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STUDY OF SERUM URIC ACID LEVELS IN ACUTE MYOCARDIAL INFARCTION AND IT'S PROGNOSTIC SIGNIFICANCE

Medicine

KEYWORDS: MI; Serum Uric acid; Killip's class; Framinghams study

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ABSTRACT

BACKGROUND: Acute myocardial infarction (AMI) is overwhelmingly the most important form of ischemic heart disease (IHD) which continues to be the leading cause of death in the industrialized and developing countries like India, despite spectacular progress in their prevention, detection and treatment over the last three decades. The relation between uric acid and cardiovascular disease is observed not only with frank hyperuricemia (defined as more than 6mg/dl in women & more than 7 mg/dl in men) but also with uric acid levels considered to be normal but at high range.

AIMS AND OBJECTIVES:

1. To study the serum uric acid level of patients of Acute Myocardial Infarction admitted to our hospital.
2. To correlate the serum uric acid levels on day 0,3,7 and compare with killips class with coefficient of correlation.
3. To study the morbidity and mortality of these patients.

METHODS: This hospital based case study was performed in Dr. PSIMS & RF, Chinoutpalli , Krishna district, Andhra Pradesh from October 2013 to September 2014. A total of 75 cases of Acute MI were studied. All the subjects were interviewed , examined and investigated as per the predesigned proforma..

RESULTS:75 cases of acute MI were included in the study. Mean SUA for discharged patients was 4.67 ±1.95 /dl and it was 7.1 ±1.45 mg/dl for the patients who died in the hospital. SUA levels were significantly higher in the patients who succumbed as compared to those who were discharged from the hospital.

CONCLUSION:

1.Serum uric levels are correlated with killip'sclassification.Patients of higher killips class have higher uric acid levels.
2.Combination of killip's class and serum uric acid levels after acute myocardial infarction is a good predictor of mortality.
3. Serum uric acid levels were high in the patients who died in the seven day follow up period and the patients who died were also in higher killip's class.

INTRODUCTION:

Acute myocardial infarction (AMI) is the most important form of ischemic heart disease (IHD) which continues to be the leading cause of death in the industrialized and developing countries like India[1].A large number of asymptomatic individuals are at serious risk of developing myocardial infarction (MI) because of their genetic predisposition, smoking behaviour and sedentary lifestyle. 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. Many studies including the National Health and Nutrition Examination Survey (NHANES) study [2]concluded that uric acid is an independent risk factor for development of cardiovascular and cerebrovascular diseases. Framingham Heart study[3]concluded that an association between hyperuricemia and cardiovascular diseases merely reflects the link between serum uric acid and other risk factors, including hypertension, renal disease, elevated lipoprotein levels and the use of diuretics.Adenosine synthesized locally by vascular smooth muscle in cardiac tissue is rapidly degraded by the endothelium to uric acid, which undergoes rapid efflux to the vascular lumen due to low intracellular pH and negative membrane potential[7] Xanthine oxidase [8] and uric acid [9]synthesis are increased in vivo under ischemic conditions, and therefore elevated serum uric acid may actas a marker of underlying tissue ischemia. Although the mechanism by which uric acid may play a pathogenetic role in cardiovascular disease is unclear, hyperuricemia is associated with deleterious effects on endothelial dysfunction, oxidative metabolism, platelet adhesiveness, and aggregation. Uric acid may have direct role in atherosclerotic process because atherosclerotic plaque contains more uric acid than control arteries. Hyperuricemia via purine metabolism may also promote thrombus formation.[4] The relation between uric acid and cardiovascular disease is observed not only with frank hyperuricemia (defined as more than 6mg/dl in women & more than 7 mg/dl in men) but also with uric acid levels considered to be normal but at high range.[5]This study was done to determine whether raised serum uric acid levels were an independent risk factor for acute MI and to determine its prognostic importance if any.

MATERIALS AND METHODS:

SOURCE OF DATA COLLECTION:

The study was conducted at Dr.PSIMS & RF, Chinoutpalli , Krishna district, Andhra Pradesh , from January 2014 to December 2016.

A total of 75cases of Acute MI were studied. All the subjects were interviewed,examined and investigated as per the predesigned proforma.

INCLUSION CRITERIA:

Patients more than 18 years of age diagnosed to have acute MI who presented to hospital within 24 hrs of onset of symptoms were included in the study. Acute MI was defined as, 'increased myocardial enzyme concentrations with typical chest pain persisting more than 30 minutes or electrocardiographic changes (including ischemic ST-

segment depression, ST- segment elevation or pathologic Q waves). Increased enzyme concentrations were defined as peak creatine phosphokinase level more than 2 times upper limit of normal.[6]

EXCLUSION CRITERIA:

1. Subjects with following conditions were excluded from the study,
2. Patients with a condition known to elevate uric acid level e.g. chronic kidney disease, gout, haematological malignancy and hypothyroidism were excluded.
3. Patients receiving drugs affecting serum UA levels (diuretics, ethambutol, pyrizinamide, salicylates, losartan, allopurinol, probenecid, atorvastatin, fenofibrate).Chronic alcoholics.

RESULTS :

TABLE 1

URIC ACID LEVELS DAY 0

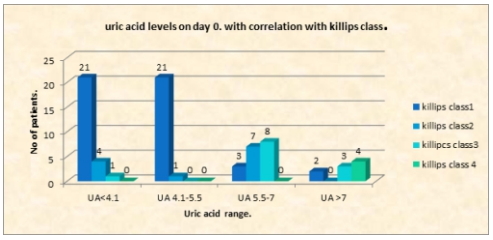


TABLE 2

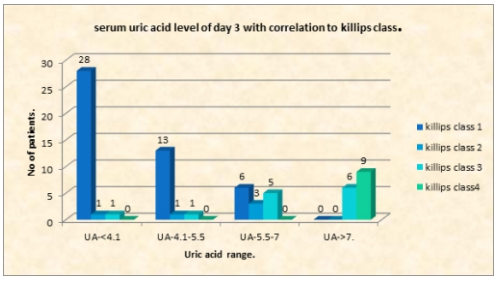
CROSS TABULATION BETWEEN SERUM URIC ACID AND KILLIPS CLASS ON DAY OF ADMISSION

KILLIPS CLASS	<4.1	4.1-5.5	5.5-7	>7	TOTAL
1	21	21	3	2	47
2	4	1	7	0	12
3	1	0	8	3	12
4	0	0	0	4	4
TOTAL	26	22	18	9	75

On applying chi-square test ,p value significant (<0.05)As p value is significant,so there is association between serum uric acid concentration and killips class on day of admission.serum uric acid levels are higher in patients who are in higher killips class.

URIC ACID LEVELS DAY 3

TABLE 3



CROSS TABULATION BETWEEN SERUM URIC ACID AND KILLIPS CLASS ON DAY 3

TABLE 4

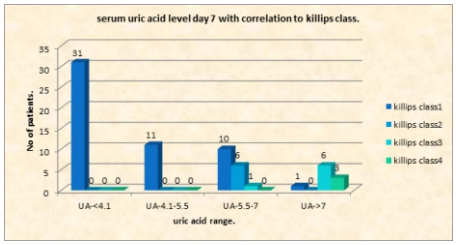
KILLIPS CLASS	<4.1	4.1-5.5	5.5-7	>7	TOTAL
1	28	13	6	0	47
2	1	1	3	0	5
3	1	1	5	6	13
4	0	0	0	9	9
TOTAL	30	15	14	15	74

On applying chi-square test ,p value significant (<0.05)

As p value is significant,so there is association between serum uric acid concentration and killips class on day of 3,serum uric acid levels are higher in patients who are in higher killips class.

URIC ACID LEVELS DAY 7

TABLE 5



CROSS TABULATION BETWEEN SERUM URIC ACID AND KILLIPS CLASS ON DAY7.

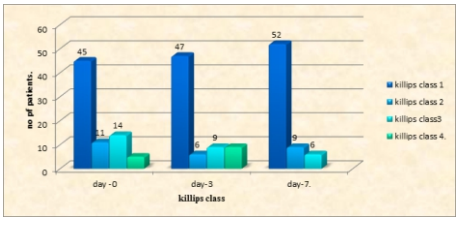
TABLE 6

KILLIPS CLASS	<4.1	4.1-5.5	5.5-7	>7	TOTAL
1	31	11	10	1	53
2	0	0	6	0	6
3	0	0	1	6	7
4	0	0	0	3	3
TOTAL	31	11	17	10	69

On applying chi-square test ,p value significant (<0.05)

As p value is significant,so there is association between serum uric acid concentration and killips class on day 7.serum uric acid levels are higher in patients who are in higher killips class.

TABLE 7



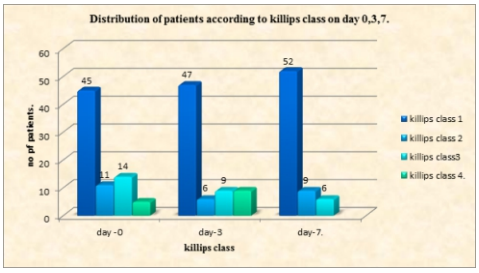
FREQUENCY DISTRIBUTION OF PATIENTS ACCORDING TO KILLIPS CLASS ON DAY 0,DAY 03,DAY 07.

TABLE 8

KILLIPS CLASS	DAY 0 N(%)	DAY 03 N(%)	DAY 07 N(%)
1	45(60%)	47(64%)	52(75%)
2	11(15%)	6 (8%)	9 (14%)
3	14(19%)	9 (13%)	6 (8%)
4	5(6%)	9 (15%)	2 (3%)
TOTAL	75	74	69

TABLE 9

From table 9, it is observed that patients have shifted from class 2 and 3 to class 1 and 4.one patient



expired till day 3,and 5 till day 7,total 6 patients died during 7 day follow up.

TABLE 10

RELATION BETWEEN SERUM URIC ACID AND MI OUTCOME

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Outcome	Discharge (n=69)	Death (n=6)	p value
Mean SUA ± SD (in mg/dl)	4.67 ±1.95	7.1 ±1.45	0.00
Range (in mg/dl)	3.2-7.8	5.8-9.6	

Mean SUA for discharged patients was4.67 ± 1.95 /dl and it was 7.1 ± 1.45 mg/dl for the patients who died in the hospital. SUA levels were significantly higher in the patients who succumbed as compared to those who were discharged from the hospital (p = 0.000).

DISCUSSION:

The well recognized risk factors like age, male sex, smoking, diabetes, hypertension, metabolic syndrome explain only a part of the MI cases. Hence a search for other risk factors is the need of the hour. Many studies have found conflicting role of uric acid in patients with cardiovascular disease It has been reported that UA promotes vascular smooth muscle proliferation and upregulates the expression of platelet-derived growth factor and monocyte chemoattractant protein-1[7]. Hypoxanthine is converted to uric acid via xanthine. This reaction can be catalyzed by xanthine hydrogenase and xanthine oxidase, the latter of which produces uric acid and superoxide. Thus, it is possible that, in certain diseased conditions, hyperuricemia is accompanied by the increased production of reactive oxygen species, which may result in the modulation of vascular contractility [8]. Consistent with this is the notion that allopurinol, a xanthine oxidase inhibitor, not only reduces the serum UA levels but also improves vascular endothelial function in patients with chronic heart failure[9]. Another possible explanation is that hyperuricemia may induce endothelial dysfunction by decreasing the production of nitric oxide in the vascular endothelial cells[10]. Adenosine synthesized locally by vascular smooth muscle in cardiac tissue is rapidly degraded by the endothelium to UA, which undergoes rapid efflux to the vascular lumen due to low intracellular pH and negative membrane potential [7]. Uric acid synthesis is increased in vivo under ischemic conditions, and therefore elevated serum UA may act as a marker of underlying tissue ischemia In conclusion, elevated serum UA may be a marker of local and systemic tissue ischemia and provides one possible explanation for a non-causal associative link between hyperuricemia and cardiovascular disease. Thus our study proves that SUA can be used as a marker forincreased risk of acute MI. Furthermore, SUA can also be used for risk stratification after acute MI.

CONCLUSION:

1. Serum uric levels are correlated with killip'sclassification.Patients of higher killips class have higher uric acid levels.

2. Combination of killip's class and serum uric acid levels after acute myocardial infarction is a good predictor of mortality.

3. Serum uric acid levels were high in the patients who died in the seven day follow up period and the patients who died were also in higher killip's class

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