

Original Research Article

Evaluating serum uric acid levels in patients with acute myocardial infarction

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ABSTRACT

Background: Elevated levels of serum uric acid are associated with increased cardiovascular morbidity and mortality. However, this association with cardiovascular diseases is still unclear, and perhaps controversial. The objective of study was to assess the serum uric acid level in patients with Acute Myocardial Infarction (AMI).

Methods: Sixty patients with AMI were studied in Department of Medicine/ Department of Cardiology, J. A. Group of Hospitals between 2016 -2018. Details of age, sex, smoking, alcohol consumption and history of ischemic heart disease (IHD) was obtained and recorded. Serum uric acid level was estimated and compared with control group (healthy subjects).

Results: Serum uric acid level was significantly higher among AMI patients (6.43 ± 2.60) as compared to control group (4.05 ± 0.95) ($p < 0.001$). Majority (46.7%) of the AMI patients had uric acid level of > 7.1 followed by 20% patients who had uric acid level between 4.5-5.9 ($p < 0.001$). Uric acid level was comparable between smoker and non-smokers ($p = 0.803$), alcoholic and non-alcoholic ($p = 0.086$), hypertensive and non-hypertensive ($p = 0.668$), patients with and without diabetes ($p = 0.278$) and patients with a history of IHD and without history of IHD ($p = 0.403$).

Conclusions: Serum uric acid may be useful for prognostication among those with pre-existing AMI.

Keywords: Cardiovascular disease, Heart failure, Hyperuricemia, Smoking

INTRODUCTION

Heart failure is a burgeoning problem worldwide, with more than 20 million people affected. The overall prevalence of HF in the adult population in developed countries is 2%.¹ Heart failure prevalence follows an exponential pattern, rising with age, and affects 6–10% of people over age 65.²

Earlier studies have shown that heart failure is often associated with hyperuricemia. Hyperuricemia is associated with worse hemodynamic measures such as increased left atrial pressure and decreased cardiac index among patients with primary pulmonary hypertension,

cor pulmonale and dilated cardiomyopathy in a small case series.³ Among those with established heart failure, hyperuricemia is a risk factor for adverse outcomes including mortality.⁴

Serum uric acid may be useful for prognostication among those with preexisting heart failure. Hyperuricemia can predict heart failure among those with preexisting hypertension. There have not been any studies that examined hyperuricemia as independent risk factors for heart failure risk among the general population. The single available study from Austria, did not account for confounders such as valvular heart disease and diuretics, and renal disease suggested that highest quintiles of

serum uric acid was associated with elevated risk for death from heart failure.⁵ Hence present study was an attempt to evaluate serum uric acid levels in patients with acute myocardial infarction.

METHODS

A prospective cross sectional study was performed on 60 patients diagnosed as a case of acute myocardial infarction (STEMI, NSTEMI) on the basis of clinical history, examination, ECG changes, biochemical marker who was admitted in ICCU in Department of Medicine/ Department of Cardiology, J.A. Group of Hospitals from 2016 -2018. As a control group 40 healthy subjects were also included.

In all the cases written informed consent was obtained from each subjects. Institutional Ethics Committee approval was obtained before starting the study.

The patients included in the study were selected consecutively among those admitted with acute STEMI having resting chest pain lasting more than 30 min, typical ischemic ST elevation in electrocardiogram (ECG) leads and rise of serum cardiac enzymes concentration (CK-MB and Troponins). We excluded patients who did not receive thrombolytic therapy during the first six hours after the onset of chest pain, were in cardiogenic shock, had previous pacemaker implantation, had a recent myocardial infarction (<3 months), had severe valvular disease, had impaired renal function (serum creatinine level >1.5 mg/dl) and was a known cases of hypothyroidism, malignancy, gout or other inflammatory diseases and were using corticosteroid or cytotoxic drugs.

Investigations including routine haemogram (Hb, total leucocyte count, differential count), renal function test, blood sugar (random, and/or fasting, post prandial), 12 leads electrocardiogram, troponin T or troponin I, serum uric acid (on day 1,3,5), lipid Profile and liver function tests were performed and results were recorded.

Hypertension was defined as resting blood pressure persistently at or above 130/80 or 140/90 mmHg. Diabetes mellitus was defined as fasting plasma glucose level ≥ 126 mg/dl, plasma glucose ≥ 200 mg/dl two hours after a 75 g oral glucose load as in a glucose tolerance test, glycated hemoglobin (HbA1C) $\geq 6.5\%$ and in serum uric acid level, normal uric acid levels was considered as 2.4-6.0 mg/dL (female) and 3.4-7.0 mg/dL (male).

All the data analysis was done using IBM SPSS ver. 20 Software. Cross tabulation and frequency distribution was used to prepare tables. Microsoft office 2010 was used to prepare the graphs. Paired sample t test and one way ANOVA and paired t test was used to compare the mean whereas categorical data was compare using Chi square test. Level of significance was assessed at 5%.

RESULTS

Majority of the subjects in case (28.3%) and control (40%) groups belonged to age group of 51-60 years ($p=0.248$). Among cases and control majority of the subjects were males (65% vs. 67.5% respectively) ($p=0.689$). Most common complaint of patients was chest pain (86.7%).

Table 1: Comparing uric acid level between groups.

Uric acid level (mg per 100 ml)	Cases	Control	P value
<4.5	12 (20)	25 (62.5)	<0.001
4.5-5.9	12 (20)	14 (35)	
6.0-7.0	8 (13.3)	1 (2.5)	
>7.1	28 (46.7)	0 (0)	
Total	60 (100)	40 (100)	

Majority of the cases had uric acid level of >7.1 (46.7%) followed by 12 patients who had uric acid level between 4.5-5.9. Among Control group maximum subjects had uric acid level <4.5 (62.5%). The comparison was highly significant ($p<0.001$). Mean Serum uric acid was significantly higher among cases (6.43 ± 2.60) as compared to control group (4.05 ± 0.95) ($p<0.001$).

No significant difference was obtained between mean uric acid level in different age groups in present study ($p=0.685$). Similarly mean uric acid level was comparable between both gender ($p=0.272$).

Table 2: Comparing serum uric acid level with risk factors.

Risk factor	IHDC	Mean	p value
Smoking	N (n=33)	6.94 \pm 3.00	0.803
	Y (n=23)	7.13 \pm 2.74	
Alcohol	N (n=55)	6.84 \pm 2.82	0.086
	Y (n=5)	9.14 \pm 2.70	
Hypertension	N (n=52)	7.09 \pm 3.02	0.668
	Y (n=8)	6.62 \pm 1.63	
Diabetes mellitus	N (n=48)	7.23 \pm 3.00	0.278
	Y (n=12)	6.22 \pm 2.18	
IHD	N (n=40)	6.81 \pm 2.86	0.403
	Y (n=20)	7.47 \pm 2.89	

Uric acid level was comparable between smoker and non-smokers ($p=0.803$), alcoholic and non-alcoholic ($p=0.086$), hypertensive and non-hypertensive ($p=0.668$), patients with and without diabetes ($p=0.278$) and patients with a history of IHD and without history of IHD ($p=0.403$).

DISCUSSION

Uric acid is an independent predictor of major adverse cardiovascular events (MACE) in patients with coronary

artery disease. High serum uric acid causes increasing platelet reactivity mediating inflammation and stimulation of smooth muscle cell proliferation, which probably worsens acute thrombosis.⁶ In present study we tried to evaluate serum uric acid levels in patients with AMI.

Majority of the subjects in case (28.3%) and control (40%) group belong to age group of 51-60 years. Behera et al in a similar study reported that mean age of presentation of STEMI patients was 50.4 ± 13.1 years.⁷ Similar reports were depicted in south Asian data where mean age of first acute myocardial infarction was 53.0 ± 11.4 .⁸

Among cases and control majority of the subjects were males (65% vs. 67.5% respectively). In line with that Harris et al found that out 100 cases there were 77% were males and 23% were females.⁵ This highlight that acute MI is predominantly observed in male population and which may be due to higher stress and social life among males.

Majority of the cases had uric acid level of >7.1 (46.7%) followed by 12 patients who had uric acid level between 4.5-5.9. Among control group maximum subjects had uric acid level <4.5 (62.5%). The comparison was highly significant ($p < 0.001$). Mean serum uric acid was significantly higher among cases (6.43 ± 2.60) as compared to control group (4.05 ± 0.95) ($p < 0.001$).

In present study we found that mean uric acid level was comparable between both gender ($p = 0.272$). Contrary to present study findings Padma et al studied 100 patients with acute myocardial infarction and compared with 100 controls. Serum uric acid level was measured on day 0, 3 and 7 of MI and results were analysed. Padma et al reported that males had higher uric acid levels as compared to females.⁹ similar finding were seen in another study.⁸

Uric acid level was comparable between smoker and non-smokers ($p = 0.803$), alcoholic and non-alcoholic ($p = 0.086$), hypertensive and non-hypertensive ($p = 0.668$), patients with and without diabetes ($p = 0.278$) and patients with a history of IHD and without history of IHD ($p = 0.403$). A study from Japan by Kojima et al noted that hyperuricemia after AMI is associated with the development of heart failure.¹⁰ However, Jularattanaporn et al noted that there was no observed association between hyperuricemia and in-hospital adverse outcomes.¹¹

Bickel et al reported that one mg/dl increase in serum uric acid levels was associated with a 26% increase in mortality.⁸ Jacobs D et al in his study found that hyperuricaemia correlated strongly as an associated risk factor in MI.¹² Author stated that serum uric acid is a variable, subject to modification by a large array of complex and often associated factors and suggested that

possible risk factors such as hyperuricaemia be assessed and treated as a routine, so as to possibly reduce the incidence of MI. However, Sokhanvar et al concluded that there was a meaningful relation between hyperuricaemia and MI wherein serum uric acid behaved as an independent variable and had no relationship with other risk factors.¹³

Present study has few limitations. First cross sectional nature of the present study was the main limitation which restricts the use of present study findings to large population. Second is the small sample size; a large randomize clinical trial is required to strengthen the present study findings.

CONCLUSION

It was found that male patients who were living in their 5th to 6th decade of life had more risk of AMI. Serum uric acid levels are elevated in systemic hypertension, diabetes mellitus and in acute myocardial infarction. Serum uric acid level of >7.1 should be suspected as the risk. However, age and gender of the patients did not have any association with serum uric acid level.

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Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Wannamethee SG, Papacosta O, Lennon L, Whincup PH. Serum uric acid as a potential marker for heart failure risk in men on antihypertensive treatment: The British Regional Heart Study. *Int J Cardiol.* 2018;252:187-92.
2. Kenchaiah S, Narula J, Vasan RS. Risk factors for heart failure. *Med Clin North Am.* 2004;88:1145-72.
3. Miller WL, Hartman KA, Burritt MF. Serial biomarker measurements in ambulatory patients with chronic heart failure: the importance of change overtime. *Circulation.* 2007;116:249-57.
4. Leyva F, Anker S, Swan JW, Godsland IF, Wingrove CS, Chua TP et al. Serum uric acid as an index of impaired oxidative metabolism in chronic heart failure. *Eur Heart J.* 1997;18:858-65.
5. Harris P, Feroz P, Jenner, Sunil Kumar. Serum uric acid as a marker of left ventricular failure in acute myocardial infarction. (IOSR-JDMS). 2015;14(11):102-09.
6. Joshi P, Islam S, Pais P, Reddy S, Dorairaj P, Kazmi K et al. Risk Factors for Early Myocardial Infarction in South Asians Compared With Individuals in Other Countries. *JAMA.* 2007;297(3):286-94.
7. Behera SK, Samal AK. Study of serum uric acid level as a prognostic marker in acute ST elevation myocardial infarction patients. *Int J Adv Med.* 2018;5:592-6.

8. Bickel C, Rupprecht HJ, Blankenberg S, Rippin G, Hafner G, Daunhauer A, et al. Serum uric acid as an independent predictor of mortality in patients with angiographically proven coronary artery disease. *Am J Cardiol*. 2002;89(1):12-7.
9. Padma V, Banupriya A. Serum uric acid levels in acute myocardial infarction. *Int J Adv Med*. 2017;4:1010-3.
10. Kojima S, Sakamoto T, Ishihara M. Prognostic usefulness of serum uric acid after acute myocardial infarction (Japanese Acute Coronary Syndrome Study). *Am J Cardiol*. 2005;96:489-95.
11. Jularattaporn V, Krittayaphong R, Boonyasirinant T, Udol K, Udompunurak S. Prevalence of Hyperuricemia in Thai Patients with Acute Coronary Syndrome. *Thai Heart J* 2008;21:86-92.
12. Jacobs D. Hyperuricaemia and myocardial infarction. *S Afr Med J*. 1972;46:367-9.
13. Sokhanavar S, Maleki A. Blood uric acid levels according to cardiovascular disease risk factors in patients with myocardial infarction. *Iranian Heart J*. 2007;8(1):43-5.

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