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Original Research Article.

Study of Uric Acid in Patient with Acute Myocardial Infarction

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ABSTRACT

Introduction: Hyperuricemia is usually defined as increase in serum uric acid level which should be at least above 6mg/dl in women and at least above 7mg/dl in men. It has been revealed that uric acid induces endothelial dysfunction by activating the HMGB1/RAGE signaling Pathway.

Methodology: In this case- control study 100 total number of population were included. This study was conducted in the Department Of Medicine, C. U. Shah Medical College, Gujarat, India. The duration of study was over a period of six months.

Results: In the present study, 26 cases & 25 control had systemic hypertension, 34 cases & 32 control had diabetic mellitus & serum uric acid level 7.272 in cases & 5.916 in control were seen.

Conclusion: This study concludes that, SUA levels were higher in patients with acute MI in comparison to normal healthy individuals. In patients with acute MI, SUA levels are elevated in systemic hypertension and diabetes mellitus. In acute MI patients, SUA can be used as a marker of short-term mortality. Hyperuricemia is found an indicator of poor prognosis in acute MI.

Keywords: Uric Acid, Myocardial Infarction, Hyperuricemia.

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INTRODUCTION

It is a known fact that uric acid is a metabolite of purines.[1] Hyperuricemia is usually defined as serum uric acid. It found at least 6 mg/dL in women and at least 7 mg/dL in men.[2-4] It has been revealed that uric acid induces endothelial dysfunction by activating the HMGB1/RAGE signaling Pathway.[5] Several researchers found that uric acid is an independent risk factor for coronary heart disease.[6] It is also used as a biomarker for inflammation.[1] Recently, it has been published in researches that in patients with progressive heart failure, the presence of elevated uric acid is a major prognostic marker predicting mortality and the requirement of heart transplantation.[7,8] The assessment of this biomarker with other parameters of cardiac function is suggested to be hopeful for managing the patients in future.[9] Some pathophysiological processes have been identified the association between the production and metabolism of uric acid and progression and outcome of heart failure.[10] It comprises the activity of xanthine oxidase pathway resulting deterioration of left ventricular function via changing calcium sensitivity of myofilaments as well as by interfering with myocardial energetic pathway.[11] Irrespective of the meditative role of xanthine oxidase pathway, uric acid itself play an important role in cardiovascular and renal dysfunction through stimulating vascular smooth muscle cell proliferation which leads to renal vascular disease, renal disease, systemic hypertension, and hemodynamic instability.[12,13]

The role of hyperuricemia on endothelial function in the human vascular bed has also been shown.[14] Hence, the increase in serum level of uric acid can be a valuable indicator for progression of heart failure which leads to inferior patients' outcome and reducing their long-term survival. However, due to myocardial infarction, the effect of hyperuricemia in acute heart failure has not been well understood.

This study aimed to compare the serum level of uric acid in patients with and without heart failure and also to determine the association between hyperuricemia and the Killip class[15], after myocardial infarction in patients with heart failure.

MATERIALS & METHODS

Study Population: In this case- control study 100 total number of population were included.

Study Area: This study was conducted in the Department of Medicine, C. U. Shah Medical College, Gujarat, India

Study Duration: The duration of study was over a period of six months.

Data Collection: Patients >18 years of age with STEMI or non-ST segment elevation MI (NSTEMI) on the basis of history, clinical examination, electrocardiographical changes and biochemical markers were included. Patients with a condition known to elevate SUA level (eg, chronic kidney disease, gout, hematological

malignancy, hypothyroidism, hyperparathyroidism) were excluded. Patients taking drugs that increase SUA (eg, salicylates [>2 g/day], ethambutol, amiloride, bumetanide, chlorthalidone, cisplatin, cyclophosphamide, cyclosporine, ethacrynic acid, thiazide diuretics, furosemide, indapamide, isotretinoin, ketoconazole, levodopa, metolazone, pentamidine, phencyclidine, pyrazinamide, theophylline, vincristine or vitamin C were also excluded, as were chronic alcoholics.

Fifty patients with acute MI (STEMI and NSTEMI) who fulfilled inclusion/exclusion criteria were studied. Fifty age- and sex-matched controls were also evaluated for their baseline SUA level.

Data Analysis: Data were analyzed by using Microsoft excel.

Table 1: Distribution of Cases According to Gender

Gender	Case	Control
Male	38	35
Female	12	15
Total	50	50

Table 2: Distribution of Cases & Control According To Age

Age	Case	Control
31-40	9	10
41-50	11	7
51-60	28	32
>60	2	1
Total	50	50

Table 3: Distribution of Cases According To Parameters

Parameters	Case	Control
Systemic Hypertension	26	25
Diabetic Mellitus	34	32
Serum Uric Acid (mg/dl)	7.272	5.916

Table 4: This Table Showing Uric Acid Level Day 1,3,5

Uric Acid Level	KLLIP Class	Mean	P Value
Day 1	I	5.51	<0.0001
	II	8.05	
	III	11.18	
	IV	14.16	
Day 3	I	5.27	<0.0001
	II	6.75	
	III	7.76	
	IV	8.05	
Day 5	I	4.97	0.001
	II	5.17	
	III	5.65	
	IV	5.96	

RESULTS

In our study 100 total populations were included. Among all these 50 were control & 50 were cases. Out of 50 cases 38 were male & 12 were female. Out of 50 controls 35 were male & 15 were female. This study found that most of the population were belongs to 51 -60 age group in case as well as in control group followed by other age group. In the present study, 26 cases & 25 controls had systemic hypertension, 34 cases & 32 control had diabetic mellitus & serum uric acid level 7.272 in cases & 5.916 in control were seen. Mean & p value of uric acid level according to KLLIP class were showing in table 4.

DISCUSSION

The present study had taken a total of 50 patients with acute MI. out of 50 patients, 38 were male and 12 were female. 50 age- and sex-matched controls were also assessed for their baseline SUA level. The findings of the present study showed that there was no significant difference with regard to age, sex, status of systemic hypertension and diabetes mellitus in patients with acute MI and healthy controls. In comparison to healthy controls, acute MI patients had statistically significant higher (P<0.0001) SUA level on the day of admission. It was also found that there was a significant relationship between SUA level and mortality. All patients who died had SUA level >7.0 mg/ dL (P=0.041 [Fisher's exact test]).

In a study by Siniša Car et al[16] observed that higher SUA level at the time of admission was related with higher in-hospital and 30-day mortality, and poorer long-term survival after acute MI. Therefore, elevated SUA level may be associated with coranary artery disease. Nadkar and Jain[17] found that SUA levels were higher in patients with acute MI and were correlated with Killip class. In this study, the majority (60%) of cases belonged to Killip class I, 22% to Killip class II, 12% to Killip class III and 6% to Killip class IV. Results showed a significant relationship (P=0.042 on day1, P=0.014 on day 3 and P=0.001 on day 5) between SUA level and patients who were found to be hypertensive on admission. The results also showed that hypertensive patients had more hyperuricemia. Kojima et al[18] found that SUA concentration was significantly correlated with hypertension (r=0.301; P=0.005). A significant relation between SUA level and Killip class was found on day of admission (Table 2). In the present study, patients in Killip class III and IV had higher levels of SUA in comparison to patients in Killip class I and II (P=0.001). The mean SUA level was higher among those cases who belonged to higher Killip class. Similar findings were observed by Kojima et al[18], Nadkar and Jain[17], and Killip and Kimball.[19] Though, Jularattanaporn et al[20] found that there was no observed relation between hyperuricemia and high TIMI risk scores or Killip class at first presentation or in-hospital adverse outcomes. Our study also found a statistically significant positive correlation (r=0.840; P=0.001) between CPK-MB on day of admission and Killip class: of 50 patients, two dies during the seven-day follow up. Among, none were in Killip class I, II or III, and both were in Killip class IV at the time of admission. In this, we found a close association between SUA concentration and Killip classification, suggestive of left ventricular failure. High SUA levels on admission were strongly related with adverse clinical outcome in patients who had acute MI. results also showed the value of SUA as a marker of short-term mortality in acute MI. Nadkar and Jain[17]

found that SUA levels were higher in patients with acute MI and correlated with Killip class. After acute MI, a combination of Killip class and SUA level is a good predictor of mortality.

CONCLUSION

This study concludes that, SUA levels were higher in patients with acute MI in comparison to normal healthy individuals. In patients with acute MI, SUA levels are elevated in systemic hypertension and diabetes mellitus. In acute MI patients, SUA can be used as a marker of short-term mortality. Hyperuricemia is found an indicator of poor prognosis in acute MI.

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