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ORIGINAL ARTICLE

Prognostic Value of Serum Uric Acid Level in Patients with ST Elevation Myocardial Infarction Undergoing Primary Percutaneous Coronary Intervention

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Abstract

Background: *All major kinds of cardiovascular disease-related mortality, such as acute, subacute, and chronic coronary artery disease, heart failure, and stroke, have been linked to serum uric acid levels. This study was conducted to evaluate prognostic value of serum uric acid in patients with ST elevation myocardial infarction (STEMI) undergoing primary percutaneous coronary intervention (PPCI).*

Methods: *This prospective observational study was conducted at Department of Cardiology at National Institute of Cardiovascular Diseases (NICVD), Dhaka, for a period of 24-months following ethical approval. A total 100 STEMI patients who underwent primary PCI were included after getting written informed consent. Based on the basal serum uric acid level, patients were categorized into tertiles. Patients with low serum uric acid (n = 50) were defined as having a value in the lower two tertiles (<6.4 mg/dl), and patients with high serum uric acid (n = 50) as having a value in the third tertile (>6.4 mg/dl). Data were collected in separated case record form and analyzed by SPSS 26.0.*

Results: *Comparing with high serum uric acid group, the low serum uric acid group had better survival and a lower incidence of other major adverse cardiac events (MACE) (P = 0.027) and advanced heart failure as well as better KILLIP class (P = 0.028), better TIMI flow (P = 0.002), and a higher ejection fraction (49.2 ± 2.7 versus 42.3 ± 2.1; P = 0.001) during in hospital and three month follow up. Logistic regression shows that, TIMI Flow (OR=7.045, 95% CI=5.383-49.569, p=0.002), LVEF (%) (OR=2.419, 95% CI=1.054-5.554, p=0.037) and serum uric acid level (OR=19.879, 95% CI=0.786-23.069, p=0.030) were significantly associated risk factors for MACE.*

Conclusion: *High serum uric acid level is associated with increased in-hospital and short-term major adverse cardiovascular events (MACE) in patients with acute STEMI undergoing primary PCI.*

Key Words: *Primary PCI, ST elevation myocardial infarction, Uric acid.*

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Introduction

A sudden coronary artery blockage causes a ST elevation myocardial infarction (STEMI), which is the most serious form of ischemic heart disease and the leading cause of mortality globally.[1]

Patients with STEMI, prompt restoration of infarct related arterial (IRA) flow is linked to better ventricular function and decreased mortality.[2] So reperfusion therapy must begin as soon as possible for patients with STEMI [3].In patients with STEMI, primary percutaneous coronary intervention (PPCI) is the recommended reperfusion method within 12 hours after symptom onset.[4]

Both in the general population and following acute coronary syndromes (ACS), elevated serum uric acid

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(eSUA) has been linked to higher mortality.[5] Patients with obesity, insulin resistance, hypertension, and cardiovascular disease (CVD) frequently have elevated serum uric acid (SUA) level. Still there is debate whether such elevation is an independent predictor of cardiovascular risk or not.[6]

Uric acid is the final oxidation product of purine metabolism, a organic molecules consisting of carbon, nitrogen, oxygen and hydrogen. Elevated levels of uric acid are indicative of increased xanthine oxidase activity.[7]

Elevated uric acid level will raise the risk of oxidative stress, local inflammation, endothelial dysfunction, and insulin resistance, proliferation of smooth muscle cells in the arteries that causes vasoconstriction. This leads to develop atherosclerotic plaques, highlighting its significant involvement in the pathogenesis of cardiovascular disease.[8]

Increased serum uric acid level is linked to lipid-rich plaques, decreased coronary flow reserve and compromised coronary microvascular function, all of which are known to be predictive of unfavourable outcomes in the future.[9] In the context of mechanically reperfused STEMI, elevated serum uric acid (eSUA) may exacerbate ischemic/reperfusion damage, coronary microvascular blockage, and greater infarct size by inducing intracellular oxidative stress and an inflammatory response.[10]

Although the exact relationship between uric acid (UA) and coronary heart disease is unknown, certain research indicates that circulating uric acid as a result of coronary reperfusion deficits may constitute a novel biomarker for predicting the prognosis of coronary heart disease.[11]

So, the purpose of this study was to determine the validity of uric acid level as a potential prognostic marker on admission and short-term major adverse cardiovascular events (MACE) and its relation to Killip classification, TIMI flow, LVEF in patients with STEMI those undergoing primary percutaneous coronary intervention (PPCI).

Methods

This prospective observational research was conducted at the Department of Cardiology, National Institute of Cardiovascular Diseases (NICVD), Dhaka, Bangladesh, from July 2022 to June 2024. The study population consisted of STEMI patients admitted to NICVD who underwent primary PCI during this period. Consecutive sampling was used to select participants based on inclusion and exclusion criteria.

The study included patients with acute STEMI undergoing primary percutaneous coronary intervention (PPCI) who were aged 18 years or older. Patients were excluded if they had known malignancies, active infections, acute inflammatory states, chronic kidney disease (CKD), liver disease, previous percutaneous coronary interventions (PCI), previous coronary artery bypass grafting (CABG), or known rheumatic heart disease (RHD) or congenital heart disease.

The study variables included demographic variables such as age, sex, and BMI. Confounding variables included hypertension, diabetes, smoking status, serum creatinine, and dyslipidemia. The independent variable was serum uric acid, while the dependent variables were major adverse cardiac events (MACE) such as cardiac death, reinfarction, and target vessel revascularization, as well as arrhythmia, stroke, left ventricular ejection fraction (LVEF), TIMI flow, and Killip class.

Data collection for the study was carried out using a pre-designed data collection sheet for statistical analysis and interpretation. Informed written consent was obtained from each participant before enrollment. Detailed patient histories and clinical examinations, including chest assessments to determine Killip score, were conducted and documented in a structured questionnaire. Demographic information, such as age, sex, and occupation, was recorded. A 12-lead resting ECG was performed upon admission using the Wuhan Zoncare Bio-medical Electronics Co. Ltd. model. Patients received standard treatment as per current guidelines, including aspirin, P2Y12 inhibitors, heparin, statins, and beta-blockers, unless contraindicated. Blood samples were collected for serum uric acid, serum creatinine, CBC and for other investigations. Serum uric acid levels were measured using an enzymatic color test on Beckman Coulter analyzers. The study population was divided into tertiles based on admission uric acid value, a value in the third tertile was defined as high uric acid group (≥6.4 mg/dl),(Group I) and in the lower two tertiles as the low uric acid group (<6.4mg/dl), (Group II). Coronary angiography and stenting of the culprit lesion were performed via transfemoral or transradial approach. All patients underwent transthoracic 2D echocardiography within 24 hours of admission using a GE VIVID 8 ultrasound system to assess left ventricular ejection fraction (LVEF) via the biplane method.

The data collection tools used in the study included a checklist prepared for each patient, an interview schedule in Bengali containing questions related to the study's

objectives, and a pretested questionnaire, which was tested on a similar group of admitted patients.

Data processing and analysis: After collecting the required data, it was checked for consistency, verified, and tabulated using SPSS version 26 (IBM Corp., Armonk, NY). Frequencies and percentages were calculated for qualitative variables, while arithmetic mean and standard deviation were used for quantitative variables. The unpaired t-test was applied to compare symmetrically distributed continuous variables, and the Chi-square test or Fisher's exact test was used for categorical variables. Multivariate logistic regression was employed to determine the odds ratio of risk factors Analysis for MACE. A p-value of <0.05 was considered statistically significant. Finally, the data were presented in tables, figures, and graphs as necessary.

Ethical consideration

The study was approved by the Ethical Review Committee, and informed consent was obtained from participants, ensuring confidentiality and their right to withdraw.

Results

Table-I

Baseline Characteristics of the studied populations

	Group I (n=50)	Group II (n=50)	*P* value
Age group (years)	56.5±3.5	54.3±4.1	0.004**
Male	36 (72)	38 (76)	0.820
Female	14 (28)	12 (24)	
Smoking habit	26 (52)	22 (44)	0.548
Hypertension	14 (28)	12 (24)	0.820
Diabetes mellitus	11 (22)	9 (18)	0.803
Dyslipidemia	8 (16)	9 (18)	1.00
BMI (kg/m^2)	22.1±2.1	21.7±2	0.418
Systolic blood pressure (mm of Hg)	124.1±16.2	123.3±16.9	0.809
Diastolic blood pressure (mm of Hg)	82.9±14.2	81.8±14.3	0.710
Pulse (beat per min)	75.5±4	75.9±4.2	0.665
Killip's class >I	10 (20)	2 (4)	0.028^s

(Within parenthesis percentage over column in total).

Baseline Characteristics of the studied populations were mentioned in (table I). In Group I, 68% of patients were above 55 years, with a mean age of 56.5±3.5 years, while 56% of patients in Group II were 55 years or below, with a mean age of 54.3±4.1 years, and age was significantly higher in Group I than Group II. In both Group I and Group II majority of the patients were male (72% vs 76%).

In Group I, 52% had a smoking habit, 28% had hypertension, 22% had diabetes mellitus, and 16% had dyslipidemia, while in Group II, 44% had a smoking habit, 24% had hypertension, 18% had diabetes mellitus, and 18% had dyslipidemia, with no significant difference found between the two groups.

Table-II

Laboratory findings of the study patients (n=100)

	Group I (n=50)	Group II (n=50)	*P* value
Random blood sugar (mmol/l)	9.22±3.51	8.99±3.57	0.746
Hemoglobin (gm/dl)	12.1±0.5	12.2±0.5	0.715
Serum Creatinine (mg/dl)	0.93±0.2	0.90±0.2	0.400
Serum uric acid (mg/dl)	6.7±0.3	5.3±0.4	<0.001

In table-II laboratory findings of the patients were mentioned. In Group I serum uric acid was found significantly higher than Group II (6.7±0.3 mg/dl vs 5.3±0.4 mg/dl). Other laboratory findings are statistically similar in both groups.

Table-III

Distribution of the patients according to angiographic findings (n=100)

	Group I (n=50)	Group II (n=50)	*P* value
PPCI of Culprit lesion			
LMCA	0 (%)	0(%)	0.786
LAD	25 (50)	22 (44)	
LCX	5 (10)	7 (14)	
RCA	20 (40)	21 (42)	
TIMI flow after procedure			
2	11 (22)	1 (2)	0.004
3	39 (78)	49 (98)	
No. of diseased vessels			
1	26 (52)	30 (60)	0.736
2	15 (30)	12 (24)	
3	9 (18)	8 (16)	

Primary PCI of culprit lesion and number of diseased vessels of both Group I and II was statistically similar. Besides, TIMI flow 3 was 78% and 98% in Group I and Group II accordingly. TIMI flow was significantly low in Group I than Group II.

Table-IV

In hospital outcome of the patients (n=100)

	Group I (n=50)	Group II (n=50)	*P* value
MACE	5 (10)	2 (4)	0.218*
Cardiac death	2 (4)	1 (2)	0.500*
TVR	1 (2)	0 (0)	0.500*
Reinfarction	2 (4)	1 (2)	0.500*
Advanced heart failure	7 (14)	1 (2)	0.030*
Arrhythmia			
Atrial fibrillation	2 (4)	1 (2)	0.50*
VT/VF on cardiac monitoring	3 (6)	1 (2)	0.30*
Stroke	(0)	(0)	-
LVEF	41.1±3.8	49.2±3.9	
<0.001**			

Regarding in hospital outcome MACE, AF and VT/VF were statistically similar and advanced heart failure was significantly

higher in group I. LVEF was significantly higher in Group II than Group I.

Table-V

Outcome of the patients (From discharge to 3 months follow up) (n=97)

	Group I (n=48)	Group II (n=49)	*P* value
MACE	7 (14.6)	3 (6.1)	0.159
Cardiac death	2 (4.2)	0 (0)	0.247
TVR	2 (4.2)	2 (4.1)	0.691
Reinfarction	3 (6.2)	1 (2)	0.309
Advanced heart failure	8 (16.7)	2 (4)	0.046
Stroke	1 (2.1)	(0)	0.495

Regarding outcome (From discharge to 3 months follow up), MACE, was statistically similar in both group.

Advanced heart failure was significantly high in Group I.

Table-VI

Short term outcome of the patients (In-hospital & 3 months follow up) in-terms of MACE (n=100)

	Group I (n=50)	Group II (n=50)	*P* value
MACE	12 (24)	4(8)	0.027
Cardiac death	4 (8)	1 (2)	0.181
TVR	3 (6)	1 (2)	0.309
Reinfarction	5 (10)	2 (4)	0.218

Regarding short-term outcome (In-hospital and after 3 months follow up), in-terms of MACE (Cardiac death, reinfarction and TVR) was found significantly higher in Group I than Group II.

Table-VII

Risk factors analysis of MACE by multivariate logistic regression analysis

Risk factors	Odds ratio	95% CI	*P* value
Age (>55)	0.409	0.019-8.845	0.569
Killip Class (>I)	1.306	0.004-24.514	0.597
TIMI Flow <3	7.045	0.839-29.569	0.056
LVEF (<40%)	2.419	1.054-5.554	0.037
Creatinine	1.023	0.077-2.294	0.415
Uric acid >6.4 mg/dl	19.879	1.786-23.069	0.030

In table-VI logistic regression shows that, LVEF (<40%) (OR=2.419, 95% CI=1.054-5.554, *P*=0.037) and serum uric acid level>6.4mg/dl (OR=19.879, 95% CI=1.786-23.069, *P*=0.030) were significantly associated risk factors for MACE.

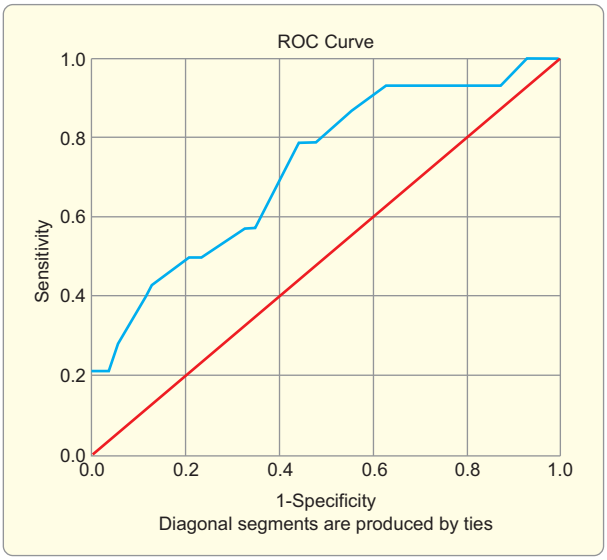

Figure 1: *ROC curve analysis of serum uric acid in the prediction of MACE in STEMI patients.*

In ROC curve analysis the highest Youden's Index was 0.344, with an optimum cut-off level for serum uric acid at 6.45, and the area under the ROC curve (AUC) was 0.724 (95% CI 0.577–0.870).

Table-VIII

Diagnostic accuracy of SUA in prediction of MACE patients with acute STEMEI undergoing PPCI.

Parameters	Value
Cut-off point	6.45
Sensitivity	78%
Specificity	54%
Positive predictive value (PPV)	22%
Negative predictive value (NPV)	94%
Accuracy	58%
Area under curve	0.724
P value	0.007

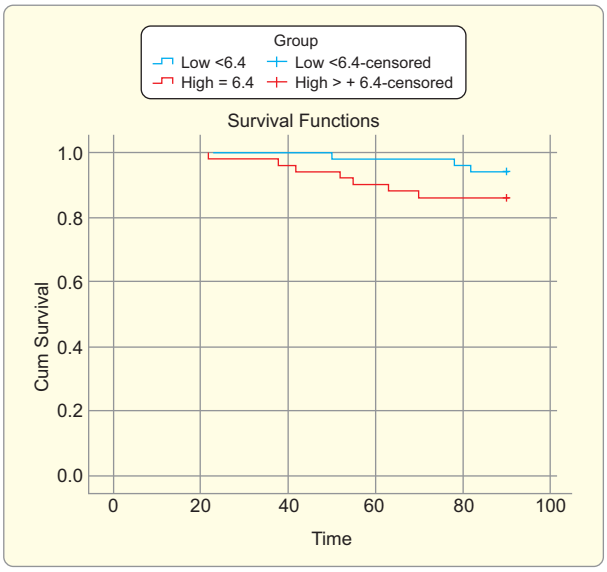

Figure 2: *Kaplan-Meier Curves of short- term (in hospital+ 3 months) composite MACE study*

Kaplan-Meier Curves showed the incidence of short-term composite MACE in high uric acid group was statistically significantly higher compared to low uric acid group (*p* value = 0.045).

Discussions

This study aimed to assess the prognostic value of SUA in STEMI patients underwent PPCI. A total of 100 patients were enrolled: 50 with SUA levels ≥6.4 mg/dl (Group I) and 50 with levels <6.4 mg/dl (Group II), selected via consecutive sampling.

In Group I and Group II mean ages of patients were 56.5±3.5 and 54.3±4.1 years, respectively. Similarly, Akanda et al. (2011) reported a mean age of 50.15±8.88 years among 637 CAD patients at NICVD,[12] while the BRAVE study found that 88% of MI cases were male, with 46% over 50 years and a mean age of 53±10 years.[13]

Previous research by Hu et al. showed significantly higher ages in hyperuricemia patients (65.6±15.1 years) compared to those with lower uric acid levels (62±13.1 years).[14] The discrepancy of mean age might result from having distinct demographic features of CAD in Bangladesh.[15]

Aging often leads to reduced renal function, diminishing uric acid clearance. Comorbid conditions like hypertension, diabetes, and heart failure, prevalent in the elderly, further affect uric acid metabolism. Medications commonly used in these patients, such as diuretics and low-dose aspirin, can also elevate uric acid levels.

In both Group I and Group II, the majority of patients were male consistent with previous studies showing a male predominance.[14,16] Studies in Bangladesh also reported a higher proportion of male patients, likely due to men having better access to healthcare and a higher prevalence of smoking, a key risk factor for ischemic heart disease (IHD).[12,13] Additionally, men generally have higher uric acid levels than women until menopause. In this study, with over 70% of patients being male, no significant difference was observed between uric acid levels and gender.

In both Group I and Group II, comorbidities such as smoking, hypertension, diabetes mellitus, and dyslipidemia were statistically similar, aligning with previous studies that found no significant effect of these factors on uric acid levels.[14,16] In terms of coronary involvement, the most commonly affected artery in both groups was the LAD followed by the RCA and the LCx which is consistent with findings from other studies.[16,17]

In the current study, Group II showed significantly better TIMI flow and better Killip class than Group I, consistent with findings by Akgül et al. Abdellatif et al.[16,17] Kaya et al.found that higher uric acid levels were linked to poorer TIMI flow restoration post-PCI.[18] This correlation may be due to lower uric acid levels being associated with reduced inflammation, oxidative stress, and better overall systemic health, contributing to improved cardiac outcomes.[19]

Atrial fibrillation (AF) and VT/VF were more frequent in Group I than Group II but did not constitute statistically significant. Hu et al. found a significant link between hyperuricemia and VT/VF after PPCI in STEMI patients, while Akgül et al. and Kaya et al. observed higher AF and VT in patients with elevated uric acid.[14,17,18] Hajizadeh et al. also observed high uric acid was associated with increased AF in acute MI patients.[20]

This study found that patients with low serum uric acid had significantly better left ventricular ejection fraction (LVEF) after PPCI compared to those with high SUA and the high SUA group had more frequent readmissions due to heart failure. Similar results were reported by Kaya et al. and Akgül et al.[17,18]

In our study in hospital MACE was higher in group I but not statistically significant, which is not consistent with Akpek et al.Small sample size may be the cause. Akpek et al. revealed that uric acid level on admission is a strong and independent predictor of poor coronary blood flow following primary PCI and in hospital MACE among patients with STEMI.[19]

Group I exhibited significantly higher rates of short-term major adverse cardiovascular events (MACE) and advanced heart failure both in-hospital and during the 3-month follow-up compared to Group II which is consistent with Akgül et al.[14] Multivariate logistic regression identified lower LVEF and elevated serum uric acid levels as significant risk factors for short-term MACE. Similarly, Akgül et al. and Omidvar et al. found that higher SUA levels correlated with increased in-hospital and short-term mortality.[17,21] Thus, this study supports SUA as a valuable prognostic marker in STEMI patients undergoing PPCI.

Conclusion

This study evaluated significant prognostic value of SUA in patients with STEMI undergoing primary PCI as low SUA group had a better Killip class, better TIMI flow after stenting, higher ejection fraction and better survival besides lower incidence of advanced heart failure and other MACE during the hospital stay and three months follow up in comparison to the high uric acid group.

Funding

No funding was received for this work.

Conflict of Interest

None

Limitation

All samples were collected from a single site ,Sample size was small and Sample was taken purposively, so randomization was not done.We did not compare the prevalence of using drugs like thiazide diuretics or other usual drugs that can change uric acid level among our groups.

Recommendation

Further randomized multicenter studies with larger sample size and longer follow-up are recommended. Routine evaluation of serum uric acid in STEMI patients should be considered in management plan

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