Study of Serum Uric Acid Levels in Acute Myocardial InfarctionPatients

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ABSTRACT: The present study has been undertaken with the following High uric acid level is a negative prognostic factor in patients with myocardial infarction. In myocardial infarction higher uric acid level increases the risk of mortality rate. There is uncertainty about the role of uric acid in acute coronary syndrome and whether it could be used as a prognostic marker in MI patients. A detailed clinical examination was performed on all patients. 100 patientswith acute myocardial infarction who fulfilled the inclusion/exclusion criteria were enrolled in the study. Thirty-three percent of patients were known diabetics in our study. Non-diabetic and diabetic patients had comparable serum uric acid levels on Day 0 This finding is consistent with a study in which there was no significant association between serum uric acid level and diabetic status. However, this finding contrasts with another study which showed that hyperuricemia is significantly associated with type 2 diabetes mellitus. Twenty-one percent of patients had a history of ischemic heart disease. There was a significant difference between serum uric acid concentration at the time of admission and h/o ischemic heart disease.

Key words: Myocardial infarction, Acute coronary, Uric acid, Troponin.

INTRODUCTION: Clinical and epidemiological studies have proved that serum uric acid (SUA) is significantly correlated with cardiovascular disease. Increased SUA is significantly associated with the occurrence and mortality of coronary artery disease [1]. But few studies have investigated serum uric acid levels in patients with acute myocardial infarction. This study was undertaken to assess the clinical value of serum uric acid levels in patients with MI by confirming the diagnosis by their clinical characteristics, ECG, and biomarkers (Troponin-T, CPK, CPK-MB). 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 an adverse event due to the association between uric acid and other cardiovascular risk factors [- 4SJ [2,3]. Several theories have been discussed, such as high serum uric acid havingan impact on increasing platelet reactivity. There is uncertainty about the role of uric acid in acute coronary syndrome and whether it could be used as a prognostic marker in MI patients. Furthermore, there is a need to find a simple and accurate prognostic marker that could be used in a remote area where fibrinolytic therapy is the first choice of acute reperfusion therapy (as part of pharmaco-invasive strategy) in nonPCI capable hospitals especially in developing countries [4-6].

Following myocardial infarction (MI) some proteins and enzymes labeled as cardiac markers (CPK - MB/ Troponin T) are released into the blood in large quantity from the necrotic heart muscle. These markers viz. CPK MB, Troponin-T, Troponin-I, and myoglobin, have a specific temporal profile in relation to MI; however, they do not correlate with myocardial function. Epidemiological studies have recently shown that uric acid may be a risk factor for cardiovascular diseases and a negative prognostic marker for mortality in subjects with pre-existing heart failure. Elevated serum uric acid is highly predictive of mortality in patients with heart failure or coronary artery disease and of cardiovascular events in patients [7,8].

MI usually occurs when coronary blood flow decreases abruptly after a thrombotic occlusion of a coronary artery previously affected by atherosclerosis. Slowly developing, high-grade coronary artery stenosis does not typically precipitate MI because of the development of a rich collateral network over time. Instead, MI occurs when a coronary artery thrombus develops rapidly at a site of vascular injury. MI occurs when the surface of an atherosclerotic plaque becomes disrupted (exposing its contents to the blood) and conditions (local or systemic) favor thrombogenesis. A mural thrombus forms at the site of plaque disruption, and the involved coronary artery becomes occluded. Histologic studies indicate that the coronary plaques prone to disruption are those with a rich lipid core and a thin fibrous [9-11]. The coagulation cascade is activated on exposure of tissue factors in damaged endothelial cells at the site of the disrupted plaque. Factors VII and X are activated, ultimately leading to the conversion of prothrombin to thrombin, which then converts fibrinogen to fibrin. Fluid phase and clot-bound thrombin participate in an auto-amplification reaction leading to further activation of the coagulation cascade. The culprit coronary artery eventually becomes occluded by a thrombus containing platelet aggregates and fibrin strands [12].

MATERIALS AND METHODS: We studied patients more than 30 years of age who were diagnosed with ST segment elevation acute myocardial infarction (STEMI) or non-ST segment elevation acute myocardial infarction (NSTEMI) based on clinical history, examination, ECG changes, biochemical markers, and admitted in Pacific Institute of Medical Sciences, Udaipur, during February 2022 – November 2022.

Inclusion criteria:
Patients brought to the hospital with a history of chest pain and diagnosed as myocardial infraction (bothSTEMI and NSTEMI) Diagnosis was confirmed by ECG.Biochemical markers like Troponin-T, Creatine Kinase (CK-MB) test CPK.

Exclusion criteria:
Any patient with a condition known to elevate uric acid levels
e.g.

- Chronic Kidney Disease.
- Gout.
- Haematological malignancy.
- Hypothyroidism were excluded.
  Also patients on drugs which increase serum uric acid
  e.g.
- Salicylates (>2 gm/d).
- Diuretics.
- Ethambutol.
- Pyrazinamide and also chronic alcoholics were excluded.

Written consent was obtained from both patients and control. Detailed history regarding symptoms and duration of the chest pain kidney disease, hypertension, diabetes, smoking, alcoholism, drug intake and treatment were elicited. A detailed clinical examination was performed in all patients. 100 patients of acute myocardial infarction who fulfilled inclusion/ exclusion criteria were emolledfor the study. A detailed history and physical examination with special reference to Killip class was carried out. All patients underwent routine investigations including Hb, CBC, renal function tests, liver function tests, ECG, chest X-ray. Patients were treated as decided by attending physician. Patients were followed up till hospital stay i.e., 7 days. Serum uric acid level was measured on day 0, 3 & 7 of ML 50 age and sex matched healthy controls were also be evaluated for baseline serum uric acid level. The study was approved by the Ethics Committee of the hospital. A detailed statistical analysis was carried out. Basal serum uric acid levels were compared with controls with unpaired 't' test. The levels of serum uric acid on day 0, 3, 7 were compared by paired 't' test. Uric acid levels and Killip class was compared with coefficient of correlation.

RESULTS:
We studied 100 patients with acute STEMI AND 50 age and sex-matched healthy controls. The comparison of the two groups and the profile of patients and their comparative uric acid levels are given in Tables 1 and 2. There was a statistically significant higher level of serum uric acid concentration in patients of MI on day of admission as compared to controls ($P < 0.05$). There was no significant difference in serum uric acid levels as regards sex, hypertension and diabetes mellitus in patients with MI; however, those with history of MI in the past had higher serum uric acid levels. Also, patients with history of IHD were in the higher Killip class (Table 3).

Serum uric acid levels were comparable on Days 0, 3 and 7 in MI group, 5.23±1.95, 5.20±2.15 and 5.28±2.52 respectively (P = NS).

Tables 4, 5, and 6 show the levels of uric acid in relation to Killip class on Days 0, 3 and 7 of admission. On all the days serum uric acid levels were higher in patients who were in higher Killip class ($P < 0.05$). All the five patients who died after 3 days of hospital stay had serum uric acid levels more than 7.0 gm/dL and all of them were Killip class IV.

DISCUSSION:
Previous studies have shown that serum uric acid increases in cardiac failure. In a study done it was shown that serum uric acid levels correlate with Killip classification. A combination of Killip class and serum uric acid level after acute myocardial infarction is a good predictor of mortality in patients who have acute myocardial infarction [13]. Using this study as a referral study, we tried to find a correlation between serum uric acid and Killip class and their prognostic value in our patients. The present study was conducted on 100 patients with acute myocardial infarction, who presented to the hospital within 24 hrs of the onset of symptoms. Fifty age and sex-matching healthy controls were also evaluated for comparison of uric acid levels. Out of 100 patients, 65 had ST-elevation myocardial infarction (STEMI), while 35 patients were of non-ST elevation myocardial infarction (NSTEMI). Sixty-one patients were thrombolysed while four were not thrombolysed due to delayed presentation. Uric acid was treated as a continuous variable and as a categorical variable, and variables were divided into quartiles according to serum uric acid concentrations same as in the referral study by Kojima et al.43 Our patients and controls were age and sex-matched [14,15]. The patients had higher serum uric acid levels probably because of acute myocardial infarction. A similar finding was seen in a referral study44 with 1124 patients who presented with acute myocardial infarction within 48 hrs. of onset of symptoms. In our study there was no difference in uric acid levels between male and female patients however in the referral study males had higher uric acid levels as compared to females43 correlation (p=0.241) between serum uric acid level and patients who were known or found to be hypertensive on admission [16,17]. This is different from other studies which showed that hypertensive patients had more hyperuricemia [18]. Thirty-three percent of patients were known diabetics in our study. Non-diabetic and diabetic patients had comparable serum uric acid levels on Day-0 This finding is consistent with study in which there was no significant association between serum uric acid level and diabetic status. However, this finding contrasts with another study which showed

that hyperuricemia is significantly associated with type 2 diabetes mellitus. Twenty-one percent of patients had a history of ischemic heart disease [19]. There was a significant difference between serum uric acid concentration at the time of admission and h/o ischemic heart disease. Serum uric acid levels were higher in patients with a history of IHD as seen in a previous study.

CONCLUSION: Uric Acid levels were high in patients with Acute Myocardial Infarction. Patients who were in higher Killip classification had higher uric acid levels Patients who died had higher uric acid levels. Patients who survived had lower uric acid levels. No significant difference in uric acid levels in diabetes mellitus patients and non-diabetic Mellitus patients. No significant difference in uric acid level in patients with systemic hypertension and patients who do not have systemic hypertension.

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ETHICAL APPROVAL:
The study was approved by the Institutional Ethics Committee.

CONFLICT OF INTEREST:
The authors declare no conflict of interest.

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