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Serum Uric Acid Level and Its Association with Risk Factors and In-Hospital Mortality in Patients with Acute Coronary Syndrome at a Tertiary Hospital in Birgunj, Nepal: A Hospital-Based Cross-Sectional Study

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ABSTRACT

Background: Conventional risk factors explain only part of the acute coronary syndrome (ACS) burden, and the role of serum uric acid (SUA) in ACS remains debated. Hospital-based data from Nepal are scarce. This study examined the association of SUA with risk factors, clinical presentation, and in-hospital outcome in patients with ACS.

Methods: This hospital-based cross-sectional study was conducted in the Emergency Department and Intensive Care Unit of National Medical College and Teaching Hospital, Birgunj, from July 2020 to June 2021. A total of 114 adults with ACS who presented within 24 hours of symptom onset were enrolled by purposive sampling. Hyperuricaemia was defined as SUA above 7 mg/dL in men and above 6 mg/dL in women. Demographic, clinical, and laboratory data were recorded on a proforma, and patients were followed until discharge or death. Data were analysed in SPSS version 23 using the Pearson chi-square test, with $p<0.05$ considered significant.

Results: The mean age was 56.8±12.3 years and 61.4% of patients were men. Exactly half (n=57) were hyperuricaemic. Hyperuricaemia was more common in patients with hypertension (p=0.014), alcohol use (p=0.037), hyperlipidaemia (p=0.002), and a family history of ischaemic heart disease (p<0.001). Hyperuricaemic patients more often had ST-elevation myocardial infarction (86.0% vs 68.4%, p=0.026) and pulmonary oedema on chest radiograph (22.8% vs 7.0%, p=0.018). Fourteen patients (12.3%) died in hospital, 12 of them with hyperuricaemia (21.1% vs 3.5%, p=0.004).

Conclusion: Hyperuricaemia was present in half of the patients with ACS, clustered with several conventional risk factors, and accompanied most in-hospital deaths. SUA is a cheap and widely available test that may support early risk assessment in resource-limited emergency settings, but these unadjusted findings from a single centre need confirmation in larger prospective studies.

Keywords: *Acute coronary syndrome; Serum uric acid; Hyperuricaemia; Risk factors; In-hospital mortality; Nepal.*

INTRODUCTION

Cardiovascular disease is the leading cause of death worldwide, and its burden is shifting steadily towards low- and middle-income countries such as Nepal. [1] Acute coronary syndrome (ACS), which covers unstable angina and myocardial infarction with or without ST-segment elevation, is its most urgent presentation and a common reason for emergency admission. [2] Smoking, dyslipidaemia, hypertension, diabetes, and physical inactivity are the established risk factors, yet they do not fully account for the rising incidence of ischaemic heart disease (IHD), including in Nepalese hospital series. [3]

Uric acid is the end product of purine metabolism, produced by xanthine oxidase, an enzyme whose activity generates oxygen-derived free radicals. Xanthine oxidase activity and urate production rise under ischaemic conditions, so a raised serum level could simply mark tissue ischaemia. Epidemiological work has linked higher SUA with cardiovascular events and death in the general population, [4,5] and several admission-based studies have reported that it predicts worse outcomes after ACS. [6,7] Other authors regard it as an innocent bystander that travels with hypertension, obesity, diabetes, and dyslipidaemia, which leaves the question of causality open.

Evidence on the relation between SUA and ACS in Nepal is limited. [8] The present study therefore examined the association of SUA with sociodemographic characteristics, conventional risk factors, clinical presentation, and in-hospital outcome among patients with ACS presenting to the emergency department of a tertiary hospital in the Terai region.

MATERIALS & METHODS

Study design and setting: This was a prospective, hospital-based, cross-sectional study conducted in the Department of Emergency Medicine and the Intensive Care Unit of National Medical College and Teaching Hospital (NMCTH), Birgunj, Parsa, Nepal, over 12 months from July 2020 to June 2021. NMCTH is a Tribhuvan University-affiliated tertiary centre in the central Terai that also serves the adjoining districts of Bihar, India.

Study population: All patients with ACS presenting to the Emergency Department, outpatient department, or Intensive Care Unit of the hospital during the study period were considered for enrolment.

Sample size and sampling technique: The sample size was calculated with the formula $n=Z^2p(1-p)/e^2$, using $Z=1.96$ for 95% confidence, an estimated prevalence (p) of 8%, and a precision (e) of 5%. This gave 113.09, rounded to 114. Participants were selected by non-probability purposive sampling.

Inclusion criteria: (i) Patients aged 18 years or above; (ii) a diagnosis of ACS supported by electrocardiogram, cardiac enzymes, and clinical signs and symptoms; (iii) presentation within 24 hours of symptom onset; (iv) written informed consent.

Exclusion criteria: (i) Previous history of ACS; (ii) chronic renal failure; (iii) gout; (iv) use of thiazide diuretics; (v) haematological abnormalities such as leukaemia or other myeloproliferative disorders; (vi) refusal to consent.

Data collection procedure: After written informed consent was obtained from the patient or a caregiver, each patient underwent a detailed history and clinical examination in the emergency room. Patients were admitted to the Intensive Care Unit after severity assessment under the emergency protocol. Sociodemographic details, chief complaints, comorbidities (hypertension, hyperlipidaemia), alcohol use, family history of IHD, troponin I, type of myocardial

infarction, chest radiograph findings, and SUA were recorded on a pre-designed proforma. Further investigation and treatment were decided by the treating cardiologist. All patients were followed until discharge or in-hospital death.

Operational definitions: Hyperuricaemia was defined as SUA above 7 mg/dL in men and above 6 mg/dL in women; patients below these thresholds were classified as having normal uric acid. Myocardial infarction was classified as ST-elevation (STEMI) or non-ST-elevation (NSTEMI) according to the universal definition. [2]

Ethical considerations: The study followed the Declaration of Helsinki and was approved by the Institutional Review Committee of NMCTH. Written informed consent was obtained from all participants or their caregivers, and confidentiality was maintained.

Data analysis: Data were analysed in IBM SPSS Statistics version 23. Continuous variables were summarised as mean±standard deviation and categorical variables as frequencies and percentages. Associations between uric acid status (normal or high) and categorical variables were tested with the Pearson chi-square test, and mean age was compared with the independent-samples t-test. All tests were two-sided and a p-value below 0.05 was taken as statistically significant. Comparisons are unadjusted.

RESULT

A total of 114 patients with ACS were included. Their mean age was 56.8±12.3 years (range 30 to 90 years). The 51 to 60 year group was the largest (28.1%), followed by the 41 to 50 and 61 to 70 year groups (23.7% each). There were 70 men (61.4%) and 44 women (38.6%), a male to female ratio of 1.6:1.

Half of the patients (n=57) were hyperuricaemic. Mean SUA was 7.36±0.91 mg/dL in the hyperuricaemic group and 5.21±0.93 mg/dL in the normal group. Hyperuricaemia was found in 30 of 70 men (42.9%) and 27 of 44 women (61.4%) (p=0.054). Patients with hyperuricaemia were slightly older (58.2±13.6 vs 55.4±10.9 years, p=0.22), and the proportion with hyperuricaemia was 60.5% (26/43) above 60 years and 43.7% (31/71) at 60 years or below (p=0.082). Neither difference reached statistical significance.

Table 1: Age and sex distribution of patients according to serum uric acid status (N=114)

Variable	Category	Normal UA (n=57) n (%)	High UA (n=57) n (%)	Total (N=114) n (%)	p-value
Age group (years)	31–40	4 (7.0)	8 (14.0)	12 (10.5)	
	41–50	18 (31.6)	9 (15.8)	27 (23.7)	
	51–60	18 (31.6)	14 (24.6)	32 (28.1)	
	61–70	11 (19.3)	16 (28.1)	27 (23.7)	
	71–80	5 (8.8)	9 (15.8)	14 (12.3)	
	81–90	1 (1.8)	1 (1.8)	2 (1.8)	
Age, years (mean±SD)	—	55.35±10.89	58.19±13.57	56.77±12.33	0.22
Sex	Male	40 (70.2)	30 (52.6)	70 (61.4)	0.054
	Female	17 (29.8)	27 (47.4)	44 (38.6)	

Hypertension was the most frequent risk factor (65 patients, 57.0%). Hyperuricaemia was present in 60.0% of hypertensive patients and 36.7% of normotensive patients (p=0.014). A family history of IHD, reported by 47 patients (41.2%), showed the strongest association: 78.7% of these patients were hyperuricaemic compared with 29.9% of those without such a history (p<0.001). Hyperlipidaemia (40 patients, 35.1%) was associated with hyperuricaemia in 70.0% against 39.2% of patients without it

(p=0.002), and alcohol use (32 patients, 28.1%) in 65.6% against 43.9% (p=0.037).

Table 2: Association of serum uric acid status with risk factors (N=114)

Risk factor	Category	Normal UA (n=57) n (%)	High UA (n=57) n (%)	p-value
Hypertension	Yes	26 (45.6)	39 (68.4)	0.014
	No	31 (54.4)	18 (31.6)	
Alcohol use	Yes	11 (19.3)	21 (36.8)	0.037
	No	46 (80.7)	36 (63.2)	
Family history of IHD	Yes	10 (17.5)	37 (64.9)	<0.001
	No	47 (82.5)	20 (35.1)	
Hyperlipidaemia	Yes	12 (21.1)	28 (49.1)	0.002
	No	45 (78.9)	29 (50.9)	

Chest pain was the leading complaint (101 patients, 88.6%), followed by vomiting (36, 31.6%) and palpitation (26, 22.8%). Chest pain was reported by 94.7% of hyperuricaemic and 82.5% of normal-UA patients (p=0.039), whereas vomiting and palpitation did not differ between the groups (p=0.227 and p=0.655). Troponin I was positive in 101 patients (88.6%) and negative in 13 (11.4%), with positivity more common in the hyperuricaemic group (94.7% vs 82.5%, p=0.039). STEMI accounted for 88 cases (77.2%) and NSTEMI for 26 (22.8%); STEMI was more frequent with hyperuricaemia (86.0% vs 68.4%, p=0.026). Chest radiographs showed pulmonary oedema in 17 patients (14.9%), 13 of them in the hyperuricaemic group (22.8% vs 7.0%, p=0.018).

Table 3: Clinical presentation, cardiac findings, and in-hospital outcome by serum uric acid status (N=114)

Variable	Category	Normal UA (n=57) n (%)	High UA (n=57) n (%)	p-value
Chest pain	Yes	47 (82.5)	54 (94.7)	0.039
	No	10 (17.5)	3 (5.3)	
Palpitation	Yes	14 (24.6)	12 (21.1)	0.655
	No	43 (75.4)	45 (78.9)	
Vomiting	Yes	15 (26.3)	21 (36.8)	0.227
	No	42 (73.7)	36 (63.2)	
Troponin I	Positive	47 (82.5)	54 (94.7)	0.039
	Negative	10 (17.5)	3 (5.3)	
Type of MI	STEMI	39 (68.4)	49 (86.0)	0.026
	NSTEMI	18 (31.6)	8 (14.0)	
Chest radiograph	Normal	53 (93.0)	44 (77.2)	0.018
	Pulmonary oedema	4 (7.0)	13 (22.8)	
In-hospital death	Yes	2 (3.5)	12 (21.1)	0.004
	No	55 (96.5)	45 (78.9)	

Fourteen patients (12.3%) died in hospital, and 12 of them were hyperuricaemic. In-hospital mortality was 21.1% (12/57) in the hyperuricaemic group against 3.5% (2/57) in the normal group (χ^2=8.14, p=0.004; unadjusted odds ratio 7.3, 95% CI 1.6 to 34.5). The wide interval reflects the small number of deaths.

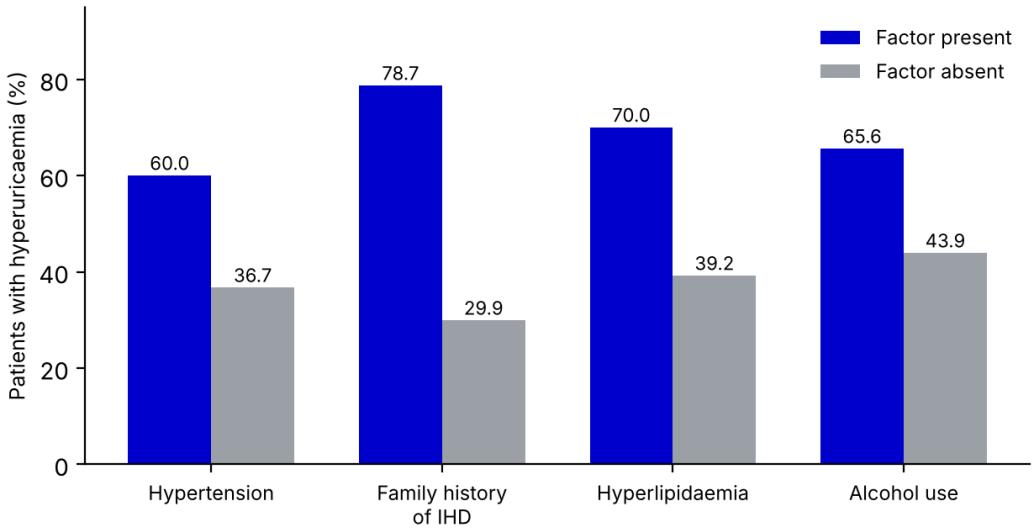

Figure 1: Proportion of patients with hyperuricaemia by presence or absence of selected risk factors

DISCUSSION

In this hospital-based study of 114 patients with ACS, half were hyperuricaemic on admission. Hyperuricaemia clustered with hypertension, a family history of IHD, hyperlipidaemia, and alcohol use, and it was more frequent in patients with STEMI, pulmonary oedema, and in-hospital death. Conventional risk factors explain only part of the ACS burden, which is why markers such as SUA have drawn sustained interest since the Framingham and NHANES I cohorts linked it to cardiovascular disease and mortality. [4,5]

The mean age of 56.8 years is close to the 58.3±11.3 years reported by Nadkar and Jain in India and a little below the 62±10 years in the series of Bickel et al. [9,10] Serum uric acid is known to rise with age in the general population. [11] In our patients the age difference between the two groups was modest and not statistically significant, although the proportion with hyperuricaemia was higher above 60 years. The findings on sex in the literature are mixed. Nadkar and Jain found no difference, Kojima et al. and Bickel et al. reported higher levels in men (Bickel et al. attributing part of this to lower values in younger women), Longo-Mbenza et al. found significantly higher levels in men, and Marak et al. found higher levels in women. [7,9,10,12,13] With sex-specific thresholds, hyperuricaemia was more common among our women (61.4% vs 42.9%), though the difference was borderline (p=0.054).

Hypertension was associated with hyperuricaemia in our cohort, in line with the Japanese Acute Coronary Syndrome Study and with population data on clustering of urate with cardiovascular risk factors. [7,14] Bickel et al. saw only a trend, and Nadkar and Jain found no difference between hypertensive and normotensive patients, so the evidence is not uniform. [9,10] The strongest association here was with a family history of IHD, which Kojima et al. and Nadkar and Jain also observed. [7,9] Hyperlipidaemia was likewise associated with hyperuricaemia, in keeping with the clustering of urate with other cardiovascular risk factors. Alcohol was associated with hyperuricaemia (p=0.037), which fits the known effect of alcoholic drinks on urate metabolism, [15] but the result is borderline: it does not survive a continuity correction (p=0.061), and Karki et al. found no association in a Nepalese cohort (p=0.11). [8]

Hyperuricaemic patients more often presented with STEMI and chest pain and were more likely to have pulmonary oedema on the admission radiograph. This agrees with the report that SUA helps to separate acute myocardial infarction from unstable

angina in hyperuricaemic patients with ACS, [16] and with the higher rate of heart failure after infarction among patients with raised urate in the series of Nadkar and Jain. [9] Most importantly, 12 of the 14 in-hospital deaths occurred in the hyperuricaemic group. Higher admission SUA has been linked to in-hospital and short-term mortality after ACS in the Japanese Acute Coronary Syndrome Study, in a Croatian cohort of 621 patients (RR 1.016 per unit, 95% CI 1.001 to 1.031), and in more recent cohorts. [7,17–19] Other studies point the same way. [6,20] Our estimate (odds ratio 7.3) is unadjusted, rests on only 14 deaths, and has a very wide confidence interval; age, renal function, infarct size, and treatment were not accounted for and may explain part of the difference.

Several mechanisms could link urate to coronary events. Xanthine oxidase activity increases during ischaemia and produces reactive oxygen species, [21] and hyperuricaemia has been shown to impair endothelial function. [22] In patients with a first episode of ACS, uric acid has been associated with endothelial dysfunction and with the severity of coronary atherosclerosis, [23] and with carotid atherosclerosis and the metabolic syndrome in Japanese adults. [24] Uric acid is also a potent antioxidant, [25] so a raised level may reflect oxidative stress rather than cause harm. Our cross-sectional data cannot separate these possibilities.

If confirmed, the practical appeal of SUA is that it is cheap and available in almost every Nepalese hospital laboratory. It could add to early risk assessment in emergency departments where advanced biomarkers and imaging are scarce.

Limitations

This was a single-centre study with a small sample selected by purposive sampling, which limits generalisability. There was no control group without ACS, so we cannot say whether SUA is higher in ACS than in healthy people. Serum uric acid was measured once, and the timing relative to symptom onset and to treatment was not analysed. Treatment details such as thrombolysis and percutaneous coronary intervention, which may change outcome, were not recorded, and only in-hospital outcome was assessed. The analyses were unadjusted bivariate comparisons with few events, and some associations were borderline.

CONCLUSION

Half of the patients with ACS in this tertiary hospital had hyperuricaemia on admission. It was associated with hypertension, family history of IHD, hyperlipidaemia, and alcohol use, and it accompanied 12 of 14 in-hospital deaths. SUA may serve as a simple adjunct for early risk assessment in resource-limited emergency settings. Larger multicentre prospective studies with a control group, multivariable adjustment, and recorded treatment details are needed to establish its prognostic value.

Declaration by Authors

Ethical Approval: Approved by the Institutional Review Committee of National Medical College and Teaching Hospital, Birgunj

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Conflict of Interest: The authors declare no conflict of interest.

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