

RESEARCH ARTICLE

Open Access Journal



## “To Assess Outcome of NSTEMI Patients with Raised Serum Uric Acid Level”

Sardar Zahid Hossain<sup>1</sup> | Md. Iktier Hassan Khan<sup>2</sup> | Md. Arif Mohammad Sohan<sup>3</sup> | Md. Ahasanul Kabir<sup>4</sup> | Md. Tufazzal Hossen<sup>5</sup> | Abu Baqar Md. Jamil<sup>6</sup> | Khan daker Harun-Rashid<sup>7</sup>

<sup>1</sup> Senior Consultant (Cardiology), Manipal AFC Hospital, Khulna, Bangladesh

<sup>2</sup> Assistant Professor (Cardiology), MBBS, D-Card (Cardiology), Shaheed Shiekh Abu Naser Specialized Hospital, Khulna, Bangladesh

<sup>3</sup> Associate Professor, Department Of Cardiology, Japan East West Medical College Hospital, Aichi Nagar, JBCS Sarani, Horirampur, Turag, Dhaka, Bangladesh

<sup>4</sup> Junior Consultant (Cardiology), Satkhira Medical College & Hospital, Khulna, Bangladesh

<sup>5</sup> Assistant Professor (Cardiology), Sheikh Hasina Medical College, Jamalpur, Bangladesh

<sup>6</sup> Assistant Professor (Cardiology), Shaheed Ziaur Rahman Medical College, Bogura, Bangladesh

<sup>7</sup> Junior Consultant (Cardiology), 250 Bedded General Hospital, Tangail, Dhaka, Bangladesh



### Abstract

**Background:** Few studies investigated serum uric acid with short and long-term major adverse outcome (MACE) in patients with NSTEMI. Non-ST Segment Elevation Myocardial Infarction (NSTEMI) is the common form of ACS and a leading global cause of premature morbidity and mortality. **Objective:** To assess in-hospital outcome of NSTEMI patients with raised serum uric acid level. **Methodology:** This was a cross-sectional observational study that was conducted in the Department of Cardiology, Manipal AFC Hospital, Khulna during the period from January, 2019 to June, 2020. Purposive consecutive sampling of 55 cases of NSTEMI with raised SUA level and 55 cases of NSTEMI with normal SUA level who were admitted in CCU included in this study. In-hospital complications and mortality were recorded while continuing standard treatment for the event. **Results:** The study population was between 30-80 years. Only 20 (18.18%) patients were below 40 years. The mean age of patients was  $55.4 \pm 11.4$  years. The mean age of the patients of group A was significantly higher than patients of Group B ( $p < 0.001$ ). Male preponderance was in both groups (81.8% versus 78.0%;  $p = 0.603$ ). Diabetes mellitus (52.7% versus 21.8%;  $p = 0.002$ ), hypertension (78.1% versus 52.7%;  $p = 0.039$ ) and dyslipidaemia (47.2% versus 12.7%;  $p < 0.001$ ) were more frequent in group A than that of group B. The mean BMI of patients was  $25.72 \pm 1.99$  kg/m<sup>2</sup>. Mean systolic BP was  $126.61 \pm 21.86$  mm of Hg and diastolic BP was  $82.07 \pm 13.11$  mm of Hg. But smoking status (69.0% versus 65.4%;  $p = 0.668$ ),

Keywords: NSTEMI, Serum Uric Acid Level, Acute coronary syndromes (ACS) Corresponding Author: Sardar Zahid Hossain Senior Consultant (Cardiology), Manipal AFC Hospital, Khulna, Bangladesh

Copyright : © 2021 The Authors. Published by Medical Editor and Educational Research Publishers Ltd. This is an open access article under the CC BY-NC-ND license

(<https://creativecommons.org/licenses/by-nc-nd/4.0/>).

## 1 | INTRODUCTION

**A**cute coronary syndromes (ACS) remain a leading cause of morbidity and mortality worldwide (1). Acute Myocardial infarction (AMI) (encompassing ST-segment elevation myocardial infarction, non-ST segment elevation myocardial infarction) is the leading cause of death in developed countries and the second leading cause of death in developing countries. Acute coronary syndromes (ACS) encompasses Unstable Angina (UA) and ST-segment elevation MI (STEMI) or non-ST-segment elevation MI (NSTEMI) (2). Myocardial infarction (MI) is one of the most common life threatening diagnoses in emergency hospital admissions. Most of the complications occur during the first few hours of hospitalization. MI is defined in pathology as myocardial cell death due to prolonged myocardial ischemia (3). The clearest separation between UA and NSTEMI is the absence or presence, respectively, of abnormal concentrations of biomarkers indicative of myocardial necrosis, either the troponins (which are structural proteins) or creatine kinase MB (which is a cardiac enzyme) (4). Angiographic, Intravascular Ultrasound (IVUS), and angioscopic studies indicate that UA/NSTEMI usually results from the disruption of an atherosclerotic plaque with a subsequent platelet-rich thrombus that obstructs microvascular blood flow and transiently or partially obstruct epicardial blood flow. Atherosclerosis is a chronic, lipid-driven inflammatory disease of the arterial wall leading to multifocal plaque development (5). As a consequence of different technical measures in developed countries, in-hospital mortality from AMI in the general population declined by half, from 15% to about 7.5% and it is now as low as 3.5% (6). By the year 2020, CAD will hold first place in the WHO's list of the leading cause of disability (7, 8). The South Asian countries of India, Pakistan, Bangladesh, Srilanka, and Nepal contribute the highest proportion of the burden of cardiovascular diseases (CVDs) compared to any other region globally (9). In India, 4% rural and 11% urban populations suffer from CAD (10). The case fatality rates for cardiovascular events in low income countries, represented largely by India, were 17%; this is much higher than in higher income

countries, which had a case fatality rate of 6.5% (11). In Bangladesh, data regarding case fatality rate of AMI are based on discrete and small scale studies. The exact proportion of ACS patients receiving early reperfusion therapy (Thrombolysis, Stenting or CABG) is also unknown. Thus, the result would be more alarming in Bangladesh if nationwide study could be conducted. Acute myocardial infarctions have a substantially increased risk of death after hospitalization, when shock, left ventricular failure (LVF) or arrhythmias occur during their hospital stay. So cardiogenic shock, LVF and arrhythmias should be treated properly. Knowledge of potential complications will help in proper management of acute MI with good outcome (12). In a recent study done in Bangladesh it has been concluded that on admission serum uric acid estimation is a predictor of in-hospital poor outcome in patients with acute myocardial infarction. As it is a cheap and noninvasive procedure, it can be routinely practiced in cardiac emergency department for risk stratification of patients (13). There have been enormous studies in multiple domains of ACS pathophysiology to detect unknown or less understood etiological and/or associated factors of ACS in the hope of a better understanding of the grave pathological condition so that newer approaches could be invented to combat the ominous impact of atherosclerotic cardiovascular disease on mankind. This study was done to observe and record the in-hospital outcome including mortality and other complications in NSTEMI patients with raised serum uric acid level and compare the outcome with that of NSTEMI patients with normal serum uric acid level.

## 2 | METHODS AND MATERIALS

This was a cross-sectional observational study that was conducted in the Department of Cardiology, Manipal AFC Hospital, Khulna during the period

---

**Supplementary information** The online version of this article (<https://doi.org/10.15520/jmrhs.v4i8.386>) contains supplementary material, which is available to authorized users.

---

from January, 2019 to June, 2020. Purposive consecutive sampling of 55 cases of NSTEMI with raised SUA level and 55 cases of NSTEMI with normal SUA level who were admitted in CCU included in this study. NSTEMI patients with raised serum uric acid (>7mg/dl in male; >6mg/dl in female) level (Group A) and normal serum uric acid level (group B) admitted within 24 hours of symptom onset were consequently enrolled. Inclusion criteria include Patients with NSTEMI, admitted within 24 hours of symptom onset, 30-75 years of age and both males and females. Exclusion criteria are NSTEMI patients admitted after 24 hours of symptom onset, NSTEMI patients treated outside before admission in CCU, NSTEMI patients with CKD, Liver disease, Malignancy, Myeloproliferative disease, Past history of myocardial infarction, Past history of revascularization (Stenting, CABG), Cardiomyopathy, valvular heart disease, patients on Hydrochlorothiazide, those who refused to enroll in this study. Both quantitative and qualitative data were collected by using pre designed questionnaire. Informed written consent was taken from the patients and/or attendant after detailed explanation of the purpose of the study. Data were collected by the investigator through face to face interview, ECG, physical examination, laboratory investigations and imaging.

**Data Analyses:** All patients were followed up hourly in CCU and 3 times in 24 hours (8.00am, 2.00pm and 8:00pm) in post-CCU up to discharge from hospital. During follow up, a 12 lead ECG was recorded daily till discharge. Development of new chest pain (post MI angina), any arrhythmia, heart failure, cardiogenic shock and in-hospital mortality were observed and recorded. All relevant data were recorded in a pre-designed questionnaire that were processed and analyzed manually and using SPSS (Statistical Package for Social Sciences) Version 22.0.

### 3 | RESULTS

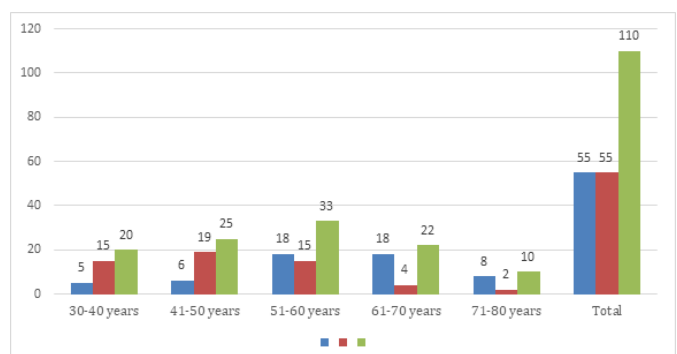
Clinical characteristics of patients are shown in (Table 1). There was no significant difference in age and the incidences of hypertension, diabetes mellitus and previous coronary artery disease between the two groups. The mean age was  $60.82 \pm$

$9.62$  years in group A and  $49.90 \pm 10.40$  years in group B. The mean age of the patients of group A was significantly higher than patients of Group B ( $p < 0.001$ ). Male preponderance was in both groups (81.8% versus 78.0%;  $p = 0.603$ ). Diabetes mellitus (52.7% versus 21.8%;  $p = 0.002$ ), hypertension (78.1% versus 52.7%;  $p = 0.039$ ) and dyslipidaemia (47.2% versus 12.7%;  $p < 0.001$ ) were more frequent in group A than that of group B. The mean BMI of patients was  $25.72 \pm 1.99$  kg/m<sup>2</sup>. Mean systolic BP was  $126.61 \pm 21.86$  mm of Hg and diastolic BP was  $82.07 \pm 13.11$  mm of Hg. But smoking status (69.0% versus 65.4%;  $p = 0.668$ ), family history of CAD (10.2% versus 20.8%;  $p = 0.476$ ) did not differ significantly. Mean serum uric acid was  $6.23 \pm 1.84$  mg/dl. Killip class did not differ significantly between group A and group B ( $p = 0.127$ ). The study showed that Killip class I was in 25 (45.0%), Killip class II in 17 (30.9%), Killip class III in 7 (12.7%) and Killip class IV in 6 (10.9%) patients in group A; whereas Killip class I was in 35 (63.6%), Killip class II in 9 (16.3%), Killip class III in 9 (16.3%) and Killip class IV in 2 (3.6%) patients in group B. Killip class did not differ significantly between group A and group B ( $p = 0.127$ ).

Table 1: Baseline characteristics of study population (n=110)

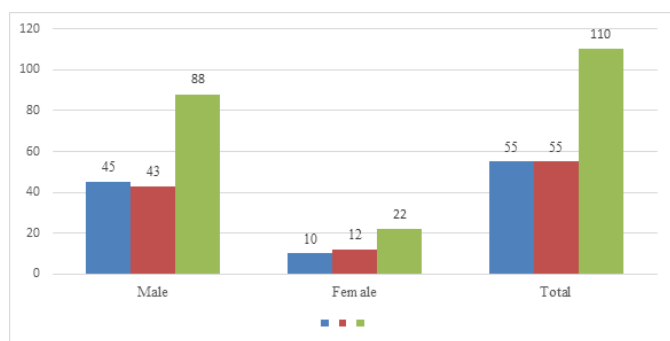
Baseline characteristics	Number (%)	Mean±SD (range)	p-value
Age (years)		55.4 ± 11.4 (30-80)	†p<0.001
Sex			
Male	88	80.0%	*p<0.001
Female	22	20.0%	
BMI (kg/m <sup>2</sup> )		25.72 ± 1.99	
Systolic BP (mm of Hg)		126.61 ± 21.86	
Diastolic BP (mm of Hg)		82.07 ± 13.11	
RBS (mmol/L)		7.62 ± 2.32	
Serum uric acid (mg/dl)		6.23 ± 1.84	

SD=standard deviation. \*Chi-Square ( $\chi^2$ ) test and †unpaired t-test were done to analyze the data. \*Chi-Square ( $\chi^2$ ) test was done to measure the level of significance.



TO ASSESS OUTCOME OF NSTEMI PATIENTS WITH RAISED SERUM URIC ACID LEVEL

**FIGURE 1:** Distribution of the patients according to age.



**FIGURE 2:** Distribution of the patients according to Sex.

**Table 2 :** Distribution of the patients by Diabetes Mellitus (n=110)

Diabetes mellitus	Study Group			p-value
	Group A (n=55)	Group B (n=55)	Total (n=110)	
Present	29	12	41	*p=0.002
Absent	26	43	69	
Total	55	55	110	

\*Chi-Square ( $\chi^2$ ) test was done to analyze the data.

**Table 3 :** Distribution of the patients by Smoking Status (n=110)

Smoking status	Study Group			p-value
	Group A (n=55)	Group B (n=55)	Total (n=110)	
Smoker	38	36	74	*p=0.668
Non-smoker	17	19	36	
Total	55	55	110	

\*Chi-Square ( $\chi^2$ ) test was done to analyze the data.

**Table 4 :** Distribution of the patients according to Blood Pressure (n=110)

Blood pressure	Study Group			p-value
	Group A (n=55)	Group B (n=55)	Total (n=110)	
Hypertensive	43	29	72	*p=0.039
Nom otensive	12	29	38	
Total	55	55	110	

\*Chi-Square ( $\chi^2$ ) test was done to analyze the data

**Table 5 :** Distribution of the patients according to Dyslipidaemia (n=110)

Dyslipidaemia	Study Group			p-value
	Group A (n=55)	Group B (n=55)	Total (n=110)	
Present	26	7	33	*p<0.001
Absent	29	48	77	
Total	55	55	110	

\*Chi-Square ( $\chi^2$ ) test was done to analyze the data.

**Table 6 :** Distribution of the patients according to family history of CAD (n=110)

Family History of CAD	Study Group			p-value
	Group A (n=55)	Group B (n=55)	Total (n=110)	
Present	32	40	72	*p=0.476
Absent	23	15	38	
Total	55	55	110	

**Table 7 :** Distribution of the patients by Killip Class of Cardiac Failure (n=110)

Killip class	Study Group			p-value
	Group A (n=55)	Group B (n=55)	Total (n=110)	
Killip class -I	25	35	60	*p=0.127
Killip class -II	17	9	26	
Killip class -III	7	9	16	
Killip class -IV	6	2	8	
Total	55	55	110	

Fisher’s exact test was done to analyze the data.

**Table 8 :** Distribution of the patients by In-hospital Mortality (n=110)

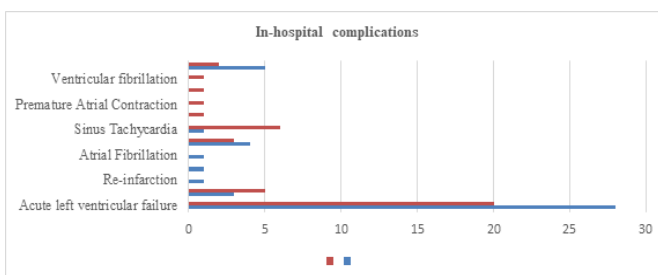
In-hospital mortality	Study Group			p-value
	Group A (n=55)	Group B (n=55)	Total (n=110)	
Death	5	2	7	*p=0.436
Survive	50	53	103	
Total	55	55	110	

\*Fisher’s exact test was done to analyze the data.

**Table 9 :** Distribution of the patients by In-hospital Complications (n=110)

In-hospital complications	Study Group		p-value
	Group A (n=55)	Group B (n=55)	
Acute left ventricular failure	28	20	*p=0.108
Post MI angina	3	5	†p=0.715
Re-infarction	1	0	†p=1.000
Atrial Flutter	1	0	†p=1.000
Atrial Fibrillation	1	0	†p=1.000
Sinus Bradycardia	4	3	†p=1.000
Sinus Tachycardia	1	6	†p=0.112
Right Bundle block	0	1	†p=1.000
Premature Atrial Contraction	0	1	†p=1.000
Ventricular tachycardia	0	1	†p=1.000
Ventricular fibrillation	0	1	†p=1.000
Cardiogenic shock	5	2	†p=0.436

\*Chi-Square ( $\chi^2$ ) test and †Fisher’s Exact test were done to analyze the data.



**FIGURE 12:** Distribution of the patients by In-hospital Complications.

This study shows revealed that cardiogenic shock [5 (10.0%) versus 2 (4.0%),  $p=0.436$ ]; acute left ventricular failure [27 (54.0%) versus 19 (38.0%),  $p=0.108$ ]; re-infarction [1 (2.0%) versus 0 (0.0%),  $p=1.000$ ]; sinus tachycardia [1 (2.0%) versus 6 (12.0%),  $p=0.108$ ]; sinus bradycardia [4 (8.0%) versus 3 (6.0%),  $p=1.000$ ]; atrial flutter [1 (2.0%) versus 0 (0.0%),  $p=1.000$ ]; atrial fibrillation [1 (2.0%) versus 0 (0.0%),  $p=1.000$ ]; right bundle block [0 (0.0%) versus 1 (2.0%),  $p=1.000$ ], ventricular tachycardia [0 (0.0%) versus 1 (2.0%),  $p=1.000$ ]; ventricular fibrillation [0 (0.0%) versus 1 (2.0%),  $p=1.000$ ] did not differ significantly between two groups. But none of the patients of both groups developed mechanical complication. In-hospital mortality was 5 (9.09%) patients in group A and 2 (3.6%) patients in group B; did not reach the level of significance ( $p>0.05$ ) and complications such as post MI angina, cardiogenic shock, acute left ventricular failure, re-infarction, sinus tachycardia, sinus bradycardia, atrial flutter, atrial fibrillation, bundle branch block, ventricular tachycardia, ventricular fibrillation did not differ significantly between the two groups ( $p>0.05$ ) (Tab-2-9 & fig-1,2,3).

#### 4 | DISCUSSION

The study population was between 30-80 years. Only 20 (18.18%) patients were below 40 years. The mean age of patients was  $55.4 \pm 11.4$  years. It was found that the study population was mainly male 88 (80.0%) and female were only 22 (20.0%). The mean age of the patients of hyperuricemia was significantly higher than patients of normal uric acid level ( $p<0.001$ ). Similar result was also observed in the study of Omidvar, et al., (14). This study showed that males were predominant in both groups (81.8% and 78.1% respectively). No significant difference of gender was seen between two groups ( $p=0.603$ ). Male preponderance of NSTMI was reported in sev-

eral other studies like Belle, et al. (15). This study showed that 37.0% of total NSTEMI patients were diabetic with 52.7% of patients of hyperuricemia was diabetic and 21.8% of patients of normal uric acid level were diabetic. The patients of hyperuricemia were more frequently diabetic than that of normal uric acid level ( $p=0.002$ ). This finding was also supported by Safi, et al., (16), which showed that hyperuricemia was significantly associated with type 2 diabetes mellitus. However, this finding was in contrast to other study by Das, et al., (17), in which there was no significant association between serum uric acid level and diabetic status. Our study revealed several risk factors such as hypertension, diabetes mellitus, smoking, dyslipidemia, and family history of CAD. These were 78.1%, 21.8%, 69.0%, 47.2% and 10.2% respectively. Dharma et al., (18) found the same risk factors such as hypertension, diabetes mellitus, smoking, dyslipidemia, and family history of IHD, which were 53%, 21%, 52%, 69%, and 24%, respectively. Omidver et al., (19) also found some risk factors like hypertension, diabetes mellitus, smoking, and dyslipidemia which were 37%, 29.3%, 46.2%, and 33.2%, respectively. Increased SUA is significantly associated with the occurrence and mortality of coronary artery disease (20). But few studies have investigated serum uric acid levels in patients with acute myocardial infarction. Several theories have been discussed, such as high serum uric acid has an impact on increasing platelet reactivity (21), mediating inflammation, and stimulation of smooth muscle cell proliferation which probably worsened the acute thrombosis complication. Previous trials suggest that uric acid might be an independent predictor of major adverse cardiovascular events (MACE) in patients with coronary artery disease or only an indirect marker of adverse event due to the association between uric acid and other cardiovascular risk factors (22). High SUA has been indicated as a risk factor for CAD and as an independent prognostic factor of poorer outcomes (occurrence of AMI, fatal AMI, sudden death, all-cause mortality) in patients with verified CAD (20). Less is known about SUA as a potential prognostic/risk factor for outcomes in patients affected specifically by AMI. A retrospective analysis observed a univariate association between higher SUA on admission (within 48

hours since the symptom onset) and higher thirty-day mortality (fourth versus first quartile SUA values) in AMI patients. It is also reported that an independent association between higher SUA and poorer long-term survival (23). Having in mind, the potential ethnic/racial specificities and cultural differences (eg, diet, alcohol consumption), we aimed to investigate SUA levels determined on admission as a potential predictor of in-hospital outcome (mortality and complications). This study revealed that 38 (69.0%) patients of group A were smoker and 36 (65.4%) patients of group B were smoker; difference was not statistically significant ( $p=0.668$ ). Zahid, et al., (24), in their study reported similar findings. This study showed that 72.0% of total NSTEMI patients were hypertensive where as 78.0% patients of group A and 52.7% patients of group B were hypertensive separately. In this study, there was no significant association ( $p=0.241$ ) between raised serum uric acid level on admission and hypertension in NSTEMI patients. This finding is different from that of other studies which showed that hypertensive patients had more hyperuricemia (Bickel et al., 2002) (25). This study revealed that 33.0% of total NSTEMI had dyslipidaemia with 47.0% patients had dyslipidaemia in group A and 12.7% patients had dyslipidaemia in group B. Patients of group A were more frequently dyslipidaemic than that of group B ( $p<0.001$ ). This study revealed that 34.5% of total NSTEMI patients had family history of CAD with 20.9% patients in group A and 27.2% patients in group B. Patients of group A had more frequent family history of CAD than that of patients of group B ( $p=0.476$ ). Belle, et al., (15), reported that 22.0% of total population of NSTEMI had family history of CAD. Omidvar, et al., (14), found 22.7% patients were diabetic in group A and 31.8% patients were diabetic in group B ( $p=0.882$ ). The present study showed that Killip class I was in 25 (45.0%), Killip class II in 17 (30.9%), Killip class III in 7 (12.7%) and Killip class IV in 6 (10.9%) patients in group A; whereas Killip class I was in 35 (63.6%), Killip class II in 9 (16.3%), Killip class III in 9 (16.3%) and Killip class IV in 2 (3.6%) patients in group B. Killip class did not differ significantly between group A and group B ( $p=0.127$ ). Biswas, et al., (26), found that Killip class-I was less frequent in hyperuricemia

group compared to normal serum uric acid group; whereas Killip class-IV was more frequent in hyperuricemia group compared to normal serum uric acid group. So, their study found that serum uric acid level is low among patients with lower Killip class and high among higher Killip class. Thus, raised serum uric acid level was associated with increased severity of heart failure and reflected the prognosis in essence with a significant  $p$  value (0.05). Hossain, et al., (24), reported in-hospital mortality of acute MI was 7.3% with 14.3% of patients with raised uric acid level and 3% of patients with normal uric acid level. In-hospital mortality of acute MI was significantly higher in patients with raised uric acid level than normal uric acid level ( $p=0.020$ ). Lazzeri, et al., (27), found that in-hospital mortality was higher in “high” SUA patients (9.0% vs. 2.5%),  $p < 0.006$  which is a bit differ from this study. In this regards Hossain et al., (24), found that cardiogenic shock and acute left ventricular failure were significantly more frequent in hyperuricemia group compared to normal serum uric acid level group ( $p=0.037$  and  $p=0.004$  respectively) whereas arrhythmia and heart block did not differ significantly between two groups ( $p=0.545$  and  $p=0.629$  respectively). The large study that showed association of mortality with raised uric acid level included all ACS patients (STEMI+ NSTEMI+ UA) and in-hospital mortality of STEMI patient is more than double than NSTEMI. In this study there were no STEMI patients and also no UA patients. So discrepancy between that study and this is justified as it is established that NSTEMI patients have comparatively lower in-hospital mortality than STEMI patients. The difference may be because of inclusion of all acute Myocardial Infarction cases irrespective of STEMI and NSTEMI in their study but in this study only NSTEMI were included. In-hospital outcome of NSTEMI patients is comparatively better than STEMI patients globally.

#### 4.1 | Conclusion

There is no significant difference between in-hospital outcome (mortality and complications) of NSTEMI patients with raised and normal serum uric acid level. So discrepancy between that study and this is justified as it is established that NSTEMI pa-

tients have comparatively lower in-hospital mortality than STEMI patients.

## REFERENCES

1. Fox KA, Cokkinons DV, Deckers J, Keil U, Maggioni A, Steg G. The ENACT study: a pan-European survey of acute coronary syndromes. *Eur Heart J*. 2000;21:1440–1449.
2. Baber U, Holmes D, Halperin J, Fuster V. Definitions of Acute Coronary Syndrome. In: V F, RA H, J N, ZJ E, editors. *Hurst's The Heart*. 14th Ed. vol. 1. McGraw Hill; 2017. p. 946–995.
3. O'gara PT, Kushner FG, Ascheim DD, Casey DE, Chung MK. ACCF/AHA guideline for the management of ST elevation myocardial infarction: a report of the American College of Cardiology Foundation. American Heart Association Task Force on Practice Guidelines *Circulation*. 2013;127:362–425.
4. Moliterno DJ, Januzzi JL. Evaluation and management of Non -ST- segment elevation Myocardial Infarction. In: V F, RA H, J N, ZJ E, editors. *Hurst's The Heart*. vol. 995. McGraw Hill; 2017. .
5. Libby P, Hansson GK. Inflammation and inflammatory diseases of the arterial tree: Players and layers. *Circ Res*. 2015;116:307–311.
6. Braunwald E. The treatment of acute myocardial infarction: The Past, the Present, and the Future *E ur. Heart J Acute Cardiovasc Care*. 2012;1:9–12.
7. Murray CJ, Lopez AD. Alternative projections of mortality and disability by cause 1990–2020: Global Burden of Disease Study. *Lancet*. 1997;349:1498–1504.
8. Ahmed M, Majumder A, Rahman A, Baqui MA. Relationship between baseline white blood cell count and angiographic severity of coronary artery disease in patients with acute coronary syndrome. *Bangladesh Heart Journal*. 2005;20(1):6–10.
9. Reddy S, Yusuf S. Emerging epidemic of cardiovascular disease in developing countries. *Circulation*. 1998;97:596–601.
10. Bangladesh Cardiac Society ACS: Guideline for management. 2004;(5).
11. Yusuf S, Rangarajan S, Teo K, Islam S, Li W. Cardiovascular risk and events in 17 low-, middle-, and high-income countries. *The New England Journal of Medicine*. 2014;371:818–827.
12. Islam M, Bhattacharjee B, Chowdhury M, Siddique AN, Karim A. Outcome of Acute Myocardial Infarction Patients Admitted in a Tertiary Care Hospital. *Medicine Today*. 2016;28:6–8.
13. Iliesiu A, Campeanu A, Dusceac D. Serum uric acid and cardiovascular disease. *Maedica*. 2010;5:186–192.
14. Omidvar B, Ayatollahi F, Alasti M. The prognostic role of serum uric acid level in patients with acute ST elevation myocardial infarction. *J Saudi Heart Assoc*. 2012;24:73–78.
15. Belle L, Cayla G, Cottin Y, Coste P, Khalife K. French Registry on Acute ST-elevation and non-ST-elevation Myocardial Infarction 2015 (FAST- MI 2015). Design and baseline data. *Arch Cardiovasc Dis*. 2017;110:366–378.
16. Safi AJ, Mahmood R, Khan A. Association of serum uric acid with type 2 diabetes mellitus. *Journal of Postgraduate Medical Institute*. 2004;18:59–63.
17. Das A, Dhandapani E, Sugumar A, Manam A. Study of serum uric acid levels in acute myocardial infarction. *International Journal of Pharma and Bio Sciences*. 2015;6:222–226.
18. Dharma S, Siswanto BB, Soerianata S, Wardeh AJ, Jukema JW. Serum uric acid as an independent predictor of cardiovascular events in patients with acute ST-elevation myocardial infarction. *J Clinic Experiment Cardiol S*. 2012;5:148–156.

19. Omidvar B, Ayatollahi F, Alasti M. The prognostic role of serum uric acid level in patients with acute ST-elevation myocardial infarction. *J Saudi Heart Assoc.* 2012;24:73–78.
20. Brodov Y, Chouraqui P, Goldenberg I, Boyko V, Mandelzweig L. Serum uric acid for risk stratification of patients with coronary artery disease. *Cardiology.* 2009;114:300–305.
21. Maxwell AJ, Bruinsma KA. Uric acid is closely linked to vascular nitric oxide activity: Evidence for mechanism of association with cardiovascular disease. *J Am Coll Cardiol.* 2011;38:1850–1858.
22. Weir CJ, Muir SW, Walters MR, Lees KR. Serum urate as an independent predictor of poor outcome and future vascular events after acute stroke. *Stroke.* 2003;34:1951–1956.
23. Kojima S, Sakamoto T, Ishihara M, Kimura K, Miyazaki S. Prognostic Usefulness of Serum Uric Acid After Acute Myocardial Infarction (The Japanese Acute Coronary Syndrome Study). *Am J Cardiol.* 2005;96:489–495.
24. Hossain MD, Ferdousi S, Islam MS, Paul D, Sultana T. Prognostic value of serum Uric Acid in-hospital Mortality and Morbidity in Patients with acute Myocardial Infarction. *University Heart Journal.* 2016;12:8–11.
25. Zahid MA, Khan H, Chowdhury AW, Sabah K, Kabir S. Demographic Profile of NSTEMI (Non ST Elevation Myocardial Infarction) Patients & Association of ST-Segment Depression and Level of Troponin I with NSTEMI Patient's In-Hospital Outcome. *Medicine Today.* 2015;27:14–19.
26. Biswas K, Halder S, Sarkar R, Roy K. A study on prognostic significance of serum uric acid in acute myocardial infarction in a tertiary care institute. *International Journal of Research in Medical Sciences.* 2016;4:4557–4562.
27. Lazzeri C, Valente S, Chiostrri M, Sori A, Bernardo P. Uric acid in the acute phase of ST elevation myocardial infarction submitted to primary PCI: Its prognostic role and relation with inflammatory markers: A single center experience. *Int J Cardiol.* 2010;138:206–219.

**How to cite this article:** Hossain S.Z., M.I.H.K., M.A.S.A., M.A.K., M.T.H., A.B.M.J. K.H.R. “To Assess Out-come of NSTEMI Patients with Raised Serum Uric Acid Level”. *Journal of Medical Research and Health Sciences.* 2021;1418–1424. <https://doi.org/10.52845/JMRHS/2021-4-8-6>