International Journal of Scientific Research in Dental and Medical Sciences

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Evaluation of the Serum Uric Acid Level as an Early Predictor of Mortality Among the Patients with Myocardial Infarction: A Prospective Study

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ARTICLE INFO

Article history:
Received 03 April 2022
Received in revised form 25 May 2022
Accepted 06 June 2022
Available online 08 June 2022

Keywords:
Body Mass Index
Myocardial Infarction
Uric Acid

ABSTRACT

Background and aim: Myocardial infarction (MI) is one of the most common causes of death through the globe. Serum uric acid (SUA) is elevated in underlying tissue ischemia. This research conducted to establish the association of SUA extents with MI patients admitted to a tertiary care hospital and to compare SUA levels with Killip Classification as an early prognostic marker in these patients.

Material and methods: Our study involved 100 consecutive AMI patients and 100 age- and sex-matched controls. SUA levels were estimated on the 0th, 3rd, and 7th days of MI and compared with the Killip class, indicative of the severity of heart failure. SUA was measured by the uricase method.

Results: The present study has been undertaken to correlate serum uric acid level as a prognostic marker in patients with myocardial infarction. In myocardial infarction, the higher the uric acid level higher is the risk of mortality. In our study, MI and hypertensive patients had significantly higher SUA levels on days 0, 3, and 7. Also, our study showed SUA levels were significantly larger in patients who belonged to a greater Killip class (Killip III and IV). Among the ten patients who died, eight had SUA levels>7.0 mg/dL during the admission. Among those ten cases, nine patients were in the higher Killip class and one in Killip I class.

Conclusions: Patients with a history of MI and hypertensive have higher SUA levels and are in the greater Killip class. A union of Killip class and SUA after AMI is a good predictor of mortality.

1. Introduction

Insufficient blood flow to the heart develops coronary heart disease (CHD) or ischemic heart disease, caused by an obstruction in the coronary artery. MI is specific to CHD. Coronary heart disease is a global epidemic and causes 25% to 30% of all deaths in developed nations. There has been a considerable increase in the prevalence of CHD in urban India and, to a lesser extent, in rural India in the past few decades due to rapid lifestyle changes. It is estimated that over.[1] Six million people in India died of CHD in 2016. CHD is associated with no modifiable risk factors such as male sex, family history, genetic factor, and modifiable risk factors like Diabetes, dyslipidemia, hypertension, smoking, sedentary habit, obesity, and stress.[1] Several novel biomarkers are recently used for the diagnosis and risk assessment in patients with MI. Among them, cardiac troponin is considered an ideal cardiac biomarker for AMI diagnosis and can independently predict death and heart failure. Other valuable biomarkers such as BNP/NT-pro BNP and CRP are also predictors of cardiovascular prognosis.[2] However, the different prognostic markers used in MI are costly and not easily accessible. Uric acid represents the oxidized output of both endogenous and exogenous purine metabolism in humans through the action of the enzyme xanthine oxidase. Uric acid can act as a major antioxidant in the human plasma or as a pro-oxidant within the cell.[3] Uric acid exhibits its antioxidant property through the interaction with peroxynitrite (ONOO−) in the extracellular space to form a strong nitric oxide donor, thus promoting vasodilatation and preventing oxidative damage.[4] However, UA was found to generate

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http://doi.org/10.30485/IJSRDMS.2022.340472.1293

superoxide and peroxide free radical, promote LDL oxidation, stimulate granulocyte adhesion to endothelium, and accumulate as a crystal within the atherosclerotic plaque.[5-9] Thus, uric acid's pro-oxidant and pro-inflammatory properties could be responsible for direct or indirect vascular injury, and elevated SUA may contribute to the pathogenesis of cardiovascular diseases. Adenosine is synthesized and released generally at a constant rate by myocardium and vascular myocytes. Adenosine on attachment to particular receptors causes relaxation of vascular smooth muscle and arteriolar vasodilatation.[10] Cardiac and visceral ischemia stimulates the increased production of adenosine, which plays a pivotal role in restoring the blood flow, thus restricting the ischemia.[11] Adenosine produced generally by the vascular smooth muscle is quickly degraded to uric acid, rapidly effluxing to the vascular lumen because of low intracellular pH and negative membrane potential.[12] Xanthine oxidase activity and uric acid production are grown in vivo under ischemic conditions, and therefore elevated SUA may be considered a marker of underlying tissue ischemia.[13] Uric acid may also trigger the RAS, contributing to vascular smooth cell growth, deterioration of arterial function, and stiffening.[14] It sounds fascinating that medical attention to chronic heart failure patients with allopurinol can reestablish the endothelial function.[15] The association between SUA and CVD is noticed not only with frank hyperuricemia (defined as >6mg/dL in the female and >7mg/dL in the male) but also with uric acid levels considered range increasing (>5.2 to 5.5 mg/dL).[15-17] Pastre search papers have indicated that SUA levels become greater in cardiac failure.[18, 19] However, very few studies have analyzed the role of SUA as a prognostic factor in MI. Therefore, we want to evaluate the role of SUA as a prognostic marker in MI and its correlation with Killip classification.

2. Material and methods

It is a medical-based prospective observational study performed in tertiary care hospital, Bhubaneswar, Odisha, for one year. One hundred consecutive patients diagnosed with myocardial infarction meeting the inclusion and exclusion criteria admitted to the ICU were considered in the present study. Patient selection was based on the following criteria:

Inclusion criteria

1. Age > 18years.
2. Study presenting to the medical in between 24 hours of symptom onset.
3. Patients of myocardial infarction were diagnosed by

(i) Elevated cardiac troponins

(ii)Anyone of the following: Typical angina or angina equivalent, ST and T segment abnormality, new-onset LBBB, and pathological Q wave.

Exclusion criteria

1. CKD
2. Gout
3. Hypothyroidism
4. Chronic alcoholic
5. Drugs responsible for increasing serum uric acid: Salicylates (>2gm/ day), diuretics, pyrazinamide, ethambutol, and methyldopa.

Associate to Killip class, patients' history was recorded, and physical examination was done. The routine investigations were done for all patients like Hb, CBC, ECG, chest X-ray, renal function tests, and liver function tests. The associated patients were followed up daily with regular physical examination and reference to vitals, presence of S3, and any new murmur, crepitation in the lung, or evidence of pulmonary edema. Any complications and resuscitative measures or procedures on the patient were noted. Hypertensive and Diabetes mellitus patients were diagnosed per the AHA (American Heart Association) 2017 guidelines and the ADA (American Diabetes Association) diagnosis criteria, respectively.20,21 Patients were followed up in the hospital at least for seven days. SUA level was measured during admission on the third and seventh days. Uric acid was measured in our laboratory by the uricase method, using COBAS Integra 400 plus analyzer. Before initiation, our institute's ethical committee approved this study. We took informed consent from all patients who were registered in the study.

3. Results

Among the 100 patients with myocardial infarction, 61% were male, and 39% were female. The average age of cases was 56.34 ±4.499 years. Uric acid levels tested on the day of admission (D0) of the cases were 5.091 ± 1.374 mg/dL. Among the cases, the male had uric acid of 5.15 ± 1.32 mg/dL, and female cases had uric acid of 5.04 ± 1.41 mg/dL, which was statistically non-significant (p = 0.705). Among the cases, the male had uric acid of 5.15 ± 1.32 mg/dL, and female cases had uric acid of 5.04 ± 1.41 mg/dL, which was statistically non-significant (p = 0.705). Uric acid levels in patients with different ECG diagnoses were recorded at the entry of all the patients and were found to be 6 (Table 1). Among the 100 cases, 19% had prior MI, and 81% had recent MI. Among the cases of prior MI, 63% were male, and 37% were female. Among the cases of recent MI, 55% were male and 45% female. The correlation coefficient between serum creatinine and uric acid was 0.189, indicating poor correlation (Fig. 1). Serum creatinine was measured at admission and compared among patients in lower (Killip's I and II) and higher Killip's class (Killip's III and IV). Patients in the higher Killip's class group had a statistically higher Cr level (p = 0.035). The uric acid level of patients and the creatinine value at the time of admission were measured. Comparison of uric acid on day 0 and Killip class P-value 0.001 of UA between Lower (I and II) & higher Killip class. Uric acid level distribution in different Killip classes. At the time of admission of patients with a history of prior MI, 10% were in Killip's I, 10% were in Killip's II, 30% were in 30%, and 50% were in Killip's IV class. However, 54% of patients with recent MI were in Killip's I, 26% were in Killip's II, 15% were in Killip's III, and 5% were in Killip's class IV (Table 3). Ten patients died during 7 day follow-up period, and all are STEMI. Among all ten patients who expired8 patients had SUA level more than 7.0 mg/dL and two patients had <7.0 mg/dL (4.8 and 6.8 mg/dL). So 80% had uric acid >7.0 mg/dL and 90% had uric acid > 6.0 mg/dL at the time of admission. Out of the ten patients who expired, one was in Killip's class I, one in Killip's class III, and eight were in Killip's class IV during the entry. Thus, 90% of expired patients were in a higher class, i.e., class III and IV, during admission. The patient who had Killip's class III moved to Killip's class IV on day 3 and expired on day 5. The patient who had Killip's class I died that day2. At the time of death, he was in Killip's class I. At the time of death, 90% of the expired patients were in Killip's class IV and one in Killip's class I. Therefore, SUA concentration is remarkably connected with Killip's class (Table 4).

Table 1. Uric acid levels in patients with different ECG diagnoses.

Patient characteristics	Uric acid day 0	P-value
Male	26.53 ± 3.48	0.778
Female	26.74 ± 3.86	-----
Obese	5.12 ± 1.41	0.688
Nonobese	4.980 ± 1.222	-----
Diabetic	4.891± 1.418	0.06
Nondaibetis	5.415± 1.249	-----
Hypertensive	5.782 ± 1.269	< 0.001
Nonhypertensive	3.913 ± 0.398	-----
History of MI		
Yes (19)	6.93± 0.96	<0.05
No (81)	4.65 ± 1.06	-----

Table 2. Comparision of uric acid level between STEMI and NSTEMI patients.

Day	STEMI	NSTEMI	P-value
URIC ACID 0 Day	5.247 ± 1.372	3.827 ± 0.3744	<0.05
URIC ACID 3 Day	5.003 ±1.485	3.827 ± 0.3744	<0.05
URIC ACID 7 Day	4.293 ± 0.989	3.827 ± 0.3744	0.126

Table 3. Comparison of various parameters between those expired and survived cases.

Factors	Expired (N=10)	Recovered (N=90)	P-value
Number (M: F)	10(4:6)	90(57:33)	0.17
BMI	27.24 ± 2.11	26.54 ± 3.76	0.569
FBS	144 ± 32.86	144.72 ± 49.69	0.964
SBP	80.8 ± 22.13	132.22 ± 22.58	<0.001
DBP	49 ± 13.83	82.13 ± 12.95	<0.001
S.creat on 0 Day	1.31± 0 .25	1.14 ± 0.2	0.008
Uric acid 0 Day	7.11± 0.89	4.87 ± 1.23	<0.001

Table 4. Comparison of Killip's class between recent and past MI cases.

Killip's class	Prior MI	Recent MI
I	2	44
II	2	21
III	6	12

IV	9	4
Total	19	81

Table 5. Comparison of uric acid level among different Killip's class on 0, 3 and 7 days.

Killip's class 0 Day	U0 Level (mg/dL)				Total
	≤4.0	4.1-5.5	5.6-7.0	>7.0	
Killip's I 0 Day	27	17	1	1	46
3 Day	42	12	2	0	56
7 Day	49	28	3	1	81
Killip's II 0 Day	6	9	7	2	24
3 Day	3	8	3	1	15
7 Day	0	3	6	0	9
Killip's III 0 Day	2	3	9	3	17
3 Day	2	1	5	0	8
7 Day	0	0	0	0	0
Killip's IV 0 Day	0	0	2	11	13
3 Day	0	1	2	15	18
7 Day	0	0	0	0	0
Total	35	29	19	17	100

4. Discussion

Our study found comparable uric acid levels between males and females. A similar non-significant difference in uric acid levels was observed between male and female study populations by Bhattacharya et al.[22] and Nadker et al.[23] (Table 1). However, Kojima et al.[24] and Prasanth et al.[25] found a significantly higher uric acid level in males compared to females. A study with a larger population is required to clarify this difference. In our study, obesity is not associated with increased uric acid levels compared to non-obese patients (Table 1). A non-significant correlation (coefficient of correlation 0.019) was found between BMI and uric acid. Bonora E et al.[26] found higher uric acid levels in the obese patient. The lower uric acid level in obese patients in our study is probably due to fewer patients with obesity (17 with BMI 30-35 and only 2 with BMI>35 kg/m^2). Our study shows a statistically significant difference in uric acid levels between hypertensives and non-hypertensives (Table 1). These correlates are with Behera S et al.[27] and Sokhanvar et al.[28] studies. This finding did not correlate with studies like Shetty et al.,[29] S Agarwal et al.[30] who found no statistical significance in uric acid levels between hypertensives and non-hypertensives. In our study, 62% of patients had Diabetes. Statistically, a difference was not found between diabetic and non-diabetic patients (Table 1). Diabetic and non-diabetic patients had comparable SUA levels on Day 0. This result is observed by Das et al.,[31] which shows no relation between SUA level and Diabetes. However, this study is in opposition to other studies by Behera S et al. 32that explained that hyperuricemia is related to T2DM. There was a significant difference between serum uric acid concentration of patients with recent MI and prior MI at the time of admission (Table 1). Serum uric acid levels were significantly higher in patients with a history of IHD. Patients who were known cases of IHD were in higher Killip's class (Similar findings were observed in the study by Kojima et al.[24] and Nadker et al.[23] Among all cases, 11% of patients had NSTEMI, and 89% had STEMI (Table 2). Patients of STEMI had significantly higher uric acid on day 0 ($p<0.01$) and day 3 ($p=0.01$). We observed higher uric acid levels in STEMI due to a higher ischemic burden. A higher but statistically non-significant uric acid level on day 0 was observed in STEMI compared to NSTEMI by Bhattacharya et al.[22] In our study, we had 81% patients with recent MI, and 19% had previous MI.patients with prior history of MI who had significantly high levels of serum uric acid. They were in a higher killips class than those with recent MI (Table 4). Previous studies have shown that serum uric acid level increases in cardiac failure.[31] In our study, serum uric acid levels correlate with the severity of cardiac failure on day 0, day 3, and day 7. Patients with a higher quartile of uric acid were in the higher Killip's class, and those in the lower quartile were in, the lower Killip class(Table 5). This finding is consistent with Behera et al.,[32] Bhattacharya et al.[22] and Das et al.[31] Ten patients died during 7 day follow-up period, and all are STEMI. Among all ten patients who died, 8 patients had serum uric acid levels of more than 7.0 mg/dL, and two patients had <7.0 mg/dL (4.8 and 6.8 mg/dL) (Table 3). So 80%of patient who died, had uric acid >7.0 mg/dL and 90% had uric acid > 6.0 mg/dL at the time of admission. Abhisekhet al.[31] observed a similar finding of the ten patients who expired; one was in Killip's class I, one in Killip's class III, and eight were in Killip's class IV at the time of admission. Thus, 90% of patients who died were in a higher class, i.e., class III and IV, at the time of admission. The patient who had Killip's class III shifted to Killip's class IV on day 3 and expired on day 5. The patient who had Killip's class I died on day 2. At the time of death, he was in Killip's class I. At the time of death, 90% of the expired patients were in Killip's class IV and one in Killip's class I. Therefore, serum uric acid concentration is significantly correlated with Killip's class. Similar findings were observed by Prasanthet al25and Das et al.[31] In our study, the patients who expired had statistically significantly higher uric acid and creatinine and lower blood pressure on admission than those who

survived. This finding is inconsistent with a study by Behera et al.[32] Bhattacharya et al.,[22] and Das et al.[31]

5. Conclusion

Our study concluded that hypertension and prior history of MI are associated with higher uric acid levels. Patients with ST-elevation MI had significantly higher day 0 and day 3 uric acid levels compared to non-ST elevation MI. We found that SUA level carries a significant predictive value on admission regarding the prognosis in patients with MI, as patients with a high uric acid level at presentation had a higher Killip class. Patients with high uric acid levels are at risk of progressing to higher Killip class even if they are in low Killip class at presentation. SUA levels and Killip's class are affected remarkably by past MI and are higher for patients with prior MI. Patients with hypotension at admission had higher mortality after myocardial infarction. A combination of Killip's class and SUA level after acute MI is a better prognosticator of mortality after AMI. Combining hypotension at the time of entrance to SUA level and Killip's class after MI will still be a better prognosticator of mortality after AMI.

Conflict of Interest

The authors declared that there is no conflict of interest.

Acknowledgements

We are thankful to all mothers who participated in this research study and give their valuable time to response all required information.

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How to Cite this Article: Jena L, Singh S, Nayak SR, Behera B, Panigrahi R, Panigrahi SK. Evaluation of the Serum Uric Acid Level as an Early Predictor of Mortality Among the Patients with Myocardial Infarction: A Prospective Study. International Journal of Scientific Research in Dental and Medical Sciences, 2022;4(2):81-86. http://doi.org/10.30485/IJSRDMS.2022.340472.1293.