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Study to Evaluate Correlation between Serum Uric Acid Levels and Severity of Coronary Artery Disease in Patients Undergoing Coronary Angiography at Tertiary Health Care Centre

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Abstract

Background: Coronary heart disease constitutes an immense public health problem. Myocardial Infarction is one of kind components of cardio vascular disease burden all around the world. **Aim:** Many epidemiologic studies have shown raised uric acid levels to predict increased risk of cardiovascular events. Studies have addressed the value of this biomarker across the whole range of patients with ACS (Acute Coronary Syndrome) especially in Indian scenarios. Thus, the aim of this study was to investigate whether there is an association between serum uric acid level with CAD (Coronary Artery Disease) risk factors and its severity in ACS patients. Also to know the prognostic value of serum uric acid levels in those patients undergoing coronary angiography for underlying coronary artery diseases. **Material and Methods:** This prospective observational study included 200 random patients admitted with Acute Coronary Syndrome in the ICU and medicine department of the institution. The presence and severity of CAD was determined by the following clinical vessel score; Absent: No coronary lesions, 1 vessel, 2 vessels and 3 vessels - disease based on the number of coronary arteries involved. The correlation of serum uric acid levels with CAD risk factors and the severity of CAD in the ACS patients was analysed statistically. **Results:** There was a significant association between uric acid and the coronary angiographic findings (P<0.001). The patients in the hyperuricemia group had more number of coronary vessels involved than the other group. **Conclusion:** The serum uric acid level is correlated with the presence and severity of CAD by coronary angiographic findings (P<0.001) in the Acute Coronary Syndrome patients. Uric acid being an inexpensive and easily available biochemical marker, it will be of much use in settings with restricted resources. Serum uric acid levels can be used to predict the severity of CAD.

Keywords: ACS (Acute Coronary Syndrome), CAD (Coronary Artery Disease), Hyperurecemia

1. Introduction

Acute Coronary Syndromes (ACS) endures a leading cause of morbidity and mortality throughout the world. Though there has been various risk stratification schemes, ACS remain poorly characterized in terms of risk prediction[1].

Coronary heart disease constitutes an immense public health problem. Myocardial Infarctionis one of kind components of cardio vascular disease burden all around the world.

Coronary heart disease mortality has now decreased over the years, but the huge burden of it associated complications is on the rise.

Majority of deaths from cardiac events, including coronary vascular disease and cerebro-vascular accidents occur in developing countries. CAD has achieved epidemic proportion in India. Comparing the Indian subcontinent

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with other countries, coronary artery disease related mortality is still high with cardiac disease manifesting 10 years earlier than the remainder of the world[2].

Patients with chronic CAD who has most commonly present with stable angina and patients with acute ischaemic coronary syndrome. The after group is composed of patients with acute Myocardial Infarction (MI) with ST- segment elevation on their describing electrocardiogram and those with unstable angina and non-ST-segment elevation MI (UA/NSTEMI).

As per WHO, Cardio Vascular Diseases (CVDs) are the number one cause of death worldwide. CVDs are a group of disorders of the heart and blood vessels and include coronary heart disease, cerebro vascular disease, rheumatic heart disease and other conditions. Four out of five CVD deaths are because of heart attacks and strokes.

Early complications of STEMI include Ventricular dysfunction, Cardiogenic shock, Infarction related arrhythmias, thromboembolism, Left ventricular aneurysm, papillary muscle rupture, ventricular free wall and septalrupture[3,4],

With the developments in antithrombotic and reperfusion therapies over the past several years, the mortality of ACS has been steadily decreasing.

Elevation of cardiac markers following M Isuch a sproteins (myoglobin) and enzymes (CKMB,Troponin T, I) are released into blood stream from necrotic heart muscle and have a temporal profile in MI. However they do not correlate with myocardial function.

For CAD and heart failure raised SUA levels strongly suggestive of mortality and incardio vascular events in patients[5,6].

Uric Acid has a pathogenic role in cardio vascular disease. Hyperuricaemia is associated with dreaded effects on endothelium dysfunction, oxidative metabolism, platelet adhesiveness, haemorheology and aggregation.

In addition to the assessment of conventional risk factors inclinical practice, the evaluation of uric acid level conceivably provides significant prognostic benefits in terms of global cardio vascular risk and management of the patients.

Uric acid is the final product of purine catabolism in humans and is gladly tested in daily clinical practice. In spite of the fact that an association between elevated uric acid level and cardio vascular disease has been recognized for more than 130 years, the role of acid uric as a risk factor or a risk marker for cardio vascular disease remains a debatable problem[7].

The correlation between raised uric acid and mortality has been investigated in the general population, and in heart failure, hypertension, diabetes, confirmed coronary artery disease and AMI patients.

Current studies recommend that uric acid is biologically active and can stimulate oxidative stress, endothelial dysfunction, inflammation and vaso constriction[8,9].

Increased SUA in cardio vascular events may be a consequence of impairment of vascular Nitric Oxide, owing to the ability of Nitric Oxide to modulate Uric acid through its influence on Xanthine Oxidase. Activation of Xanthine Oxidase through free radical release causes leucocyte and end othelial cell activation[10]. Progressive elevation in Uric acid levels are coalition with increased vascular tone and depressed myocardial contractility via increase in Xanthine Oxidase activity.

Diagnostic cardiac catheterization and coronary angiography are considered the gold standard in the assessment of anatomy and physiology of the heart and its associated vasculature.

Cardiac catheterization and coronary angiography are indicated to evaluate the extent and severity of cardiac disease in symptomatic patients and to determine if medical, surgical or catheter based interventions are warranted.

As a predictor for mortality and morbidity following acute Myocardial Infarction this is one such study in which the prognostic role of serum uric acid in those patients undergoing coronary angiography and its correlation with severity of the CAD.

2. Materials and Methods

This will be a prospective study of aprrox 200 patients admitted for Coronary Angio Graphy between October 2018 to December 2020.

2.1 Type of Study

Prospective study will be done in department of Medicine, and cath-lab a tertiary care centre.

2.2 Eligibility Criteria

Inclusion

- All patients with Acute Coronary Syndrome admitted for CAG.

- Both failed and successful thromolysed cases were included.

Exclusion

- Patients taking oral uricosuric agents
- chronic kidney disease patients
- Gout
- Hematological malignancy
- Patients on drugs such a spyrizinamide, diuretics, salicylate

2.3 Study Design

- **Study Type/Design:** Prospective observational study**.**
- **Study Settings**: The study will be done in Department of Medicine and Cathlab and ICU CARE

2.4 Study Population

- **200**

2.5 Study Method

Patients were selected for study according to the inclusion and exclusion criteria mentioned above.

Detailed history regarding smoking, alcoholism, diabetes mellitus, hypertension, Drug intake was enquired.Vital signs, waist/Hipratio,15 lead-ECG findings were noted.

Blood sugar values, Fasting lipid profile and Fasting Serum uric acid were noted. After thrombolysis patients were followed up till they leave the hospital.

During the hospital stay they were closely monitored for development of complications like Heart failure, Cardiogenic shock, Arrhythmias, Thromboembolism and sudden cardiac death.

ECG was interpreted as ST elevation in:

L1,aVL,V1-V6	:	Extensive anterior
L1,aVL	:	High lateral
L1,aVL,V5-6	:	Antero-Lateral
V1-V4	:	Anteroseptal
LII,LIII,aVF	:	Inferior
LII,LIII,aVF,rV4	:	Inferior+Right ventricular
LII,LIII,aVF,V8,V9	:	Inferior+Posterior

2.6 Diabetes Mellitus

Patients were considered as diabetic only when they were known diabetic on treatment or fasting blood sugar>126 mg%.

2.7 Hypertension

Patients were considered hypertensive only when they were known hypertensive on treatment or Systolic BP>140 mmHg and Diastolic BP>90 mg%.

2.8 Central Obesity

Waist Hip Ratio	>1 for men Waist
Hip Ratio	>0.8 for women

2.9 Dyslipidemia

Total cholesterol	>	200 mg%
LDL cholesterol	>	160 mg%
HDL cholesterol	<	40 mg%
Triglycerides	>	200mg%

2.10 Serum Uric Acid

Estimated by Uricase method**.**

Considered elevated if serum level >7 mg% for male, >6 mg% for female.

2.11 Methodology

Based on the criteria patients were selected and all subjects underwent detailed history and clinical examination. Patients with hyperuricemia were defined as serum uric acid concentrations ≥7.0 mg/dL in men and ≥6.0 mg/dL in women. Patient also underwent the following base line investigations: CBC, Blood sugar levels, renal function test, serum electrolytes and cardiac enzymes (eg. Trop I, CPK-MB). The presence and severity of CAD was determined by the following clinical vessel score: Absent: no coronary lesions, 1 vessel, 2 vessel and 3 vessel - disease based on the number of coronary arteries involved. The degree of stenosis was defined as the greatest percentage reduction of luminal diameter in any view compared with the nearest normal segment and was determined visually.

2.12 Statistical Analysis

The data was coded and entered into Microsoft Excel spreadsheet. Analysis was doneusing SPSS version 20 (IBM SPSS Statistics Inc., Chicago, Illinois, USA) Windows software program. Descriptive statistics included computation of percentages, means and standard deviations. The unpaired t-test [for quantitative data within two groups] and Analysis of Variance (ANOVA)

[for quantitative data within three groups] was used for quantitative data comparison of all clinical indicators. Chi-square test was used for qualitative data whenever two or more than two groups were used to compare. Level of significance was set at $P \leq 0.05$.

3. Results

Table 1. Age and severity of CAD

	N	Mean	Std. Deviation
S	121	56.57	11.328
D	56	58.52	11.527
T	23	57.91	10.887

Table 2. Sex and severity of CAD P value = 0.008(S)

			sex		Total
			F	M	
Severity of CAD	S	N	52	69	121
		%	43.0%	57.0%	100.0%
	D	N	12	44	56
		%	21.4%	78.6%	100.0%
	T	N	5	18	23
		%	21.7%	78.3%	100.0%
Total		N	69	131	200
		%	34.5%	65.5%	100.0%

Table 3. Dyspnoea and severity of CAD

			dyspnoea		Total
			N	Y	
Severity of CAD	S	N	114	7	121
		%	94.2%	5.8%	100.0%
	D	N	18	38	56
		%	32.1%	67.9%	100.0%
	T	N	0	23	23
		%	0.0%	100.0%	100.0%
Total		N	132	68	200
		%	66.0%	34.0%	100.0%

P value = 0.001(S)

Table 4. HTN and severity of CAD

			HTN		Total
			N	Y	
Severity of CAD	S	N	68	53	121
		%	56.2%	43.8%	100.0%
	D	N	13	43	56
		%	23.2%	76.8%	100.0%
	T	N	5	18	23
		%	21.7%	78.3%	100.0%
Total		N	86	114	200
		%	43.0%	57.0%	100.0%

P value = 0.001(S)

Table 5. Cardiac markers and severity of CAD

			cardiacmarkers		Total
			HIGH	LOW	
Severity of CAD	S	N	40	81	121
		%	33.1%	66.9%	100.0%
	D	N	49	7	56
		%	87.5%	12.5%	100.0%
	T	N	20	3	23
		%	87.0%	13.0%	100.0%
Total		N	109	91	200
		%	54.5%	45.5%	100.0%

P value = 0.001(S)

Table 6. Type of MI and severity of CAD

			type of MI		Total
			NSTE MI	STE MI	
Severity of CAD	S	N	60	61	121
		%	49.6%	50.4%	100.0%
	D	N	22	34	56
		%	39.3%	60.7%	100.0%
	T	N	7	16	23
		%	30.4%	69.6%	100.0%
Total		N	89	111	200
		%	44.5%	55.5%	100.0%

Table 7. UA and severity of CAD

		UA day1	UA day3
S	N	121	121
	Mean	5.9595	5.7777
	Std. Deviation	1.24737	1.07047
D	N	56	56
	Mean	8.8946	8.4661
	Std. Deviation	1.08232	1.13990
T	N	23	23
	Mean	11.8739	11.6391
	Std. Deviation	.85239	1.01835
	Pvalue	0.001(S)	0.001(S)

Table 8. Mortality and severity of CAD

			mortality		Total
			N	Y	
Severity of CAD	S	N	121	0	121
		%	100.0%	0.0%	100.0%
	D	N	47	9	56
		%	83.9%	16.1%	100.0%
	T	N	14	9	23
		%	60.9%	39.1%	100.0%
Total		N	182	18	200
		%	91.0%	9.0%	100.0%

P value = 0.001(S)

Table 9. HTN and UA at 3 day

	N	Mean	Std. Dev	P value
N	86	6.36	1.89	0.001(S)
Y	114	7.83	2.31	

4. Discussion

Unlike many disabling medical conditions, cardio vascular disease which is already one of the most common cause of deaths is almost entirely preventable. Acute Coronary Syndrome is the most serious consequence of cardio vascular disease. We also evaluated the correlation of serum uric acid with various CAD risk factors in the selected ACS patients. We did not find any significant association between SUA and gender of the patient. Age as a CAD risk factor was also not significantly associated with SUA concentration in our study. In current study there is a significant relation (p = 0.001) between serum uric acid level and patients who was known or found to be hypertensive on admission. In current study showed that known hypertensive patients had more hyperuricemia. Similar findings were also seen in a study by Schmidt et al, where compared with normotensive patients SUA levels were significantly elevated in hypertensive patients[11]. By investigating our patients undergoing coronary angiography, we found that hyperuricemia was associated with the presence of angiographic documentation of CAD in the whole population of ACS patients who were studied. However, we found that there was an increase in the severity of CAD scaled by clinical vessel score with the increase in the SUA levels. Hyper uricemia has also been studied as a marker of poor prognosis and increased mortality in general population, other co-morbidities and established IHD patients. Kojima S et al. also studied that serum uric acid predicted the development of heart failure and long term mortality in acute MI. This risk was even stronger in women[12,13,14,15,16]. Our study found that there was a significant association between serum uric acid and dyslipidemia (p = 0.002) and previous history of coronary artery disease (p = 0.004). In current study mortality rate increases as severity of CAD increases, its almost nil in single vessel disease but increases to 39.1% in triple vessel disease.

5. Conclusion

Serum uric acid aptly raised in patients with Acute Coronary Syndrome. There is a significant correlation between serum uric acid and CAD risk factors like hypertension and diabetes. The serum uric acid level is correlated with the presence and severity of CAD by coronary angiographic findings. There is significant association of Hyperuricemia with infarction pattern and severity of coronary artery disease in those patients undergoing coronary angiography. Hyperuricemia has poor prognostic value in those patients undergoing coronary angiography for underlying coronary artery diseases.

6. References

1. Kolansky DM. Acute Coronary Syndromes: morbidity, mortality and pharmaco economic burden. Am J Manag

Care. 2009; 15(suppl):S36–4. PMid: 19286476. https://doi.org/10.1016/j.yebeh.2009.03.006

2. Alpert JS, et al. Definition of myocardial infarction-a global consensus document of The Joint ESC/ACC/AHA/WHF/WHO Task Force. 2007 (in press).
3. BirnbaumY, Chamoun AJ, Anzuini A, Lick SD, Ahmad M, Uretsky BF: Ventricular free wall rupture following acute myocardial infarction. Coron Artery Dis. 2003; 14:463–70. PMid: 12966268. https://doi.org/10.1097/00019501-200309000-00008
4. Reynen K, Strasser RH. Images in clinical medicine. Impending rupture of the myocardial wall. N Engl J Med. 2003; 348:e3. PMid: 12621147. https://doi.org/10.1056/ENEJMicm020410
5. Gertler MM, Garn SM, Levine SA. Serum uric acid in relation to age and physique in health and in coronary heart disease. Ann Intern Med. 1951; 34:1421. PMid: 14838504. https://doi.org/10.7326/0003-4819-34-6-1421
6. Kohn PM, Prozan GB. Hyperuricemia-relationship to hypercholesterolemia and acute myocardial infarction. JAMA. 1959; 170:1909. PMid: 13672785. https://doi.org/10.1001/jama.1959.03010160025007
7. Culleton BF, Larson MG, Kannel WB, Levy D. Serum uric acid and risk for cardiovascular disease and death: The Framingham Heart Study. Ann Intern Med. 1999; 131:7–13. PMid: 10391820. https://doi.org/10.7326/0003-4819-131-1-199907060-00003
8. Master AM. Acute coronary artery diseases; history, incidence, differential diagnosis and occupational significance. Am J Med. 1947 May; 2(5):501–16. doi:10.1016/0002-9343(47)90096-x.PMID:20294530 https://doi.org/10.1016/0002-9343(47)90096-X
9. Rusnak J, Fastner C, Behnes M, Mashayekhi K, Borggrefe M, Akin I. Biomarkers in Stable Coronary Artery Disease. Curr Pharm Biotechnol. 2017;18(6):456-–71. doi:10.2174/1389201018666170630120805.PMID:28669333
10. Leyva F, Anker SD, Godsland IF. Uric acid in chronic heart failure: A marker of chronic inflammation. European Heart Journal. 1998; 19(12):1814–22. PMid: 9886724. https://doi.org/10.1053/euhj.1998.1188
11. Schmidt MI, Watson RL, Duncan BB, et al. Clustering of dyslipidemia, hyperuricemia, diabetes and hypertension and its association with fasting insulin and central and overall obesity in a general population. Atherosclerosis risk in communities study investigators. Metabolism. 1996; 45(3):699–706. https://doi.org/10.1016/S0026-0495(96)90134-1
12. Demir B, Caglar IM, Ugurlucan M, Ozde C, Oktay Tureli H, Ciftci S. The relationship between severity of Calcific Aortic Stenosis and Serum Uric Acid Levels. Angiology. 2012; 63(8):603–8. PMid: 22261436. https://doi.org/10.1177/0003319711433198
13. Kojima S, Sakamoto T, Ishihara M, Kimura K, Miyazaki S,Yamagishi M. Prognostic usefulness of serum uric acid after acute Myocardial Infarction (the Japanese Acute Coronary Syndrome Study). Am J Cardiol. 2005; 96(4):489–95. PMid: 16098298. https://doi.org/10.1016/j.amjcard.2005.04.007
14. Nadkar MY, JainVI. Serum Uric Acid in Acute Myocardial Infarction. JAPI. 2008; 56(1):759–62. https://doi.org/10.1057/mds.2009.50
15. Baruah M, Nath CK, Chaudhury M, Devi R, Shaker A, Ivvala. Astudy of Serum Uric Acid and C-Reactive Protein in Acute Myocardial Infarction. International J of Basic Medical Sciences and Pharmacy. 2012; 2(1):21–4.
16. Kojima S, Sakamoto T, Ishihara M, et al. Prognostic usefulness of serum uric acid after acute Myocardial Infarction (Japanese Acute Coronary Syndrome).

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