"THE IMPORTANCE OF SERUM URIC ACID LEVELS AND KILLIP CLASSIFICATION IN PREDICTING PROGNOSIS OF ACUTE MYOCARDIAL INFARCTION."

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M. D.
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Under the Guidance of

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LIST OF ABBREVIATIONS USED

ATP- Adenosine Triphosphate.

BNP- B-type Natriuretic Peptide.

CABG- Coronary Artery Bypass Graft.

CAG- Coronary Angiography.

CO- Cardiac Output.

cTn- Cardiac Troponin

CVD- Cardiovasular Disease.

ECG- Electrocardigram.

FBS- Fasting Blood Sugar.

HDL- High Density Lipoprotein.

HF- Heart Failure.

IFN gamma - Interferon gamma.

IHD- Ischemic Heart Disease.

IL-6- Interleukin-6.

LBBB- Left Bundle Branch Block.

LDL- Low Density Lipoprotein.

LV- Left Ventricle.

LVEDP- Left Ventricle End Diastolic Pressure.

LVEF- Left Ventricle Ejection Fraction.

MI- Myocardial Infarction.

NO- Nitric oxide.

Nos- Nitric oxide synthase.

NSTEMI - Non ST Segment Elevation Myocardial Infarction.

NYHA- New York Heart Association.

AMI- Acute Myocardial Infarction.

PCI- Percutaneous Coronary Intervention.

P^H- Potential of Hydroogen.

STEMI- ST Segment Elevation Myocardial Infarction.

SUA- Serum Uric Acid.

TNF-alpha- Tumour Necrosis Factor- alpha.

UA- Uric acid.

XDH- Xanthine Dehydrogenase.

XO- Xanthine oxidase.

ABSTRACT

Background and objectives:

There is evidence that high uric acid is a negative prognostic marker in patients with mild to severe heart failure. Furthermore there is a need to find a simple, less expensive but accurate marker that could be useful in rural areas where fibrinolytic treatment is still the first choice of acute reperfusion therapy. Our study tried to correlate between serum uric acid levels and killip classification in predicting prognosis in acute myocardial infarction patients.

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Methodology:

The present one year cross-sectional study was done in the Department of Medicine and Cardiology, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. A total of 100 acute myocardial infarction patients were included in the study. Patients were subjected to clinical examination, electrocardiogram and serum uric acid was measured on day 0, 3 and day 5. Serum uric acid levels and killip class of the patients were compared to assess the prognosis.

Results:

Majority of patients had abnormal uric acid on day 1.Patients with abnormal uric acid levels on all the 3 days were more in killip class III and IV as compared to class I and II. Among 18 patients who expired, 16 were in killip class III and IV (8 in each class).The mean serum uric acid levels of expired patients were raised on all the 3 days with maximum on day 1. Serum uric acid

level was positively correlated with serum triglyceride level. Hyperuricemia was associated with left ventricle dysfunction.

Conclusion and interpretation:

Serum uric acid levels are raised in acute myocardial infarction patient. There is a positive correlation between rising serum uric acid levels with higher killip class. Combination of killip class and serum uric acid level after acute myocardial infarction is a good predictor of mortality. Hyperuricemia is positively correlated with serum triglyceride level and left ventricle dysfunction.

Keywords:

Serum uric acid, acute myocardial infarction, killip class, Triglyceride, left ventricle dysfunction.

CONTENTS

LIST OF TABLES

LIST OF GRAPHS

LIST OF FIGURES

INTRODUCTION

Ischaemic heart disease is a condition in which there is an inadequate supply of blood and oxygen to a region of the myocardium, it occurs due to an imbalance between myocardial oxygen supply and demand. The most common cause of myocardial ischemia is atherosclerotic disease of coronary artery (or arteries) that is sufficient to cause a regional reduction in myocardial blood flow and inadequate perfusion of the myocardium supplied by the involved coronary artery.

IHD causes more deaths and disability and incurs greater economic costs than any other illness in the developed world. IHD is the most common, serious, life - threatening illness in the United States, where 13 million persons have IHD, more than 6 million have angina pectoris, and more than 7 million have sustained a myocardial infarction. Genetic factors, an energy-rich and high-fat diet, smoking, and a sedentary lifestyle are associated with the emergence of IHD.

In the United States and Western Europe, it is increasing among low-income groups, but primary prevention has delayed the disease to later in life in all social strata groups. Despite of these sobering statistics, it is worth noting that epidemiologic data show a decline in the rate of deaths due to IHD, about half due to prevention by risk factor modification and half of which is attributable to treatment.

Obesity, insulin resistance, and type 2 diabetes mellitus are increasing and are strong risk factors for IHD. With urbanization in countries with emerging economies and a growing middle class, elements of the energy-rich Western diet are being adopted. As a result, the prevalence of risk factors for IHD are increasing rapidly in those regions such that a majority of the global burden of IHD occurs there.

Population that appear to be particularly affected are men in South Asian countries, especially India and the Middle East. In light of the projection of large increase in IHD throughout the world, IHD is likely to become the most common cause of death worldwide by 2020.[1]

Cardiovascular diseases have been gaining importance in developing countries like India recently because of increased incidence of the disease. It is the first among top 5 causes of deaths in Indian population (rural vs. urban, economically backward vs. developed states, men vs. women and at all ages vs middle age) [2]

In 2000, there were an estimated 29.8 million people with IHD in India, out of a total estimated population of 1.03 billion, or a nearly 3% overall prevalence[3,4] .

According to World Bank estimates, CVD had a 31% share in the total burden of disease in 2001 [5].

In 2003, the prevalence was estimated to be 8-10% in urban areas and 3-4% in rural areas according to population based cross sectional surveys[6, 7].

Left ventricular dysfunction is the single and the most important predictor of mortality following STEMI.[8, 9]

In 1967, Killip and Kimball [10] proposed a prognostic classification scheme on the basis of the presence and severity of rales detected in patients presenting with STEMI.

Killip classification is a strong independent predictor of all-cause mortality in patients with non–ST-elevation acute coronary syndromes.[11]

There are some markers indicating unfavourable prognosis in patients with acute myocardial infarction. Serum uric acid is one of the markers that has been

evaluated in research. The role of uric acid as a prognostic marker or as a risk factor for cardiovascular disease is still controversial. Uric acid is produced by the activity of the enzyme xanthine oxidase and is the final product of purine metabolism.[12]

Xanthine oxidase produces oxidants in this process that may have an important role in cardiovascular disease. Some studies have suggested that uric acid can cause inflammation and intracellular stress leading to endothelial injury and enhancement of vasoconstrictor effects .[13]

According to the Japanese Acute Coronary Syndrome Study[14], there was a strong correlation between serum uric acid concentration and Killip classification in patients of acute myocardial infarction. Patients who had high uric acid concentrations developed short-term adverse events.

Though various cardiac biomarkers are available, occasionally there may be a need to find a simple & reliable prognostic marker in developing countries where fibrinolytic therapy is still the primary mode of reperfusion therapy,that may be due to nonavailability of percutaneous coronary intervention or due to financial constraints.

We undertook this study to note the levels of serum uric acid in Acute Myocardial Infarction, to correlate the serum uric acid levels with Killip classification in predicting the prognosis in these patients.

OBJECTIVE OF THE STUDY

To study the importance of Serum Uric Acid and Killip classification in Predicting Prognosis in Acute myocardial infarction patients.

REVIEW OF LITERATURE

Myocardial infarction is the leading cause of mortality and morbidity in present days. Myocardial infarction is a common presentation of ischemic heart disease(IHD).Ischemic heart disease is a condition in which there is an inadequate supply of oxygen and blood to a portion of the myocardium. The most common cause of myocardial ischemia is atherosclerotic disease of an epicardial coronary artery which is enough to cause a regional reduction in blood flow to the myocardium and inadequate perfusion of the myocardium supplied by the involved coronary artery.

By reducing the lumen of the coronary arteries, atherosclerosis limits appropriate increases in perfusion when the demand for flow is augmented, as occurs during exertion or excitement. When the luminal reduction is severe, myocardial perfusion in the basal state is reduced.

Epicardial coronary arteries are the major site of atherosclerotic disease. The major risk factors for atherosclerosis includes low plasma high-density lipoprotein (HDL),high levels of plasma low-density lipoprotein (LDL),hypertension, diabetes mellitus and cigarette smoking, that disturbs the normal functions of the vascular endothelium. These functions include maintaining an antithrombotic surface, local control of vascular tone, and control of adhesion of inflammatory cells and diapedesis. The loss of these defense mechanism leads to luminal thrombus formation, inappropriate constriction and abnormal interactions between blood cells, especially platelets and monocytes, and the activated vascular endothelium. Functional changes in the vascular milieu result in the subintimal collections of fat, fibroblasts, smooth muscle cells, and intercellular matrix that define the atherosclerotic plaque. This process develops at an irregular rates in different segments of the epicardial coronary

tree and eventually leads to segmental reductions in cross-sectional area, i.e., plaque formation.[15]

The mechanism of plaque rupture is a complex process. Lipid related inflammation within the plaque result in degradation and weakening of the plaque tissue. On the other hand, collagen synthesis and proliferation of smooth muscle cell exert reparative and stabilizing effects. These biologic features of the plaque determine to a large extent whether or not a plaque will be vulnerable, and set the stage for rupture and a trigger induce a rupture event. New studies have emerged on a possible role for inflammation and also repair in local arterial wall remodeling in terms of dilation and shrinkage, with consequences for the geometry of the entire vessel [16]

The inflammatory process within plaques most likely represents a protective phenomenon which act to eliminate accumulations of noxious agents from the arterial wall. For example, phagocytosis of lipids is basically a protective mechanism, but excess uptake and death of foam cells can result in expansion of the soft atheroma. Another example is provided by the T-cell cytokine IFN- gamma, which may inhibit growth of native plaques and restenosis lesions by inhibiting smooth muscle cells proliferation and also collagen synthesis [17]. However, in the advanced lipid plaque which requires the support of an intact fibrous cap, the same effect appears to be dangerous. Therefore, although protective, the inflammatory process in these advanced plaques has a worse side-effect, can cause destabilization and plaque rupture. This leads to coronary thrombosis and myocardial infarction.

REVISED DEFINITION OF MYOCARDIAL INFARCTION [63]

The term acute MI should be used when there is evidence of myocardial necrosis in a clinical setting consistent with acute myocardial ischemia. Under these conditions any of the following criteria meet the diagnosis for MI:

- Detection of a rise and/or fall in cardiac biomarker values (preferably cTn), with at least one value above the 99th percentile of the URL and with at least one of the following:

- Symptoms of ischemia
- New or presumed new significant ST-segment T wave (ST-T) changes or new LBBB
- Development of pathologic Q waves on the ECG
- Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.
- Identification of an intracoronary thrombus by angiography or autopsy

- Cardiac death with symptoms suggestive of myocardial ischemia and presumed new ischemic changes on the ECG or new LBBB but death occurred before cardiac biomarkers were determined or before cardiac biomarker values would be increased.

- PCI-related MI is arbitrarily defined by elevation of cTn values (to >5 Å~ the 99th percentile of the URL) in patients with normal baseline values (≤99th percentile of the URL) or a rise in cTn values >20% if the baseline values are elevated and are stable or falling.In addition, either (1) symptoms suggestive of myocardial ischemia, (2) new ischemic changes on the ECG,

(3) angiographic findings consistent with a procedural complication, or (4) imaging demonstration of new loss of viable myocardium or new regional wall motion abnormality is required.

- Stent thrombosis associated with MI when detected by coronary angiography or autopsy in the setting of myocardial ischemia and with a rise and/or fall in cardiac biomarker values and at least one value higher than the 99th percentile of the URL.
- CABG-related MI is arbitrarily defined by elevation of cardiac biomarker values (to >10 Å~ the 99th percentile of the URL) in patients with normal baseline cTn values (≤99th percentile of the URL). In addition, either (1) new pathologic Q waves or new LBBB, (2) angiographically documented new graft or new native coronary artery occlusion, or (3) imaging evidence of new loss of viable myocardium or new regional wall motion abnormality is required.

Clinically, Myocardial infarction can be further sub classified into a ST elevation MI (STEMI) versus a non-ST elevation MI (non-STEMI) based on ECG changes.

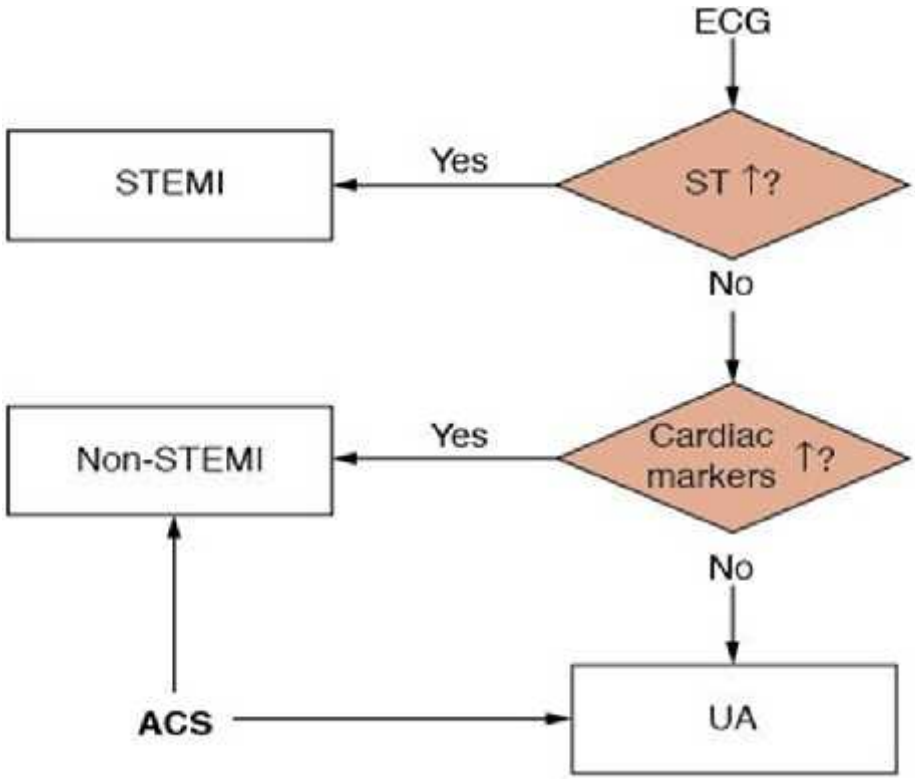


Figure 1 : Clinical Classification of Myocardial Infarction.

A 2007 consensus document classifies myocardial infarction into five main types:[63]

Type 1 – Spontaneous myocardial infarction related to ischemia due to a primary Coronary event such as plaque erosion and/or rupture , fissuring, or dissection.

Type 2 – Myocardial infarction secondary to ischemia due to either increased oxygen demand or decreased supply, e.g. coronary artery spasm, coronary embolism, anemia, arrhythmias, hypertension, or hypotension.

Type 3 – Sudden unexpected cardiac death, including cardiac arrest, often with symptoms suggestive of myocardial ischemia, accompanied by presumably new ST elevation, or new LBBB, or evidence of fresh thrombus in a coronary artery by

angiography and/or at autopsy, but death occurring before blood samples could be Obtained, or at a time before the appearance of cardiac biomarkers in the blood.

Type 4 – Associated with coronary angioplasty or stents.

Type 4a – Myocardial infarction associated with PCI

Type 4b – Myocardial infarction associated with stent thrombosis as documented by angiography or at autopsy.

Type 5 – Myocardial infarction associated with CABG

The most common major mechanical complications of Acute STEMI include cardiogenic shock, right ventricular infarction, acute mitral regurgitation, ventricular septum rupture, and free wall rupture.

The electrical complications include Bradyarrhythmias (viz. sinus bradycardia, second-degree atrioventricular block, complete heart block, bundle-branch block), Tachyarrhythmias (viz. sinus tachycardia, atrial fibrillation, accelerated idioventricular rhythm, ventricular tachycardia, ventricular fibrillation).

Mechanical complications should be suspected in any patient with STEMI in whom circulatory collapse occurs. Immediate hemodynamic, angiographic, and echocardiographic evaluations are necessary in patients with cardiogenic shock. It is important to exclude mechanical complications because primary therapy of such lesions usually requires immediate operative treatment, with intervening support of the circulation by intra-aortic balloon counterpulsation.

Cardiogenic shock[18] is the most severe clinical expression of left ventricular failure and is associated with extensive damage to the left ventricular myocardium in more than 80% of STEMI patients in whom it occurs; the

remainder have a mechanical defect such as ventricular septal or papillary muscle rupture or predominant right ventricular infarction. In the past, cardiogenic shock was reported to occur in up to 20% of patients with STEMI, but estimates from recent large trials and observational databases report an incidence in the range of 5% to 8%.

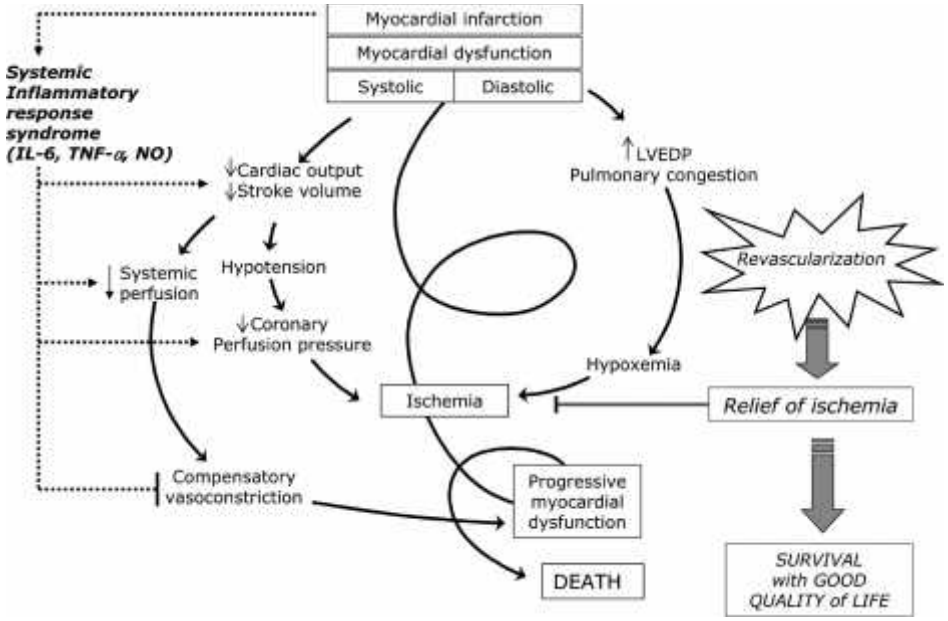


Figure 2. Current concept in the pathophysiology of Cardiogenic Shock

Myocardial injury causes systolic and diastolic dysfunction. A decrease in CO leads to a decrease in systemic and coronary perfusion. This exacerbates ischemia and causes cell death in the infarct border zone and the remote zone of myocardium. Inadequate systemic perfusion triggers reflex vasoconstriction, which is usually insufficient. Systemic inflammation may play a role in limiting the peripheral vascular compensatory response and may contribute to myocardial dysfunction. Whether inflammation plays a causal role or is only an epiphenomenon remains unclear. Revascularization leads to relief of ischemia. It has not been possible to demonstrate an increase in CO or LVEF as the mechanism of benefit of revascularization;

however, revascularization does significantly increase the likelihood of survival with good quality of life.[18]

Uric Acid in Disease

Uric acid is the final breakdown product of purine degradation in humans. It is a weak acid with pK_as of 5.75 and 10.3.Urates, the ionized forms of uric acid, predominate in plasma extracellular fluid and synovial fluid, with 98% existing as monosodium urate at pH 7.4. Although purine nucleotides are synthesized and degraded in all tissues, urate is produced only in tissues that contain xanthine oxidase, primarily the liver and small intestine.

Urate production varies with the purine content of the diet and the rates of purine biosynthesis, degradation, and salvage. Normally, two-thirds to three-fourths of urate is excreted by the kidneys, and most of the remainder is eliminated through the intestines.

Hyperuricemia

Hyperuricemia can result from increased production or decreased excretion of uric acid or from a combination of the two processes. Sustained hyperuricemia predisposes some individuals to develop clinical manifestations including gouty arthritis, urolithiasis, and renal dysfunction (uric acid nephropathy).

Hyperuricemia is defined as a plasma (or serum) urate concentration >405 mol/L (6.8 mg/dL). The risk of developing gouty arthritis or urolithiasis increases with higher urate levels and escalates in proportion to the degree of elevation. Hyperuricemia is present in between 2 and 13.2% of ambulatory adults and is even more frequent in hospitalized individuals.

Hyperuricemia and Metabolic Syndrome

Metabolic syndrome is characterized by abdominal obesity with visceral adiposity, impaired glucose tolerance due to insulin resistance with hyperinsulinemia, hypertriglyceridemia, increased low density lipoprotein cholesterol, decreased high-density lipoprotein cholesterol, and hyperuricemia.

Hyperinsulinemia reduces the renal excretion of uric acid and sodium. Not surprisingly, hyperuricemia resulting from euglycemic hyperinsulinemia may precede the onset of type 2 diabetes, hypertension, coronary artery disease, and gout in individuals with metabolic syndrome.[19]

Elevated levels of uric acid (UA) predict mortality and the need for heart transplantation in patients with congestive heart failure.[20]

HF Potential Mechanisms for Increased Uric Acid in Heart Failure

UA is a metabolic byproduct of purine metabolism. Serum UA may increase in the failing circulation because of increased generation, decreased excretion, or a combination of the 2 factors. There are several possible contributors to increased UA production in HF, including increased abundance and activity of XO, [65] increased conversion of xanthine dehydrogenase (XDH) to XO, [66] or increased XO substrate resulting from enhanced ATP breakdown to adenosine and hypoxanthine. As UA is excreted primarily by the kidney, decreased renal perfusion could lead to increased UA levels.

To the extent that HF leads to tissue ischemia (in advanced HF) and a rise in serum lactate, renal UA excretion can be further impaired as lactate competes with urate via an organic anion exchanger in the proximal tubule.

Pathophysiological Role of the Xanthine Oxidase Pathway in Heart Failure

There is increasing evidence that strongly supports a direct pathophysiological role for the metabolic pathway leading to UA production in the failing circulation.[21, 22] In this regard, the two terminal steps in urate production are catalyzed by XO, which also produces a molecule of superoxide for each reaction . XO is the product of the xanthine oxidoreductase gene that encodes XDH, an 150 Kda protein, which functions as a homodimer. XDH is converted to XO by proteolytic cleavage or sulfhydryl modification.[23]

The elevation in serum UA may reflect increased XO pathway activity and in turn the generation of superoxide and resultant oxidative stress via the XO system.[24] XO is upregulated within the heart in both experimental [25, 26] and human [27] heart failure. Much had previously been made of the difficulty in identifying XO within the hearts of certain mammalian species, including humans.[28] Nevertheless, it is clear that XO, which is produced in highest abundance in the liver and gut, may circulate in the blood and adhere to endothelium in distant sites.[29] Moreover, XO is expressed in cardiac myocytes, as shown by immunohistochemistry and may participate in intracrine signaling.[30]

From a functional standpoint, XO activity participates in both mechanoenergetic uncoupling and vascular dysfunction in the failing circulation. Mechanoenergetic uncoupling is the process whereby cardiac energy consumption remains the same or increases while cardiac work falls dramatically, and is increasingly being perceived as a potential key lesion in the failing heart. Inhibition of XO with allopurinol restores depressed myocardial energetics toward normal, and this effect can be mimicked by the antioxidant ascorbate.[30]

Furthermore, several recent studies have demonstrated that XO inhibition improves endothelial dysfunction in patients with congestive heart failure in association with reduction in circulating markers of oxidative stress, [31, 32] thereby providing evidence that XO inhibition reduces oxidant

Pathophysiological Role of Uric Acid in Heart Failure

Beyond XO activity, recent experimental studies suggest that UA itself may have a role in cardiovascular and renal pathophysiology. This might seem surprising, as UA can function as an antioxidant, both by itself and by promoting superoxide dismutase activity[33,34] and might therefore be considered potentially protective. However, UA potently stimulates vascular smooth muscle cell proliferation in vitro, an effect mediated by stimulation of mitogen-activated protein kinases, cyclooxygenase-2, and platelet-derived growth factor.[35, 36] Furthermore, rats with mild experimentally induced hyperuricemia develop intrarenal vascular disease with increased renin expression, systemic and glomerular hypertension, and renal injury in the absence of intrarenal crystal deposition.[37,38] These hemodynamic and structural changes can be prevented if UA elevation is prevented by Allopurinol generation.

Interaction of Xanthine Oxidase and Uric Acid With Nitric Oxide Pathways

Both XO activity and UA may also affect cardiac and renal nitric oxide signaling, [30, 39] which exerts key cardiac and vascular effects. The impact of XO inhibition to restore depressed myocardial energetics requires intact NO pathway activity.UA may also impair NO production directly, as suggested by the finding that UA infusion into forearm veins of humans attenuates acetylcholine-stimulated vasodilation.[40]

Likewise, the hypertension associated with hyperuricemia in rats is associated with reduced expression of macula densa neuronal nitric oxide synthase (NOS) and can be partially reversed by the NOS substrate L-arginine.[39]

This finding has interesting implications for cardiac function, as neuronal NOS plays a key role in modulating cardiac excitation contraction coupling by facilitating sarcoplasmic reticulum calcium release.[41]

Clinical Utility of Uric Acid Measurements.

From a clinical perspective, there raises the issue of whether serum UA levels should be routinely measured in HF patients. Indeed this should be more likely, and one which will require evaluation in the context of measurement of brain natriuretic peptide (BNP), a serum marker that also possesses prognostic and diagnostic value in HF patients.[42] Much in the same way as BNP has been evaluated, it will be of great value to assess whether UA levels change in response to HF therapy in a manner that predicts clinical outcome.[43]

UA levels are ready for clinical use, the observation that UA levels possess prognostic information adds an extremely intriguing finding to mounting evidence that XO and UA play pathophysiological roles in HF and its precursor, Hypertension.

Indeed, the amassing data have led to the planning of a clinical trial entitled a Phase II-III Prospective, Randomized, Double-Blind, Placebo-Controlled Efficacy and Safety Study of Oxypurinol added to Standard Therapy in Patients with NYHA Class III-IV Congestive Heart Failure (OPT-CHF), initiated in 2003,

which will test clinical outcomes using a composite endpoint comprising measures of heart failure morbidity, exercise capacity, and mortality.

The findings of Anker and colleagues,[44] therefore, not only bring to light a potentially new diagnostic test but also provide a novel line of evidence that the XO pathway and/or UA itself may be of pathophysiological importance in heart failure progression. The available evidence has established a link between Hyperuricemia and cardiovascular disease and this may be causal**.** Without waiting for the resolution of causality arguments, one can start using serum uric acid concentration as an inexpensive cardiovascular risk marker.

Uric Acid as a Marker of Subclinical Ischemia

Adenosine is synthesized and released by cardiac and vascular myocytes. Binding to specific adenosine receptors causes relaxation of vascular smooth muscle and arteriolar vasodilatation.[44] Adenosine makes a small contribution to normal resting vascular tone, since competitive antagonism at the adenosine receptor by methylxanthines, such as theophylline, reduce blood flow response to ischemia in the forearm vascular bed.[45] Under conditions of hypoxia and tissue ischemia, vascular adenosine synthesis and release are upregulated, causing significantly increased circulating concentrations.[46] Cardiac and visceral ischemia promote generation of adenosine, which may serve as an important regulatory mechanism for restoring blood flow and limiting the ischaemia.[47]

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.[48]

Xanthine oxidase activity [49] and uric acid synthesis [50] are increased in vivo under ischemic conditions, and therefore elevated serum uric acid may act as a marker of underlying tissue ischemia. In the human coronary circulation, hypoxia, caused by transient coronary artery occlusion, leads to an increase in the local circulating concentration of uric acid.[51] Study of tourniquet-induced lower limb exsanguination in patients undergoing surgery shows a five-fold increase in systemic vascular xanthine oxidase activity during reperfusion, and a significant elevation of serum uric acid, which persists for at least two hours.[52]

These findings are also consistent with the inverse relation between baseline serum uric acid concentration and maximal lower limb blood flow in patient with cardiac failure, where higher concentration could predict subclinical ischaemia.[53]

In conclusion therefore, elevated serum uric acid may be a marker of local or systemic ischaemia and provide one possible explaination for a non-causal associative link between hyperuricaemia and cardiovascular disease.

Bickel C et al reported that one mg/dl increase in serum acid levels was associated with a 26% increase in mortality.[54]

Yıldız et al [55] and Nihat Kalay et al [56] in their separate studies found that serum uric acid levels were higher in patients with Slow Coronary Flow compared to controls. Serum uric acid has also been suggested as a risk factor for occurrence and a predictor of poorer outcomes in acute stroke.[57, 58, 59]

Siniša Car [60] et al. in their study found that higher serum uric acid determined on admission was associated with higher in-hospital mortality and thirty-day mortality and poorer long-term survival after AMI.

According to the Japanese Acute Coronary Syndrome Study[14], there was a close correlation between serum uric acid concentration and Killip classification in patients of acute myocardial infarction. Patients who developed short-term adverse events had high uric acid concentrations.

The Losartan Intervention For Endpoint reduction in hypertension (LIFE) study demonstrated that lowering serum uric acid concentrations by losartan was associated with a beneficial effect on cardiovascular outcome.[61] The uric acid lowering effect of atorvastatin may have contributed to the decrease in cardiovascular mortality in the Greek Atorvastatin and Coronary Heart Disease Evaluation (GREACE) study.[62] Therefore, any drug interventions, such as therapy to decrease serum uric acid level in addition to coronary reperfusion, may have a favourable effect on mortality in patients who have Acute Myocardial Infarction.

The Killip classification.[10]

This is a system used in individuals with an acute myocardial infarction and heart failure, in order to risk stratify them. Individuals with a low Killip class are less likely to die within the first 30 days after their myocardial infarction than individuals with a high Killip class. Patients were ranked by Killip class in the following way:

Killip class I : Includes individuals with no clinical signs of heart failure.

Killip class II : Includes individuals with rales or crackles in the lungs, an S3, and elevated jugular venous pressure.

Killip class III: Describes individuals with frank acute pulmonary edema.

Killip class IV: Describes individuals in cardiogenic shock or hypotension (measured as systolic blood pressure lower than 90 mmHg), and evidence of peripheral vasoconstriction (oliguria,cyanosis or sweating).

The Killip classification system and mortality rate :

Killip class I: .Mortality rate was found to be at 6%.

Killip class II: Mortality rate was found to be at 17%.

Killip class III:Mortality rate was found to be at 38%.

Killip class IV:Mortality rate 67%

Cardiovascular Conditions and Risk Factors Associated with Elevated Uric Acid[64]

Hypertension and prehypertension, obstructive sleep apnea, renal disease(including reduced glomerular filtration rate and microalbuminuria), vascular disease (carotid, peripheral, coronary artery), stroke metabolic syndrome (including abdominal obesity, hypertriglyceridemia, low level of high-density lipoprotein cholesterol, insulin resistance, impaired glucose tolerance, elevated leptin level), and vascular dementia, preeclampsia, inflammation markers (C-reactive protein, plasminogen activator inhibitor type 1, soluble intercellular adhesion molecule type 1), endothelial dysfunction, oxidative stress, sex and race (postmenopausal women, blacks), and demographic (movement from rural to urban communities, westernization, immigration to western cultures) are the conditions and risk factors associated with elevated uric acid.

Our study was done to note the levels of serum uric acid in acute myocardial infarction, to correlate serum uric acid levels with Killip classification and to note any

relationship between serum uric acid level and mortality following acute myocardial infarction, to study the relation between serum uric acid and cardiac troponin in acute myocardial infarction.

In the present study, we found a close relation between serum uric acid concentrations and Killip classification suggestive of left ventricular failure. High uric acid concentrations on admission were strongly associated with adverse clinical outcome in patients who had acute myocardial infarction.

METHODOLOGY

The present study was conducted in the Department of Medicine and Cardiology, KLES Dr. Prabhakar Kore Hospital and Medical Research centre, belgaum from January 2014 to December 2014.

Study Design:

The study design was a one year Cross-Sectional study.

Study Period:

The present study was carried out from January 2014 to December 2014.

Source of Data:

All patients admitted with acute myocardial infarction within 24 hrs of onset of symptoms in Department of Cardiology & Medicine.

Sample size:

A total of 100 patient with acute myocardial infarction were included in the study

Sampling Procedure:

Based on this formula a sample size of 100 patients was considered.

Sample Size Calculation: $z\alpha^2pqd^2$

$$n = 4pq/d^2$$

Where, zα (constant),

p -sensitivity (60)-as obtained from previous studies, q-(100-p),

d -absolute error (10)

P=60%, q=40%, d=10

Sample size= 100

Inclusion Criteria:

- Patients more than 18 years of age, ECG findings and biochemical markers suggestive of acute myocardial infarction.
- Both ST segment elevation myocardial infarction (STEMI) and Non-ST segment elevation myocardial infarction (NSTEMI) will be included in the study.

Exclusion Criteria:

- Known causes of elevated uric acid level (chronic kidney disease, gout, hematological malignancy, hypothyroidism).
- Patients on drugs which increase serum uric acid e.g. salicylates (2gm/d, hydrochlorthiazide, pyrazinamide).
- Chronic alcoholics.

Ethical clearance

Prior to the beginning, the study was approved by the Institutional Ethics Committee, Jawaharlal Nehru Medical College, Belgaum.

Informed consent:

The patients who fulfilled the selection criteria were informed about the nature of study in detail and a written informed consent was obtained (Annexure-I).

Data collection:

Patients were interviewed and demographic data, history of present illness, other comorbid conditions, personal history were obtained. Further these patients underwent clinical examination followed by systemic examination. These findings were noted on a predesigned and pretested proforma (Annexure-II).

INVESTIGATIONS CARRIED OUT

- Complete blood count ,
- Serum uric acid on day 0, 3 & day 5.
- Fasting Lipid Profile,
- Urea and creatinine,
- 12-Lead ECG,
- CPK-MB, Trop-I.(as and when required)
- Other relevant investigations.

Statistical methods

The data obtained was coded and entered into the Microsoft Excel Spreadsheet (Annexure III). The categorical data was expressed in terms of rates, ratios and percentages and comparison was done using chi-square test. The continuous data was expressed as mean ± standard deviation and comparison was done using independent sample 't' test. A probability value ('P' value) of less than or equal to 0.05 was considered as statistically significant.

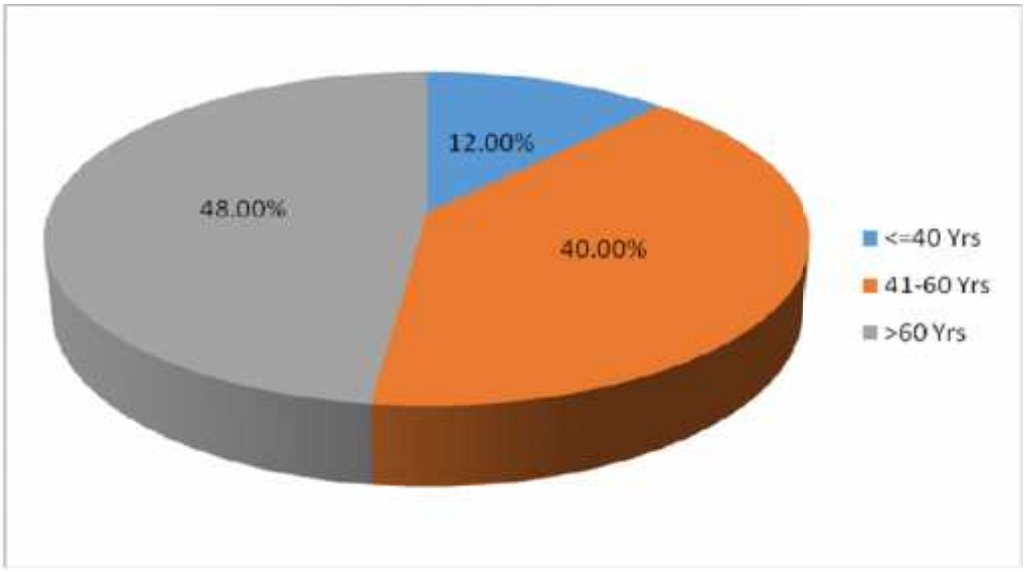
RESULTS

The present one year cross-sectional study titled "THE IMPORTANCE OF SERUM URIC ACID LEVELS AND KILLIP CLASSIFICATION IN PREDICTING PROGNOSIS OF ACUTE MYOCARDIAL INFARCTION" was carried out in Department of Medicine and Cardiology at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. A total of 100 acute Myocardial Infarction patients were studied.

The findings/observations and final results were tabulated as follows:

TABLE 1: AGE DISTRIBUTION

AGE GROUP (YEARS)	NUMBER	PERCENT
<=40	12	12.0
41-60	40	40.0
>60	48	48.0
Total	**100**	**100.0**

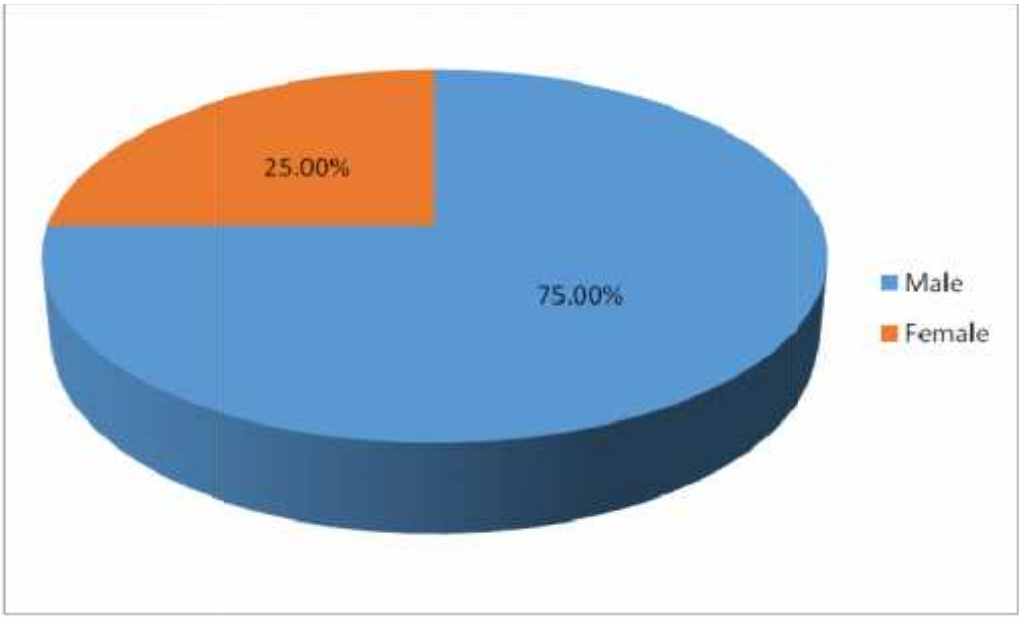


Graph 3: Age distribution

Patients age ranged from 28-88 years, maximum number of cases were in the age group of more than 60 years that is 48 patients (48%), between age group 41-60 years 40 patients (40%), and below 40 years 12 patients (12%).

TABLE 2: SEX DISTRIBUTION

SEX	NUMBER	PERCENT
MALE	75	75.0
FEMALE	25	25.0
Total	**100**	**100.0**



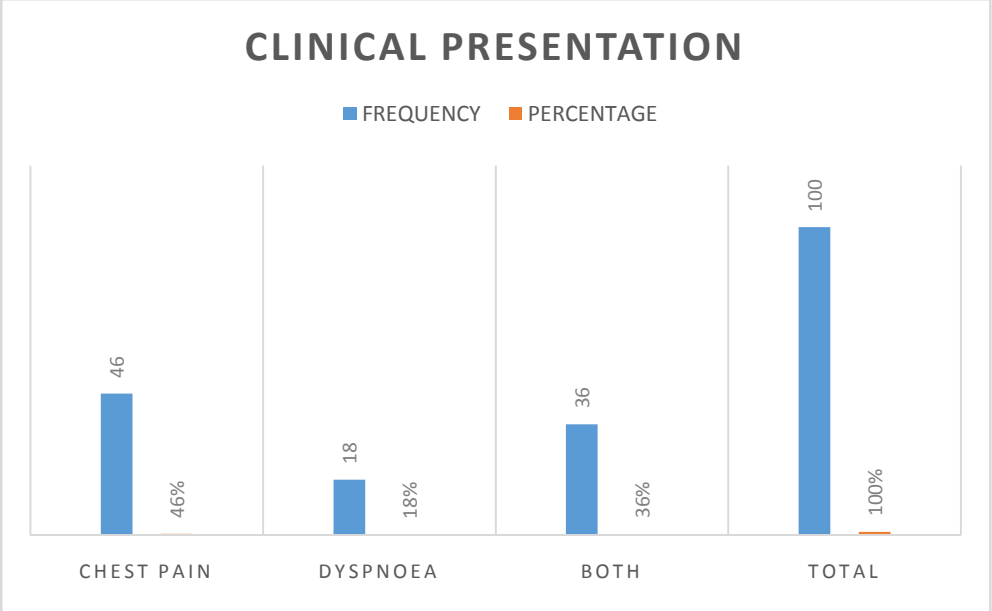
Graph 4: Gender distribution.

Out of 100 patients, 75 (75%) were males and 25 (25%) were females accounting a ratio of Male to Female 3:1.

INFERENCE: Male preponderance was seen.

TABLE 3: CLINICAL PRESENTATION

CLINICAL PRESENTATION	FREQUENCY	PERCENTAGE
CHEST PAIN	46	46%
DYSPNOEA	18	18%
BOTH	36	36%
TOTAL	**100**	**100%**

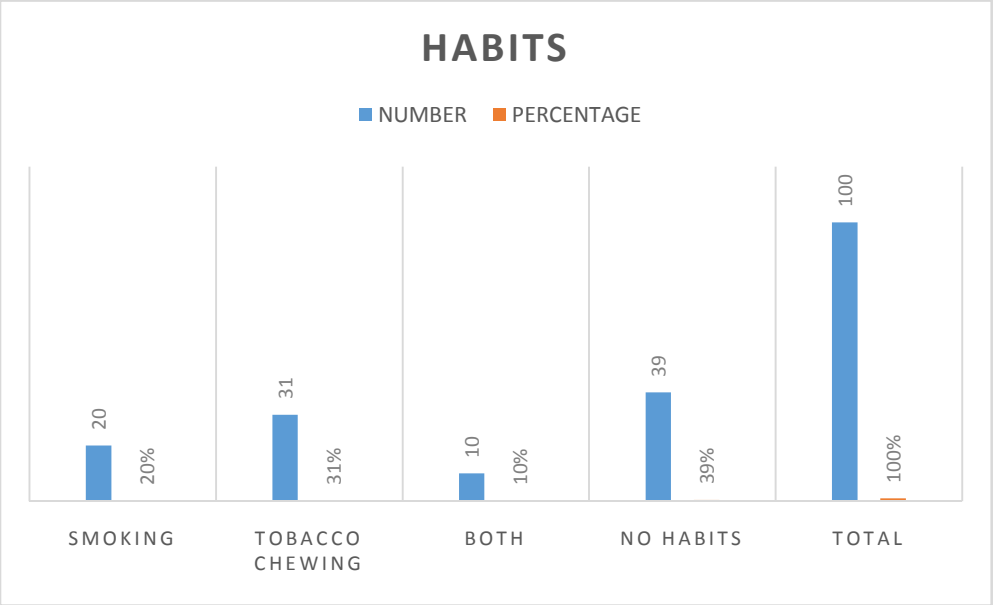


Graph 5: Clinical presentation.

In our study, majority of the patients that is 46 (46%) presented with chest pain, 18 patients (18%) presented with dyspnoea, followed by 36 patients (36%) who presented with combination of both symptoms (chest pain & dyspnoea).

TABLE 4: HABITS

HABITS	NUMBER	PERCENTAGE
TOBACCO CHEWING	31	31%
SMOKING	20	20%
BOTH	10	10%
NO HABITS	39	39%
TOTAL	**100**	**100%**

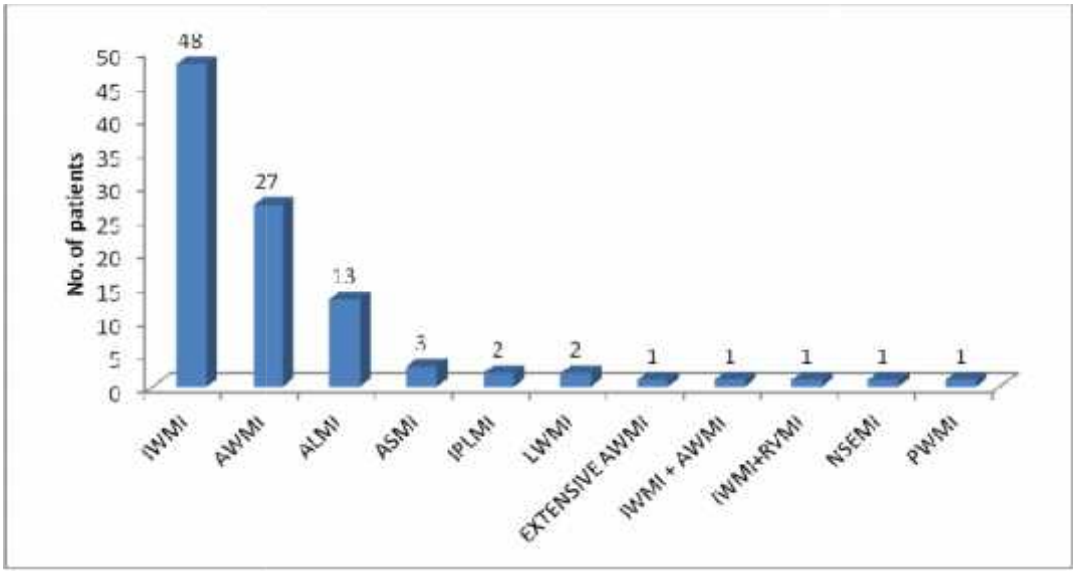


Graph 6: Habits.

Out of 100 patients 20 (20%) were smokers and 31 (31%) were tobacco chewer, and 10 (10%) were both smoker and tobacco chewers. 39 patients did not give history of any habits.

TABLE 5: ECG FINDINGS

MYOCARDIAL INFARCTION (MI)	NUMBER	PERCENTAGE
INFERIOR WALL MI	48	48.0
ANTERIOR WALL MI	27	27.0
ANTEROLATERAL MI	13	13.0
ANTEROSEPTAL MI	3	3.0
INFEROPOSTEROLATERAL MI	2	2.0
LATERAL WALL MI	2	2.0
AWMI + IWMI	1	1.0
INFERIOR WALL MI WITH RIGHT VENTRICLE EXTENSION	1	1.0
EXTENSIVE ANTERIOR WALL MI	1	1.0
NSTEMI	1	1.0
POSTERIOR WALL MI	1	1.0
Total	**100**	**100.0**

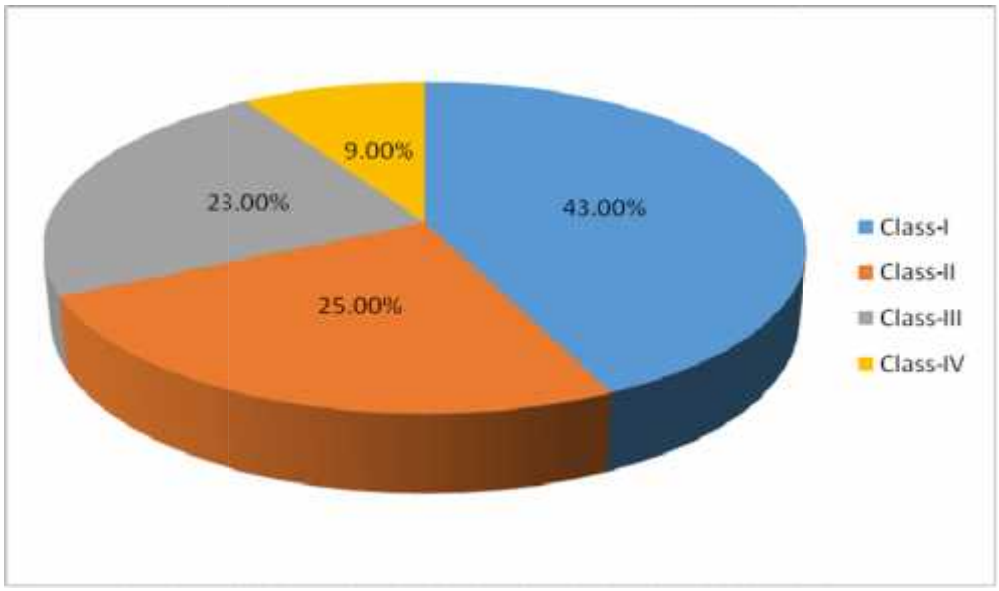


Graph 7: ECG findings.

In the present study, ECG tracing showed following pattern of MI -48 patients (48%) had INFERIOR WALL MI, 27 patients (27%) had ANTERIOR WALL MI, 13 patients (13%) had ANTEROLATERAL MI, 3 patients (3%) had ANTEROSEPTAL MI, 2 (2%) patients had INFEROPOSTEROLATERAL MI, 2 patients (2%) LATERAL WALL MI, and remaining 1 each in EXTENSIVE AWMI, AWMI+ IWMI, IWMI WITH RIGHT VENTRICAL EXTENSION, NON ST ELEVATION MI and POSTERIOR WALL MI.

TABLE 6: DISTRIBUTION OF PATIENTS KILLIP CLASS WISE

KILLIPS CLASS	NUMBER	PERCENT
I	43	43.0
II	25	25.0
III	23	23.0
IV	9	9.0
Total	**100**	**100.0**

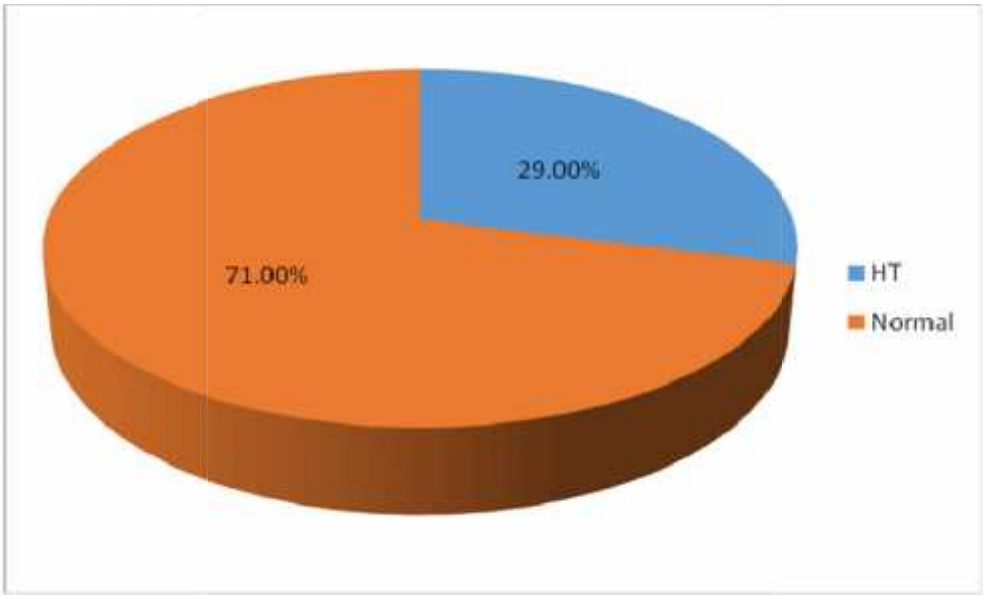


Graph 8 : Distribution of patients, killip class wise.

We observed that 43 patient (43%) were in killip class I, 25 patients (25%) in killip class II, 23 patients (23%) in killip class III and 9 patients (9%) in killip class IV

TABLE 7: HYPERTENSION DISTRIBUTION.

HYPERTENSION	NUMBER	PERCENT
NO	71	71.0
YES	29	29.0
Total	**100**	**100.0**

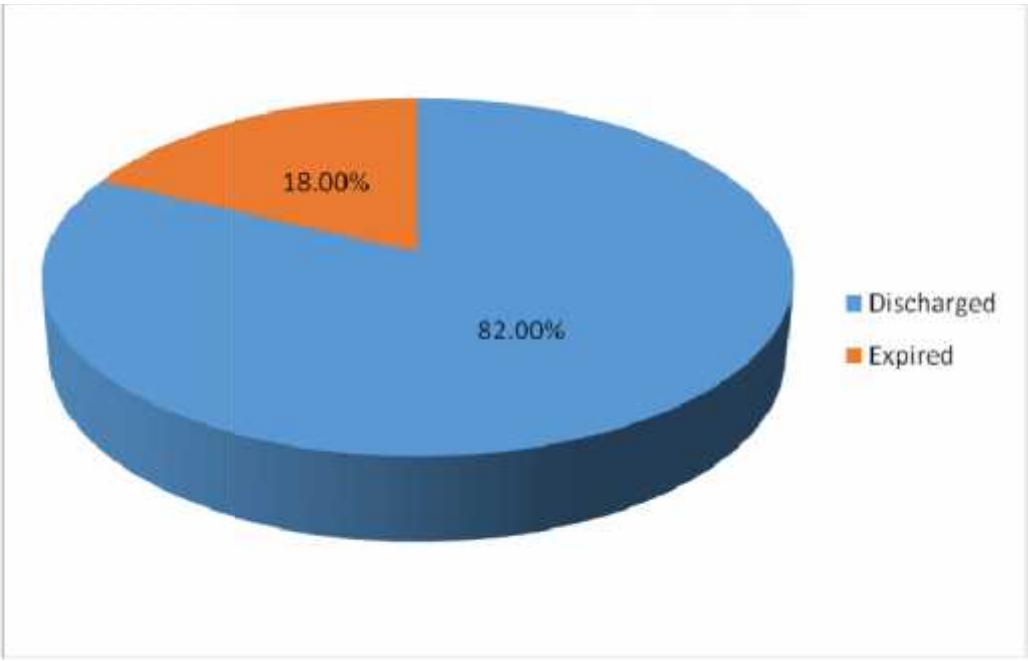


Graph 9: Hypertension distribution.

In the present study we observed that 71 (71%) patients did not have hypertension, remaining 29 patients (29%) had hypertension, who were on treatment. The normotensive to hypertensive ratio was 1:2.44.

TABLE 8: OUTCOME OF PATIENTS

OUTCOME	FREQUENCY	PERCENT
DISCHARGED	82	82.0
EXPIRED	18	18.0
TOTAL	**100**	**100.0**



Graph 10 : Outcome of patients.

In our present study 82 patient (82%) got discharged, remaining 18 patients (18%) expired.

TABLE 9: CORRELATION OF AGE WITH OUTCOME

AGE (YEARS)	OUTCOME		TOTAL
	DISCHARGE	EXPIRED	
<=40	10	2	12
	83.3%	16.7%	100.0%
41-60	31	9	40
	77.5%	22.5%	100.0%
>60	41	7	48
	85.4%	14.6%	100.0%
TOTAL	**82**	**18**	**100**
	82.0%	**18.0%**	**100.0%**

P-VALUE: 0.628

The present study shows that maximum number of deaths that is 9 (22.5%) were in age group 41- 60 years, 7 in age group more than 60 years and minimum number of deaths that is 2 (16.7%) in age group less than 40 years.

Among survivors 41 patients (85.4%) were in age group of more than 60 years, 31 patients (77.5%) between age group 41-60 years and 10 patients (83.3%) below 40 years of age. P -value statistically being insignificant.

TABLE 10: CORRELATION OF GENDER WITH OUTCOME

GENDER	OUTCOME		TOTAL
	DISCHARGE	EXPIRED	
FEMALE	19	6	25
	76.0%	24.0%	100.0%
MALE	63	12	75
	84.0%	16.0%	100.0%
TOTAL	**82**	**18**	**100**
	82.0%	**18.0%**	**100.0%**

P-VALUE 0.549

When outcome was compared to gender, 18 patients expired (12 males; 6 females), 82 patients got discharged (63 males; 19 females).

P -value being statistically insignificant.

TABLE 11: CORRELATION OF OUTCOME WITH KILLIP CLASS

KILLIP CLASS	OUTCOME		TOTAL
	DISCHARGE	EXPIRED	
I	43 100.0%	0 .0%	43 100.0%
II	23 92.0%	2 8.0%	25 100.0%
III	15 65.2%	8 34.8%	23 100.0%
IV	1 11.1%	8 88.9%	9 100.0%
TOTAL	82 82.0%	18 18.0%	100 100.0%

P-VALUE: <0.001

Out of 18 patients who expired, 16 were in killip class III and IV (8 in class III and another 8 in class IV) only 2 were in class II and none in class IV.

Also, 82 patients who survived 68 patients were in killip class I and II (43 in class I and 25 in class II) and remaining 23 patients in killip class III and only 9 in killip class IV. **This shows that as the killip class increases mortality increases. (p -value <0.001 being statistically significant).**

TABLE 12: CORREATION OF OUTCOME WITH PATTERN OF MYOCARDIAL INFARCTION

MYOCARDIAL INFARCTION		OUTCOME		
		DISCHARGE	EXPIRED	Total
ALMI		13 100.0%	0 .0%	13 100.0%
ASMI		3 100.0%	0 .0%	3 100.0%
AWMI		24 88.9%	3 11.1%	27 100.0%
EXTENSIVE AWMI		1 100.0%	0 .0%	1 100.0%
IPLMI		0 .0%	2 100.0%	2 100.0%
IWMI		38 79.2%	10 20.8%	48 100.0%
	IWMI+ AWMI	0 0.0%	1 100.0%	1 100.0%
	IWMI WITH RV EXTENSION	0 .0%	1 100.0%	1 100.0%
	LWMI	1 50.0%	1 50.0%	2 100.0%
	NSTEMI	1 100.0%	0 .0%	1 100.0%
	PWMI	1 100.0%	0 .0%	1 100.0%
Total		82 82.0%	18 18.0%	100 100.0%

P –VALUE: 0.006

Our study showed comparison based on pattern of myocardial infarction with outcome, more patients died, that is 10 (20.8%) in INFERIOR WALL MI, 3(11.1%)

in ANTERIOR WALL MI, 2 (100%) in INFEROPOSTEROLATERAL MI, 1 each in AWMI + IWMI, IWMI with RV extension and LATERAL WALL MI.

38 patients (79.2%) who got discharged had IWMI, 24 (88.9%) had AWMI,13 (100%) had ALMI, 3 (100%) had ASMI and 1 each in EXTENSIVE ANTERIOR WALL MI, LATERAL WALL MI, NON ST ELEVATION MI.

P -value being statistically insignificant.

TABLE 13: CORRELATION OF OUTCOME WITH HYPERTENSION

HYPERTENSION	OUTCOME		TOTAL
	DISCHARGE	**EXPIRED**	**TOTAL**
Yes	23 79.3%	6 20.7%	29 100.0%
No	59 83.1%	12 16.9%	71 100.0%
TOTAL	**82** **82.0%**	**18** **18.0%**	**100** **100.0%**

P- VALUE: 0.775

6 (20.7%) patients died in our present study who had hypertension and 12 (16.9%) patients who died were normotensives.

59 (83.1%) patients got discharged who were normotensive and 23 (79.3%) patients with hypertension got discharged

P -value (0.775) being statistically insignificant.

TABLE 14: CORRELATION OF KILLIP CLASS WITH AGE

AGE GROUP	KILLIP I	II	III	IV	TOTAL
< 40 YEARS	**3**	**5**	**1**	**1**	**10**
41-60 YEARS	**14**	**11**	**11**	**3**	**39**
>60 YEARS	**25**	**10**	**11**	**5**	**51**
					100

P-VALUE = 0.466

In our study killip class was compared with age, in killip class I, 25 patients were in age group more than 60 years, 14 patients in age group between 41-60 years and 3 in age group less than 40 years.

In killip class II, 10 patients were in age group more than 60 years, 11 in age group between 41-60 years and 5 in age group less than 40 years.

In killip class III, out of 23 patients (11in age group more than 60 years, 11 between 41-60 years and only 1 in age group less than 40 years).

In killip class IV, out of 9 patients (5 in age more than 60,3 in 41-60 and only 1 in less than 40 years).

P -value being statistically insignificant.

TABLE 15: CORRELATION OF KILLIP CLASS WITH GENDER

GENDER	KILLIP I	II	III	IV	TOTAL
MALE	**33**	**21**	**15**	**16**	**75**
FEMALE	**10**	**4**	**8**	**3**	**25**
					100

P-VALUE = 0.488

Killip classs when compared with gender, majority (43) were in class I (male 33; female10), 25 in class II (male21; female 4), 23 in class III (male 15; female 8) and 19 in class IV (male 16; female 3).

P -value being statistically insignificant.

TABLE 16: CORRELATION OF KILLIP CLASS WITH HYPERTENSION

	KILLIP I	II	III	IV	TOTAL
HYPERTENSION	**13**	**6**	**6**	**4**	**29**
NORMOTENSION	**13**	**19**	**17**	**5**	**71**
					100

P-VALUE = 0.701

Majority of hypertensive patient (13) were in killip class I, 6 each in class II and III and remaining 4 in class IV.

In normotension group majority (19) patients were in killip class II, 17 in class III, 13 in class I and 5 in class IV.

P -value being statistically insignificant.

TABLE 17: CORRELATION OF KILLIP CLASS WITH PATTERN OF MI

	KILLIP I	II	III	IV	TOTAL
ALMI	**9**	**2**	**2**	**0**	**13**
ASMI	**1**	**1**	**1**	**0**	**3**
AWMI	**11**	**11**	**5**	**0**	**27**
EXTEN AWMI	**0**	**0**	**1**	**0**	**1**
IPLMI	**0**	**0**	**1**	**1**	**2**
IWMI	**19**	**11**	**12**	**6**	**48**
IWMI+AWMI	**0**	**0**	**0**	**1**	**1**
IWMI WITH RV EXTENSION	**0**	**0**	**0**	**1**	**1**
LWMI	**1**	**0**	**1**	**0**	**2**
NSEMI	**1**	**0**	**0**	**0**	**1**
PWMI	**1**	**0**	**0**	**0**	**1**
					100

Comparison based on pattern of myocardial infarction with killip class showed that in killip class I- 19 had IWMI, in class II- 11 had IWMI, in class III- 12 had IWMI and class IV- 6 had IWMI, remaining pattern of MI with killip class is depicted in the above table.

TABLE 18: CORELATION OF KILLIP CLASS WITH CAG (CORONARY ANGIOGRAPHY)

CAG	KILLIP I	II	III	IV	TOTAL
SINGLE VESSEL DISEASE	21	16	12	1	50
DOUBLE VESSEL DISEASE	4	2	5	2	12
TRIPLE VESSEL DISEASE	0	0	1	0	1
TOTAL	25	18	18	3	63

P- VALUE = 0.121

An effort to correlate coronary angiography with killip class showed 25 patients in class I (SVD-21, DVD-4), 18 patients in class II (SVD-16, DVD-2), 18 in class III (SVD-12, DVD-5, TVD -1) and 3 in class IV (SVD-1, DVD-2).

In remaining 37 patients CAG was not performed due to one or the other reason.

P -value being statistically insignificant.

TABLE 19: LEVT VENTRICLE FUNCTION BY 2D ECHO

EJECTION FRACTION	NUMBER	PERCENTAGE	IMPRESSION
>50%	52	52%	**NORMAL**
40-49%	28	28%	**MILD LVD**
30-39%	17	17%	**MODERATE LVD**
<30%	3	3%	**SEVERE LVD**
TOTAL	**100**	**100%**	

In present study of 100 patients, 52 (52%) had normal ejection fraction, however 28 patients (28%) had mild left ventricle dysfunction with EF 40-49%. 17 patients (17%) had moderate LV dysfunction with EF 30-39% and only 3 patients (3%) had severe LV dysfunction with EF less than 30%.

TABLE 20: 2D ECHO CORRELATION WITH URIC ACID DAY 1

	URIC ACID DAY 1	N	MEAN	STD. DEVIATION
2D ECHO	**ABNORMAL**	**46**	**39.24**	**7.38**
	NORMAL	**54**	**48.89**	**5.55**

P –VALUE: <0.001

On comparison of 2D ECHO findings with uric acid levels, we found significant correlation. Patient with elevated serum uric acid levels had significant LV dysfunction.

P -value being statistically significant.

TABLE 21: 2D ECHO CORRELATION WITH OUTCOME

2D ECHO	OUTCOME		TOTAL
	DISCHARGED	EXPIRED	
NORMAL	52 100.0%	0 .0%	52 100.0%
MILD LVD	27 96.4%	1 3.6%	28 100.0%
MODERATE LVD	3 100.0	14 82.4%	17 100.0%
SEVERE LVD	0 0.0%	3 100.0%	3 100.0%

P-VALUE<0.001

When we compared 2D ECHO findings with outcome, majority of the patients who expired had moderate to severe left ventricle dysfunction with only 1 had mild LV dysfunction.

P-value <0.001 being statistically significant.

TABLE 22: THROMBOLYSIS THERAPY

THROMBOLYSED	NUMBER	PERCENTAGE
YES	**69**	**69%**
NO	**31**	**31%**
TOTAL	**100**	**100%**

In our study out of 100 patients, majority of patients 69 (69%) were thrombolysed, rest 31 were not thrombolysed due to one or the other reason.

TABLE 23: CORONARY ANGIOGRAPHY (CAG)

CAG	NUMBER	PERCENTAGE
SINGLE VESSEL DISEASE	50	50%
DOUBLE VESSEL DISEASE	12	12%
TRIPLE VESSEL DISEASE	1	1%
TOTAL	**63**	**63%**

In our study out of 100 patients,63 patients (63%) underwent coronary angiography which revealed single vessel disease in 50 patients (50%), double vessel disease in 12 patients (12%) and only 1 patient (1%) had triple vessel disease.37 patients did not undergo CAG for varied reasons.

LABORATORY PARAMETERS

TABLE 24: FASTING BLOOD SUGAR.

FBS (mg/dl)	NUMBER	PERCENTAGE
<126	**92**	**92%**
≥126	**8**	**8%**
TOTAL	**100**	**100%**

We observed that majority of the patients 92 (92%) had fasting blood sugar less than 126 and only 8 patients (8%) had blood sugar more than 126.

FASTING LIPID PROFILE

TABLE 25: TOTAL CHOLESTEROL

TOTAL CHOLESTEROL(mg/dl)	NUMBER	PERCENTAGE
<200	**84**	**84%**
≥200	**16**	**16%**
TOTAL	**100**	**100%**

84 patients (84%) had cholesterol of less than 200 mg/dl and in 16 patients (16%) was more than 200 mg/dl.

TABLE 26: HIGH DENSITY LIPOPROTEIN

HDL (mg/dl)	NUMBER	PERCENTAGE
<40	6	6%
≥40	94	94%
TOTAL	**100**	**100%**

Majority of our patients that is 94 patients (94%) had HDL of more than or equal to 40 mg/dl and only 6 patients (6%) had HDL levels less than 40 mg/dl

TABLE 27: LOW DENSITY LIPOPROTEIN

LDL(mg/dl)	NUMBER	PERCENTAGE
<100	90	90%
≥100	10	10%
TOTAL	**100**	**100%**

It was observed that in 90 patients (90%) the LDL was less than 100 mg/dl and in remaining 10 patients (10%) it was equal to or more than 100 mg/dl.

TABLE 28: TRIGLYCERIDES

TRIGLYCERIDES(mg/dl)	NUMBER	PERCENTAGE
<150	40	40%
≥150	60	60%
TOTAL	**100**	**100%**

60 patients (60%) had triglycerides below 150 mg/dl, 40 patients (40%) had equal to or more than 150 mg/dl.

TABLE 29: TRIGLYCERIDE CORRELATION WITH URIC ACID DAY 1

	URIC ACID DAY 1	N	MEAN	STD. DEVIATION
TRIGLYCERIDE	**ABNORMAL**	**46**	**200.94**	**48.57**
	NORMAL	**54**	**144.32**	**40.53**

P-VALUE: <0.001

Comparison between triglycerides levels and uric acid levels on day 1 showed significant correlation. Patients who had high triglycerides levels had high uric acid levels on day1.

P -value being statistically significant.

TABLE 30: TRIGLYCERIDE CORRELATION WITH OUTCOME

TRIGLYCERIDE (mg/dl)	OUTCOME		TOTAL
	DISCHARGE	EXPIRED	
<150	41	0	41
	100%	.0%	100%
≥150	41	18	59
	69.5%	30.5%	100.0%
TOTAL	82	18	100
	82.0%	18.0%	100%

P-VALUE: <0.001

Triglycerides levels correlated with outcome showed that 18 patients (30.5%) who expired all had high triglycerides levels that is equal to or more than 150 mg/dl

It is statistically significant p-value: 0.001

TABLE 31: CORELATION BETWEEN TROPONIN I AND KILLIP CLASS

KILLIP CLASS	TROPONIN-I (n)	MEAN	STD. DEVIATION
I	**43**	**4.61**	**10.51**
II	**25**	**2.92**	**3.35**
III	**23**	**8.83**	**9.93**
IV	**9**	**4.15**	**5.05**
TOTAL	**100**	**5.12**	**8.84**

P -VALUE: 0.118

In the present study mean troponin levels were higher in killip class III and IV as compared to class I and II. However it was statistically not significant.

TABLE 32: CORRELATION OF TROPONIN-I WITH URIC ACID DAY 1

	URIC ACID DAY 1	N	MEAN	STD. DEVIATION
TROPONIN-I	**ABNORMAL**	**46**	**6.27**	**7.94**
	NORMAL	**54**	**4.14**	**9.52**

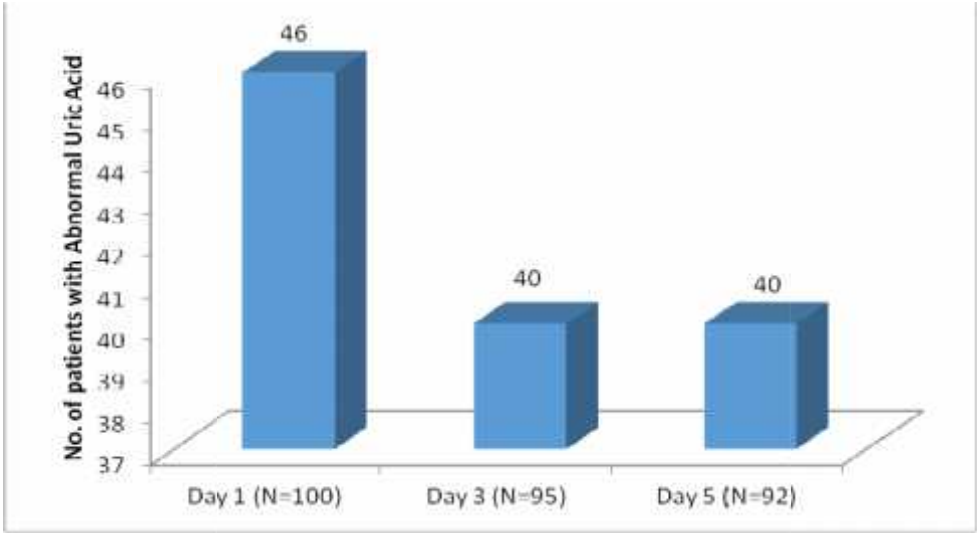
P- VALUE: 0.232

Correlation between troponin-I levels with uric acid day on day 1 was not significant.

P -value being insignificant**.**

TABLE 33: SERUM URIC ACID (SUA)

ABNORMAL URIC ACID	FREQUENCY	PERCENT	MEAN	STD DEVIATION
Day 1	46/100	46.0	6.43	1.99840
Day 3	40/95	42.11	6.51	2.09945
Day 5	40/92	43.48	6.28	2.10630



Graph 11 : Serum uric acid.

In our study maximum number of patients ie., 46 patients (46%) had abnormal uric acid on day1, 40 patients each on day 3 and day 5. The mean serum uric acid levels on day 1, 3 and 5 were 6.43±1.99, 6.51±2.09 and 6.28±2.10 respectively.

TABLE 34: CORRELATION OF SERUM URIC ACID ON DAY 1 WITH AGE.

AGE GROUP	URIC ACID DAY 1		TOTAL
	NORMAL	ABNORMAL	
<=40	6	6	12
	11.1%	13.0%	12.0%
41-60	18	22	40
	33.3%	47.8%	40.0%
>60	30	18	48
	55.6%	39.1%	48.0%
TOTAL	**54**	**46**	**100**
	100.0%	**100.0%**	**100.0%**

P-value: 0.249

The above table shows that on day 1 more number of patient ie.,22 (47.8%) with abnormal uric acid were in age group 41-60.

P -value being statistically insignificant.

TABLE 35: CORRELATION OF SERUM URIC ACID ON DAY 3 WITH AGE.

AGE GROUP	URIC ACID DAY3		TOTAL
	NORMAL	ABNORMAL	
<=40	5 9.1%	6 15.0%	11 11.6%
41-60	20 36.4%	18 45.0%	38 40.0%
>60	30 54.5%	16 40.0%	46 48.4%
TOTAL	**55** **100.0%**	**40** **100.0%**	**95** **100.0%**

P -value: 0.343

The above table shows that on day 3, more number of patients ie.18 (45.0%) with abnormal uric acid were in age group 41-60 years.

P -value being statistically insignificant.

TABLE 36: CORRELATION OF SERUM URIC ACID ON DAY 5 WITH AGE.

AGE GROUP	URIC ACID DAY 5		TOTAL
	NORMAL	ABNORMAL	
<=40	4	7	11
	7.7%	17.5%	12.0%
41-60	18	18	36
	34.6%	45.0%	39.1%
>60	30	15	45
	57.7%	37.5%	48.9%
TOTAL	**52**	**40**	**92**
	100.0%	**100.0%**	**100.0%**

P -value: 0.115

The above table shows that on day 5 more number of patients ie., 18 (34.6%) with abnormal uric acid were in age group 41-60 years.

P -value being statistically insignificant.

TABLE 37: ABNORMAL SERUM URIC ACID CORRELATION WITH GENDER

	MALE (N/TOTAL)	FEMALE (N/TOTAL)	TOTAL	FEMALE TO MALE RELATIVE RISK (95% CI)	P-VALUE
Day 1	33/75(44%)	13/25(52%)	46/100	1.182 (0.750 – 1.863)	0.487
Day 3	29/72(40.28%)	11/23(47.83%)	40/95	1.187 (0.712 – 1.980)	0.523
Day 5	30/69(43.48%)	10/23(43.48%)	40/92	1.000 (0.584 – 1.713)	1.000

The above table shows abnormal serum uric acid levels in percentage, female patients had percentage wise more serum uric acid on day 1 and day 3 as compared to males. However it was almost same on day 5 in both the gender.

It was statistically insignificant.

TABLE 38: CORRELATION OF SERUM URIC ACID DAY 1 WITH KILLIP CLASS

KILLIP CLASS	Uric Acid Day 1		
	Normal	Abnormal	Total
I	39	4	43
	72.2%	8.7%	43.0%
II	11	14	25
	20.4%	30.4%	25.0%
III	2	21	23
	3.7%	45.7%	23.0%
IV	2	7	9
	3.7%	15.2%	9.0%
Total	54	46	100
	100.0%	100.0%	100.0%

P value: 0.00

Patients with abnormal uric acid on day 1 were more in KILLIP class III and p- value was 0.000 which was statistically significant.

TABLE 39: CORRELATION OF URIC ACID DAY 3 WITH KILLIP CLASS

KILLIP CLASS	Uric Acid Day 3		
	Normal	Abnormal	Total
1	41	2	43
	74.5%	5.0%	45.3%
II	12	12	24
	21.8%	30.0%	25.3%
III	1	19	20
	1.8%	47.5%	21.1%
IV	1	7	8
	1.8%	17.5%	8.4%
Total	55	40	95
	100.0%	100.0%	100.0%

P -value: 0.00

Patients with abnormal uric acid on day 3 were more in KILLIP class III and p- value was .000 which was statistically significant.

TABLE 40: CORRELATION OF SERUM URIC ACID DAY 5 WITH KILLIP CLASS

KILLIP CLASS	Uric Acid Day 5		
	Normal	Abnormal	Total
1	38	5	43
	73.1%	12.5%	46.7%
II	13	10	23
	25.0%	25.0%	25.0%
III	1	18	19
	1.9%	45.0%	20.7%
IV	0	7	7
	.0%	17.5%	7.6%
Total	40	52	92
	100.0%	100.0%	100.0%

P -value: 0.00

Patients with abnormal uric acid on day 5 were maximum in KILLIP class III and p-value was .000 which was statistically significant.

TABLE 41: CORRELATION OF HYPERTENSION WITH ABNORMAL SERUM URIC ACID

	Hypertension Present (N/TOTAL)	**Hypertension Absent (N/TOTAL)**	**Total (N/TOTAL)**	**Female to Male Relative Risk (95% CI)**	**p-value**
Day 1	12/29(41.4%)	34/71(47.9%)	46/100	0.864 (0.526 – 1.420)	0.554
Day 3	13/29(44.8%)	27/66(40.9%)	40/95	1.096(0.667 – 1.80)	0.722
Day 5	14/29(48.3%)	26/63(41.3%)	40/92	1.17(0.725 – 1.89)	0.529

The above table shows, abnormal uric acid levels were more in hypertensive group on day 3 & 5 but it was statistically insignificant.

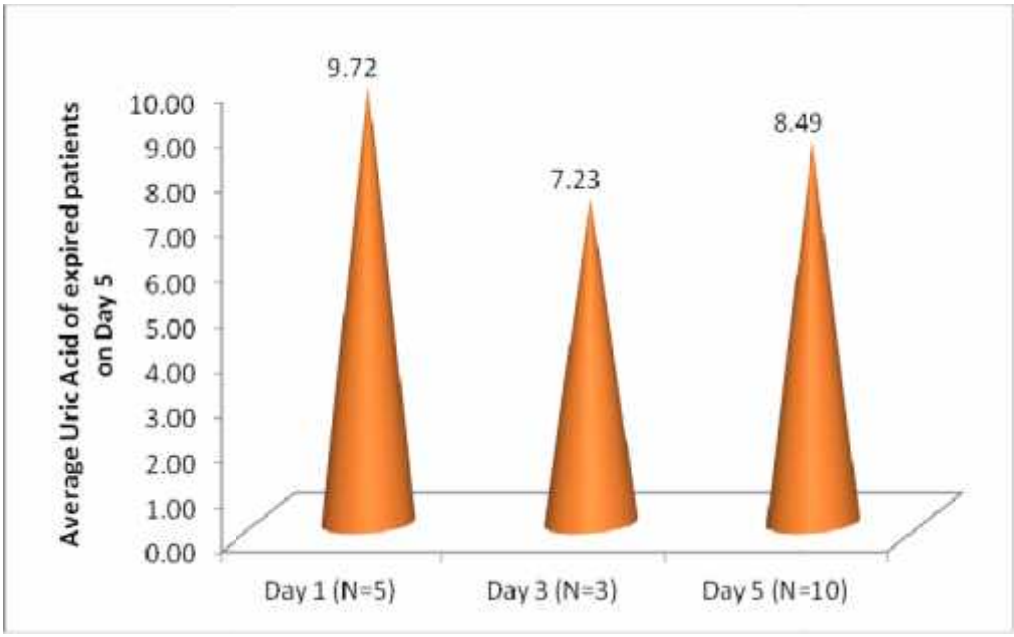
Similarly, abnormal uric acid level was more in non-hypertensive group on day, it was statistically insignificant.

TABLE 42: OUTCOME IN RELATION WITH ABNORMAL SERUM URIC ACID.

OUTCOME	N	TOTAL	Mean	Std. Deviation	t-value	p-value
URIC ACID DAY 1	5	100	9.72	1.95	4.123	<0.001
Expired						
Survived	95		6.21	1.84		
URIC ACID DAY 3	3		7.23	3.16	0.645	0.521
Expired		95				
Survived	92		6.44	2.08		
URIC ACID DAY 5	10		8.4900	.92550	3.820	<0.001
Expired		92				
Survived	82		6.0890	1.95372		

The above table shows, mean abnormal uric acid levels were more in expired group on day 1, day 3 and day 5 compared to survived group but it was statistically significant only on day 1 and day 5.

GRAPH: AVERAGE URIC ACID OF EXPIRED PATIENT AT END OF DAY5



Graph 12 : Average uric acid of expired patient at end of day 5.

The above graph shows that the average uric acid levels of expired patients were raised on all 3 days with maximum on day 1.

TABLE 43: CORRELATION OF KILLIP CLASS WITH MEAN URIC ACID LEVELS

	Killp class	N	Mean	Std. Deviation
URIC ACID DAY 1	I	43	4.7605	.99286
	II	25	6.9520	1.36660
	III	23	8.1739	1.55043
	IV	9	8.0333	2.14126
	Total	100	6.3880	1.99840
URIC ACID DAY 3	I	43	4.9535	.82875
	II	24	6.8875	1.36487
	III	20	8.6300	2.34433
	IV	8	7.8750	1.92854
	Total	95	6.4621	2.09945
URIC ACID DAY 5	I	43	4.8163	.97295
	II	23	6.7348	1.42880
	III	19	8.3684	1.46175
	IV	7	9.0286	1.46937
	Total	92	6.3500	2.01170

The above table shows that Mean uric acid levels at day 1, 3 and day 5 are significantly higher in Killip class III and IV as compared to I & II.

P -value < 0.01 being statistically significant.

DISCUSSION

In the present study of 100 patients with acute myocardial infarction, the importance of serum uric acid levels and killip class was studied to know the prognosis in these patient.

In our study, patients age ranged from 28-88 years, maximum number of patients that is 48 (48%) were in the age group of more than 60 years, between age group 41-60 years 40 (40%) patients, below 40 years 12 (12%) patients .There were more cases of myocardial infarction after the age of 40 years as compared to below 40 years of age. This is almost similar to the study done by Shetty et al.[67] It also correlates with study by Omidvar et al.[68]

Taking Gender in to consideration, we observed in our study that males (75%) were more as compared to females (25%). Similar conclusion was drawn by Shetty et al.[67]

The clinical presentation of patients, majority of them presented with history of chest pain (46%) followed by dyspnoea (18%), 36% of the patients presented with both the symptoms (chest pain and dyspnoea). This is almost similar to the study by Ersan Tatli et al [69]and Omidvar et al.[68] In both these studies patient presented with typical history of chest pain suggestive of myocardial infarction were taken in to account.

61 patients (61%) gave history of habits like smoking (20%), tobacco chewing (31%) and both (10%), 39 patients (39%) did not have any habits. This is in contrast to the study by Ersan Tatli et al, [69] they observed 95% of their patient had history of smoking.

In our present study, 12 lead ECG showed that maximum number of patients 48 (48%) had IWMI. This is in contrast to study by Ersan Tatli et al, [69] where maximum number of patients had AWMI (65%).

In our study majority of patients were in killip class I (43%), class II (25%), class III (23%) and only (9%) in class IV. Similar observation was drawn by Nadkar et al [70] who had maximum cases in killip class I. However in contrast to this, a study by Shetty et al [67]who had maximum number of cases in killip class II.

We observed that 29 patients had history of hypertension and were on treatment and 71 patients were normotension. This is in contrast to the study by Shetty et al [67] where more than 50% of their patients had hypertension.

In our study 82 (82%) patients were discharged after 5 days and 18 (18%) died during the hospital stay. This is in contrast to the study by Nadkar et al [70] and Shetty et al.[67]

When an attempt was made to correlate age with outcome of patients, we observed that maximum number ie., 9 deaths in age group between 41-60 years, 7 in age more than 60 years and only 2 in age below 40 years. To best of our knowledge when we tried to search for studies to compare age with outcome, information is lacking.

When we tried to compare outcome of patients in relation with gender, 12 males expired out of 75 and 6 females expired out of 25. Again the outcome in relation with gender alone, literature is lacking.

Similarly the outcome of patients in relation with killip class was done by us and we found that maximum number of patients who expired belong to class III and

IV (8 in each class) and those who survived, maximum were in killip class I (43) and II (23). This shows that as killip class increases mortality increases. P-value being statistically significant (0.001). This is similar to the study by Nadkar et al [70] and Shetty et al [67] where all the patients who expired were in killip class III and IV.

Correlation with pattern of myocardial infarction and outcome revealed that maximum number ie., 10 patients had IWMI who expired. Study by Dharma et al [71] have observed the pattern or area of myocardial infarction but not correlated with outcome of patients.

We have tried to compare outcome of patients with or without hypertension alone, only 6 (20.7%) patients out of 18 who expired had hypertension, which was statistically insignificant. Whereas study in Iran 2007 by sokhanvar and Maliki[72] found significant correlation with hypertension but they have correlated other risk factors with hypertension like gender and hyperuricemia.

Age with killip class comparison revealed that in all age group maximum number of patients were in killip class I and II as compared to class III and IV. It was statistically insignificant. However studies by various author, the comparison of age with killip class is lacking.

Similarly Gender and killip class comparison revealed that in both the gender maximum number of patients were in killip class I and II as compared to class III and IV. It was statistically insignificant. Studies by various author, the comparison of gender with killip class is lacking.

Comparison of hypertension with killip class showed that maximum number of hypertensive patients were in killip class I and II as compared to class III and IV. It

was statistically insignificant. Similar attempt by Nadkar et al [70] and Shetty et al [67] was done with hypertension, however other variables were also taken in to account in their study but same was not compared with killip class.

We also tried comparison of pattern of myocardial infarction with killip class, patients with IWMI were observed in all the 4 killip class.The literature with respect to pattern of MI and killip class is missing.

63 patients out of 100 were subjected to coronary angiography and same was when compared with killip class, more number of patients with SVD and DVD were in killip class I and II as compared with class III and IV and only 1 patient had TVD who was in class III. We did not find significant correlation between coronary angiography findings and killip class, Literature on correlation between killip class and coronary artery disease is lacking.

All 100 patients in the study were subjected to 2D ECHO and we observed that 52 patients (52%) had normal left ventricle function and remaining 48 patients (48%) had LV dysfunction ranging from mild to severe. When we compared 2D ECHO findings with serum uric acid levels on day 1, we found significant correlation. Patient with elevated serum uric acid levels had significant LV dysfunction. P -value (<0.001) being statistically significant. Also on comparing 2D ECHO with outcome of patients, result was statistically significant (p-value <0.001). Study by Chen et al [73] and Shetty et al [67] found similar observation when they evaluated LV dysfunction by 2D ECHO and compared with uric acid. Patients with moderate to severe LV dysfunction had high uric acid levels.

Out of 100 patients, 69 (69%) were thrombolysed and remaining 31 were not thrombolysed for one or the other reason. A study by Dharma et al [71] have used

thrombolytic therapy in their patients and same was compared to serum uric acid levels and found significant correlation. In our study, this attempt was not made as only 69 patients were thrombolysed which may not reflect the true correlation.

In the present study of 100 patients more patients that is 92 (92%) had normal fasting blood sugar level and only 8 (8%) had fasting blood sugar levels equal to or more than 126 mg/dl. In our study we excluded patient with known history of type 2 Diabetes mellitus so as to avoid the bias that the Diabetics patients may have effect on serum uric acid levels. An explaination by proposed mechanism in various studies by Quiniones et al, [81] Muscelli et al, [75] hyperinsulinemia may affect the excretion of uric acid via the kidneys, this may indirectly influence the elevated level of serum uric acid.

In our study we observed that maximum number ie., 84 (84%) patients had total cholesterol within normal range and only 16 patients (16%) had high cholesterol levels. HDL level when analysed 94 patients (94%) had normal values and only 6 patients (6%) had abnormal levels. LDL level showed normal values in 90 patients (90%) and only 10 patients (10%) had abnormal levels. Triglycerides when analysed majority (60%) had high levels and remaining 40% had normal levels. We observed significant correlation between triglyceride levels and outcome of patients with myocardial infarction. P-value statistically being significant (0.001). Study by Wannamethee et al, [76] and Chen et al [73] also showed significant correlation between serum uric acid level and triglyceride level.

Comparison of Troponin-I level with killip class showed that mean troponin-I levels were more in killip class III and IV as compared to class I and II. However it

was not statistically significant. Studies comparing troponin and killip class are lacking.

Serum uric acid estimation revealed that more number of patients 46 (46%) out of 100 had abnormal levels on day1, 40 patient (42.11%) out of 95 on day 3 and 40 patients (43.48) out of 92 on day 5. The mean serum uric acid levels on day 1, 3 and 5 were 6.43±1.99, 6.51±2.09 and 6.28±2.10 respectively. It was statistically not significant. Similar observation was also seen in study by Nadkar et al. [70] We also tried to compare uric acid levels of expired patients, the average uric acid levels of expired patients were raised on all 3 days with maximum on day 1. Study by Omidvar et al [68] and Car et al[77] showed strong relation between serum uric acid at the time of admission and in hospital and short term mortality. Trkulja V [78] found on admission serum uric acid predict outcome.

An attempt was made to compare serum uric acid level on day 1, 3 and 5 with different age group, which showed that on all the 3 days more number of patients with abnormal uric acid levels were in the age group 41-60 years. However it was statistically not significant. This is in contrast to the study by PK Sarma [79] who observed raising trend of serum uric acid as age advances. They had 62 patients who were above 60 years of age with abnormal uric acid levels.

Comparing serum uric acid level with gender revealed that abnormal uric acid levels were more in females on day 1 and 3 but it was same on day 5, however it was statistically not significant. Study by Dharma et al [71] also found no significant difference between uric acid levels in both the gender.

An attempt was made to compare serum uric acid levels with killip class showed that on all 3 days patient with abnormal uric acid were more in class III. P-

value being statistically significant (0.00). Kojima et al, [80] Nadkar et al, [70] Shetty et al [67] have observed similar correlation between serum uric acid level and killip class. In contrast to this, a study by Jularattanaporn et al [74] observed no association between hyperuricemia and killip class.

When we tried to correlate serum uric acid levels with hypertension, we did not find any significance between the two group (hypertension & normotension). This was similar to the study by Shetty et al. In contrast to this, another study by Sokhanvar and Maleki in 2007 in Iraq [72] found significant correlation between hyperuricemia and hypertension.

In our study we tried to correlate serum uric acid level with outcome of patients, we found significant correlation between uric acid and mortality, however it statistically significant on day 1 and day 5. Dharma et al [71]in there study found adverse cardiovascular outcome with abnormal serum uric acid level. Omidvar et al [68] and Car et al [77] observed association between abnormal uric acid level and short term mortality. Kojima S et al [80] in their study conducted in japan suggest that hyperuricemia after MI is associated with the development of heart failure. Bickel et al [54] showed that increased uric acid level is associated with overall mortality.

In our present small sample study of 100 patients, who were evaluated, comparing various variables, we found significant correlation between abnormal serum uric acid levels with higher killip class (III and IV) and when mortality was taken in to account, elevated levels of serum uric acid was seen in those patients who expired (18 patients). This simple, easily available, inexpensive laboratory parameter (estimation of serum uric acid) may help in settings where facilities are lacking, can be a useful biomarker when compared with killip class to prognosticate patients of

acute myocardial infarction. To address this issue whether efficacy of serum uric acid level in these patients is significant or not, a detailed study with large sample size may be required.

SUMMARY

In the present study of 100 patients titled "THE IMPORTANCE OF SERUM URIC ACID LEVELS AND KILLIP CLASSIFICATION IN PREDICTING PROGNOSIS OF ACUTE MYOCARDIAL INFARCTION" during the period from January 2014 to December 2014 in the department of General Medicine and Cardiology, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. The findings of the study has been summarized as follows:

- There were more cases of myocardial infarction above 40 years as compared to below 40 years of age.
- In our study, males (75%) were more as compared to females (25%).
- The commonest presentation was chest pain (46%) followed by dyspnoea (18%), 36% of the patients presented with both the symptoms (chest pain and dyspnoea).
- Majority of the patients had IWMI (48%).
- Out of 100 patients only 29 had hypertension.
- In our study 52% patients had normal LV function and 48% had mild to severe LV dysfunction.
- Out of 100 patients, 60% had high triglycerides levels and 40% had normal levels.
- Majority of patients had abnormal uric acid on day 1 as compared to day 3 and 5.

- On all the 3 days, majority of patients with abnormal uric acid were in age group 41-60 years.
- Patients with abnormal uric acid levels on all the 3 days were more in killip class III and IV as compared to class I and II.
- Out of 100 patients, 18 expired during the hospital stay.
- Among 18 patients who expired, 16 were in killip class III and IV (8 in each class).
- The mean serum uric acid levels of expired patients were raised on all the 3 days with maximum on day 1.

CONCLUSION

In the present study of 100 patients with acute myocardial infarction we observed significant correlation with various factors.

Based on the findings of the present study the prominent features are:

- In patients with acute myocardial infarction, patients with **hyperuricemia had higher mortality**.
- There was a positive correlation with elevated serum uric acid levels with killip class. (**p-value being statistically significant**)
- There was strong correlation between hyperuricemia and left ventricle dysfunction. (p-value being **statistically significant**)
- Hyperuricemia is an indicator of poor prognosis in acute myocardial infarction.
- Comparison between triglycerides levels and uric acid levels on day 1 showed **statistically significant** correlation.
- High triglycerides levels were also associated with poor outcome of the patients.(p-value was statistically significant)
- We didn't find significant correlation with various factors like age, gender, pattern of myocardial infarction, hypertension, blood sugar levels, total cholesterol, HDL, LDL, Troponin-I levels.

We feel it is worth to study by adjusting co-morbid condition/ confounding factors and comparing them with serum uric acid levels and killip class to know whether these factors have true association or not.

Owing to our small sample size study (100 patients), a large sample size may be required to overcome these bias.

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ANNEXURE I – CONSENT FORM

Title of Research Study:

"THE IMPORTANCE OF SERUM URIC ACID LEVELS AND KILLIP CLASSIFICATION IN PREDICTING PROGNOSIS OF ACUTE MYOCARDIAL INFARCTION".

Principal Investigator:-

Dr. Rockey Katheria, Post Graduate Student,

Department of General Medicine, J.N. Medical College,

Belgaum.

Introduction and Purpose:-

The study is to determine the correlation between Serum Uric Acid and Killip's classification in acute myocardial infarction patients.

Procedure:

If you agree to be part of the research study, you will be asked the history of any chest pain, associated with sweating, radiating to left shoulder/neck and will be subjected to relevant examination and investigations like ECG and Uric acid. You will also have to give blood and urine samples for the necessary investigations.

Risk and Benefits:

The only risk and possible discomfort you might get is while taking blood from my arm for the investigations. It may cause swelling, pain, redness, bruising or infection (rarely happens) at the site from where the blood is drawn.

The benefit is that it helps in predicting prognosis of acute myocardial infarction patient.

Alternatives:

Taking part in this study is voluntary. You may choose not to take part in this study, or if you decide to take part you can later change your mind and withdraw from the study. Your decision will not change the present or future health care or other services that you receive. The study doctor or sponsor may stop your participation in this study at any time. If you choose not to take part in the study, you will receive the standard treatment for patients with your condition.

Privacy and Confidentiality:

All information collected about you during the course of this study will be kept confidential to the extent permitted by law. The code numbers will identify you in this research record. Information from this study may be published but your identity will be confidential in any publication.

Institution / Sponsor's policy / compensation:

In case of any injury related to the study, treatment will be made available at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. There is no compensation or payment for such medical treatment by law.

Financial incentives for participation:

You will not be paid / offered any gifts /incentives for participating in the study.

Authorization to publish the results:

The results of the study would be forwarded to the KLE University, Belgaum as part of requirement towards the completion of MD degree, review and publishing.

Questions: During study/ in future you may contact following persons for any questions.

1. **Dr. Rockey Katheria,**
Investigator,
PG in General Medicine,
JNMC, Belgaum.ph.no-9986532167

2. **Dr. VIJAY.G.SOMANNAVAR,**
Professor,
Dept of General Medicine,
JNMC, Belgaum. Ph.no-9845710945

3. **Dr. S.V.PATTED,**
Professor,
Dept of Cardiology,
JNMC, Belgaum.

4. **Dr. P. V. PATIL,** MD, PhD, DHA,FIC(Path)
Professor,
Dept. of Pathology, J.N. Medical College,

Consent Statement:

"THE IMPORTANCE OF SERUM URIC ACID LEVELS AND KILLIP CLASSIFICATION IN PREDICTING PROGNOSIS OF ACUTE MYOCARDIAL INFARCTION".

I voluntarily agree to take part in this study by signing below. I may withdraw at any time. I am not giving up any of my legal rights by signing this form. My signature below indicates that I have read, or it has been read to me, this entire consent form, and have had all my questions answered.

Name of the Participant: _____ Signature /Thumb print: _____

Name of the Witness: _____ Signature/ Thumb print: _____

Investigator Name: _____ Signature :_____

Date: Place:

ANNEXURE II – PROFORMA

Case No:

NAME: **AGE/SEX:**

IP No. **ADDRESS:**

OCCUPATION

COMPLAINTS AT PRESENTATION: **YES** **NO**

PAST HISTORY:

TREATMENT HISTORY:

PHYSICAL EXAMINATION:

GENERAL CONDITION:

Pallor: Yes/No

Icterus: Yes/No

Lymphadenopathy: Yes/No

Cyanosis: Yes/No

Clubbing: Yes/No

Edema: Yes/No

VITALS:

Temperature:

Pulse:

Respiratory rate:

Blood pressure:

SYSTEMIC EXAMINATION:

R. S.:

C.V.S.:

P.A.:

C.N.S.:

Killip Class:

ANNEXURE - III – MASTER CHART

KEY TO MASTER CHART

-	-	Absent.
+	-	Present.
N	-	Nil.
M	-	Male.
F	-	Female.
Y	-	Yes.
DOA	-	Date of admission.
DOD	-	Date of discharge.
IP NO	-	Inpatient Number.
FBS	-	Fasting Blood Sugar.
HDL	-	High Density Lipoprotein.
LDL	-	Low Density Lipoprotein.