CORRELATION BETWEEN SERUM URIC ACID AND KILLIP CLASS IN ACUTE MYOCARDIAL INFARCTION

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For the Award of the Degree of

M.D. (GENERAL MEDICINE) - BRANCH – I

GOVERNMENT KILPAUK MEDICAL COLLEGE

CHENNAI

MAY 2018

BONAFIDE CERTIFICATE

This is to certify that "**CORRELATION BETWEEN SERUM URIC ACID AND KILLIP CLASS IN ACUTE MYOCARDIAL INFARCTION** is a bonafide work done by **Dr. KARTHIKEYAN V A.** Post graduate student, Department of General Medicine, Kilpauk Medical College, Chennai-10, under my guidance and supervision in partial fulfillment of rules and regulations of the TamilNadu Dr. M.G.R Medical University, for the award of M.D. Degree Branch I (General Medicine) during the academic period from MAY 2015 To MAY 2018.

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DECLARATION

I solemnly declare that this dissertation "**CORRELATION BETWEEN SERUM URIC ACID AND KIILLIP CLASS IN ACUTE MYOCARDIAL INFARCTION"** was prepared by me at Government Kilpauk Medical College and Hospital, Chennai, under the guidance and supervision of **Prof.Dr.K.V.RAJALAKSHMI, M.D.,** Professor and Head of the Department, Department of Internal Medicine, Government Kilpauk Medical College and Hospital, Chennai. This dissertation is submitted to **The Tamil Nadu Dr. M.G.R. Medical University, Chennai** in partial fulfillment of the University regulations for the award of the degree of **M.D. Branch I (General Medicine)**.

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CERTIFICATE – II

This is to certify that this dissertation work titled entitled dissertation "**CORRELATION BETWEEN SERUM URIC ACID AND KIILLIP CLASS IN ACUTE MYOCARDIAL INFARCTION"** of the candidate **Dr.KARTHIKEYAN V.A.** with Registration Number **201511156** for the award of **M.D** degree in the branch of **GENERAL MEDICINE**. I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows **5%** of plagiarism in this dissertation.

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LIST OF ABBREVATIONS

ACS	Acute coronary syndrome
AHA	American Heart Association
ACC	American college of Cardiology
cTn	Cardiac troponin
CABG	Coronary Artery Bypass grafting
CHF	Congestive heart failure
ECG	ElectroCardioGram
ESC	European Society of Cardiology
EF	Ejection Fraction
LAD	Left Anterior Descending artery
LCX	Left Circumflex artery
LCA	Left Coronary Artery
LBBB	Left Bundle Branch Block
MI	Myocardial Infarction
STEMI	ST Elevation Myocardial Infarction.
NSTEMI	Non ST Elevation Myocardial Infarction
AWMI	Anterior Wall Myocardial Infarction
LWMI	Lateral Wall Myocardial Infarction
IWMI	Inferior Wall Myocardial Infarction
RVMI	Right Ventricular Myocardial Infarction.
Trop	Troponin
UA	Unstable Angina
TIMI	Thrombolysis In Myocardial Infarction
WHO	World Health Organization
CK	Creatine Kinase
S3	Third Heart Sound

CONTENTS

INTRODUCTION

Coronary Artery disease is epidemic in our country. It is reported that CHD accounts for 32% of adult death in 2010-2013 in our country. In India there has been 4 fold increase in prevalence of coronary heart disease in last 40 year. Important risk factors are diabetes, hypertension, smoking, dyslipidemia, abdominal obesity, unhealthy diet, physical inactivity. Rapid urbanisation and change in life style in last two decade have led to growing burden of coronary heart disease.

Acute Coronary Syndrome (ACS) is a broad collection of condition

- Acute Myocardial Infarction which manifest either as ST Elevation MI (STEMI) and Non ST Elevation myocardial infarction (NSTEMI).
- Unstable angina(UA).

Notably, the clinical presentation and symptoms may be similar for all these syndromes.

The primary goals of treatment in ACS are relieve or limit ischemia, prevent reinfarction, improves outcome and well being.

Acute Myocardial infarction manifests as either ST segment elevation myocardial infarction (STEMI) or Non ST segment elevation myocardial infarction (NSTEMI).Risk stratification of acute myocardial infarction are done by various clinical assessment and score .KILLIP Classification is used assess the severity and prognosis in acute myocardial infarction .Serum uric acid which is a metabolite of purine often used as a biomarker of inflammation. Hyperuricemia are associated with various diseases such as chronic kidney disease, stroke, cardiovascular disease. The purpose of this study is to assess the correlation between serum uric acid and killip class in acute myocardial infarct.

AIM AND OBJECTIVES

AIM:

To study the association between Serum Uric acid level and killip class in acute myocardial infarction.

OBJECTIVES

1. To assess the serum uric acid in level in acute myocardial infarction.
2. To study the association between serum uric acid level an killip class in acute myocardial infarction

REVIEW OF LITERATURE

ACUTE MYOCARDIAL INFARCTION

The current universal definition for acute myocardial infarction adopted by ESC/ACF (American Cardiology Foundation) as evidence of myocardial necrosis in a clinical setting consistent. Under these condition any of following criteria meet the diagnosis of MI

1. Detection of rise and/or fall in cardiac biomarkers preferably troponin c with at least one value above the 99th percentile of upper reference limit with one of the following

   - Ischemic symptoms.
   - New significant ST segment T wave changes or new left bundle branch block.
   - Pathological Q wave on the ECG.
   - Imaging evidence of new loss of viable myocardium or regional wall motion abnormality.
   - Intracoronary thrombus identification by angiography or autopsy.

2. PCI related MI-Elevation of cardiac troponin to >5 times of 99th percentile of upper normal limit.

3. CABG related MI-Elevation of cardiac troponin to >10 times of 99^{th} percentile of the upper normal limit.

The patients included in acute myocardial infarction are either have

- ST segment elevation MI(STEMI)
- Non ST segment elevation MI(NSTEMI).

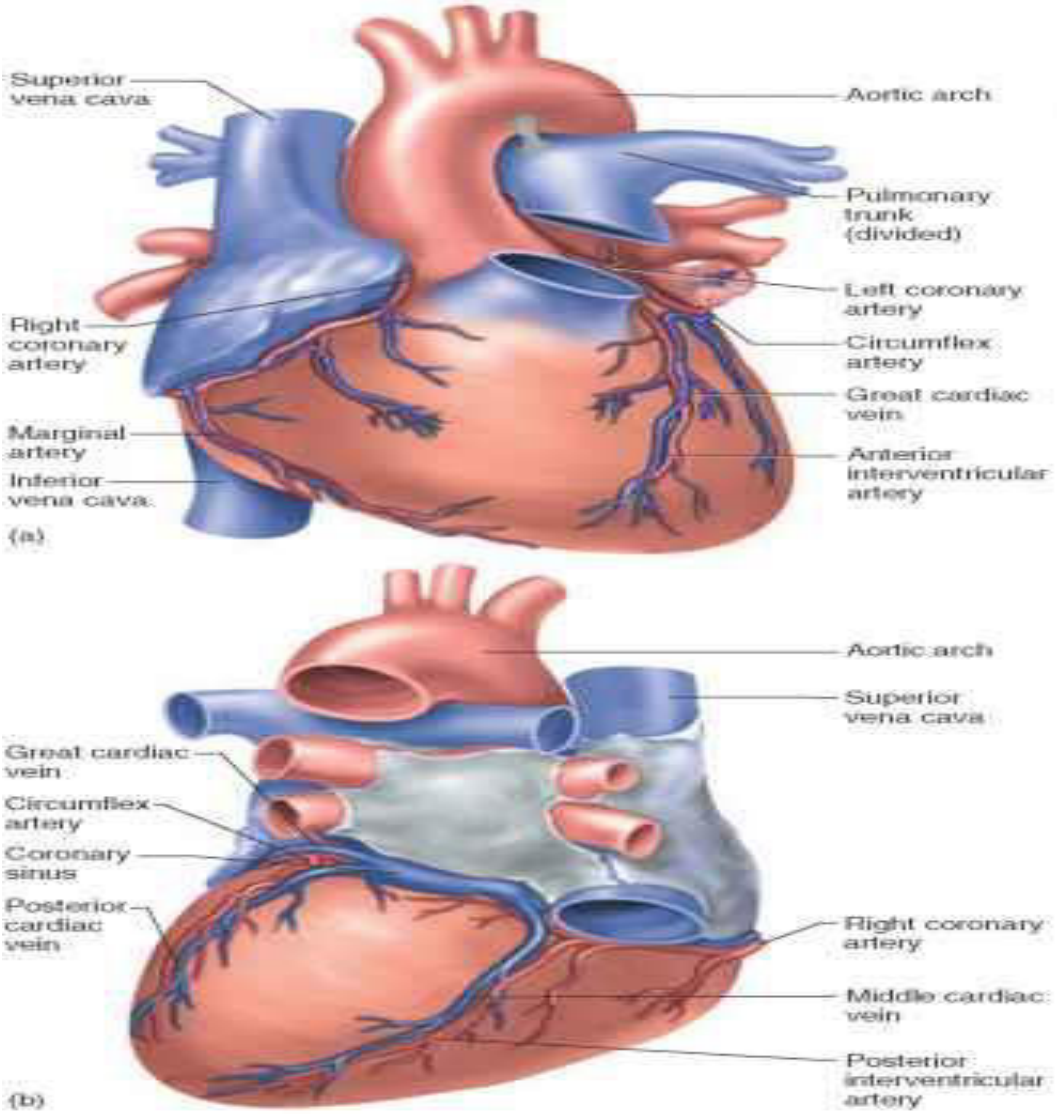
EPIDEMIOLOGY

- Acute Myocardial Infarction is the leading cause of mortality in both developed and developing nation such as India.
- A significant proportion of these patients will die from sudden cardiac death due to ventricular arrhythmia prior to arriving at the hospital.
- The success of the medical community's concerted efforts has led to 26% reduction in mortality since 1990
- The risk factors are hypertension, diabetes, smoking, dyslipidemia, abdominal obesity, sedentary life style, male gender.
- 90% of acute myocardial infarction are attributed to modifiable risk factors.
- Overall survival rates in majority of good Indian centers are > 90%.
- There are various predictors of mortality in acute myocardial infarction such as killip class,TIMI score by baseline clinical data.

BLOOD SUPPLY OF HEART

- Coronary artery are the first branch that arises from the aorta, it arise from the sinuses of Valsalva just above the aortic valve.
- **Right coronary artery (RCA)** arises from the anterior sinus and supplies

1. Right atrium.
2. Greater part of Right ventricle.
3. Posterior region of inter ventricular septum
4. Conduction system in 40% of the patients
5. Small part of left ventricle adjacent to posterior inter ventricular groove.


Superior vena cava
Aortic arch
Pulmonary trunk (divided)
Left coronary artery
Circumflex artery
Great cardiac vein
Anterior interventricular artery
Right coronary artery
Marginal artery
Inferior vena cava
(a)
Aortic arch
Superior vena cava
Great cardiac vein
Circumflex artery
Coronary sinus
Posterior cardiac vein
Right coronary artery
Middle cardiac vein
Posterior interventricular artery
(b)

- **Left coronary artery (LCA)** arises from left posterior sinus, divides into the left anterior descending artery and circumflex artery. It supplies

1. Left atrium.
2. Greater part of left ventricle.
3. Anterior region of interventricular septum
4. Small part of right ventricle adjacent to anterior inter ventricular groove.
5. Part of left branch of AV bundle.

PATHOPHYSIOLOGY OF STEMI

- STEMI occurs due to sudden decrease in coronary blood flow after thrombotic occlusion of coronary artery.
- STEMI occurs when coronary artery thrombus develops quickly at the site of vascular injury produced by various factors such as smoking, hypertension, accumulation of lipid.
- In many cases atherosclerotic plaque surface will be disrupted which leads to thrombogenesis by activation of coagulation cascade, generation of thromboxane A2, activation of platelets.
- Other condition that can cause STEMI are

1. Severe coronary vasospasm.

2. Coronary artery embolisation.

3. Spontaneous coronary dissection.

- These conditions should be considered in a patient whose clinical findings suggest a process other than acute plaque rupture.
- The factors responsible for myocardial damage are territory supplied by culprit vessel, duration of occlusion, percentage of occlusion, blood supplied by collaterals, myocardial oxygen demand,

PATHOPHYSIOLOGY OF NSTEMI

- Pathogenesis of NSTEMI four processs:

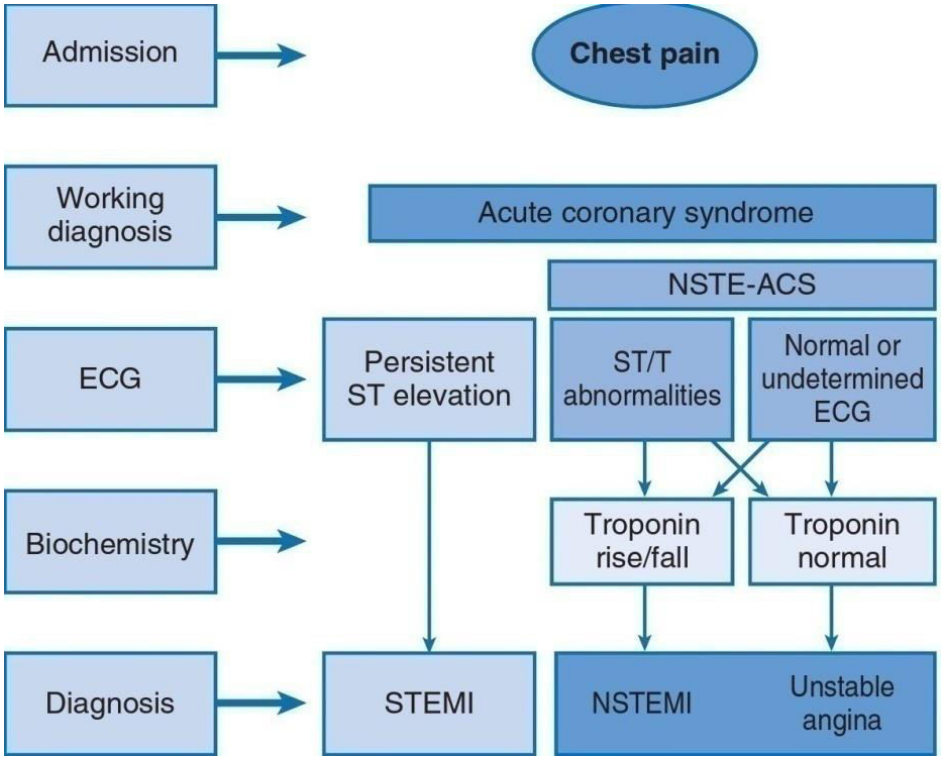
1. Rupture of unstable atheromatous plaque.

2. Vasoconstriction of coronary artery.

3. Imbalance between myocardial oxygen supply and demand

4. Gradual intra luminal narrowing of epicardial coronary artery

- Plaque ruptue or erosion leads to formation of superimposed thrombus along with impaired myocardial perfusion and vasoconstriction leads to myocardial necrosis.
- Activation of coagulation cascade and platelet plays a important role in both STEMI and NTEMI.



SPECTRUM OF ACUTE CORONARY SYNDROME

ATHEROSCLEROSIS

- Endothelial cells activated by risk factors which include hyperlipoproteinemia, usually express adhesion and chemo attractant molecules, recruit inflammatory leucocytes such as monocytes and T lymphocytes. Extracellular lipid begins to accumulate in intima.
- Fibrofatty stage- monocyte recruited to artery wall becomes macrophages and express scavenger receptors that bind modified lipoproteins. Macrophages become lipid laden foam cells by engulfing modified lipoproteins. Leucocytes and resident vascular wall cells can secrete cytokines and growth factors that increase leucocyte migration and smooth muscle cell migration and proliferation.
- As lesion progress, inflammatory meditors causes expression of tissue factors, a potent pro-coagulant and matrix degrading proteinases that weaken fibrous cap of plaque.
- When fibrous cap rupture at the point of weakness,coagulation factors have access to thrombogenic tissue factors causing thrombosis and non occlusive plaque.

- If balance between prothrombotic and firinolytic mechanism prevailing at that particular region and at that particular time is unfavourable occlusive thrombus causing myocardial infarction.

FEATURES OF VULNERABLE PLAGUE:

Lipid rich core (> 30-40% of plague).

Fibrous cap covering the lipid rich core.

Thikness of plague<100 μm.

Many macrophages.

Few smooth muscle cells.

Outward remodelling preserving the lumen.

Neovascularisation from vasa vasorum.

Adventitial/perivascular inflammation.

CLINICAL FEATURES

- **Predisposing factors**

1. **50%** of the patients with STEMI usually have an identifiable precipitating factors and prodromal symptoms.
2. Unusually heavy exercise and emotional stress would precipitate STEMI.
3. Other precipitating factors are lung infections, hypoxemia, pulmonary embolism, hypoglycaemia, administration of ergot, serum sickness, sympathomimetics, allergy.
4. Onset of STEMI have a circadian periodicity, with peak incidence of events occurring in the morning, these early morning hours are associated with increase cortisol and catecholamines, aggregation of platelet. These circadian rhythm will be absent in patients on beta blockers and aspirin.

- **Symptoms**

1. Chest pain generally lasts more than half an hour, pain usually described as constricting, crushing, compressing or sensation of heaviness on chest.

2. Chest pain localised to retrosternal region and spread to both anterior chest wall commonly left side, pain radiates to left shoulder, neck, jaw and interscapular region.

- Nausea and vomiting can occur, due to vagal reflex activation, stimulation of left ventricular receptors. This symptoms are commonly seen inferior wall myocardial infarction. This symptoms can be confused with gastritis, peptic ulcer, cholecystitis.
- Other associated symptoms are dizziness, palpitations, cold perspiration, sense of impending doom.
- **ATYPICAL PRESENTATION**

1. Features of heart failure.
2. Classical angina pain.
3. Atypical pain location.
4. Sudden onset of mania and psychosis
5. Syncope.
6. Features resembling of stroke.
7. Apprehension and nervousness.

- Clinical features of NSTEMI resembles same as STEMI.

PHYSICAL EXAMINATION

- Physical examination is important in determining other sources of chest pain, assessing prognosis and establishing a baseline that will aid in early recognition of complications.
- The goal is to determine hemodynamic instability, presence of cardiogenic pulmonary edema, or mechanical complication such as papillary muscle dysfunction, free wall rupture, ventricular septal defect and to exclude other causes of chest pain.
- Physical examination should include intial assessment of vital signs,oxygenation, bilateral blood pressure and jugular venous pulse, bilateral crepitation for pulmonary edema, murmur or friction rub and gallop for mechanical complication and heart failure, and neurological examination.
- Fundus examination may provide underlying vascular status due to hypertension and diabetes. Abdomen examination may provide right heart failure features such as hepatomegaly and positive abdominojugular reflex. Examination of extremities will be useful in identifying peripheral vascular disease and cyanosis in severe LV failure.

DIAGNOSTIC TESTS

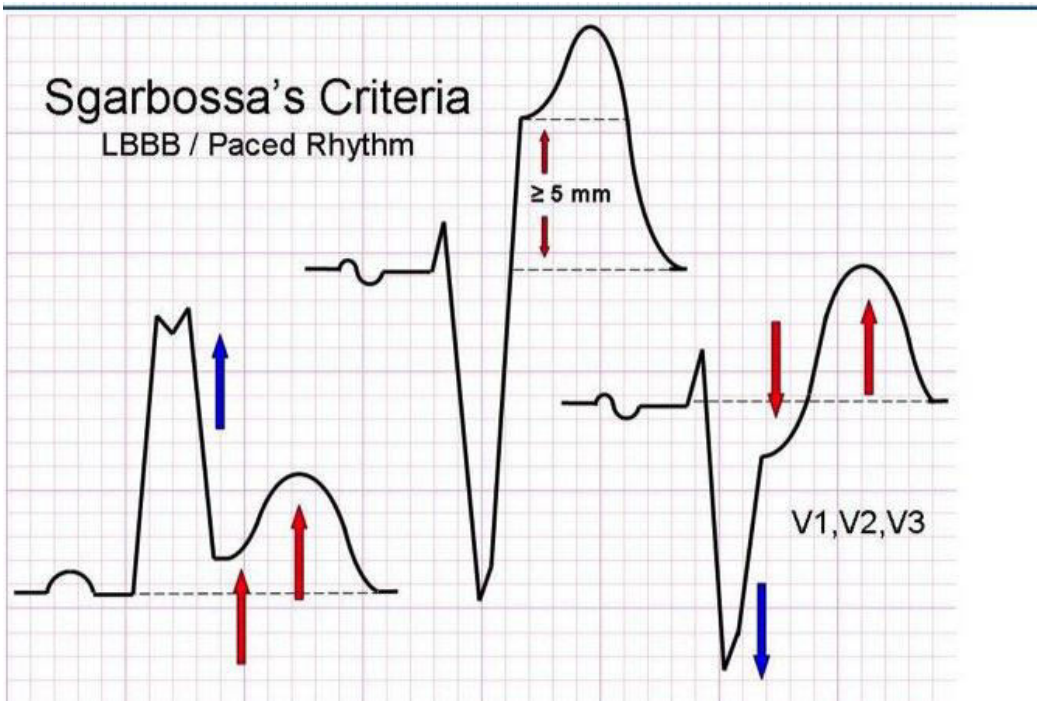
ELECTROCARDIOGRAM (ECG)

- The ECG should be performed and interpreted within 10 mins of presentation.
- Serial standard 12-lead ECG is extremely useful in detection and localisation of myocardial infarction
- Analysis of the constellation of ECG showing ST changes will be useful in identifying the site of occlusion in the infarct artery.
- In addition to diagnostic and prognostic information,serial 12-lead ECG monitoring will provide non invasive information about the success of reperfusion therapy.
- **ECG CRITERIA FOR DIAGNOSIS STEMI:**
    - New ST ELEVATION at J point in two or more contiguous leads with (in the absence of LBBB)
    - >1mm(.1Mv) in all leads except V2-V3.
    - In leads V2-V3 - >2mm(.2mv) in men > 40 years of age.

      >2.5mm(.25mv) in men <40 years of age.

      >1.5mm(.15mv) in women.

- In the setting of an old LBBB, acute myocardial infarction is diagnosed by Sgarbossa criteria
    1. ST-segment elevation >1mm and concordant with QRS complex-5 points.
    2. ST-segment depression >1mm in lead V1,V2,V3 -3 points.
    3. ST-segment elevation >5mm and discordant with QRS complex-2points.
- Score more than 3 has specificity of 98% for acute myocardial infarction.



- **ECG CRITERIA FOR DIAGNOSIS OF NSTEMI**

1. New ST- segment depression (downsloping or horizontal) >0.05mv in two contiguous leads.

2. T-wave inversion >.1mv in two contiguous leads with prominent R wave

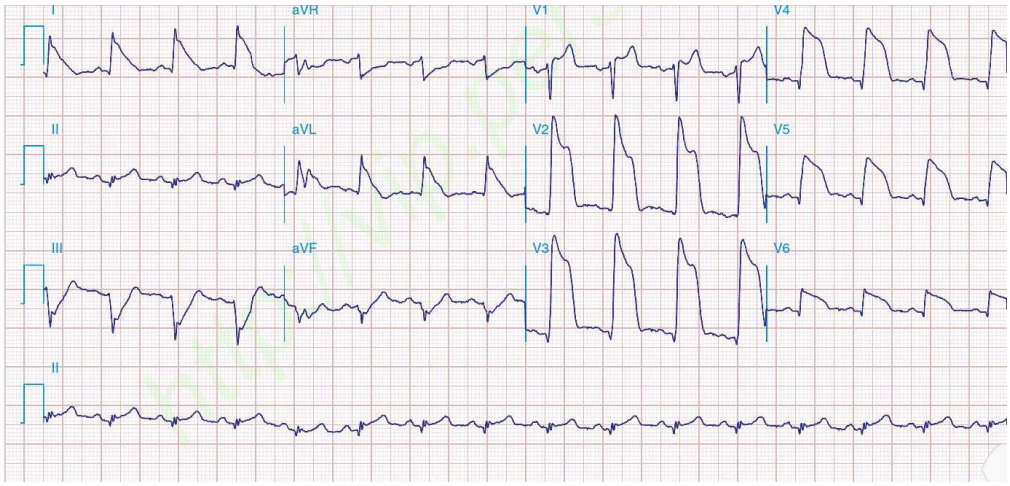
or with R/S ratio >1

ST Elevation, MI Location, and Possible Complications

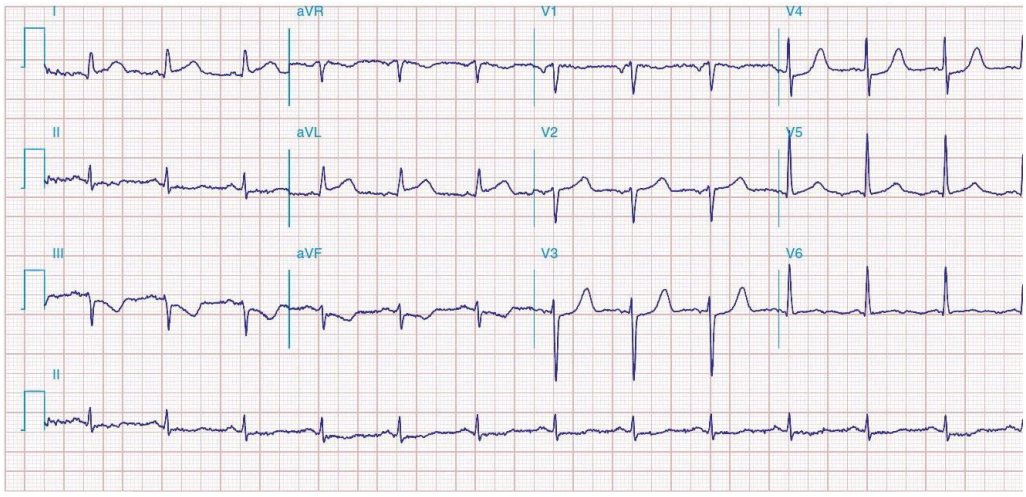
Leads with ST Elevation	Location of the MI	Infarct-Related Artery	Possible Complications
II, III, aVF	Inferior wall of the left ventricle	RCA in 85% to 90%	RCA = VT/VF, RVMI, bradyarrhythmias including sinus bradycardia, hypotension and AV block, LV dysfunction, postero-medial papillary muscle dysfunction, or rupture
		LCx in 10% to 15%	Dominant LCx: VT/VF, LV dysfunction, AV block but no RVMI or papillary muscle dysfunction
I and aVL	High lateral	LCx or first diagonal branch of LAD	VT/VF, LV dysfunction
V_1–V_4	Anteroseptal	LAD	VT-VF, extensive LV dysfunction, RBBB ± fascicular block, cardiogenic shock
V_5–V_6 + I, aVL	Lateral	LCx	VT/VF, LV dysfunction
ST depression in V_1–V_3 ± tall R waves	Posterior or straight posterior	LCx , RCA also possible	VT/VF, LV dysfunction
II, III, aVF + V_{3R}, V_{4R}, or V_{5R}	RVMI	Proximal RCA	VT/VF, AV block, bradycardia, hypotension, atrial infarction

AV, atrioventricular; LAD, left anterior descending; LCx, left circumflex; LV, left ventricular; PM, papillary muscle; RBBB, right bundle branch block; RCA, right coronary artery; RVMI, right ventricular MI; VT/VF, ventricular tachycardia/ventricular fibrillation.

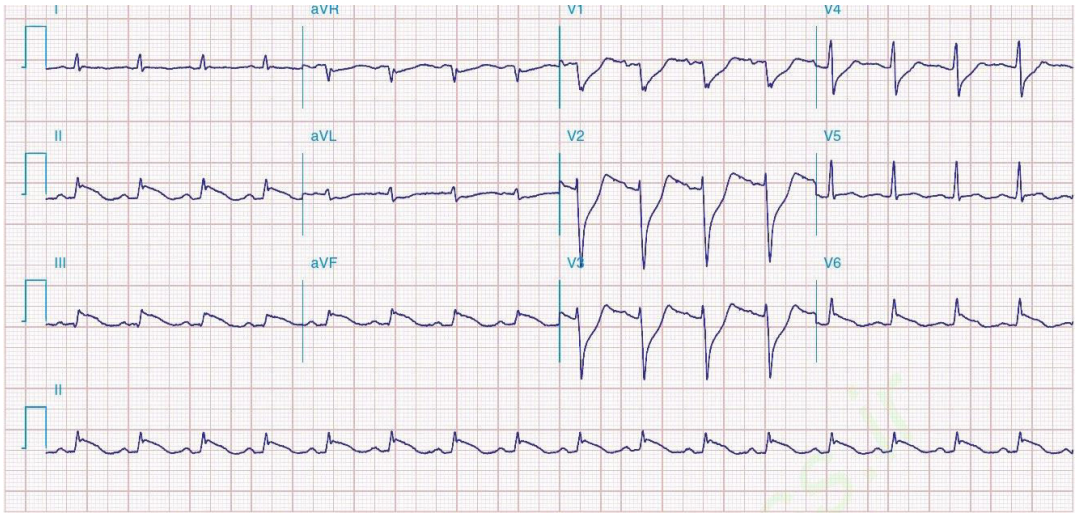
LOCALISATION OF MI.



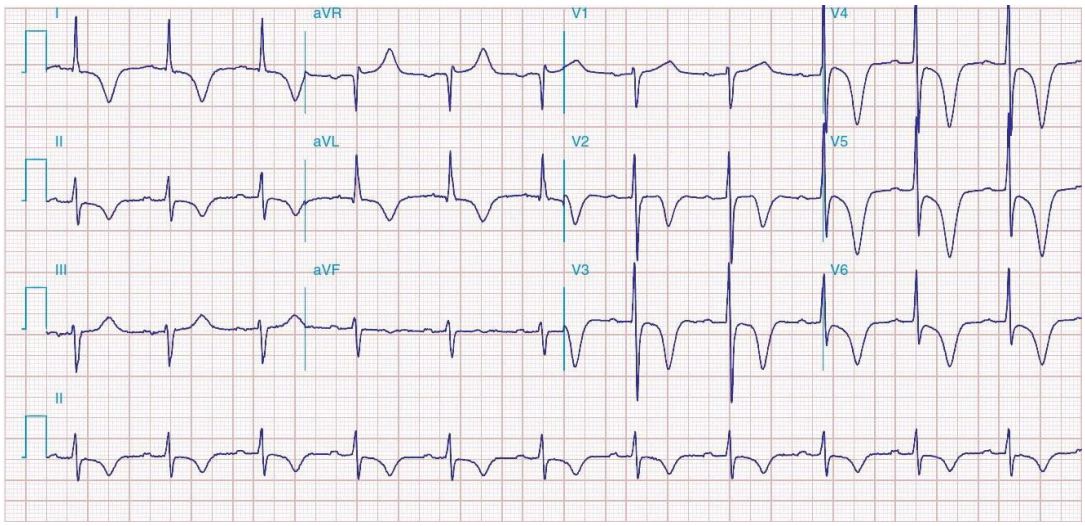
EXTENSIVE ANTERIOR WALL MI.



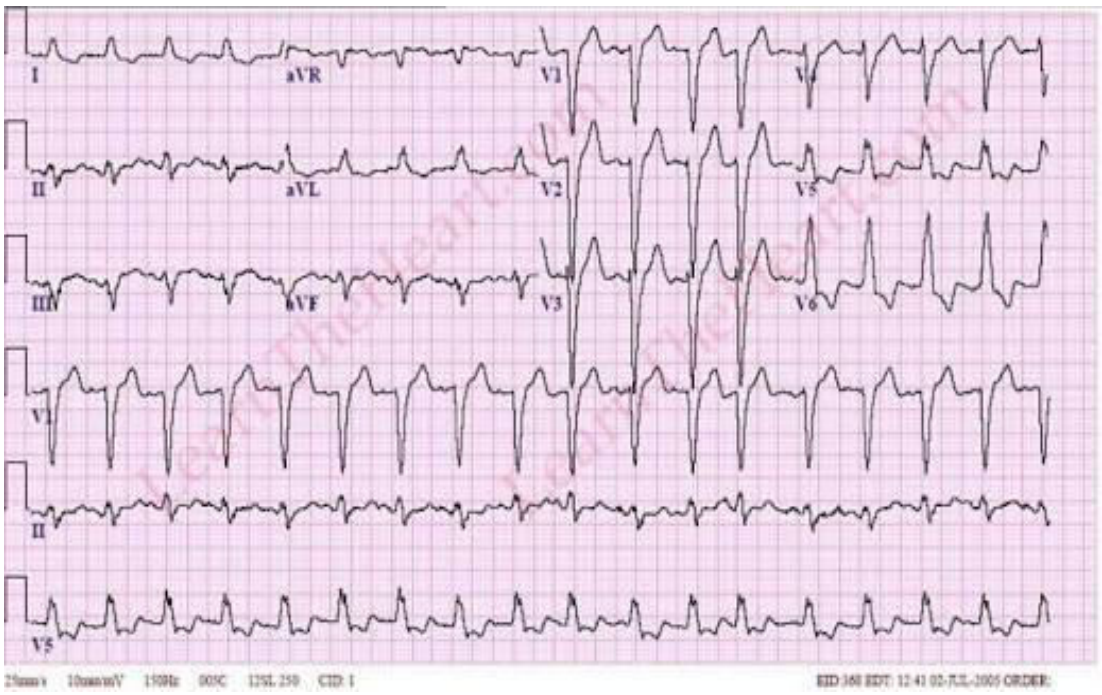
ACUTE LATERAL WALL MI



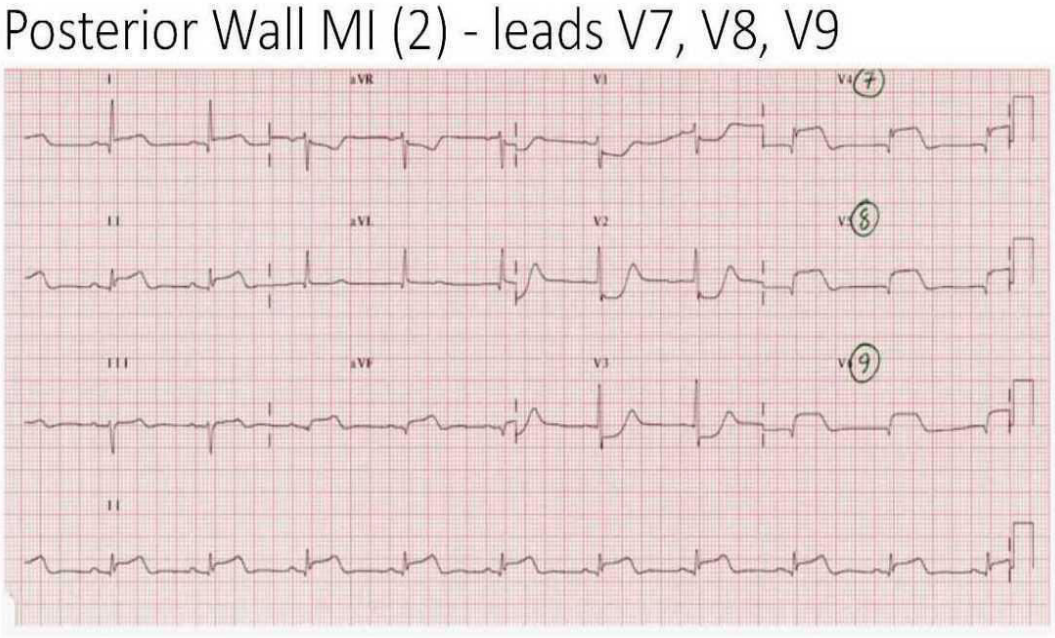
ACUTE INFERIOR WALL MI



NSTEMI OF ANTERIOR WALL



LEFT BUNDLE BRANCH BLOCK.



- Posterior myocardial infarction is an entity that is often unrecognized, it should be suspected in the patients with inferior of lateral wall myocardial infarction.
- The “reverse mirror test” is useful to identify posterior wall myovardial infarction, ST segment depression in leads V1to V3 is actually a ST elevation in posterior wall. The prominent R wave is actually represent posterior Q wave.
- Inferoposterior or inferolateral myocardial infarction involves the right coronary artery(RCA) or left circumflex obtuse marginal branch.
- There are condition that mimic electrocardiographic changes of myocardial infarction, it is termed as “pseudoinfarction”. They are

1. Ventricular hypertrophy.
2. Preexitation syndrome.
3. Primary myocardial disease.
4. Pneumothorax, Pulmonary embolism.
5. Primary and metastatic tumors of heart.
6. Amyloid disease, cardiac sarcoidisis.
7. Intracranial hemorrhage.
8. Hyperkalemia.
9. Pericarditis.

IMAGING

CHEST RADIOGRAPHY:

- A standard chest radiography (CXR) should be included in initial evaluation. Pulmonary edema in CXR us important prognostic and therapeutic implications. prominent vascular markings in CXR reflects elevated LV end diastolic pressure.

ECHOCARDIOGRAPHY:

- The evaluation of a patient with a non diagnostic ECG, Finding of echocardiography such as regional wall motion abnormality can support the diagnosis of myocardial infarction. It also useful in localising the territory at risk.
- LV function assessed in echocardiography correlates well with measurements in angiography are useful in establishing prognosis in myocardial infarction.
- Echocardiography can help in detecting in mechanical complication of MI such as mitral regurgitation, ventricular septal rupture, cardiac tamponade.

CARDIAC BIOMARKERS

Markers of myocardial necrosis:

Myocardial injury can be detected by presence of circulating proteins in serum which is released by damaged myocardium. Most commonly used serum markers are cardiac specific troponin and creatine kinase MB.

Cardiac specific troponins:

The preferred marker to detect myocardial injury is cardiac troponin which consists of three subunits that regulate calcium mediated contractile process of striated muscle. Troponin I binds to actin and inhibits interaction of actin-myosin. Troponin T binds to tropomysin thereby attaching the troponin complex to thin filaments. Only cardiac specific isoforms of troponin T(cTnT) and troponin I(cTnI) are exclusively expressed in cardiac myocyte.

In myocardial infarction, cardiac specific troponin T and I begin to rise 3 hours after onset of chest discomfort. It may persist for 7 to 10 days in myocardial infarction patients because of continuous proteolysis of contractile apparatus in necrotic myocardium.

Other causes of elevated troponin are myocarditis, pericarditis, cardiac contusion/trauma, aortic dissection, endocarditis, post cardiac arrest, pulmonary embolism, cardiac arrhythmia, sepsis, renal failure.

Creatine kinase MB:

Creatine kinase MB can be used as a alternative assay in the absence of cardiac troponin assay. Even improve test system for quantitative determination of creatine kinase MB based on immunological determination did not substantially increase the sensitivity for detection of minor myocardial injury.

Other biomarkers:

C-reactive proteins and BNP assay can be used for risk stratification but there is no clear guidance available for specific therapeutic maneuvers in the setting of myocardial infarction in response to these biomarkers.

Other laboratory investigations:

Serum lipid profile should be done in all patients with acute myocardial infarction within 24hours of symptoms. Ratio of total cholesterol to HDL cholesterol is no longer used for risk assessment.

Elevation in WBC count usually occurs after 2 hours of chest pain, reaches the peak 2 to 4 days after infarct and returns to normal range in 7 days. increased risk of adverse outcome are seen in patients with higher WBC count in patients with acute MI. The haemoglobin value at time of admission with myocardial infarction independently predicts major cardiovascular events.

RISK STRATIFICATION :

Whereas risk factors for the development of atherothrombosis provides in insights into disease mechanism and the opportunity for primary and secondary prevention therapy, analysis of risk for adverse outcome after presentation is important in guiding management and therapeutic decisions.

Analysis usually uses a combination of various clinical, ECG and biochemical parameter. Five simple baseline parameter can be used to predict 30-day mortality more than 90% of patients:

1. Age.
2. Systolic blood pressure.
3. Killip class.
4. Heart rate.
5. Localisation of infarct.

KILLIP CLASSIFICATION

KILLIP class was proposed by Thomas killip III and John Kimball in 1967 involved bedside stratification. This risk stratification was based on bedside clinical examination in patients with acute myocardial infarction and it is used to identify who are at highest risk of death and potential benefits of specialised care in intensive coronary care unit.[20]

- **Killip I**: with no clinical signs of heart failure.
- **Killip II**: with rales in the lungs, third heart sound (S3), and elevated jugular venous pressure
- **Killip III**: with acute pulmonary edema (APE).
- **Killip IV**: with cardiogenic shock or arterial hypotension (measured as systolic blood pressure < 90 mmHg), and evidence of peripheral vasoconstriction (oliguria, cyanosis, and diaphoresis).

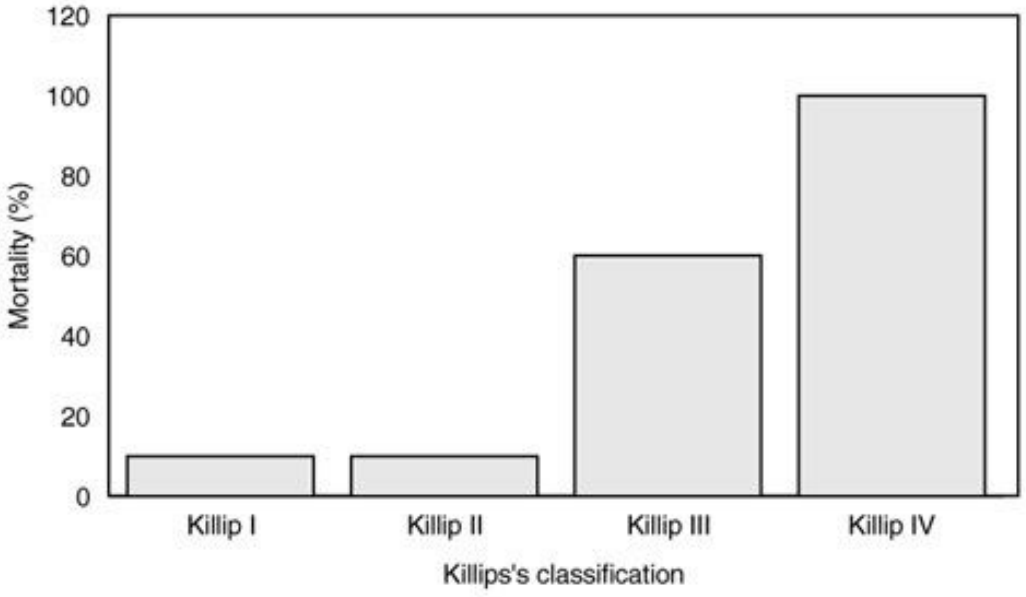
  The 30 day mortality rate of killip class are

  Killip I: 6% of mortality.

  Killip II: 17% of mortality.

  Killip III: 38% of mortality.

  Killip IV: 81% of mortality.[20]



The other classification used in acute myocardial infarction are

- Simplified forrester & Diamond hemodynamic classification(STEMI).
- The GRACE risk score for Acute Coronary syndrome (not STEMI alone).
- TIMI(The Thrombolysis In Myocardial Infarction) score.

Simplified Forrester & Diamond hemodynamic classification in STEMI		
Class 1	No hypotension	No pulmonary congestion
Class 2	No hypotension	Pulmonary congestion
Class 3*	Hypotension	No pulmonary congestion
Class 4 *	Hypotension	Pulmonary congestion

SIMPLIFIED FORRESTER CLASSIFICATION

TIMI Risk Score for STEMI

Historical	
Age 65-74	2 points
≥ 75	3 points
DM/HTN or angina	1 point
Exam	
SBP < 100	3 points
HR >100	2 points
Killip II-IV	2 points
Weight < 67 kg	1 point
Presentation	
Anterior STE or LBBB	1 point
Time to rx > 4 hrs	1 point
Risk Score = Total	(0 -14)

Risk Score	Odds of death by 30D*	
0	0.1	(0.1-0.2)
1	0.3	(0.2-0.3)
2	0.4	(0.3-0.5)
3	0.7	(0.6-0.9)
4	1.2	(1.0-1.5)
5	2.2	(1.9-2.6)
6	3.0	(2.5-3.6)
7	4.8	(3.8-6.1)
8	5.8	(4.2-7.8)
>8	8.8	(6.3-12)

*referenced to average mortality
(95% confidence intervals)

TIMI SCORE.

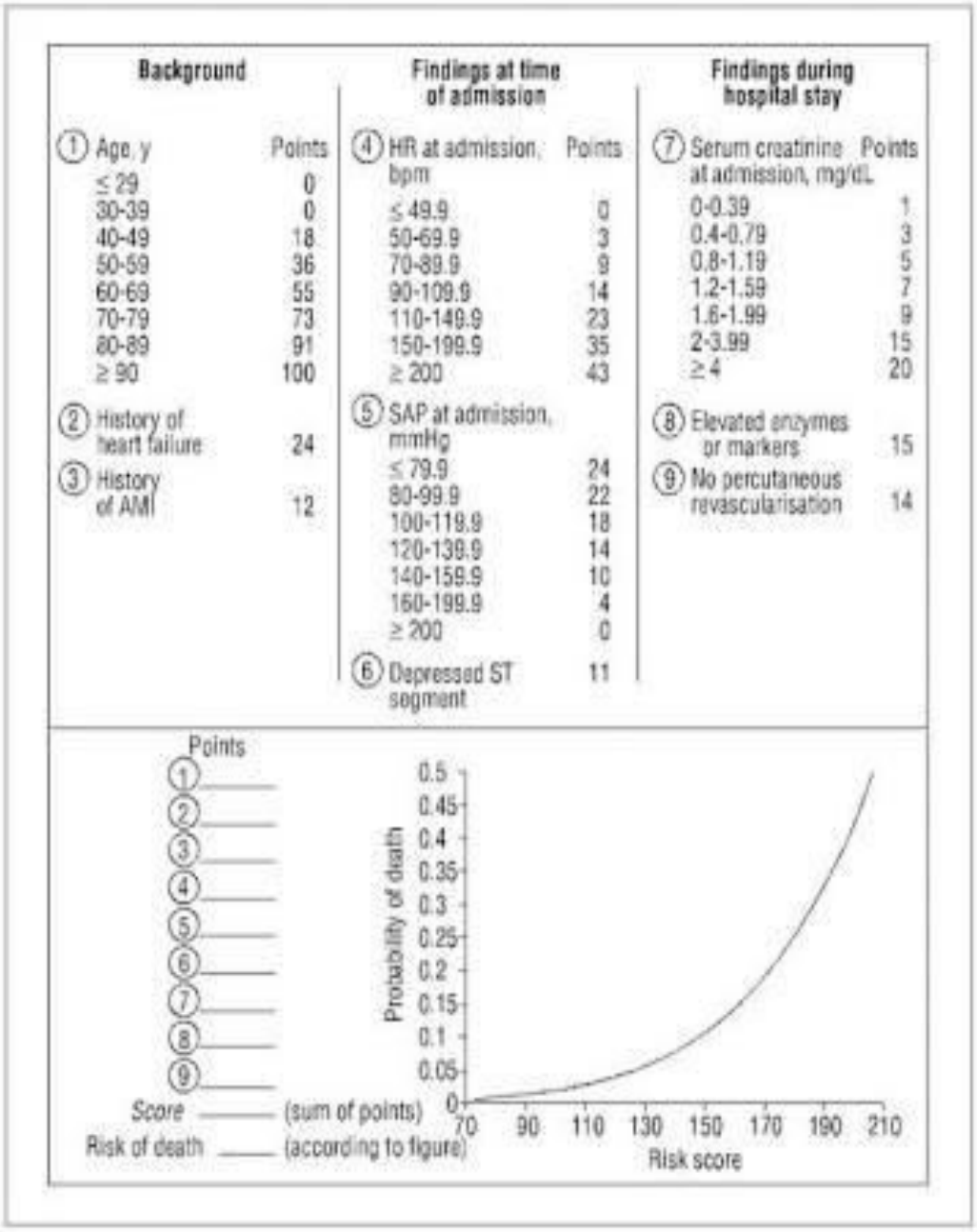
The Grace risk score for ACS:

GRACE score calcultes mortality rate in hospital and 6 month in acute coronary syndrome.

It uses data like

- Enzyme elevation.
- Pci done/ not.
- Serum creatinine.
- Killip class.
- ST depression.

Risk category (tertile)	GRACE risk score	In-hospital death (%)
Low	≤108	<1
Intermediate	109–140	1–3
High	>140	>3
Risk category (tertile)	**GRACE risk score**	**Post-discharge to 6-month death (%)**
Low	≤88	<3
Intermediate	89–118	3-8
High	>118	>8


Background
Findings at time of admission
Findings during hospital stay
1 Age, y
Points
≤ 29 0
30-39 0
40-49 18
50-59 36
60-69 55
70-79 73
80-89 91
≥ 90 100
2 History of heart failure 24
3 History of AMI 12
4 HR at admission, bpm
Points
≤ 49.9 0
50-69.9 3
70-89.9 9
90-109.9 14
110-149.9 23
150-199.9 35
≥ 200 43
5 SAP at admission, mmHg
≤ 79.9 24
80-99.9 22
100-119.9 18
120-139.9 14
140-159.9 10
160-199.9 4
≥ 200 0
6 Depressed ST segment 11
7 Serum creatinine at admission, mg/dL
Points
0-0.39 1
0.4-0.79 3
0.8-1.19 5
1.2-1.59 7
1.6-1.99 9
2-3.99 15
≥ 4 20
8 Elevated enzymes or markers 15
9 No percutaneous revascularisation 14
Points
1
2
3
4
5
6
7
8
9
Score (sum of points)
Risk of death (according to figure)
Probability of death
0.5
0.45
0.4
0.35
0.3
0.25
0.2
0.15
0.1
0.05
0
70 90 110 130 150 170 190 210
Risk score

INFARCT LOCATION

- Prognosis of myocardial infarction is also related to extent of myocardium at risk and also site of coronary occlusion.the ECG reflects the infarct location(TABLE 2). Patients with left main stem occlusion rarely reaches the hospital for reperfusion therapy.
- Occlusion of proximal left anterior descending artery proximal to first septal branch is assocated with high early and late mortality (window- maker).
- Large inferior wall myocardial infarction result of dominant right coronary artey occlusion are also a high risk especially when right ventricle is involved.
- Other location such as apical due to distal left anterior descending artery, lateral myocardial infarction due to diagonal branch occlusion or small inferior wall infarction due to distal right or circumflex occlusion have better outcome.
- Strictly posterior wall myocardial infarction may escape routine ECG leads or evident only by st depression in V1- V4 usually have a good outcome.

TREATMENT

Treatment of STEMI:

PRE HOSPITAL CARE:

In STEMI, pre hospital care has a very important role because many deaths occur within one hour of onset that usually results from ventricular fibrilliation. So immediate of resuscitative action and quick transportation to hospital is very important. Emergency medical service system should have three components: emergency medical dispatch, first response and EMS ambulance service. The EMS should have expanded capability to record a pre hospital 12- lead ECG.

GENERAL TREATMENT:

Aspirin:

Aspirin is very effective in all acute coronary syndrome. It is initial management strategy for all patients with suspected STEMI. In order to achieve therapeutic blood level,162 to 325 mg tablet should be chewed to increase buccal absorption.

Pain management:

Most commonly used analgesics in myocardial infarction are morphine, pentazocine, mepiridine. The ideal dose of morphine in STEMI is 4 to 8mg in intravenous route repeated at interval of 5 to 15 minutes. It also has beneficial effect in acute pulmonary edema because of peripheral arterial and venous dilatation.

Nitrates:

Their ability to enhance blood flow of coronaries and to decrease preload by venodilation, sublingual nitrates are indicated in acute coronary syndrome once hypotension and right ventricular infarct were ruled out.

Beta blockers:

This drug reduces the pain, prevent life threatening arrhythmia, and also reduces infarct size. Beta blockers are contraindicated in patients with hypotension, bradycardia or patients with significant heart block. Metoprolol is common drug used in this category.

Oxygen:

Hypoxemia is common in STEMI due to ventilation-perfusion abnormality as a sequelae of left ventricular failure. So patients should be treated with oxygen for a period of 24 to 48 hours. Patients with severe pulmonary edema may need endotracheal intubation and mechanical ventilation in order to correct hypoxemia.

Reperfusion therapy:

Fibrinolysis:

It recanalizes the coronary artery thrombotic occlusion, thereby it resore the coronary flow and reduces infarct size. Fibrinolysis improves both short term and long term survival.

Assessment of reperfusion done by TIMI flow grade, TIMI frame count, electrocardiogram, echocardiography.

TIMI flow grade-when assessed after 60 to 90 minutes after fibrinolytic therapy TIMI grade 3 has lowest mortality and grade 0 and 1 has highest mortality.

In ECG, resolution of ST segment is a strong predictor of positive outcome.

FIBRINOLYTIC AGENT	DOSE	FIBRIN SPECIFICITY	FIBRINOGEN DEPLETION	ANTIGENIC	PATENCY RATE (90-MIN TIMI 2 OR 3 FLOW)
Fibrin Specific					
Tenecteplase (TNK)	Single IV weight-based bolus†	++++	Minimal	No	85%
Reteplase (r-PA)	10 units + 10-unit IV boluses given 30 min apart	++	Moderate	No	84%
Alteplase (t-PA)	90-min weight-based infusion‡	++	Mild	No	73-84%
Non–Fibrin Specific					
Streptokinase§	1.5 million units IV given over 30-60 min	No	Marked	Yes¶	60-68%

CONTRAINDICATION FOR FIBRINOLYTICS

Absolute contraindication are

- Previous intracranial hemorrhage (ICH).
- History of structural cerebral vascular lesion.
- History of intracranial malignancy.
- Ischemic stroke within 3 month of period.
- Suspicion of aortic dissection.
- Active hemorrhage or bleeding diathesis (excluding menses).
- History of head injury or facial injury within 3 months.
- Recent Intracranial, intraspinal surgery within 2 months.
- Severe uncontrolled hypertension ,not responding to emergency management.
- For streptokinase, prior treatment in previous 6 months.

Relative contraindication are

- History of intracranial pathology.
- Previous ischaemic stroke > 3 months.
- Recent history of trauma.
- Prolonged CPR more than 10 minutes.
- Major surgery < 3 weeks.
- Active peptic ulcer disease.

- Recent internal bleeding within 2 to 4 weeks.
- Systolic BP >180, Diastolic > 110mm Hg.
- History of severe or poorly controlled blood pressure.
- Dementia.
- History of non compressible vascular puncture such as IJV and subclavian lines.

Percutaneous coronary intervention(PCI):

Current recommendations according to ACC/AHA guidelines in STEMI:

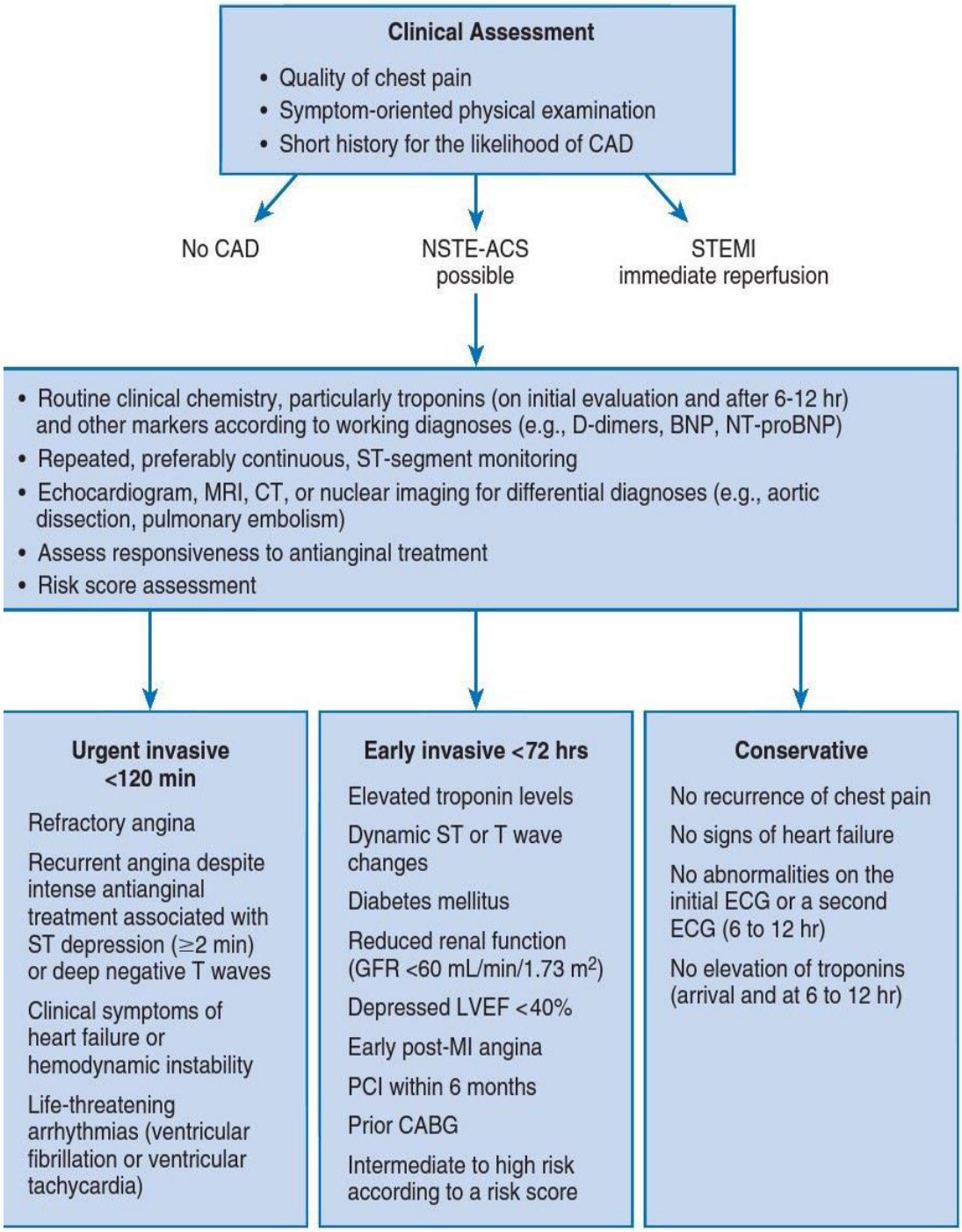
Primary PCI

- symptoms of STEMI within 12 hours.
- Severe heart failure and cardiogenic shock
- Contraindications for thrombolysis with symptoms of STEMI

Delayed PCI:

- Fibrinolytic failure
- Hemodynamically stable stenosis after 24 hours of symptoms.

Treatment of NSTEMI:



Complications of myocardial infarction:

Complication of acute myocardial infarction include mechanical, ischemic, arrhythmic, embolic and inflammatory.

Circulatory failure, one of the mechanical complications is the mos common cause of death in acute myocardial infarction.

Mechanical complications:

- Cardiac failure.
- Cardiogenic shock.
- Mitral valve regurgitation.
- Papillary muscle dysfunction.
- Ventricular septal rupture.
- Free wall rupture.

Ischemic:

- Angina.
- Reinfarction.
- Extension of infarct**.**

Arrhythmic complication:

- Atrial and ventricular arrhythmia .
- Sino nodal or AV nodal dysfunction.

Embolic complication:

- Peripheral and central nervous system embolism.

Inflammatory:

- Pericarditis.
- Dressler’s syndrome.

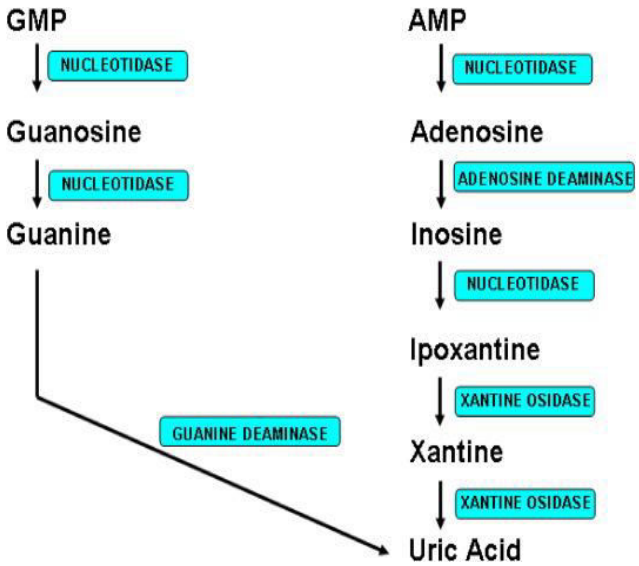
SERUM URIC ACID IN ACUTE MYOCARDIAL INFARCTION

Uric acid-production and metabolism:

- Uric acid production and metabolism is a complex processes involving various factors that regulate hepatic production, as well as renal and gut excretion of this compound.
- Uric acid is the end product of exogenous and endogenous purine metabolism.[19]
- The exogenous pool varies significantly with diet, and animal proteins contribute significantly to purine pool
- The endogenous production of uric acid is mainly produced from the liver, intestines and other tissues like muscles,kidneys and the vascular endothelium.
- The chemical formula of Uric acid is **C5H4N4O3 (7,9-dihydro-1H-purine-2,6,8(3H)-trione)**
- Molecular weight of uric acid is 168 Da.[19]
- Various enzymes are involved in the conversion of the two purine nucleic acids, adenine and guanine, to uric acid.
- First step, adenosinemonophosphate (AMP) is converted to inosine by two different mechanisms; either by removal of an amino group

by deaminase to form inosine monophosphate (IMP) then followed by dephosphorylation with nucleotidase to form inosine, or by removal of a phosphate group by nucleotidase to form adenosine then followed by deamination to form inosine.

- Guanine monophosphate (GMP) is converted to guanosine by the enzyme nucleotidase.
- The nucleosides, inosine and guanosine, then converted to purine base hypoxanthine and guanine, respectively by enzyme purine nucleoside phosphorylase (PNP).
- Hypoxanthine is then oxidized to form xanthine by enzyme xanthine-oxidase(XO), and guanine is deaminated to form xanthine by the enzyme guanine deaminase.
- Xanthine is again oxidized by xanthine oxidase to form the final product, uric acid.



- At physiologic pH, uric acid is a weak acid with pKα of 5.8. Uric acid exists majorly as urate, which is the salt of uric acid. When urate concentration increases in blood, uric acid crystal formation increases.
- The normal value of uric acid in human blood is 1.5 to 6.0mg/dL in women and 2.5 to 7.0 mg/dL in men.
- The solubility of uric acid in water is low, and in humans, the average concentration of uric acid in blood is close to the solubility limit (6.8 mg/dL). When the level of uric acid is higher than 6.8 mg/dL, crystals of uric acid form as monosodium urate (MSU).[19]
- Uric acid concentration can be measured in serum, plasma, urine and also in exhaled breath condensate.
- Various methods are

1. phosphotungistic acidmethods (PTA).
2. Uricase methods.
3. High-performance liquid chromatography methods.
4. Dry chemistry systems and biosensor methods.

- The production and catabolism of purines are relatively constant between 300 and 400 mg per day. Two third of uric acid is excreted by kidney and remaining one third is excreted by gastrointestinal tract.

URIC ACID AND CARDIOVASCULAR DISEASE

An association between high uric acid and cardiovascular disease has been reported in the 19th century itself. Since then number of studies have been conducted and supported this relationship and considered uric acid as an independent risk factor for cardiovascular events, such as coronary vascular disease, cerebrovascular disease, and congestive heart failure in high risk population (subjects with diabetes mellitus, hypertension, hyperlipidemia) However, the importance of a link between high uric acid and cardiovascular events in the general population still remains to be clarified.

Chen JH, Chuang SY, Chen HJ, Yeh WT, Pan WH et al repored a study involving a large group of patients with hypertension and/or diabetes, a serum uric acid level more than 7 mg/dl was associated with increased cardiovascular mortality[12].

Hyperuricemia is also significantly correlated with an increased mortality rate in patients with congestive heart failure in some studies.[11]

URIC ACID IN ACUTE MYOCARDIAL INFARCTION:

Many studies were conducted in various places to prove the association of uric acid in acute myocardial infarction in predicting mortality and severity. Some studies have shown close relationship between serum uric acid and killip class in acute myocardial infarction.

1. C.-W. Liu et al. / International Journal of Cardiology 226 (2017) Relationship of serum uric acid and Killip class on mortality after acute ST-segment elevation myocardial infarction and primary percutaneous coronary intervention.Hyperuricemia increased the1-year mortalityof STEMI patients in Killip class I, but not of patients in Killip classes II–IV. An interaction of hyperuricemia and Killip class significantly affects the mortality of STEMI patient.
2. Study of serum uric acid level in patients of acute myocardial infarction 1Dr.Anil Katdare, 2 Dr.A.L.Kakrani, 3Dr.Sridevi, 4 Dr.Vivek Vilas Manade. This hospital based case study was performed in the parent institute. A total of 75cases of Acute MI were studied. Mean SUA for discharged patients was 4.67 ±1.95 /dl and it was 7.1±1.45 mg/dl for the patients who died in the hospital. There was correlation between serum uric acid level after acute myocardial infarction and age and body mass index.

The causes of increased serum uric acid level in acute myocardial infarction:

- Uric acid are produced in endothelium by rapid degradation of adenosine which is synthesized in vascular smooth muscle, then undergo rapid efflux to the vascular lumen due to low intracellular pH and negative membrane potential.
- The activity of xanthine oxidase and production of uric acid is increased in ischaemic condition hence elevated uric acid can be used as a marker of tissue ischaemia.[10]
- Hyperuricaemia is also associated with some harmful effects on endothelial dysfunction, oxidative metabolism, platelet adhesiveness, haemorheology, and aggregation.
- Some study suggest that uric acid may also have a negative effect on cardiovascular disease by causing inflammation, which is clearly involved in the pathogenesis of cardiovascular disease.
- Uric acid, as a general marker of cell death and hyperuricemia is associated with obesity, dyslipidemia, hypertension, insulin resistance, male gender, aging, menopause, excessive alcohol intake and diuretic use.
- Elevated Uric acid level reflects increased xanthine oxidase pathway activity, which has the ability to contribute in the progression of left ventricular dysfunction by interfering with myocardial energetics and myofilament calcium sensitivity.

SUMMARY

Acute myocardial infarction is life threatening condition. Prompt action is essential as it has very high mortality in hospital and long term mortality and morbidity. So risk stratification forms the crux of management protocol since physicians has to know which patients are likely to develop serious and life threatening complications.

Killip class is a bed side assessment test which is useful in predicting mortality in acute myocardial infarction. Hyperuricemia is associated in increased cardiovascular mortality in high risk patients. There are few studies which states the association between killip class and uric acid level.So our goal is to find out the any quantal relationship between killip class and serum uric acid in acute myocardial infarction in our population.

MATERIALS AND METHODS

STUDY DESIGN:

Cross sectional study (Descriptive)

STUDY PERIOD:

Data collection done for a period of 6 months between april 2017 to september 2017

PLACE OF STUDY:

Govt.Kilpauk Medical College and hospital, Chennai-10.

STUDY POPULATION:

Patients >18 years of age with STEMI or non-ST segment elevation MI (NSTEMI) on the basis of history, clinical examination, electrocardiographical changes and biochemical markers.

SAMPLE SIZE : 70

INCLUSION CRITERIA :

Patients >18 years of age with -STEMI or non-ST segment elevation MI (NSTEMI) on the basis of history, clinical examination, electrocardiographical changes and biochemical markers.

1. History (resting chest pain lasting more than30 min)
2. Electrocardiographical changes
    - New or presumed new significant ST-segment T-wave (ST-T) changes or new left bundle branch block (LBBB),Development of pathologic Q waves in the electrocardiogram(ECG)
3. Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality
4. Biochemical markers:
    - Rise of serum cardiac enzymes concentration (CK-MB and Troponins).

EXCLUSION CRITERIA :

- Patients with a condition known to elevate SUA level (eg, chronic kidney disease, gout, hematological malignancy, hypothyroidism, hyperparathyroidism)
- Patients taking drugs that increase SUA

  salicylates [>2 g/day], ethambutol, amiloride, bumetanide, chlorthalidone, cisplatin, cyclophosphamide, cyclosporine, ethacrynic acid, thiazide diuretics, furosemide, indapamide, isotretinoin, ketoconazole, levodopa, metolazone, pentamidine, phencyclidine, pyrazinamide, theophylline, vincristine or vitamin C.

METHODOLOGY

- Patients more than 18 years of age diagnosed to have acute MI who presented to hospital within 24 hours of onset of symptoms were included in the Study.
- Patient with increased myocardial enzyme concentrations with typical chest pain persisting more than 30 minutes with
    1. electrocardiographic changes (including ischemic ST-segment depression, ST-segment elevation or pathologic Q waves).
    2. Increased enzyme concentrations were defined as (Creatine kinase,Troponin) peak level more than 2 times upper limit of normal.
- Complete history taking and physical examinations was done, patient with exclusion criteria was identified and excluded in the study.After getting informed consent from the patient, they were included in study.
- The data of each patient will be collected in a special proforma,which includes patient's name, age, sex,demographic details and presenting complaints.

- Blood pressure,random sugar,urea,creatinine will be taken immediately after admission,killip classification applied at the time of admissions.
- Baseline Serum Uric acid level will be done by withdrawing 4ml of blood.uric acid level in serum is measured by uricase method with COBRA INTEGRA/COBAS C SYSTEM.
- Cardiac enzyme assay were done in patients with NSTEMI.
- Reference level for uric acid

  Male -3.1-7.0 mg/dl

  Female- 2.5 to 5.6 mg/dl
- The data of each patient will be collected in specific proforma(ANNEXURE 2) which includes patient's name, age, sex, demographic details, presenting complaints, risk factors and all clinical data.
- All the relevant data and values are then entered in master chart in Microsoft excel format an then analyzed statiscially.

STATISTICAL ANALYSIS

The data was collected in the master chart obtained in the Microsoft excel format.

The collected was analysed with SPSS 16.0 version. To describe about the data descriptive statistics frequency analysis, percentage analysis were used for categorical variable and the mean were used for continuous variable. To find the significant of two variables by unpaired t test. To find the correlation between the two variables kruskal wallis test were used with p value less than .05 is considered as significant.

RESULTS

The total patients recruited in our study were 70. The following charts depicit frequency distributions.

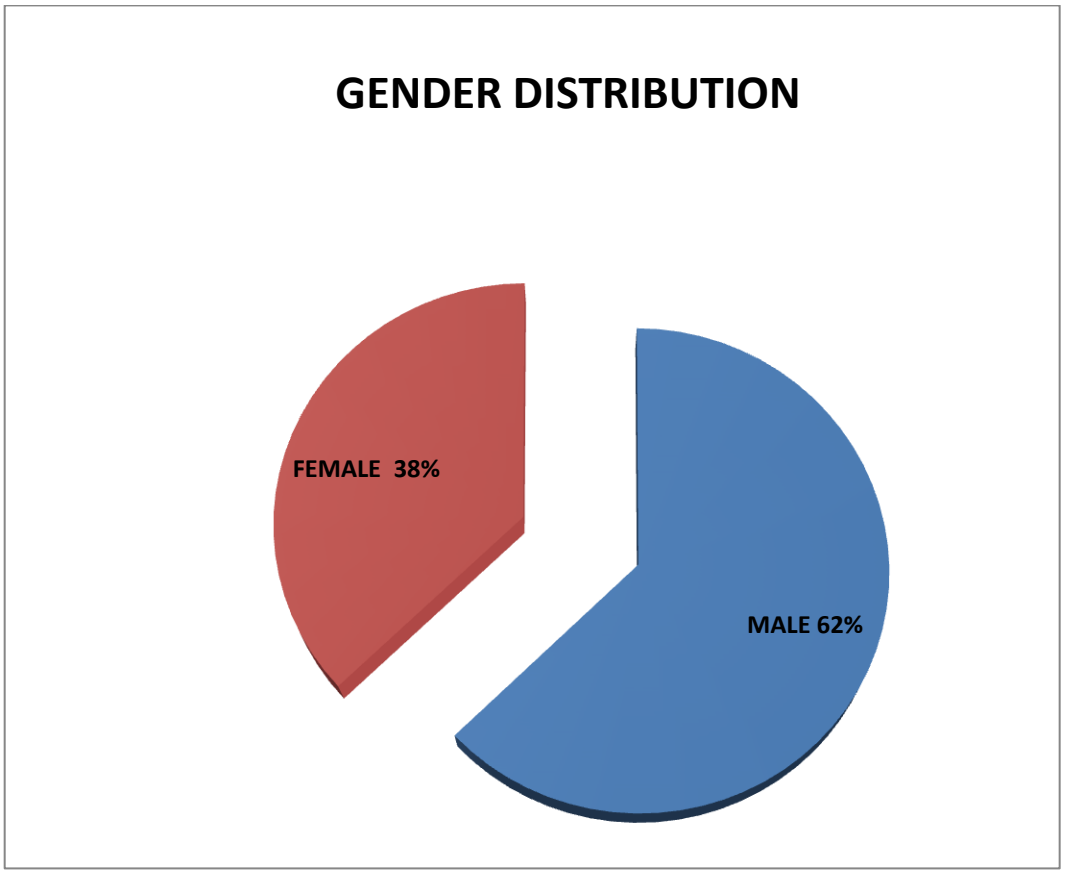
These are the frequency distributions of various variable used in our study.

GENDER:

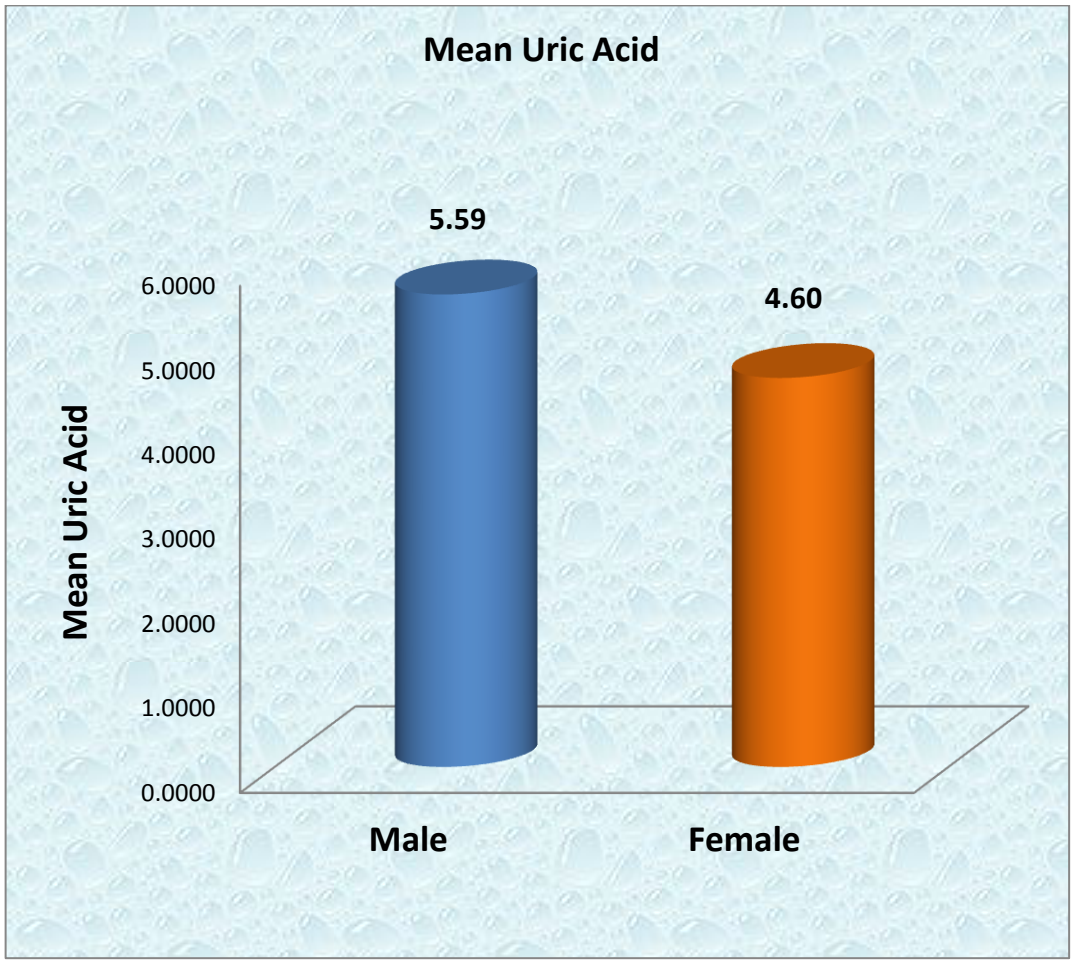
In a total of 70 patients participated in our study 44 patients were male and 26 patients were females. This distribution shows the predominance of males in acute myocardial infarction.

GENDER

	Frequency	Percent
F	26	37.1
M	44	62.9
Total	70	100.0


GENDER DISTRIBUTION
FEMALE 38%
MALE 62%

ASSOCIATION BETWEEN SERUM URIC ACID AND GENDER:



Mean uric acid levels was more among males (5.59) and less among females (4.6). This difference is statistically significant by unpaired t test.(**p=0.004**)

AGE DISTRIBUTION:

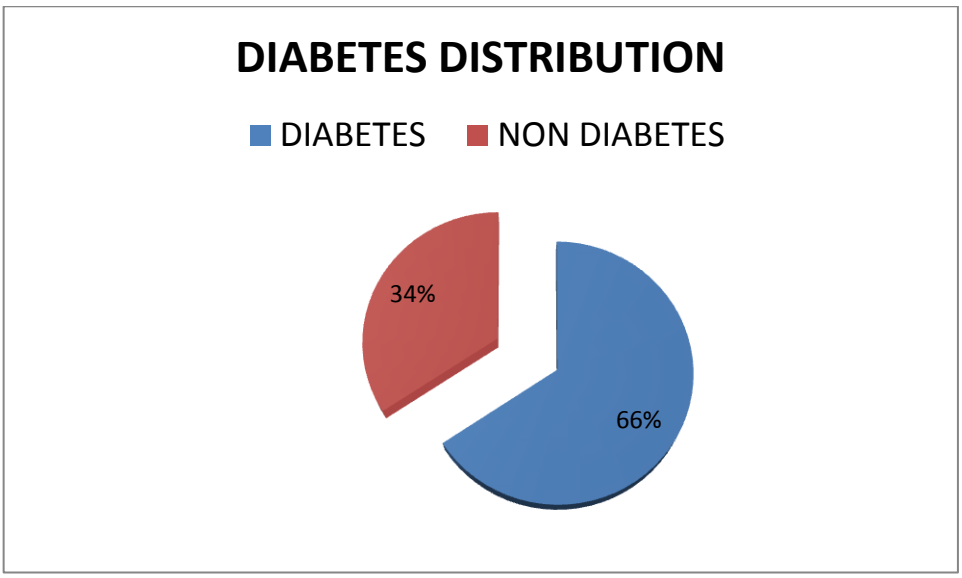
	N	Minimum	Maximum	Mean	Std. Deviation
AGE	70	35	69	53.77	8.054
Valid N (listwise)	70				

The mean age of the patients in our study is 54years. The minimum age of patient is 35years in our study.

DISTRIBUTION OF DIABETES:

In a total of 70 patients,46 patients were diabetes and 24 were non diabetic. It clearly shows acute myocardial infarction are common in diabetes patients.

	Frequency	Percent	Valid Percent	Cumulative Percent
N	24	34.3	34.3	34.3
Y	46	65.7	65.7	100.0
Total	70	100.0	100.0	

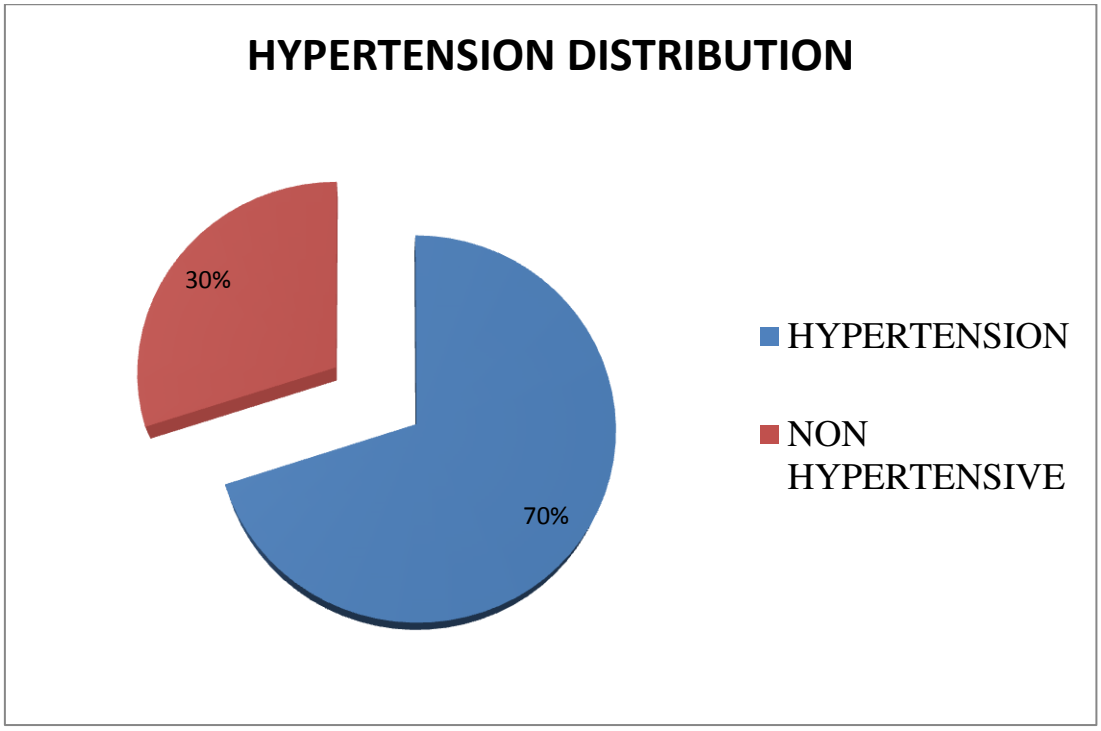


DISTRIBUTION OF HYPERTENSION:

In total of 70 patients included in our study 49 patients were hypertensive ant the remaining 21 patients were not hypertensive.

HYPERTENSION

	Frequency	Percent	Valid Percent	Cumulative Percent
NO	21	30.0	30.0	30.0
YES	49	70.0	70.0	100.0
Total	70	100.0	100.0	



Association between serum uric acid hypertension / diabetes

Kruskal-Wallis Test

Variable	Mean serum uric acid	Std. Deviation	Mean Rank	P value
Both diabetes and hypertension present	5.546429	1.5469162	39.07	
Either diabetes or hypertension present	5.030769	1.3442184	33.05	0.485
No hypertension / diabetes	4.766667	.4163332	34.00	
Total	5.225714	1.4173547		

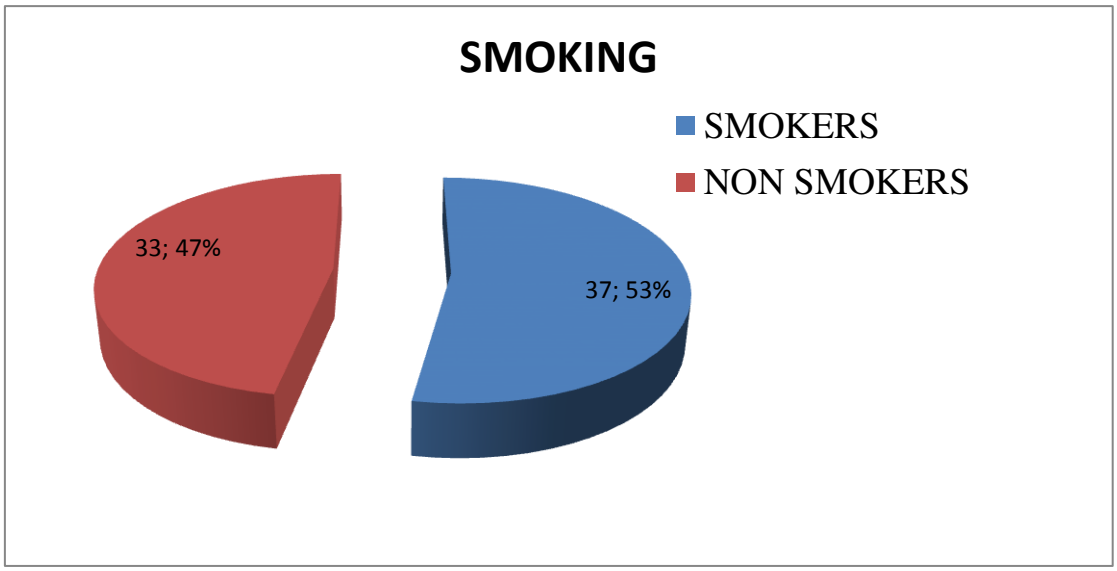
No significant association was found between mean serum uric acid levels and presence of diabetes or hypertension by kruskal wallis test (p=0.485)

SMOKING DISTRIBUTION:

Out of 70 patients in our study,37 patients had smoking habits and remaining 33 were non smokers. Out of 44 male patients 37 were smokers.

SMOKING

	Frequency	Percent	Valid Percent	Cumulative Percent
N	37	52.9	52.9	52.9
Y	33	47.1	47.1	100.0
Total	70	100.0	100.0	



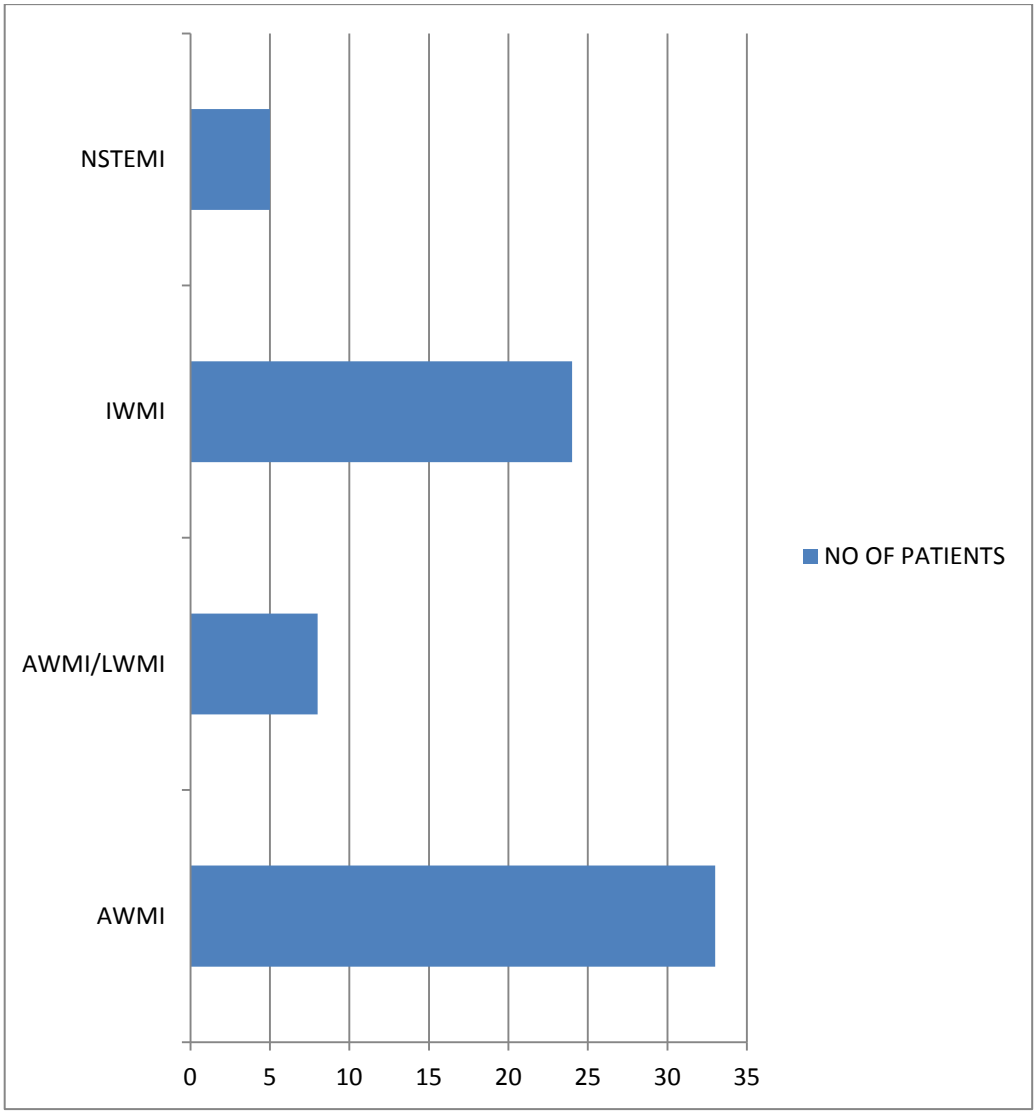
ACUTE MYOCARDIAL INFARCTION TYPES:

AWMI: Anterior wall myocardial infarction.

LWMI: Lateral wall myocardial infarction.

IWMI: Inferior wall myocardial infarction.

NSTEMI: Non ST elevation myocardial infarction.



Out of 70 patients, 33 patients had AWMI,8 patients were both AWMI/LWMI, 24 patients were IWMI,5 patients had NSTEMI.

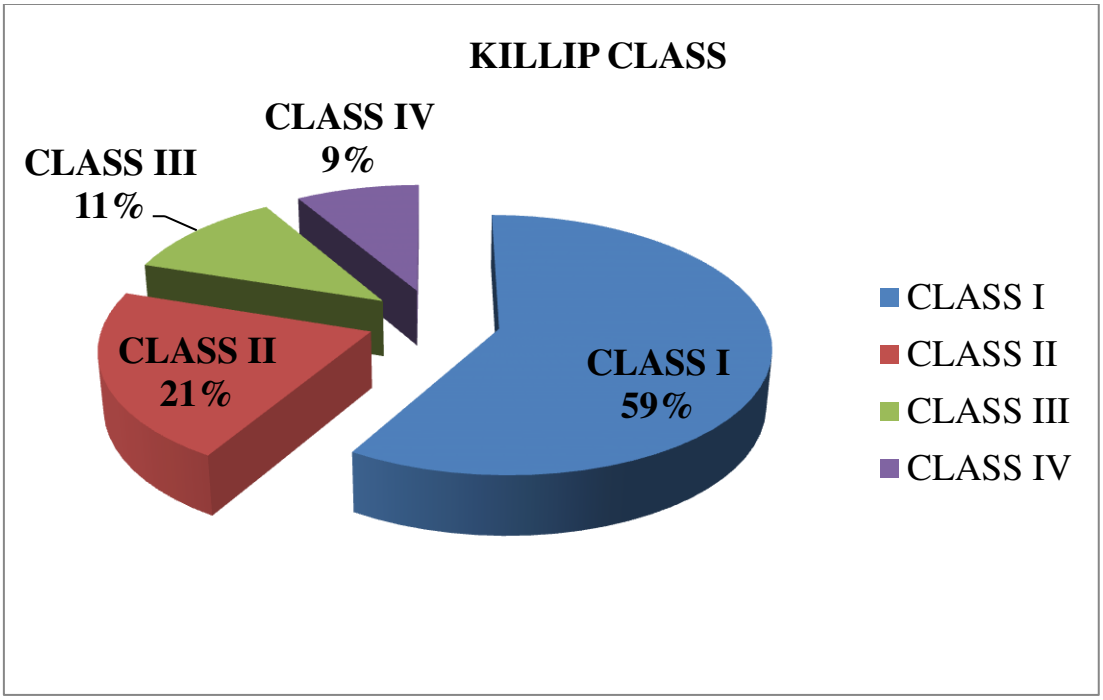
AWMI/IWMI/LWMI/NSTEMI

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	AWMI	33	47.1	47.1	47.1
	AWMI/LWMI	8	11.4	11.4	58.6
	IWMI	24	34.3	34.3	92.9
	NSTEMI	5	7.1	7.1	100.0
	Total	70	100.0	100.0	

KILLIP CLASSIFICATION:

KILLIP CLASS

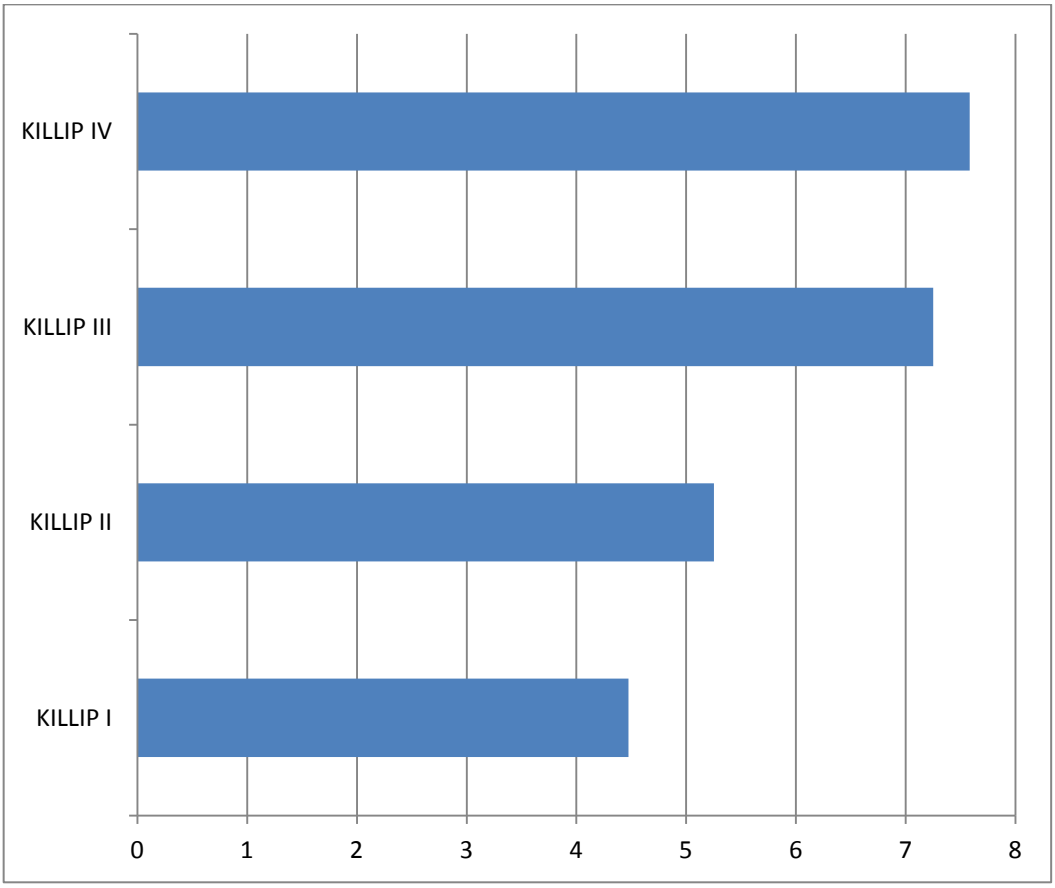
	Frequency	Percent	Valid Percent	Cumulative Percent
CLASS I	41	58.6	58.6	58.6
CLASS II	15	21.4	21.4	80.0
CLASS III	8	11.4	11.4	91.4
CLASS IV	6	8.6	8.6	100.0
Total	70	100.0	100.0	



Out of 70 patients in our study killip class I and II was around 80%, killip class III and IV was around 20%

Association between serum Uric Acid levels and Killip classification:

Killip class	Mean	Mean Rank	P value
I	4.475	25.06	0.000
II	5.253	38.33	
III	7.250	62.06	
IV	7.583	64.33	



Kruskal-Wallis Test

Ranks

	Killip class coded	N	Mean	Mean Rank	P value
SERUM URIC ACID	1.00	41	4.475610	25.06	0.000
	2.00	15	5.253333	38.33	
	3.00	8	7.250000	62.06	
	4.00	6	7.583333	64.33	
	Total	70	5.225714		

Uric acid level was significantly higher among patients in class IV (7.58), in class III(7.25) than patients class II (5.25) and class I (4.47). Mean difference was statistically significant by kruskal wallis test (p=0.000).

DISCUSSION

Acute myocardial infarction is a spectrum of disorder which include ST elevation myocardial infarction (STEMI) and non ST elevation myocardial infarction(NSTEMI).STEMI is usually associated with short term and as well long term mortality. The diagnosis of STEMI and NSTEMI is done by clinical features and characteristic ECG changes such as new onset or presumed new changes in ST segment and new onset LBBB and elevation of cardiac biomarkers such as troponin I and T, creatine kinase MB.

Acute myocardial infarction is associated with high death rate within 24 hours. And most of the death occur within one hour of onset of symptoms. So risk stratification has a important role in the management of acute myocardial infarction. Blood pressure, localisation of infarct, killip class, TIMI score are some methods useful in risk stratification and estimation of mortality in intensive coronary unit.

Killip classification is bedside evaluation test to predict the mortality in acute coronary syndrome. It has 4 class in which class III and IV has a higher mortality than the class I and II. The mortality rate of killip class IV is 81%. The other scores such as TIMI(Thrombolysis In Myocardial Infarction) was used to predict mortality in only in STEMI.[20]

Serum uric acid is a product of purine metabolism and hyperuricemia is also a independent risk factor for cardiovascular disease in high risk population such as diabetes, hypertension, dyslipidemia.[13] Some studies shows that uric acid level more than 7gm/dl are associated with the high mortality rate in cardiac failure.[11] In Japan, study conducted on STEMI patients reported that hyperuricemia increases 1-year mortality rate in killip class IV.[22] Our study is conducted to assess the relationship between serum uric acid level and killip class in acute myocardial infarction.

In our study total of 70 patients were included, who were admitted in ICCU. All patients were included in study after getting consent, detail history and physical examination and after ruling out the exclusion criteria. Out of 70 patients 44 patients were male and 26 patients were females. This distribution shows the predominance of males in acute myocardial infarction. This results are similar to other study regarding male predominance in acute myocardial infarction.

Uric acid of male patients is significantly higher than the female patient, which is similar to other studies. In our study the risk factors such as diabetes, hypertension, smoking were taken into consideration. Out of 70 patients 49 patients were hypertensive and 46 patients has diabetes, 37 patients had smoking habits, smoking is common in males in our

population. Out of 70 patients in our study 33 patients had AWMI,8 patients were both AWMI/LWMI, 24 patients were IWMI,5 patients had NSTEMI.

Out of 70 patients in our study, 80% of the patients were under killip class I and II at the time of admission, 20% of the patients were on killip class III and IV at the time of admission. Uric acid level was significantly higher among patients in class IV (7.58), in class III (7.25) than patients class II (5.25) and class I (4.47). Mean difference was statistically significant by kruskal wallis test (p=0.000). The results in our study shows that serum uric acid level are high in killip class III and IV in acute myocardial infarction patients. Combination of serum acid and killip classification will be useful in assessing the prognosis in acute myocardial infarction patients.

CONCLUSION

In general, serum uric acid level is significantly higher in male patients than the female patients.

Diabetes and hypertension remains the major risk factors for acute myocardial infarction.

Hyperuricemia is associated with killip class III and IV in Acute myocardial infarction patients.

Further study on combination of killip class and serum uric acid level in predicting mortality will be informative and useful.

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PROFORMA

Name Age: Sex

Educational status: Occupation:

Address:

Ht in cms

Wt in kgs:

BMI [kg/m2]:

Clinical presentation on admission:

Time of onset of symptoms:

- Chest pain Typical/Atypical
- Syncope.
- Palpitation.
- Shortness of breath.
- Cerebral symptoms.

- **PAST HISTORY:** yes no
- Diabetes

- Hypertension
- Myocardal infarction
- Angina pectoris
- Cerebrovascular diseases
- Drug intake

- **PERSONAL HISTORY:**

- Diet: vegetarian/non vegetarian:
- Smoking/ Alcohol /Tobacco chewing:
- Sedentary habits:
- **Menstrual and Obstetric History**:

- **GENERAL EXAMINATION**
- VITALS PR: BP: JVP :
- Anemia:
- Jaundice
- Cyanosis:
- Clubbing:
- EDEMA

EXAMINATION OF CARDIOVASCULAR SYSTEM

- **EXAMINATION OF RESPIRATORY SYSTEM:**

- **ABDOMEN EXAMINATION**

- **CENTRAL NERVOUS SYSTEM**

INVESTIGATIONS

- **ECG:** **ECHO**
- **CBC:**
- **CARDIAC ENZYMES:**
- **RFT:**
- **LFT:**
- **SERUM URIC ACID LEVEL:**

S.NO	NAME	AGE	SEX	HT	DM	SMOKING	AWMI/IWMI/LWMI/NSTEMI	KILLIP CLASS	SERUM URIC ACID
1	SIVAKUMAR	48	M	N	Y	Y	AWMI	CLASS I	4.9
2	ABRAHAM	58	M	Y	Y	N	IWMI	CLASS I	4.6
3	SRIDHAR	45	M	N	N	Y	IWMI	CLASS I	4.3
4	ABDUL RAHIM	35	M	Y	Y	Y	IWMI	CLASS I	3.3
5	AROKIAMARY	69	F	Y	N	N	IWMI	CLASS I	5.6
6	RAVIKUMAR	52	M	N	Y	N	AWMI	CLASS I	4.1
7	MURUGAN	55	M	Y	Y	Y	AWMI/LWMI	CLASS II	6.2
8	SARASWATHI	61	F	Y	Y	N	AWMI	CLASS II	3.4
9	PRAKASH	35	M	N	N	Y	AWMI	CLASS I	4.9
10	SUBBAIYA	49	M	Y	Y	N	IWMI	CLASS I	4
11	TAMILARASI	52	F	Y	Y	N	AWMI	CLASS I	4
12	KANNAN	51	M	Y	N	Y	AWMI	CLASS I	4.1
13	LATHA	60	F	Y	Y	N	AWMI/LWMI	CLASS II	6.2
14	DURAISAMY	55	M	N	Y	Y	AWMI	CLASS I	4.3
15	ILAYARAJA	52	M	Y	N	Y	AWMI	CLASS I	6.6
16	VENKATESAN	61	M	Y	Y	N	AWMI/LWMI	CLASS IV	7.6
17	KUMARESAN	54	M	Y	Y	Y	AWMI	CLASS II	6.1
18	RAJAMMAL	58	F	Y	Y	N	IWMI	CLASS I	5.8
19	CHELLAYA	51	M	N	Y	Y	NSTEMI	CLASS I	4.4
20	SHANMUGAM	48	M	Y	Y	Y	IWMI	CLASS I	4.2
21	RAJASEKAR	62	M	Y	N	Y	AWMI/LWMI	CLASS II	5.8
22	KUMAR	48	M	N	N	Y	IWMI	CLASS I	5.1
23	LOGANATHAN	62	M	Y	N	Y	AWMI	CLASS I	4.8
24	LAKSHMI	56	F	Y	N	N	IWMI	CLASS I	3.9
25	PERUMAL	60	M	Y	N	Y	IWMI	CLASS I	4

26	SEETHALAKSHMI	68	F	Y	Y	N	AWMI	CLASS I	4.2
27	KALAISELVI	55	F	N	Y	N	AWMI	CLASS I	3.4
28	NANDHAGOPAL	63	M	Y	Y	Y	AWMI	CLASS III	6.8
29	GANGAIAMMAL	51	F	Y	Y	N	AWMI/LWMI	CLASS III	7.8
30	KUMAR	35	M	N	Y	Y	AWMI	CLASS I	6.9
31	KAMALAMMAL	54	F	Y	Y	N	IWMI	CLASS II	3.8
32	GOMATHI	66	F	Y	Y	N	AWMI	CLASS I	3.4
33	PARAMESHWARAN	51	M	Y	Y	Y	AWMI/LWMI	CLASS II	4.8
34	LAKSHMINARAYAN	68	M	Y	N	Y	AWMI	CLASS IV	8.1
35	PUSPHALATHA	51	F	Y	N	N	IWMI	CLASS I	4.2
36	CHELLAPPAN	60	M	Y	N	Y	AWMI	CLASS II	4.3
37	ANBALAGAN	54	M	Y	Y	N	AWMI	CLASS II	4.8
38	VALLI	49	F	N	Y	N	IWMI	CLASS I	2.8
39	NAGARAJ	54	M	Y	N	Y	AWMI	CLASS I	4.1
40	MOHAMED JINNA	58	M	Y	N	Y	IWMI	CLASS I	4.8
41	VASUKI	59	F	Y	Y	N	AWMI	CLASS I	3.8
42	DHARMARAJ	40	M	Y	N	Y	AWMI	CLASS I	4.3
43	GOVUNDRAJ	49	M	Y	Y	Y	AWMI/LWMI	CLASS IV	7.2
44	RANI	54	F	Y	N	N	IWMI	CLASS II	5.1
45	KAUVERI	66	F	N	Y	N	IWMI	CLASS I	5
46	DURAISAMY	50	M	Y	Y	Y	AWMI	CLASS II	5.5
47	KOMALA	49	F	N	Y	N	IWMI	CLASS I	4
48	RAJESHWARI	60	F	Y	N	N	NSTEMI	CLASS I	3.4
49	GOPI	54	M	Y	Y	Y	AWMI/LWMI	CLASS IV	7.8
50	KATHIRVEL	38	M	N	Y	Y	AWMI	CLASS I	4.8
51	BHAVANI	65	F	Y	N	N	NSTEMI	CLASS I	3.2

52	GUNASEKAR	55	M	Y	Y	N	AWMI	CLASS II	5.2
53	SENTHIKUMAR	48	M	N	Y	Y	IWMI	CLASS II	5.5
54	MAHADEVAN	53	M	Y	Y	N	AWMI	CLASS III	6.5
55	JAGATHAMBAL	61	F	Y	Y	N	AWMI	CLASS I	6
56	ESWARI	56	F	Y	N	N	IWMI	CLASS IV	6.4
57	SHANTHI	50	F	N	Y	N	IWMI	CLASS I	4.2
58	KANNAN	39	M	N	Y	Y	IWMI	CLASS I	4
59	MEENAKSHI	56	F	N	Y	N	AWMI	CLASS I	3.8
60	DEVARAJ	57	M	Y	Y	Y	AWMI	CLASS III	7.5
61	GOPAL	47	M	Y	N	Y	AWMI	CLASS III	7
62	SARSWATHI	58	F	Y	Y	N	NSTEMI	CLASS I	6.4
63	SRINIVASAN	62	M	Y	Y	N	IWMI	CLASS IV	8.4
64	RAVI	40	M	N	Y	Y	AWMI	CLASS III	7.8
65	RAMACHANDRAN	51	M	Y	N	N	AWMI	CLASS II	6.1
66	AMBIKA	55	F	N	Y	N	IWMI	CLASS I	5.1
67	MANICKAM	53	M	N	Y	N	AWMI	CLASS III	6.8
68	VEERAMANI	61	M	Y	N	N	IWMI	CLASS II	6
69	JOSEPH	40	M	N	Y	Y	AWMI	CLASS III	7.8
70	ANTHONIAMMAL	64	F	Y	N	N	NSTEMI	CLASS I	4.8

HT-HYPERTENSION

DM- DIABETES MELLITUS

Y-YES

N-NO

AWMI-ANTERIOR WALL MYOCARDIAL INFARCTION

IWMI-INFERIOR WALL MYOCARDIAL INFARCTION

LWMI- LATERAL WALL MYOCARDIAL INFARCTION

NSTEMI-NON ST ELEVATION MYOCARDIAL INFARCTION

PATIENT CONSENT FORM

Study detail: **"correlation between Serum Uric acid level and killip class in acute myocardial infarction "**

Study centre : KILPAUK MEDICAL COLLEGE, CHENNAI
Patients Name :
Patients Age :
Identification Number :

Patient may check (✓) these boxes

I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask question and all my questions and doubts have been answered to my complete satisfaction. ☐

I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected. ☐

I understand that sponsor of the clinical study, others working on the sponsor's behalf, the ethical committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study. ☐

I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well-being or any unexpected or unusual symptoms. ☐

I hereby consent to participate in this study. ☐

I hereby give permission to undergo complete clinical examination and diagnostic tests including hematological, biochemical, radiological tests. ☐

Signature/thumb impression:

Patients Name and Address: place date

Signature of investigator :

Study investigator's Name : place date

சுயஒப்புதல் படிவம்

ஆய்வுசெய்யப்படும்தலைப்பு:

இடம்: பொது மருத்துவத்துவ துரை

அரசு கீழ்பாக்கம் மருத்துவ கல்லூரி மருத்துவமனை

சென்னை

பங்குபெறுபவரின் பெயர்:

பங்குபெறுபவரின் வயது: ***பங்குபெறுபவரின் எண்:***

மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. நான் இவ்வாய்வில் தன்னிச்சையாக பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த சட்டசிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகிக்கொள்ளல்லாம் என்றும் அறிந்து கொண்டேன்.

இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்து மேலும் ஆய்வு மேற்கொள்ளும்போதும் இந்த ஆய்வில்பங்கு பெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். இந்த ஆய்வின் மூலம் கிடைக்கும் தகவலையோ, முடிவையோ பயன்படுத்திக்கொள்ள மறுக்க மாட்டேன்.

இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக்கொள்கிறேன். இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதியளிக்கிறேன்.

பங்கேற்பவரின் கையொப்பம் *ஆய்வாளரின் கையொப்பம்*

இடம்:

தேதி:

INSTITUTIONAL ETHICS COMMITTEE
GOVT. KILPAUK MEDICAL COLLEGE,
CHENNAI-10
Protocol ID. No.07/2017 Meeting held on 17.04.2017

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval "**Correlation between Serum Uric acid level and Killip Class in Acute Myocardial infarction"** submitted by Dr.Karthikeyan.V.A, M.D. (General Medicine), PG Student, GKMC, Chennai-10

The Proposal is APPROVED

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

DEAN
Govt. Kilpauk Medical College,
Chennai-10.