A DISSERTATION ON

"MODIFIED SELVESTER QRS SCORE IN PREDICTING SUCCESSFUL ST SEGMENT RESOLUTION IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION RECEIVING THROMBOLYTIC THERAPY WITH STREPTOKINASE"

Dissertation Submitted To

THE TAMILNADU Dr. M.G.R MEDICAL UNIVERSITY,

In partial fulfillment of the rules and regulations, for the award of the

M.D. DEGREE IN GENERAL MEDICINEBRANCH –I

REGISTRATION NUMBER: 200120103003

GOVT.CHENGALPATTU MEDICAL COLLEGE & HOSPITAL,

CHENGALPATTU-603001

MAY 2023

CERTIFICATE

This is to certify that the dissertation titled **"MODIFIED SELVESTERQRS SCORE IN PREDICTING SUCCESSFUL ST SEGMENT RESOLUTION IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION RECEIVING THROMBOLYTIC THERAPY WITH STREPTOKINASE"** is the bonafide original work of **Dr. ASWIN.C** in partial fulfillment of the requirements for **M.D. Branch-1 (General Medicine)** Examination of the Tamil Nadu Dr.M.G.R. Medical University to be held in May2023. The period of study was from April 2021 to March 2022.

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DECLARATION

I, **Dr. ASWIN.C** solemnly declare that dissertation titled **"MODIFIED SELVESTER QRS SCORE IN PREDICTING SUCCESSFUL ST SEGMENT RESOLUTION IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION RECEIVING THROMBOLYTIC THERAPY WITH STREPTOKINASE"** a bonafide record of work done by me in the Department of Internal Medicine, Government Chengalpattu Medical College, and Hospital during April 2021 to March 2022 under the guidance of **Prof. Dr. R. NARMADHA LAKSHMI, M.D, Dch,** Professor ofGeneral Medicine, Government Chengalpattu Medical College and Hospital, Chengalpattu. This dissertation is submitted to Tamil Nadu Dr. M.G.R. MedicalUniversity, in partial fulfillment of the University regulations for the award of M.D. Degree (Branch 1) General Medicine- May 2023.

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Chengalpattu Medical College
Registration No: ECR/774/Inst/TN/2015
IEC Approval Letter – Amended 11.01.2021

No. IEC-CMC/Approval/19/2021 **Dated: 30.03.2021**

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Title of Work: Modified selvester QRS score in predicting successful resolution of ST segment elevation in patients with acute myocardial infarction receiving thrombolytic therapy with streptokinase

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC e-meeting held as per ICH-GCP guidelines, New Drug and clinical trials Rule March 2019 requirements & ICMR – EC Guidance during COVID – 19 Pandemic on 25.03.2021 at the Medical Education Unit, Chengalpattu Government Medical College, Chengalpattu at 11.00 AM for the IEC-Chengalpattu Medical College members.

The Members of the committee, the Secretary and the Chairperson are pleased to inform you that your proposed project mentioned above is **Approved** in its presented form.

1. This approval is valid for three years or the duration of the project whichever is less.
2. Should inform the IEC in case of any changes in study procedure, methodology, sample size investigation, investigator or guide or any other changes.
3. Should not deviate from the area of work for which you had applied for ethical clearance.
4. Should inform the IEC immediately, in case of any adverse events or serious adverse reactions, if encountered during from study seven days.
5. Should abide to the rules and regulations of the institution(s).
6. Should complete the work within the specific period and if any extension is required, you should apply for the permission again for extension period.
7. A yearly progress report of the project has to be submitted to the IEC for review.
8. Should submit a copy of the trial work to the ethical committee on completion of the study.

(Dr.A.Anitha)
Member Secretary Ethics Committee

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APPROVAL FROM COLLABORATING DEPARTMENT

From

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Postgraduate Student,

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To

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Through proper channel,

Respected sir/ Madam,

SUB: Dissertation on " **MODIFIED SELVESTER QRS SCORE IN PREDICTING SUCCESSFUL RESOLUTION OF ST SEGMENT ELEVATION IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION RECIEVING THROMBOLYTIC THERAPY WITH STREPTOKINASE** " - request for permission to do the study. Applied for approval of the project by Institutional ethics committee, CMCH, Chengalpattu- regarding.

I joined M.D GENERAL MEDICINE in the academic year 2020-2021 I am planning to do a study **"MODIFIED SELVESTER QRS SCORE IN PREDICTING SUCCESSFUL RESOLUTION OF ST SEGMENT ELEVATION IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION RECIEVING THROMBOLYTIC THERAPY WITH STREPTOKINASE "** among patients coming for treatment at CMCH, Chengalpattu, request you to grant me the permission to do the study.

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Thanking you

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Date: 22/3/2021

Yours faithfully,

ABSTRACT

BACKGROUND: Cardiovascular illnesses are a major cause of death for people and are a growing burden on society. We also observed a radical shift from conservative strategies of bed rest to re perfusion strategies of thrombolysis and PCI directed therapies. The main aim of this reperfusion strategies are to restore the coronary blood flow and achieve myocardial perfusion. This can be detected by the early and complete resolution of ST segment elevation after treatment. So, this makes it extremely important to predict the ST segment resolution with thrombolysis before initiating treatment. This modified Selvester score is one such score which helps us in this aspect.

AIM: To determine whether modified Selvester QRS scoring system would predict the resolution of ST segment elevation in patients with acute myocardial infarction receiving thrombolytic therapy with streptokinase.

MATERIALS AND METHODS: This study was conducted in Government Chengalpattu medical college with study population of 61. Information gathered about the patient's type of MI, Fasting lipid profile, Diabetes, Hypertension. Baseline ECG was taken and QRS score was calculated with the initial ECG and 90 minutes ECG after thrombolysis was taken. STE (ST Elevation) in initial ECG taken as STE1 and 90 minutes after thrombolysis taken as STE2 and the difference between the two taken as ST segment resolution (STR). The patients are divided into resolution (STR more than or equal to 50%) and non resolution group (STR less than 50%) group. Based on the value QRS <4 predicting the resolution group and other characteristics are studied.

RESULTS: The mean age of the whole group is 56.07 ±11.845 with 43 being male (70%) and 18 (30%) being female and older individuals are seen more in the non-resolution group than the resolution group and QRS score is high in the older age which shows as the age advances the chance of non-resolution. Sex, Smoking,

Alcohol, Hypertension shows no statistical relation with respect to ST resolution and QRS score respectively. However, Diabetics (50% in the study) showed a significant relation to QRS score and had a high QRS score but they did not show any correlation with the ST resolution. Cardiac Failure has a significant correlation with respect to QRS score and ST resolution. The patients with cardiac failure have a high QRS score (69% of patients) and there was ST non resolution in 74% of patients. The patients with the type of MI or Dyslipidemia did not have any significance in this study. There was also a negative correlation between Ejection fraction and QRS score.

CONCLUSION: Our study concluded that high QRS score noted with increasing age, diabetics, cardiac failure and have a more risk of non resolution. Also a significant negative corelation observed between EF and QRS score is noted. Thus, QRS score would reliably predict the ST segment resolution in patients with STEMI receiving thrombolytic therapy. QRS score >4 says the patient has higher chances of ST non resolution.

Key words: Selvester score, QRS score, Myocardial infarction

CONTENTS

INTRODUCTION

INTRODUCTION

Coronary Artery disease is a pathological process affecting the coronary arteries. Coronary artery disease includes angina pectoris, Myocardial Infarction and Silent Myocardial infarction. Our understanding of Myocardial Infarction had evolved over years in its causes, diagnosis, and treatment.

Cardiovascular illnesses are a major cause of death for people and are a growing burden on society.. The major risk factors include sedentary life style, Diabetes Mellitus, Hypertension, cigarette smoking, alcoholism. We also observed a radical shift from conservative strategies of bed rest to re perfusion strategies of thrombolysis and PCI directed therapies.

The main aim of this reperfusion strategies are to restore the coronary blood flow and achieve myocardial perfusion. This can be detected by the early and complete resolution of ST segment elevation after treatment. So, this makes it extremely important to predict the ST segment resolution with thrombolysis before initiating treatment. This modified Selvester score is one such score which helps us in this aspect. Originally Selvester score[31] was designed based on 54 criteria making it 32 points and subsequently multiple versions of the score were published and we chose a simplified form[26] of the score with 29 points.

Myocardial infarction can be diagnosed based on the clinical history, biochemical markers, electrocardiography, and echocardiography. But echocardiography remains as a very important tool because they are non-invasive, easy to use, low cost.

Although with improvements in the treatment, the mortality rate in patients who develop cardiogenic shock remains high. So, an early detection ofthe Myocardial

infarction helps us to improve the outcomes. Elderly patients also contribute to high-risk mortality group of Myocardial infarction and they are prone more for recurrent infarctions and dysfunctions and are affected more than young people.

AIM AND OBJECTIVES

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To determine whether modified Selvester QRS scoring system would predict the resolution of ST segment elevation in patients with acute myocardial infarction receiving thrombolytic therapy with streptokinase.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

India has one of the highest cardiovascular burdens around the world.

The prevalence rates among the urban population are higher compared to the rural population and the prevalence rate estimated over the past decades were 1.6 to 7.4 % in rural population and 1% to 13.2 % in urban population[1].

The INTERHEART study showed that the Cardiovascular risk factors such as abdominal obesity, Hypertension, diabetes are increasing among Indians even at younger age than other groups. The prevalence rates areincreasing over the past 25 decades particularly among the urban population.

Cardiovascular diseases are among the rise than the infectious diseases. In India the peak period is 51 to 60 years which is a decade earlier than the developed countries.

ANATOMY OF CORONARY VESSELS

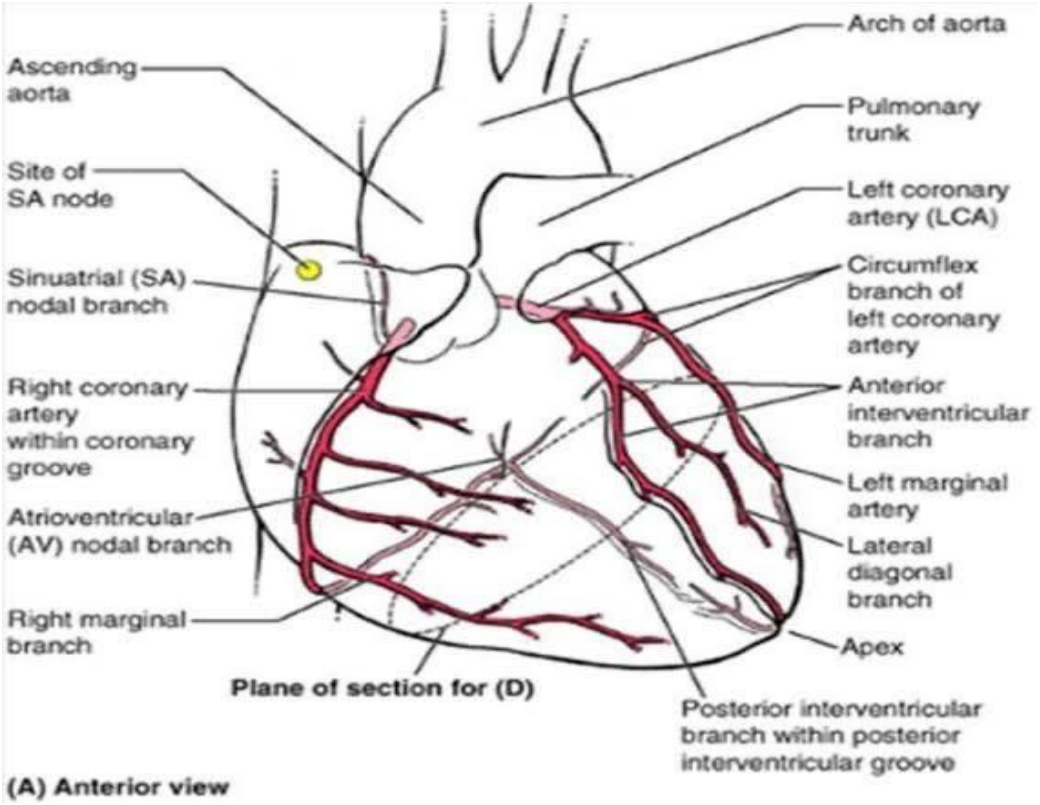
CORONARY ARTERIES

The coronary arteries arise from the coronary sinus at the base of heart and divides into three main branches

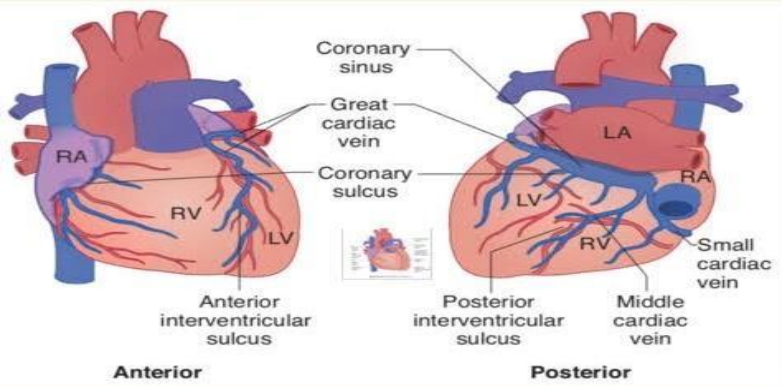
Right coronary artery and divides into atrial branch, marginal branch, SA nodal, AV nodal, septal, and posterior descending branches.

Left main coronary artery into

1. Left Anterior descending that gives diagonal and septal branches.
2. Left circumflex artery that gives marginal and sometimes the posterior descending branches.



VENOUS DRAINAGE OF TH E HEART



CORONARY BLOOD FLOW

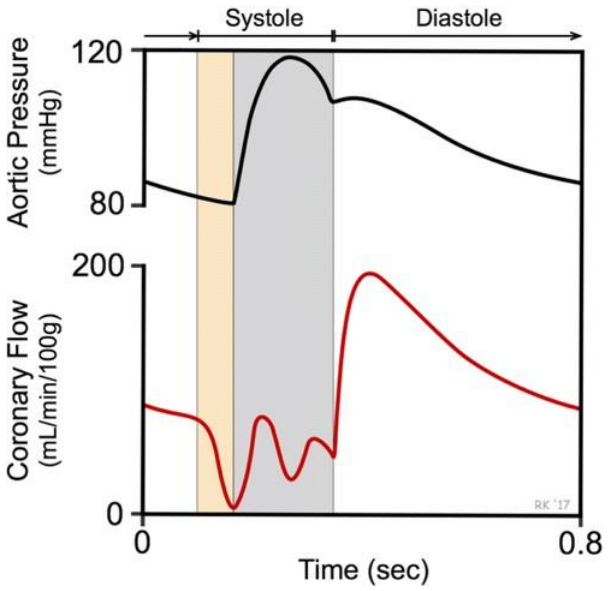
Coronary arteries are broadly classified into intramuscular and epicardial vessels. The epicardial vessels are superficial and functions to conduct the blood and the intramuscular arteries are smaller and provide higher resistance to control the blood flow.

Coronary blood flow is 5% of cardiac output which is around 225 to 250 ml/min. At rest 60 to 70 % of oxygen is delivered to the heart as it has more mitochondria occupying the myocytes. The coronary blood flow is tightly coupled to the oxygen demand due to its high basal oxygen consumption.

Whenever the cardiac activity and oxygen consumption is increased it is compensated by the increase in coronary blood flow.

The extra vascular compression during the systole affects the coronaryblood flow, so most of the coronary blood flow occurs during the diastole.

Because of the extra vascular compression the endocardium is more susceptible to ischemia particularly at low perfusion pressures.



Coronary blood flow to the right ventricle is not much affected duringsystole owing to the pressure differences between aorta and right ventricle during systole and diastole. Auto regulation between 60 to 200 mm Hg perfusion pressures helps to maintain the normal coronary blood flow.

FACTORS AFFECTING THE CORONARY BLOOD FLOW

Chemical factors

Causing coronary vessel vasodilation:

- Adenosine
- Nitric oxide
- Lack of oxygen
- Increased local factors such as K^+, lactate, pH, CO_2.

Neural factors

Sympathetic:

Alpha receptors causing coronary vessel vasoconstriction. Beta 1 receptor causing coronary vessel vasodilation.

Parasympathetic: Vagal stimulation causing coronary vasodilation.

CORONARY ARTERY DISEASE SPECTRUM

Coronary artery disease includes Stable Ischemic Heart Disease and Acute coronary syndrome.

Acute coronary syndrome includes

- Unstable Angina (UA) – Patient has negative biomarkerswith non-ST segment elevation
- Non-ST Elevation Myocardial Infarction – Patient haspositive biomarkers with non-ST segment Elevation
- ST Elevation Myocardial Infarction – Patient has positivebiomarkers with ST segment elevation.

<table>
<tr><th></th><th>SIHD</th><th>UA</th><th>NSTEMI</th><th>STEMI</th></tr>
<tr><td>Definition</td><td>Ischemia on activity</td><td>Ischemia without necrosis</td><td>Necrosis

(Non-Transmural)</td><td>nsmural necrosis</td></tr>
<tr><td rowspan="2">Diagnosis</td><td rowspan="2">Symptoms,

ECG, stress testing</td><td>Negative Biomarkers</td><td colspan="2">Positive Biomarkers</td></tr>
<tr><td colspan="2">No ECG ST – segment elevation</td><td>ECG ST segmentelevation</td></tr>
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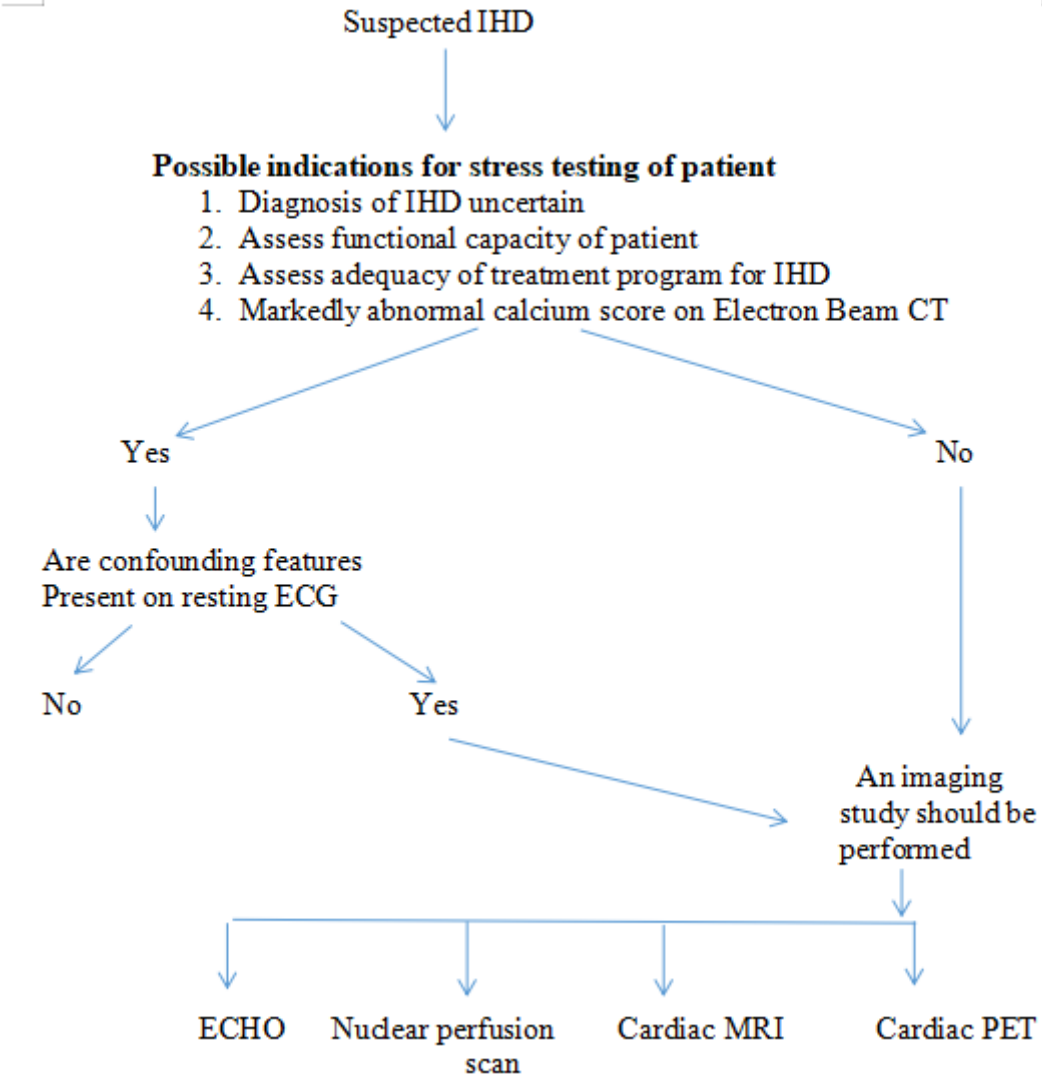
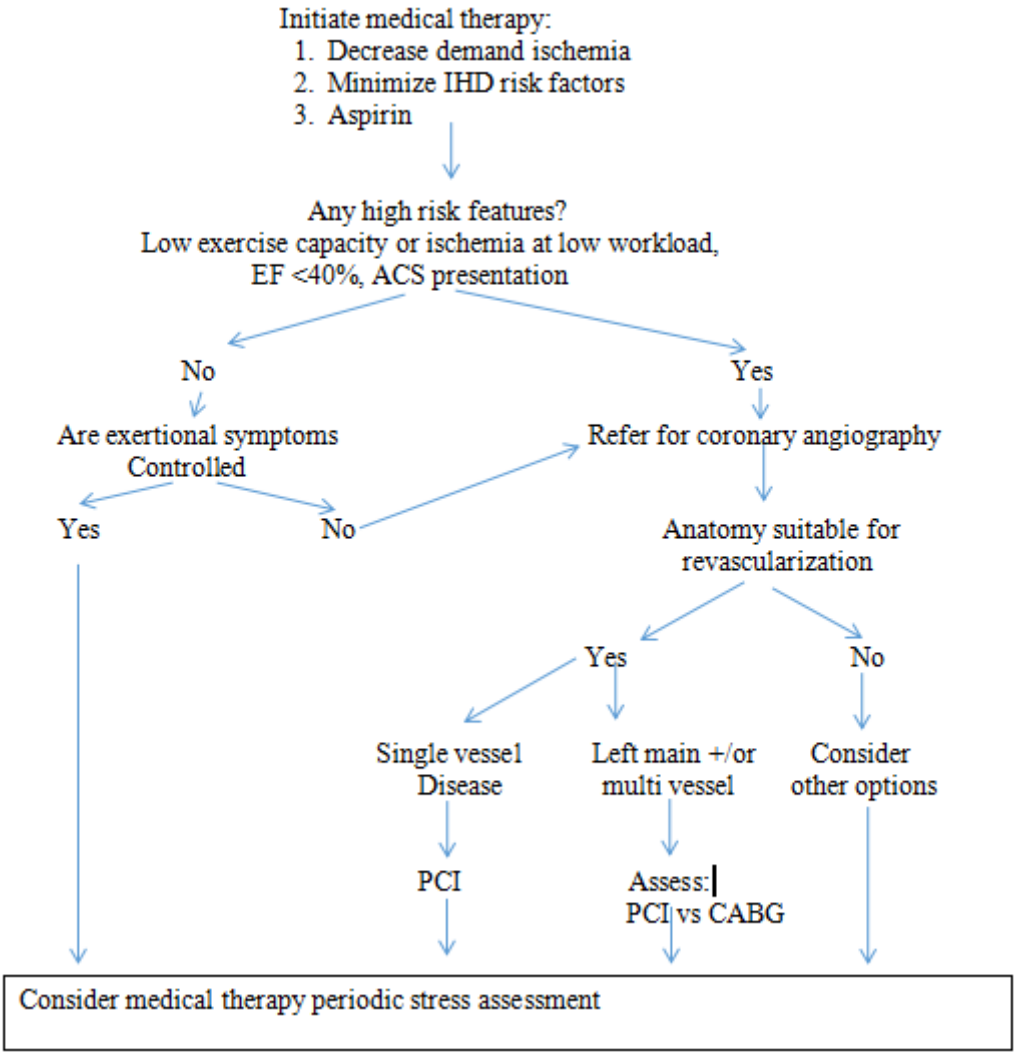
Evaluation of patient with suspected Ischemic Heart disease



Management of a patient with ischemic heart disease



MYOCARDIAL INFARCTION

Definition[2]

The term acute myocardial injury with clinical evidence of acute myocardial ischemia and with detection of rise and/or fall of cardiac troponins with at least one value above 99th percentile URL with at least one of the following:

- Symptoms of myocardial ischemia;
- Development of pathological Q waves;
- New ischemic ECG changes;
- Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality in a pattern consistent withan ischemic etiology
- Identification of a coronary thrombus by angiography orautopsy

ECG changes suggestive of acute MI in the absence of bundle branch block or left ventricular hypertrophy

New elevation of ST segment at J point in two contiguous leads with ≥ 1 mm in all leads except V2 and V3 where the following points apply ≥ 2 mm in men ≥ 40 years; 2.5mm in men < 40 years or 1.5mm in women regardless of age

(or) a new left bundle branch block

Reciprocal ST segment changes in the other leads makes the diagnosis of MI more specific.

ECG changes suggestive of acute MI in the setting of Left Bundle branch block (LBBB)

Sgarbossa scoring system is used in case of LBBB

1. ST-segment elevation ≥ 1 mm in a lead with positive QRS complex (5 points)

2. ST-segment depression ≥ 1 mm in V1, V2, or V3 (3 points)

3. ST-segment elevation ≥ 5 mm in a lead with negative QRS complex (2 points)

Score of > 3 is suggestive of MI.

Criteria 1 is more specific than criteria 3 and the presence of criteria 1itself is more specific for diagnosis.

CLASSIFICATION OF MYOCARDIAL INFARCTION[3]

Type	**Classification**	**Diagnostic criteria**
1	Spontaneous MI	Plaque rupture,ulceration,fissuring,erosion or dissection resulting in coronary thrombus
2	Supply/demand mismatch	Mismatch between myocardial oxygen demand and supply driven by a process other than coronary artery disease
3	Suspected MI related death	Cardiac disease suggestive of an ischemic process without cardiac biomarker evidence of MI
4a	PCI-related MI	Rise in cardiac biomarkers accompanied by symptoms, electrocardiographic,angiographic,or imaging evidence of ischemia after PCI
4b	Stent thrombosis	Confirmed stent thrombosis in context of ischemia and dynamic cardiac biomarker changes
5	CABG related MI	Rise in cardiac biomarkers accompanied by electrocardiographic, angiographic or imaging evidence of ischemia after CABG

RISK FACTORS

Non modifiable risk factors:

1. **Age**

The prevalence increases after the age of 35 in both men and women.

The lifetime risk of developing CAD after the age of 40 in both men and women are 49% and 32% respectively[4].

2. **Sex**

Men are at increased risk than women. Estrogen in women seems toplay a protective role.

3. **Family history**

Family history of CAD are associated with increased risk of PrematureCAD in patients less than 50 years of age.

Modifiable risk factors:

1. **Hypertension**

Hypertension is a major risk factor by mechanical and oxidative stress over the vessel wall. A reduction in systolic blood pressure by 10 mm Hg and Diastolic blood pressure by 5 mm Hg can lead to the absolute risk reduction of CAD related events.

2. **Diabetes mellitus**

The heart disease rate is 2.5 times and 2.4 times higher in men andwomen with diabetes respectively than non-diabetes. A 0.5% reduction inHbA1C conferred a 20 % reduction in the risk [(5)].

3. **Dyslipidemia:**

Elevated cholesterol and triglycerides serve as an important risk factor. The Seven Countries Study showed that serum cholesterol is an important risk factor for the incidence of CAD at levels more than 220 mg/dl. The level of low- density lipoprotein (LDL) is directly associated with CAD. Very low-density lipoprotein (VLDL) is shown to be associated with premature atherosclerosis. High Density lipoprotein (HDL) is protective against the occurrence of CAD. HDL levels should be more than 30 mg/dl to exert a protective effect and LDL levels should ideally be less than 200 mg/dl.

Dyslipidemia can promote atherosclerosis and thus lowering LDL with statinsserves as both primary and secondary prevention.

4. **Diet**

The increased intake of saturated fat and trans-fat increases the risk ofCAD. Even the increase in the intake of salt, refined sugars, high content of sucrose and fructose increasing the risk is noteworthy here. Thus, the intake ofmono and poly unsaturated fatty acids are recommended.

5. **Obesity and lifestyle factors.**

Obesity is an important risk factor for the development of CAD, Hypertension, diabetes. Thus, weight reduction confers clinical benefits for the individuals. Sedentary life style itself leads to obesity. Exercising around 150 minutes per week is recommended.

6. **Smoking:**

Smokers are 2 - 4 times at increased risk and even passive smokers has an

increased risk. Behavioral and pharmacological approaches are practiced to prevent smoking.

7. **Other novel risk factors:**

- Increased homocysteine levels increases the risk.
- Chronic kidney disease [6] is an independent risk factor as it increases the endothelial dysfunction.
- Non-Alcoholic Fatty Liver Disease (NAFLD) increases the risk of CAD.
- Type B malnutrition with increased calorie and low micro nutrient intake.

CAUSES OF MYOCARDIAL INFARCTION [7]

- Atherosclerosis - an important cause.
- Coronary artery embolization from infective endocarditis, mural thrombi,prosthetic valves, calcium deposits, neoplasm.
- Viral infections with Coxsackie B as an uncommon cause.
- Coronary occlusion due to syphilitic aortitis, Takayasu arteritis, Systemic lupuserythematosus, Kawasaki disease.
- Cocaine abuse.
- Spontaneous coronary dissection.

ATHEROSCLEROSIS

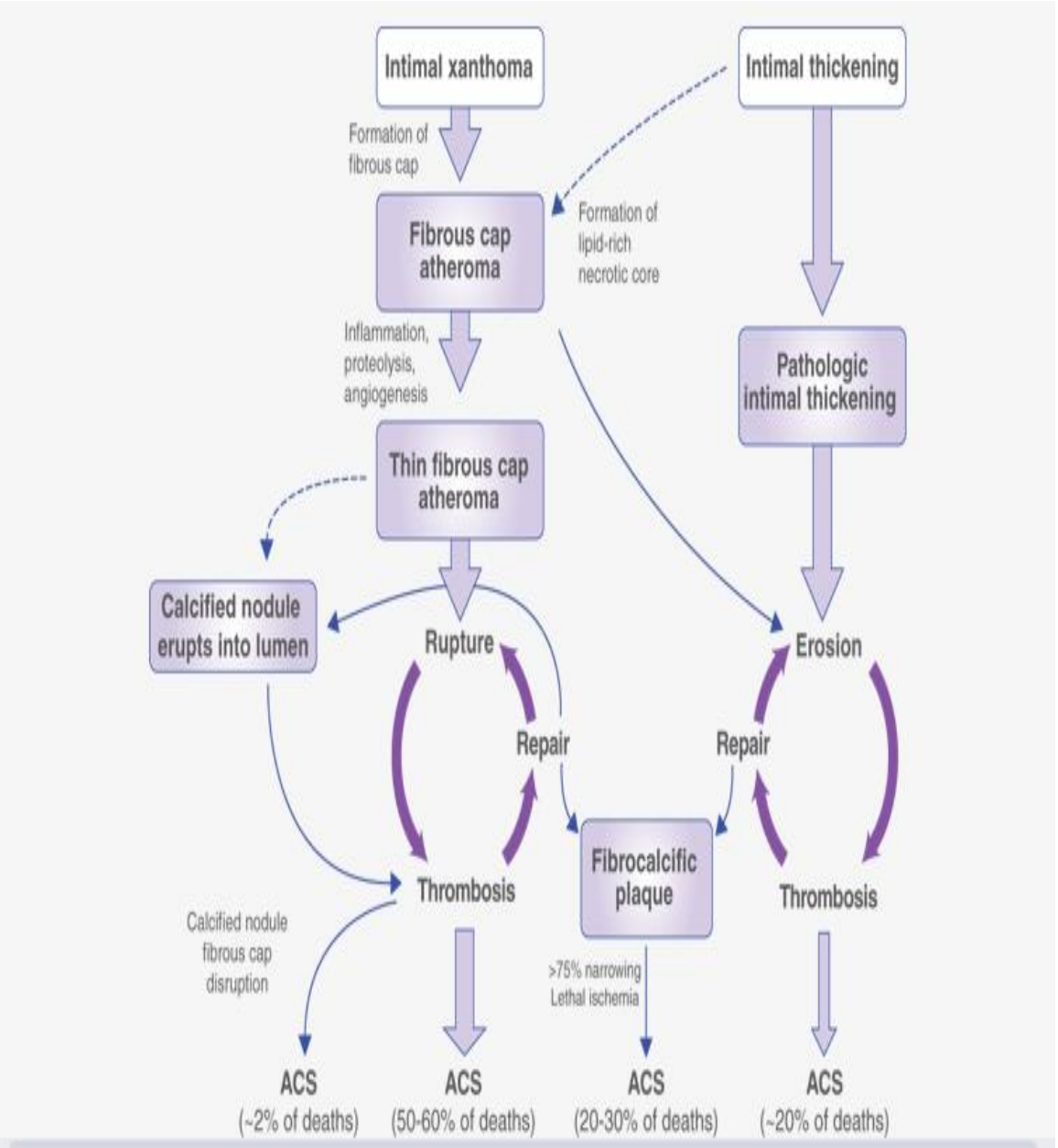
Is an important cause of myocardial infarction and is a disease of large and medium sized vessels. It has been characterized by

- endothelial dysfunction
- buildup of lipids, cholesterol,calcium within the intimal layer
- Vascular inflammation

Atheromatous plaque:

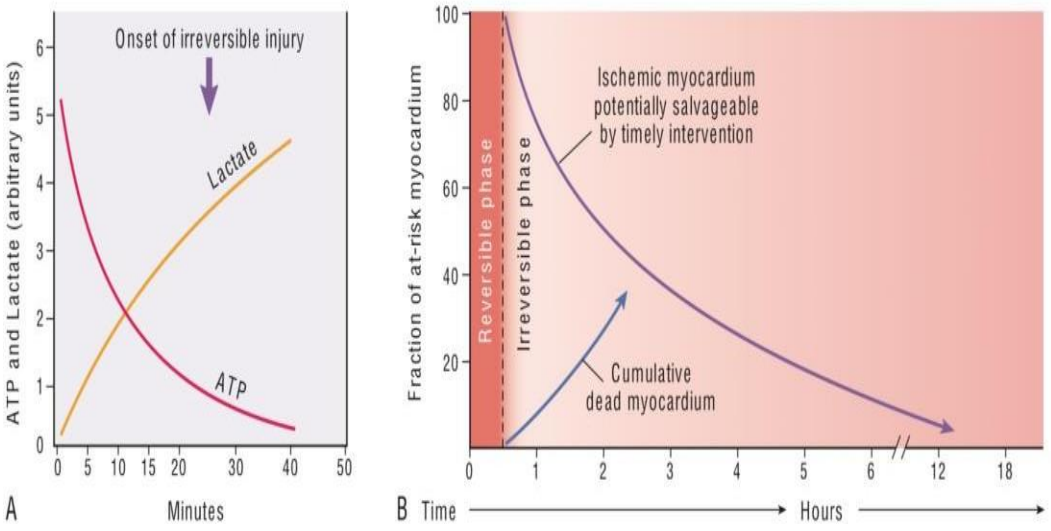
Atheromatous plaque contains a soft yellow core of lipid (mainly cholesterol) covered by a firm white fibrinous cap. It obstructs the blood flow and weakens the media layer and they themselves rupture causing acute thrombosis.

The risk factors include age, gender, hypertension, hyperlipidemia, diabetes, cigarette smoking.


Intimal xanthoma
Intimal thickening
Formation of fibrous cap
Fibrous cap atheroma
Formation of lipid-rich necrotic core
Inflammation, proteolysis, angiogenesis
Pathologic intimal thickening
Thin fibrous cap atheroma
Calcified nodule erupts into lumen
Rupture
Erosion
Repair
Repair
Fibrocalcific plaque
Thrombosis
Thrombosis
Calcified nodule fibrous cap disruption
>75% narrowing Lethal ischemia
ACS (~2% of deaths)
ACS (50-60% of deaths)
ACS (20-30% of deaths)
ACS (~20% of deaths)

PATHOPHYSIOLOGY OF ST ELEVATION MYOCARDIALINFARCTION

Myocardial infarction is commonly due to thrombotic occlusion of a coronary vessel caused by a rupture of plaque. Coronary arterial obstruction diminishes blood flow to a region of myocardium causing ischemia, rapid myocardial dysfunction, and myocyte death. The early biochemical changes include loss of ATP and accumulation of lactate.

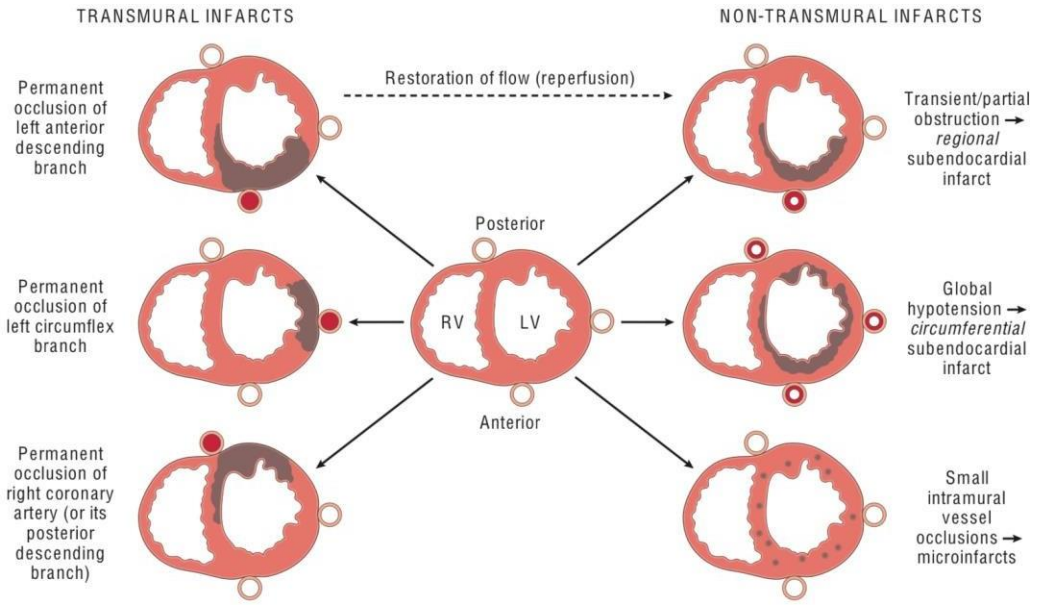


The progression of ischemic necrosis occurs from the sub endocardium. With more extended ischemia, a wavefront of cell death movesthrough the myocardium to enclose the transmural thickness.

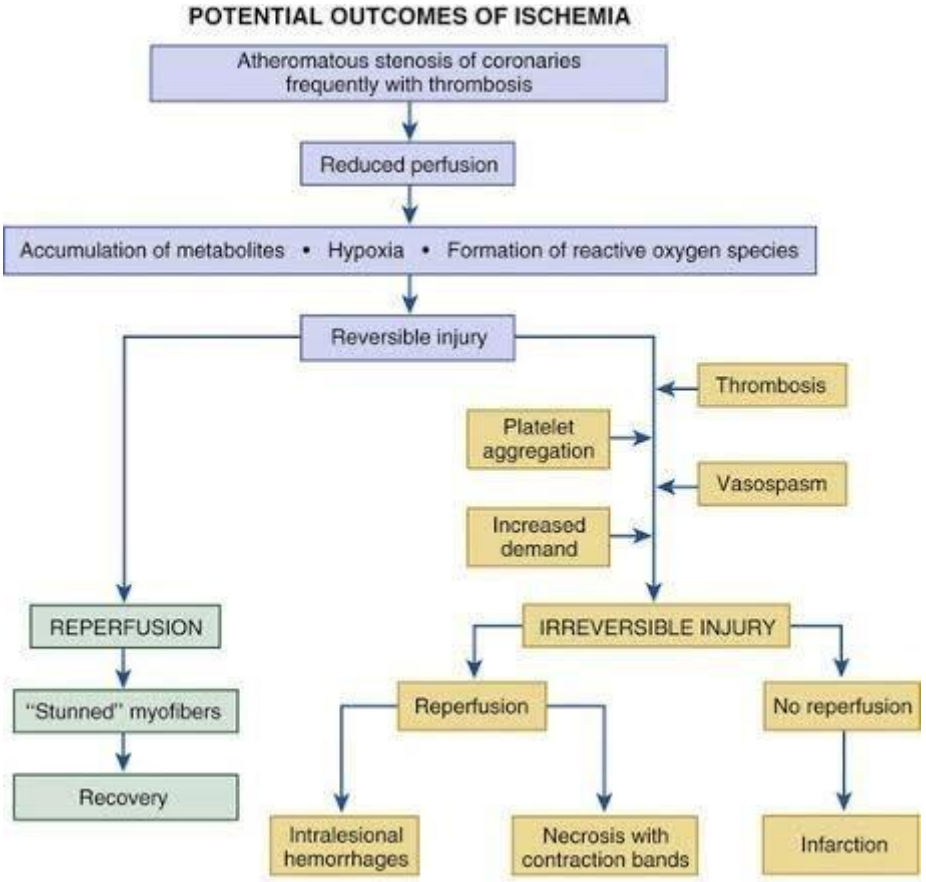
The atherosclerotic plaque is vulnerable as it contains a high number of inflammatory cells with fibrinous caps. Rupture of these plaques exposes to thrombogenic materials which activates the coagulation factors and platelets which triggers the formation of thrombus and further occlusion leading to myocardial necrosis.

Infarcted myocardium heals by formation of scar and is dependent on the inflammatory cascade triggered by the dead cells. Activation of the Renin Angiotensin system and activation of transforming growth factors converts fibroblasts to myofibroblasts promoting deposition of extra cellular matrix proteins leading to ventricular remodeling in the form of dilation and hypertrophy of the viable segments worsening the dysfunction.

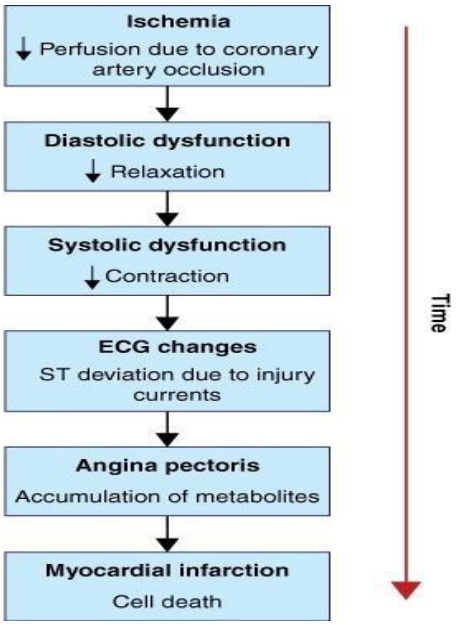
PATTERNS OF INFARCTION



Re perfusion is the restoration of blood flow to the ischemic myocardium and the goal is to salvage the cardiac muscle at risk and limitinfarcts size and alters the morphology of lethally injured cells.



ISCHEMIC CASCADE



- **ISCHEMIC PRE-CONDITIONING AND COLLATERALFLOW**

(8) In addition to the duration of ischemia, there are other factors that affect the consequences of coronary occlusion. If the myocardium is subjected to repetitive short ischemic episodes, it increases its protection against subsequent prolonged ischemic episodes and can reduce the level of ST-segment deviation. The mechanisms for the preconditioning are complicated and not fullyelucidated. They involve a much lower consumption of ATP, delayed development of acidosis and slower accumulation of lactate in preconditioned myocardium causing a reduced energy requirement and delayed myocyte death.

- **Myocardial stunning[9].**

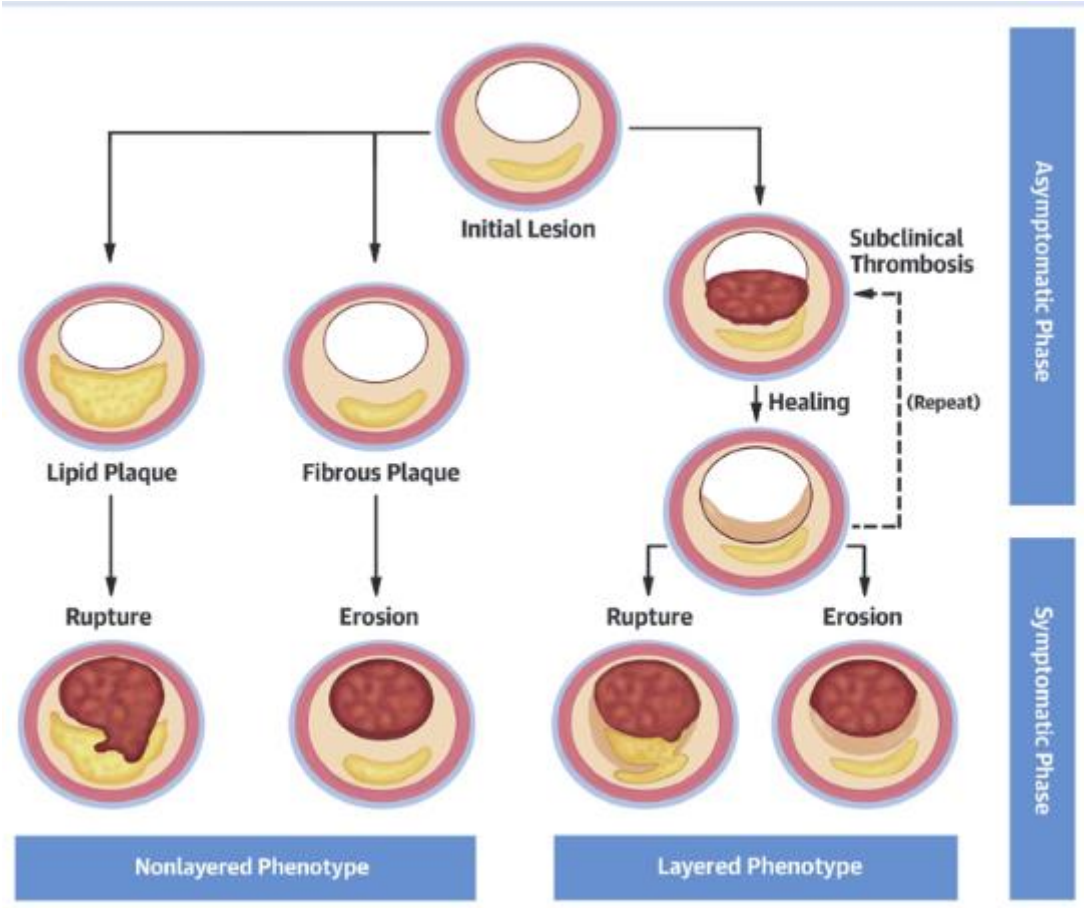
According to Braunwald and Kloner, stunned myocardium is "prolonged, post ischemic dysfunction of viable tissue recovered by reperfusion." Myocardial stunning can sohappen even in the absence of MI. The length and intensity of ischemia as well as the effectiveness of blood flow restoration determine the recovery of cardiac function.

- **Myocardial hibernation[10]**

The prolonged reduction in blood flow brought on by a tight coronary stenosis might also result in a decline in myocardial function. The myocardium's blood supply in the region distal to the stenosis is too low for itto function normally, but it is still adequate for it to survive. Myocardial hibernation is a phenomenon that has been labeled as "a state of persistently impaired myocardial and LV function at rest due to reduced coronary blood flow that can be partially or completely restored to normal if

the myocardial oxygen supply/demand relationship is favorably altered, either by improvingblood flow and/or by decreasing the demand." In contrast to infarcted myocardium the myocardium that is hibernating can regain its function.

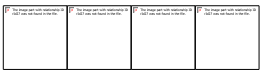
PROGRESSION OF A PLAQUE



CLINICAL FEATURES

Symptoms:

- Severe retrosternal pain with radiation to jaw, shoulder and back.
- Pain is usually severe, squeezing pain and is associated withsweating, palpitations, dyspnoea
- ST elevation Myocardial Infarction may have atypical presentations in women, chronic kidney disease, diabetes.
- Some patients may have anginal equivalents like dyspnoea,syncope, unexplained hypotension, palpitations.
- The severity of angina is given by Canadian Cardiovascular society functional classification and the functional status is assessed by New York Heart Association classification.



NEW YORK HEART ASSOCIATION CLASSIFICATION

Class I	No limitation of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation or dyspnoea.
Class II	Slight limitation of physical activity. Comfortable at rest but ordinary physical activity results in fatigue, palpitation or dyspnoea.
Class III	Marked limitation of physical activity. Comfortable at rest but less than ordinary activity results in fatigue, palpitation or dyspnoea.
Class IV	Unable to carry out any physical activity without discomfort. Symptoms at rest. If any physical activity is undertaken, discomfort is increased.

Canadian Cardiovascular society classification

Class	Level of symptoms
Class I	'Ordinary activity does not cause angina' Angina with strenuous or rapid or prolonged exertion only
Class II	'Slight limitation of ordinary activity' Angina on walking or climbing stairs rapidly, walking uphill or exertion after meals, in cold weather, when under emotional stress, or only during the first few hours after awakening
Class III	'Marked limitation of ordinary physical activity' Angina on walking one or two blocks on the level or one flight of stairs at a normal pace under normal conditions[a]
Class IV	'Inability to carry out any physical activity without discomfort' or 'angina at rest'

Signs:

- Physical findings are directed towards identifying hemodynamic instability, pulmonary congestion, mechanical complications of Myocardial Infarction.
- Cardiogenic shock can be identified in the presence of feeblepulses, raised jugular venous pulse.

KILIP CLASSIFICATION OF HEART FAILURE

STAGE	DEFINITION
1	No clinical signs of heart failure
2	Crackles in lungs, s3, and elevated jugular venous pressure
3	Acute pulmonary edema
4	Cardiogenic shock or hypotension (measured as Systolic Blood pressure <90 mm Hg), and evidence of vasoconstriction (oliguria, cyanosis, or sweating)

COMPLICATIONS OF MYOCARDIAL INFARCTION

1. Ventricular dysfunction
2. Cardiogenic shock
3. Congestive Cardiac failure
4. Right ventricular infarction
5. Arrhythmia
   - Ventricular premature beats
   - Ventricular tachycardia and fibrillation
   - Accelerated idioventricular rhythm
   - Supraventricualr arrhythmia
6. Pericarditis
7. Thromboembolism
8. Left Ventricular aneurysm

INVESTIGATIONS

CARDIAC ENZYMES:

Cardiac enzymes are released from the injured and necrotic myocardium. They included the CK-MB, Cardiac troponins, Myoglobin.

MYOGLOBIN:

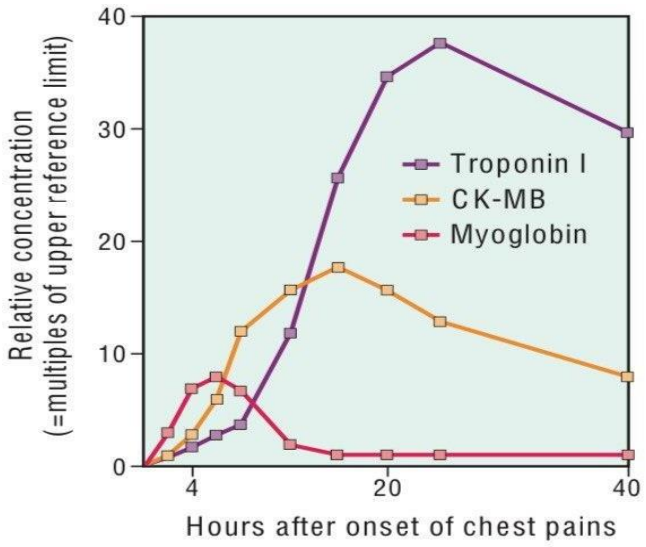
It originates from cardiac and skeletal muscles. It is an early markerto rise in Myocardial Infarction within 2 hours and returns to normal by 1 day.

CK-MB[11]:

- It is detectable within 3-6 hrs
- Can detect early re infarction.
- It can give false positive results with muscle trauma, cardiacsurgery.

Cardiac Troponins:

- They are released within 3 to 4 hours and return to normal by10 to 14 days.
- It is the best marker for MI with skeletal muscle injury.

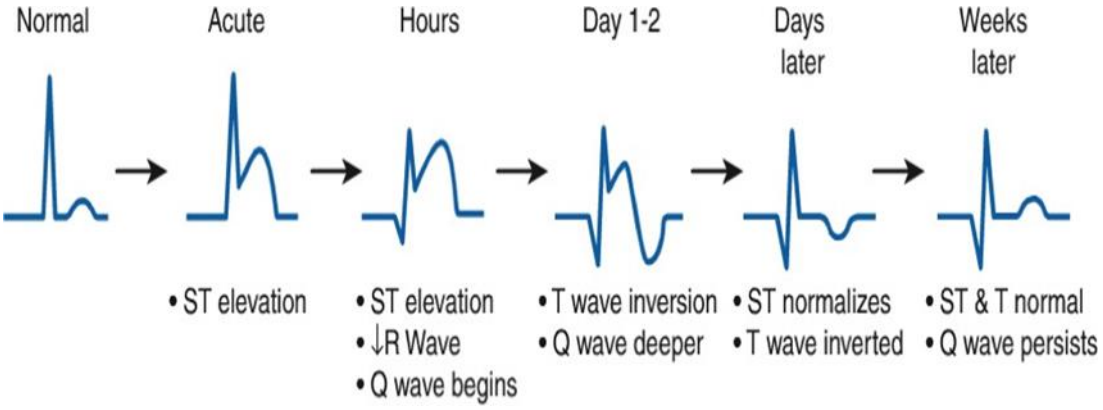


Electrocardiography in Myocardial Infarction:

Though Coronary angiogram is the valuable method to locate the artery affected in Myocardial Infarction ECG can be used in identifying thevessel involved.

The series of ECG changes are:

- Tall, Positive, hyper acute T waves are the earliest change.
- This is followed by elevation of the ST segments.
- T-wave inversion develops over hours to days.
- And finally, diminished R-wave amplitude or Q waves occur, representing significant myocardial necrosis and replacement by scartissue.



ECHOCARDIOGRAPHY:

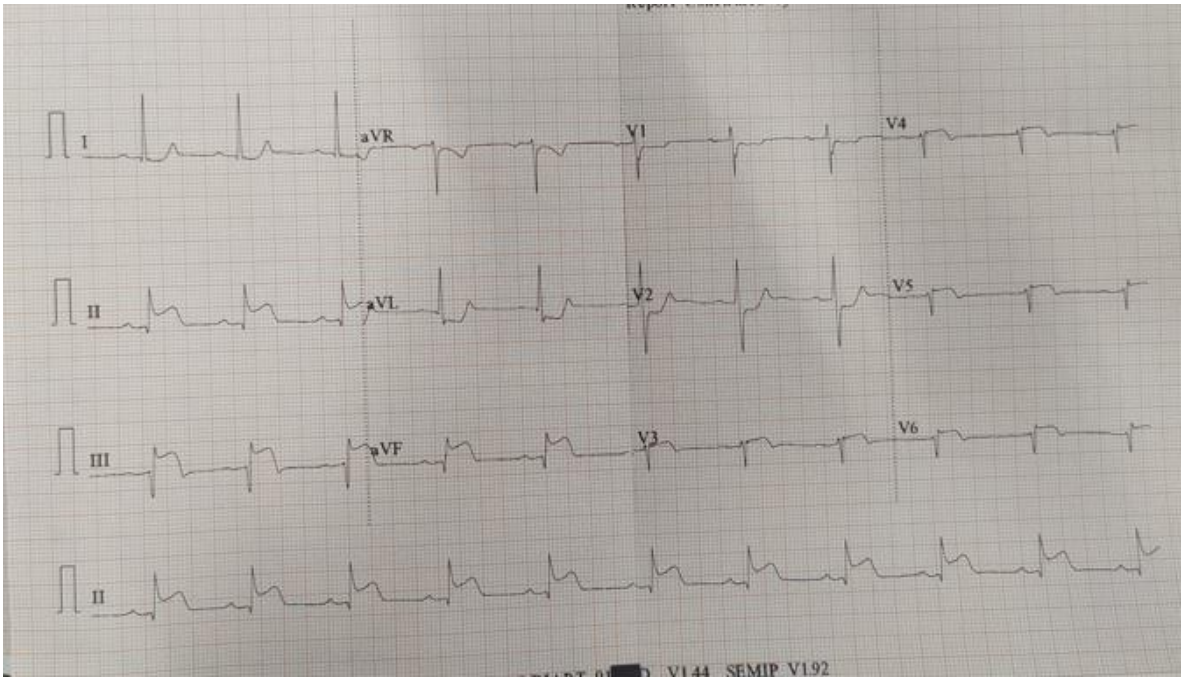
- This could show irregularities in wall motion, chamber size, myocardial fibrosis, edema, ventricular wall thinning, hypertrophy,and other things.
- Color Doppler can be used to assess cardiac hemodynamics. It canalso be used to show how an ischemia progresses into an infarction.

CORONARY ANGIOGRAPHY

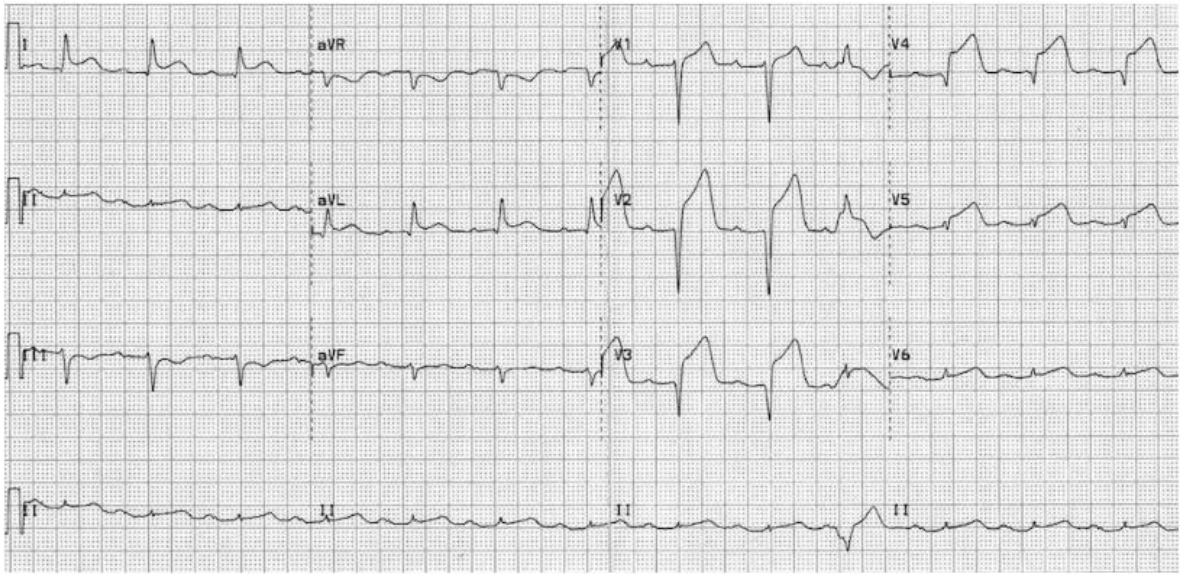
Angiography is essential for determining the size, location, and severity of lesions as well as for planning revascularization (PCI or CABG). On coronary angiography, substantial coronary lesions can be seen in more than 80% of patients with UA/NSTEMI.

Location of MI	**Leads affected**	**Vessel involved**
Anterior wall	V2 to V4	Left Anterior Descending artery (LAD) – Diagonal branch
Septal wall	V1 and V2	Left Anterior Descending artery (LAD) – septal branch
Inferior wall	II, III, aVF	Right coronary artery (RCA) – Posterior Descending branch
Lateral wall	I, aVL, V5, V6	Left coronary Artery (LCA) – Circumflex branch
Posterior wall	V1 to V4	Left Coronary Artery (LCA) – Circumflex branch Right Coronary Artery (RCA) – Posterior Descending branch

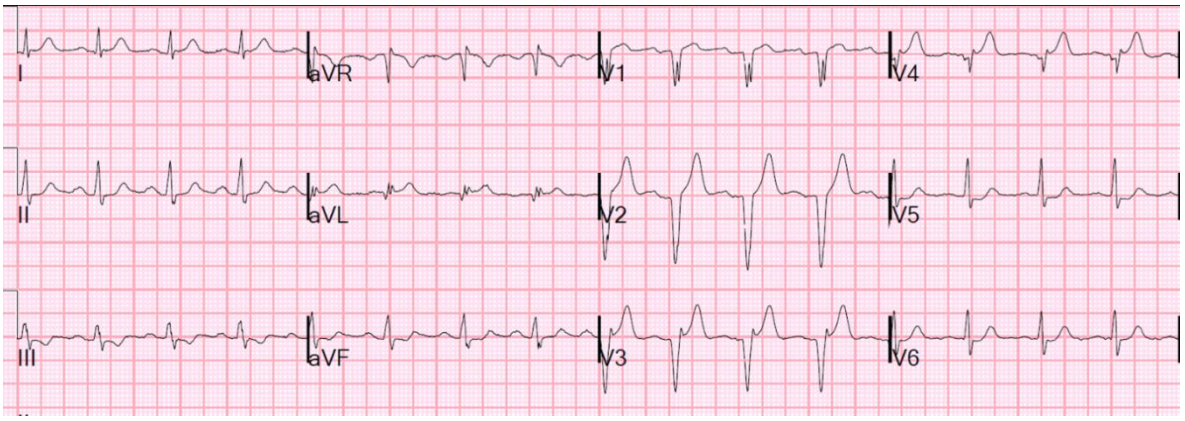
INFERIOR WALL MYOCARDIAL INFARCTION



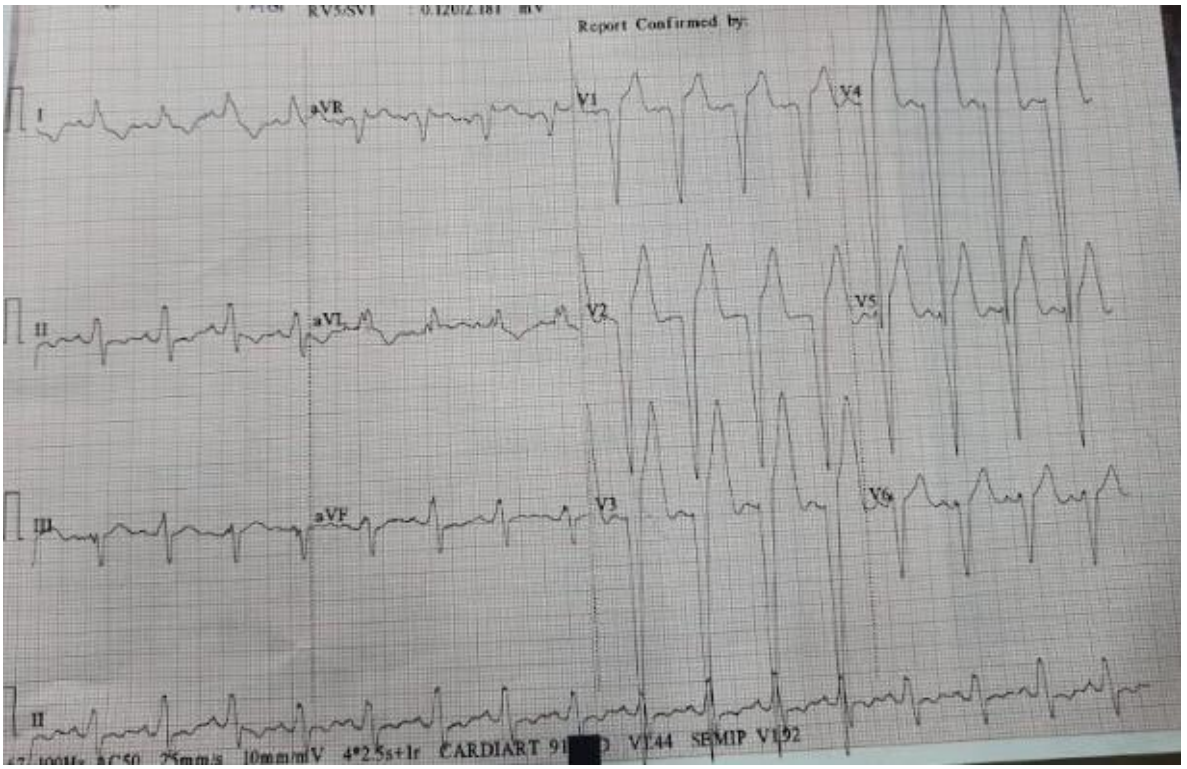
ANTERIO LATERAL WALL INFARCTION



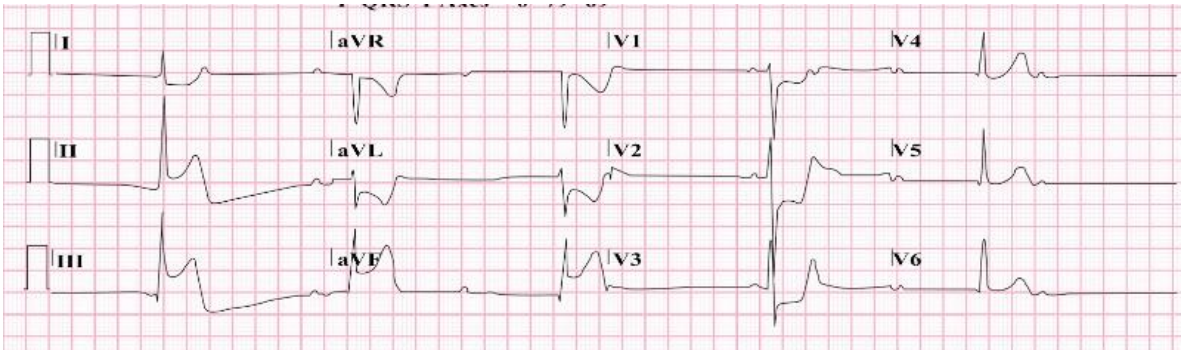
ANTERIOR WALL INFARCTION



LBBB



INFEROPOSTERIOR MYOCARDIAL INFARCTION



ESTIMATING INFARCT SIZE[12]

Myocardial infarct size is important to measure since it has asubstantial impact on prognosis.

Several clinical approaches have been tested to see if they can accurately predict infarct size.Planning the rehabilitation program and forecasting the severity of the infarction both require early assessment of infarct size.

Dual single photon emission computed tomography (dual SPECT)using 201 T1 and technetium-99m pyrophosphate is a new technique for estimating infarct size.

However, due to its widespread availability, the 12-lead ECG is still the gold standard for detecting the presence and location of infarcts and are non- invasive, affordable, and repeatable.

Computer simulations that demonstrate an orderly and predictableseries of changes that happens in QRS associated with infarcts of various locations and sizes are based on studies of the activation sequence in QRS complexes in both canine and human hearts. Selvester et al. improved the outcomes of these computer simulations to provide both qualitative and quantitative criteria for estimating infarct size through several pilot investigations in patients with localized wall motion abnormalities revealed by ventriculography.

SELVESTER QRS SCORING SYSTEM[13]

Selvester et al. first suggested the Selvester QRS Score, an electrocardiographic (ECG) approach for evaluating MI size, and Wagner andPalmeri later improved it. The Selvester QRS score, which was introduced forthe first time in 1972, transforms variations in cardiac electrical activity into data that aids in identifying the size and location of infarcts.

Research has revealed that the myocardial infarct size predicted bythis scoring method and the size determined at autopsy have a reliable and strong association. Numerous investigations have shown that the estimated QRS score in patients after a MI gave important information about prognosis. A higher QRS score is a sign that ventricular tachyarrhythmias are more common, that more defibrillatory shocks are required, and that the effectiveness of cardiac resynchronization therapy is lowered. This QRS scoring technique is used to calculate an electrocardiographic estimate of the extent of a myocardial infarct. Ten ECG leads (1, II, aVL, aVF, V1-V6) are weighted based on Selvester findings about their capacity to indicate infarct size[15]. Some of the original grading factors are not taken into consideration.

Complete 50-Criteria, 31-point QRS Scoring System*

Lead	Maximum Lead Points	Criteria	Points
I	(2)	Q ≥ 30ms	(1)
		R/Q ≤ 1	(1)
		R ≤ 0.2mV	(1)
II	(2)	Q ≥ 40ms	(2)
		Q ≥ 30ms	(1)
aVL	(2)	Q ≥ 30ms	(1)
		R/Q ≤ 1	(1)
aVF	(5)	Q ≥ 50ms	(3)
		Q ≥ 40ms	(2)
		Q ≥ 30ms	(1)
		R/Q ≤ 1	(2)
		R/Q ≤ 2	(1)
V1			
Anterior	(1)	Any Q	(1)
Posterior	(4)	R/S ≥ 1	(1)
		R ≥ 50ms	(2)
		R ≥ 1.0mV	(2)
		R ≥ 40ms	(1)
		R ≥ 0.6mV	(1)
		Q and S ≤ 0.3mV	(1)
V2			
Anterior	(1)	Any Q	(1)
		R ≤ 10ms	(1)
		R ≤ 0.1mV	(1)
		R ≤ RV_1 mV	(1)
Posterior	(4)	R/S ≥ 1.5	(1)
		R ≥ 60ms	(2)
		R ≥ 2.0mV	(2)
		R ≥ 50ms	(1)
		R ≥ 1.5mV	(1)
		Q and S ≤ 0.4mV	(1)
V3	(1)	Any Q	(1)
		R ≤ 20ms	(1)
		R ≤ 0.2mV	(1)
V4	(3)	Q ≥ 20ms	(1)
		R/S ≤ 0.5	(2)
		R/Q ≤ 0.5	(2)
		R/S ≤ 1	(1)
		R/Q ≤ 1	(1)
		R ≤ 0.7mV	(1)
V5	(3)	Q ≥ 30ms	(1)
		R/S ≤ 1	(2)
		R/Q ≤ 1	(2)
		R/S ≤ 2	(1)
		R/Q ≤ 2	(1)
		R ≤ 0.7mV	(1)
V6	(3)	Q ≥ 30ms	(1)
		R/S ≤ 1	(2)
		R/Q ≤ 1	(2)
		R/S ≤ 3	(1)
		R/Q ≤ 3	(1)
		R ≤ 0.6mV	(1)

*When more than one criterion in the brace is met, the one with the most points is selected (1 point = 3% of myocardium infracted).

Several modifications of the QRS scoring system are available. Onesuch simplified score is the modified QRS score which is a 29-point score which is shown below.

MODIFIED SELVESTER QRS SCORE

TABLE 3. *Modified QRS Scoring System*

Lead	Duration (msec)		Amplitude ratios		Max points
I	Q ≥ 30	(1)	R/Q ≤ 1	(1)	2
II	Q ≥ 40	(2)			
	Q ≥ 30	(1)			2
aV_L	Q ≥ 30	(1)	R/Q ≤ 1	(1)	2
	Q ≥ 50	(3)	R/Q ≤ 1	(2)	
aV_F	Q ≥ 40	(2)			
	Q ≥ 30	(1)	R/Q ≤ 2	(1)	5
	Any Q	(1)			
V_1	R ≥ 50	(2)			
	R ≥ 40	(1)	R/S ≥ 1	(1)	4
	Any Q or R ≤ 20	(1)			
V_2	R ≥ 60	(2)			
	R ≥ 50	(1)	R/S ≥ 1.5	(1)	4
V_3	Any Q or R ≤ 30	(1)			1
V_4	Q ≥ 20	(1)	R/Q or R/S ≤ 0.5	(2)	
			R/Q or R/S ≤ 1	(1)	3
V_5	Q ≥ 30	(1)	R/Q or R/S ≤ 1	(2)	
			R/Q or R/S ≤ 2	(1)	3
V_6	Q ≥ 30	(1)	R/Q or R/S ≤ 1	(2)	
			R/Q or R/S ≤ 3	(1)	3

When confounding factors like left or right ventricular hypertrophy, left or right bundle branch block, or left anterior or posterior fascicular block are removed, the modified Selvester QRS scoring system reaches an acceptable level of specificity. The entire 29-point scoring system[14]obtained 98% specificity when a score of more than 2 points was needed to identify myocardial infarction, with each criterion exhibiting at least 95% specificity.

This scoring system's usefulness for identifying and evaluatinginfarct size in some individuals will likely be constrained by the confounding variables, which will likely reduce its specificity. However, it is necessary to establish the specificity. The QRS scoring system also permits respectable levels of inter and intra observer variation. There was 91% or more intra observer agreement and 92% or more inter

observer agreement for each of the37 criteria.

PROGNOSTIC VALUE OF MODIFIED SELVESTER QRS SCORE

Patients with MI who have higher QRS scores also have greater fatality rates. Patients with scores of 10 or more had survival rates of 81% and 52% at one- and five-year intervals respectively. Patients with scoresof 0 had a survival rate of 95% at the end of one year and 88% at the end of five years[14].

ECG assessment is simple to do and non-invasive, even though the extent of the infarct can be accurately predicted using magnetic resonance imaging, single photon emission computed tomography, or positronemission tomography.

Pathological Q-waves are the most dependable electrocardiographic indicator of an old myocardial infarction; however, they are unreliable since they do not represent the affected myocardial segments. In determining the magnitude of the infarct, Selvester QRS score has proven to be

more accurate than several other scoring systems, including the Minnesota score, novacode, and cardiac infarction injury score. The recovery of LV function following an acute MI has also been successfully predicted using this scoring method, which also provides the most information about the prognosis.

As a result, the MODIFIED SELVESTER QRS grading systemcan be an extremely useful clinical tool for clinicians.

ROLE OF ST SEGMENT RESOLUTION IN MI:

Quick methods are needed to judge the efficacy of reperfusion in clinical studies. Recently, ST segment resolution has become much more significant from this standpoint. ST segment resolution are widely utilised in clinical practise and several research studies shows it as an easy way to predictreperfusion in patients receiving reperfusion treatment for acute ST elevation myocardial infarction.

The ST segment resolution is used to assess the reperfusion when coronary angiogram is not available and the ST segment resolution is better thanTIMI score[16] in evaluating the outcome in primary angioplasty. Thus, it is a good predictor of myocardial reperfusion.

TIMI FLOW GRADE[17]

This classification estimates the coronary blood flow in the infarct-related artery after thrombolysis.

TIMI 0	Refers to the absence of any antegrade flow beyond a coronary occlusion
TIMI 1	Flow is minimal antegrade coronary flow beyond the occlusion, although filling of the distal coronary bed is incomplete
TIMI 2	Flow is delayed or sluggish antegrade flow with complete filling of the distal territory
TIMI 3	Flow is normal flow, which fills the distal coronary bed completely.

Many studies have shown the prognostic value of ST-segment resolution (STR) and isused to assess the efficacy of reperfusion therapy in myocardial infarction. An incomplete (<50%) STR is a marker of failed thrombolysis.

MANAGEMENT OF MYOCARDIAL INFARCTION

Includes

1. Prehospital care

Includes recognition of symptoms and rapid deployment of the emergency team and transportation of the patient to the hospital facility

2. Management in emergency department

- Aspirin 160 - 325 mg loading dose, supplemental oxygen
- Relief of discomfort by sublingual nitroglycerin upto three doses of 0.4mg, morphine, intravenous beta blockers.
- Fibrinolysis and PCI

3. Hospital phase management

- Bed rest, diet, preventing constipation
- Sedation

4. Pharmacotherapy

- Anti thrombotic agents (anti platelet and anticoagulant)
- Beta blockers
- Inhibition of Renin Angiotensin- Aldosterone system

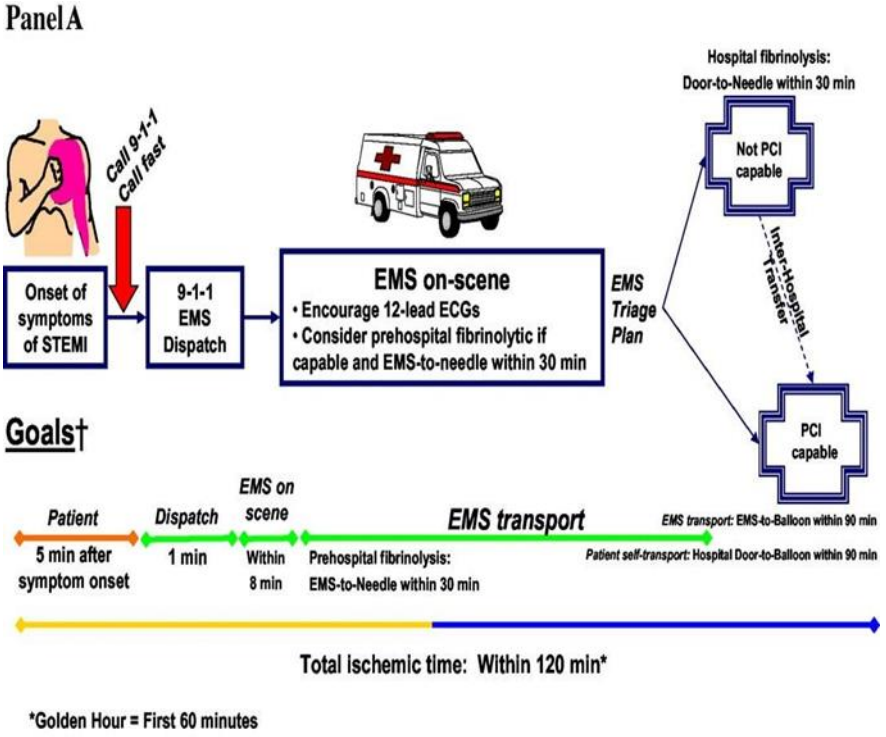
5. Managing complications

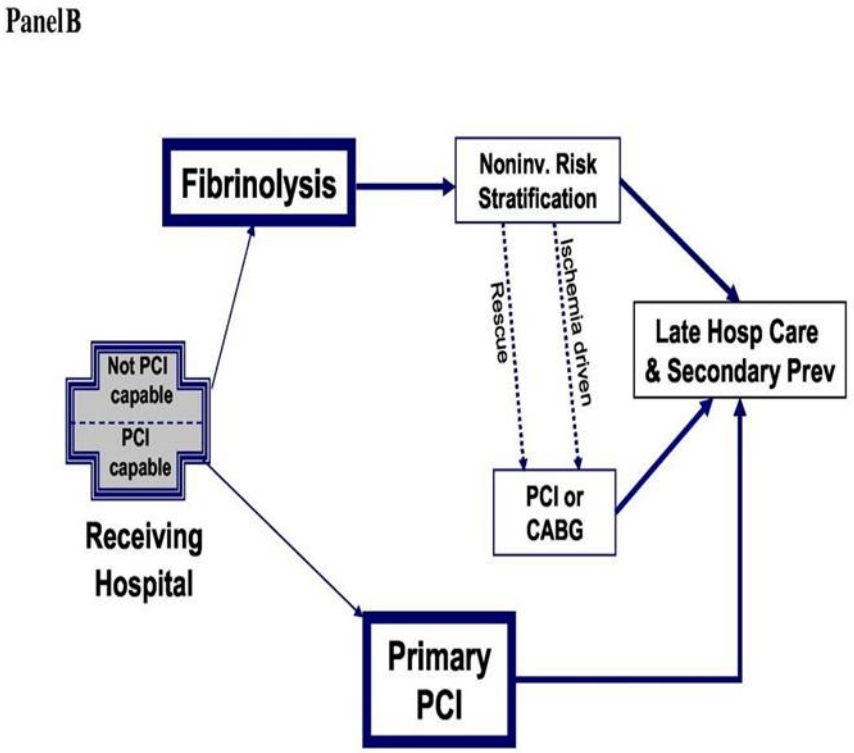
6. Post infarction risk stratification and management

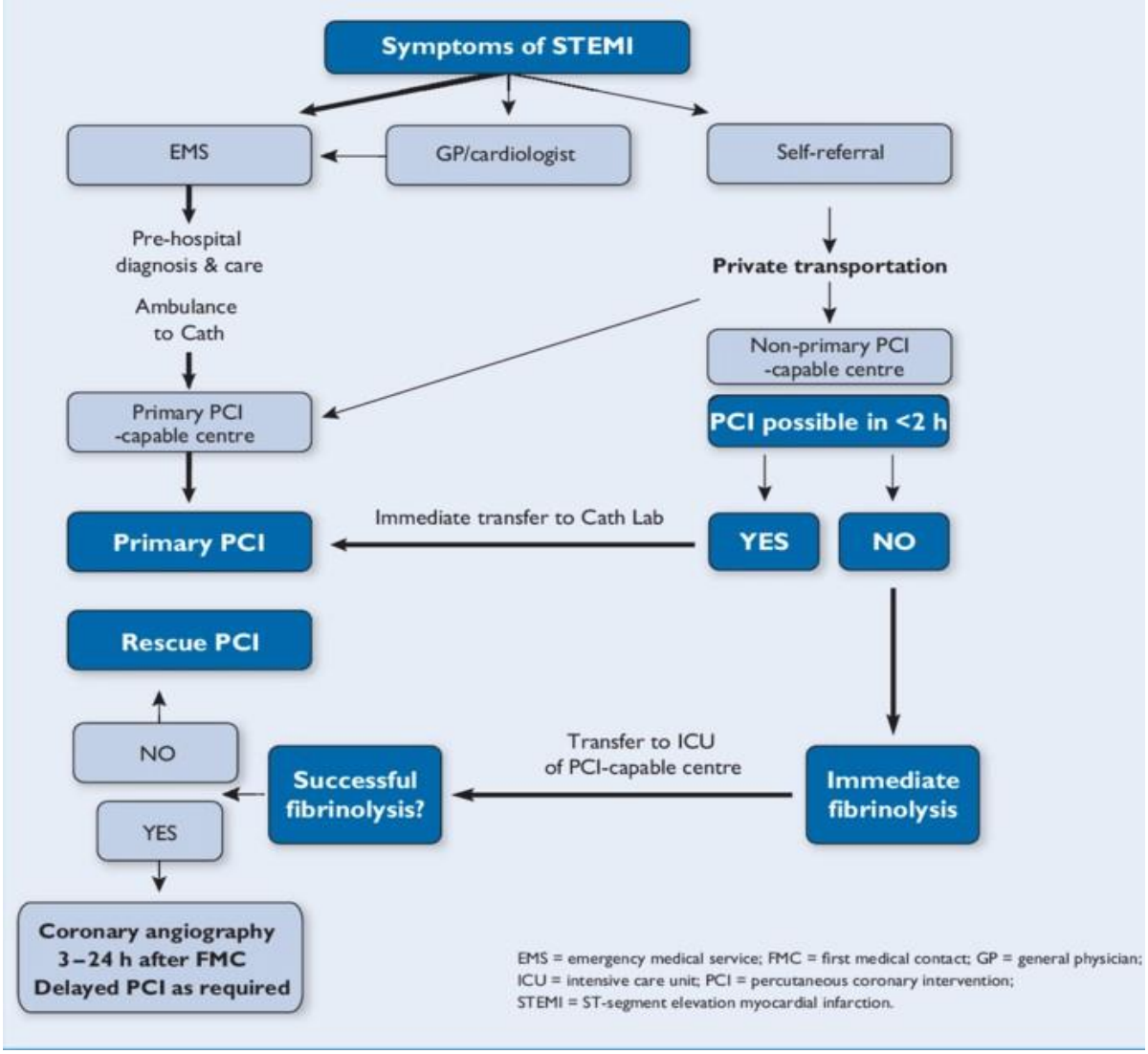
7. Secondary Prevention

8. Managing other risk factors for atherosclerosis.

Pre hospital care







RISK STRATIFICATION IN MI:

The prognosis of ACS in patients varies. Young individuals with newly developed angina, no ECG abnormalities, no increased biomarkers, and no hemodynamic instability are at one extreme of the continuum. After 10 days from the onset of symptoms, these patients should have non-invasive testing (TMT, stress echocardiography), and treated accordingly. On the other extremeare individuals who require an early invasive technique such as PCI as they have a history of recurring or rest angina, recent changes in their ECG, increased biomarkers, or are hemodynamically unstable.

TIMI RISK SCORE[18]

Age ≥ 75	3
Age 65 – 75	2
DM or HTN or Angina	1
SBP < 100 mmHg	3
HR > 100 bpm	2
Killip II – IV	2
Weight < 67 kg	1
Anterior STE or LBBB	1
Time to treatment > 4 hours	1

This estimates the risk of all-cause mortality of 30 days in the patients

THROMBOLYTIC AGENTS

1st Generation:

- Urokinase, Streptokinase

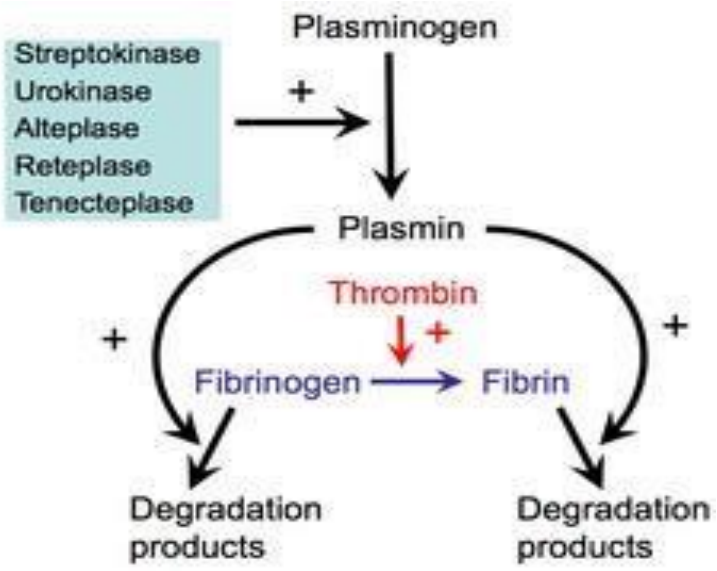
2nd Generation

- Recombinant tissue plasminogen activator (r- t PA, alteplase,duteplase)

3rd Generation

- Reteplase, Lanoteplase, Tenecteplase,
- Staphylokinase, Recombinant glycosylated plasminogen activator

MECHANISM OF ACTION:



COMPARASION OF THROMBOLYTIC AGENTS[21]:

Characteristic	Streptokinase (first generation)	Alteplase (second generation)	Third generation	
			Tenecteplase	Reteplase
Plasma half-life (min)	18-23	3-8	18-20	15-18
Metabolism	Hepatic	Hepatic	Hepatic	Renal
Fibrin specificity	Nonspecific	High	Very high	Moderate
Infusion/bolus dosing	Infusion (1 h)	Bolus + 90 min infusion	Single bolus	Double bolus
Weight-based dosing	No	Yes	Yes	No
Efficacy (TIMI 2 or 3 flow at 90 min)	60%-68%	75%-84%	85%	84%

TIMI: Thrombolysis in myocardial infarction

STREPTOKINASE:

Because t-PA is ten times more expensive than streptokinase, thelatter remains the fibrinolytic of choice in many developing countries. It has been demonstrated in numerous studies that streptokinase is equally effectiveas other fibrin-specific agents in reducing mortality. Streptokinase is the mostused thrombolytic agent in developing countries like India.

Streptokinase is produced by beta-haemolytic streptococci. Although it lacks intrinsic enzymatic activity, it joins plasminogen in a stable, noncovalent 1:1 complex. As a result, plasminogen undergoes a conformationalchange that reveals the active site where arginine 560 on free plasminogen is broken down to make free plasmin.

There is no question that early thrombolysis lowers mortality by 20–50%. [19]This is accomplished by restoring blood flow to the ischemic myocardium, which reduces the infarct size by 15% to 30%. Although 0.5% of people who take thrombolytics experience bleeding, the risk of bleeding was

comparable across the thrombolytic and placebo groups in multiple significant trials.

INDICATIONS OF THROMBOLYSIS:

- Symptoms and signs consistent with Myocardial infarction
- Time of symptom onset 12 hours or less
- New Left bundle branch block
- True Posterior wall MI
- In facilities where invasive strategy is not an option

CONTRAINDICATIONS OF THROMBOLYSIS

ABSOLUTE CONTRAINDICATIONS:

- Recent intracranial haemorrhage (ICH)

  Structural cerebral vascular lesion

  Intracranial neoplasm
- Ischemic stroke within three months
- Possible aortic dissection
- Active bleeding or bleeding diathesis (excluding menses)
- Significant head injury or facial trauma within three months
- Recent Intracranial or spinal surgery
- Severe uncontrolled hypertension

RELATIVE CONTRAINDICATIONS:[45]

- History of severe and poorly controlled hypertension
- Severe hypertension at presentation (systolic blood pressure >180mmHg or diastolic blood pressure >110mmHg)
- Prolonged (>10 minutes) cardiopulmonary resuscitation (CPR) or majorsurgery within three weeks.

- History of ischemic stroke.
- Pregnancy
- Active peptic ulcer
- Concurrent therapy of anticoagulants is associated with an elevated international normalized ratio (INR) higher than 1.7 or a prothrombintime (PT) longer than 15 seconds.

ADVERSE EFFECTS OF STREPTOKINASE[20]:

- Hypotension is the most common complication
- Bleeding manifestations in the form of bleeding gums,haemorrhagic Cerebrovascular accidents.
- Allergic manifestations in the form of skin rashes.
- Rarely, bradycardia and arrythmias.

STREPTOKINASE IN MYOCARDIAL INFARCTION:

The GISSI trial[22] recommended Streptokinase can be used in allmyocardial infarction with no contraindications and showed early presentationto the hospital had decreased the mortality. GISSI trial was followed by multiple other trials including GUSTO, GISSI -2 and ISIS-3 which compared streptokinase with other t-PA and showed no significant difference in the mortality rates with streptokinase compared with other t-PA.

Streptokinase also preserves LV function in MI patients[23]. TheISIS-2 trial unequivocally proves that the early survival benefit brought on byfibrinolytic therapy in acute myocardial infarction persists for many years following treatment. Fibrinolytic therapy has survival advantages that persist for at least 10 years after treatment[12].

PERCUTANEOUS TRANSLUMINAL CORONARY ANGIOPLASTY:

Though studies have shown that PTCA is superior to streptokinase interms of reinfarction and avoids the haemorrhagic manifestations of thrombolysis is not readily available and operator dependent procedure.

FAILED THROMBOLYSIS:

Thrombolytic agents have not achieved reperfusion in at least 15–40% of patients[24]. Failed thrombolysis is associated with increased risk of death and ventricular dysfunction. The most useful method in the diagnosis of failed thrombolysis is the standard 12 lead ECG. Angiographic TIMI-3 flow is

only achieved in 25 to 50% of patients with thrombolysis. Repeat

thrombolysisproves no benefit and they are successfully managed with rescue angioplasty.

MATERIALS AND METHOD

STUDY POPULATION:

This study was conducted among 61 patients in Chengalpattu Medical College Hospital who are diagnosed with Acute myocardial infarctionreceiving thrombolytic therapy with streptokinase.

STUDY DURATION:

One year from April 2021 to March 2022

SAMPLE SIZE: 61

INCLUSION CRITERIA:

Patients diagnosed with new onset acute ST Elevation Myocardial Infarction in Chengalpattu medical college between 30-80 years of age presenting within 24 hours of onset of symptom and eligible for reperfusiontherapy (presenting within 12 hours of symptom onset or presenting thereafter with persistent symptoms) were included in the study. The diagnosis of STEMIwas based on ECG criteria[2] for ST segment elevation in a patient presenting with typical history of angina and elevated CK-MB.

EXCLUSION CRITERIA:

- Patients with bundle branch block, paced rhythm, fascicularblock.
- ECG signs of ventricular hypertrophy.
- Patients with cardiogenic shock.
- Patients with other causes of ST elevation such asPericarditis.

Blood samples are collected for Blood sugar, urea, creatinine, FastingLipid profile, CK- MB, complete hemogram.

CRITERIAS:

Hypertension if Systolic BP >140 mmHg and Diastolic BP >90 mmHg. Diabetic if Fasting Glucose >126 mg/dl and 2-hour post prandial glucose > 200 mg/dl.

Dyslipidemia if LDL cholesterol >100 mg/dl, TGL>150 mg/dl, HDL<40 mg/dl in men and <50 mg/dl in women.

METHODOLOGY:

The patients were diagnosed with acute myocardial infarction based on a standard 12 lead ECG and after the confirmation of diagnosis of myocardial Infarction are thrombolysed by Streptokinase 1,50,000 IU intravenously by infusion over 60 minutes and 90 minutes after the initiation of treatment patientwas taken another standard 12 lead ECG.

1. QRS score calculated according to the modified Selvester scoringsystem [26] based on 37 criteria giving a total of 29 points. QRS score of >4 is considered as non-resolution.
2. ST segment elevation, measured 20 ms after the J point. The height (in mm) of ST segment elevations was measured in leads I, aVL and V1 through V6 for anterior infarctions, and in leads II,III, aVF, V5 and V6 for inferior infarctions. The sum of all measured ST segment elevations was expressed as STE1.
3. In the ECG after 90 minutes of initiation of treatment, the sum ofthe measured ST elevations is taken as STE2.
4. The difference between the STE1 and STE2 is taken as STsegment resolution (STR)

5. Patients were also classified into resolution group (STR is ≥ 50%of STE1) and non-resolution group (STR is < 50% of STE1)

STATISTICAL ANALYSIS:

Analysis was performed in SPSS 24 software. All continuous variableswere presented as mean ±standard deviation if they were normally distributed.Comparisons between the two individual groups were performed using the unpaired *t*-test (parametric). Pearson's correlation coefficient test was performed to study the correlation between the QRS score and the STR. All tests were two-sided and a probability value of $p < 0.05$ was considered statistically significant.

RESULTS:

TABLE 1: BASE LINE CHARACTERISTICS OF WHOLESTUDY COHORT AND THE TWO STUDY GROUPS WITHRESPECT TO ST SEGMENT RESOLUTION

	Whole CohortN=61	Resolution Groupn=33	Non-Resolution Group n=28	p-value
Age	56.07 ±11.845	51.82 ±11.015	61.07 ±10.951	**0.002**
Male	43 (70.5%)	21 (48.8%)	22 (51.9%)	0.202
Female	18 (29.5%)	12 (66.6%)	6 (33.7%)	
Smoking	24 (39.3%)	10 (41.6%)	14 (58.4%)	0.117
Alcohol	28 (45.9%)	12 (42.8%)	16 (57.9%)	0.105
Hypertension	28 (45.9%)	13 (46.4%)	15 (53.5%)	0.268
Diabetes	31 (50.8%)	14 (45.1%)	17 (54.9%)	0.154
Cardiac Failure	23 (37.7%)	6 (26.1%)	17 (73.9%)	**0.01**
Anterior	40 (65.6%)	17 (42.5%)	23 (57.5%)	0.12
Inferior	21 (34.4%)	16 (76.2%)	5 (23.8%)	
Dyslipidaemia	26 (42.6%)	16 (61.5%)	10 (38.5%)	0.315

TABLE 2: BASE LINE CHARACTERISTICS OF WHOLE STUDYCOHORT AND THE TWO STUDY GROUPS WITH RESPECT TO QRS SCORE.

	WHOLE COHORT	QRS<4	QRS>4	p-value
Age	56.07 ±11.845	52.18 ±11.574	60.96 ±10.442	**0.003**
Smoking	24 (39.3%)	13 (54.1%)	11 (45.9%)	0.842
Alcohol	28 (45.9%)	16 (57.1%)	12 (42.9%)	0.839
Hypertension	28 (45.9%)	13 (46.4%)	15 (53.6%)	0.178
Diabetes	31 (50.8%)	13 (41.9%)	18 (58.1%)	**0.027**
Cardiac Failure	23 (37.7%)	7 (30.4%)	16 (69.6%)	**0.002**
Anterior	40 (65.6%)	18 (45%)	22 (55%)	0.20
Inferior	21 (34.4%)	16 (76.1%)	5 (23.9%)	
Dyslipidaemia	26 (42.6%)	15 (57.6%)	11 (42.4%)	0.791

TABLE 3: ELECTROCARDIOGRAPHIC DATA OF THE OVERALL COHORT AND ST RESOLUTION

	WHOLE COHORT N=61	RESOLUTION GROUP n=33	NON-RESOLUTION GROUP n=28	p - value
QRS SCORE	4.57 ±2.807	3 ±1.436	6.43 ±2.911	**0.001**
STE1	21.59 ±12.106	18.55 ±10.025	25.18 ±13.485	**0.032**
STR	9.049 ±6.727	12.212 ±7.44	5.321 ±2.89	**0.020**
EJECTION FRACTION	40.98 ±7.300	45.06 ±5.172	36.18 ±6.515	**0.001**

TABLE 4: ELECTROCARDIOGRAPHIC DATA OF THE OVERALL COHORT AND QRS SCORE

	WHOLE COHORT N=61	QRS <4	QRS >4	p – value
STE1	21.59 ±12.106	18.18 ±9.712	25.89 ±13.566	**0.012**
STR	9.049 ±6.727	11.12 ±7.64	6.296 ±4.140	**0.003**
EJECTION FRACTION	40.98 ±7.300	44.44 ±5.106	36.63 ±7.386	**0.001**

A total of 61 patients were enrolled in the study. The baseline characteristics of the whole group and the two study groups in relation to STsegment resolution are shown in Table 1 and in relation to QRS score are shown in Table 2.

The mean age of the whole group is 56.07 ±11.845 with 43 being male(70%) and 18 (30%) being female and older individuals are seen more in the non-resolution group than the resolution group ($p < 0.05$) as shown in table 1 and QRS score is high in the older age ($p < 0.05$) which shows as the age

advances the chance of non-resolution which indirectly means the chances of reperfusion decreases. Sex ($p = 0.202$, $p = 0.842$), Smoking (0.117, 0.839), Alcohol (0.105, 0.839), Hypertension ($p=0.268$, $p=0.178$) shows no statisticalrelation with respect to ST resolution and QRS score respectively.

However, Diabetics (50% in the study) showed a significant relation to QRS score ($p = 0.027$) and had a high QRS score but they did notshow any correlation with the ST resolution ($p=0.154$). Cardiac Failure has a significant correlation with respect to QRS score ($p=0.002$) and ST resolution ($p=0.01$). The patients with cardiac failure have a high QRS score (69% of patients) and there was ST non resolution in 74% of patients. The patients withthe type of MI or Dyslipidemia did not have any significance in this study.

Table 3 shows the comparison of QRS score and ST resolution. QRS score was lower in resolution group (3 ±1.436) than the non-resolution group (6.43 ±2.91) and is statistically significant($p=0.001$). The STE1 was also less inthe resolution (18.55 ±10.025) than non-resolution group (25.18 ±13.485). STR was less in the non-resolution group ($p<0.05$). Ejection fraction are also less in non-resolution group than resolution group. There was a significant negative correlation between the QRS score and STR with $r = - 0.37$ as shown in Table 4.There was also a negative correlation between Ejection fraction and QRS with ($p=0.01$).

Table – 5 EFFECT OF SMOKING ON QRS SCORE AND STSEGMENT RESOLUTION

	QRS <4	QRS >4	p-value
SMOKER	54.1%	45.9%	0.842
NON-SMOKER	56.8%	43.2%	

TABLE 6 EFFECT OF SMOKING ON ST SEGMENTRESOLUTION

	RESOLUTION GROUP	NON RESOLUTION GROUP	p-value
SMOKER	41.7%	58.3%	0.117
NON-SMOKER	62.2%	37.8%	

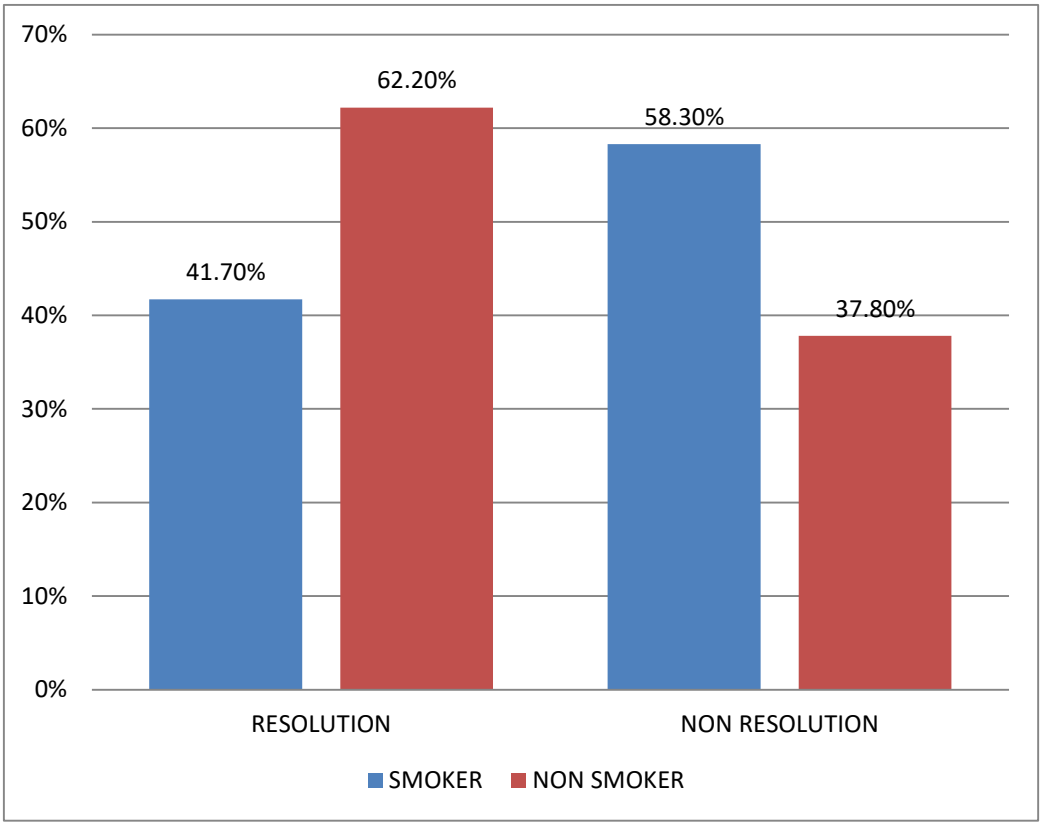


Table – 7 EFFECT OF ALCOHOL ON QRS SCORE

	QRS <4	QRS >4	p-value
ALCOHOLIC	57.1%	42.9%	0.839
NON-ALCOHOLIC	54.5%	45.5%	

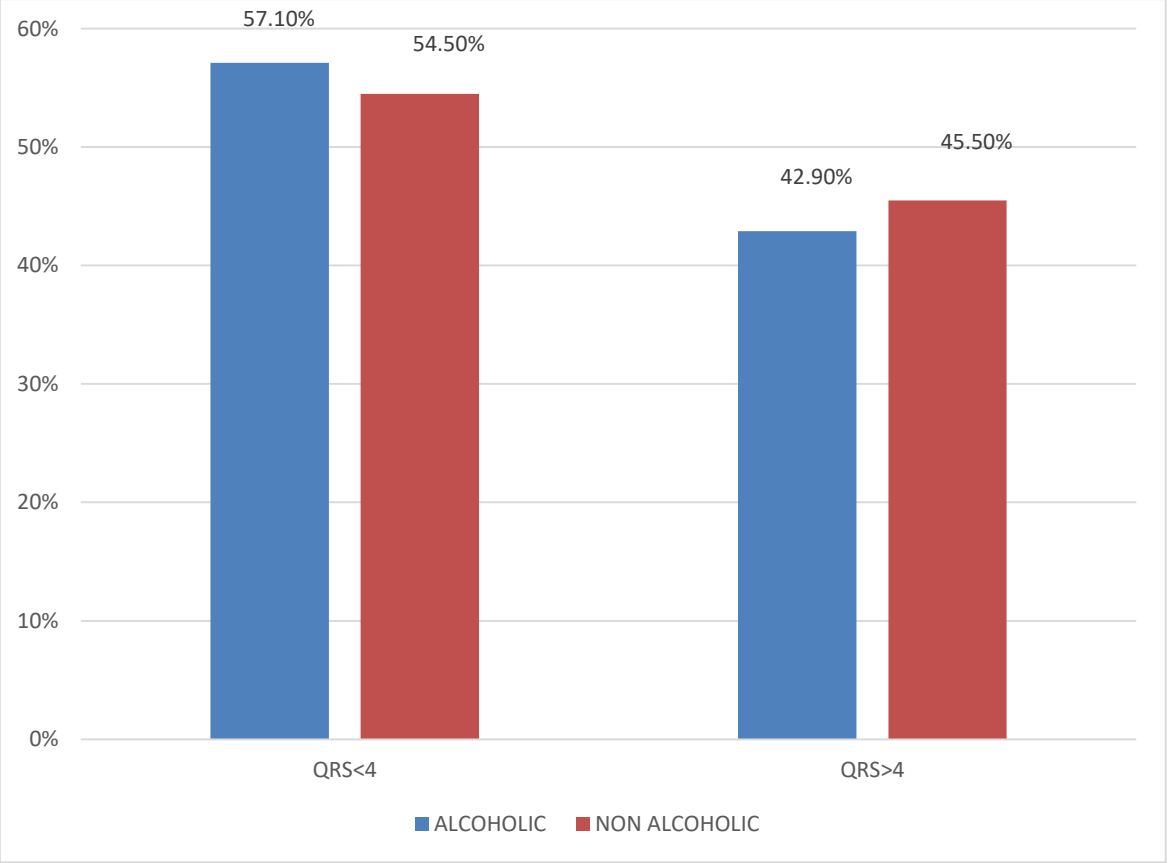


Table – 8 EFFECT OF ALCOHOL ON ST SEGMENT RESOLUTION

	RESOLUTION GROUP	NON RESOLUTION GROUP	p-value
ALCOHOLIC	57.1%	42.9%	0.117
NON-ALCOHOLIC	36.4%	63.6%	

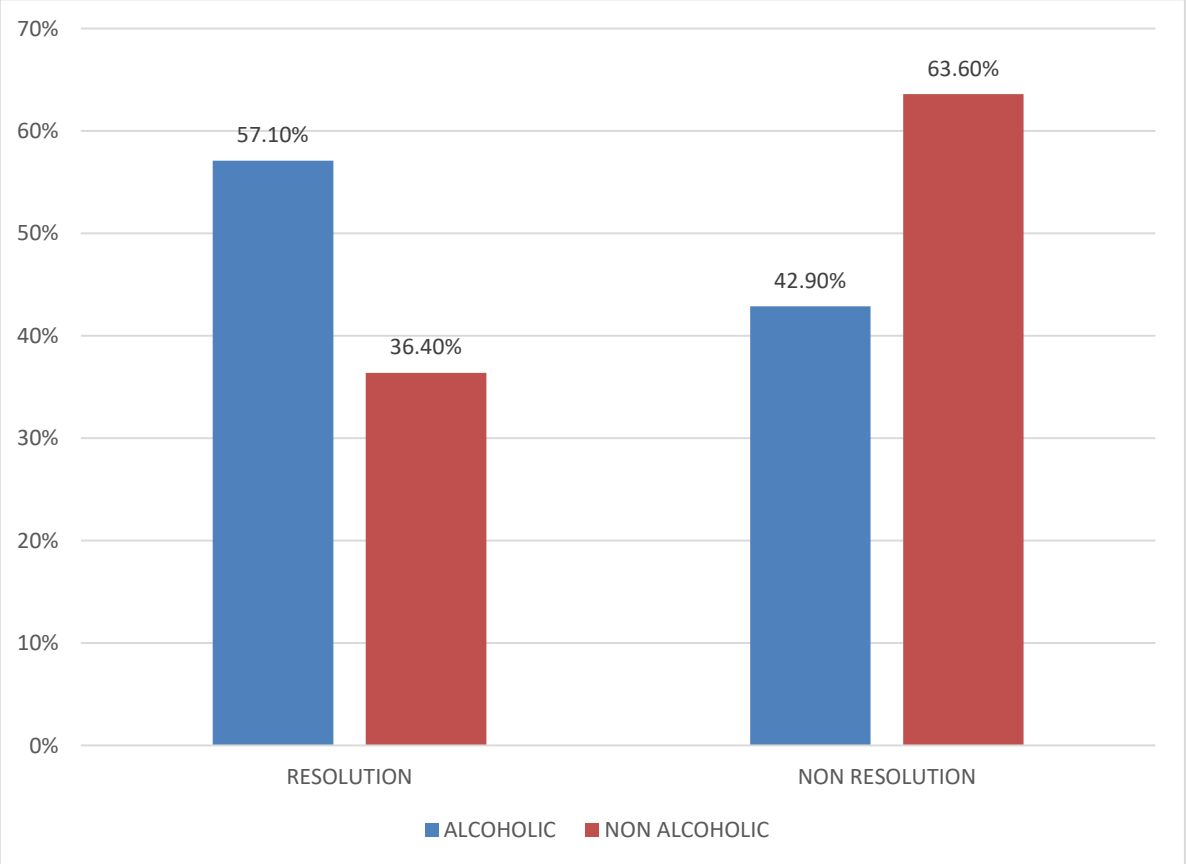


Table – 9 Effect of hypertension on QRS score

	QRS <4	QRS >4	p-value
HYPERTENSIVES	46.4%	53.6%	0.178
NON- HYPERTENSIVES	63.6%	36.4%	

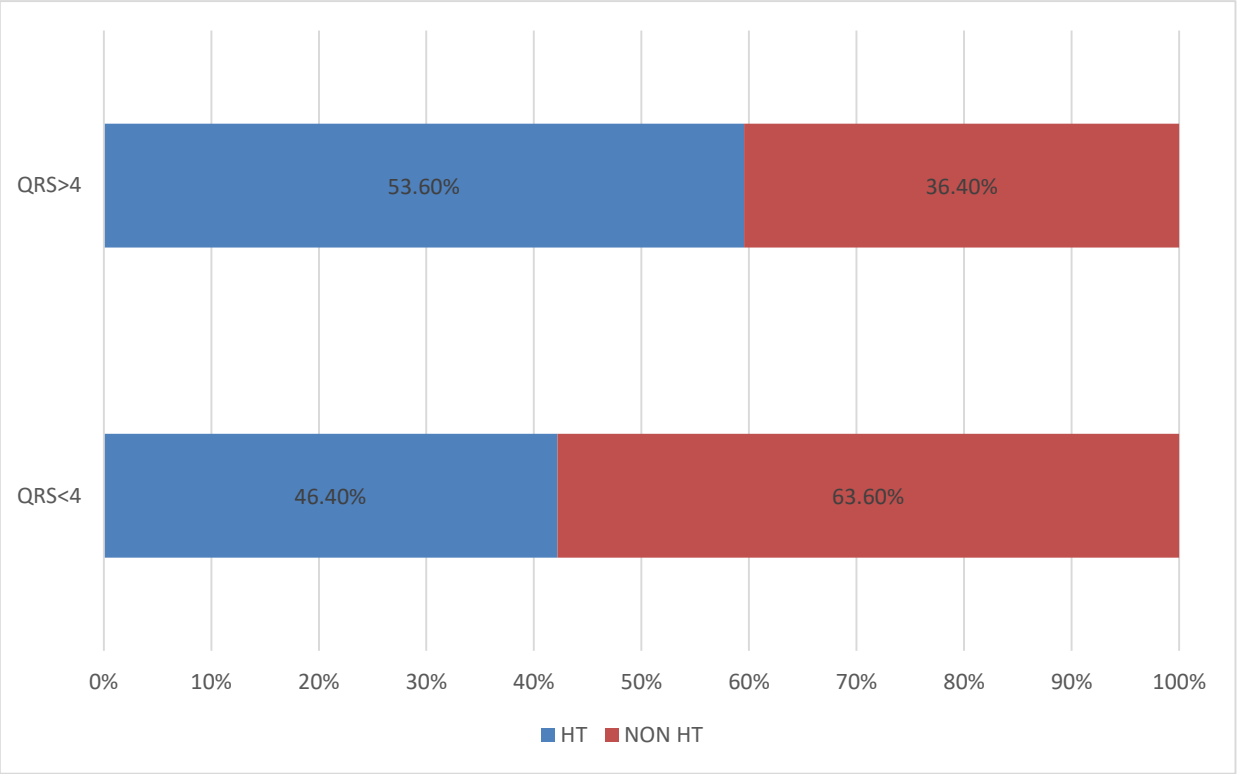


Table - 10 Effect of hypertension on ST SEGMENT RESOLUTION

	RESOLUTION GROUP	NON-RESOLUTION GROUP	p-value
HYPERTENSIVES	46.4%	53.6%	0.268
NON-HYPERTENSIVES	60.6%	39.4%	

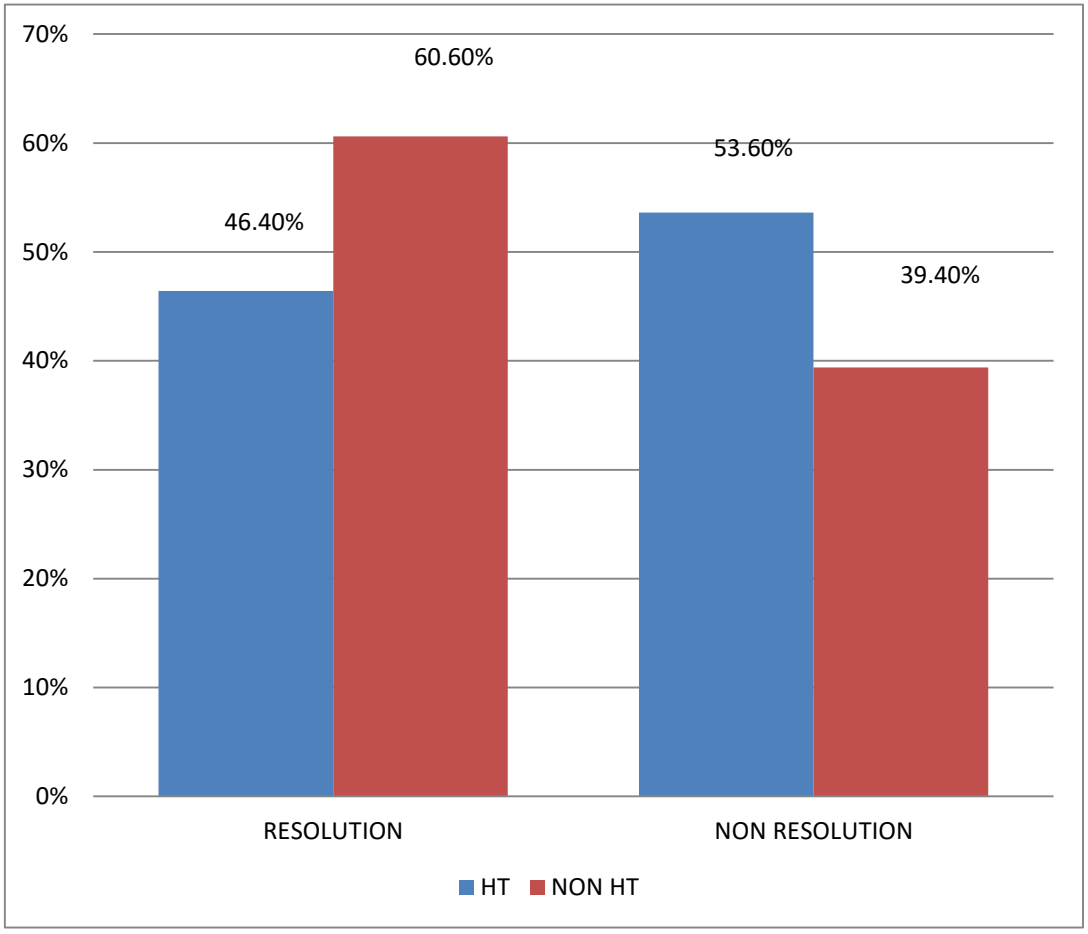


Table – 11 Effect of Diabetes on QRS score

	QRS <4	QRS >4	p-value
DIABETIC	41.9%	58.1%	0.027*
NON DIABETIC	70%	30%	

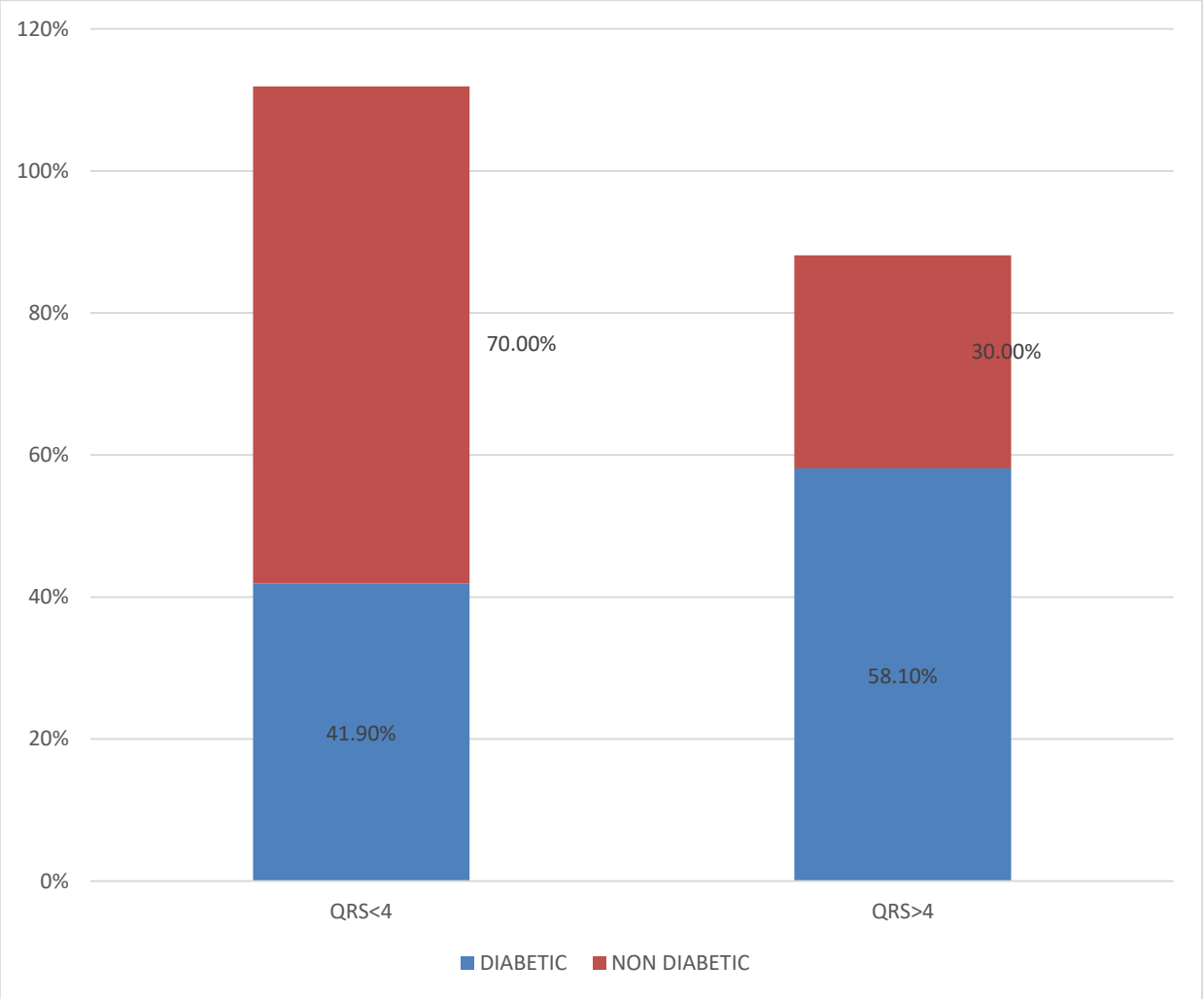


Table – 12 Effect of Diabetes on ST segment resolution

	RESOLUTION GROUP	NON-RESOLUTION GROUP	p-value
DIABETIC	45.2%	54.8%	0.154
NON-DIABETIC	63.3%	36.7%	

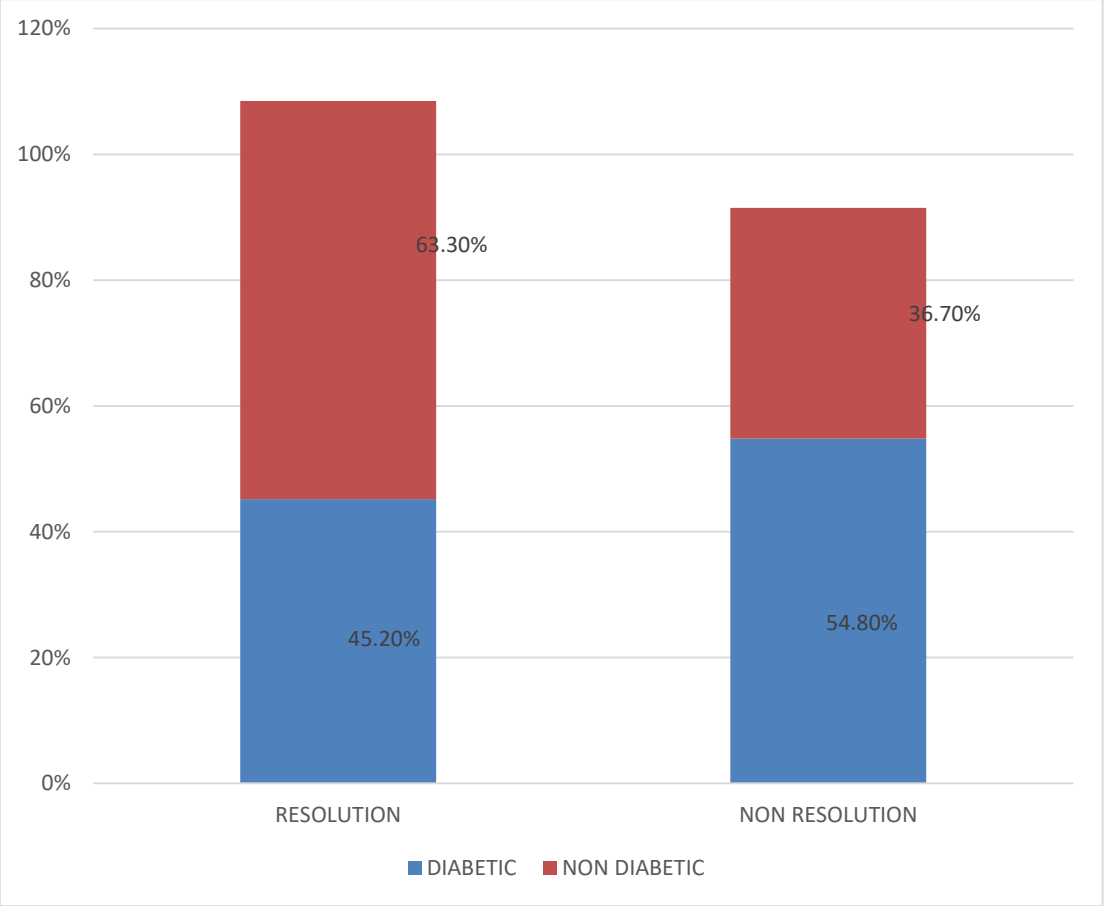


Table – 13 EFFECT OF CCF ON QRS score

	QRS <4	QRS >4	p-value
CCF	30.4%	69.6%	0.002*
NO CCF	71%	29%	

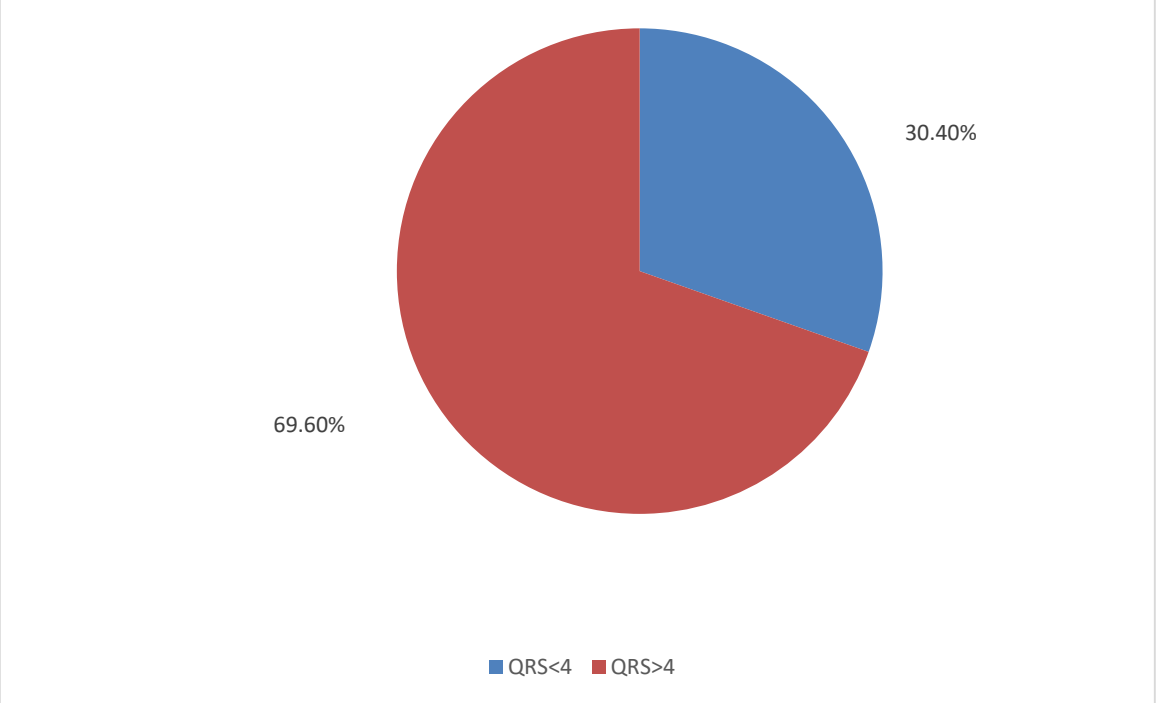


Table – 14 EFFECT OF CCF ON ST segment resolution

	RESOLUTION GROUP	NON-RESOLUTION GROUP	p-value
CCF	26.1%	73.9%	0.001*
NO CCF	71%	29%	

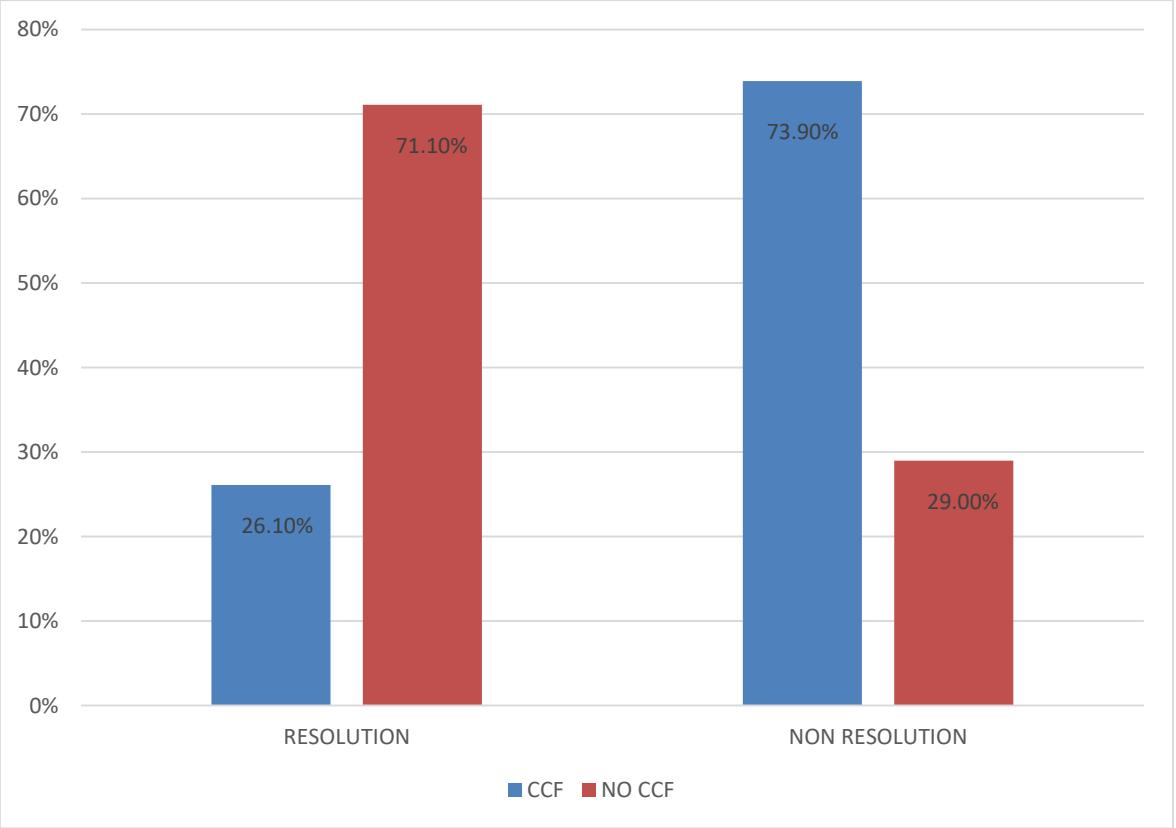


Table – 15 Effect of type of MI on QRS SCORE

	QRS <4	QRS >4	p-value
ANTERIOR	45%	55%	0.02*
INFERIOR	76.2%	23.8%	

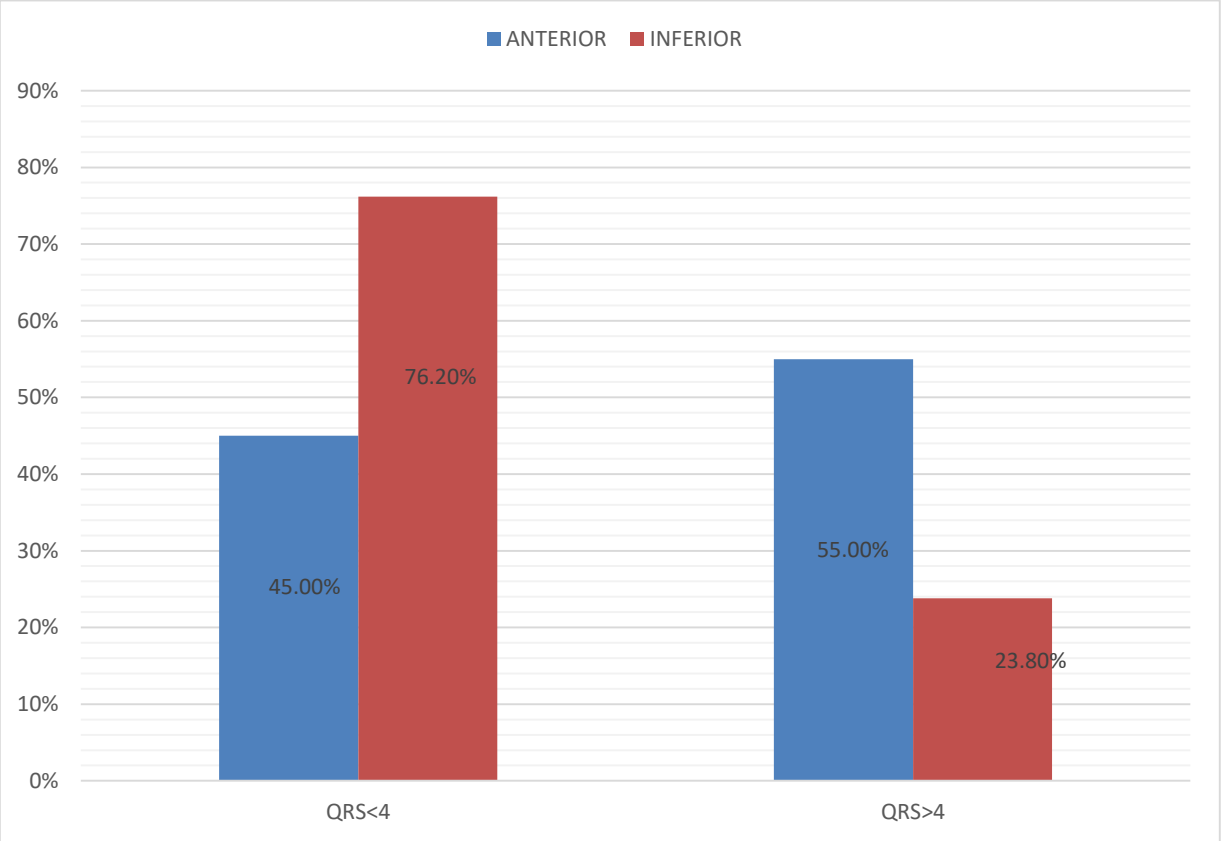


Table – 16 Effect of type of MI on ST segment resolution

	RESOLUTION GROUP	NON-RESOLUTION GROUP	p-value
ANTERIOR	42.5%	57.5%	0.012*
INFERIOR	76.2%	23.8%	

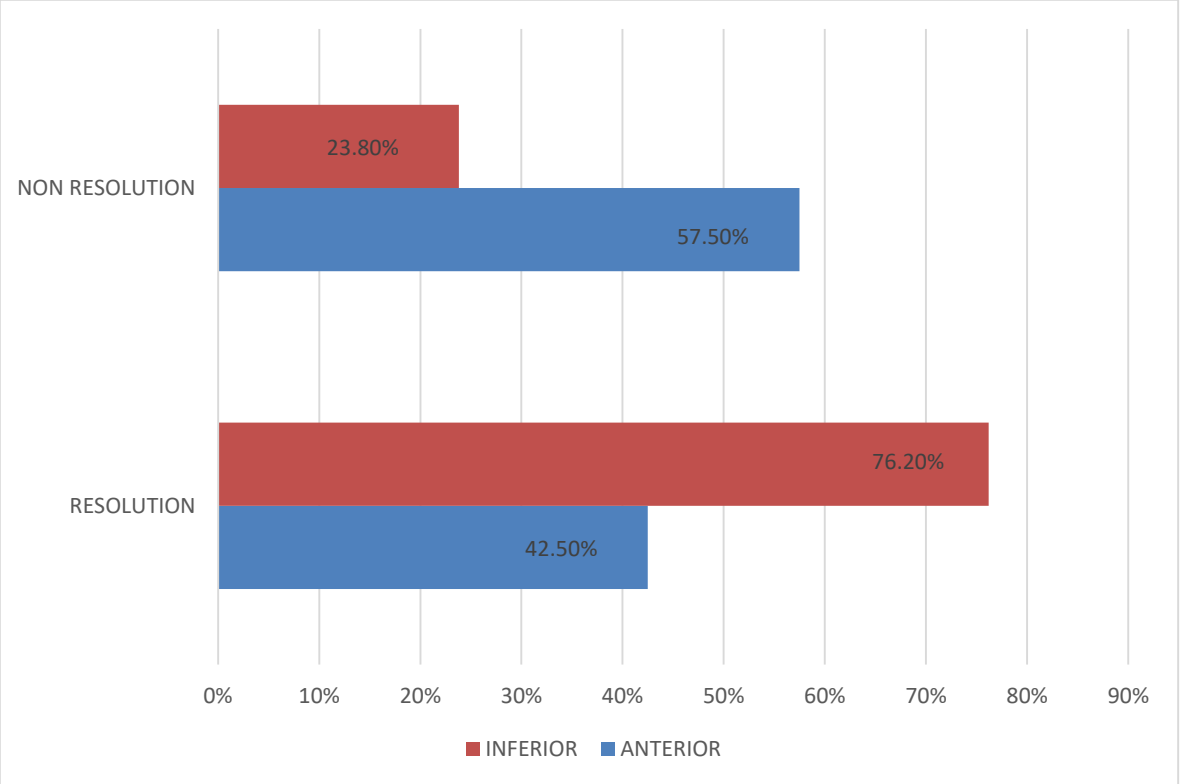


Table – 17 Effect of Dyslipidaemia on QRS score

	QRS <4	QRS >4	p-value
DYSLIPIDEMIA	57.7%	42.3%	0.791
NO DYSLIPIDEMIA	54.3%	45.7%	

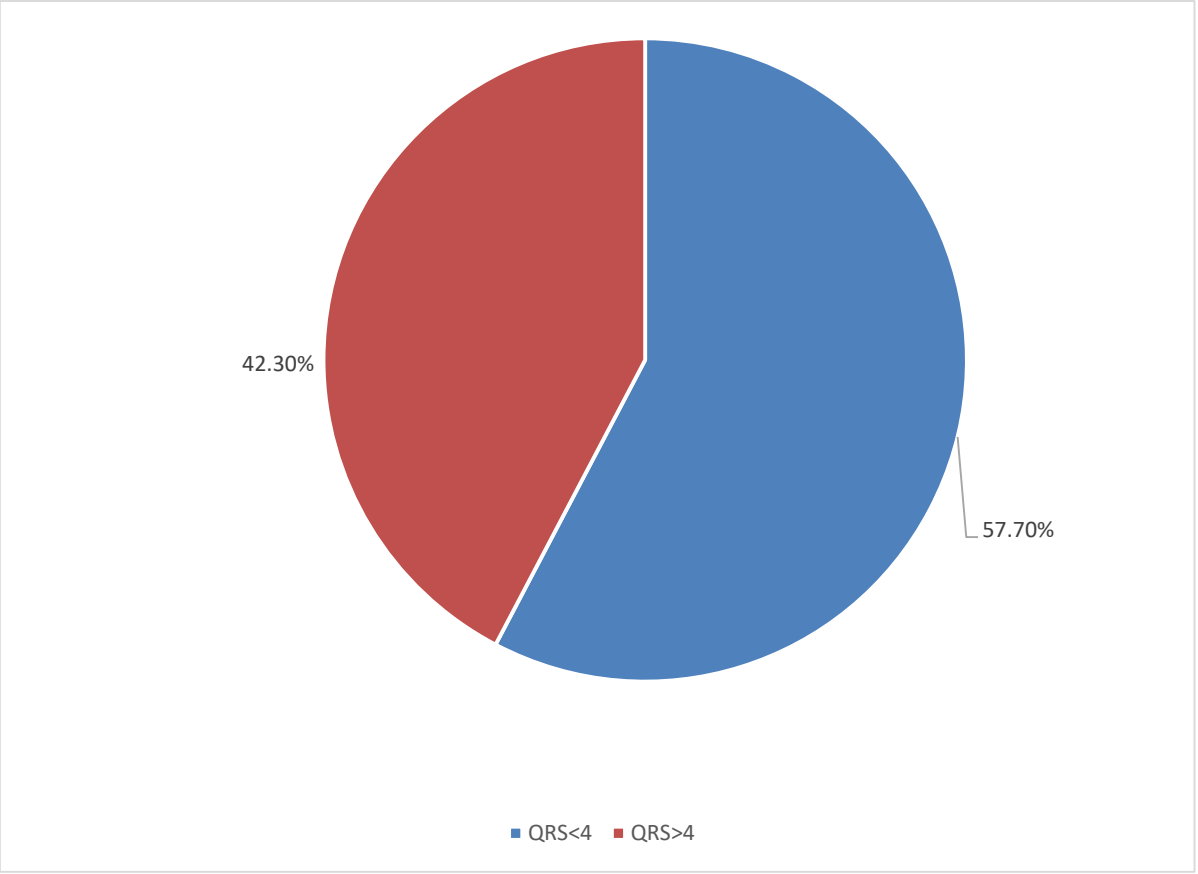
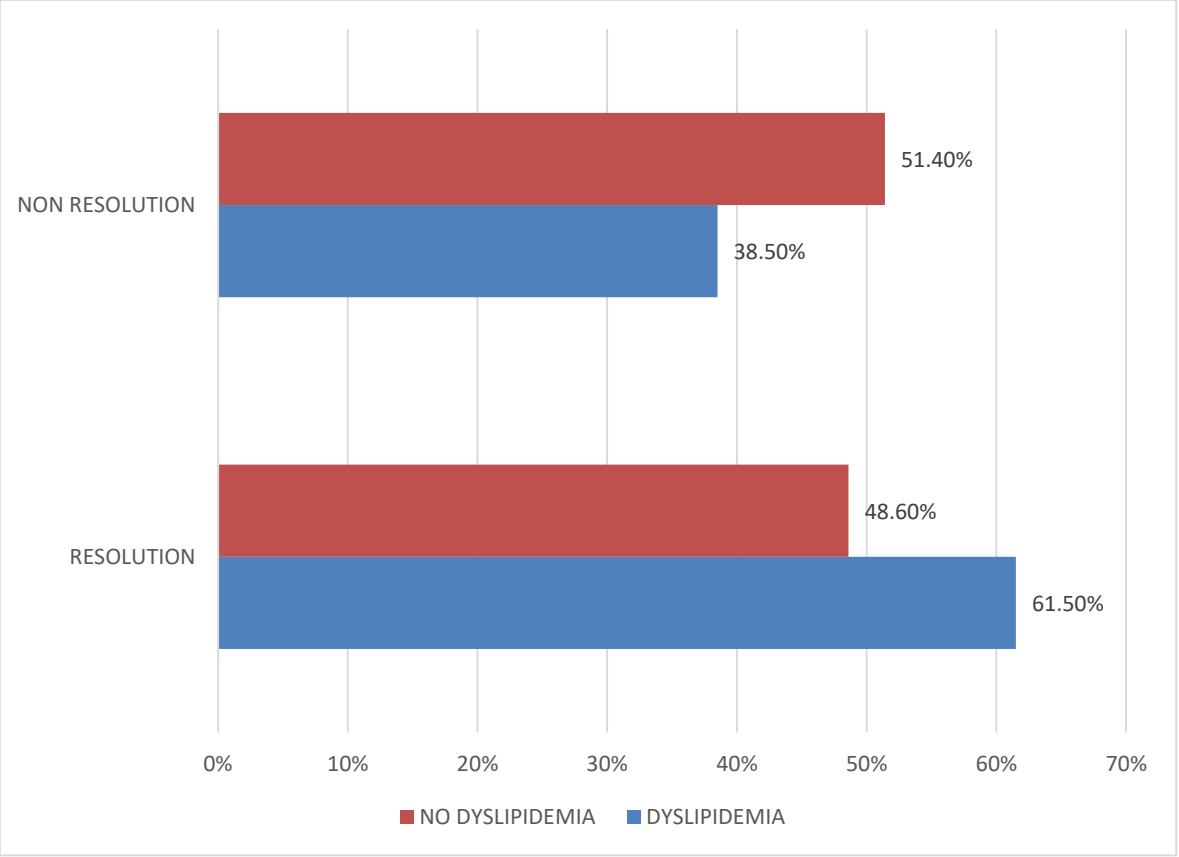


Table – 18 Effect of Dyslipidaemia on ST segment resolution

	RESOLUTION GROUP	NON-RESOLUTION GROUP	p-value
DYSLIPIDEMIA	61.5%	38.5%	0.315
NO DYSLIPIDEMIA	48.6%	51.4%	



DISCUSSION:

Patients who arrive at our hospital diagnosed with acute MyocardialInfarction are treated more commonly with thrombolytic agents. The thrombolytics used in the treatment are Streptokinase (1.5 million IU), Alteplase (15 mg i.v bolus followed by 50 mg over 30 minutes followed by 35mg over 60 minutes), retepase (10 units + 10 units i.v bolus). Various other treatment options also are available for MI such as the Percutaneous Transluminal Coronary Angioplasty. Streptokinase is still having popularity due to its cost effectiveness, readily available and hence we used streptokinasewith our study. Various other studies also had proved the benefits of using streptokinase and is well accepted method of revascularisation.

Here ST segment resolution is taken as a reliable tool to predict the revascularisation after the thrombolysis. Many clinical trials have also shown that ST segment non resolution is associated with increased risk of mortality and higher risk of complications[26].In this context the presence of Q waves before reperfusion can predict the slower and incomplete resolution of ST segment. Selvester QRS score[13] was initially developed to predict the infarct size using ECG using 10 ECG leads and Modified Selvester score which is a 29-point system which is a simplified form is used in our study to quantify theQRS score in early stage of the disease to determine the effectiveness of reperfusion after treatment.

We adopted a cut off ≥ 50% of ST resolution and <50% of ST resolution as a criterion for successful reperfusion and failed reperfusion respectively. Our study demonstrates that this modified QRS scoring can reliably predict the patients who can have ST segment resolution after thrombolysis. Patients with QRS score >4 would

predict the non-resolution of ST elevation after treatment.

This is important to predict the ST resolution in order to prevent unnecessary thrombolysis in patients especially in the situation where they present beyond the window period. But sometimes even when the patients present beyond the window period there is a doubtful role of thrombolysis. Therefore, it keeps alluring to recognise patients who stand to benefit the mostwith thrombolytics using the QRS score.

The other benefit of our study is in the situation of silent Myocardial Infarction which are incidentally diagnosed in elderly, diabetic patients withoutmuch symptoms or other angina equivalents it becomes a cumbersome job to decide on the benefit of thrombolysis to achieve reperfusion. Thus, in this setting it becomes increasingly important to predict the ST resolution with the thrombolytics. QRS score gives a window of opportunity to decide whether patient could benefit from the use of thrombolytics.

Even in some situations where patient present within window period some patients especially the elderly has the risk of bleeding. So, we can analysethis risk vs benefit from the use of QRS score in predicting the effectiveness of thrombolysis. Elderly patients have a higher QRS score likewise diabetics alsohave a high QRS score showing that diabetes have a risk of failed thrombolysis. Even patients with heart failure and low Ejection Fraction also have a high risk of failed thrombolysis.

Studies[32,33]showed that the QRS score was more in patients with a high 2-year mortality after treated by PCI. The myocardial salvage achieved bythe reperfusion of

the occluded arteries by PCI is less time dependent than thrombolysis. So as time progresses the efficacy of thrombolysis decreases but this problem is not seen much with PCI[34].So it is important to decide on the efficacy of thrombolysis after treatment in the setting.

CONCLUSION

1. Higher age group has higher QRS score and has a more risk ofrisk of non-ST resolution.
2. Sex, Smoking, Alcohol, Hypertension shows no statistical relation with respect to ST resolution and QRS score.
3. Diabetics shows a statistical significance in relation to QRSscore and had a high QRS score but they did not show any correlation with the ST resolution.
4. Cardiac Failure has a significant correlation with respect to QRS score and ST resolution. The patients with cardiac failure have ahigh QRS score and there was high risk of ST non resolution.
5. The patients with the type of MI or Dyslipidemia did not have any significance in this study.
6. QRS score was lower in resolution group than the non- resolution group. There was a significant negative correlation between the QRS score and STR. Thus, a low QRS score have more chances of ST resolution (reperfusion) than high QRS scorewho have a less chance of ST resolution and have failed perfusion.
7. There was also a negative correlation between Ejection fraction and QRS score.
8. Thus, QRS score would reliably predict the ST segment resolution in patients with STEMI receiving thrombolytic therapy. QRS score >4 says the patient has higher chances of STnon resolution.

SUMMARY

This study was conducted in Government Chengalpattu medical college with study population of 61 with aim to determine whether Modified Selvester QRS score would reliably predict resolution of ST segment elevation in patients with acute Myocardial Infarction receiving thrombolytic therapy with streptokinase.

Information gathered about the patients type of MI, Fasting lipid profile, Diabetes, Hypertension. Baseline ECG was taken and QRS score was calculated with the initial ECG and 90 minutes ECG after thrombolysis was taken.STE in initial ECG taken as STE1 and 90 minutes after thrombolysis taken as STE2 and the difference between the two taken as ST segment resolution (STR). The patients are divided into resolution (STR more than or equal to 50%) and non resolution group (STR less than 50%) group. Based on the value QRS <4 predicting the resolution group and other characteristics are studied.

Our study concluded that high QRS score noted with increasing age, diabetics, cardiac failure and have a more risk of non resolution. Also a significant negative corelation observed between EF and QRS score is noted. Thus, QRS score would reliably predict the ST segment resolution in patients with STEMI receiving thrombolytic therapy. QRS score >4 says the patient has higher chances of STnon resolution.

BIBILOGRAPHY

1. Gupta R, Joshi P, Mohan V, Reddy KS, Yusuf S. Epidemiology and causation of coronary heart disease and stroke in India. 2008; 94:16–26.

2. Thygesen K, Alpert J, Jaffe A, et al. Fourth Universal Definition of Myocardial Infarction (2018). *J Am Coll Cardiol.* 2018 Oct, 72 (18) 2231–2264.

3. Fourth Universal Definition of Myocardial Infarction (2018) Kristian Thygesen, Joseph S. Alpert, Allan S. Jaffe, Bernard R. Chaitman, Jeroen J. Bax, David A. Morrow, Harvey D. White, The Executive Group of the Joint European Society of Cardiology (ESC)/American College of Cardiology (ACC)/American Heart Association (AHA)/World Heart Federation (WHF) Task Force for the Universal Definition of MyocardialInfarction

4. Sanchis-Gomar F, Perez-Quilis C, Leischik R, Lucia A. Epidemiology ofcoronary heart disease and acute coronary syndrome. Ann Transl Med. 2016 Jul;4(13):256.

5. Giugliano D, Chiodini P, Maiorino MI, Bellastella G, Esposito K. Cardiovascular outcome trials and major cardiovascular events. Does glucose matter? A systematic review with meta-analysis. J Endocrinol Invest. 2019 Oct;42(10):1165-1169.

6. Cai Q, Mukku VK, Ahmad M. Coronary artery disease in patients withchronic kidney disease: a clinical update. Curr Cardiol Rev. 2013 Nov;9(4):331-9.

7. Saleh M, Ambrose JA. Understanding myocardial infarction. *F1000Res*.

2018;7:F1000 Faculty Rev-1378. Published 2018 Sep 3.

8. Sudhindra Rao, B. S. Patil Rao Predictors Of Failed Thrombolysis In Acute Myocardial Infarction Vol 3, No5 (2012).

9. Braunwald E, Kloner R. The stunned myocardium: prolonged, postischemic ventricular dysfunction. Circulation, 66(6):1146–9, 1982.

10. Rahimtoola S. The hibernating myocardium. Am Heart J,117(1):211–21, 1989.

11. Guzy PM. Creatine phosphokinase-MB (CPK-MB) and the diagnosis of myocardial infarction. West J Med. 1977 Dec;127(6):455-60.

12. Yoshiaki Maruyama, MD, Third Department of Internal Medicine, Saitama Medical Center, Saitama Medical School. 1981 Kamodatsujimichi- machi,Kawagoe city, Saitama 350-0844, Japan New Method of EstimatingMyocardial Infarct Size UsingTechnetium-99m Pyrophosphate and Thallium-201

13. Selvester RH, Wagner GS, Hindman NB. The Selvester QRS scoring system for estimating myocardial infarct size. The development and application of the system. Arch Intern Med. 1985 Oct;145(10):1877- 81.

14. Abdel-Salam Z, Wafa S, Kamel S, Nammas W. The modified Selvester QRS score: can we predict successful ST segment resolution in patients with myocardial infarction receiving fibrinolytic therapy? Cardiol J. 2010;17(4):367-73.

15. Evaluation of a QRS scoring system for estimating myocardial infarct

size. I. Specificity and observer agreement.G S Wagner, C JFreye, S T Palmeri, S F Roark, N C Stack, R E Ideker, F E Harrell,Jr, and R H Selvester

16. Harkness JR, Sabatine MS, Braunwald E, Morrow DA, Sloan S, Wiviott SD, Giugliano RP, Antman EM, Cannon CP, Scirica BM. Extent of ST-segment resolution after fibrinolysis adds improved riskstratification to clinical risk score for ST-segment elevation myocardial infarction. Am Heart J. 2010 Jan;159(1):55-62..

17. Appleby, M.A., Michaels, A.D., Chen, M. *et al.* Importance of the TIMI frame count: implications for future trials. *Trials* **1**, 31 (2000).

18. Morrow DA, Antman EM, Parsons L, de Lemos JA, Cannon CP, Giugliano RP, McCabe CH, Barron HV, Braunwald E. Applicationof the TIMI risk score for ST-elevation MI in the National Registryof Myocardial Infarction 3. JAMA. 2001 Sep 19

19. Br Heart J. PMCID: PMC1232158 High dose intravenous streptokinase in acute myocardial infarction -short and long term prognosis. 1986 March; 55(3): 231–239.

20. Sawar S, Khan B, Khan AT, Noor N, Rahman F, Rehman N. Complications of streptokinase in patients with acute ST segment elevation myocardial infarction: No reason to withhold this effectivetherapy. J Postgrad Med Inst 2019; 33(4): 293-8.

21. Chopra A, Patted SV, Parikh M, Agarwal R, Jaishankar K, Modi N. Use of thrombolytic agents for ST-elevation myocardial infarction care in India: An

expert consensus. J Pract Cardiovasc Sci 2021; 7:182-9

22. Effectiveness of intravenous thrombolytic treatment in acute myocardial infarction. Gruppo Italiano per lo Studio della Streptochinasi nell'Infarto Miocardico (GISSI). Lancet. 1986 Feb22.

23. Intravenous streptokinase for acute myocardial infarction. Effects on global and regional systolic function. AHA ,1988; 78: 258-266

24. de Belder MA Acute myocardial infarction: failed thrombolysis *Heart* 2001;**85:**104-112

25. Teramoto T, Sasaki J, Ueshima H, Egusa G, Kinoshita M, Shimamoto K, Daida H, Biro S, Hirobe K, Funahashi T, Yokote K, Yokode M; Japan Atherosclerosis Society (JAS) Committee for Epidemiology and Clinical Management of Atherosclerosis. Diagnostic criteria for dyslipidemia. Executive summary of Japan Atherosclerosis Society (JAS) guideline for diagnosis and preventionof atherosclerotic cardiovascular diseases for Japanese. Thrombolysis. 2007 Aug;14(4):155-8.

26. Wagner GS, Freye CJ, Palmeri ST et al. Evaluation of a QRS scoring system for estimating myocardial infarct size. Specificity and observer agreement. Circulation, 1982; 65: 342–347. 9. De Lemos J

27. Bhatia L, Clesham GJ, Turner DR. Clinical implications of ST-segment non-resolution after thrombolysis for myocardial infarction.J R Soc Med. 2004 Dec;97(12):566-70.

28. Mechanic OJ, Gavin M, Grossman SA. Acute Myocardial Infarction. [Updated 2022 Aug 8]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls

Publishing; 2022

29. Sreeniwas Kumar A, Sinha N. Cardiovascular disease in India: A 360 degree overview. *Med J Armed Forces India*. 2020;76(1):1-3. doi:10.1016/j.mjafi.2019.12.005

30. Huffman MD, Prabhakaran D, Osmond C, et al. Incidence of cardiovascular risk factors in an Indian urban cohort results from theNew Delhi birth cohort. *J Am Coll Cardiology*. 2011;57(17):1765-1774. doi:10.1016/j.jacc.2010.09.08

31. Strauss DG, Selvester RH. The QRS complex-a biomarker that ”images” the heart: QRS scores to quantify myocardial scar in thepresence of normal and abnormal ventricular conduction. J Electrocardiography, 2009; 42: 85–96.

32. Liu, Qiana,,b; Zhang, Yonga; Zhang, Pengqiangb; Zhang, Junbob; Cao, Xiaojiaob; He, Shanshanc; Yang, Donghuib. Both baseline Selvester QRS score and change in QRS score can predict prognosis in patients with acute ST-segment elevation myocardial infarction afterpercutaneous coronary intervention. Coronary Artery Disease: August 2020 - Volume 31 - Issue 5 - p 403-410.

33. Uyarel H, Cam N, Okmen E et al. Level of Selvester QRS score is predictive of ST-segment resolution and 30-day outcomes in patientswith acute myocardial infarction undergoing primary coronary intervention. Am Heart J, 2006; 151: 1239.e1–1239.e7.

34. Schömig A, Ndrepepa G, Mehilli J et al. Therapy-dependent influence of time-to-treatment interval on myocardial salvage in patients with acute

myocardial infarction treated with coronary arterystenting or thrombolysis. Circulation, 2003; 108: 1084.

STUDY PROFORMA:

Name and age:

Sex:

Occupation:

Presenting complaints: Chest pain - Duration

Breathlessness NYHA class Other complaints

Past History: Type 2 DM / Hypertension/ CAD

Personal History: Smoker / Alcoholic

Family History of IHD:

General Examination: Pallor/ Pedal oedemaHR: BP:

CVS examination: Presence of S3 Gallop/Basal Crepitations

Type of MI:

Anterior:Anterolateral /Anteroseptal

Inferior: Inferior wall

Infero posterior

KILLIP CLASS: 1/ 2/3/4

Presentation time after symptoms: in hours.

Laboratory investigations: Haemoglobin:

Lipid profile:

RBS:

FBS /PPBS (if diabetic):

STE – 1:

STE - 2:

STR: > 50% Resolution (Resolution group) / < 50% Resolution (Non Resolution group)

QRS Score:

EF %:

Lead	**Duration (msec)**		**Amplitude ratios**		**Max points**
I	Q≥30	(1)	R/Q≤1	(1)	2
II	Q≥40 Q≥30	(2) (1)			2
aVL	Q≥30	(1)	R/Q≤1	(1)	2
aVF	Q≥50 Q≥40 Q≥30	(3) (2) (1)	R/Q≤1 R/Q≤2	(2) (1)	5
V1	Any Q R≥50 R≥40	(3) (2) (1)	 R/S≥1	 (1)	4
V2	AnyQ orR≤20 R≥60 R≥50	(3) (2) (1)	R/S≥1.5	(1)	4
V3	Any Q orR ≤ 30	(1)			1
V4	Q≥20	(1)	R/QorR/S≤0.5 R/Q or R/S ≤1	(2) (1)	3
V5	Q≥ 30	(1)	R/QorR/S≤1 R/Q or R/S ≤2	(2) (1)	3
V6	Q≥ 30	(1)	R/QorR/S≤1 R/Q or R/S ≤3	(2) (1)	3

STE 1:

STE 2:

STR: Resolution group/Non resolution group

QRS score:

INFORMED CONSENT FORM

Title of the Study : **" MODIFIED SELVESTER QRS SCORE IN PREDICTING SUCCESSFUL RESOLUTION OF ST SEGMENT ELEVATION IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION RECIEVING THROMBOLYTIC THERAPY WITH STREPTOKINASE "**

Name of the Participant :

_____.

Name of the Principal (Co-

Investigator):_____.

Name of the Institution: Chengalpattu Medical college and Hospital.

Name and address of the sponsor / agency (ies) (If any) :

_____

_____

____.

Documentation of the informed consent

I _____have read the information in this form (or it has been read to me). I was free to ask any questions and they have been answered. I am over 18 years of age and, exercising my free power of choice, hereby give my consent to be included as a participant in **"MODIFIED SELVESTER QRS SCORE IN PREDICTING SUCCESSFUL RESOLUTION OF ST SEGMENT ELEVATION IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION RECIEVING THROMBOLYTIC THERAPY WITH STREPTOKINASE".**(title of the study).

1. I have read and understood this consent form and the information provided to me.
2. I have had the consent document explained to me.
3. I have been explained about the nature of the study.
4. I have been explained about my rights and responsibilities by the investigator.
5. I have been informed the investigator of all the treatments I am taking or have taken in the past _____ months including any native (alternative) treatment.
6. I have been advised about the risks associated with my participation in this study.*
7. I agree to cooperate with the investigator and I will inform him/her immediately if I suffer unusual symptoms.*
8. I have not participated in any research study within the past _____month(s).*
9. I have not donated blood within the past _____months----add if the study involves extensive blood sampling.*
10. I am aware of the fact that I can opt out of the study at any time without having to give any reason and this will not affect my future treatment in this hospital.*
11. I am also aware that the investigator may terminate my participation in the study at any time, for any reason, without my consent.
12. I hereby give permission to the investigators to release the information obtained from me as result of participation in this study to the sponsors, regulatory authorities, Govt. agencies, and IEC. I understand that they are publicly presented.
13. I have understand that my identity will be kept confidential if my data are publicly presented .
14. I have had my questions answered to my satisfaction.
15. I have decided to be in the research study.

I am aware that if I have any question during this study, I should contact the investigator. By signing this consent form I attest that the information given in this

document has been clearly explained to me and understood by me, I will be given a copy of this consent document.

signature of the investigator :

signature of the participant :

சுயஒப்புதல் படிவம்
ஆய்வுசெய்யப்படும் தலைப்பு : "கடுமையான மாரடைப்பு நோயாளிகலில் ஸ்ட்ரெப்டோகினேஸுடன் த்ரோம்போலிடிக் சிகிச்சையைப் பெறுதல் நோயாளிகளுக்கு ஸ்டி பிரிவு உயரம் குறைப்பதை கணிப்பதில் மாற்றியமைக்கப்பட்ட செல்வெஸ்டர் மதிப்பெண்ணின் பங்கு."

பங்குபெறுபவரின் வயது:
பங்குபெறுபவரின் எண் :

மேலேகுறிப்பிட்டுள்ள மருத்துவஆய்வின் விவரங்கள் எனக்கு விளக்கபட்டுள்ளது. நான் இவ்வாய்வில் தன்னிச்சையாக பங்கேற்கின்றேன். எந்தகாரணத்தினாலோ, எந்த சட்டசிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகிக்கொள்ளலாம் என்றும் அறிந்துகொண்டேன். இந்த ஆய்வு சம்பந்தமாகவோ, இதைசார்ந்து மேலும் ஆய்வுமேற்கொள்ளும்போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர், என்னுடைய மருத்துவஅறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவை இல்லை என அறிந்துகொள்கிறேன். இந்த ஆய்வின் மூலம் கிடைக்கும் தகவலையோ,முடிவையோ பயன்படுத்திக் கொள்ள மறுக்கமாட்டேன். இந்த ஆய்வில் பங்குகொள்ள ஒப்புக்கொள்கிறேன். இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்று உறுதியளிக்கிறேன்.

பங்கேற்பவரின் கையொப்பம்:

சாட்சியாளரின் கையொப்பம்:

இடம்:
தேதி:

பங்கேற்பவரின் பெயர் மற்றும் விலாசம்:

ஆய்வாளரின் கையொப்பம் :

இடம்: தேதி:

MASTERCHART

S.no	Name and age	TIME (Hrs)	Diabetes	Hypertension	SMOKER	Alcoholic	Ccf	dyslipidemia	Type AnTERIOR / iNFERIOR	Killip class	Ste 1	Ste 2	<50 %	>50 %	QRS SCORE	EF %
1.	Geetha kumari 44/f	4	S	S	N	N	S	S	Anterior	2	52	21		S	2	37
2	Rani 45/f	6	N	S	N	N	S	S	Inferior	1	21	6		S	3	35
3	Kokila 55/f	1	S	N	N	N	N	N	Anterior	1	73	45	S		7	47
4	Murugan 62/m	3	S	S	S	S	N	N	Anterior	1	7	4	S		6	49
5	Krishnan 44/m	3	S	S	S	S	S	N	Inferior	1	15	6	S		4	42
6	Sagunthala 65/f	10	S	N	N	N	S	N	Inferior	1	7	3		S	3	34
7	Ezhumalai 60/m	4	N	N	N	S	N	S	Inferior	1	6	2		S	1	50
8	Adhikesavan 50/m	5	N	N	S	S	N	S	Inferior	1	6	5	S		2	40
9	Chandran75/m	9	N	N	S	S	S	N	Anterior	1	45	32	S		7	33
10	Sagayam 60/m	6	N	N	S	S	N	S	Anterior	1	15	6		S	2	48
11	Chellammal 65/f	3	S	S	N	N	N	S	Inferior	1	5	2		S	1	45

12	Subban 75/m	12	N	N	S	S	S	N	Inferior	3	7	5	S		10	33
13	Ramu 44/m	15	S	N	N	N	S	N	Anterior	2	28	20	S		5	33
14	Kesavan 48/m	4	N	N	S	S	S	S	Anterior	2	15	11	S		3	42
15	Revathi 50/f	5	N	N	N	N	N	N	Inferior	1	15	6		S	2	45
16	Annammal 70/f	6	S	S	N	N	N	S	Anterior	1	18	7		S	2	49
17	Ravi 47/m	4	N	S	N	N	N	S	Inferior	1	14	6		S	5	55
18	Lakshmi 56/f	7	S	S	N	N	S	N	Anterior	2	35	13		S	6	36
19	Kanambal67/ f	8	S	S	N	N	S	S	Inferior	1	12	5		S	3	33
20	Kasi 53/m	9	N	S	S	S	S	N	Anterior	1	34	30	S		7	34
21	Mani 46/m	2	S	S	S	S	N	N	Anterior	1	32	12		S	3	44
22	Babyammal 68/f	4	N	S	N	N	N	N	Anterior	1	23	19	S		2	50
23	Dhanam 68/f	8	S	N	N	N	N	N	anterior	1	32	24	S		7	32
24	velmurugan 59/m	10	N	N	N	N	N	S	anterior	1	41	15		S	2	45
25	Muniyammal 72/f	13	S	S	N	N	S	N	Anterior	2	42	33	S		8	26

26	Kanagammal 61/f	9	N	S	N	N	N	S	Anterior	1	24	10		S	5	42
27	Tamilarasi 57/f	12	S	S	N	N	N	S	Inferior	1	15	5		S	3	45
28	Selvi 69/f	6	N	S	N	N	N	N	Inferior	1	14	10	S		2	33
29	Tamilarasan 49/m	3	N	N	S	S	N	S	Anterior	1	28	11		S	2	45
30	Kannan 48m	14	N	N	N	S	N	S	Anterior	1	26	17	S		7	33
31	Thanigachalam 65/m	5	S	S	N	N	S	N	Anterior	2	28	19	S		10	28
32	Lakshmi 63/f	10	S	S	N	N	S	S	Anterior	2	24	15	S		7	32
33	Parthian 32/m	2	N	N	S	N	N	S	Inferior	1	10	3		S	2	50
34	Muthiah 47/m	7	S	N	S	S	N	N	Anterior	1	13	5		S	3	48
35	Joseph 60/m	10	S	S	N	S	S	N	Anterior	2	27	19	S		10	32
36	Baskar 49/m	5	N	S	N	S	N	N	Anterior	1	21	12		S	3	48
37	Michael 36/m	3	N	N	S	S	N	N	Inferior	1	15	5		S	3	52
38	kanthasamay 48/m	8	S	N	S	S	S	S	Anterior	2	30	23	S		9	32
39	Mannar 50/m	12	S	N	N	S	N	S	Anterior	1	23	15	S		2	48

40	kannabiran 45/m	10	S	S	S	S	S	N	Anterior	2	28	20	S		10	33
41	raja 67/m	12	S	N	N	N	N	S	Inferior	1	15	10		S	6	45
42	Sivaraman 65/m	10	N	S	S	N	S	N	Anterior	2	28	21	S		12	33
43	Ibrahim 52/m	9	N	N	N	N	N	N	Inferior	1	9	4		S	6	45
44	Santhanam 60/m	4	S	N	N	N	N	N	Anterior	1	22	10		S	3	48
45	Xavier 55/m	7	N	N	S	N	N	N	Inferior	1	11	4		S	2	45
46	Kanniyappan 72/m	5	S	S	N	N	N	N	Anterior	1	23	10		S	3	45
47	Balaji 62/m	6	S	N	N	S	S	S	Anterior	1	18	14	S		6	34
48	Varadarajan 44/m	7	N	N	N	S	N	N	Inferior	1	10	3		S	1	50
49	Rajalakshmi 40/f	4	N	N	N	N	N	N	Anterior	1	15	6		S	2	45
50	Muniyappan 76/m	8	S	N	N	N	S	N	Inferior	I	9	6	S		6	30
51	Raja 54/m	16	N	N	S	S.	N	S	Anterior	1	21	6		S	3	46
52	Kalimuthu 62/m	5	S	N	S	S	N	N	Anterior	1	23	13	S		8	42
53	Rajagopal 35/m	3	S	N	N	S	S	N	Anterior	1	17	6		S	2	48

54	Manigandan 63/m	13	N	S	S	S	S	S	Anterior	2	30	18	S		5	33
55	kannabiran 68/m	14	N	S	N	N	N	N	Anterior	1	28	19	S		2	42
56	kalimuthu 78/m	8	S	S	S	N	N	S	Anterior	1	19	10	S		6	34
57	Ilavarasan 45/m	12	N	N	S	S	N	N	Inferior	1	15	5		S	3	41
58	Nagarani 50/f	6	S	S	N	N	N	S	Anterior	1	21	12		S	6	50
59	Rajan 32/m	4	N	N	S	N	N	N	Inferior	1	15	4		S	3	45
60	Siva 78/m	12	S	S	S	S	S	S	Anterior	2	23	17	S		10	33
61	Pandiyan 39/m	5	N	N	N	S	N	N	Anterior	1	19	8		S	3	48

CCF – Congestive Cardiac Failure l HTN – Hypertension

Sex: M- Male, F- Female

EF – Ejection Fraction

ABBREVIATIONS:

NSTEMI - NON ST ELEVATION MYOCARDIAL INFARCTION

SIHD - STABLE ISCHEMIC HEART DIASEASE

UA - UNSTABLE ANGINA

STEMI - ST ELEVATION MYOCARDIAL INFARCTION

STE 1 - SUM OF ST SEGMENT ELEVATION IN THE FIRST ECG

STE 2 - SUM OF ST SEGMENT ELEVATION IN THE 90 MINS ECG

STR - DIFFERENCE BETWEEN STE 1 AND STE 2

SK - STREPTOKINASE

GUSTO - GLOBAL UTILISATION OF STREPTOKINASE AND TISSUE PLASMINOGEN ACTIVATOR FOR OCCLUDED CORONARY ARTERIES

IRIS - INTERNATIONAL STUDY OF INFARCT SURVIVAL COLLABORATIONGROUP

T PA - TISSUE PLASMINOGEN ACTIVATOR

GISSI - GRUPPO ITALINOPER SO STUDIO DELLA SOPRAVVIENZANCIL INFARCTO

CAD - CORONARY ARTERY DISEASE

TMT - TREADMILL TEST.

PCI - PERCUTANEOUS TRANSLUMINAL CORONARY ANGIOPLASTY

ECG - ELECTROCARDIOGRAPHY

CABG - CORONARY ARTERY BYPASS GRAFT