" TIMI SCORE IN ACUTE ST ELEVATION MYOCARDIAL INFARCTION AND ITS CORRELATION WITH SINGLE QUANTITATIVE TROPONIN T AND EJECTION FRACTION < 40%"

A Dissertation Submitted to

THE TAMILNADU DR. M.G.R MEDICAL UNIVERSITY

CHENNAI

In Partial Fulfillment of the Regulations

for the Award of the Degree of

M.D. (GENERAL MEDICINE) - BRANCH – I

GOVERNMENT KILPAUK MEDICAL COLLEGE

CHENNAI-10

April – 2016

BONAFIDE CERTIFICATE

This is to certify that **"TIMI SCORE IN ACUTE ST ELEVATION MYOCARDIAL INFARCTION AND ITS CORRELATION WITH SINGLE QUANTITATIVE TROPONIN T AND EJECTION FRACTION < 40% "** is a bonafide work performed by **Dr. DILIP KUMAR R**, post graduate student, Department of Internal Medicine, Kilpauk Medical College, Chennai-10, under my guidance and supervision in fulfillment of regulations of the Tamil Nadu Dr. M.G.R Medical university for the award of M.D. Degree Branch I (General Medicine) during the academic period from July 2013 to April 2016.

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DECLARATION

I, **Dr. DILIP KUMAR R,** declare that, I carried out this work on, **" TIMI SCORE IN ACUTE ST ELEVATION MYOCARDIAL INFARCTION AND ITS CORRELATION WITH SINGLE QUANTITATIVE TROPONIN T AND EJECTION FRACTION < 40% "** at the Department of Medicine, Kilpauk medical college & hospital during the period of January 2015 to August 2015. This is submitted to The Tamilnadu Dr.M.G.R.Medical University, Chennai in partial fulfillment of the requirement for the award of M.D degree (Branch –I) General Medicine.

Place : Chennai **Dr. DILIP KUMAR R**

Date:

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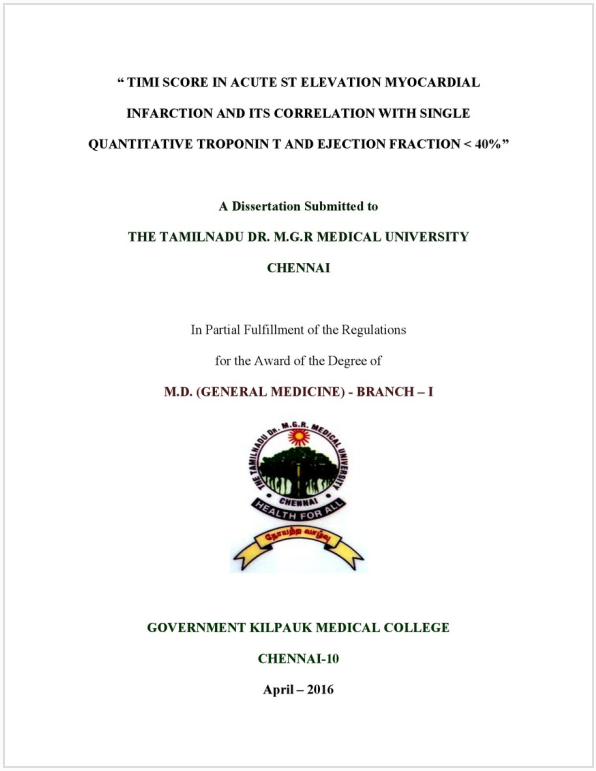

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LIST OF ABBREVATIONS

Sl No	ABBREVIATION	EXPANSION
1.	ACS	Acute Coronary Syndrome
2.	AMI	Acute Myocardial Infarction
3.	AHA	American Heart Association
4.	ACC	American College of cardiology 7,
4.	cTn	Cardiac Troponins
5.	CABG	Coronary Artery Bypass Grafting
6.	CHF	Congestive Heart Failure
7.	ECG	ElectroCardioGram
8.	ESC	European Society of Cardiology
9.	EF	Ejection Fraction
10.	LAD	Left Anterior Descending artey
11.	LCx	Left Circumflex artery
12.	LCA	Left Coronary Artery
13.	LBBB	Left Bundle Branch Block
14.	MI	Myocardial Infarction
15.	STEMI	ST Elevation Myocardial Infarction

16.	NSTEMI	Non ST Elevation Myocardial Infarction
17.	AWMI	Anterior Wall Myocardial Infarction
18.	ASMI	AnteroSeptal Myocardial Infarction
19.	ALMI	AnteroLateral Myocardial Infarction
20.	IWMI	Inferior Wall Myocardial Infarction
21.	RVMI	Right Ventricular Myocardial Infarction
22.	PWMI	Posterior Wall Myocardial Infarction
23.	STE	ST Elevation
24.	UA	Unstable Angina
25.	Trop	Troponin
26.	TIMI	Thrombolysis In Myocardial Infarction
27.	WHO	World Health Organization
28.	CK	Creatine Kinase
29.	S3	Third Heart Sound

TABLE OF CONTENTS

INTRODUCTION

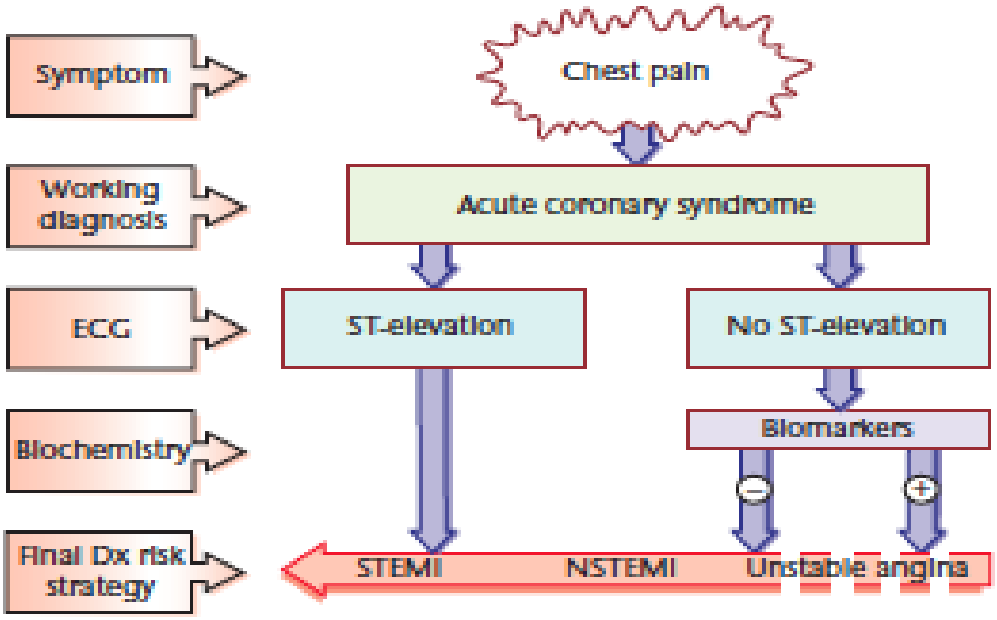
ACUTE CORONARY SYNDROMES :

Acute Coronary Syndromes (ACS) represents a broad collection of conditions :

1. Acute Myocardial infarction manifested by ST Elevation MI , also called as Q wave MI and Non ST Elevation MI wave also called as non Q wave MI.
2. Unstable Angina (UA)

As opposed to STEMI which results from complete and prolonged occlusion of an epicardial vessel. Notably, the clinical presentation and symptoms may be similar for these syndromes.

The primary goals of treatment in NSTE-ACS are to relieve and/or limit ischemia , prevent infarction or reinfarction, and improve outcomes.



DEFINITIONS :

1. STEMI is diagnosed in an appropriate clinical setting with the finding of >= 1mm or >= 0.1cm elevation of the ST segment in at least two contiguous leads of an electrocardiogram (ECG) associated with cardiac biomarker elevation.

2. NSTEMI/UA is diagnosed in an appropriate clinical setting when there is no ST segment elevation . ECG may show ST segment depression or T wave abnormalities, but may be normal, with (NSTEMI) or without (UA) myocardial necrosis demonstrated by cardiac biomarker elevation.1

The current universal definition for acute myocardial infarction (AMI) adopted by the ESC/ACF(American Cardiology Foundation) /AHA WHO Task Force defines AMI as evidence of myocardial necrosis in the appropriate clinical setting meeting any of the following criteria2: elevated cardiac biomarkers, preferably serum troponin, with minimum one value of the biomarker >99% of the upper reference limit (URL) with one of the following:

1. Ischemic symptoms
2. New ischemic ECG changes (new changes in the ST-T segment or evidence of new left bundle branch block)
3. New pathologic Q waves
4. Evidence of new infarction by cardiac imaging
5. Intracoronary thrombus noted on angiography or autopsy

Post PCI – greater than 3 times normal elevation of cardiac biomarkers signify PCI related myocardial infarction.

Post CABG – greater than 5 times the normal elevation of cardiac biomarkers with new Q waves/ LBBB, graft/native coronary artery occlusion , or infarction by cardiac imaging signify CABG related myocardial infarction.

EPIDEMIOLOGY :

ACS represents almost 1.6 million hospitalizations each year. This syndrome requires early diagnosis and aggressive management to minimize myocardial damage . Despite maximal medical therapy , at the end of 1 year , those patients diagnosed wih NSTEMI and treated for it are still at a risk of death (5.91%), risk of recurrent myocardial infarction (10.87%) and recurrent need for a revascularization (49-61%). It is important , in fact extremely necessary to note that even though the short term mortality for ST Elevation MI is higher when compared to Non ST Elevation MI , the long term mortality is still the same.

There are a lot of tools necessary to assess ACS . Our discussion will pertain to STEMI , and it has a Very high short term and even high long term mortality as well. All tools including Physical and laboratory tools are essential to make a diagnosis of STEMI , but this is to emphasize the use of TIMI score (Thrombolysis In Myocardial Infarction – TIMI) for STEMI to assess the values of Cardiac Biomarkers and Ejection fraction (both of which in turn are significant predictors of infarct size – morbidity/mortality in short and long term).

TIMI score for STEMI uses simple bedside data along wit ECG to determine the 30 day mortality rate , but again we emphasize the use of the score to predict Troponin T biomarker and Ejection fraction , so that it can be used to predict those values at the time of admission itself . (one step ahead)

AIMS AND OBJECTIVES

AIMS :

To assess the TIMI score for acute STEMI and its relationship with Quantitative Troponin T and LV Ejection Fraction .

OBJECTIVES :

1. To assess the relationship between TIMI score for acute STEMI and Single Quantitative Troponin T at 12 hours following admission.
2. To assess the relationship between TIMI score for acute STEMI and EF < 40% at 48 hours post admission.

REVIEW OF LITERATURE

ST-segment elevation myocardial infarction (STEMI) represents a pathophysiologic process among the acute coronary syndromes (ACS) that is different from that of and non–ST-segment elevation myocardial infarction (NSTEMI) and Unstable angina . (UA)

• The therapeutic paradigm for STEMI mandates a rapid decision regarding reperfusion.

• Time to reperfusion (i.e., “door to balloon” or “door to needle”)is an important component of these therapies and has become a major benchmark for institutional quality of cardiovascular care.

Definition

• Pathologic definition: cardiac myocyte cell death, usually due to prolonged myocardial ischemia.

• Clinical definition of myocardial infarction (MI) that has been established by the WHO/AHA/ESC/ACC requires the rise and/or fall of cardiac biomarkers for myocardial necrosis in addition to one of the following1:

1. Ischemic symptoms

2. Electrocardiogram (ECG) changes consistent with ischemia or 3. pathologic Q waves

4. Confirmation of infarction on imaging

5. Autopsy evidence of myocardial cell death is also sufficient.

• STEMI can be differentiated clinically from NSTEMI by the presence of changes in ECG specific to them (i.e., ST elevation, new left bundle branch block [LBBB]).

Classification

• The designation of STEMI includes not only classic ST elevation but also new LBBB.

• The descriptors "Q wave" and "non-Q wave MI" have lost favor because the majority of STEMIs are Q-wave MIs.

• A consensus statement by the ACC/AHA/ESC/WHF published in 2012 refines and broadens the classification of MI and includes categories based on procedure associated MIs, demand ischemia, and sudden cardiac death.

Epidemiology

• An estimated 50,00,000 patients in India will suffer a STEMI annually.

• 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 a 26% reduction in mortality since 1990.

• Overall survival rates across the majority of good Indian Centres are >90%.

• However, the death rate remains high among the subgroup of patients who develop cardiogenic shock or other mechanical complications of STEMI, with mortality in excess of 50%.

Etiology

• Any condition or event that results in interruption of coronary flow sufficient to produce cell death in the myocardium can lead to MI.

• Usually can result from an acute change in the preexisting coronary plaque that leads to thrombotic mediators getting activated and subsequent clot formation with obstruction to blood flow.

• Although there is a spectrum in terms of the degree of artery obstruction and cell death, it is the significant or complete coronary occlusion, which leads to STEMI.

• Other conditions that can lead to STEMI include

1. Severe coronary vasospasm
2. Embolization
3. Spontaneous coronary dissection

These conditions should be considered in the patient whose clinical findings suggest a process other than acute plaque rupture.

Pathophysiology

• Most commonly due to coronary artery occlusion by thrombus, which often forms in situ at the site of an atheromatous plaque.

• The mechanisms involved vary by age and gender.

1. Plaque rupture causes the majority of events in men and older women.

2. Plaque erosion is a more common mechanism in younger women.

• Involves mild-to-moderate immature plaques (i.e., those that do not significantly impede coronary flow at baseline) with thin fibrous caps and lipid-rich cores that rupture in the acute setting of inflammation, shear forces, and local rheologic

factors.

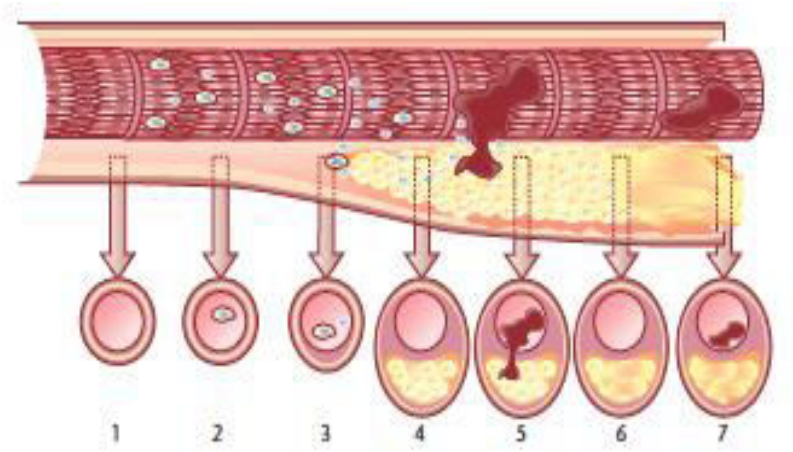
• This initiates a sequence of platelet aggregation, fibrin deposition, and vasoconstriction, forming the classic fibrin-rich red thrombus, which completely occludes the involved artery, predisposing to STEMI.

• Left untreated, the mortality rate of uncomplicated STEMI can exceed 30%.

• Mechanical complications are more common when a STEMI is untreated.

• In addition, the heart undergoes the detrimental process of remodeling.

ATHEROSCLEROSIS:

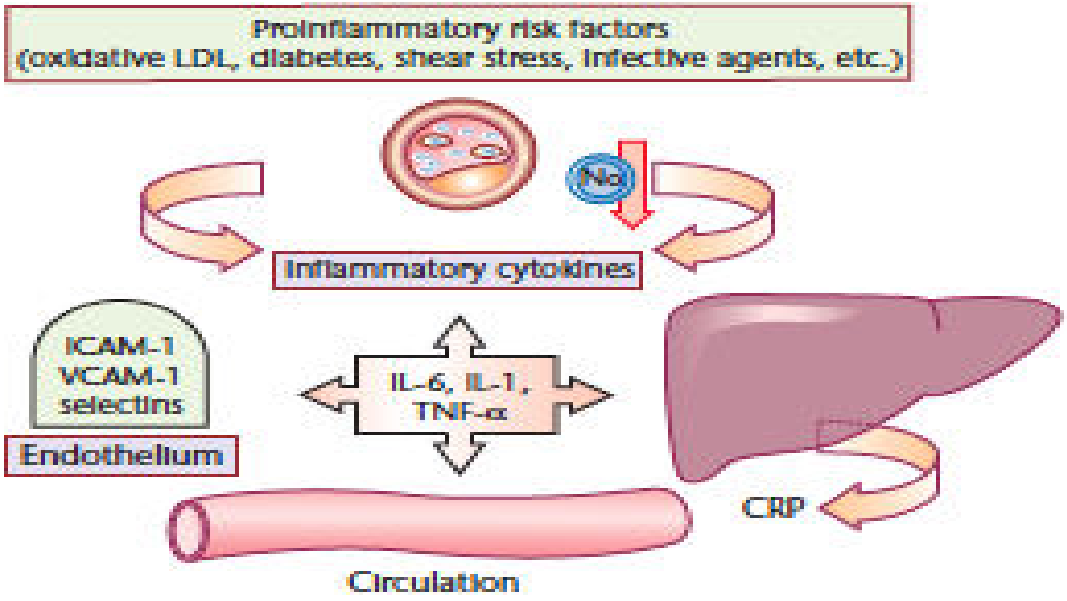


1, Normal artery.

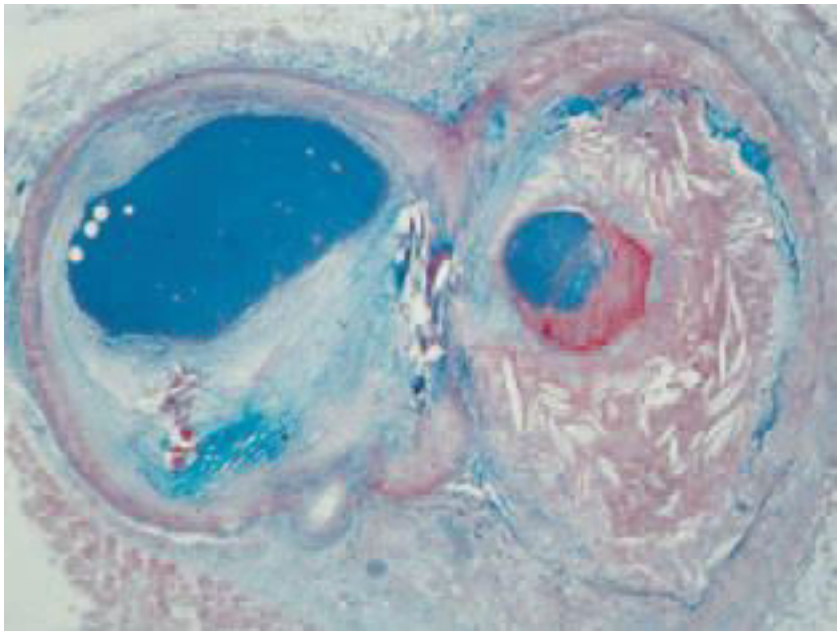
2.Endothelial cells , activated by

risk factors which may include hyperlipoproteinaemia, usually express adhesion as well as chemoattractant molecules that recruit inflammatory leucocytes such as monocytes and T lymphocytes. Extracellular lipid begins to accumulate in intima at this stage. 3, Evolution to fibrofatty stage. Monocytes recruited to artery wall become 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 inflammatory cytokines and growth factors that amplify leucocyte recruitment and cause smooth muscle cell migration and proliferation. 4, As lesion progresses, inflammatory mediators cause expression of tissue

factor, a potent pro-coagulant, and of matrix-degrading proteinases that weaken fibrous cap of plaque. If fibrous cap ruptures at point of weakening, coagulation factors in blood can gain access to thrombogenic, tissue factor-containing lipid core, causing thrombosis on non-occlusive atherosclerotic plaque. If balance between prothrombotic and fibrinolytic mechanisms prevailing at that particular region and at that particular time is unfavourable, occlusive thrombus causing ACS may result. 6, When thrombus resorbs, products associated with thrombosis such as thrombin and mediators released from degranulating platelets, can cause healing response, leading to increased collagen accumulation and smooth muscle cell growth. In this manner, the fibrofatty lesion can evolve into an advanced fibrous and often calcified plaque, one that may cause significant stenosis, and produce symptoms of stable angina pectoris. 7, In some cases, occlusive thrombi arise not from fracture of fibrous cap but from superficial erosion of endothelial layer. Resulting mural thrombus, again dependent on local prothrombotic and fibrinolytic balance, can cause acute myocardial infarction.



The pathophysiology of atherosclerosis with respect to lesion development, progression and destabilization. Biomarkers with distinct pathophysiological profile can be used to assess disease activity.



"Atherothrombosis : A variable mix of chronic atherosclerosis and acute thrombosis. Cross-sectioned arterial bifurcation illustrating a collagen-rich (blue-stained) plaque in the circumflex branch (left), and a lipid-rich and ruptured plaque with a non-occlusive thrombus superimposed in the obtuse branch (right). Ca, calcification; T, thrombus; C, contrast in the lumen."

FEATURES OF A VULNERABLE PLAQUE :

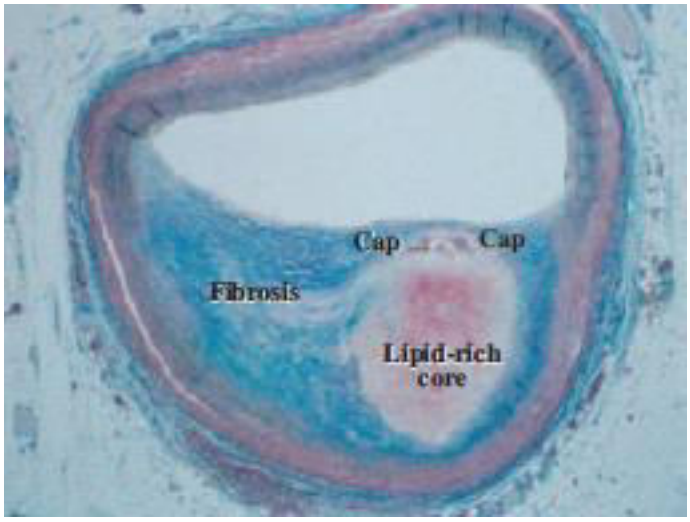
1. Large lipid-rich core (> 30–40% of plaque)
2. Fibrous cap covering the lipid-rich core
3. Thin (thickness < 100 μm)
4. Many macrophages (inflammation)

5. Few smooth muscle cells (apoptosis)

6. Outward remodelling preserving the lumen

7. Neovascularization from vasa vasorum

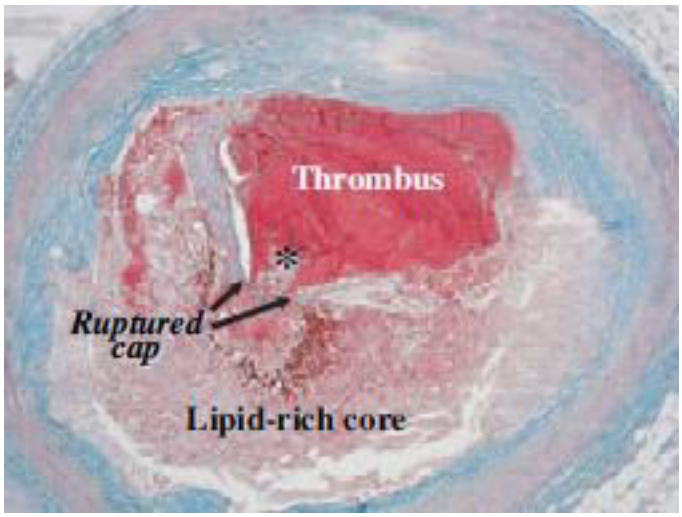
8. Adventitial/perivascular inflammation



Vulnerable plaque. Cross-section of a coronary artery containing a plaque assumed to be rupture-prone, consisting of a relatively large lipid-rich core covered by a thin

and fragile fibrous cap. Trichrome stain, rendering collagen blue and lipid colourless.

RUPTURED PLAQUE :

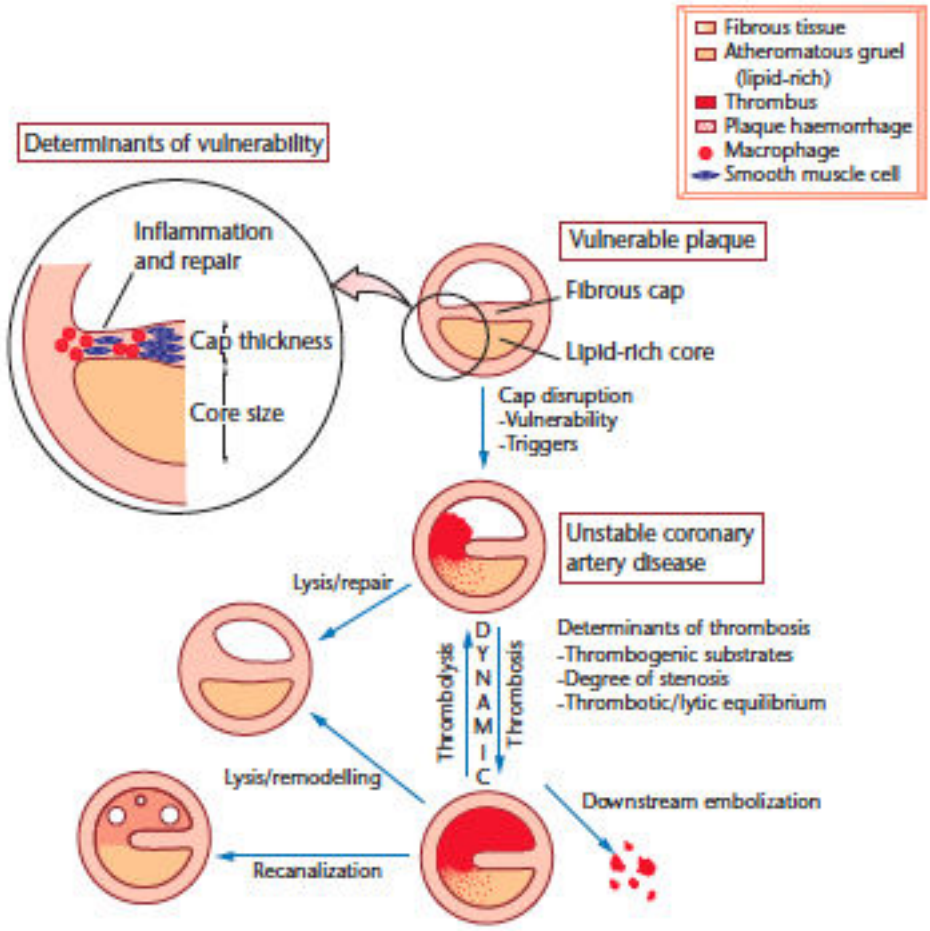


Plaque rupture. Cross-sectioned coronary artery containing a lipid-rich atherosclerotic plaque with occlusive thrombosis superimposed. The fibrous cap covering the lipid-rich core is ruptured (between arrows) exposing the thrombogenic core to the blood in the lumen. Atheromatous plaque content is displaced through the gap in the cap into the lumen (cholesterol crystals at asterisk), clearly indicating

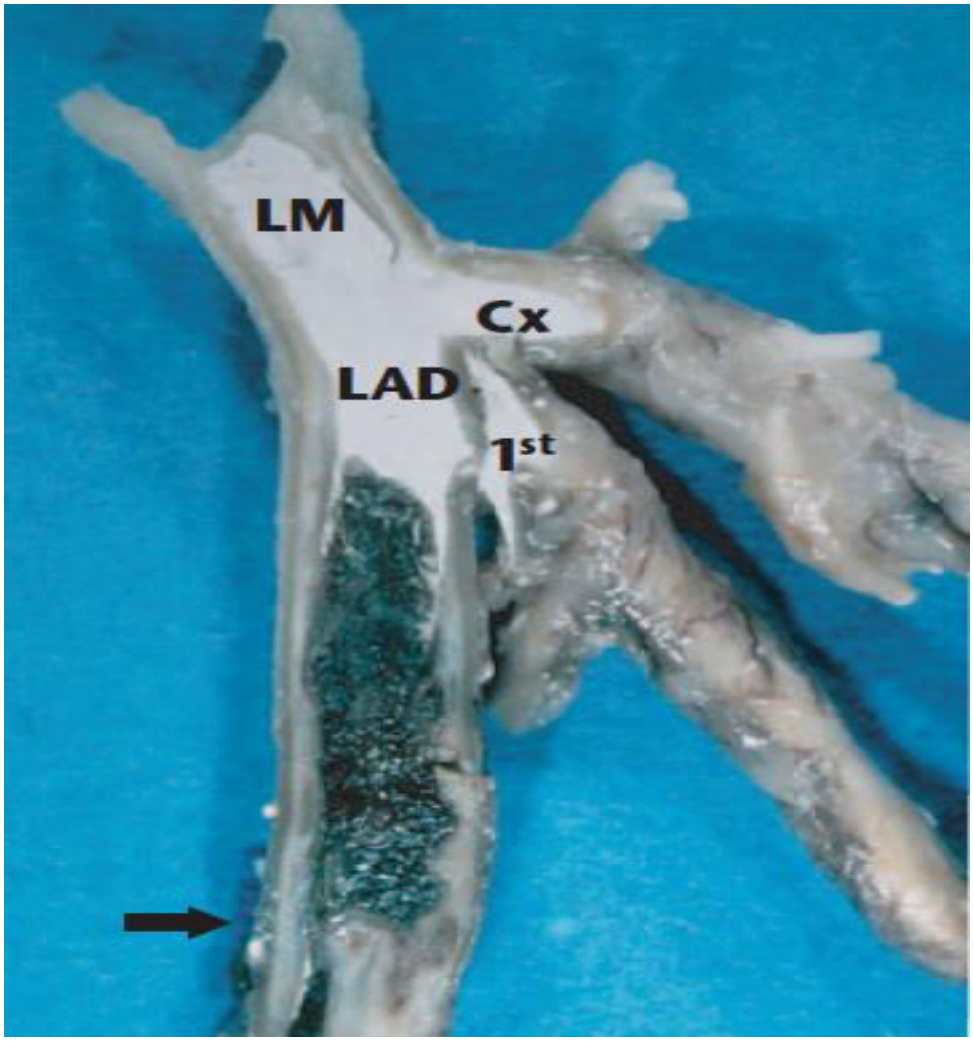
the sequence of events: plaque rupture preceded thrombus

formation. Trichrome stain, rendering luminal thrombus and

intraplaque haemorrhage red and collagen blue.



Plaque vulnerability, rupture and thrombosis. Lipid accumulation, cap thinning, macrophage infiltration, and local loss of smooth muscle cells destabilize plaques, making them vulnerable to rupture. It is unknown whether rupture of a vulnerable plaque is a random (spontaneous) or triggered event. The thrombotic response to plaque rupture is dynamic and depends on local (e.g. exposed substrate and shear forces) as well as systemic factors (e.g. platelets, coagulation and fibrinolysis).



Thrombotic burden. Thrombosed coronary artery cut open longitudinally, illustrating a voluminous erythrocyte-rich stagnation thrombosis (dark black on screen) that has developed secondarily to blood stagnation caused by an occlusive platelet-rich thrombus (white) formed on top of a severely stenotic and ruptured plaque (arrow). The white material in the lumen is contrast medium injected postmortem. LM, left main stem; LAD, left anterior descending

coronary artery; Cx, circumflex branch; 1st; first diagonal branch.

DIAGNOSIS

Clinical Presentation

• Patients presenting with a suspected ACS should undergo rapid evaluation.

• A focused history, physical, and ECG interpretation should be performed within 10 minutes of arrival in the emergency department to allow for timely reperfusion

when appropriate.

History

• Quickly acquire adequate historical information in the setting of diagnostic ECG changes to initiate treatment and mobilize, when appropriate, the team for percutaneous revascularization.

• Chest discomfort is the most common symptom.

Typically progressive, substernal to left-sided, and often similar in quality to typical angina.

Usually intense and prolonged, lasting more than 20 to 30 minutes.

Unlike UA/NSTEMI, rest and nitroglycerin usually do not provide significant relief.

• Review absolute and relative contraindications to thrombolytic therapy

(Table-1) which are intended as recommendation for clinical decision making, but other contraindications may be present and decisions must be made on a case-by-case

basis.4

• Review issues regarding primary percutaneous coronary intervention (PCI), including allergy to contrast agents, issues relating to vascular access (peripheral vessel disease or previous peripheral revascularization procedures), previous cardiac catheterizations and complications, history of renal dysfunction, central nervous system disease, pregnancy, or bleeding diathesis.

• Traditional risk factors are weak predictors of the likelihood of acute infarction as the presenting etiology.

TABLE -1 :

The following Table – 1 shows the contraindications to thrombolysis . This is important since the patients we select must be thrombolysis eligible !

Absolute Contraindications.

- Any prior intracerebral haemorrhage
- Known structural cerebrovascular lesion (eg. AVM)
- Known intracranial neoplasm
- Prior ischaemic stroke (w/in 3 months)
- Active internal bleeding (excl. menses)
- Suspected aortic dissection or pericarditis

Relative Contraindications.

- Severe uncontrolled hypertension (>180/100 mmHg)
- Prior ischaemic stroke (>3 months)
- Known intracranial pathology
- Current anticoagulation w/ INR > 2-3
- Known bleeding diathesis
- Recent trauma (past 2 weeks)
- Prolonged CPR > 10mins
- Major surgery <3 weeks
- Non-compressible vascular punctures (eg. Subclavian or IJ lines)
- Recent internal bleeding (2-4 weeks)
- Previous streptokinase therapy
- Pregnancy
- Active peptic ulcer disease

PHYSICAL EXAMINATION :

• Important in determining other potential sources of chest pain, assessing prognosis, and establishing a baseline that will aid in the early recognition of complications.

• The goal is to determine hemodynamic stability, the presence of cardiogenic pulmonary edema, or mechanical complications of MI (papillary muscle dysfunction, free wall rupture, and ventricular septal defect [VSD]) and exclude other etiologies of acute chest discomfort.

• Should include assessment of vital signs and oxygenation with bilateral blood pressures as well as jugular venous pressure; pulmonary examination for pulmonary edema; cardiac examination for arrhythmia, murmurs, gallops, or friction rub; vascular examination for evidence of peripheral vascular disease and pulse deficits; and neurologic examination (especially prior to the administration of thrombolytics).

Differential Diagnosis

• The inherent risks of both thrombolytic therapy and primary PCI mandate that alternative diagnoses be considered in patients with chest pain.

• In particular, administration of thrombolytic agents in certain conditions, such as aortic dissection, may lead to death.

• In the situation when the diagnosis is uncertain, primary PCI offers a distinct advantage as the initial reperfusion strategy.

• Differential diagnosis of chest pain:

Life-threatening: aortic dissection, pulmonary embolus, perforated ulcer, tension pneumothorax, and Boerhaave syndrome (esophageal rupture with mediastinitis).

Other cardiac and noncardiac causes: pericarditis, myocarditis, vasospastic angina, gastroesophageal reflux disease, esophageal spasm,

costochondritis, pleurisy, peptic ulcer disease, panic attack, biliary or pancreatic pain, cervical disc or neuropathic pain, and somatization and psychogenic pain disorder.

• Differential diagnosis of ST elevation on ECG:

1. pericarditis
2. pulmonary embolism
3. aortic dissection with coronary artery involvement
4. normal variant
5. early repolarization
6. left ventricular (LV) hypertrophy with strain
7. Brugada syndrome
8. Myocarditis
9. Hyperkalemia
10. Others include HOCM, Bundle branch blocks, Prinzmetal Angina etc.

DIAGNOSTIC TESTS :

• The ECG should be performed and interpreted within 10 minutes of presentation.

• The ECG should be repeated every 20 to 30 minutes for up to 4 hours if

the patient has persistent symptoms when there is clinical suspicion for AMI, but the ECG is nondiagnostic.

• Hyperacute T waves, seen as either tall or deeply inverted T waves, may be an early sign of AMI that warrants close monitoring.

• Recognition of the limitations of the ECG in AMI is also important as up to 10% of patients with an acute STEMI may have a normal ECG as certain myocardial segments of the left ventricle are not adequately represented, particularly the

posterior and lateral walls, which are supplied by the left circumflex artery.

ECG criteria for diagnosis of STEMI:

≥1 mm (0.1 mV) of ST-segment elevation (STE) in two or more contiguous limb leads, or

2 mm in two contiguous precordial leads for men and 1.5 mm in two precordial leads for women

• The location and degree of STE determine the occluded anatomy and prognosis and can alert the physician to potential complications of MI.

Location	Anatomy of occlusion	ECG	1-year mortality (%)*
Proximal left anterior descending	Proximal to first septal perforator	ST↑ V1–6, I, aVL and fascicular bundle or bundle branch block	25.6
Mid left anterior descending	Proximal to large diagonal but distal to first septal perforator	ST↑ V1–6, I, aVL	12.4
Distal left anterior descending or diagonal	Distal to large diagonal or to diagonal itself	ST↑ V1–V4 or ST↑ I,V5,6 aVL, V5–V6	10.2
Moderate to large inferior (posterior, lateral, right ventricular)	Proximal right coronary artery or left circumflex	ST↑ II, III, aVF and any or all of the following: 1) V1, V3R, V4R 2) V5V6 3) R > S in V1, V2	8.4
Small inferior sdsds	Distal righ coronary artery or left circumflex branch occlusion	ST↑ II, III, aVF only	6.7

*Based on GUSTO-I cohort population in each of the 5-year categories, all receiving reperfusion therapy.

• Special considerations:

The presence of a new LBBB in the setting of acute chest symptoms suggests occlusion of the proximal left anterior descending (LAD).

Patients presenting with this finding should be managed in the same manner as the patient with a classic STEMI.

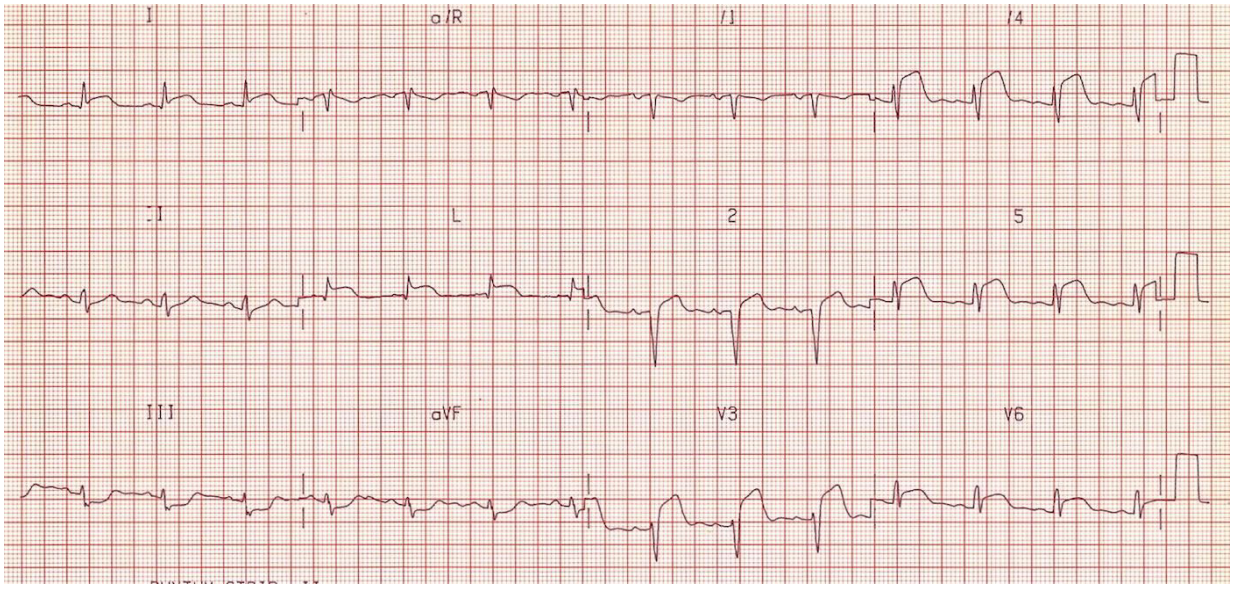
In the setting of an old LBBB or an RV-paced rhythm, an acute injury pattern may be supported by the Sgarbossa criteria5:

1. STE ≥1 mm in the presence of a positive QRS complex (ST elevation is concordant with QRS).

2. ST-segment depression ≥1 mm in lead V1, V2, or V3.

3. STE ≥5 mm in the presence of a negative QRS complex (ST elevation

is discordant with QRS).

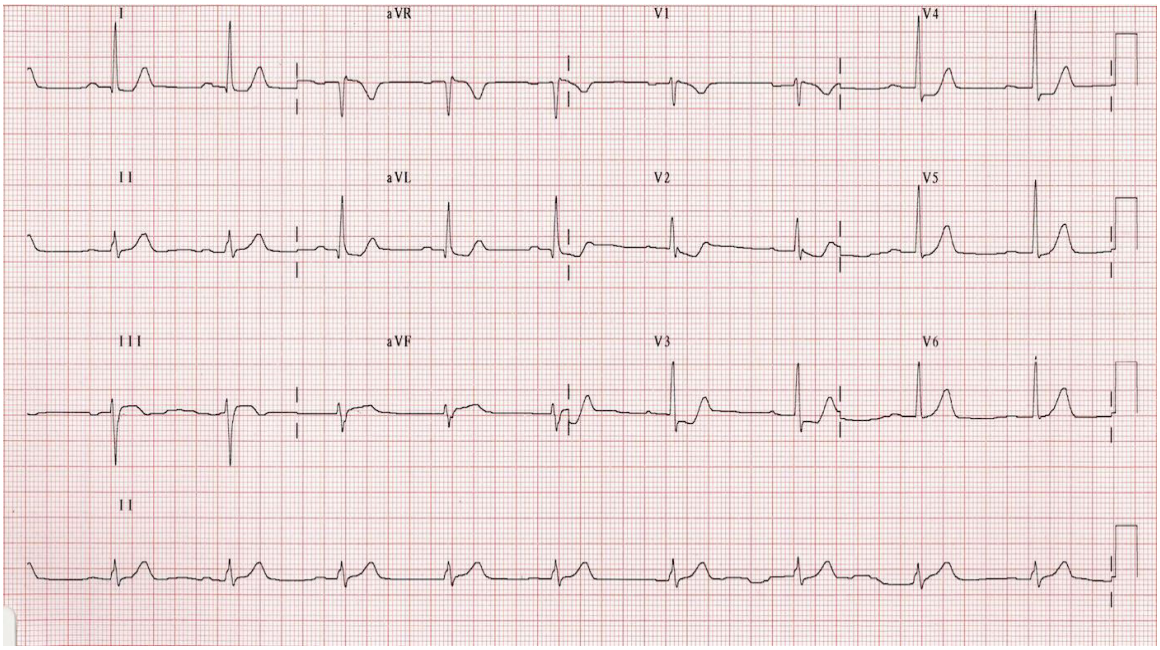


Example of a STEMI – ECG shows extensive Anterolateral MI with reciprocal changes in inferior leads.

Posterior MI is an entity that is often unrecognized and should be suspected by the clinician in the setting of inferior or lateral wall infarct. Isolated posterior MI is uncommon.

The “reverse mirror test” is useful to demonstrate that the ST-segment depression in leads V1 to V3 is actually ST elevations in the posterior wall.

The prominent R waves in these leads represent posterior Q waves.



Example of a Inferoposterior MI in RCA territory .

Inferoposterior and posterolateral MIs typically involve the right coronary artery (RCA) or obtuse marginal branch of the left circumflex coronary artery (LCx), respectively.

Posterior leads (V7 to V9) may be placed to help distinguish posterior MI from anterior ischemia or reciprocal depression in all patients presenting with ST depression in leads V1 to V3.

ST elevation in the inferior leads should always prompt a right-sided ECG to assess for right ventricular (RV) infarction. ST elevation in leads V3R and V4R suggests RV involvement.

RV infarction should also be suspected on a standard 12-lead ECG when

there is STE in V1 along with changes indicating inferior MI.

The finding of ST elevation in lead III greater than in lead II also suggests RV infarct.

Proximal RCA lesions typically involve the RV, as RV marginal branches arise early from the RCA.

Although the principle for the revascularization of RV infarct is the same for other STEMIs, other aspects of treatment are unique, including maintenance of adequate preload and the cautious use of nitrate and β-blocker therapy to avoid hypotension. ST segments in pericarditis normalize before there is T-wave inversion, whereas the T waves invert before ST normalization in STEMI.

STE in pericarditis is typically diffuse, does not correlate with a particular vascular territory, and does not exhibit reciprocal ST depressions.

PR-segment depression in acute pericarditis may also differentiate these two conditions. Pericarditis may present later in the course of AMI and should be differentiated from recurrent ischemia or stent thrombosis.

Imaging

• A standard portable chest radiograph (CXR) should be included in the initial evaluation protocol.

Pulmonary edema on CXR has important prognostic and therapeutic implications.

Should be reviewed for mediastinal widening, suggesting acute aortic dissection, prior to initiating thrombolytic therapy.

If clinical suspicion is high; however, normal mediastinal width does not exclude dissection.

• The evaluation of a patient with chest pain in the setting of a nondiagnostic ECG (i.e., LBBB of unknown duration, paced rhythm) may be aided by an echocardiogram. Segmental wall motion abnormalities suggest myocardial ischemia or infarction, assuming no baseline wall motion abnormalities and can help localize the territory at risk.

Laboratories

• Cardiac biomarkers are important in the diagnosis and prognosis of STEMI but have a limited role in the initial decision-making process.

Markers used for determining the presence of myocardial necrosis—including creatine kinase MB, troponin, and myoglobin.

• Standard laboratory evaluation should include a basic metabolic profile,

magnesium level, liver function, lipid profile, complete blood count, and coagulation studies.

The center of our article TROPONIN T biomarker has been discussed in detail in the coming review.

Risk Stratification in ST-segment elevation
myocardial infarction :

Whereas risk factors for the development of atherothrombosis
provide insights into disease mechanisms and the opportunity for primary and secondary prevention therapy, analysis of the risk for adverse outcome after presentation with ST-segment elevations is critically important in guiding management and therapeutic decisions.

Analysis usually uses a combination of clinical, ECG and biochemical parameters. Five rather simple baseline parameters can be used to predict more than 90% of the 30-day mortality: age, systolic blood pressure, Killip class, heart rate and infarct localization.

Killip class

The haemodynamic impact of the evolving myocardial infarction is clinically evident by the symptoms of shock.The Killip classification is widely used and linked to outcome. Killip class IV (‘cardiogenic shock’) is found in about 5% of AMI patients and is associated with extremely high mortality.

Infarct location

The prognosis of myocardial infarction is related to the extent of myocardium at risk and, related to this, to the site of coronary occlusion. The ECG reflects the infarct location (Table 12.5). Patients with main stem occlusion only rarely reach the hospital for reperfusion therapy. Occlusion of the proximal left anterior descending coronary artery proximal to the first septal branch is associ ated with high early and late mortality (‘widow-maker’). Large inferior myocardial infarctions as a result of occlusion of a dominant right coronary artery are also a high risk, particularly when the right ventricle is involved. Other locations, such as apical (distal left anterior descending), lateral (diagonal branch), or small inferior infarctions (distal right or circumflex), show ST-segment elevations in only a few leads and have a better outcome. Strictly

posterior myocardial infarctions (marginal branch of left circumflex) may escape routine ECG leads or only be evident through ST depression in V1 to V4, but usually have a good outcome.

Location	Anatomy of occlusion	ECG	1-year mortality (%)*
Proximal left anterior descending	Proximal to first septal perforator	ST↑ V1–6, I, aVL and fascicular bundle or bundle branch block	25.6
Mid left anterior descending	Proximal to large diagonal but distal to first septal perforator	ST↑ V1–6, I, aVL	12.4
Distal left anterior descending or diagonal	Distal to large diagonal or to diagonal itself	ST↑ V1–V4 or ST↑ I,V5,6 aVL, V5–V6	10.2
Moderate to large inferior (posterior, lateral, right ventricular)	Proximal right coronary artery or left circumflex	ST↑ II, III, aVF and any or all of the following: 1) V1, V3R, V4R 2) V5V6 3) R > S in V1, V2	8.4
Small inferior sdsds	Distal righ coronary artery or left circumflex branch occlusion	ST↑ II, III, aVF only	6.7

*Based on GUSTO-I cohort population in each of the 5-year categories, all receiving reperfusion therapy.

Acute myocardial infarction based on electrocardiographic entry criteria with angiographic correlation.

ECG criteria

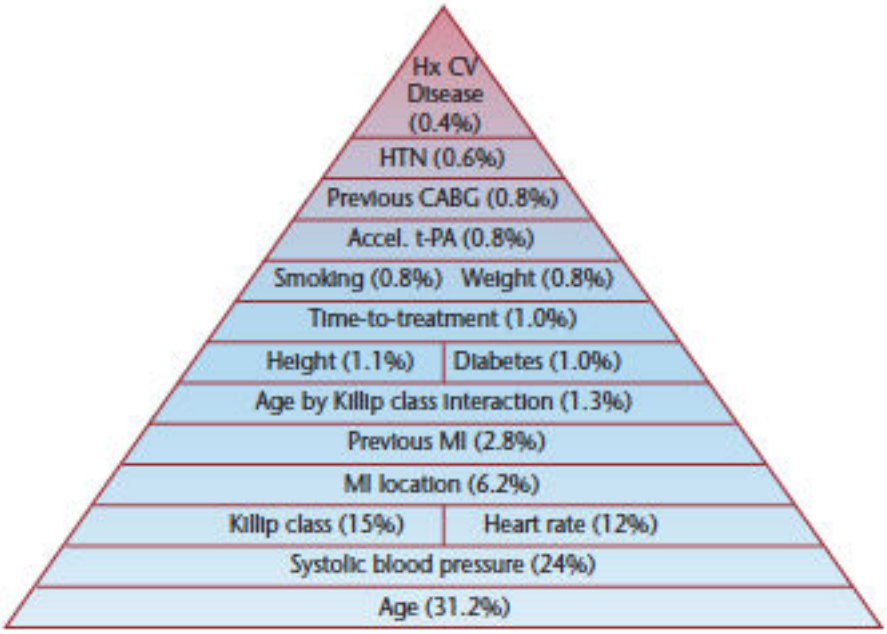
The ECG allows the rough location of the infarct artery

and identification of the extent of the territory at risk. The development of a bundle branch block or atrioventricular block in anterior myocardial infarctions suggests involvement of a proximal septal artery and is associated with increased mortality. Atrioventricular blocks in inferior

myocardial infarctions are frequent and mostly transient.

Biomarkers on presentation

Blood sampling for serum markers must be routinely performed in the acute phase, but one should not wait for the results to initiate reperfusion treatment. The finding of elevated markers of necrosis may sometimes be helpful in deciding to give reperfusion therapy (e.g. in patients with left bundle branch block), but should retard decisionmaking. Elevation of markers of necrosis (troponin) on arrival in hospital is associated with adverse outcome [167].



Multivariate model of 30-day mortality according to GUSTO I trial.

• Multiple proven risk assessment tools utilize information obtained during the history, physical examination, and diagnostic evaluation, which provide an estimate of 30-day mortality following AMI.

• The Killip classification uses bedside physical examination findings including an

S3 gallop, pulmonary congestion, and cardiogenic shock[6]

• The Forrester classification uses hemodynamic monitoring of cardiac index and pulmonary capillary wedge pressure (PCWP)[7]

• The most recent prognostic system, the thrombolysis in myocardial infarction (TIMI) risk score , combines history and examination findings in patients with STEMI treated with thrombolytics.[8]

• This is a different risk score than that used for risk stratification in the setting of UA/NSTEMI.

KILLIP SCORE FOR ACUTE MI :

Class	Definition	Mortality (%)
I	No CHF	6
II	S3 and/or basilar rales	17
III	Pulmonary edema	30–40
IV	Cardiogenic shock	60–80

FORRESTER CLASSIFICATION SYSTEM FOR ACUTE MI :

This System uses PCWP as mainstay :

Class	Cardiac index (L/minute/m²)	PCWP (mmHg)	Mortality (%)
I	≥2.2	<18	3
II	≥2.2	≥18	9
III	<2.2	<18	23
IV	<2.2	≥18	51

But we can use a simplified forrester class : (clinically)

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***

THE THROMBOLYSIS IN MYOCARDIAL INFARCTION (TIMI) SCORE FOR ST ELEVATION MYOCARDIAL INFARCTION (STEMI) :

Criteria	Points
Age > 75 y.o.	3
Age between 65-74	2
Diabetes mellitus or arterial hypertension or angina	1
Systolic blood pressure < 100 mmHg	3
Heart rate > 100 bpm	2
Killip class II-IV	2
Weight < 67 kg	1
Anterior ST segment elevation or left bundle branch block	1
Time to treatment > 4 hours	1

Max points – 14

Minimum Points – 0

Entry criteria:

1. Chest pain for more than 30 minutes.
2. ST elevation.
3. Symptom onset less than 6 hours.
4. Fibrinolytic eligible.

These were important In our discussion and analysis as they form the criteria for inclusion and if not the subjrcts are excluded.

30 day mortality rate based on TIMI score :

0 Points:	0.8%
1 Point:	1.6%
2 Points:	2.2%
3 Points:	4.4%
4 Points:	7.3%
5 Points:	12%
6 Points:	16%
7 Points:	23%
8 Points:	27%
9-14 Points:	36%

It is important to note that cardiac biomarkers especially the cardiac troponins and also the Ejection Fraction (EF) are not included in the scoring system.

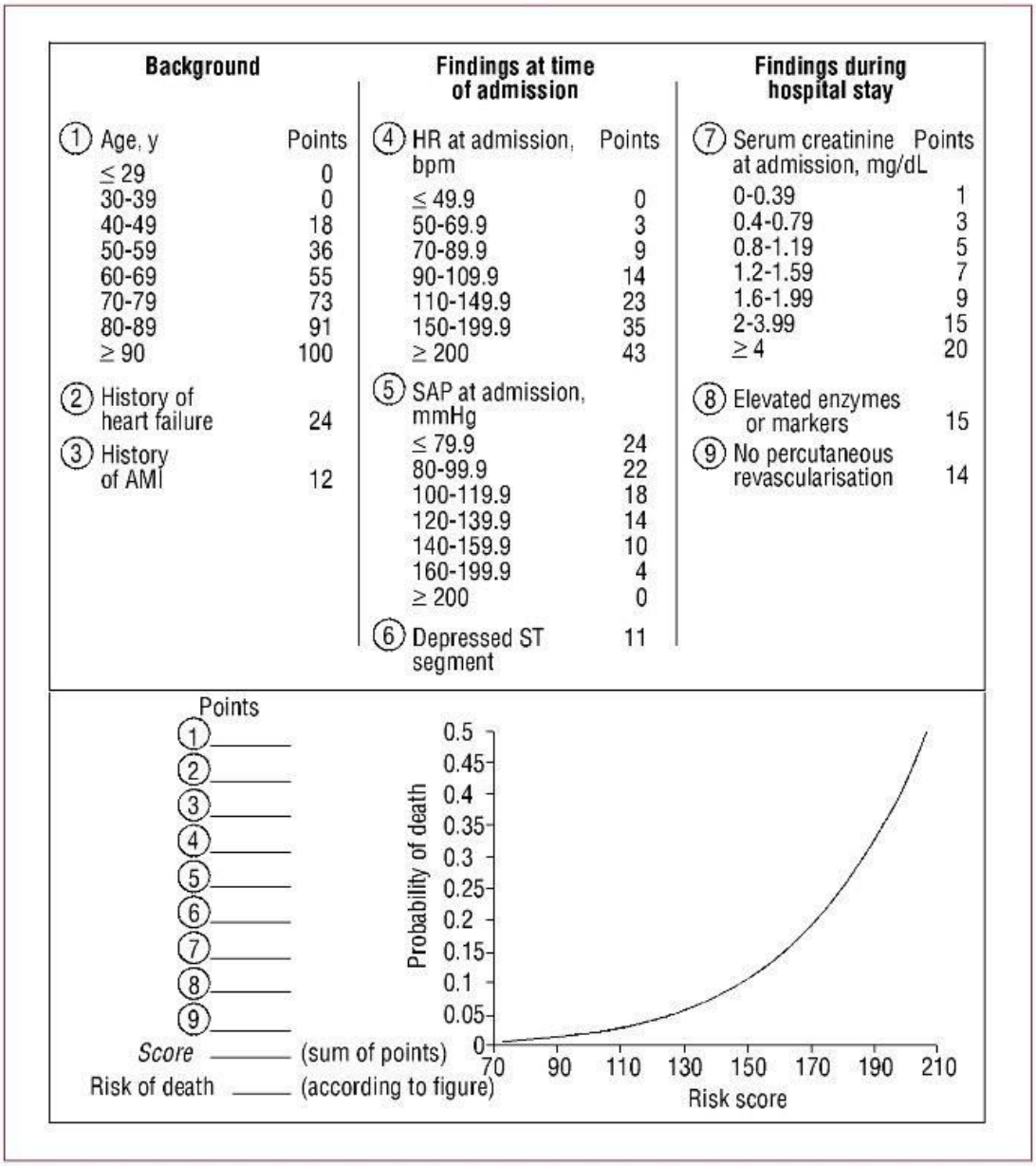
Since the Scoring system provides indirect (Qualitative) ways of assessing them , we wanted to know whether there was any quantitative relationship if at all to provide resource limited clinicians a great information about the predictions .

For example low SBP, tachycardia and Killip 2,3,4 of TIMI score may be a clue towards Low EF.

The same may also predict large infarct and a high troponin levels at 72 hrs .

Our aim is to translate such data into a quantitative one that can be used by all.

The GRACE risk score for ACS (not STEMI alone) :



Background		Findings at time of admission		Findings during hospital stay	
① Age, y	Points	④ HR at admission, bpm	Points	⑦ Serum creatinine at admission, mg/dL	Points
≤ 29	0	≤ 49.9	0	0-0.39	1
30-39	0	50-69.9	3	0.4-0.79	3
40-49	18	70-89.9	9	0.8-1.19	5
50-59	36	90-109.9	14	1.2-1.59	7
60-69	55	110-149.9	23	1.6-1.99	9
70-79	73	150-199.9	35	2-3.99	15
80-89	91	≥ 200	43	≥ 4	20
≥ 90	100	⑤ SAP at admission, mmHg		⑧ Elevated enzymes or markers	15
② History of heart failure	24	≤ 79.9	24	⑨ No percutaneous revascularisation	14
③ History of AMI	12	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		

GRACE score calculates in hospital and 180 day mortality rate.

It also uses other data like

1. Enzyme elevation
2. PCI done/not
3. S. Creatinine
4. ST depression

These are not used in the TIMI score.

The data is as follows according to the literature :

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

CARDIAC BIOMARKERS :

Markers of myocardial necrosis :

Pathohistological studies in patients with unstable angina have disclosed focal cell necroses in the myocardium distal to the culprit artery. These were attributed to repetitive thrombus embolization [10,11]. Focal cell necroses, or so-called minor myocardial injuries, are very infrequently detectable by routine creatine kinase (CK) and CK-MB measurements. Even improved test systems for the quantitative determination of CK-MB based on immunological determination, which are superior to enzyme activity measurements, did not substantially increase the sensitivity for the detection of minor myocardial injury. Myoglobin is a marker which rises

earlier than CK-MB in AMI but has similar limitations with respect to specificity.

These biochemical limitations of CK-MB and myoglobin measurements for the detection of minor myocardial

injury have been overcome by the introduction of troponin measurements .In the early 1990s, the cardiac isoforms of troponin T and troponin I were introduced into clinical practice. The troponin complex is formed by three distinct structural proteins

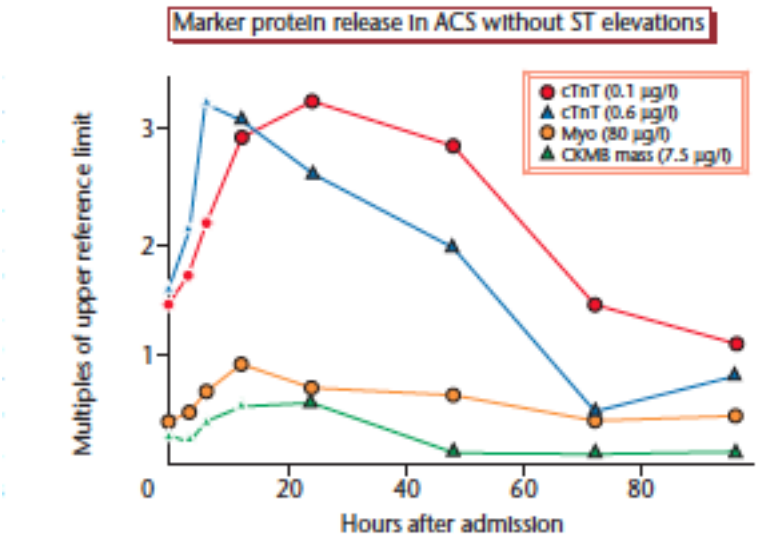
(troponins I, C and T) and is located on the thin filament of the contractile apparatus in both skeletal and cardiac muscle tissue regulating the

calcium-dependent interaction of myosin and actin. Cardiac isoforms for all three troponins, however, are encoded each by different genes and can be distinguished by monoclonal antibodies recognizing the amino acid sequence distinct for the cardiac isoform[12]. However, only the cardiac isoforms of troponin T and troponin I are exclusively expressed in cardiac myocytes.

Accordingly, the detection of cardiac troponin T and troponin I is highly specific for myocardial damage, attributing these markers the role of a new gold standard. In conditions of 'false-positive' elevated CK-MB, such as skeletal muscle trauma, troponins will clarify any cardiac involvement.

In patients with a myocardial infarction, a first rise of troponins in peripheral blood can be observed as early as 3–4 hours because of its release from a cytosolic pool, followed by a prolonged appearance of up to 2 weeks related to continuous proteolysis of the contractile apparatus in the necrotic myocardium. The high proportional rise of troponins, relative to the low plasma troponin concentrations in healthy controls, allows the detection of myocardial damage in about one-third of patients presenting with unstable angina even without elevated CK-MB[13-21.]

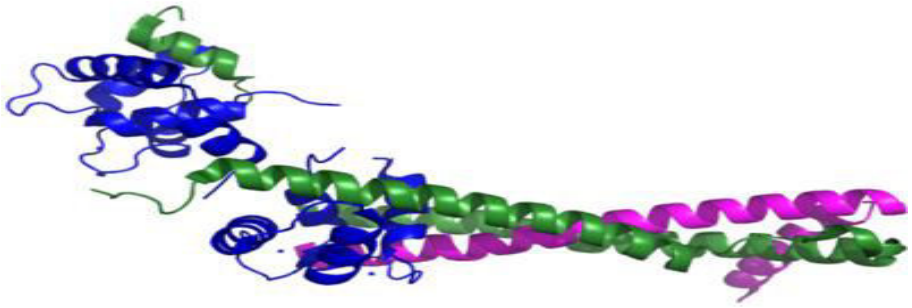
	MW (kDa)	Specificity	Sensitivity	First rise after AMI	Peak after AMI	Return to normal
CK-MB mass	85.0	++	+	4 h	24 h	72 h
Myoglobin	17.8	+	+	2 h	6–8 h	24 h
Troponin T	33.0	+++	+++	4 h	24–48 h	5–21 days
Troponin I	22.5	+++	+++	3–4 h	24–36 h	5–14 days



Important biomarkers used in clinical practice and their rise and fall

Troponins for risk stratification

Troponin T molecule is shown below :



It has been demonstrated in numerous clinical trials that troponin T and troponin I are strongly associated with increased risk both in the acute phase of hospitalization and during long-term follow-up. In the first report on troponin T in a small cohort of patients with

unstable angina, it was demonstrated that the risk of death and myocardial infarction during hospitalization was increased even in the presence of antiplatelet therapy with aspirin and heparin [19.] In a substudy of the FRISC (Fragmin during InStability in Coronary artery disease) trial the prognostic value was shown to correlate with the absolute concentrations of troponin T over a 5-month period [19]. The peak value during the first 24 hours provided the best independent prognostic information and the absence of troponin T was superior to CK-MB for identification of the low-risk group [22]. Furthermore, the combination of the troponin T test with a predischarge exercise test represents an excellent risk assessment for

unstable coronary disease[23]. During the 5-month follow-up, death and

myocardial infarction occurred at a rate of only 1%, if both the troponin test and the predischarge exercise test were normal, whereas the event rate was as high as 50% when both tests were abnormal.

Moreover, the prognostic potential of troponin T in the entire spectrum of patients with ACS, including myocardial infarctions, was evaluated in a substudy of the GUSTO (Global Use of Strategies To Open occluded coronary arteries) IIA trial[24]. A single measurement within 2 hours after admission was highly predictive of 30-day mortality and other major complications. The prognostic value was independent of ECG findings and was superior

to CK-MB measurements.

For elevations of cardiac troponin I, a similar prognostic impact was evidenced as for troponin T elevations. In the TIMI IIIB trial including patients with unstable angina and non-Q-wave myocardial infarction, the mortality rate was closely related to troponin I levels reaching 7.5% after 42 days follow-up in patients with the highest

troponin I values[20].

“IT HAS NOT BEEN PROVEN IN ANY TRIAL THAT ONE TROPONIN IS SUPERIOR TO ANOTHER “

Currently, the diagnostic threshold for troponin T may be maintained between 0.06 and 0.10 μg/l, depending on the local laboratory performance.

CAUSES OF ELEVATED TROPONINS OTHER THAN MYOCARDIAL INFARCTION :

1. Myocarditis
2. Pericarditis
3. Cardiac contusion /trauma
4. Aortic dissection
5. Endocarditis
6. Cardiac Surgery
7. Pulmonary Embolism
8. Stroke
9. CPR
10. Defibrillation
11. Chronic Severe Heart Failure
12. Cardiac Arrhythmias
13. Sepsis
14. Renal failure
15. Burns

16. HOCM
17. Takatsubo cardiomyopathy
18. Extreme exertion
19. Post Cardiac Surgery
20. Infiltrative diseases like amyloidosis
21. Transplant Vasculopathy
22. Post PCI
23. Post CABG
24. Medications – Doxorubicin, Trastuzumab , Snake venom
25. Critical Illness

Note that the troponin elevation In unstable angina is very minimal to be detected in routine testing (current lab standards). So by definition UA does not cause DETECTABLE troponin elevation rather than it does not mean that they donot rise.

LV EF as a Predict0r of Increased/Higher Risk :

The risk of mortality that was associated with a decreased LV-EF after Myocardial Infarction and especially after a ST Elevation MI has been recognized for long .An LV Ejection fraction of < 40% was found to be an independent mortality predict0r in the MPR group ar0und the 1980's.[26] During the 1990's, in the CAMI study- Canadian Assessment of MI , the odds ratio 1 year mortality after MI was 9.28 for patients with LV Ejection Fraction ≤ 40% compared with the patients with a LV Ejection Fraction > 50%, 2.64 for patients with LV Ejection Fraction of 30–40%, and however the risk was not at all significantly elevated in patients with LV Ejection fraction of 40–50%[27]. Thus, in a overview there tends to be a rough LV Ejection Fraction threshold of ~40% for an elevated risk post-MI .

More latest studies have confirmed this. In the ATRAMI study – Aut0nomic T0ne and Reflexes After Myocardial Infarction which has enr0lled 1286 patients with recent MI, patients with LV-EF of 35–50% had a relative risk of 2.48 for mortality related to cardiac component compared with patients with LVEF > 50%, whereas in patients with LVEF < 35%, the relative risk was 7.3.[28]

"Left ventricular ejection fraction, thus is a strong predictor of arrhythmic death. Direct evidence for this has been provided by the randomized trials

of ICD implantation for primary prevention in patients with low LV-EF. For instance, the Multicenter Automatic Defibrillator Implantation Trial II (MADIT-II) randomized 1243 patients with previous MI and LV-EF ≤30% to ICD or conventional medical therapy[29]." During average period of follow-up of 20 months, the mortality rate was significantly lower in the defibrillator group (14.2 vs. 19.8%).

"Similar findings were provided by the Sudden Cardiac Death in Heart Failure Trial (SCD-HeFT), which randomized 2531 patients with NYHA class II or III heart failure and LVEF ≤ 35% to placebo, amiodarone, or a single-lead ICD in addition to conventional therapy[30]. The cause of heart failure was ischaemic in 52%. ICD therapy was associated with a decreased risk of death of 23% without interaction with the heart failure cause. Since the ICD affects only arrhythmic death, this mortality reduction is attributed to a reduction in Sudden Cardiac Death.

The ICD trials were particularly designed for Primary prevention to actually evaluate the importance of ICD in people with high risk , defined by decreases LV Ejection fraction, and *not* to look for various variables including LV Ejection Fraction as risk stratifiers. "Thus, they sh0w that a reduced LV Ejection Fraction is associated with an elevated SCD risk and that Implantable cardioverter Defibrillator therapy improves survival, but usually don't establish LV-EF as optimal risk stratification variable."

The increased Sudden Cardiac Death risk 0f patients with reduced LV-EF is confirmed by studies. Thus, in a multivariable analysis of the Multicenter Unsustained Tachycardia Trial (MUSTT), reduced LV-EF was associated with increased risk for arrhythmic death or cardiac arrest.[31]

"Recent studies have replicated these findings: In the ISAR-Risk, which studied 2443 acute Myocardial Infarction survivors, an LVEF ≤ 30% predicted all-cause mortality, cardiac mortality, and Sudden Cardiac Death at 5 years[32]." Sensitivity and specificity for Sudden Cardiac Death prediction were 22.1 and 95.4%, respectively. However, the positive predictive accuracy was only 12.0%. The Risk Estimation Following Infarction, Noninvasive Evaluation (REFINE) cohort study evaluated the utility of a combined assessment of the different risk stratifiers in 332 patients early after Myocardial Infarction with LV-EF < 50% for prediction of cardiac death or resuscitated cardiac arrest.[33"] A reduced LV-EF (≤30%) was a good predictor for cardiac death or resuscitated cardiac arrest (hazard ratio 3.3, $P = 0.005$). However, the area under the receiver-operating characteristic curve was moderate (0.62) indicating the limitations of LV-EF as a sole predictor."

“Thus in short , the LV Ejection Fraction < 40% can be taken as a independent risk factor for mortality but with a pinch of salt. “

Since TIMI score equates to mortality data at 30 days and also uses variables that predict Low EF , our goal is to find out if there are any quantitative relationship between both.

SUMMARY

ACS is a multifactorial disease of utmost emergency. The subtype ST Elevation MI is a cardiac/medical emergency and prompt action is essential as it has a very high in hospital and equally high long term morbidity/mortality. Risk Stratification forms the crux of management protocol since one has to know which patients are likely to develop serious and essentially life threatening complications.

There are lot of individual variables like Troponin T quantitative , Age , Angiographic localization ,Ejection fraction etc and there are scores (Forrester, TIMI score , GRACE score) which bring one or more individual variables together into the scoring system to form the risk assessement strategy.

Of note , the TIMI score is the one which is entirely clinical and bedside (except for the ECG – the only investigation , which in turn can also be done bedside) .

The TIMI score for STEMI traditionally calculates the 30 day mortality rate and it also uses clinical data that are indirect markers of infarct size and Cardiac function.

So, Our goal is to find out any quantal relationship existing between TIMI score for STEMI and these individual investigation related

variables and of note the TROPONIN T and LV EJECTION FRACTION <40%. In other wors our goal is to find out the relationship between bedside data and laboratory parameters , but rather quantitatively.

These relations if existing and if delineated , can provide a ital. clue to the clinician to determine the trop t level and patients with LV-EF <40% at the time of admission itself !!

MATERIALS & METHODS

STUDY POPULATION :

New patients admitted with ACS- Acute ST Elevation Myocardial Infarction in the casualty of Govt Kilpauk Medical College and Hospital , Chennai.

STUDY TYPE :

Cross sectional Study. (Descriptive)

STUDY PERIOD :

Data collection done for a period of 6 months between Dec 2014 to May 2015.(over a period of 6 months)

PLACE OF STUDY :

Govt .Kilpauk Medical College and Hospital

Sample Size : 50

DEFINITION :

We use WHO citeria to define STEMI :

1. Evidence of ST elevation in ECG (0.1 mm ST elevation In atleast 2 contiguous leads)
2. Ischemic type of chest pain . (central – compressing/crushing/burning type of pain with or without radiation to Left or both shoulders/ arms / hands , jaw forehead etc)
3. Rise and fall of cardiac markers .

Supporting evidence – Echocardiographic evidence of new regional wall motion abnormalities, Q waves in ECG and/or angiographic evidence of vessel obstruction) .

(1) is a must with at least 1 of the other criteria with atleast one supporting evidence – chosen by us for convenience of this study .

ENTRY CRITERIA : (for a patient to be eligibile for TIMI score calculation)

1. Chest pain for more than 30 minutes.
2. ST elevation.
3. Symptom onset less than 6 hours.
4. Fibrinolytic eligible.

INCLUSION CRITERIA :

All new/fresh patients in the age group > 18 years presenting with acute ST Elevation Myocardial Infarction meeting the entry definition / entry criteria and getting admitted in Govt. Kilpauk Medical College and hospital .

EXCLUSION CRITERIA :

1. Age < 18 years
2. Prior History/ records of ACS .
3. Prior History of PCI/CABG
4. Evolved MI
5. NSTEMI/UA

6. Patients with history /records suggestive of heart failure.
7. Patients with history /records suggestive of CKD
8. Patients with history / records suggestive of or with new evidence of Cardiomyopathies.
9. Severe burns
10. Severe Sepsis
11. Suspected cardiac trauma
12. Suspected aortic dissection
13. Suspected myocarditis/pericarditis
14. Post Cardiac Transplantation.
15. Symptom duration > 6 hours
16. Chest pain < 30 minutes
17. Fibrinolytic ineligible or patients with contraindications to fibrinolysis.
18. Patients who are not willing to participate in the study or from whom we cannot obtain consent.

METHODOLOGY :

1. The data of each patient will be collected in a specific proforma (ANNEXURE 2) which includes patient's name , age, sex, demographic details , presenting complaints, risk factors and all the clinical data.

2. Baseline clinical data will be collected from all patients including proper history- History of Diabetes , Hypertension , angina , Chest pain duration and character, associated symptoms and other necessary history.
3. The collected data of each patient will be collected in a specific proforma (ANNEXURE 1) which includes patient's name , age, sex, demographic details , presenting complaints, risk factors and all the clinical data.
4. The ECG and if necessary a 2D Echocardiogram will be performed for all patients to confirm STEMI.
5. Blood sample will be collected at around 12 hours post admission in all STEMI patients and Troponin T Quantitative levels will be determined by ECLIA (ElectroChemiLuminescent Assay) method and will be entered in the proforma . Fasting Lipid Profile will also be sent and the results entered in a proforma.
6. Reference normal Troponin T is <0.01 ng/mL
7. Repeat 2D Echocardiogram will be done for all patients , 48 hours post admission to determine EF and the value will then be recorded in the proforma.
8. All the relevant data and values are then entered In a masterchart in Microsoft excel Format and then analyzed satistically.

STATISTICAL ANALYSIS

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

The collected data was analysed with SPSS 16.0 version.To describe about the data descriptive statistics frequency analysis, percentage analysis were used for categorical variables and the mean & S.D were used for continuous variables. To find the significant difference between the bivariate samples in Independent groups the Mann-Whitney U test was used. To assess the relationship between the variables Spearman's rank Correlation was used. In both the above statistical tools the probability value .05 is considered as significant level.

RESULTS

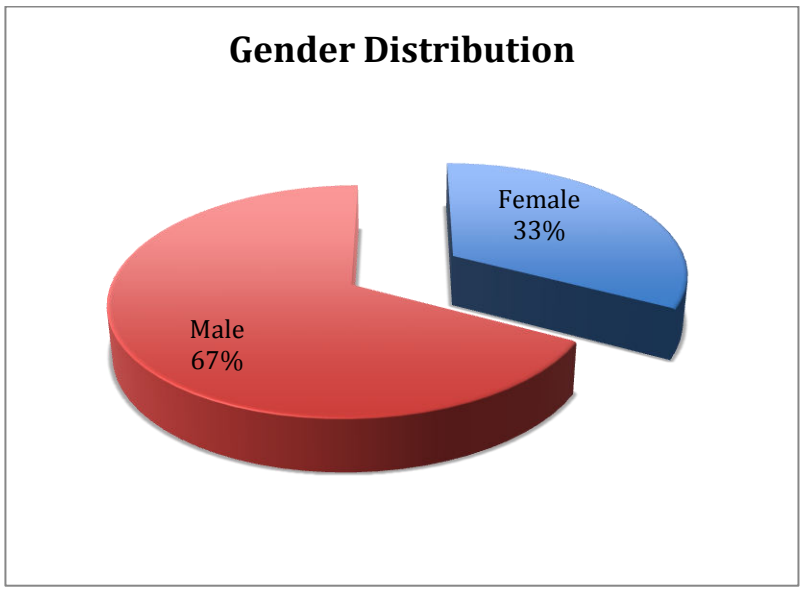
The total patients recruited in our study were 52. The following charts depict frequency distributions.

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

GENDER DISTRIBUTION :

In a total of 52 patients enrolled in our study 17 were females and 35 were males and it clearly shows the predominance of males in STEMI.

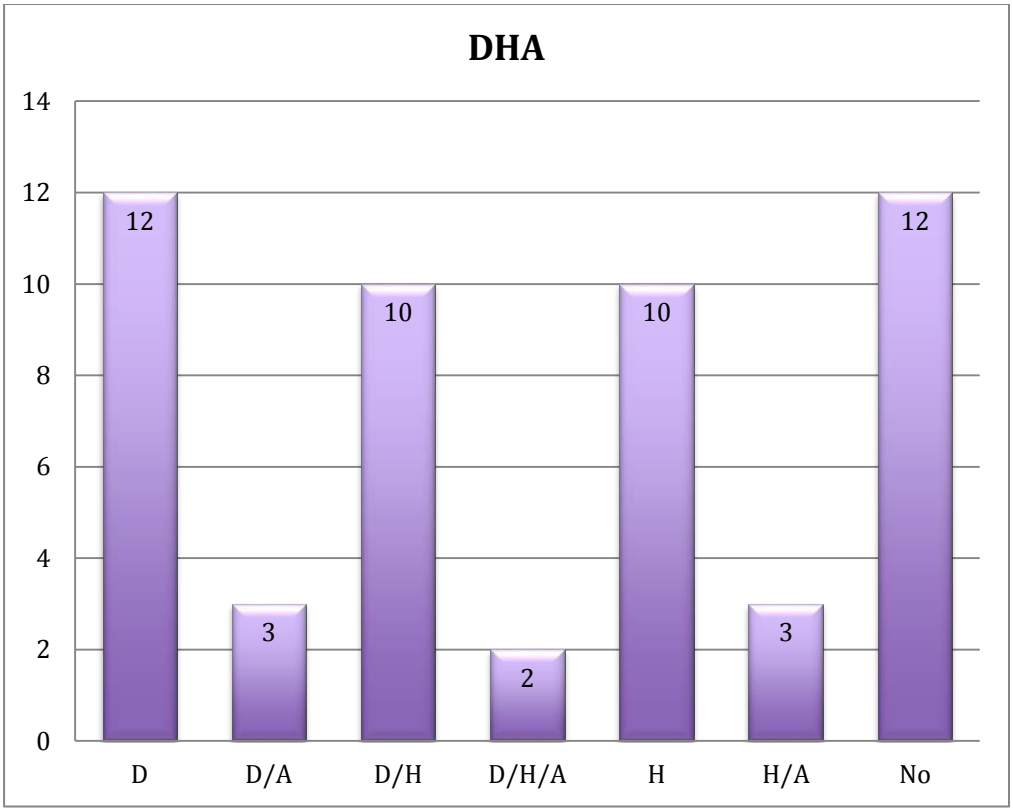
	Frequency	Percent
Female	17	32.7
Male	35	67.3
Total	52	100.0



2. **DISTRIBUTION OF DIABTES (D) , HYPERTENSION(H) AND ANGINA(A) PATIENTS (ALONE AND IN COMBINATION** :

In our study the frequency of diabetes alone was higher together with patients having neither diabetes , hypertension or angina .

	Frequency	Percent
D	12	23.1
D/A	3	5.8
D/H	10	19.2
D/H/A	2	3.8
H	10	19.2
H/A	3	5.8
No	12	23.1
Total	52	100.0

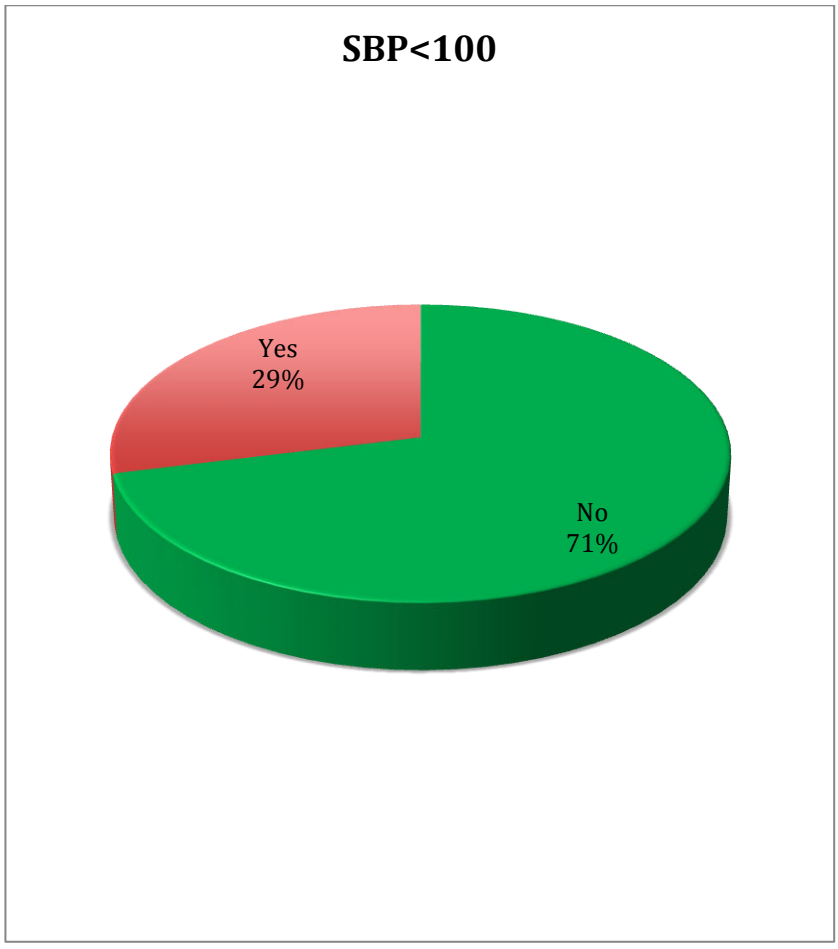


Key : D- Diabetes , H- Hypertension, A- Angina.

3. PATIENTS WITH SYSTOLIC BP < 100 mm of Hg :

15 patients (28.8%) had a systolic BP of < 100 mm Hg .

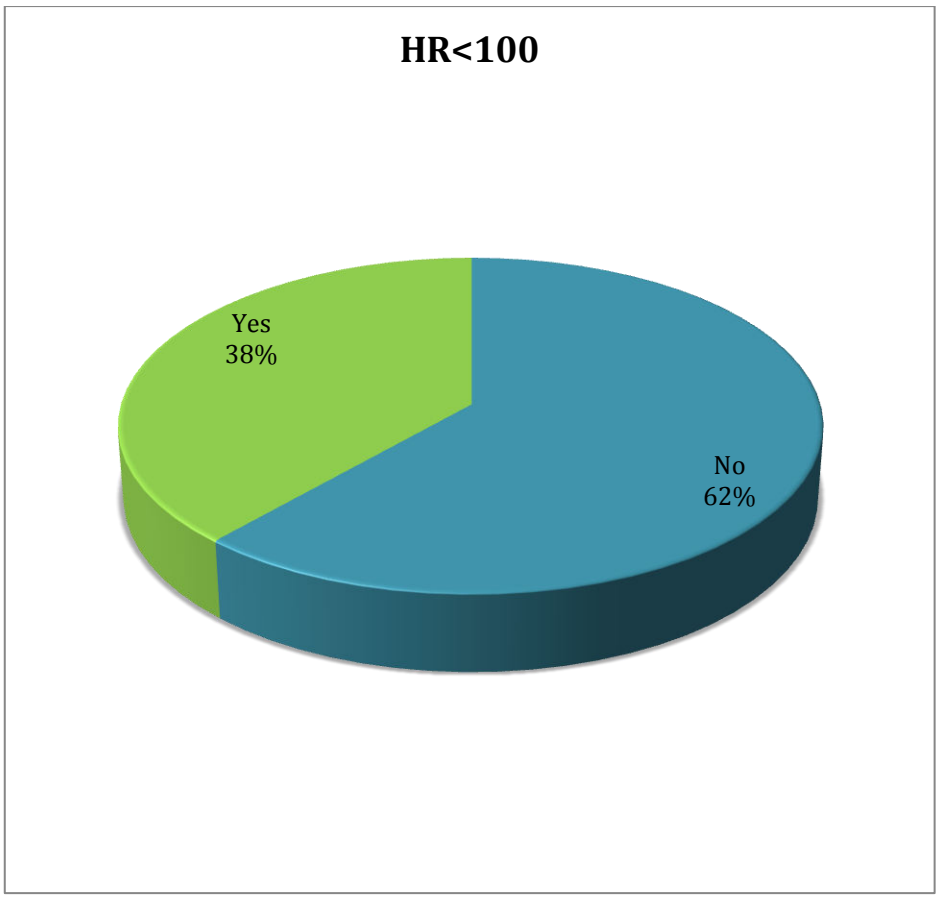
	Frequency	Percent
No	37	71.2
Yes	15	28.8
Total	52	100.0



4. PATIENTS WITH HEART RATE < 100 bpm :

20 patients (38.5%) of patients had a Heart rate of > 100 beats per minute.

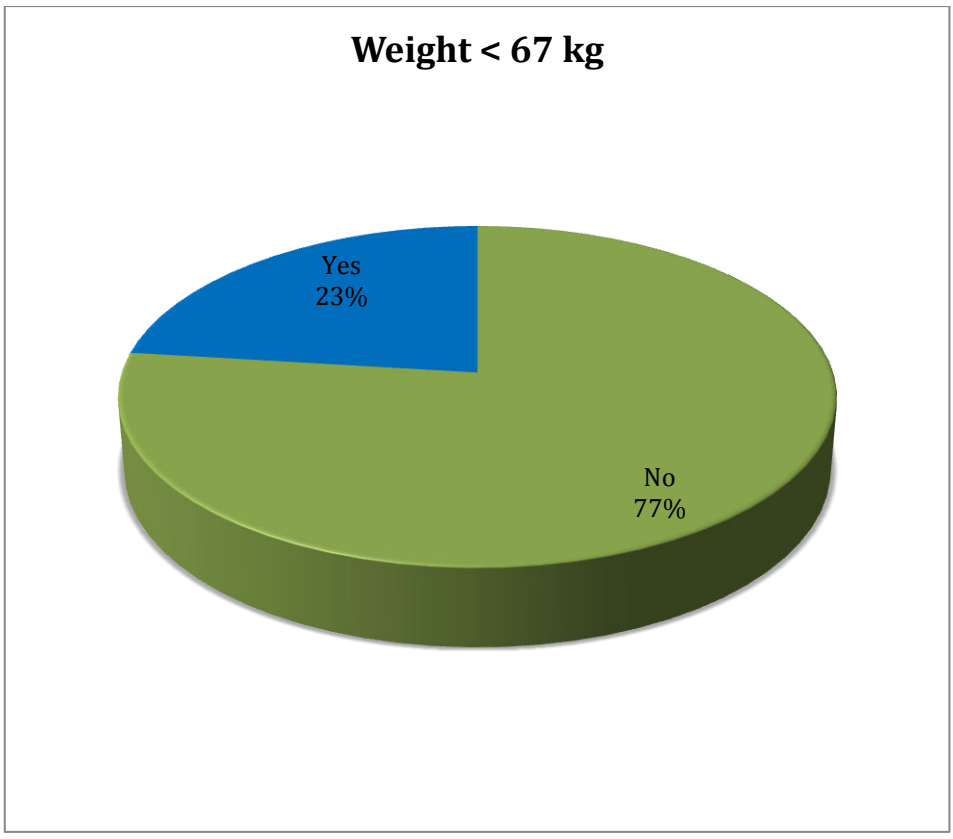
	Frequency	Percent
No	32	61.5
Yes	20	38.5
Total	52	100.0



5. PATIENTS WITH WEIGHT <67kg :

12 patients (23.1%) had weight < 67 kg.

	Frequency	Percent
No	40	76.9
Yes	12	23.1
Total	52	100.0



6. KILLIP CLASS 2,3.4 :

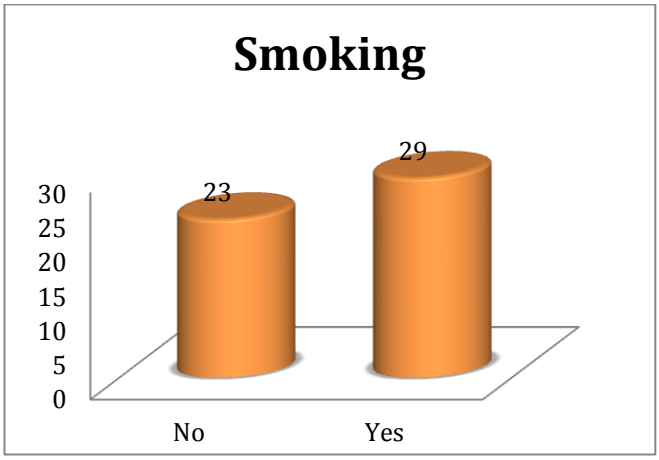
25 patients (48.1%) had a killip score of 2,3 or 4 .

	Frequency	Percent
No	27	51.9
Yes	25	48.1
Total	52	100.0

7. PATIENTS WHO SMOKE :

29 patients out of 52 (55.8%) were smokers .

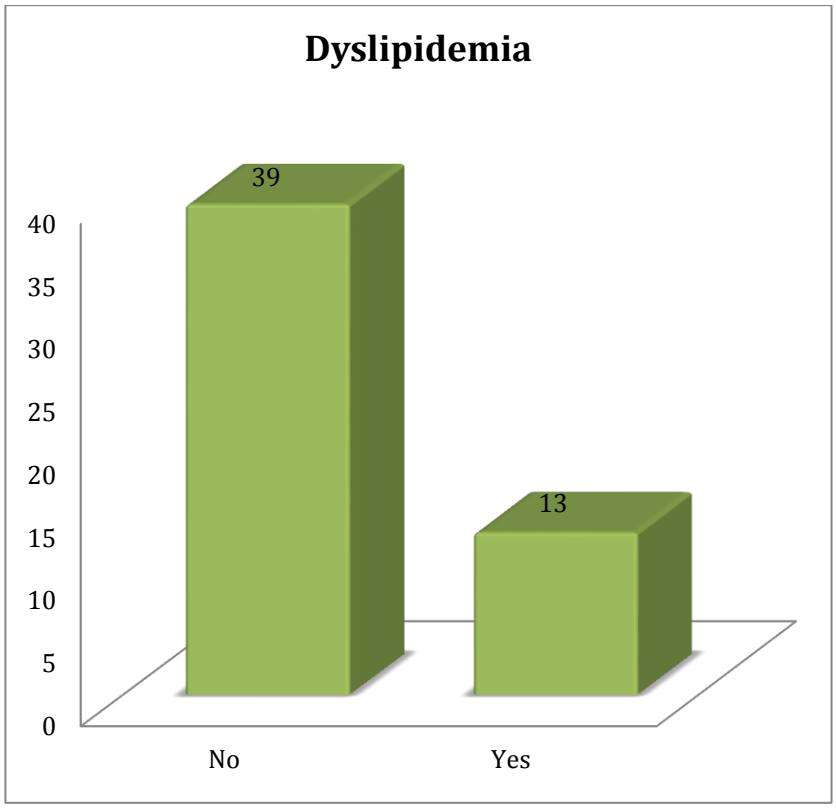
	Frequency	Percent
No	23	44.2
Yes	29	55.8
Total	52	100.0



8. PATIENTS WHO ARE DYSLIPIDEMIC :

13 patients (25%) in our study were dyslipidemic.

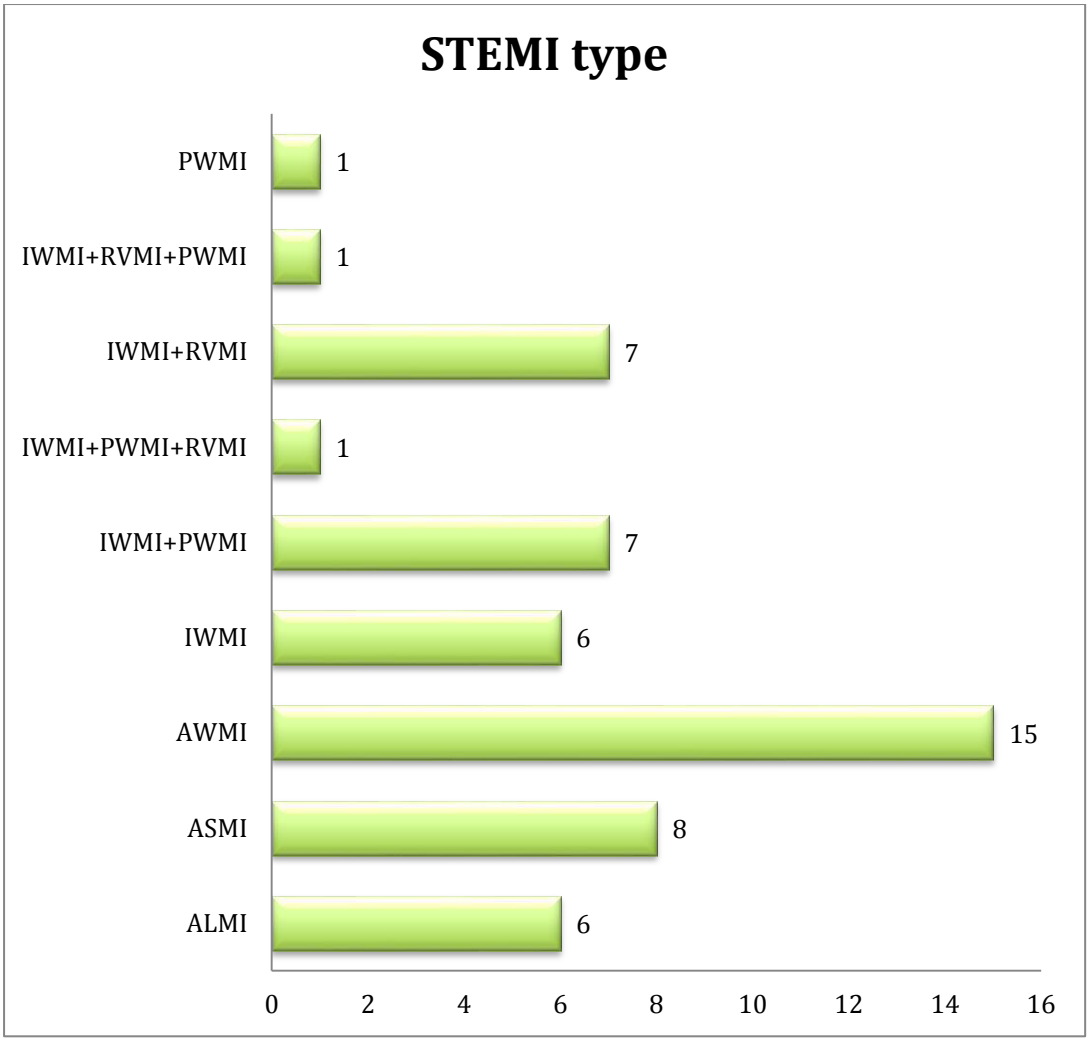
	Frequency	Percent
No	39	75.0
Yes	13	25.0
Total	52	100.0



9. STEMI TYPE :

Out of 52 patients 22 patients were found out to be Inferior Wall MI or combinations of IWMI and 15 were found to have Anterior wall MI and only 1 patient had an isolated Posterior Wall MI.

	Frequency	Percent
ALMI	6	11.5
ASMI	8	15.4
AWMI	15	28.8
IWMI	6	11.5
IWMI+PWMI	7	13.5
IWMI+PWMI+RVMI	1	1.9
IWMI+RVMI	7	13.5
IWMI+RVMI+PWMI	1	1.9
PWMI	1	1.9
Total	52	100.0



KEY : PWMI- Posterior wall Myocardial Infarction

RVMI- Right Ventricular Myocardial Infarction

IWMI- Inferior Wall Myocardial Infarction

AWMI- Anterior Wall Myocardial Infarction

ALMI- AnteroLateral Myocardial Infarction

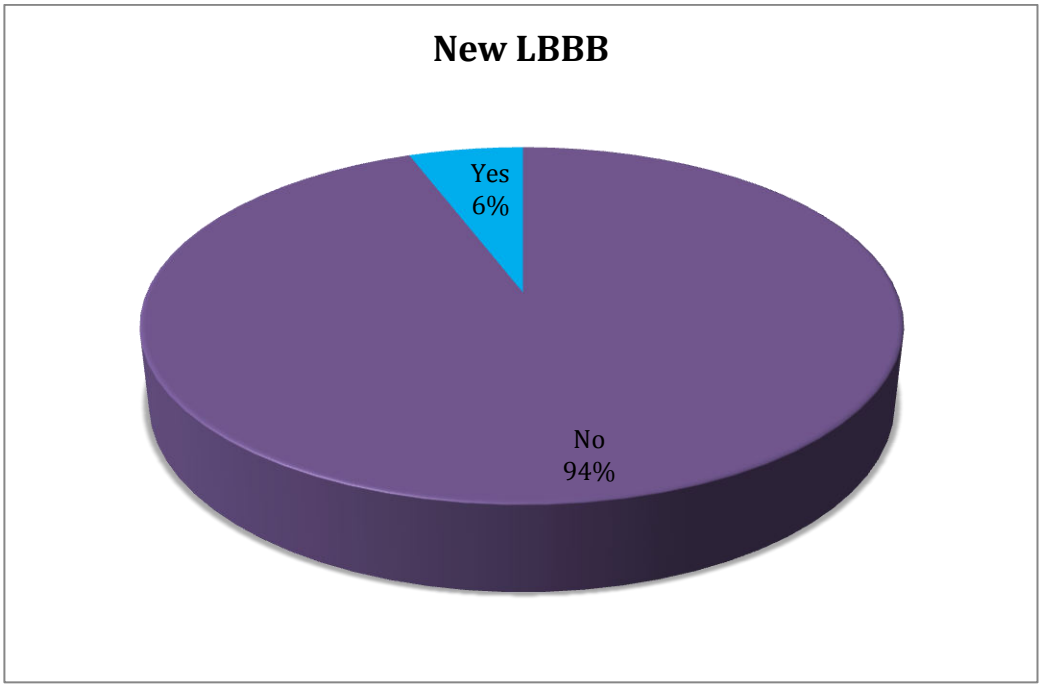
ASMI- AnteroSeptal Myocardial Infarction

STEMI- ST Elevation Myocardial Infarction

10. PATIENTS WITH NEW LBBB :

Only 3 patients in our study had a new onset LBBB (Left Bundle Branch Block) in ECG.

	Frequency	Percent
No	49	94.2
Yes	3	5.8
Total	52	100.0



11. PATIENTS WITH TIME TO TREAT > 4 HOURS

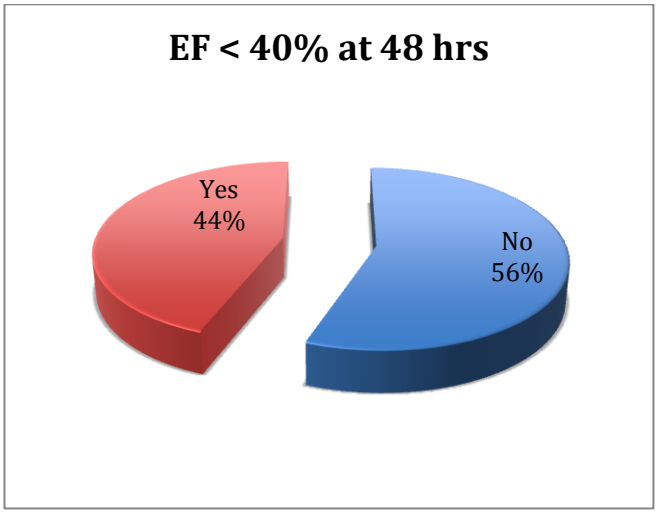
In our study all the enrolled patients were treated with thrombolysis before 4 hours .

	Frequency	Percent
No	52	100.0

12. PATIENTS WITH EJECTION FRACTION < 40% :

23 patients (44.2%) in our study had an EF of < 40%.

	Frequency	Percent
No	29	55.8
Yes	23	44.2
Total	52	100.0

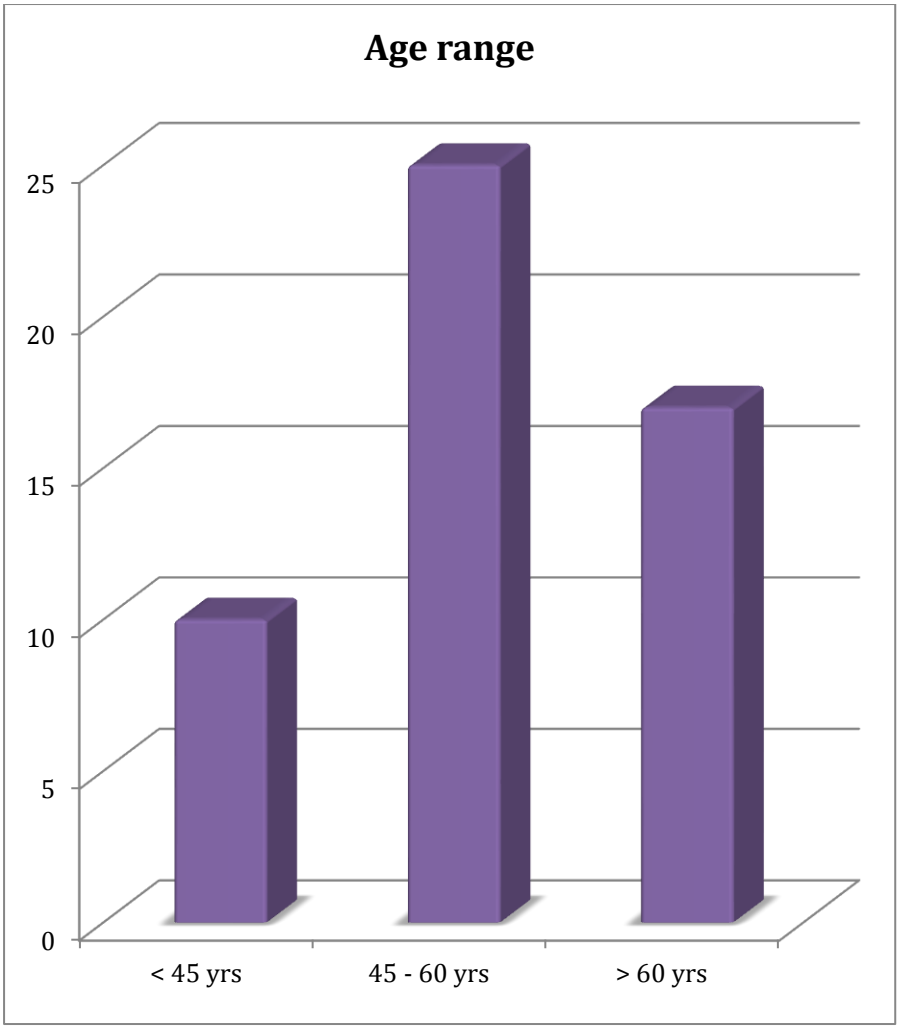


13. AGE RANGE :

The age range in our study id depicted in the following table and bar diagram.

	Frequency	Percent
< 45 yrs	10	19.2
45 - 60 yrs	25	48.1
> 60 yrs	17	32.7
Total	52	100.0

It can be noted that the maximum events in our study occurred in the 45-60 age group .



Age range – Note thst the maximum number of events occurred in the 45-60 age roup in our study (NB:This may be biased as the we take in to account only the number of patients reaching the hospital . It does not take into account the patients who do not report after an MI or patiemts who die before reaching the hospital)

14. THROMBOLYSIS IN MYOCARDIAL INFARCTION SCORE :

	Frequency	Percent
0	6	11.5
1	6	11.5
2	7	13.5
3	3	5.8
4	9	17.3
5	2	3.8
6	7	13.5
7	2	3.8
8	2	3.8
9	6	11.5
10	2	3.8
Total	52	100.0

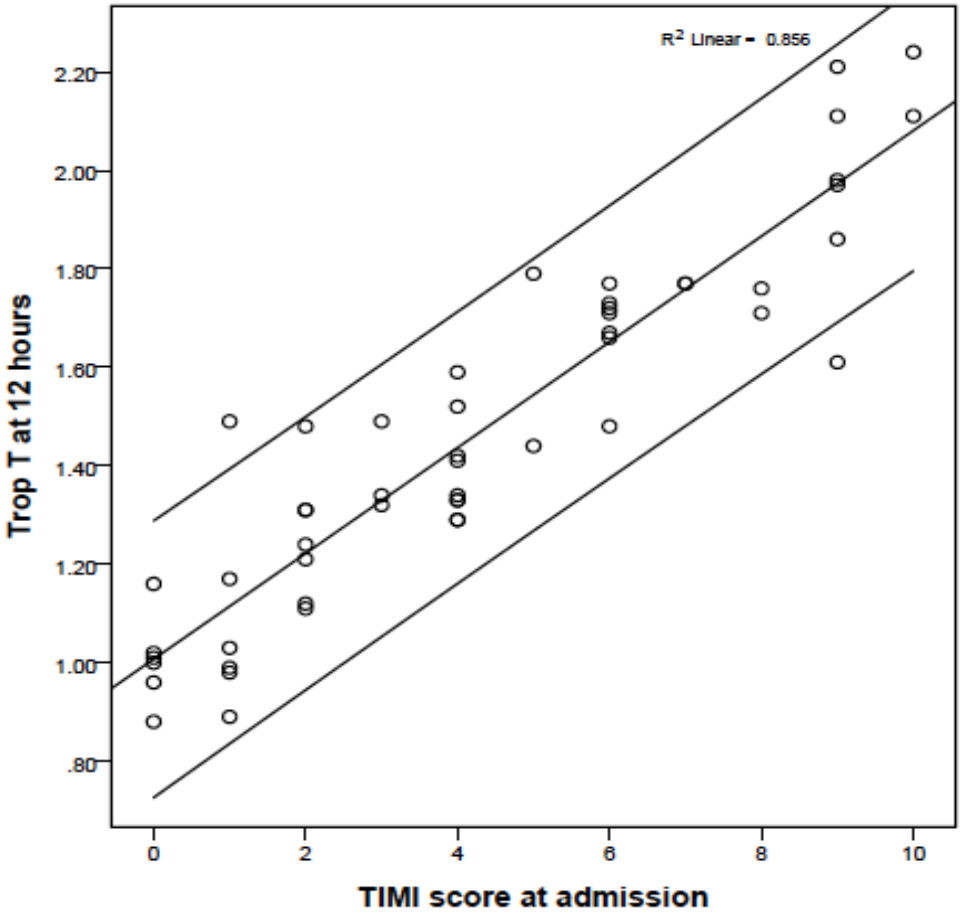
STATISTICAL ANALYSIS :

The total patients enrolled in our study were 52 . The maximum age is 76 and the minimum is 36. In the male patients maximum age recorded was 71 and the minimum age was 36 where as in the females it is 76 and 42 respectively.

1. CORRELATION OF TIMI SCORE AT THE TIME OF ADMISSION AND SINGLE QUANTITAIVE TROPONIN T AT 12 HOURS :

TIMI score for acute STEMI which was calculated at the time of admission was correlated with Single Quantitative Troponin T at 12 hours using non parametric correlations using correlation coefficient . It was then statistically tested using spearman's rho (sig two tailed) and the correlation is taken to be significant at a p value of 0.05 .

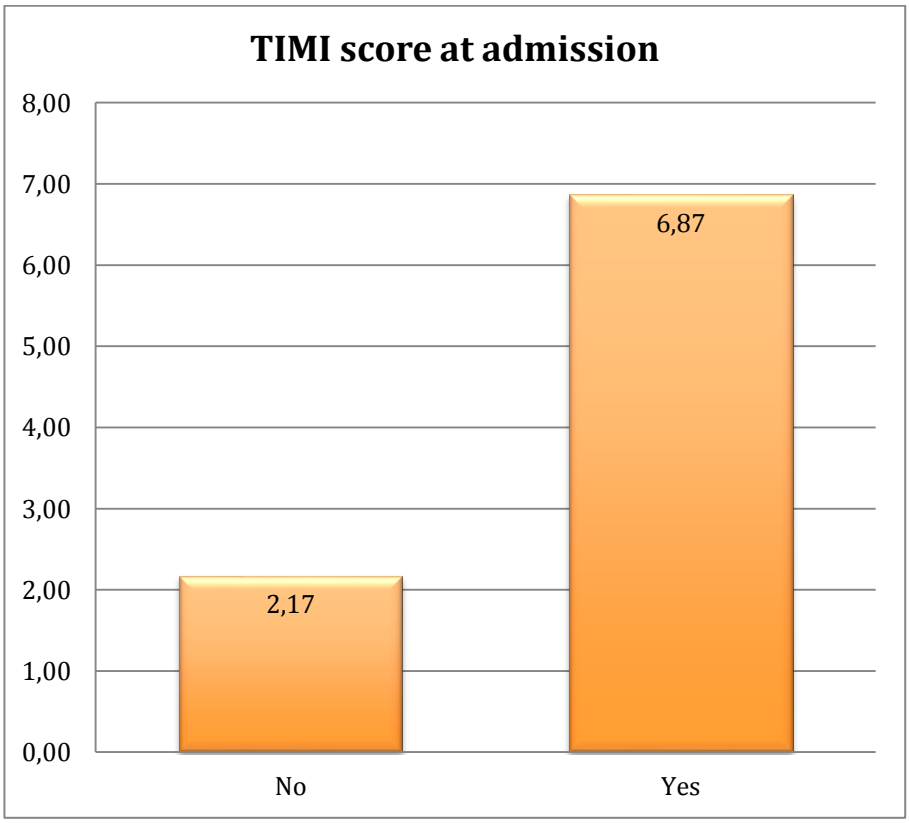
In our study the correlation between TIMI score for acute STEMI at the time of admission and with Single Quantitative Troponin T at 12 hours was found to be highly significant at p=0.005.



From the graph , it was evident at that the correlation between TIMI score at the time of admission and TIMI score at 48 hours is statistically significant .

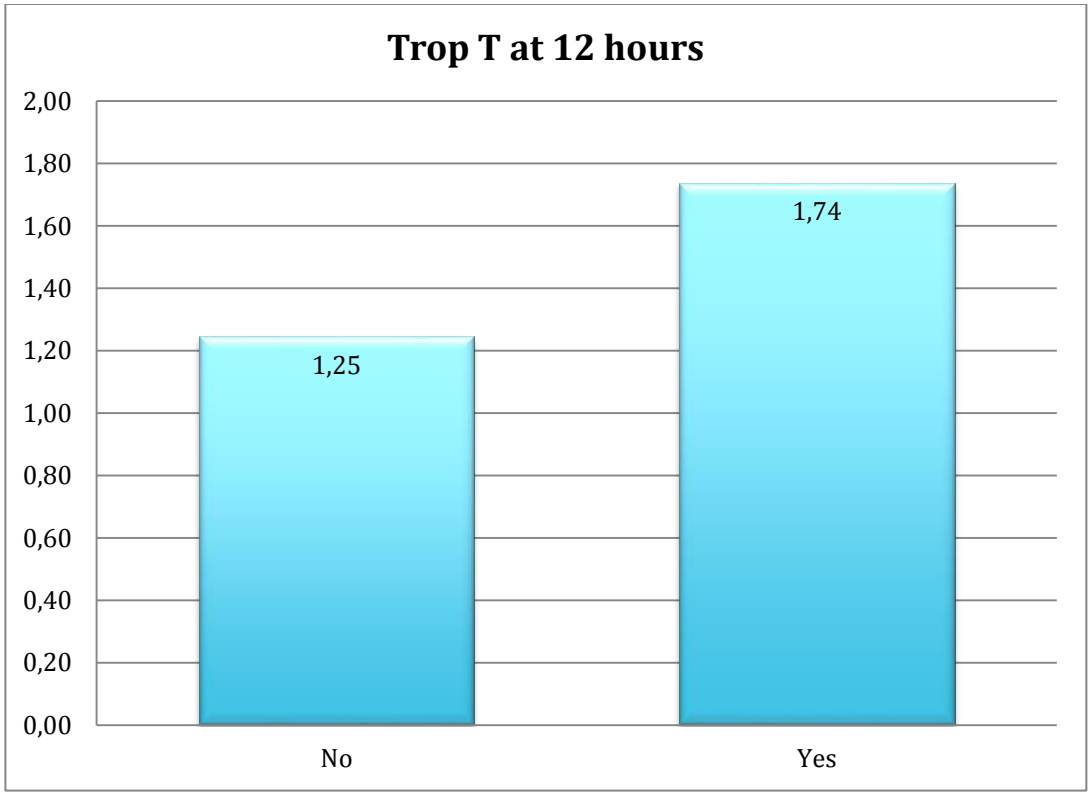
2. ASSOCIATION BETWEEN TIMI SCORE AT THE TIME OF ADMISSION AND EJECTION FRACTION < /> 40 % at 48 hours :

The association between TIMI score at the time of admission and EF < 40 % was tested using Mann – Whitney U test. Those with EF> 40% at 48 hours had a mean TIMI score of 2.17 during admission and those with EF <40% at 48 hours had a mean TIMI score of 6.87 during admission . 23 patients had a EF of < 40% at 48 hours and 29 patients had a EF>40% at 48 hours. The association was found to be statistically significant at p=0.005.



3. ASSOCIATION BETWEEN TROPONIN T AT 12 HOURS AND EJECTION FRACTION < /> 40% AT 48 HOURS :

The association between Troponin T at 12 hours and Ejection fraction < 40 or >40% was tested again using Mann Whitney U test. Those with Ejection fraction<40% at 48 hours had a mean Troponin T of 1.74 ng/mL at 12 hours and those with EF > 40% at 48 hours had a mean Troponin T level of 1.74 at 12 hours. The association was found o be statistically significant at p=0.005.



The association between all three variables is depicted in the chart below.

EF <40%	**Mean TIMI score at admission**
No	2.17
Yes	6.87

	Mean Trop T at 12 hours
No	1.25
Yes	1.74

All the aforementioned statistics confirms that the correlation between

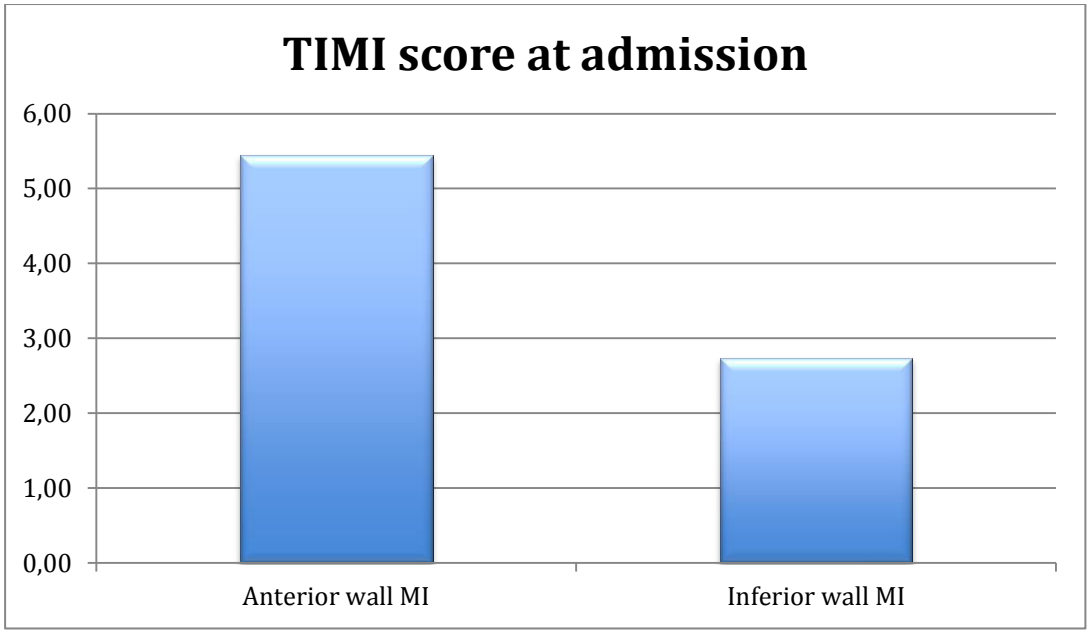
1. TIMI score at the time of admission and Troponin T at 12 hours
2. The association between Troponin T at 12 hours and EF <40% at 48 hours
3. The association between TIMI score at the time of admission and EF<40% at 48 hours.

These are also found to be highly significant statistically at p=0.005.

Further Statistical data were obtained to confirm the results and exclude the possible confounders like smoking and duslipidemia .

1. **CORRELATION BETWEEN TIMI SCORE AT THE TIME OF ADMISSION AND THE TYPE OF MI :**

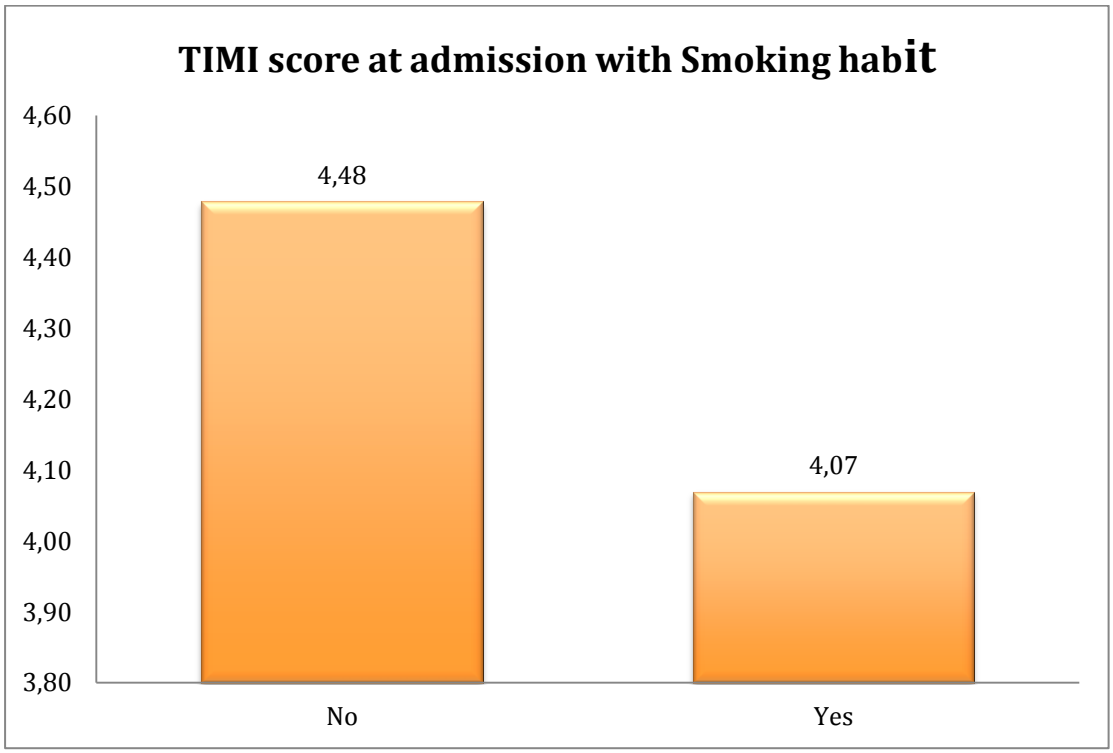
The TIMI score at the time of admission was correlated with the type of MI . First MI was classified into two types- those involving anterior wall (AWMI,ASMI.ALMI) and those involving Inferior wall (IWMI,IWMI+RVMI, IWMI+RVMI+PWMI,IWMI+PWMI, isolated PWMI). This analysis was actually done to find out the correlation as well as VALIDATE THE TIMI SCORE with its variables as one of the variable is the presence of AWMI. The mean TIMI score for 5.45 for AWMI's and 2.74 for IWMI's at the time of admission. The results were tested using Mann Whitney U test and was found to be statistically significant at p=0.001.



And also since points awarded for AWMI was only 1 in the TIMI score , the difference in our study was actually around 2.71. This maybe due to the fact that AWMI is usually severe with involvement of other variables like SBP, HR amd Killip class used in the TIMI score. That might need a separate Study and further evaluation.

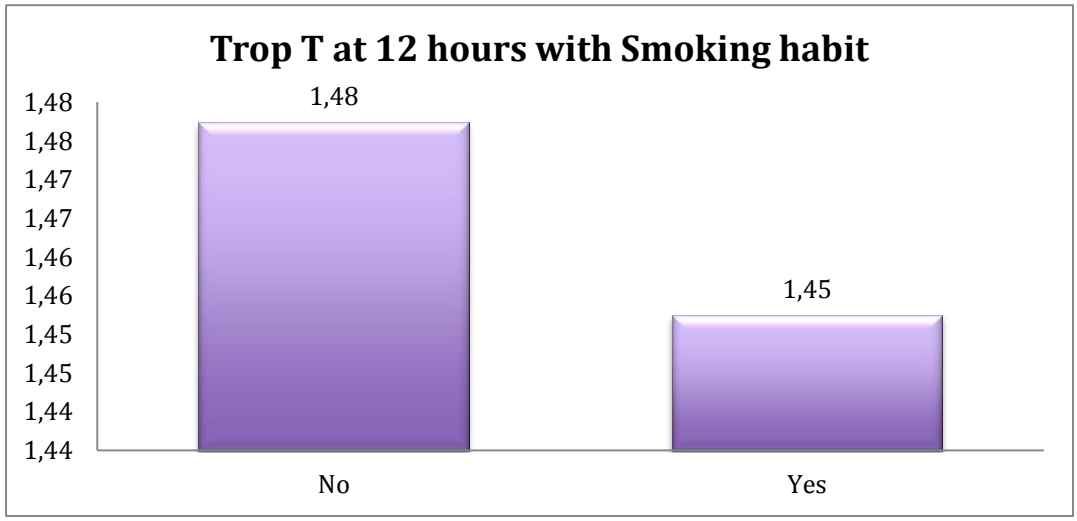
2. TIMI score and its association with Smoking :

This was done actually to find out smoking as a potential confounder in altering one or more components of the TIMI score . In our study 29 were smokers and 23 were non smokers out of a total of 52 patients. The mean TIMI score in Smokers were found to be 4.07 and mean TIMI score in non smokers were found to be 4.48 and the results are tested with Mann Whitney U test and are found to be Statistically NOT significant at p=0.774.



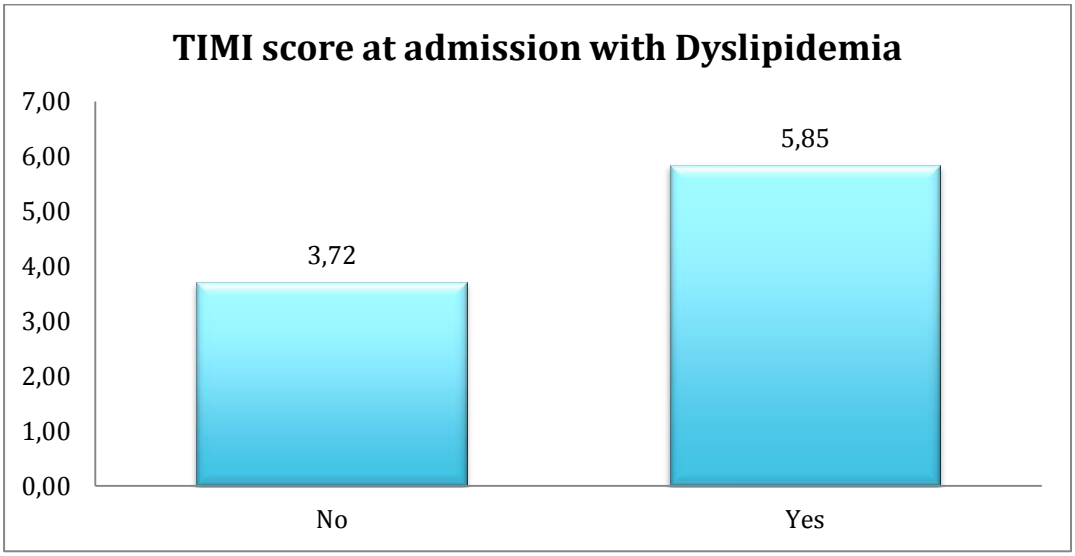
3. Troponin T and its association with Smoking :

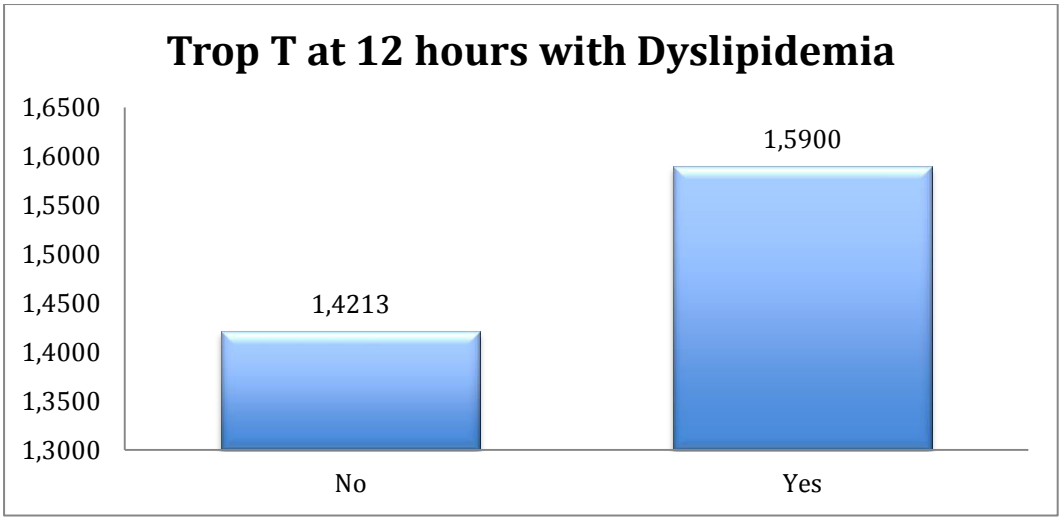
This was done actually to find out smoking as a potential confounder in altering the Troponin T values considerably. It was found that the smokers had a mean Troponin T value of 1.45 ng/mL at 12 hours and non smokers had a mean troponin T value of 1.48 ng/mLat 12 hours and the results were tested using mann Whitney U test and it is found to be statistically NOT significant at p= 0.956.



4. **<u>Association of dyslipidemia with TIMI score at admission and Troponin T at 12 hours :</u>**

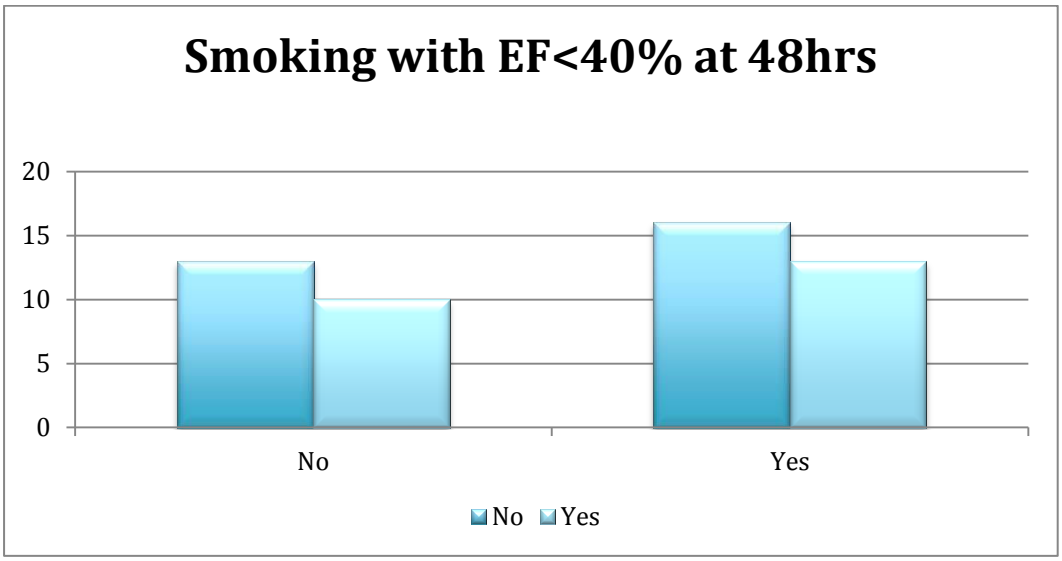
This was also done to find out whether dyslipidemia affects TIMI score and Troponin T . It is found out that patients with dyslipidemia had a mean TIMI score of 5.85 at the time of admission at p=0.048 and a mean Troponin T value of 1.59 ng/mL at 12 hours at p=0.131 and both were statistically not significant as p>0.01 was considered to be statistically insignificant. Patients without dyslipidemia had a mean TIMI score of 3.72 at the time of admission at p=0.048 and a mean Troponin T value of 1.42ng/mL at 48 hours at p=0.131. Both were tested using Mann Whitney U test . The results were depicted below.

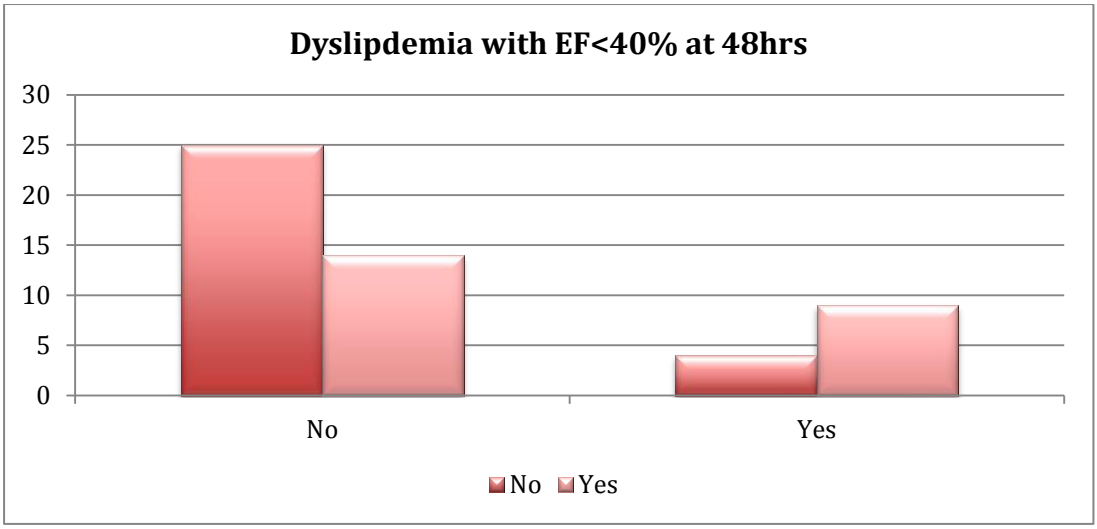




5. Association of EF<40% with Smoking and dyslipidemia :

The association of Smoking and dyslipidemia with EF<40% were tested individually using Chi square tests and the association was found to be statistically not significant at p=0.922 (for smoking and EF>40%) and p=0.036 (for dydlipidemia and F<40%).




Dyslipdemia with EF<40% at 48hrs
30
25
20
15
10
5
0
No
Yes
No
Yes

DISCUSSION

Acute Coronary Syndrome is a spectrum of disorders which include ST Elevation Myocardial Infarction (STEMI), Non ST Elevation Myocardial Infarction(NSTEMI) and Unstable Angina (UA). STEMI is usually associated with significant short term as well as long term mortality . STEMI is diagnosed by characteristic ECG changes along with clinical features and elevation of cardiac biomarkers.[1]

There are a lot of prognostic markers available for ST Elevstion Myocardial Infarction . But the Thrombolysis In Myocardial Infarction (TIMI) score is clinical and was widely known to predict prognosis in the form of mortality at 30 days. TIMI score[8] uses simple clinical data , and its calculation is simple and entirely bedside and it it can be assessed through simple clinical examination and history [4]

The Troponin T which is used as a biomarker for diagnosing STEMI , can also be used as a prognostic marker based on the value of Troponin T at various points post MI[13]. Usually the levels of Troponin T at 12, 24 ,48 or 72 hours can be taken to predict the prognosis. Troponin T is releases from the cardiac myocytes upon death/lysis usually during infarction) . So the absolute Troponin T levels can predict the size of the infarct in MI and especially STEMI. The infarct size is a well known

predictor of severity and it is directly proportional to the mortality and morbidity and hence widely used as a prognostic marker .

The Ejection fraction[32] has long been recognized as a prognostic indicator. Roughly an Ejection fraction of <40% has been shown by a number of studies as an indicator of poor prognosis and increased long term and short term mortality and morbidity. Even though its usefulness has been questioned in recent times , it is still regarded as an important prognostic variable.

There are numerous other prognostic variables but in our study we use only TIMI score[30] , Troponin T and Ejection fraction <40%. Since TIMI score is entirely clinical we evaluated the associations between these three variables so that we can predict the other two using TIMI score itself which is calculated at the time of admission. There were no previous studies made in regard to this association.

Total of 52 patients were enrolled in our study over a period of 6 months and 17 were females and 35 were males . We obtained a standard questionairre mentioning all the data and variables used in the TIMI score as well as ejection fraction <40% obtained at 48 hours and Quantitative Troponin T obtained at 12 hours. We considered smoking and dyslipidemia as potential confounders and hence added to the list . **The collected data was analysed with SPSS 16.0 version**

In our study the frequency of diabetes alone was higher together with patients having neither diabetes , hypertension or angina , 15 patients (28.8%) had a systolic BP of < 100 mm Hg, 20 patients (38.5%) of patients had a Heart rate of > 100 beats per minute, 12 patients (23.1%) had weight < 67 kg, 25 patients (48.1%) had a killip score of 2,3 or 4, 29 patients out of 52 (55.8%) were smokers , 13 patients (25%) in our study were dyslipidemic , 22 patients were found out to be Inferior Wall MI or combinations of IWMI and 15 were found to have Anterior wall MI and only 1 patient had an isolated Posterior Wall MI, Only 3 patients in our study had a new onset LBBB (Left Bundle Branch Block) in ECG. In our study all the enrolled patients were treated with thrombolysis before 4 hours .

23 patients (44.2%) in our study had an EF of < 40%. The maximum age is 76 and the minimum is 36. In the male patients maximum age recorded was 71 and the minimum age was 36 where as in the females it is 76 and 42 respectively.

The correlation and association was done between all possible variables . Correlation was done between TIMI score at the time of admission and Single Quantitative Troponin T at 12 hours. In our study the correlation between TIMI score for acute STEMI at the time of admission and with Single Quantitative Troponin T at 12 hours was found to be highly significant at p=0.005. Association between TIMI

score and EF<40% at 48 hours was tested and the association was found to be statistically significant at p=0.005. Those with EF> 40% at 48 hours had a mean TIMI score of 2.17 during admission and those with EF <40% at 48 hours had a mean TIMI score of 6.87 during admission . 23 patients had a EF of < 40% at 48 hours and 29 patients had a EF>40% at 48 hours. The association between Troponin T at 12 hours and EF<40% at 48 hours was then tested and the association was found o be statistically significant at p=0.005. "Those with Ejection fraction<40% at 48 hours had a mean Troponin T of 1.74 ng/mL at 12 hours and those with EF > 40% at 48 hours had a mean Troponin T level of 1.74 at 12 hours."

55

The TIMI score at the time of admission was then correlated with the type of MI . The mean TIMI score is 5.45 for AWMI's and 2.74 for IWMI's at the time of admission. The results were found to be statistically significant at p=0.001.

The association between potential confounders – smoking and dyslipidemia was then done. The mean TIMI score in Smokers were found to be 4.07 and mean TIMI score in non smokers were found to be 4.48 and the results were found to be Statistically NOT significant at p=0.774. The association between Smoking and Troponin T was then done. It was found that the smokers had a mean Troponin T value of 1.45

ng/mL at 12 hours and non smokers had a mean troponin T value of 1.48 ng/mLat 12 hours and the results were tested using mann Whitney U test and it is found to be statistically NOT significant at p= 0.956.

The association between TIMI score and dyslipidemia was then done. It is found out that patients with dyslipidemia had a mean TIMI score of 5.85 at the time of admission at p=0.048 and a mean Troponin T value of 1.59 ng/mL at 12 hours at p=0.131 and both were statistically not significant as p>0.01 was considered to be statistically insignificant. Patients without dyslipidemia had a mean TIMI score of 3.72 at the time of admission at p=0.048 and a mean Troponin T value of 1.42ng/mL at 48 hours at p=0.131

Lastly , the association of EF <40% with smoking and dyslipidemia was sought for. The association of Smoking and dyslipidemia with EF<40% were tested individually using Chi square tests and the association was found to be statistically not significant at p=0.922 (for smoking and EF>40%) and at p=0.036 (for dydlipidemia and F<40%).

Since our study is totally new (TIMI score have not been compared with Troponin T or Ejection fraction) and have not been performed in the past , we did not find any control studies to compare our study with their experiences. We consider this as a potential disadvantage of our study and so this study might need further studies to confirm

positive and negative associations found in our study. We believe that will happen in the near future.

CONCLUSION

1. TIMI score calculated at the time of admission has significant correlation with Single Quantitative Troponin T at 12 hours post admission. (p=0.005)
2. TIMI score calculated at the time of admission also has significant association with Ejection Fraction < 40% at 48 hours post admission . (p=0.005)
3. Single Quantitative Troponin T obtained 12 hours post admission has significant association with Ejection Fraction < 40% at 48 hours post admission (p=0.005)
4. There is no statistically significant association between smoking and TIMI score at admission , Troponin T at 12 hours or Ejection Fraction < 40% at 48 hours.
5. There is no statistically significant association between dyslipidemia and TIMI score , Troponin T at 12 hours or Ejection Fraction<40% at 48 hours.

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ANNEXURE 1 – PROFORMA

PROFORMA

PERSONAL DETAILS :

NAME :

AGE :

SEX :

IP NO :

UNIT :

DATE OF ADMISSION :

DATE OF DISCHARGE :

ADDRESS :

CONTACT :

EDUCATION :

PROFESSION :

INCOME :

COMPLAINTS :

PAST HISTORY :

HISTORY OF	YES (IF YES DURATION)	NO
DIABETES MELLITUS		
SYSTEMIC HYPERTENSION		
DYSLIPIDEMIA		
CORONARY ARTERY DISEASE / HEART FAILURE		
CONGENITAL HEART DISEASE		
PRIOR ACUTE CORONARY SYNDROME		
CHRONIC KIDNEY DISEASE		
PRIOR SURGERY/PROCEDURES (INCLUDING PCI/CABG)		
HYPOTHYROIDISM		
MALIGNANCY		
CEREBROVASCULAR ACCIDENT/THROBOEMBOLIC EVENTS		
OTHERS (IF ANY)		

PERSONAL HISTORY :

PERSONA	YES(IF YES- DURATION)	NO
SMOKING/TOBACCO		
ALCOHOL		
DRUG DEPENDENCE		

FAMILY HISTORY :

GENERAL EXAMINATION AND VITAL SIGNS :

SL NO	EXAMINATION	FINDINGS
1	CONSCIOUSNESS	
2	ORIENTATION	
3	TEMPERATURE	
4	HEART RATE	
5	BLOOD PRESSURE	
6	SATURATION	
7	RESPIRATORY RATE	
8	PALLOR	
9	CYANOSIS	
10	CLUBBING	
11	ICTERUS	
12	PEDAL EDEMA	
13	LYMPHADENOPATHY	
14	JVP	
15. OTHERS (IF ANY)		

SYSTEM EXAMINATION :

SYSTEM	FINDINGS
CARDIOVASCULAR SYSTEM	
RESPIRATORY SYSTEM	
ABDOMEN	
CENTRAL NERVOUS SYSTEM	
OTHERS :	

ELECTROCARDIOGRAM DURING ADMISSION :

2D ECHOCARDIOGRAPHY DURING ADMISSION :

TIMI (THROBOLYSIS IN MYOCARDIAL INFARCTION) SCORING FOR STEMI :

SL NO	RISK FACTOR	POINTS AWARDED	TICK IF YES	POINTS
1	AGE 65-74 YRS	2		
2	AGE > 74 YRS	3		
3	DIABETES MELLITUS / HYPERTENSION/ ANGINA (ANY ONE)	1		
4	SYSTOLIC BLOOD PRESSURE < 100 MM HG	3		
5	HEART RATE > 100	2		
6	KILLIP CLASS II-IV	2		
7	WEIGHT< 67 KG	1		
8	ANTERIOR ST ELEVATION OR LEFT BUNDLE BRANCH BLOCK	1		
9	TIME TO TREAT > 4 HOURS	1		
TOTAL SCORE (OUT OF 14)				

TROPONIN T LEVEL AT 72 HRS :

FASTING LIPID PROFILE :

Sl no	LIPID	VALUE
1.	Total cholesterol	
2.	HDL	
3.	LDL	
4.	Triglycerides	
5.	VLDL	
6.	TC/HDL ratio	

2D ECHOCARDIOGRAM AT 48 HOURS :

ANNEXURE -2

MASTER CHART

Sl no	Name	Age	Sex	Diabetes(D), Hypertension (H), Angina(A)	SBP<100 Mm hg	HR> 100 bpm	Killip Class 2,3,4	Weight <67 kg	STEMI type	New LBBB	Time to treat > 4 hours	Smoking	Dyslipidemia	TIMI score at admission	Trop T at 12 hours	EF<40%at 48 hrs
1.	Srinivasan	43	M	No	Yes	Yes	Yes	No	AWMI	No	No	No	Yes	8	1.76	Yes
2.	Venugopal	56	M	D/H	No	No	No	No	IWMI	No	No	Yes	Yes	2	1.21	No
3.	Sankaran	61	M	D/A	No	No	No	No	IWMI+ RVMI	No	No	Yes	No	2	1.31	No
4.	AdhiRaj	52	M	No	No	Yes	Yes	No	ASMI	Yes	No	Yes	Yes	6	1.72	Yes
5.	Mangalam	59	F	D/A	Yes	Yes	Yes	Yes	ALMI	No	No	No	No	9	2.21	Yes
6.	Siva Kumar	36	M	No	Yes	Yes	Yes	Yes	AWMI	No	No	Yes	Yes	9	1.97	Yes
7.	Meenakshi	52	F	D/H	No	No	No	No	IWMI	No	No	No	No	2	1.31	No
8.	Sundaram	40	M	D	No	No	No	No	IWMI+PWMI+RVMI	No	No	Yes	No	1	0.98	No
9.	Adikesavan	69	M	D/H/A	Yes	No	No	Yes	IWMI+PWMI	No	No	Yes	No	5	1.79	No
10.	Mary	62	F	D	Yes	Yes	Yes	Yes	AWMI	No	No	No	No	10	2.11	Yes
11.	Alamelu	76	F	D/H/A	No	Yes	Yes	Yes	ASMI	No	No	No	No	10	2.24	Yes
12.	Sekar	51	M	H/A	No	No	Yes	No	ASMI	No	No	Yes	No	4	1.59	Yes
13.	Rizwan	41	M	No	No	No	No	No	IWMI+ RVMI	No	No	No	No	0	0.96	No
14.	Gowri	49	F	H	No	No	Yes	No	AWMI	No	No	No	No	4	1.52	No
15.	Raja	54	M	D/H	Yes	No	No	Yes	IWMI+PWMI	No	No	Yes	Yes	5	1.44	No
16.	Jeevaraj	44	M	H	No	No	No	No	ASMI	No	No	Yes	No	2	1.48	No
17.	Subraja	68	F	H	Yes	Yes	Yes	No	ALMI	No	No	No	No	7	1.77	Yes
Sl no	Name	Age	Sex	Diabetes(D), Hypertension (H), Angina(A)	SBP<100 Mm hg	HR> 100 bpm	Killip Class 2,3,4	Weight <67 kg	STEMI type	New LBBB	Time to treat > 4 hours	Smoking	Dyslipidemia	TIMI score at admission	Trop T at 12 hours	EF<40%

18.	Anthony	53	M	H	No	Yes	Yes	No	AWMI	No	No	Yes	No	6	1.67	Yes
19.	Chinna Ponnu	68	F	D/H	Yes	No	Yes	Yes	IWMI+ RVMI	No	No	No	Yes	9	1.61	Yes
20.	Azharrudin	58	M	No	No	No	Yes	No	ASMI	No	No	Yes	No	3	1.32	No
21.	Singaram	64	M	D/H	No	No	No	No	AWMI	No	No	Yes	No	1	1.49	No
22.	Pichai	59	M	H	Yes	Yes	No	NO	IWMI	No	No	No	No	6	1.66	Yes
23.	Akilandam	55	F	D/H	No	No	No	No	IWMI+ RVMI	No	No	No	No	0	1.16	No
24.	Saraswathy	59	F	No	No	No	No	No	IWMI+P WMI	No	No	No	Yes	0	0.88	Yes
25.	Rangan	68	M	H	Yes	No	Yes	Yes	IWMI+ RVMI	No	No	Yes	No	8	1.71	Yes
26.	Azhagar	71	M	D/H	No	Yes	Yes	Yes	AWMI	No	No	Yes	Yes	9	1.86	Yes
27.	Tamilselvi	66	F	D/A	No	No	No	No	AWMI	No	No	No	No	4	1.34	No
28.	Saminathan	71	M	D	No	Yes	Yes	No	ASMI	No	No	Yes	No	6	1.73	Yes
29.	Aruna	55	F	H/A	No	No	No	No	AWMI	No	No	No	No	0	1.02	No
30.	Adhithyan	61	M	D	No	Yes	No	No	ALMI	No	No	Yes	No	3	1.34	No
31.	Ilayaraja	58	M	D/H	Yes	No	No	No	IWMI+P WMI	No	No	Yes	No	4	1.33	Yes
32.	Sadhiq Basha	55	M	No	No	No	No	Yes	IWMI	No	No	Yes	No	1	0.99	No
33.	Ramesh	46	M	D	Yes	Yes	Yes	No	AWMI	Yes	No	Yes	Yes	9	1.98	Yes
Sl no	Name	Age	S e x	Diabetes(D), Hypertensio n (H), Angina(A)	SBP<1 00 Mm hg	HR> 100 bpm	Killip Class 2,3,4	Weight <67 kg	STEMI type	New LBBB	Time to treat > 4 hours	Smoking	Dyslipidemia	TIMI score at admission	Trop T at 12 hours	EF<4 0%
34.	Saravanan	38	M	No	Yes	Yes	No	No	ALMI	No	No	Yes	No	6	1.77	Yes
35.	Arivukozhu ndhu	66	F	D	No	No	No	No	ASMI	Yes	No	No	No	4	1.29	No
36.	Adalarasan	48	M	H	No	No	Yes	No	AWMI	No	No	Yes	No	4	1.33	Yes
37.	Yasir Khan	56	M	D	Yes	No	Yes	No	IWMI+P	No	No	Yes	No	6	1.48	Yes

									WMI							
38.	Arikrishnan	54	M	D	No	No	No	No	IWMI+ RVMI+P WMI	No	No	No	No	1	1.17	No
39.	Munusamy	41	M	No	No	No	No	No	ASMI	No	No	Yes	No	1	0.89	No
40.	Arokia Mary	56	F	H	Yes	Yes	Yes	No	AWMI	No	No	No	Yes	9	2.11	Yes
41.	Anthony	42	M	No	No	Yes	Yes	No	IWMI+ RVMI	No	No	Yes	No	4	1.29	No
42.	Anandhan	46	M	H	No	No	No	No	IWMI+P WMI	No	No	Yes	No	0	1.01	No
43.	Rajesh	39	M	No	No	Yes	No	No	ALMI	No	No	Yes	No	3	1.49	No
44.	Kalaiselvi	42	F	D	No	No	Yes	No	IWMI	No	No	No	No	2	1.11	No
45.	Nandha Kumar	67	M	D/H	No	Yes	Yes	Yes	AWMI	No	No	No	No	7	1.77	Yes
46.	Maheswari	61	F	D/H	No	No	No	Yes	PWMI	No	No	No	No	1	1.03	No
47.	Siva Sridharan	47	M	H	No	Yes	No	No	AWMI	No	No	No	No	4	1.41	No
Sl no	Name	Age	S e x	Diabetes(D), Hypertension (H), Angina(A)	SBP<100 Mm hg	HR> 100 bpm	Killip Class 2,3,4	Weight <67 kg	STEMI type	New LBBB	Time to treat > 4 hours	Smoking	Dyslipidemia	TIMI score at admission	Trop T at 12 hours	EF<40%
48.	Manickam	50	M	D	No	Yes	Yes	No	ALMI	No	No	Yes	Yes	6	1.71	Yes
49.	Sasikala	61	F	H/A	No	No	Yes	No	AWMI	No	No	No	Yes	4	1.42	No
50.	Mala	69	F	D	No	No	No	No	IWMI+ RVMI	No	No	No	No	2	1.12	No
51.	Murugan	53	M	No	No	No	Yes	No	IWMI+P WMI	No	No	Yes	No	2	1.24	No
52.	Raman	51	M	D	No	No	No	No	IWMI	No	No	Yes	Yes	0	1.00	No

KEYS TO MASTER CHART

M- Male

F- Female

D- Diabetes

H- Hypertension

A- Angina

SBP- Systolic Blood Pressure

HR- Heart Rate

STEMI- ST Elevation Myocardial Infarction

TIMI – Thrombolysis In Myocardial Infarction

LBBB- Left Bundle Branch Block

Trop T- Troponin T

EF- Ejection fraction

AWMI- Anterior Wall Myocardial Infarction

ASMI- AnteroSeptal Myocardial Infarction

ALMI- AnteroLateral Myocardial Infarction

IWMI- Inferior Wall Myocardial Infarction

RVMI- Right Ventrivular Myocardial Infarction

PWMI- Posterior Wall Myocardial Infarction

ANNEXURE – 3

ETHICAL COMMITTEE CLEARANCE CERTIFICATE

INSTITUTIONAL ETHICAL COMMITTEE
GOVT.KILPAUK MEDICAL COLLEGE,
CHENNAI-10
Protocol ID No.14/01/2015 Dt.20.01.2015
CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval TIMI score in patients presenting with acute stelevation myocardial infarction and their correlation with quantitative troponin T and Ejection fraction. -For Project Work-submitted by Dr. Dillip Kumar, PG in General Medicine, KMC, 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.

GOVT. KILPAUK MEDICAL COLLEGE * Kilpauk, Chennai-10. * JAN 2015

20/1/15

CHAIRMAN,
Ethical Committee
Govt.Kilpauk Medical College,Chennai

19/1/2015

6 Comp.> ME 1> Ethical Committee