Section: Biochemistry

Original Research Article

COMPARATIVE STUDY OF SERUM URIC ACID LEVELS IN VARIOUS ACUTE CORONARY SYNDROMES AND ITS PROGNOSTIC SIGNIFICANT USING KILLIP CLASSIFICATION

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Abstract

Background: Apart from indicating in the renal profile, serum uric acid is also elevated in cardiovascular disease because it is accumulated in the lumen of blood vessels. Hence, serum uric acid (SUA) is a good predictor of cardiovascular diseases. **Materials and Methods:** 50 (fifty) patients fulfilling the acute coronary syndrome were compared with 50 (fifty) healthy controlled group. Cardiovascular parameters, including serum uric acid (SUA), in both groups, and significant results were noted. **Result:** The serum uric acid (SUA) level was elevated in acute coronary syndrome patients in this comparative study as per the killip class on baselines; the 3rd and 7th day p value ($p<0.001$) was highly significant. **Conclusion:** It is concluded that elevation of SUA is a strong and independent predictor for CVD and mortality.

INTRODUCTION

Uric acid is the end product of purine metabolism in the human body. Its production is facilitated by the enzyme xanthine oxidase (XO). XO utilizes oxygen and purine to produce O2− and H2O2 as well as uric acid through oxidative hydroxylation. In the endothelium XO can produce large amounts of reactive oxygen species (ROS) under pathophysiologic conditions, such as atherosclerosis and ischemia-reperfusion injury.[1] Elevated serum uric acid (SUA) levels are linked to unfavorable outcomes in cardio-vascular diseases.[2] Uric acid rapidly increases within the tissues, which is subsequently discharged into the vascular lumen. Upon arrival, a reduction in intracellular PH and a reversal of negative membrane potential occurs.[3] It is reported that there is an elevation in the synthesis of UA and an increase in the activity of xanthine oxidase during instances of myocardial ischemia. Hyperuremia frequently co-occurs with metabolic syndrome, comprises of hypertension, diabetes, dyslipidemia, and obesity. All of these are established risk factors for coronary heart disease. It is also proven that elevated uric acid levels pose a significant risk factor for the development of cardiovascular disease related to mortality.[4] Hence, an attempt is made to evaluate the levels of uric acid in acute coronary syndrome patients and to study the prognosis of these patients.

MATERIALS AND METHODS

50 (fifty) adult patients who regularly visited Government Medical College Hospital Bhadradri Kothagudem, Telangana-507101 were studied.

Inclusive Criteria

50 adult patients fulfilling the standard diagnostic criteria for acute coronary syndrome [ST segment elevated MI(STEMI), non-ST segment elevated MI (NSTEMI), unstable angina(UA)] on the basis of clinical history, signs, ECG changes, and biomarkers (Troponin T and I) (CK-MB) were included, and patients who gave consent in writing were selected for the study.

Exclusion Criteria

Patients with kidney disease, gout, hematological malignancy, hypothyroidism, and chronic alcoholic patients on treatment with salicylates, diuretics, ethambutol, and pyrazinamide were excluded from the study.

Method: 50 (fifty) patients fulfilling the standard diagnostic criteria for acute coronary syndrome were compared with a 50 (fifty) adult controlled group. After informed consent and ethical clearance the patients underwent routine investigations, including Hb%, CBC, renal profile, liver function test, lipid profile, ECG, and chest x-rays. They were followed up for a minimum of 7 days in the hospital. Serum uric acid level was measured on days 0, (first), 3rd and 7th acute coronary syndrome. Significant comparative results were noted.

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Statistical Analysis: Various parameters of acute syndrome in both groups were compared, and significant results were noted. The statistical analysis was carried out in SPSS software. The ratio of males and females was 2:1.

RESULTS

[Table 1] Comparison of acute coronary syndrome patients versus a controlled (healthy) group

- Age (years): 56.20 (±12.6) in ACS patients and 54.12 (± 9.6) in the controlled group; t test was 0.9 and p>0.35 (p value is insignificant).
- Sex proportion: 34 male and 16 female in ACS patients and 33 male and 17 female in the controlled group (34:16 vs. 33:17).
- Serum uric acid (mg/dl): on the first day (baseline study), 6.42 (± 2.05) in ACS patients and 4.20 (± 0.50) in the controlled group; the t test was 7.43 and p<0.001 (p value is highly significant).

[Table 2] Comparative study of serum uric acid levels at baseline on day 0, 3rd day, 7th day, and KILLIP CLASS

- Level of Uric on base line/zero day: 4.55 (± 1.22) in 20 patients, 7.8 (± 0.85) in 15 patients, 8.37 (± 1.36) in 8 patients, 9.25 (± 2.0) in 7 patients, F value 36.2, and p<0.001.
- Uric acid level: On the 3rd day, 4.53 (± 1.05) in 20 patients, 6.73 (± 1.06) in 15 patients, 7.60 (± 0.66) in 8 patients, and 9.05 (± 1.62) in 7 patients had an F value of 36.4 and p<0.001.
- Uric acid level on 7th day (mg/dl) was 4.20 (± 1.0) in 20 patients, 6.30 (± 1.53) in 15 patients, 7.20 (± 0.65) in 8 patients, and 8.30 (± 1.55) in 7 patients. The F value was 24.9 and p<0.001.

[Table 3] Comparison of serum uric acid levels at baseline on the 3rd and 7th days and various acute coronary syndromes

- Level of uric acid (mg/dl) on the base line (zero day): 7.24 (± 2.10) in STEMI and 6.74 (± 1.67) in NSTEMI; 3.78 (± 1.10) UA F value: 62.3 and p<0.001.
- 3rd day: 6.72 (± 1.88) in STEMI group, 6.50 (± 1.50) in NSTEMI, 4.02 (± 1.10) in UA, F value 48.2 and p<0.001.
- 7th day: 6.15 (± 1.86) in the STEMI group, 6.13 (± 1.65) in the NSTEMI group, and 3.80 (± 1.12) in the UA. F value was 36.8 and p<0.001.

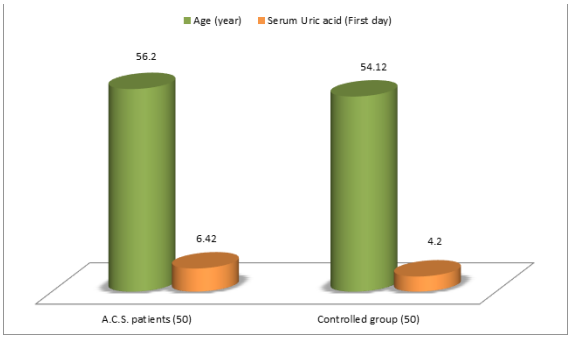

Figure 1: Comparison of acute coronary syndrome patients and healthy (controlled)

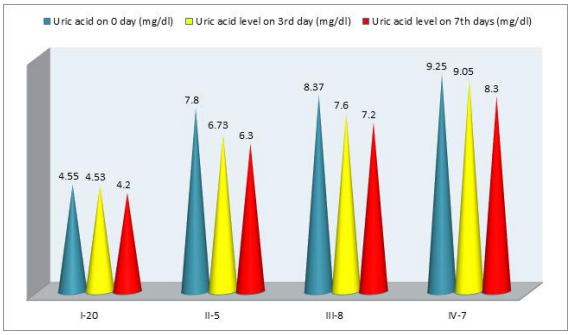

Figure 2: Comparative study of serum acid levels at baseline day 0 at 3rd day, 7th day and KILLIP CLASS

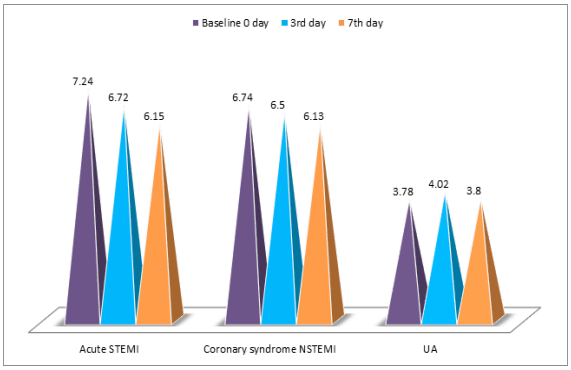

Figure 3: Comparative study of serum uric acid level at baseline, 3rd day and 7th day and various acute coronary syndromes

Table 1: Comparison of acute coronary syndrome patients and healthy (controlled)

Details	A.C.S. patients (50)	Controlled group (50)	t test	p value
Age (year)	56.20 (± 12.6)	54.12 (± 9.6)	0.9	p>0.35
Sex M/F	34:16	33:17	--	Not significant
Serum Uric acid (First day)	6.42 (±2.05)	4.20 (±0.50)	7.43	P<0.001

Table 2: Comparative study of serum acid levels at baseline day 0 at 3rd day, 7th day and KILLIP CLASS

No. of patients	KILLIP CLASS					
	I 20	II 15	III 8	IV 7	F value	P value
Uric acid on 0 day (mg/dl)	4.55 (± 1.22)	7.8 (± 0.85)	8.37 (± 1.36)	9.25 (± 2.0)	36.2	P<0.001
Uric acid level on 3rd day (mg/dl)	4.53 (± 1.05)	6.73 (±1.06)	7.60 (± 0.06)	9.05 (± 1.62)	36.4	P<0.001
Uric acid level on 7th days (mg/dl)	4.20 (± 1.0)	6.30 (± 1.55)	7.20 (± 0.65)	8.30 (± 1.55)	24.9	P<0.001

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Table 3: Comparative study of serum uric acid level at baseline, 3rd day and 7th day and various acute coronary syndromes

Uric acid level at (mg/dl)	Acute STEMI	Coronary syndrome NSTEMI	UA	f	p value
Baseline 0 day	7.24 (± 2.10)	6.74 (±1.67)	3.78 (±1.10)	62.3	P<0.001
3rd day	6.72 (±1.88)	6.50 (±1.50)	4.02 (±1.10)	48.2	P<0.001
7th day	6.15 (±1.86)	6.13 (±1.65)	3.80 (±1.12)	36.8	P<0.001

STEMI = ST segment Elevation Myocardial Infarction
NSTEMI = Non ST elevation Myocardial Infarction

DISCUSSION

In the present comparative study of serum uric acid (SUA) in acute coronary syndrome and its prognostic significance using the KILLIP classification. The SUA level was 6.42 (± 2.05) in acute coronary syndrome (ACS) patients and 4.20 (± 0.50) in healthy; the t test was 7.43 and p<0.001 [Table 1]. SUA levels at baseline on day 0 (zero) at 3rd day, 7th day, as per the Killip class, patients were divided into 20, 15, 8, and 7; the SUA level was elevated in the 15th, 8th and 7th groups on the baseline study. Similar elevations of SUA were also observed on 3rd and 7th days [Table 2]. In the comparative study of SUA at base line 3rd day and 7th day and various acute ACS base line study on STEMI was 7.24 (± 2.10), 6.72 (± 1.88) on 3rd day and 6.15 (± 1.86) and in NSTEMI group, 6.74 (± 1.67) in base line study, 6.50 (± 1.50) on 3rd day, 6.13 (± 1.68) on 7th day and p value was highly significant [Table 3]. These findings are more or less in agreement with previous studies.[5-7]

There was no significant relationship between age and sex in the present study. It indicates that SUA elevation may be due to sedentary life or diabetes. The patients had a higher SUA level, probably because of an acute myocardial infarction.[8] It is reported that SUA may be useful for prognostic marker among those with pre-existing heart failure.[9] SUA is produced by the enzymatic activity of XO and is the main end product of the metabolism of purines, which in turn are derived mostly from diet, de novo biosynthesis, and the breakdown of nucleic acids. SUA levels reflect degree XO activation. During SUA production, oxygen-free radicals are generated, and therefore SUA is a simple and useful clinical indicator of excess oxidative stress.[10] In humans, one of the tissues with the highest activity of XO is the capillary endothelium, and the endothelium of small arteries is an important source of oxygen-free radical production within the endothelium.[11] SUA is also one of the strongest determinants of plasma oxidative capacity, with free radical scavenging activity in human serum. Hence, in atherosclerosis, elevated SUA is found which leads to myocardial infarction due to the higher activity of XO in capillaries, which obstructs the blood flow.

However, hyper-SUA can also directly cause vascular injury at high concentrations. SUA promotes vascular smooth muscle proliferation, platelet aggregation, cell apoptosis, and local inflammation.[12] It is also associated with tubule interstitial inflammation, morphological and functional changes in the glomeruli and renal arterioles, and increased salt-sensitivity hyper-uricaemic or salt-sensitive kidney-dependent hypertension. All these mechanisms suggest SUA is potentially associated with cardiovascular disease.

CONCLUSION

The present comparative study of SUA levels in various acute coronary syndromes and their prognostic significance using the KILLIP classification. It showed that there is a direct relationship between SUA levels and myocardial infarction. Higher levels of SUA in association with the KILLIP class are a good predictor of severity and short-term mortality after myocardial infarction. Hence, patients with elevated SUA must be given the utmost care to avoid myocardial infarction and mortality as well.

The present study demands a genetic, nutrition, patho-physiological, and pharmacological study because the mechanism of SUA towards heart disease is still unclear.

Limitation of study: Owing to the tertiary location of the research center, the small number of patients, and the lack of the latest techniques, we have limited findings and results.

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