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Evaluate the role of serum uric acid in acute myocardial infarction as a prognostic marker

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Abstract
The uric acid role as a prognostic factor in patients with acute ST elevation of myocardial infarction is contentious. The aim of this research was to show the association between the amount of serum uric acid and mortality during the induction time. Patients older than 18 years of age have been identified as ST segment elevation acute myocardial infarction (STEMI) or non-ST segment elevation with reference to clinical records, reviews, ECG shifts, biochemical indicators, and Krishna Hospital, Karad. Patients of high Killip class showed higher serum uric acid levels than to patients with lower Killip class. Serum uric acid level when paired with Killip class is a strong indicator of heart failure incidence and short-term mortality after STEMI.

Keywords: uric acid, myocardial infarction, serum, acute myocardial, ST elevation

Introduction
The cardiac proteins (CPK, MB / T&I troponin) are released from the necrotic heart muscle in significant quantities into the blood. *Viz*. The signs. CPK-MB, troponin-T, troponin-I, and myoglobin have different MI-related temporal profiles that may not interact with myocardial activity. In recently, epidemiological studies have shown that uric acid may be a risk factor for heart disease and a negative prophylactic marker for mortality in pre-existing subjects with heart failure. High mortality from serum uric acid is predictive in patients with heart failure or coronary artery disease and in patients with heart events [1]. There is evidence that elevated uric acid in patients with moderate to serious heart disease is a harmful prognostic factor [2]. While the occurrence of hyperuricaemia in these patients almost always includes a worsening of renal failure [3].

Hence, dissecting the functions performed by decreased renal function and elevated uric acid in influencing these patients' prognoses is challenging. Many research indicates uric acid may have a detrimental effect on cardiovascular disease by inducing inflammation, which is specifically implicated in cardiovascular disease pathogenesis [4, 5].

Aim
To measure the levels of serum uric acid (SUA) calculated after admission as a possible indicator of short-term mortality (7 days) in patients with acute myocardial infarction (AMI).

Objectives
To determine if uric acid in AMI is substantially elevated when opposed to normal subjects. A poor prognostic predictor of AMI is for validating hyperuricemia.

Review of Literature
Uric acid is a heterocyclic nitrogen, carbon, oxygen, and hydrogen complex containing formula $C_5H_4N_4O_3$. Uric acid is produced as purine nucleotides break down in the body. Hyperuricemia (HU) is a biochemical abnormality. Not a disease, Uric acid concentration in excess of urate solubility (>6.8-7mg/dl) in men and >6.0mg/dl in women, estrogen is uricosuric, dramatic rise in the incidence of HU and gout is seen ingenetically susceptible host, western diet, ageing population, obesity. Does it have any beneficial role? Any evolutionary advantage? Biological reason for losing uricase activity is not understood fully, It helps to maintain BP during upright posture, when salt intake is low: It activates Renin-angiotensin system, it is mild neurostimulant. Intelligence, Anti-oxidant and neuroprotective. Decreased incidence of Parkinson's, MS and Alzheimer's- effect on astroglial cell, UA contributes to 50% anti-oxidant activity in blood, Contributes to longevity of life in hominids. Hyperuricemia is associated with Hypertension /prehypertension. Consider Pre-glomerular arteriopathy in young hypertensive with established HTN in whom ULT does not respondin Renal disease, Diabetes/IGT. Cardiovascular risk, Obesity, Hyperlipidemia/low HDL/high TGL Increased mortality (Gout and nothyperuricemia per set).

In HEROES study by G LAZAROS *et al* in European study to cardiology [6] They studied 522 patients admitted in hospital from January 2007 to 2009 with AMI. Hyperuricemia is a biochemical abnormality, not a disease. Indications for lowering uric acid is clear. Drug therapy to lower UA (ULT) for CVD prevention is not advised as per current evidence [7].

The vast majority of acute myocardial infarction are related to rupture or fissuring of atherosclerotic plaque in coronary arteries with superimposed thrombus. Thrombotic occlusion is present in about 90% of patients with transmural Q wave infarction, when coronary angiography is performed within 4-6 hrs of the onset of pain. In a minority of patients with Q wave infarct, coronary artery spasm may play a pathogenic role. Only two thirds of patients with non-Q wave myocardial infarction have occluding thrombi in the infarct related artery.

In these patients, a coronary thrombus may have undergone spontaneous lysis. Coronary artery spasm may have played a role or there may have been increased myocardial oxygen demand [8, 9].

In this study we studied the relation of UA in cases and controls in relation to age and sex. Study found that on day 0 uric acid levels in males- In age group of <40yrs, mean UA levels in cases was 6.36 mg% in Killip's class I, no cases were found in Killip's class II, III and IV in this age group whereas in control group mean UA level was 5.40mg%.Similarly in age group of 40-50 mean UA levels in cases was 6.25mg%, 6.30mg%, 8.6mg%, 9.8mg% in Killip's class I, II, III, IV respectively whereas in controls it was 5.56mg%. Similarly in age group of 51- 60, mean UA levels in cases was 6.04mg%, 7.35mg%, 10.1mg% in Killip's class I, II, IV respectively, no cases fell in Killip's class III, whereas in control group mean UA levels was 6.04mg%.In the age group of 61-70, in cases mean UA level was 5.78mg%, 7.2mg%, 8.37mg%, 9.9mg% in Killip's class I, II, III, IV respectively where as in control group mean UA level was 6.52mg%.In age group of 71-80, in cases mean UA levels was 7.33mg%,7.0mg%,8.35mg%, 8.85mg% in Killip's class I, II, III, IV respectively and in control group mean UA levels was 5.98mg%. In age group of >80 mean UA in cases group was 7.2mg%, 8.7mg%, 8.7mg% in Killip's class I, III, IV respectively, no cases fell in Killip's class II and in control group mean UA levels was 7.3mg%. Study found that on day 0 uric acid levels in females- In age group of <40, in cases mean UA was 5.7mg% in Killip's class I, no cases fell in Killip's class II, III, IV and in control group mean UA was 5.04mg%. In age group of 40-50, mean UA levels in cases was 5.85mg% in Killip's class I, no cases fell in Killip's class II, III, IV and in control group was 5.57mg%. In age group of 51-60, in cases mean UA levels was 5.34mg%, 7.85mg%, 8.5mg%, 8.5mg% in Killip's class I, II, III and IV respectively and in control mean UA was 6.29 mg%. In age group of 61-70, in cases mean UA levels was 6.5mg%, 7.1mg%, 9.76mg% in Killip's class I, II, and IV respectively no cases fell in Killip's class III and in controls mean UA level was 5.30 mg%. In age group of 71-80, in cases mean UA levels was 6.46mg%, 6.4mg%, 7.2mg%, 9.0mg% in Killip's class I, II, III, IV respectively and in controls mean UA levels 6.7mg%. In age group of > 80, mean UA in cases was 5.1mg% in Killip's class I, no cases fell in class II, III, IV and I controls mean UA levels was 5.84mg%. The study observed here that there was rising trend of UA levels as the Killip's class increased in AMI as compared to controls in males, females and all the age groups.

Materials and Methods

100 cases of acute myocardial infarction (STEMI AND NSTEMI) and 100 cases as controls. Patients above the age of 18 that have been classified on the basis of clinical reports as ST segment elevation acute myocardial infarction (STEMI) or non-ST segment elevation acute myocardial infarction (NSTEMI), examination, ECG changes, biochemical markers and connection to Krishna Hospital, Karad. Diagnosed cases of acute myocardial infarction both (STEMI and NSTEMI) of more than 18 years of age. Total no of 100 patients diagnosed to have acute myocardial infarction getting admitted in Krishna hospital were selected for this study along with 100 controls, the study has been accepted by the institute's ethics committee, consent of both the groups was taken for participation in study. Institute and the researcher beared the expenses of uric acid estimation in both the groups. UA levels in controls were evaluated by random sampling method. All the subjects in control group were having mixed dietary habit. The patients after meeting the inclusion criteria and exclusion were evaluated by taking detail clinical history and examination and serum uric acid levels on day 0, 3, 7 was measured.

Observation and Tables

Table 1: Age and sex wise distribution

Age in years	Cases group (n=100)		Control group (n=100)	
	Males	Females	Males	Females
	No. (%)	No. (%)	No. (%)	No. (%)
< 40	7	3	18	6
40-50	6	0	10	8
50-60	12	17	10	12
60-70	16	17	10	6
70-80	9	8	6	4
>80	4	1	8	2
Total	54(54%)	46(46%)	62(62%)	38(38%)
Mean ± SD	60.59±12.04		58.90±14.15	

Value of $\times^2 = 21.14$, d. f.=15, $p<0.05$, significant

As seen in table 1, there is a important correlation between age and sex by the use of Chi-square test (i.e.$p<0.05$) in case and control group.

Table 2: Type of MI in Cases group

Type of MI	No. (%)
Stemi	80(80%)
Nstemi	20(20%)
Total	100

As seen in table 2, out of 100 cases studied 80 cases were of STEMI and 20 were NSTEMI signifying majority of cases of AMI were of STEMI.

Table 3: Wall involved in MI in Cases group

Wall	No. (%)
Anterior	55(55%)
Posterior	12(12%)
Inferior	42(42%)
RV	11(11%)

As seen in table 3, of 100 cases 55% of anterior wall MI, followed by inferior wall 42% followed by posterior wall 12% and then right ventricular 11%.

Table 4: Relation between uric acid level and Killip's class and death

Killip's class	Uric acid day 0			Uric acid day 3			Uric acid day 7		
	<6.5	6.5-8.5	>8.5	<6.5	6.5-8.5	>8.5	<6.5	6.5-8.5	>8.5
	No. of deaths			No. of deaths			No. of deaths		
I	0	0	0	0	0	0	0	0	0
II	0	0	0	0	0	0	0	0	0
III	0	1	7	0	2	3	0	0	1
IV	0	3	12	0	0	5	0	0	2
Total	0	4	19	0	2	8	0	0	3

In this table studied relation between level of uric acid on day1, day 3 and 7 and no. Of deaths. Uric acid levels were grouped in 3 groups of <6.5, 6.5-8.5, >8.5.Total no. Of deaths were 23 of 100 cases studied No deaths were observed in patients in whom the uric acid was <6.5. 2 cases who had uric acid between 6.5-8.5 expired on day 1 and 2 cases expired on day 3 respectively, all the 4 cases were in Killip's class III and IV. In patients with S.UA of >8.5 19 deaths was observed. Of these 11 patients died before day 3, 5 patients died between day 3 and 7 and 3 patients died on day 7. All patients were in class III of Killip, of which 7 were in class III and 12 were in class IV.

Table 5: Uric Acid Day 0 in cases

Day 1	No. (%)
3.0-3.9	1(1%)
4.0-4.9	5(5%)
5.0-5.9	20(20%)
6.0-6.9	28(28%)
7.0-7.9	19(19%)
8.0-8.9	12(12%)
9.0 and above	15(15%)
Total	100

As seen in table 5, out of 100 cases studied 1% cases were in range of UA 3.0-3.9, 5% were in range of 4.0 to 4.9,20% were in range of 5.0-5.9,28% in range of 6.0-6.9,19%in range of 7.0-7.9,12% were in range of 8.0-8.9 and 15% were in range of 9.0and above. Hence this shows that majority of patients of acute myocardial infarction have uric acid in range of 6.0-6.9.

Discussion

In this study it was seen that mean age in case group and mean age in control group is did not differ significantly and hence the two groups were considered age matched. There were significant no. of patients in the age Group of 50-70 Years and hence we conclude that age itself is a independent risk factor for AMI. Muhammad Shabbir *et al* study concluded that majority of patients of acute myocardial infarction lied in mean age of 57± 14 years. our findings correlate with this study. In this study 55% patients had anterior wall MI, 42% patients had inferior wall MI,12% patients had posterior wall MI and 11% had right ventricular wall MI. Signifying incidence of MI was highest in the anterior myocardial wall. According to a study by Myung Hwan Bae, MD *et al* also the incidence of anterior wall was 52% in their study. Our findings correlate with this study. In this study 86% patients of STEMI were actively thrombolysed and the remaining 14% could not be thrombolysed and hence were managed with heparin alone. Overall, following the course of treatment, 73% patients of AMI were discharged and 23 % patients expired Association of Killip's class with mortality seen in cases of STEMI / NSTEMI. In this study, there is a significant association seen between Killip's class and mortality in STEMI and NSTEMI (i. e. $p<0.05$). Higher the Killip's class of AMI greater is the mortality.

Conclusion

Study and control group were matched in age and sex for UA estimation to rule out any difference between two groups. Serum uric acid was statistically slightly higher (7.03 mg percent) in AMI patients relative to control groups, and in patients with acute myocardial infarction we infer that serum uric acid is substantially elevated. For patients with higher level of Killip's class (III, IV) serum uric acid was increased with the lowest rate of anterior wall MI. High uric acid (8.5 mg percent) and higher Killip's class (III, IV) were correlated with higher mortality (23/23 deaths), thereby associating lower uric acid rates with higher Killip's class (III and IV) and higher mortality in AMI patients. And uric acid should be used for instances of acute myocardial infarction as a short term prognostic marker.

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