuricemia with dyslipidemia in AMI patients. Its seen that SUA may intensify many pathophysiological processes associated with the risk of CAD and may have synergistic interactions with lipid profiles causing CAD. Henceforth detection and treatment of disordered lipid and SUA metabolism in AMI patients should be given high priority in clinical settings.

In this study the proportion of AMI patients with hyperuricemia was high. SUA was significantly associated with dyslipidemia in AMI patients. In clinical practice, further investigation would be warranted for more comprehensive strategic management to deal with dyslipidemia and hyperuricemia in AMI patients.

Multicenter prospective study with large sample size should be done for further evaluation of relationship of SUA with dyslipidemia in AMI. Inclusion of important variables like apo lipoprotein B, ratio of apo lipoprotein B to A1 may provide a better assessment in this context. Community based interventions should be aimed to convey awareness regarding detection and treatment of SUA and lipid profiles along with lifestyle changes in AMI patients. These can help greatly to prevent AMI or retard further AMI complications.

Limitations

This was cross sectional study where causal relationships cannot be established. This was small sample study done by purposive sampling which cannot be generalized to the entire diabetic population. This study did not include important indicators of CAD like Apo lipoprotein B, ratio of Apo lipoprotein B to A1.

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