**Research Article**

# Serum uric acid as an indicator of bad prognosis in Myocardial infarction

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## ABSTRACT

***Background:*** *Cardiovascular disease (CVD) has become a major health issue in India & is probably one of the most preventable disease. Studies have quoted serum uric acid (SUA) & killip class as bad prognostic indicator in patients of myocardial infarction (MI).*

***Objective:*** *This study aims to find the role of SUA & killip classification as prognostic marker in patients of MI.*

***Material & methods:*** *Fifty patients of acute MI fulfilling the inclusion and exclusion criteria were compared with 50 age and sex matched healthy controls. A detailed history and physical examination with special reference to Killip class was carried out. Patients were followed up till hospital stay & then in OPD. SUA was measured on day 0, 3, 7 and also on day 30 of MI. SUA levels & Killip class was compared with coefficient of correlation.*

***Results:*** *There was a statistically significant higher level of SUA in patients of acute MI on day of admission as compared to controls ($p < 0.05$). At the time of admission, patients in killip class III & IV had higher SUA levels than those in class I & II. Four patients who died during follow up were in class IV. SUA levels are positively correlated to killip class i.e higher the SUA levels higher is Killip class.*

***Conclusion:*** *Higher SUA & higher killip class can be considered as indicator of bad prognosis in patients of MI.*

***Keywords:*** *Serum uric acid, killip classification, myocardial infarction, prognostic marker, cardiovascular disease*

## Introduction

Myocardial infarction (MI) commonly known as a heart attack is the interruption of blood supply to part of the heart, causing heart cells to die. This is most commonly due to occlusion of a coronary artery following the rupture of a vulnerable atherosclerotic plaque, which is an unstable collection of lipids and macrophages in the wall of coronary artery. The resulting ischemia and oxygen shortage, if left untreated for a sufficient period of time, can cause damage or death (infarction) of myocardium. In 2002, 12.6 percent of deaths worldwide were from ischemic heart disease. [1] In India, cardiovascular disease (CVD) is the leading cause of death. [2] Although a relatively new epidemic in India, it has quickly become a major health issue with deaths due to CVD expected to double during 1985-2015. [3-4]

Classical symptoms of acute MI include sudden chest pain typically radiating to the left arm or left side of the neck, breathlessness, vomiting, palpitations, sweating, and anxiety (often

described as a sense of impending doom). MI can occur without any warning symptoms. These are called silent MI. Some may be associated with "atypical' symptoms, such as heartburn, nausea, or sudden light-headedness and sweating. These are more common in women, diabetics, and people older than 65 years. [5] Risk factors for atherosclerosis are generally the risk factors of MI. [6] Older age, Male sex, Tobacco smoking, Hypercholesterolemia (more accurately hyperlipoproteinemia, especially high- LDL and low-HDL), Hyperhomocysteinemia (high homocysteine), a toxic blood amino acid that is elevated when intakes of vitamins B2, B6, B12 and folic acid are insufficient), Diabetes (with or without insulin resistance), High blood pressure, Obesity (defined by a body mass index of more than 30 kg/m² or alternatively by waist to hip ratio >0.9 in women & >1.0 in men).[6]

Many of these risk factors are modifiable; so many heart attacks can be prevented by maintaining a healthier lifestyle. Physical activity, for example, is associated with a lower risk profile. [7] Non-modifiable risk factors include age, sex, and family history of an early heart attack (before the age of 60), which is thought of as reflecting a genetic predisposition.

Diagnosis is made when there is a rise and/or fall of cardiac biomarkers, along with supportive evidence in the form of typical symptoms, suggestive electrocardiographic changes, or imaging evidence of new loss of viable myocardium or new regional wall motion abnormality. [8]

Over recent years there has been renewed debate about the nature of the association between raised SUA concentration and CVD. [9] Several large studies have identified the value, in populations, of SUA concentration in predicting the risk of cardiovascular events, such as MI. [10, 11] The First National Health and Nutrition Examination Survey [12] (NHANES I) Epidemiologic Follow-up Study was done to study the importance of hyperuricemia as a risk factor in persons with ischemic heart disease. It was done among 5,421 persons. Baseline data were collected in 1971-1975 and follow-up was through 1987. It was observed that no associations were seen among men, but, among women, the serum uric acid level was predictive of mortality from all causes and from ischemic heart disease. [12]

Cross-sectional population-based study of epidemiological follow-up data from the First National Health and Nutrition Examination Survey (NHANES I) from 1971-1975 (baseline) and data from NHANES I Epidemiologic Follow-up Study (NHEFS) carried to determine the association of SUA levels with cardiovascular mortality concluded that increased SUA levels are independently and significantly associated with risk of cardiovascular mortality. [13]

Torun et al [14] noted that the risk of death due to ischemic heart disease increased by 77% (men) and by 300% (women) when SUA levels were in the highest quartile (> 416 µmol/L or 7 mg/dL) compared with the lowest quartile (< 321 µmol/L or 5.4 mg/dL).

Some studies have correlated higher SUA levels with Killip class as a bad prognostic marker in patients of MI. [15,16,17]

SUA levels, killip class can be used as indicator of bad prognosis in patients of MI. Killip classification is indicator of severity of heart failure. Killip classification include four classes where Class I is No sign of pulmonary or venous congestion, Class II is Moderate heart failure, as crackles in the lungs with S3 gallop & elevated JVP, Class III is severe

heart failure, pulmonary edema, Class IV is cardiogenic shock with systolic BP <90 mm Hg with peripheral vasoconstriction, peripheral cyanosis, mental confusion & oliguria. This study aims to find the role of SUA & Killip class as an indicator of bad prognosis in patients of MI

**Material and methods**

The present study on SUA in acute MI consists of 50 cases of MI taken from ICCU of Rajindra Hospital, Patiala. Study was undertaken jointly by Department of Medicine, and Department of Biochemistry, Government Medical College and Rajindra Hospital, Patiala. Inclusion criteria is Patients fulfilling the diagnostic criteria of acute MI (on basis of history, physical examination, ECG changes and serum biochemical, cardiac markers), patients age >18 years, patients who have given consent in writing. Exclusion criteria is patients of age <18 years, Any patient with a condition known to elevate uric acid levels e.g. gout, hematological malignancy, hypothyroidism, patients unwilling or unable to give informed written consent. Fifty patients of acute MI fulfilling the inclusion and exclusion criteria were enrolled for the study. A detailed history and physical examination with special reference to Killip class was carried out. All patients went routine investigation including Hb, TLC, DLC, renal function tests (blood urea and serum creatinine), ECG, and other tests as required. Patients were followed up till hospital stay & then in OPD. Serum uric acid was measured on day 0, 3, 7 and also on day 30 of MI. Statistical analysis was carried out and the levels of uric acid on day 0, 3, 7 and 30 were compared. Uric acid levels & Killip class was compared with coefficient of correlation. 50 age and sex matched healthy controls were evaluated for comparison.

**Results**

Present study was conducted in 50 patients of acute MI, who presented to hospital within 24 hrs of onset of symptoms. Out of 50 patients, 37 had ST-elevation myocardial infarction, while 13 patients were of non-ST elevation myocardial infarction. Thirty three patients were thrombolysed while four were not thrombolysed due to delayed presentation. Fifty age and sex matched healthy controls were also evaluated for comparison. The mean age of control gp is 58.9±13.2 years (range of 40-85 years). The mean age of patients of MI was 57.8 ± 11.5 years (range of 40-85 years) (Table 1). The maximum number of patients, (42%) was in 50-60 years of age group. Mean SUA level was 7.05 ± 1.06 in age gp <50 years, 6.60±1.04 in age gp 50-60 years, and 7.25 ± 2.04 in age gp >60 years. Out of total 50 patients, 35 patients (70%) were males; whereas 15 patients (30%) were females. SUA ranged from 5.4 – 14.4 mg/dl (mean 7.17±1.61 mg/dl) for males and 5.8 – 8.0 mg/dl (mean 6.34±0.665) for females (Table2).

SUA was measured on day 0, 3, 7 and also on day 30 (Table 3) of MI and levels were compared. The mean SUA levels were 6.928±1.443, 6.166±1.264, 5.636±1.19, 5.02±0.684 on day 0, 3, 7, 30 respectively. SUA ranged from 3.8- 7.2 mg/dl in controls with mean value of 5.098± 0.864 mg/dl. There was a statistically significant higher level of SUA in patients of AMI on day of admission as compared to controls ($p < 0.05$). SUA gradually decrease with time as is clear from values mentioned. On day 30, there was no statistical significant relation ($p=0.656$) between the two suggesting that SUA levels on day 30 were comparable to that in control group. Correlation of SUA & Killip class is observed on day 0, 3, 7, 30 (Table 6). On day 0, out of total 50 patients, 15 (30%)

were in Killip class I, 15 (30%) patients were in Killip class II, 12 (24%) patients in Killip class III and 8 (16%) patients in Killip class IV. On day 3, the number of patients in Killip class I increased following treatment and clinical improvement and were 24 (48%) in Killip class I and 16 (32%), 8(16%), 2 (4%) in Killip class II, III and IV respectively. On day 7, the respective number was 36 (75%), 9 (18.75%), 2 (4.16%) and 1 (2.08%) in Killip class I, II, III and IV. On day 30, thirty nine (90.7%) patients were in Killip class I and 4 (9.3%) patients in Killip class II while no patient was in class III and IV. The SUA levels on day 0 ranged between 4.1-5.5 in 3 patients, 5.6-7mg/dl in 29 patients & >7mg/dl in 18 patients. On day 3, 23 patients had SUA between 4.1- 5.5mg/dl, 17 patients had SUA between 5.6-7mg/dl & was >7mg/dl in 10 patients. On day 7, 25 patients had SUA between 4.1-5.5, 22 patients were having SUA between 5.6-7 & only 1 patient had >7mg/dl SUA. On day 30, 7 patients had SUA levels ≤4mg/dl, 30 patients had SUA levels between 4.1-5.5, 6 patients had SUA between 5.6-7mg/dl & no patients was having >7mg/dl SUA. At the time of admission, patients in killip class III & IV had higher SUA levels than those in class I & II. Two patients were lost at follow up on day 7 and the five more patients were lost at day 30. Four patients who died during follow up were in class IV. Comparison of SUA levels was also done in hypertensive & non hypertensive patients (Table 4), diabetic & non diabetic patients (Table 4). Serum uric acid ranged from 5.5 – 9.0 mg/dl (mean 6.64±1.90) in hypertensive patients and 5.4 – 14.4 mg/dl (mean 7.29±0.878) for non-hypertensive patients. In diabetic patients, SUA levels ranged from 5.5-14.4 mg/dl with mean value 7.02± 2.079 mg/dl and in non diabetic patients it ranged from 5.4 to 9.0 mg/dl with mean value 6.85± 0.896 mg/dl. SUA levels comparison was also done with lipid levels (Table 5) in patients of acute MI. Out of 50 patients, 32 patients had LDL cholesterol below 100 mg/dl and 18 patients had LDL cholesterol between 100-200 mg/dl These patients had mean SUA levels of 6.59 ± 0.82 and 7.53 ± 2.05 mg/dl respectively. Considering HDL cholesterol, out of total 50 patients, 5 patients had HDL cholesterol below 30 mg/dl and 40 patients had HDL cholesterol between 30-50 mg/dl and 5 patients had HDL cholesterol more than 50 mg/dl. These patients have mean SUA levels of 9.84± 2.63, 6.62±0.81 and 6.48±0.59 mg/dl respectively. Triglyceride levels were below 150 mg/dl in 24 patients, between 150- 250 mg/dl in 21 patients & >250 mg/dl in 5 patients. These patients have mean SUA levels of 6.56±0.82, 7.34±1.95 and 6.96±1.003 mg/dl respectively.

**Table 1: Distribution of patients of acute myocardial infarction according to age**

| Age(years) | No. of cases | No. Of controls |
|---|---|---|
| **<50** | **13** | **14** |
| **50-60** | **21** | **17** |
| **>60** | **16** | **19** |
| **Range( in years)** | **40-85** | **40-85** |
| **Mean ±SD** | **57.8±11.5** | **58.9±13.2** |
| | **p>0.05** | |

**Table 2: Comparison of serum uric acid levels in male and female patients of acute MI**

| Gender | No. of patients | Mean SUA ± SD |
|---|---|---|
| **Male** | 35 | 7.17 ± 1.61 |
| **female** | 15 | 6.34± 0.665 |
| | | P =0.059 |

**Table 3: Comparison of Serum Uric Acid levels in patients & controls on Day 0, 3, 7 & 30**

| | Day 0 | Day 3 | Day 7 | Day 30 |
|---|---|---|---|---|
| | Mean ±S.D. | Mean ±S.D | Mean ±S.D | Mean ±S.D |
| **patients** | 6.928±1.443 | 6.166±1.264 | 5.636±1.19 | 5.023±0.684 |
| **control** | 5.098±0.864 | 5.098±0.864 | 5.098±0.864 | 5.098±0.864 |
| | P<0.0001 | p< 0.05 | p< 0.05 | P= 0.656 |

**Table 4: Comparison of serum uric acid levels in hypertensive and non hypertensive, diabetic & non diabetic patients of MI**

| | Hypertensive | Non hypertensive | Diabetic | Non Diabetic |
|---|---|---|---|---|
| No. of patients | 28 | 22 | 19 | 31 |
| Mean SUA ± SD | 6.646±1.904 | 7.286± 0.878 | 7.021± 2.079 | 6.854±0.896 |
| | p>0.05 | | p>0.05 | |

**Table 5: Comparison of Serum uric acid levels with LDL, HDL cholesterol and Triglyceride in patients of MI**

| | LDL C | | | HDL C | | | TG | | |
|---|---|---|---|---|---|---|---|---|---|
| | <100 | 100-200 | >200 | <30 | 30-50 | >50 | <150 | 150-250 | >250 |
| **No. of patients** | 32 | 18 | 0 | 5 | 40 | 5 | 24 | 21 | 5 |
| **Mean SUA ± SD** | 6.59± 0.817 | 7.527± 2.046 | - | 9.84± 2.628 | 6.62± 0.813 | 6.48± 0.597 | 6.562± 0.822 | 7.338± 1.945 | 6.96± 1.003 |
| | P=0.0259 | | | P<0.05 | | | P= 0.20 | | |

**Table 6: Serum uric acid levels and Killip class on day 0, 3, 7 & 30 in patients of MI**

| | SUA levels | Killip class I | Killip class II | Killip class III | Killip class IV | total | Coefficient of correlation r, & p value |
|---|---|---|---|---|---|---|---|
| **Day 0** | ≤4.0 | 0 | 0 | 0 | 0 | 0 | r=0.749, p<0.05 |
| | 4.1-5.5 | 3 | 0 | 0 | 0 | 3 | |
| | 5.6-7.0 | 12 | 14 | 3 | 0 | 29 | |
| | >7.0 | 0 | 1 | 9 | 8 | 18 | |
| | **total** | **15** | **15** | **12** | **8** | **50** | |
| **Day 3** | ≤4.0 | 0 | 0 | 0 | 0 | 0 | r=0.813, p<0.05 |
| | 4.1-5.5 | 20 | 3 | 0 | 0 | 23 | |
| | 5.6-7.0 | 4 | 10 | 3 | 0 | 17 | |
| | >7.0 | 0 | 3 | 5 | 2 | 10 | |
| | **total** | **24** | **16** | **8** | **2** | **50** | |
| **Day 7** | ≤4.0 | 0 | 0 | 0 | 0 | 0 | r=0.729, p<0.05 |
| | 4.1-5.5 | 25 | 0 | 0 | 0 | 25 | |
| | 5.6-7.0 | 11 | 9 | 2 | 0 | 22 | |
| | >7.0 | 0 | 0 | 0 | 1 | 1 | |
| | **total** | **36** | **9** | **2** | **1** | **48** | |
| **Day 30** | ≤4.0 | 7 | 0 | 0 | 0 | 7 | r=0.373, p<0.05 |
| | 4.1-5.5 | 29 | 1 | 0 | 0 | 30 | |
| | 5.6-7.0 | 3 | 3 | 0 | 0 | 6 | |
| | >7.0 | 0 | 0 | 0 | 0 | 0 | |
| | **Total** | **39** | **4** | **0** | **0** | **43** | |

## Discussion

Present study was conducted in 50 patients of acute MI, who presented to hospital within 24 hrs of onset of symptoms. Fifty age and sex matched healthy controls were also evaluated for comparison.

**Distribution of patients of Acute MI according to age:** The mean age of patients of MI was 57.8 ± 11.5 years with range of 40-85 years (Table 1). Mean age

was compared and p value was >0.05 which shows that there was no significant difference between age of patients & controls. Mean SUA level was 7.05 ± 1.06 in age group(gp) <50 years, 6.60±1.04 in age gp 50-60 years, and 7.25 ± 2.04 in age gp >60 years. The p value for SUA levels in patient of different age is 0.382 showed that SUA levels are similar in different age gp. The study conducted by Jacobs [18] concluded similar results. But in contrast, Sokhanvar [19] et al, reported that there is increase in SUA levels as the age advances.

**Sex wise distribution of patients and serum uric acid levels:** Sex wise distribution of patients and SUA levels on day 0 showed that there is no statistical difference (p = 0.059) in SUA levels between male and female patients of AMI (table 2). Sokhanvar et al, [19] and Nadkar et al [16] also concluded in their studies that there is no difference in SUA levels in male and female patients of MI. This is in concordance with the present study. But the study conducted by Jacobs [18] noted SUA ranged from 3.5 – 12.4 mg/dl (mean 6.6) for males and 3.0 – 14.0 mg/dl (mean 7.2) for females and concluded that in patients of MI females have higher levels of SUA as compared to males. In contrast study done in Japan by Kojima [15] et al, it was observed that males had higher SUA levels as compared to females.

**Serum uric acid levels in patients and controls & killip class:** SUA in 50 patients of MI was compared with 50 age and sex matched healthy controls (Table 3). On day 0, SUA levels ranged from 5.4-14.4 mg/dl in patients with mean value of 6.92± 1.44 mg/dl and it ranged from 3.8-7.2 mg/dl in controls with mean value of 5.09± 0.86 mg/dl. There was a statistically significant higher level of SUA in patients of AMI on day of admission as compared to controls ($p < 0.05$). On day 3, 7, & 30 the mean SUA levels were 6.166±1.264, 5.636±1.19, 5.02±0.684 respectively. SUA ranged from 3.8- 7.2 mg/dl in controls with mean value of 5.098± 0.864 mg/dl. SUA gradually decreased with time in patients of MI & on day 30 levels were comparable to that in control group i.e. the levels come to normal at day 30. Hence, patients had higher SUA level on day 0 probably because of AMI. Similar finding was observed in a study by Kojima [15] et al with 1124 patients who presented with acute MI within 48 hrs of onset of symptoms. The study conducted by Nadkar [16] et al, also concluded that SUA levels are higher in patients with acute MI as compared to healthy sex and age matched controls.SUA levels are correlated with Killip class (table 6). Coefficient of correlation r= 0.749 and p value <0.05 on day 0 shows linear positive correlation between Killip class and SUA. Similarly on day 3, 7 and 30 there is a positive correlation between Killip class and SUA ($p<0.05$). Also patients of Killip class III and IV had higher levels of uric acid as compared to patients of class I and II. Out of 50 patients, four patients expired during 30 day follow up & all of them were in Killip class IV with >7mg/dl SUA at the time of admission. SUA levels are positively correlated to killip class i.e. higher the SUA levels higher is Killip class i.e SUA correlate with severity of cardiac failure. This is consistent with studies of Cicoira[21] et al Anker[22] et al who reported association of SUA with severity of cardiac failure. Also Kojima[15] et al in Japan, (Japanese Acute Coronary Syndrome Study) observed a close correlation between SUA and Killip classification in patients of acute MI. It was observed that patients who developed short-term adverse events had high SUA concentrations & reported that SUA, Killip class, age, and peak creatine

phosphokinase level were significant predictors of mortality. It was also noted that patients with angiographically confirmed coronary artery disease with SUA levels in the upper quartile were five times more likely to die than those in the lowest quartile. Nadkar [16] et al studied 100 patients with acute MI and 50 sex and age matched healthy controls and found a significant correlation between SUA concentration and Killip classification in patients of acute MI. SUA level was measured on day 0, 3 & 7 of MI. It was observed that there is statistically significant higher level of SUA concentration in patients of MI on the day of admission as compared to controls & patients with history of MI in the past had higher SUA levels. They observed that on all the days SUA were higher in patients who were in higher Killip class & the five patients who died after 3 days of hospital stay had SUA level more than 7.0 gm/dL and all of them were Killip class IV. It was concluded that SUA levels are higher in patients of acute MI & correlated with Killip class & combination of Killip class and SUA level after acute MI is a good predictor of mortality after acute MI.

**Comparison of serum uric acid in hypertensive and non hypertensives:** Out of 50 patients of AMI, 28(56%) were hypertensive while 22 (44%) were previously non hypertensive. Mean value was compared (table 4) & the p value is 0.12 showing there is no significant relation between serum uric acid levels in patients who are known or found to be hypertensive and non hypertensives. The study conducted by Nadkar [16] et al also concluded similar results that in patients of acute MI there is no significant relation between SUA levels in hypertensive & non-hypertensive. But study by Kojima **[15]** et al, observed that SUA levels were higher in patients who were previously hypertensive or found to be hypertensive on admission.

**Comparison of serum uric acid in diabetic and non-diabetic:** Out of 50 patients of AMI, 19 patients were diabetic and 31 patients were non-diabetic. The mean value was compared (table 4) & the p value is 0.695 showing there is no statistical significant relation between SUA levels of patients of AMI in diabetic and non-diabetic group. Nadkar **[16]** et al also concluded similar results that in patients of AMI there is no significant relation between SUA levels in diabetic versus non-diabetic group. Tuomilheto **[20]** et al also concluded in their study that SUA levels are comparable in diabetic and non diabetic patients of acute MI.

**Comparison of Serum uric acid levels with Lipid levels in patients of MI:** Out of 50 patients, 32 patients had LDL cholesterol below 100 mg/dl and 18 patients had LDL cholesterol between 100-200 mg/dl. The mean value was compared (table 5) & P value is 0.0259 showing significant positive correlation between uric acid and levels of LDL cholesterol in patients of acute MI i.e. higher the LDL cholesterol higher is the serum uric acid levels. Considering HDL cholesterol, out of total 50 patients, 5 patients had HDL cholesterol below 30 mg/dl and 40 patients had HDL cholesterol between 30-50 mg/dl and 5 patients had HDL cholesterol more than 50 mg/dl. The data is analysed using analysis of variance (ANOVA) and P value is <0.05 showing significant negative correlation between uric acid levels and levels of HDL cholesterol in patients of acute MI i.e. higher the HDL cholesterol lower the serum uric acid levels. Triglyceride (TG) were below 150 mg/dl in 24 patients, between 150- 250 mg/dl in 21 patients & >250 mg/dl in 5 patients. The

data is analysed using analysis of variance (ANOVA) and P value is 0= 0.200 showing no significant relation between SUA and triglyceride levels in patients of acute MI. Hence present study showed that in patients of MI, SUA levels are raised in patients having low HDL-cholesterol, high LDL-cholesterol. But Wenli [23] et al reported that increased uric acid levels are associated with increased lipid profile, Lippi[24] et al reported higher TG levels in hyperuricemic males & females, Peng [25] et al reported higher LDL –C, TG, total cholesterol association with higher SUA levels & inverse relation with HDL- C.

SUA concentration is significantly correlated with killip class. Patients with higher SUA & higher killip class have higher chances of mortality & that this combination can be considered as indicator of bad prognosis.

**References**


1. WHO 2004 – World Health Report 2004 – Changing History
2. Mukherjee AK. Prediction of coronary heart disease using risk factor categories. J Indian Med Assoc 1995 Aug;93(8):312-5.
3. Rastogi T, Vaz M, Spiegelman D, Reddy K S, Bharathi A V, Stampfer M J. Physical activity and risk of coronary heart disease in India. Int. J Epidemiol 2004 Aug;33(4):759-67.
4. Gupta R. Escalating Coronary Heart Disease and Risk factors in south Asians. Indian heart journal. 2007;59(3):214-17.
5. Mallinson T. Myocardial Infarction-Focus on First Aid. Magazine 2010;15(15).
6. Yusuf S, Hawken S, Ounpuu S, Bautista L, Franzosi MG, Commerford P et al. INTERHEART Study Investigators, Obesity and the risk of myocardial infarction in 27,000 participants from 52 countries: a case-control study. Lancet 2005;366(9497):1640–9.
7. Jensen G, Nyboe J, Appleyard M, Schnohr P. Risk factors for acute myocardial infarction in Copenhagen, II: Smoking, alcohol intake, physical activity, obesity, oral contraception, diabetes, lipids, and blood pressure. Eur Heart J 1991;12(3):298–308.
8. Thygesen K, Alpert JS, White HD. Universal definition of myocardial infarction. Eur Heart J 2007;28(20): 2525–38.
9. Dobson A. Is raised serum uric acid a cause of cardiovascular disease or death? Lancet 1999;354:1578.
10. Agamah ES, Srinivasan SR, Webber LS, Berenson GS. Serum uric acid and its relation to cardiovascular disease risk factors in children and young adults from a biracial community: the Bogalusa Heart Study. J Lab Clin Med 1991;118:241–9.
11. Bonora E, Targher G, Zenere MB, Saggiani F, Cacciatori V, Tosi F, et al. Relationship of uric acid concentration to cardiovascular risk factors in young men. Role of obesity and central fat distribution. The Verona Young Men Atherosclerosis Risk Factors Study. Int J Obes Relat Metab Disord 1996; 20:975–80.
12. Freedman DS, Williamson DF, Gunter EW, Byers T. Relation of serum uric acid to mortality and ischemic heart disease. The NHANES I Epidemiologic Follow-up Study. Am J Epidemiol 1995;141(7):637-44.
13. Fang J, Alderman MH. Serum uric acid and cardiovascular mortality the NHANES I epidemiologic follow-up study, 1971–1992 National Health and Nutrition Examination Survey. JAMA. 2000;283(18):2404-2410.
14. Torun M, Yardim S, Simsek B, Burgaz S. Serum uric acid levels in

cardiovascular diseases. J Clin Pharm Ther 1998;23:25–9.
15. Kojima S, Sakamoto T, Ishihara M, Kimura K, Miyazaki S, Yamagishi M, et al. Prognostic usefulness of serum uric acid after acute myocardial infarction (Japanese Acute Coronary Syndrome Study). Am J Cardiol 2005;96:489-95.
16. Nadkar MY, Jain VI. Serum uric acid in acute myocardial infarction. J Assoc Physicians India. 2008;56:759-62.
17. Shetty, Shobha, A. Harish Rao, and Sampath Kumar AK. Serum Uric Acid as a prognostic biomarker & its correlation with Killip Class in Acute Myocardial Infarction. International Journal of Biomedical Research 2013; 4(7):312-16.
18. Jacobs D. Hyperuricemia and myocardial infarction. S Afr Med J 1972;46:367.
19. Sokhanvar S, Maleki A. Blood Uric Acid Levels According to Cardiovascular Disease Risk Factors in Patients with Myocardial Infarction. Iranian Heart Journal 2007;8(1):43-45.
20. Tuomilheto J, Zimmet P, Evawolf. Plasma Uric acid level and its association with Diabetes Mellitus and some Biologic Parameters in a Biracial Population of FIJI. Am J Epidemiol 1988;127:321-36.
21. Cicoira M, Zanolla L, Rossi A, Golia G, Franceschini L, Brighetti G et al. Elevated Serum Uric acid levels are associated with diastolic dysfunction in patients with dilated cardiomyopathy. Am Heart J 2002;143:1107-11.
22. Anker SD, Doehner W, Rauchhaus M, Sharma R, Francis D, Knosalla C, et al. Uric acid and survivalin chronic heart failure: validation and application in metabolic, functional and haemodynamic staging. Circulation 2003;22:1991–97.
23. Wenli Lu, Song K, Wang Y, Zhang Q, Li W, Jiao H et al. Relationship between serum uric acid and metabolic syndrome: an analysis by structural equqtion modeling. Journal of clinical lipidology 2012;6:159-167.
24. Lippi G, Montagnana M, Luca Salvagno G, Targher G, Cesare Guidi G. Epidemiological association between uric acid concentration in plasma, lipoprotein(a), and the traditional lipid profile. Clin Cardiol 2010;33(2);76-80.
25. Peng TC, Wang CC, Kao TW, Chan JYH, Yang YH, Chang TW, et al. Relationship between hyperuricemia & lipid profiles in US adults. Biomed research international. doi 10.11.55/2015/1275 /127596.

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