A STUDY OF COMPARISON OF QT DISPERSION IN ACUTE MYOCARDIAL INFARCTION BETWEEN EARLY REPERFUSION AND LATE REPERFUSION THERAPY

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M.D. (GENERAL MEDICINE) - BRANCH – I

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BONAFIDE CERTIFICATE

This is to certify that **"A Study of Comparison of QT dispersion in acute myocardial infarction between early reperfusion and late reperfusion therapy"** is a bonafide work performed by **Dr KARTHIK. N.**, post graduate student, Department of Internal Medicine, Kilpauk Medical College, Chennai-10, under my guidance and supervision in fulfilment 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 May 2010 to April 2013.

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DECLARATION

I solemnly declare that this dissertation **"A study of Comparison of QT dispersion in acute myocardial infarction between early reperfusion and late reperfusion therapy"** was prepared by me at Government Kilpauk Medical College and Hospital, Chennai, under the guidance and supervision of **Dr. G. Balan M.D.**, Professor and Unit Chief, Department of Internal Medicine, Government Kilpauk Medical College and Hospital, Chennai.

This dissertation is submitted to **The Tamil Nadu Dr. M.G.R. Medical University, Chennai** in partial fulfilment of the University regulations for the award of the degree of **M.D. Branch I (General Medicine)**.

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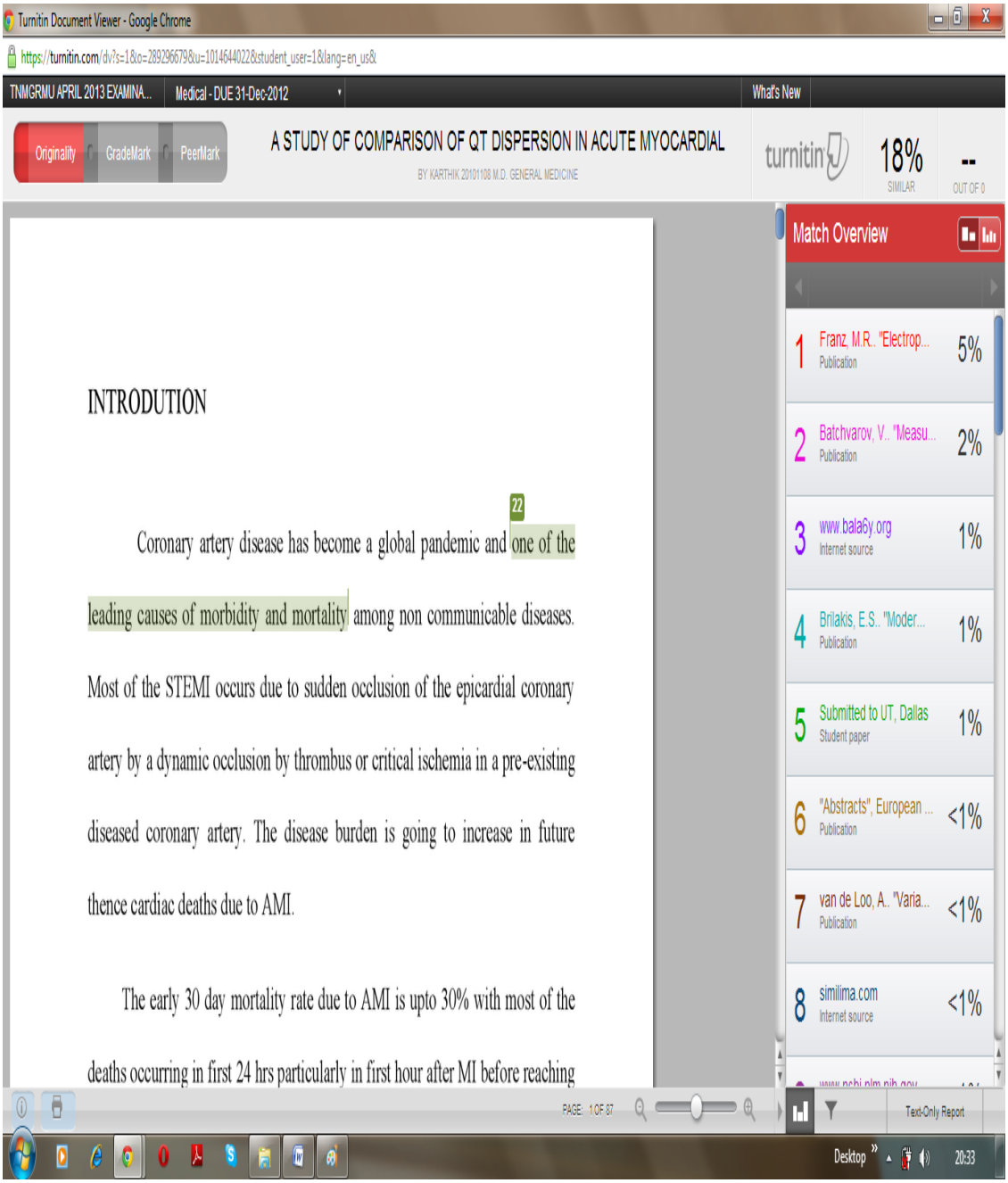
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Coronary artery disease has become a global pandemic and one of the leading causes of morbidity and mortality among non communicable diseases. Most of the STEMI occurs due to sudden occlusion of the epicardial coronary artery by a dynamic occlusion by thrombus or critical ischemia in a pre-existing diseased coronary artery. The disease burden is going to increase in future thence cardiac deaths due to AMI.
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CONTENTS

A STUDY OF COMPARISON OF QT DISPERSION IN ACUTE MYOCARDIAL INFARCTION BETWEEN EARLY REPERFUSION AND LATE REPERFUSION THERAPY

ABSTRACT:

INTRODUCTION: Coronary artery disease has become a global pandemic and one of the leading causes of morbidity and mortality among non communicable diseases. The complications leading to death in acute myocardial infarction such as malignant ventricular arrhythmias are very much preventable. QT dispersion (maximum QT interval minus minimum QT interval) was proposed as an index of the spatial dispersion of ventricular recovery. QTd measurement is an attempt by which we can distinguish homogenous myocardium from inhomogenous myocardium. Hence QT dispersion provides a cheap, simple and non invasive method to measure underlying dispersion of ventricular excitability.

MATERIALS AMD METHODS : This is an observational study conducted in Government Kilpauk medical college hospital, Department of medicine for a period of 1 year. A total of 30 early thrombolysed cases and 30 late thrombolysed cases were recruited for the study. QT dispersion was studied before thrombolysis, after thrombolysis , 2nd day , 5th day and 6th week post thrombolysis in both groups and later compared between both groups. Similarly

QTd was compared between treatment successful and failed cases. The obtained data was tabulated, statistical analysis done and co-relation was studied.

RESULTS AND CONCLUSION:

1. QTd was higher among those who were thrombolysed late as compared to early group.

2. QTd was lower in those with successful thrombolysis.

3. Incidence of arrythmias was high among late group than early indicating early successful thrombolysis reduces the occurrence of arrythmias.

INTRODUTION

Coronary artery disease has become a global pandemic and one of the leading causes of morbidity and mortality among non communicable diseases. Most of the STEMI occurs due to sudden occlusion of the epicardial coronary artery by a dynamic occlusion by thrombus or critical ischemia in a pre-existing diseased coronary artery. The disease burden is going to increase in future thence cardiac deaths due to AMI.

The early 30 day mortality rate due to AMI is upto 30% with most of the deaths occurring in first 24 hrs particularly in first hour after MI before reaching hospital. Most of these deaths are increasingly occurring among the young during the productive period of life [(1)].

The complications leading to death in acute myocardial infarction such as malignant ventricular arrhythmias (like ventricular tachycardia and ventricular fibrillation) are very much preventable [(2)].

Despite the sobering statistics in the occurrence of AMI and its complications there is a decline in the deaths in the early hours after MI due to the good treatment.

The use of sophisticated battery of tests, like continuous Holter Monitoring, Microvolt T wave alternans, Domain ventricular late potentials are not available to most of the people [3].

QT dispersion (maximum QT interval minus minimum QT interval) was proposed as an index of the spatial dispersion of ventricular recovery. QTd measurement is an attempt by which we can distinguish homogenous myocardium from inhomogenous myocardium. In other words increased QT dispersion reflects the disparity of ventricular recovery times. Hence QT dispersion provides a cheap, simple and non invasive method to measure underlying dispersion of ventricular excitability [6].

There is an absolute need for the development of affordable parameters to detect and identify the risk of development of ventricular arrhythmias associated with acute myocardial infarction and to prevent sudden cardiac deaths associated with it [4, 5].

AIMS AND OBJECTIVES

1. To study QT dispersion in acute myocardial infarction and its comparison after thrombolysis between early and late reperfusion therapy.
2. To compare QT dispersion between successful thrombolysis and failed thrombolysis.

REVIEW OF LITERATURE

HISTORICAL REVIEW

Coronary artery disease and myocardial infarction

There are evidences of human sufferings from coronary artery disease from the time of civilizations. Ancient Egyptian, Roman, Greeks and Biblical literature have clear evidences[7]. It was Hippocrates who first said sudden cardiac deaths could be due to disease of the heart.

In 1768, classical angina pectoris was described by Heberden. In1809, imbalance between myocardial oxygen supply and need was postulated by Burns. Hammer was the first one to demonstrate antemortem coronary thrombosis in 1878.

In early 19 century Osler said heart continues to function even after infarction. Later studies by Obrastzow and Straschesko in 1912 showed AMI is not always fatal and is usually due to thrombosis and can be diagnosed before death [8]. In 1945 Radner visualised coronary arteries and in 1948 anticoagulant therapy was used. The use of enzymes to diagnose myocardial infarction was first demonstrated by Ladue. In 1962, Mason performed first coronary angiography. Later Effler and Favoloro started doing endarterectomy and grafting.

It was only recently that the full understanding of the development of cascade of events leading to the complete coronary artery occlusion by fibrin platelet thrombus postulated and the reperfusion in the form of fibrinolysis or PCI started.

Historical aspects of ECG and QT dispersion

Augustus recorded first electrocardiogram by using capillary Electrometer[9]. Later William Einthoven developed string galvanometer and was awarded noble prize in 1924 for his work on electrocardiogram and published reports done with it. Later Mackenzie and Lewis proposed the advantages of electrocardiograph in diagnosing arrhythmias[10]. In 1928 Parkinson and Bedford[11] showed the evolutionary electrocardiographic changes after infarction.

In 1957, Jerwell and Lange Neilson described sudden cardiac death associated with long QT interval [12]. In 1963, Romono and Ward described a similar condition with ventricular arrhythmias [13]. Zabel et al [14] hypothesized that QT dispersion is an indirect measure of heterogenecity of ventricular repolarisation. My et al [15] said QT interval dispersion is a marker of susceptibility of ventricular arrhythmias.

Bazett showed QT interval is influenced by heart rate and it is related to the ventricular systole. This has led to the development of formula which corrects QT interval for heart rate, corrected QT interval [16].

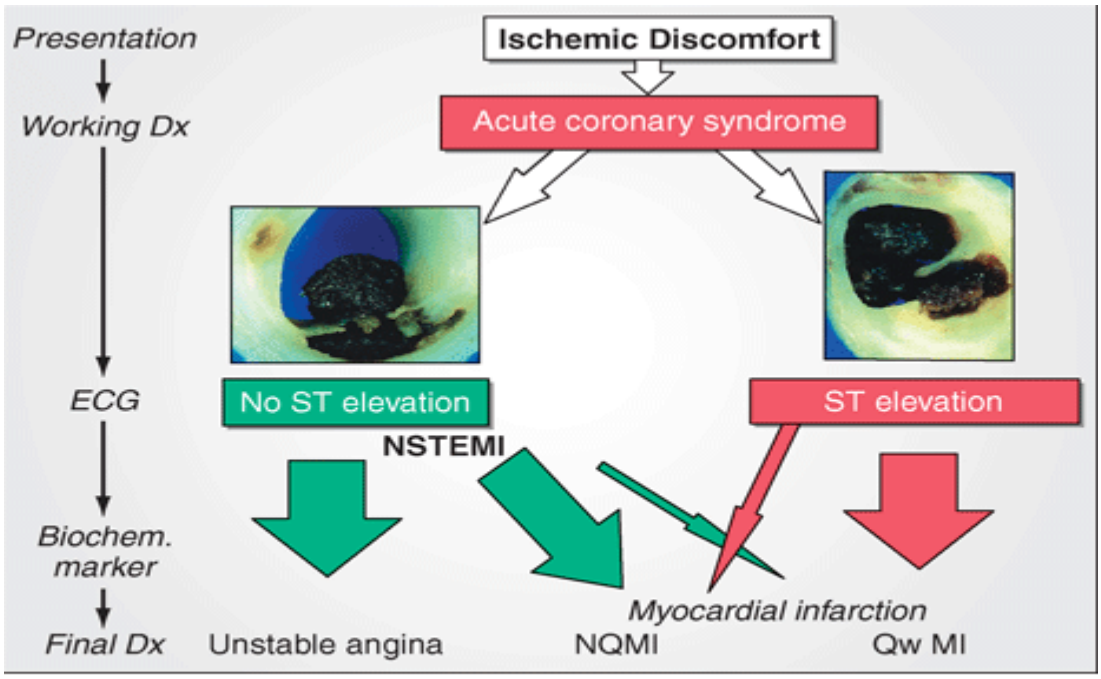
Acute myocardial infarction

Acute myocardial infarction is a leading killer disease globally and is one of the most common diagnoses in hospitalised patients in both developed and developing countries. In United States 650,000 new AMI and 450,000 recurrent AMI occur each year [17]. It is an indirect indicator of society's development. Acute MI is caused when there is sudden and total occlusion of epicardial coronary artery leading to myocardial necrosis. Myocardial infarction occurs when ischemia exceeds the critical threshold for a considerable duration, far beyond myocardial cellular repair mechanisms designed to maintain normal function.

This critical ischemia results when there is an interruption of supply of oxygen and nutrients due to the occlusion of artery by thrombus over an ulcerated, fissured atherosclerotic plaque [18]. This sudden cessation of blood flow to coronary artery leads to cessation of aerobic metabolism, depletion of creatinine phosphate and onset of anaerobic glycolysis sets in. Irreversible injury starts occurring after 20 minutes in the absence of significant collaterals [19].

Hibernating myocardium is a late consequence of ischemia after re-establishing normal coronary arterial flow, which protects the heart from subsequent ischemic episodes resulting in chronic contractile dysfunction. Myocardial stunning is a regional depression in myocardial contractility after resolution of ischemia in the absence of tissue necrosis. This may take considerable time to recover leading to reversibly dysfunctional myocardium occurring in the setting of ACS [20, 21].

Myocardial infarction is classified based on the anatomical, regional and diagnostical clinical. Morphologically or anatomically it is divided into non-transmural and transmural types. A transmural MI is one in which the full thickness of the myocardial segment(s) extending from endocardium to epicardium through myocardium is affected. A non-transmural MI is where the ischemic necrosis does not involve full thickness of myocardial segment(s), limiting itself to the endocardium, myocardium or epicardium. It is the endocardial and subendocardial region that is least perfused. Earlier classification of AMI based on ECG as Q wave and non Q wave MI was based on the presence of Q waves on ECG. However the presence or absence of Q waves on ECG did not distinguish transmural AMI from non-transmural AMI [25].



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Now universal definition of myocardial infarction is stated and accordingly MI should be used in the setting of myocardial necrosis consistent clinically with symptoms of AMI. Accordingly AMI is classified into

- Type 1 Spontaneous MI in the setting of primary coronary event (eg. Plaque rupture, fissuring, ulceration or dissection).
- Type 2 MI in the setting of mismatch between supply and demand (anaemia, coronary spasm, arrhythmias, hypotension)
- Type 3 Sudden unexpected cardiac death, including cardiac arrest with symptoms suggestive of AMI and ECG features of new ST

elevation and new onset left bundle branch block or definite new thrombus by coronary angiography.

- Type 4a MI associated with Percutaneous coronary intervention.
- Type 4b MI associated with post stent thrombosis.
- Type 5 MI associated with CABG [24].

Myocardial infarction can also be classified into STEMI and NSTEMI based on the presence or absence of ST segment elevation respectively on surface 12 lead ECG, however both represent a continuum of the same spectrum of ACS. The management protocol also differs for STEMI and NSTEMI.

Prevalence

Coronary artery disease has become a leading cause of mortality worldwide in most industrialised nations and increasing even more in developing countries [22]. By 2020 WHO estimates 20 million CVD deaths an year and by 2030 it is expected to increase to 24 million. It is estimated that 650,000 new cases of MI and 450,000 recurrent MI occur each year in United States alone. However, the mortality caused by MI also has improved considerably due to better management.

The incidence of MI is increasing due to an aging society coupled with a sedentary lifestyle.

RISK FACTORS

Smoking

Tobacco consumption in any form is an important risk for the development of CVD for both sexes in any age and a preventable cause of death [23]. A strong dose- response relation exists between cigarette smoking and CHD, the risk increases two to three fold in active smokers and increases by 20 to 30 % and interacts with other multiple risk factors [26]. Oxidative stress plays the central pivotal role by decreasing nitric oxide synthesis in endothelial cells. Other mechanisms are nicotine and carbonmonoxide induced direct damage to endothelial cells, reduces HDL – C, increased platelet aggregation and fibrinogen levels, vascular reactivity and hypertension.

Hypertension

High B.P confers silent cardiovascular risk, many studies have clearly showed a strong, positive and linear relation between CVD risk with both systolic and diastolic blood pressure [27]. Wide pulse pressure which reflects arterial stiffness is an independent risk factor in predicting new and recurrent MI [28]. Isolated systolic hypertension due to stiff artery and hypertension is associated with metabolic syndrome. Various mechanisms by which hypertension has its effects are increased myocardial wall stress and oxygen requirement, endothelial dysfunction, endothelial permeability

to lipoproteins, triggering acute plaque rupture and finally but not the least increased oxidative stress.

Diabetes mellitus

Hyperglycemia, insulin resistance and diabetes mellitus are major cardiovascular risk factors. Diabetes mellitus, both type 1 and type 2 is independent risk factors for CVD [29, 30]. Hyperglycemia produces accelerated atherosclerosis even before the development of frank diabetes mellitus. Diabetes mellitus patients have two to eight fold increase in cardiovascular risk which makes upto two thirds of death in such patients. Diabetes abolishes the protection offered in a premenopausal woman and equals and even doubles the CVD risk [32, 33]. Diabetes in association with metabolic syndrome is an added CVD risk. Potential mechanisms by which it acts are low HDL-c, high oxidised LDL-c and small LDL, high TGL, increased platelet aggregability, increased hsCRP and PAI-I, enhanced glycoprotein oxidation leads to accelerated atherosclerosis. Tight glycemic control reduces the microvascular complications and exercise increases the insulin sensitivity thus decreases cardiovascular mortality.

Dyslipidemia

Elevated total cholesterol, LDL-c, high triglycerides and a low HDL-c below 40 mg/dl increase the risk of myocardial infarction. Increased LDL-

c sustains and accelerates atherosclerosis; many genetic diseases with elevated LDL-c have increased CVD risk and premature onset of atherosclerosis [34]. In most studies, therapy with statins not only slows down but also regress the atherosclerotic lesions of coronary artery [35, 36]. Low HDL is becoming an increasing problem in CHD due to sedentary life style; there are studies showing increase in HDL-c cholesterol with the use of nicotinic acid and a reduction of CVD risk due to the same.

Gender

Incidence and prevalence of coronary risk is increased in men when compared with Pre-menopausal woman. In post menopausal women the margin narrows. Oestrogen therapy increases the HDL-c and there by decreases the coronary risk. Estrogen decreases LDL-c, total cholesterol and has a favourable effect on fibrinogen, homocystine, plasma viscosity etc.

Family history

History of premature cardiac death in a family increases the individual's risk of CHD. The exact cause is multi-factorial, genetic and acquired habits like smoking, high fatty diet, shared environments, monogenic factors. Onset of CHD in a male relative at or below 55 years and in female relative at or below 65 years is a documented risk. The

predictive value becomes stronger with increase in number of relatives having premature coronary events [37, 38].

Physical inactivity

Physical inactivity bears its direct effect on CHD and stroke, doubles the risk. Physical activity has shown to bring in an improvement and even regression in plaque size, as demonstrated by angiogram. Moderate physical activity has been well documented in reducing the coronary events by improving myocardial oxygen requirement and electrical stability, increasing insulin sensitivity and decreasing its requirement, reducing obesity, increases HDL-c, reduces blood pressure and increasing fibrinolysis.

Obesity

Obesity is an independent and a major risk factor for coronary artery disease according to AHA [39]. Obesity accelerates the rate of atherosclerosis and progression of coronary artery disease. It increases the risk of diabetes, insulin resistance, high B.P and leads to elevated level of inflammation, dyslipidemia, decreased level of HDL-c, increases platelet aggregability and LVH.

Additional emerging risk factors

These are mainly markers of inflammation and atherosclerosis

Fibrinogen

Fibrinogen is a marker of atherogenesis, helps in sustaining atherosclerosis by increasing blood viscosity and increasing platelet aggregability. Higher levels of fibrinogen are seen in smokers, obese individual, those on OCPs and thence increase CVD [40, 41].

C- reactive protein

CRP is considered as the novel marker of inflammation, and increased CRP levels have well co-related with the full spectrum of CHD [24]. hsCRP has come as a novel marker in assessing and monitoring CHD. CRP is an acute phase reactant produced in liver and seen to increase in diabetes, hypertension, obesity, dyslipidemia, metabolic syndrome, chronic inflammations and infections [42].

PATHOGENESIS

Acute myocardial infarction is due to the sudden abrupt occlusion of coronary artery by a thrombus complicating artery previously affected by atherosclerosis which results in cessation of blood flow completely. If the cessation of blood flow persists more than 20 minutes irreversible injury of the myocardium sets in. However when the occlusion is a slow process STEMI does not occur because of rich collaterals.

The development of atherosclerotic plaque starts and sustains from decades before the development of MI; it is said that the process of atherogenesis starts from the intrauterine period. The site of atherosclerosis usually will be at specific arterial sites such as bifurcations, branches, curvatures, which increases turbulence and decrease shear stress which make the endothelium prone to injury. In a majority of cases thrombosis occurs at the site where there is thinning of fibrous cap usually at the shoulder region of the lipid rich core plaque. Fibrous cap thinning occurs due to action of proteases, elastases, metalloprotienases, collagenases, increased smooth muscle cell death and decreased matrix production [43, 44]. After the formation of initial platelet monolayer, many agonists like ADP, epinephrine, serotonin cause platelet aggregation and release of thromboxane A2 causes vasoconstriction. The presence of glycoprotein IIb/IIIa causes platelet cross linking.

Myocardial cell death occurs first in the most distal area away from the site of occlusion usually the endocardium or subendocardial layer of myocardium; later if the occlusion persists cell death extends to the myocardium and to epicardium. If blood flow is re-established in the occluded coronary artery most of the affected myocardium can be salvaged.

The damage caused depends upon distinct factors

1. The territory supplied by the affected vessel
2. The duration of occlusion.
3. Whether or not it is completely occluded.
4. The presence of collaterals to the affected area. 5. Oxygen demand in the affected area.
5. Endogenous factors that produces early lysis .
6. The adequacy of myocardial perfusion in the infarct territory after flow is re-established in an occluded artery.

Less commonly STEMI occurs due to hypercoagulable conditions, cocaine abuse, connective tissue disorders, and embolism originating from cardiac chambers [(45)].

Signs and symptoms

Myocardial infarction presents with typical clinical features, with sometimes symptoms varying from a simple epigastric pain to sudden cardiac death, particularly in diabetes, where asymptomatic MI is commonly seen due to autonomic neuropathy. Severity of AMI cannot be assessed clinically based on symptoms. Despite this varied clinical presentation of AMI there are some characteristic symptoms as such as -

chest pain – crushing, squeezing, heaviness over the chest, tight band around the chest.

- Radiation of pain to both shoulder, arm, jaws, or back
- Shortness of breath
- Epigastic discomfort
- Excessive sweating or diaphoresis
- Nausea, vomiting
- Syncope or presyncope, palpitations
- Altered sensorium and shock

AMI can occur at any time but usually seen to cluster around early morning or within a few hours after awakening due to the circadian variations in the vascular tone. Many a times it is associated with precipitating factors like anxiety, emotional stress, and physical exertion or during hospitalisation due to various illnesses.

On examination patient may be tossing over the bed in order to relieve the pain, cold and clammy skin, cool peripheries, tachycardia, soft s1, presence of s3, s4 (indicates ventricular dysfunction), transient midsystolic or late systolic apical systolic murmur (indicating mitral valvular apparatus dysfunction). About one third to half of inferior wall MI patients have bradycardia and hypotension due to parasympathetic

overactivity. Temperature may be minimally raised on the first day due to release of cytokines and carotids pulse may be weak owing to reduced stroke volume which regains over time. After some time, pericardial rub may be heard.

In evaluating any patient with chest pain one should thoroughly assess the severity of complaints, aim to identify potential precipitating factors (accelerated hypertension) and evidence of complications like hypotension, mitral regurgitation, congestive cardiac failure [47]. Diagnosis of AMI can be suspected in a patient who has classical symptoms and has the risk factors for it but a definitive diagnosis is possible only after confirmatory tests. These tests are electrocardiography, cardiac enzyme study, echocardiography.

Electrocardiography

Electrocardiograph has to be taken in all patient presenting with chest pain who has risk factors preferably as rapidly as possible to evaluate, locate, and determine the prognosis in a case of ACS and to plan the modality of treatment [48]. The 12 lead serial electrocardiogram is also taken to follow the changes occurring over time. New transient or persistent ST segment abnormalities that develop during characteristic symptoms of ACS suggest acute ischemia. A normal or near normal ECG has 80-90% negative

predictive value regardless of patients symptoms [49]. A 12 lead ECG is poor in examining right ventricle, posterior wall for which one should take additional right sided leads like v3R and v4R, posterior leads like v7, v8, v9 may improve sensitivity for diagnosing right ventricle and posterior wall infarction .The 12 lead ECG is used to classify patients into 3 groups –

1. ST segment elevation or development of new onset bundle branch block (amicable for reperfusion therapy).
2. ST depression or T wave inversion.
3. Those with no ST T wave changes and are non diagnostic.

However identification of STEMI in a pre existing LBBB is difficult and one should utilise Sgarbossa criteria [49] by –

1. ST segment elevation > 1mm concordant with the QRS complex.
2. ST segment depression >1mm in leads v1, v2 and v3.
3. ST segment elevation >5mm discordant with the QRS complex.
4. Additional diagnostic changes are 1. Pathologic Q wave in lead 1, aVL, v5, v6 2. Precordial R wave regression 3. Late notching of S wave in v1 to v4 4. ST segment deviation in the same direction as major QRS deflection [50].

Leads representing their respective walls

- Anterior wall V3 – V4

- Extensive anterior V2 – V5
- Anteroseptal V1 – V3
- Anterolateral V4 – V6, I, AVL
- Lateral I, II, AVL
- Inferior II, III, AVF
- Posterior V1, V2 (Reciprocal)

But normal ECG does not rule out acute myocardial infarction.

Serum Cardiac biomarkers

Certain proteins which are present inside myocardial cell leak out into peripheral circulation due to damage to the cardiac myocyte membrane. The rate of their appearance in the circulation depends upon their intracellular location, molecular weight and local lymphatic and blood flow. Rapid bedside assays for serum cardiac biomarkers are now available which help in planning management strategy particular in non diagnostic ECG. Now the most widely used cardiac enzymes are cardiac troponin I and troponin T, and MB subtype of creatinine kinase. Troponin I and T are released within 4hrs from injury, reaches peak by 12 hrs and remain elevated upto 7 to 14 days (trop I) 8 to 21 days (trop T) [(51)]; CKMB starts rising by 4 to 6 hrs reaches peak by 12 – 24 hrs and remains elevated for 1 to 3 days. CKMB is more useful in the setting of recurrent infarction occurring within a week [(52, 53)]. Other cardiac markers like myoglobin, high fatty binding protein (H-

FABP), due to less specificity, are not used and needs further study [54]. IMA- ischemia modified albumin is one other molecule, for which further studies regarding the specificity are awaited [55]. Since these enzymes are elevated immediately and take sometime to appear in the circulation patients presenting with chest pain are treated as having AMI and then further evaluation done for more precise diagnosis [56].

Echocardiography and Imaging

Two dimensional echocardiography, which demonstrates wall motion abnormalities caused due to decreased oxygen and nutrition supply is an easy and non invasive procedure that can be used in an emergency room. One of the earliest protective mechanisms which myocardium uses is to turn off contraction process which is energy requiring, and this is seen in echo as wall motion abnormality. Although it can even detect the portion of the myocardium affected, differentiation between an old scar and a new akinesia is difficult and limits its usage. Other uses are assessment of LV function thence prognosis, presence of pericardial effusion; with the help of colour Doppler, ventricular septal defect and mitral regurgitation can also be assessed.

There are a number of imaging modalities available like radionuclide imaging techniques but cumbersome technique, lack of specificity and

sensitivity limit their usage. Myocardial perfusion imaging using technetium 99 sestamibi scan demonstrates cold spot in an affected area but it cannot differentiate between an old scar and new infarct and this limits its overall diagnostic utility.

TREATMENT

Treatment aims at salvaging myocardium to the maximum and reducing complications arising out of it .The main aim of treatment in emergency room is to allay the discomfort and to identify patients who are candidates for urgent reperfusion.

Control of discomfort

Nitrates

Sublingual nitrate 0.4mg upto three times can be given in a patient with AMI in a gap of 5 minutes; if there is further recurrence of pain with an ongoing ischemia, intravenous nitroglycerin is considered [(63)]. Nitroglycerin acts as a nitric oxide donor to the coronary artery smooth muscle and causes vasodilation thus increasing circulation and alleviates pain, but it is better avoided in those on phosphodiesterase inhibitors for a minimum period of not less than 24 hrs to prevent dangerous hypotension.

Morphine

Morphine, an opioid analgesic, is very effective in relieving pain associated with STEMI. It is given intravenously in a dose of 4 to 8 mg and repeated in doses of 2 to 4 mg in intervals of 5 to 15 mins till the pain is alleviated. It reduces anxiety, sympathetic overactivity with a consequent reduction in heart's metabolic demand, causes peripheral arterial and venous dilatation and alleviates symptoms due to pulmonary edema. Due to its vagotonic actions it has a tendency to cause transient bradycardia, hypotension which can be managed by the use of atropine.

Beta blockers

Beta blockers decrease the need for analgesics, relieve pain, reduce the infarct size and occurrence of life threatening arrhythmias. The commonly employed regimen is metoprolol 5mg intravenous injection every 2- 5 mins for a minimum of 3 doses; after the last dose, if there are no contraindications, an oral regimen of 50mg every 6 hourly is started for 48 hrs followed by 100mg 12 hourly. However one should avoid the use of beta blocker in Killip class 2 or higher due to risk of developing cardiogenic shock [57, 58].

Antiplatelets

Aspirin has shown to markedly reduce mortality and thus should be given as soon as possible in those without an allergy [59]. Aspirin irreversibly inhibits cyclooxygenase -1 and thromboxane A2 in platelets and prevents the further occurrence of clots. It should be a non enteric coated chewable aspirin for a rapid and immediate benefit; dissolved soluble preparations or sublingual administration can also be used in a dose of 150 to 325 mg. In addition, the antiplatelet agent clopidogrel in the dose of 300 mg improves outcomes in those who are conservatively managed [61]. Newer antiplatelets like prasugrel and ticagrelor can also be used particularly in those who undergo percutaneous coronary interventions.

Reperfusion

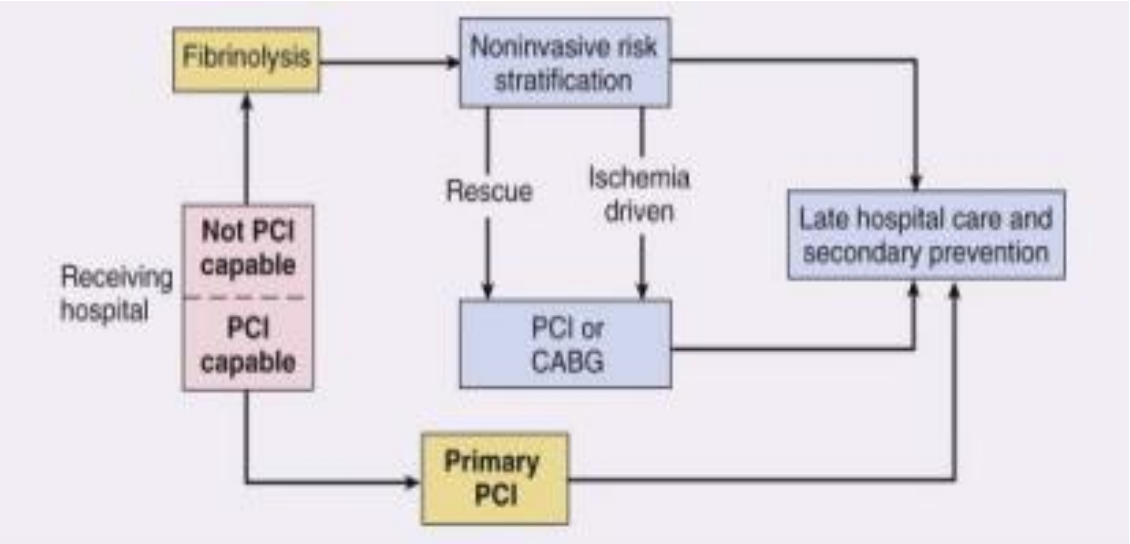
The central dogma of the treatment of AMI is reperfusion and currently we are in the era of reperfusion [62]. The main goal of treatment for the patients who presents with ECG features of STEMI and new onset LBBB is to adequately re-establish and restore blood flow to the affected ischemic myocardium. This can be achieved either by use of thrombolytics, percutaneous coronary intervention or by bypass surgery.

The patients who present with features suggestive of acute coronary syndrome are classified based on the presence or absence of ST segment

elevation and further by the presence or absence of cardiac biomarker. Initially all the patients receive the same treatment in the form of antiplatelets, sorbitrate and anticoagulation. Patients without ST segment elevation are presumed to suffer from non ST segment elevation myocardial infarction (NSTEMI) and unstable angina (UA) and are believed to have critical occlusion with diminished and preserved distal blood flow to the coronary bed. So in this scenario the main aim is to prevent progression of thrombus. After initial hemodynamic stabilisation they are offered coronary angiography and revascularisation. If the patient is hemodynamically unstable, they are taken up for immediate coronary angiography and subsequent revascularisation. Thrombolytic therapy usage is contraindicated in this subset of patients [(67)].

Percutaneous coronary intervention restores blood flow completely and achieves TIMI 4 blood flow in upto 95% as compared to 50–60 % with thrombolytics; with further passage of time, the clot becomes more and more mature and it is less amenable for lysis with thrombolytics [(64)].

In a subset of patients, restoration of flow within a critical limit prevents the development of Q waves or myocardial necrosis and is known as ‘’ aborting’’ the myocardial infarction.



COMPLICATIONS

Complications of acute myocardial infarction are mainly divided into mechanical, electrical and ischemic types. Cardiogenic shock due to severe LV dysfunction results in high number of deaths and long term morbidity secondary to recurrent hospitalisation and decreased quality of life.

Mechanical complications

The common major mechanical complications include cardiogenic shock due to LV dysfunction, right ventricular infarction and failure, acute mitral regurgitation, Ventricular septal rupture, free wall rupture, pseudoaneurysm.

1. Cardiogenic shock

It is the common complication expected in AMI, and occurs due to loss of contractile elements. Pump failure is now the primary cause of in hospital mortality [65]. Individuals who have infarctions more distally may have preserved ventricular function due to the compensation by the un-infarcted segments. Prior MI, old age, triple vessel disease, anterior wall infarction are a few risk factors [65,66] Killip and Kimball originally proposed a classification which helps in prognostification based on the hemodynamic profile of the patients, varying from no evidence of congestive cardiac failure to cardiogenic shock. They divided patients into four classes and in hospital mortality for class 1 is 0 -5% and for class 4 is 85-95%.

Killips class	Characteristics	In-Hospital mortality
Class 1	no evidence of failure	0-5 %
Class 2	Rales, raised JVP, s3	10-20 %
Class 3	Pulmonary edema	35-45 %
Class 4	Cardiogenic shock	85-95 %

The occurrence of cardiogenic shock is around 7% according and mortality is upto 80% [68].

Patients with cardiogenic shock should be put on intraaortic balloon pump temporarily to improve coronary perfusion, cardiac output and reduce

afterload. Early revascularisation procedures like percutaneous coronary intervention and coronary artery bypass grafting should be done for those who develop shock within 36 hrs following STEMI, and fibrinolytics should be given for patients who are unsuitable for invasive treatment if there are no contraindications.

Other modalities of treatment which can be considered for those who are refractory are ventricular assist devices and extracorporeal membrane oxygenator which acts as a bridge therapy for those who are awaiting transplantation. *Should we emergently revascularise occluded coronaries for cardiogenic shock (SHOCK) trial* evaluated the effect of early revascularisation and showed a decrease in all cause mortality in those who underwent early revascularisation [(65)].

2. Right ventricular infarction and failure

Involvement of right ventricle is a sequelae of inferior wall myocardial infarction as a result of proximal right coronary occlusion [(69)]. However, hemodynamically significant RV dysfunction occurs in less than 10% in RVMI.

The triad of hypotension, elevated JVP and clear lung fields in the setting of inferior wall MI, in addition to ECG findings of ST elevation in right sided leads (v3R and v4R) with RV wall hypokinesia diagnoses

RVMI. Moreover patients with RVMI may display Kussmaul's sign. Patients with RVMI have a better outcome because right ventricle is relatively resistant to ischemia and dysfunction improves over a period of 24 to 48 hours spontaneously [(69)].

Treatment for RVMI includes volume loading with normal saline in order to attain a pulmonary artery wedge pressure of 18 to 20 mm of Hg. It is always better to monitor these patients with pulmonary artery catheter because overzealous use of fluids leads to RV filling and further LV compromise thence cardiac output. If patients are not responding to these measures, one should use inotropes like dobutamine (which improves cardiac index) and right ventricle assist devices. Reperfusion preferably with primary PCI improves right ventricle function and mortality [(70)].

3. Mitral regurgitation

Mitral regurgitation is a known complication after acute myocardial infarction, with mostly mild to moderate MR occurring in 13 to 45% of patients. However severe MR due to papillary muscle rupture is life threatening and accounts for 5% of mortality in AMI [(65)]. Rupture of posteromedial papillary muscle is more common than anterolateral due to the presence of dual blood supply in the latter; the rupture may be partial or complete. Rupture of right ventricular papillary muscle is unlikely and when

it does leads to massive tricuspid regurgitation and right heart failure. Most patients have single vessel disease or small infarction.

Clinically it manifests as pulmonary edema with new onset holosystolic murmur and increasing severity of heart failure develops within 2 to 7 days after MI. 2D echocardiography demonstrates ruptured papillary muscle and colour Doppler can recognise acute mitral regurgitation which is helpful in differentiating it from ventricular septal defect.

Treatment includes prompt identification and aggressive surgical repair & is catastrophic if treated conservatively. Available surgical therapies include valve repair, replacement with prosthetic valve in association with revascularisation & they improve mortality [71].

4. Ventricular Septal rupture

Ventricular Septal rupture occurs in 1 to 3 % and accounts for 5% of mortality in AMI. VSR in apical portion is seen in anterior infarction and those in basal inferior septum are seen in inferior wall infarction. It confers a very high early mortality [72]. It varies in size, which determines the shunt and hemodynamic deterioration and chances of survival. Ventricular septal defect usually is associated with bundle branch blocks or complete heart block.

VSR is commonly seen between 3 to 7 days, with the development of holosystolic murmur better heard at the lower sternal border and invariably is associated with thrill and biventricular failure. Surgical treatment should be offered to all the patients in the form of open closures or with patch closure for small ruptures. Due to large infarct and associated multivessel disease, results are not as gratifying as mitral regurgitation [73, 74].

5. Free wall rupture

Free wall rupture occurs in 2 to 4% of patients of acute myocardial infarction and accounts for 12 to 15% of early AMI deaths [75, 76]. Free wall rupture is seen more commonly in elderly, women, diabetics and hypertensives. It usually occurs between 1 to 4 days, involving the left ventricle more commonly than right ventricle and most often involves the lateral wall. Fibrinolysis offers no benefit but decreased incidence is seen in patients who undergo PCI.

The clinical presentation varies from chest discomfort and unexplained hypotension & the outcome is usually catastrophic with imminent death

The therapeutic goal is to perform emergency open surgical repair. Inotropes and intra-aortic balloon pump can be used for initial stabilisation.

6. Pseudoaneurysm

Pseudoaneurysm develops due to incomplete ventricle rupture; more commonly a contained rupture of LV free wall occurs, where organising thrombus and pericardium form pseudoaneurysm which communicates with the ventricle via a narrow neck. Aneurysm clinically remains silent, gradually becomes bigger and at times can embolize thrombus into systemic circulation causing strokes. This condition is usually diagnosed by echocardiography.

Spontaneous rupture of pseudoaneurysm is a possibility and therefore surgical treatment should be undertaken for all patients irrespective of the size [77].

7. True ventricular aneurysm

True ventricular aneurysms are commonly seen in patients with apical infarction as compared to other areas. This is a true aneurysm because it has all the three layers and is usually formed by a scar tissue which has a wide neck.

The risk of developing aneurysm is greatest in those patients who are not reperfused in whom it is seen in upto 10 to 30 %, as compared to 5% in reperfused patients, and is seen particularly in anterior wall transmural MI

[78]. It produces dyskinesia and paradoxical expansile wall motion abnormality and there- by causing mechanical disadvantage to left ventricle decreasing its stroke volume. Chronic aneurysm leads to heart failure, tachyarrythmias and systemic embolization. Death is often sudden, presumably due to tachyarrythmias [79].

Persistent ST elevation despite reperfusion even after 6 weeks in a patient with acute myocardial infarction should arouse suspicion for the presence of chronic aneurysm although definitive diagnosis is done by echocardiography which demonstrates the location of aneurysmal segment. Complications arising out of aneurysms should be managed effectively. Congestive cardiac failure should be treated adequately with diuretics, ACE inhibitors, and digoxin. Drugs like ACE inhibitors, aldosterone antagonists, beta blockers should be started as soon as possible to prevent the cardiac remodelling. To prevent the formation of thrombus and subsequent systemic embolization, patients should be put on long term anticoagulants like warfarin and target international normalised ratio (INR) of 2 to 3 has to be maintained.

8. Arrhythmic complications

One of the common complications seen in the setting of acute myocardial infarction is the development of arrhythmias, and is seen even

before patient reaches the hospital. There are various causes leading to arrhythmias but by far the common is electrical instability which gives rise to re-entry circuits in the electrically inhomogenous ischemic myocardium [80, 81]. Primary ventricular fibrillation is the commonest cause of sudden cardiac death in AMI. Premature ventricular depolarisations (PVC) is by far the most common arrhythmia and sometimes can come as a warning prior to the development of ventricular fibrillation, particularly multifocal PVCs, couplets and runs, PVCs with R on T phenomena. Observation and monitoring is good enough as prophylactic suppression of PVCs has not shown any proven benefit in prevention of ventricular arrhythmias and may be even detrimental [82]. Accelerated idioventricular rhythm is also seen very frequently in upto 20 %, usually following successful reperfusion. However, its occurrence is frequent even in those who are not reperfused making it a poor marker for reperfusion induced arrhythmias. Sustained ventricular tachycardia is also fairly common both in early and late course of STEMI, particularly in late phase of STEMI with poor LV systolic function. Abolition of VT and correction of underlying precipitating factors is very important as progression to ventricular fibrillation is commonly seen, and if resistant and refractory, use of implantable anti tachycardia devices is mandatory. The incidence of ventricular fibrillation is approximately 2% to 4% and is decreasing in the post reperfusion era. Secondary ventricular

fibrillation occurs in patients with LV failure. Amiodarone may be used to treat ventricular arrhythmias in the dosage of 150 mg as loading dose followed by 1mg/min for first 6 hrs and 0.5mg/min for next 18hrs. Lidocaine in the dosage of 1mg/kg for a maximum of 100 mg followed by 1-4mg/min can be used in those refractory to amiodarone.

Polymorphic ventricular tachycardia is a rare complication which can be treated with amiodarone and lignocaine.

Patients with poor LV reserve with recurrent ventricular tachycardia can be treated with implantable defibrillators, particularly those who have ejection fraction below 30 %.

Bradyarrythmias like transient sinus bradycardia, atrioventricular block are more common in inferior and RVMI. Sinus bradycardia seen in inferior wall MI is due to heightened vagal tone which can be treated using atropine. AV block occurs in the setting of AMI particularly in inferior and right ventricular infarction. Generally SupraHis blocks have narrow complex with stable rhythm seen usually in inferior MI as compared to infra His conduction disturbances which are unstable and have wide complex escape rhythm.

Treatment for higher degrees of block like mobitz type 2, trifasicular blocks with significant symptoms and complete AV block is transvenous pacing to improve hemodynamics.

Supraventricular arrhythmias: Sinus tachycardia is, by far, the most common type and occurs due to sympathetic overactivity and anxiety. Atrial fibrillation is seen in 15% of patients, representing ongoing atrial ischemia, which portends bad prognosis; treatment depends upon the ventricular response. In patients with rapid ventricular rate, immediate cardioversion is done to prevent the further aggravation of ischemia. If ventricular response is moderate then beta blockers or calcium channel blockers can be administered. Atrial fibrillation in AMI is associated with a higher mortality and increased incidences of stroke [83, 84].

9. Embolic complication

The overall incidence of development of thrombus in STEMI has decreased over the past decades to less than 5% due to the widespread use of aggressive antithrombotic strategy [85] with risk of developing mural thrombus the highest in anterior wall MI. Early in the phase of AMI, the endocardial layer is inflamed and makes the surface thrombogenic while in chronic phase, due to presence of wall motion abnormalities, ventricular aneursyms and atrial fibrillation predispose to the formation of mural

thrombus. The risk of systemic embolisation is quite high in large mobile thrombus and in those associated with akinetic or dyskinetic myocardium.

Treatment includes anticoagulation by intravenous heparin with a target aPTT of 1.5 – 2 times that of control followed by warfarin with INR of 2-3 for a minimum 3 to 6 months.

10. Pericarditis

Pericarditis in acute myocardial infarction is seen in upto 8 to 10 %. Pericarditis can be seen from as early as first day to 6 weeks, and usually presents with pain that is difficult to differentiate with ischemic pain but the radiation of pain to both the trapezius ridge is characteristic of acute pericarditis. Pain of pericarditis increases with deep inspiration & supine position and gets relieved or decreased by sitting and leaning forward. Due to the involvement of epicardium in a transmural myocardial infarction, leads to acute fibrinous pericarditis. Pericardial friction rub on auscultation is pathognomonic and its presence correlates with a larger infarct.

Although the presence of generalised ST elevation with concavity upward (saddle shaped) favours the diagnosis, ECG features of evolving MI produces a diagnostic dilemma.

Aspirin in high doses 650 mg given every 4-6 hourly is the treatment of choice [87].

Dressler's syndrome occurs within 1 to 8 weeks after myocardial infarction and is also known as post myocardial infarction syndrome. The exact pathogenesis is not known, but an immunological process has been suggested. Clinically presents with fever, malaise, chest discomfort, leuckocytosis and elevated ESR. Aspirin is the drug of choice while steroid is best to be avoided within first 4 weeks post MI.

11. Ischemic complications

These include recurrent infarction, post infarct angina and infarct extension.

Recurrent infarction can be due to reocclusion of the infarct related artery (IRA) or infarction occurring in a separate territory**.** Re occlusion of the infarct related artery is a complication mostly seen in thrombolysed patients**,** diabetes mellitus and prior MI and portends a poor prognosis [87].

Multi vessel and triple vessel coronary heart disease is very common among diabetic & dyslipidemic patients; so recurrent MI occurring in them is difficult to diagnose when it occurs in first week after an AMI. In fact, angiographic evidence of complicated plaque in non infarct related arteries is very common.

Post infarct angina is defined as angina occurring anytime before 30 days post AMI. Incidence is highest among those thrombolysed as compared to revascularised patients. Pathogenesis is said to be due to coronary artery spasm, and has a very high early and late mortality.

The genesis and representation of electrocadiographic representation

The P wave

The atrial activation is done by the sino atrial node. Time taken for the complete activation of the atrium is 0.099 ± 0.012 secs, producing a normal atrial activation of 0.11 secs. Right atrial activity begins first due to its close location to SA node, reflecting as proximal or ascending limb of P wave in the frontal plane leads and ending at the apex of P wave with a duration ranging from 0.02 to 0.04 sec. Activation of the left atrium begins 0.03 secs later than right atrial activation and constitute descending limb of P wave in standard lead II with a duration of left atrial activation ranging from 0.05 sec to 0.06 sec. Thus normal P wave is formed by composite deflection of right and left atrial activation. The normal morphology of P wave is usually best studied in standard lead II and V1 because the frontal P wave axis is usually directed to the positive pole of this lead. The normal P wave is usually in range of 0.08 sec. to 0.10 sec in duration, and maximum normal amplitude being less than 2.5mm.

PR Segment

The PR segment begins from the end of P wave and ends with the onset of QRS complex. It is a flat isoelectric baseline and forms the part of PR interval, beginning from the onset of P wave to the onset of QRS complex. The normal PR interval duration ranges from 0.12 -0.2 seconds.

PR interval represents temporal bridge between atrial and ventricular activation [88].

The genesis of QRS complex

QRS complex in ECG represents ventricular depolarisation. Activation of the ventricles begins in the left subendocardial region of the lower third of the interventricular septum, spreading transversly from left to right. It is opposed by a smaller activation force which occurs almost synchronously but fractionally later, and arises in right subendocardial region of the interventricular septum, spreading transversly through the septum from right to left. The larger left to right force dominates, counteracting the smaller right to left force resulting in an effective vector which is directed transversly from left to right through the lower third of the interventricular septum. This is sometimes referred to as septal force or septal vector.

Activation of the interventricular septum is then followed by activation of the free walls of both the ventricles, occurring transversely from subendocardial to subepicardial regions of free walls of both ventricles. The larger right-to-left force of the free left ventricular wall dominates and counteracts smaller left-to-right force of smaller right ventricular wall, resulting in an effective or net resultant vector which is directed from right to left through the free wall of left ventricle. To explain this in simplified terms, activation of the ventricles may be depicted as a small initial vector from left to right through the interventricular septum, followed by larger vector from right to left through the free wall of left ventricle.

The T wave

The T wave is normally inscribed in the same direction as the QRS complex. It has asymmetric limbs, the proximal limb being shallower than the distal limb. It has relatively blunt apex.

The ST segment

The ST segment represents ventricular repolarization, almost leaving the baseline immediately after its origin from the end of the QRS complex. Thus very little or none of it tends to be isoelectric. The ST segment usually

merges smoothly and imperceptibly with proximal limb of T wave, making it difficult to separate them.

The U wave

The U wave is a small rounded deflection which occurs immediately after the T wave. It is normally in the same direction as the T wave. It is usually best seen in leads V2 – V4. The deflection may be so small making accurate recognition difficult.

The genesis of U wave is uncertain and remains controversial.

The Q-T interval

The Q-T interval is the interval from the starting of QRS complex to the end of T wave, representing the total duration of the ventricular activity i.e., the sum of ventricular depolarization and repolarization. The Q-T interval may vary in different parts of ventricles, but the Q-T interval measured from surface electrocardiogram represents largest Q-T interval.

Measurement of Q-T interval

Measuring Q-T interval may be difficult, because it may be difficult to determine the exact beginning and end of the interval. The beginning of QRS complex is best determined in a lead with an initial Q wave, commonly in order to avoid mistakes of ignoring an initial part of QRS

complex which may be isoelectric in a particular lead and hence not discernible, the Q wave being, in effect, incorporated within the PR interval. The precise end of T wave may, at times, also be difficult to determine; this is because the end of T wave may be obscured by a superimposed U wave or, in the case of sinus tachycardia, by the ensuing P wave. When the Q-T interval is measured from a lead where the U wave is prominent, the nadir or notch between T and U wave is taken as the end of T wave. This is not necessarily the true end point of the T wave but it will do for practical purposes.

The significance of the Heart rate in relation to Q-T interval

The Q-T interval varies with heart rate, shortening with tachycardia and lengthening with bradycardia. In other words Q-T interval proportionally changes with the R-R interval, decreasing with shortening of R-R interval and increases with prolongation of R-R interval. This change occurs due to change in refractory period of myocardium and ventricular repolarisation & indicates that correction of the Q-T should be done in relation to heart rate for purposeful analysis [(88)].

Correction of the Q-T interval: The Q-Tc interval and its calculation

The Q-T interval is corrected for what it would theoretically be at rate of 60 beat per minute [(88)]. Q-Tc interval is the term used to denote corrected

QT interval. Traditionally various formulas have been formulated to correct Q-T interval but Bazetts formula formulated by Bazett is in vogue.

Bazetts formula is

$$Q\text{-}Tc = Q\text{-}T/\sqrt{(R\text{-}R)}$$

The Q-T interval is calculated and the R-R interval is calculated between two consecutive R waves and it should be in seconds.

The Q – Tc may be considered as a constant, K thus,

Normal value for K = 0.39 Sec ± 0.04 Sec.

Normal range thus ranges from 0.35 Sec to 0.43 Sec.

Conditions associated with abnormal Q-Tc are many. Prolonged Q-Tc is seen in acute myocardial infarction, during sleep, hypocalcemia, acute myocarditis, drugs like quinidine, procainamide, tricyclic antidepressants, hypothermia, hypertrophic cardiomyopathy, and certain congenital long Q-T syndromes like Jervall–Lange Neilson syndrome, Romano-Ward syndrome etc.

Shortened Q-Tc is seen in digitalis effect, hypercalcemia, hyperthermia and vagal stimulation.

Surface 12 lead ECG represents the longest Q-T interval and Q-T interval varies from lead to lead on surface ECG by upto fifty milliseconds, being longest in mid precordial leads V2 and V3. This variation represents Q-T dispersion relating itself to electrical instability and predisposing to ventricular arrhythmias [88].

Q-T dispersion and its electrophysiological basis

Q-T dispersion is defined as the difference between maximum and minimum Q-T interval in 12 lead electrocardiogram.

Dispersion of repolarisation of ventricles is widely considered as a representation of non homogenous recovery of excitability, in other words, heterogeneity in ventricular repolarisation. It is commonly expressed as difference of the various repolarisation measured. The importance of this simple electrophysiological measurement was said to be associated with the facilitation of ventricular re-entry arrhythmias [89, 90, 91, 92 & 93]. These experimental findings suggested dispersion of ventricular repolarisation as an important arrythmogenic substrate, and were later extended when dispersion of repolarization along the trans mural axis of the myocardium was shown to play a key role in the genesis of arrhythmias specifically associated with the acquired or congenital long QT syndrome [95, 96, 97]. QT dispersion represents dispersion of ventricular repolarization, and therefore,

is a potential measure of substrates for re-entry tachycardia. Evidences obtained from various studies reinforce the significance of increased repolarization heterogeneity in the genesis of re-entry and malignant ventricular arrhythmias.

T wave genesis and dispersion

The basic principle behind the genesis of the T wave is very essential in order to understand pathophysiological meaning of QTd and its genesis [97]. T wave recorded in the 12 lead surface ECG is the result of temporal and spatial voltage gradient within the ventricular myocardium representing the phase of cellular repolarisation but sometimes particularly in bundle branch block and premature ventricular beats it also includes phase of depolarisation.

These voltage gradients are complex geographical representation of the cellular Repolarization and its dispersion in time and space. It has been proven time and again that the T wave from the body surface itself is a direct expression of dispersion of ventricular repolarization. By far, the bulk of the T wave represents the greater portion of ventricular repolarization gradients and only a small fraction of terminal portions of T wave represents repolarization differences in the voltage or time domain. The small amount

of cells which are still repolarising, minute gradients still persist but escape detection in some ECG leads [97].

T wave is usually ignored in the standard method of QTd measurement. If we measure only the temporal difference the that occurs between the ends of the shortest and the longest QT interval between the 12 ECG leads; we miss out on the most important reflection of ventricular repolarization dispersion is the T wave proper. Hence, all the variables expressing either the T-wave area or the T-wave width have to be more tightly related to dispersion of ventricular repolarization. From this thought process we can hypothesize that disparities of repolarization in the voltage - domain, more importantly than in the time – domain (as depicted by QTd), can be a better appropriate index of repolarization dispersion. This idea of voltage domain dispersion of repolarization is experimentally supported by Behrens et al who showed in isolated rabbit hearts that the voltage differences in amplitude – normalized monophasic action potential (MAP) recordings closely correlated with the shape of the concurrent T wave which was recorded from the tissue bath chamber. Of that interestingly the T – wave peak seem to occur precisely at the time of the maximal difference in MAPs.

The role of early after repolarisation and genesis of QT dispersion and arrhythmias

Without understanding early after depolarisation it is very difficult to study dispersion of ventricular repolarisation. EADs occurs more commonly in mid myocardial M cells and purkinje cells when they are treated with agents which prolong APD. EADs are nothing but the prolongation of repolarisation occurring in the phase 3 of action potential, which prolong ventricular repolarisation occurring in the myocardium, sometimes excessively reaching a voltage level that triggers premature depolarization. They occur predominantly due to reduction of net outward current which may be due to either an increase of inward current and/or decrease of outward current. In other words, EADs occurs when balance of current shifts inward during phase 2 or 3 of action potential. EADs precipitate Torsade de pointes (Tdp) arrhythmias in the setting of either congenital and acquired long QT syndromes by triggering mechanism or by their ability to sustain ectopic depolarizations within the ventricles[98, 99]. This is based on studies that EADs can be reproduced using drugs that prolong the action potential duration by blocking the potassium outward channel, which may trigger repetitive depolarization and TdP arrhythmias.

By MAP catheter technology it is possible to record the recordings which furnishes direct insight into the pathophysiology in intact animal

hearts and even in patients. An example from the available literature induced prolonged QT intervals and episodes of (TdP). The MAP tracings show conspicuous prolongation of repolarisation and a prominent EAD that precedes the polymorphous rhythm. This action potential (AP) prolongation and EAD is associated with change in polarity of the T wave and the origin of a giant U wave. The marked prolongation of the AP also leads to exaggeration of dispersion of ventricular repolarization that may play a role in any function re entry present in sustained triggered arrhythmias. This possibly explains EADs developing in the selected transmural M cells can actually set the stage for re-entry by exaggerating transmural dispersion of repolarisation (TDR). It is noteworthy to remind that EAD is also a possible explanation in the origin of ventricular arrhythmias in cases of heart failure and cardiac hypertrophy.

Therefore, EAD appears to play an important role in both triggering and sustaining serious ventricular arrhythmias.

Measurement of QT dispersion

Measurement of QT dispersion, especially its accuracy is mainly determined by the precision with which the QT intervals are noted in individual leads. However, we know that measurement of the QT interval is unreliable. The main problem stems predominantly because of the

difficulties of the exact determination of the T-wave end. Independent of whether the measurement is performed by a human observer or by a computer algorithm, the main source of error arise from specific morphological patterns of the T wave, merges of the T wave with the U wave, low amplitude of the T-wave signal, and, at fast heart rate, merges of the T wave with the P wave.

The morphology of the T wave can influence any measurement of the QT interval, and frequently cited beliefs that computer algorithms are less affected than humans are not justified. The shape of the descending limb of the T wave obviously influences the determination of the T- wave end by any down slope tangent computer algorithm, as is any threshold algorithm by the amplitude of the T wave. The amplitude of the T wave very strongly influences the reliability of automatic [100, 101] and manual measurement.

It is often very difficult, almost impossible, to separate the end of the T wave from the start of U wave and also to mark the end of the T wave with a better accuracy than that of few tens of milliseconds. In1952, Lepeschkin et al explained various patterns of T- and U-wave merges and differentiated them into 16 patterns. They also suggested methods for finding the end of the T wave when it is lost within the U wave. It was also shown that, depending on the particular pattern of T-U wave merging, either

the point of intersection of the tangent to the down slope steepest point and the isoelectric line, or the nadir between the T and the U wave is actually closer to the real end of the T wave.

However, most of the available methods are not suitable for certain pattern of T-U merging, for example when U wave is augmented and/or when T wave is inverted or flat. This ECG pattern is usually observed in patients with severe hypokalemia and be mistakenly taken as a grossly prolonged interval that often comes before torsades de pointes tachycardia.

Q-T interval after Acute Myocardial Infarction

In early as 1953 Elk et al reported prolongation of QT interval in the early phase of myocardial infarction. But the relation between serial changes in QT interval and outcome of MI was undefined. Schwartz et al in 1975 demonstrated risk of sudden death in patients with prolonged QT interval in healed MI. Later, similar association were found between a prolonged QT interval and high risk of sudden death even in acute myocardial infarction.

Time related changes in the QT interval after acute Myocardial Infarction

Doroghazi RM and Childer's in 1978[(104)] first studied the sequential QT interval changes in AMI. They obtained Serial ECG's after infarction for 14 days and the changes in the QTc was analysed retrospectively.

They observed prolonged QT at some point of time in first five days and by day six, QT interval returned to the site of peri-infarction in most cases. Abnormal QT Interval was observed in 37% of patients with inferior wall infarction and 40% of patients with anterior wall infarction. QT interval returned to normal at the end of 5-6 days irrespective to the degree of initial QT and did not prolong further thereafter. The mean QTc was found to be greatest on day two in the majority of the patients. They also stated the magnitude of QT prolongation was higher among non-transmural infarction as compared to transmural infarction which was statistically insignificant.

Rate dependence of dispersion of repolarization

A common point of discussion and uncertainty in dispersion of repolarisation and QT dispersion from ECG is how to correct QTd and other variables of heart rate. Earlier clinical studies of QTd used a Bazett Formula based algorithm for both QT dispersion and JT dispersion. Correction of rate of QT and JT dispersion was done under the assumption that dispersion of ventricular repolarization showed almost similar rate dependence as its duration. The adaptation of QT interval rate physiologically has been shown previously by others to lay within the rate adaptation characteristics of action potential duration. Therefore, many algorithms have been recommended for clinical rate correction of the QT interval. In contrast however, the dispersion of ventricular repolarization is shown to be rate

independent in an experimental study using multiple MAPs from an isolated rabbit heart which was paced at different heart rates. This independence of rate of QTd was confirmed in normal patients and also seen in patients with minor heart diseases. Long QT syndromes both congenital and acquired should not be included in this rule because this particular pathophysiological situation exhibits markedly increased dispersion of ventricular repolarization at slow heart rates.

Unfortunately, before many studies of rate dependence of dispersion of ventricular repolarization was done and analysed, rate correction of QTd with reporting QTc dispersion had been widely done. QTc dispersion had even been advocated as an improved risk stratifier in patients. However, it is possible that QTd dispersion was contaminated by rate correction due to addition of the unnecessary heart rate variable and also the well known over correction of QT in patients with faster heart rates. Heart rate an independent risk stratifier by itself may ironically introduce an independent risk marker that is then unknowingly and falsely attributed to the measurement of QTc dispersion [97].

Therefore, rate correction of QTc dispersion and other ventricular dispersion variables should be avoided unless such variables are found to be directly influenced by heart rate.

QTd from surface ECG was developed as a simple noninvasive clinical risk marker to reflect dispersion of ventricular repolarization at the level of myocardium. Although its exact derivative is not yet fully understood or determined, QTd may depend on a composite of inhomogeneous repolarization forces which includes the T-wave vector and a changing component of local influences believed to be the main explanation for QTd. For many theoretical reasons, QTd may not represent the most useful ECG variable, actually reflecting the actual dispersion of ventricular repolarization. But it is very clear that dispersion of ventricular repolarization at the myocardial level is an important arrhythmogenic substrate. The future will show whether other non invasive methods in the making, which more directly relate to the pathophysiological substrate and are methodologically more robust, will be able to improve stratification of patients at risk for life –threatening arrhythmias.

MATERIALS AND METHODS

This is an observational study conducted in Government Kilpauk medical college hospital, Department of medicine in collaboration with Department of Cardiology, for a period of 1 year. A total of 60 cases admitted with acute myocardial infarction and who are thrombolysed were selected for the present study from January 2011 to January 2011.

Among 60 patients studied 30 cases are those who are thrombolysed in less than 3 hours after the onset of chest pain and rest 30 are those who presented late and thrombolysed later than 3 hours after onset of chest pain.

Patients who fulfil inclusion and exclusion criteria were enrolled for the study after obtaining written informed consent. The study protocol was approved by the ethical committee of Govt. Kilpauk medical college hospital, Chennai-10 for research studies.

Inclusion criteria

Patients admitted in ICCU of Govt. Kilpauk medical college hospital with complaints suggestive of acute myocardial infarction and 2 lead ECG showing ST elevation are included in this study. The patients who are thrombolysed are included in the study. Age group included was everyone above 18 years who had acute myocardial infarction.

Exclusion criteria

Medical conditions and patients who were on drugs which prolongs the QT interval are excluded from study like

1. Electrolyte imbalance
2. Patients in atrial fibrillation.
3. Unmeasurable T waves.
4. Patients with bundle branch block.
5. Drugs affecting QT interval- antiarrythmics, macrolide antibiotics, cisapride and other prokinetic drugs.
6. Patients with contraindications to thrombolysis.

Data collection

Detailed history was taken from the patients that includes onset of symptoms, past medical history and thorough physical examination and systemic examination was done and entered in proforma specially designed for this study.

Blood investigations

In all patients with myocardial infarction, routine investigation like complete blood count and urine examination was done. Biochemical parameters like random blood sugar, fasting lipid profile and cardiac enzymes like creatinine phosphokinase (CPK, CK-MB) was done.

ECG Recording:

ECG recordings were done on admission before thrombolysis and 90 minutes after thrombolysis, day 2 & day 5 and 6 weeks of follow up. ECG was recorded with an ECG recorder speed of 25mm/sec.

Measurement of QT dispersion:

QT interval was measured in all leads from the beginning of QRS complex to end of T wave. In the presence of U wave, QT interval was measured till nadir of curve between T and U waves. Each QT interval was corrected for the patient's heart rate using Bazett's formula.

(QTc = QT/√ RR (sec)) (QTc is the corrected QT interval).

QT dispersion on each electrocardiogram is "the difference between the maximal and minimal QT interval in any of the leads measured". Accordingly QTc dispersion is defined as "the difference between maximal and minimal heart rate corrected QT interval''.

Cases were further divided into

1. Those who presented early in less than 3 hours after AMI and thrombolysed early and those who are thrombolysed later than 3 hours.

2. Successful and failed thrombolysed group.(failed thrombolysis based on clinical and ECG criteria).
3. Ventricular Arrhythmia and No Ventricular Arrhythmia group.

The obtained data's were entered and tabulated in the Master chart and statistical analysis done using SPSS software. Univariate analysis was done with paired t- test and pearson product moment correlation co-efficient. P value < 0.05 was considered to be statistically significant.

OBSERVATIONS AND ANALYSIS

TABLE 1: AGE AND SEX DISTRIBUTION OF EARLY REPERFUSED CASES.

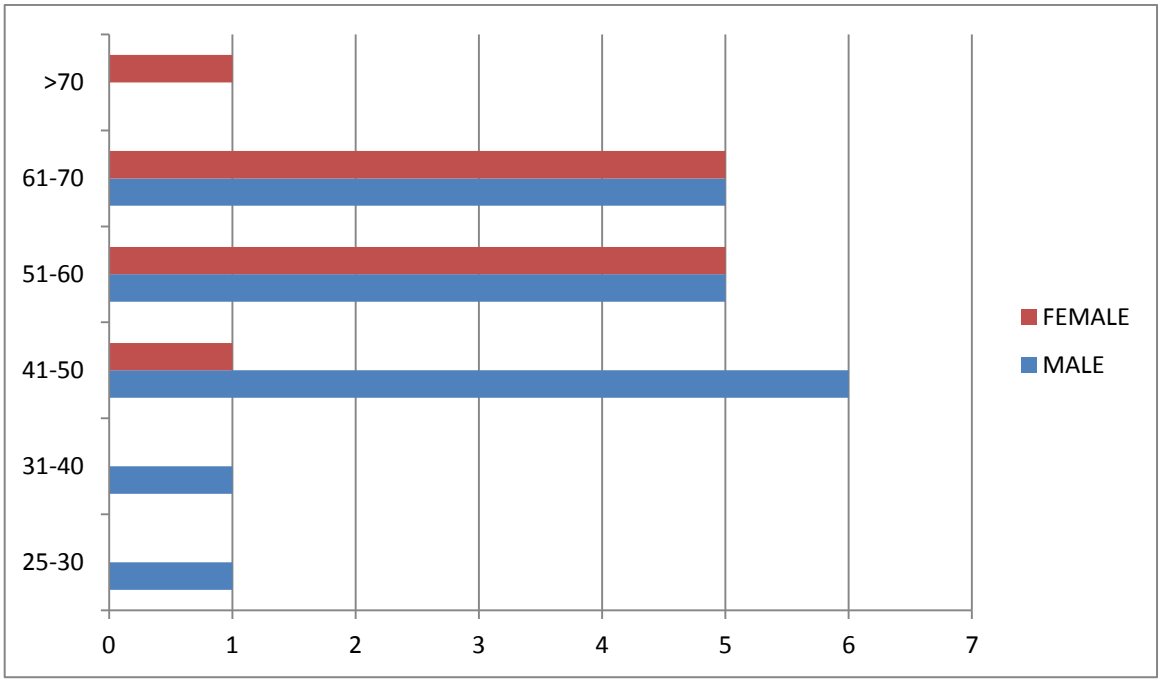
AGE	MALE	FEMALE	TOTAL
25- 30	1	0	**1**
31-40	1	0	**1**
41-50	6	1	**7-**
51-60	5	5	**10**
61-70	5	5	**10**
>70	0	1	**1**
	18	**12**	**30**

Out of 30 cases of early thrombolysed acute myocardial infarction studied

18 were men

12 were women

CHART 1: AGE AND SEX DISTRIBUTION OF EARLY THROMBOLYSED CASES



The average age of presentation of myocardial infarction among those who were thrombolysed early is 55.8 years.

The maximum incidence of MI in men is seen in the age group of 41-50 years.

The maximum incidence of MI among women is seen in the age group of 51-60 and 61-70 years equally in the study group.

TABLE 2: AGE AND SEX DISTRIBUTION OF CASES IN LATE THROMBOLYSIS

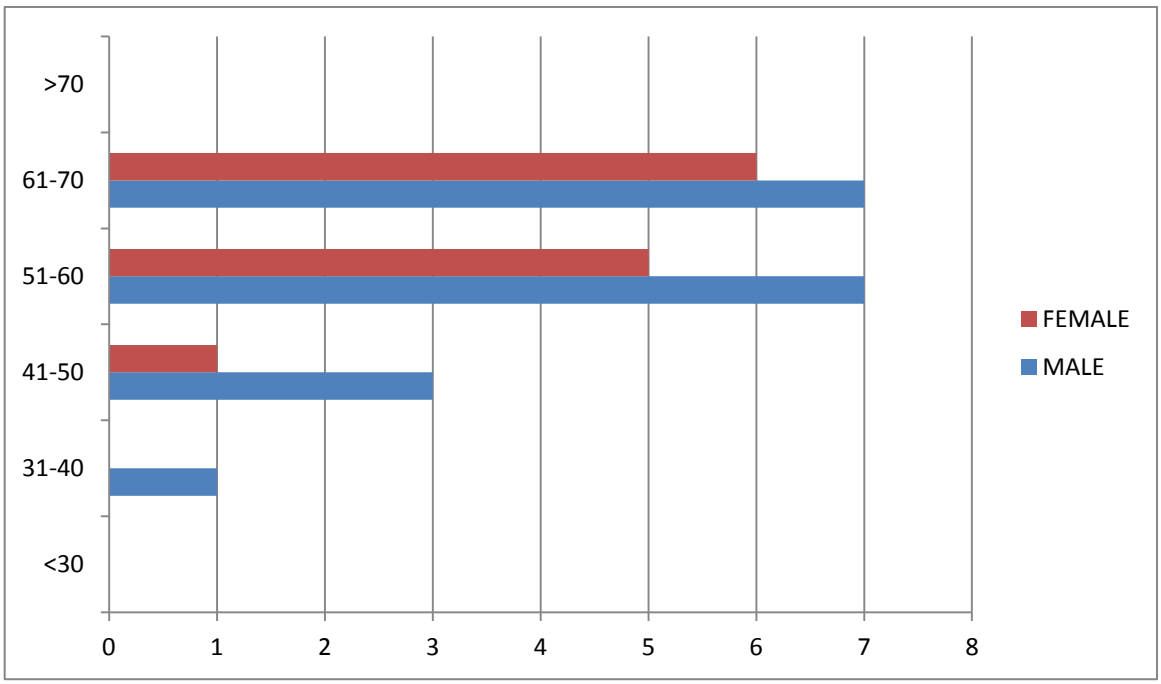
AGE	MALE	FEMALE	TOTAL
<30	0	0	**0**
31-40	1	0	**1**
41-50	3	1	**4**
51-60	7	5	**12**
61-70	7	6	**13**
>70	0	0	**0**
	18	**12**	**30**

Out of 30 cases of late thrombolysed acute myocardial infarction studied

18 were men

12 were women

CHART 2: AGE AND SEX DISTRIBUTION OF CASES IN LATE THROMBOLYSIS

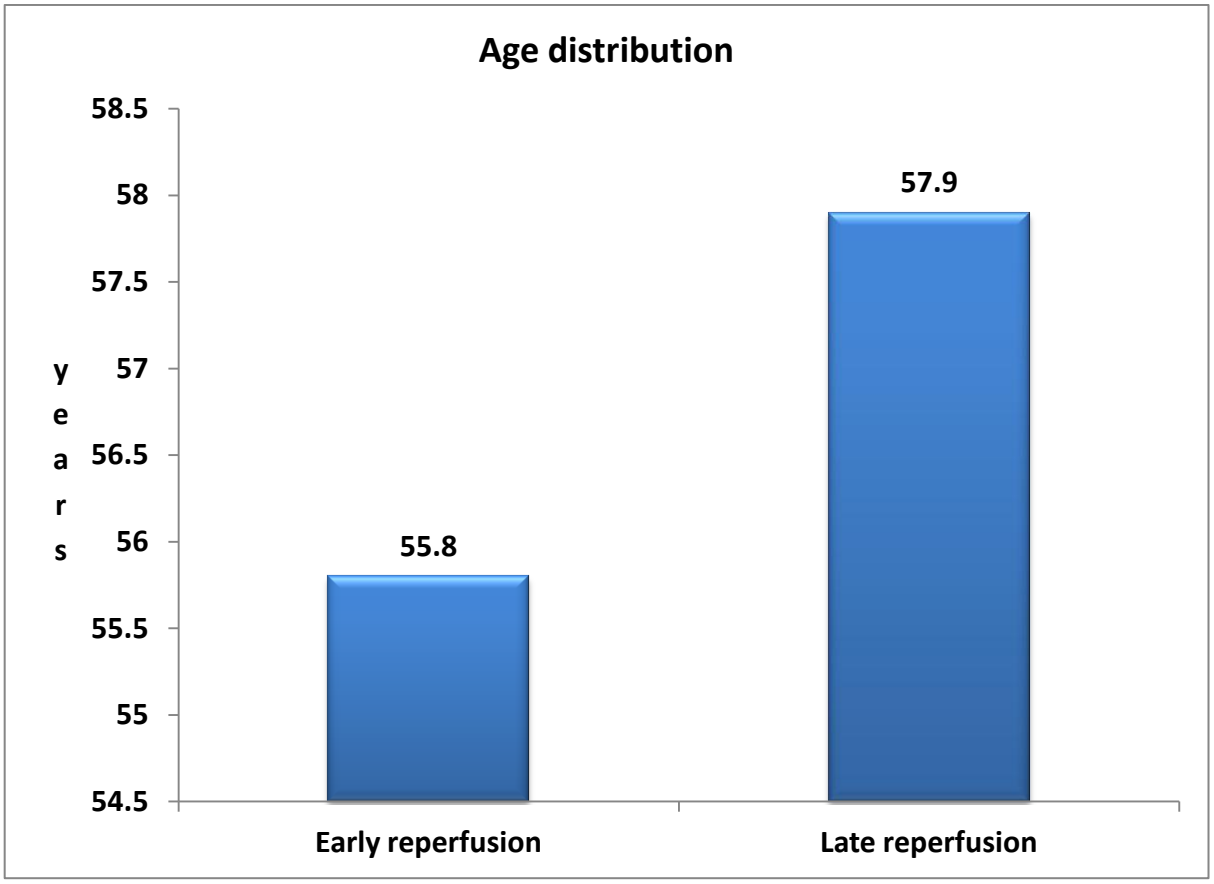


The average age of presentation of myocardial infarction among those who were thrombolysed late is 57.9 years.

The maximum incidence in men is seen in the age group of 51-60 and 61-70 years equally.

The maximum incidence among women is seen in the age group of 61-70 years.

CHART 3: MEAN AGE OF PRESENTATION OF CASES IN BOTH EARLY AND LATE GROUP



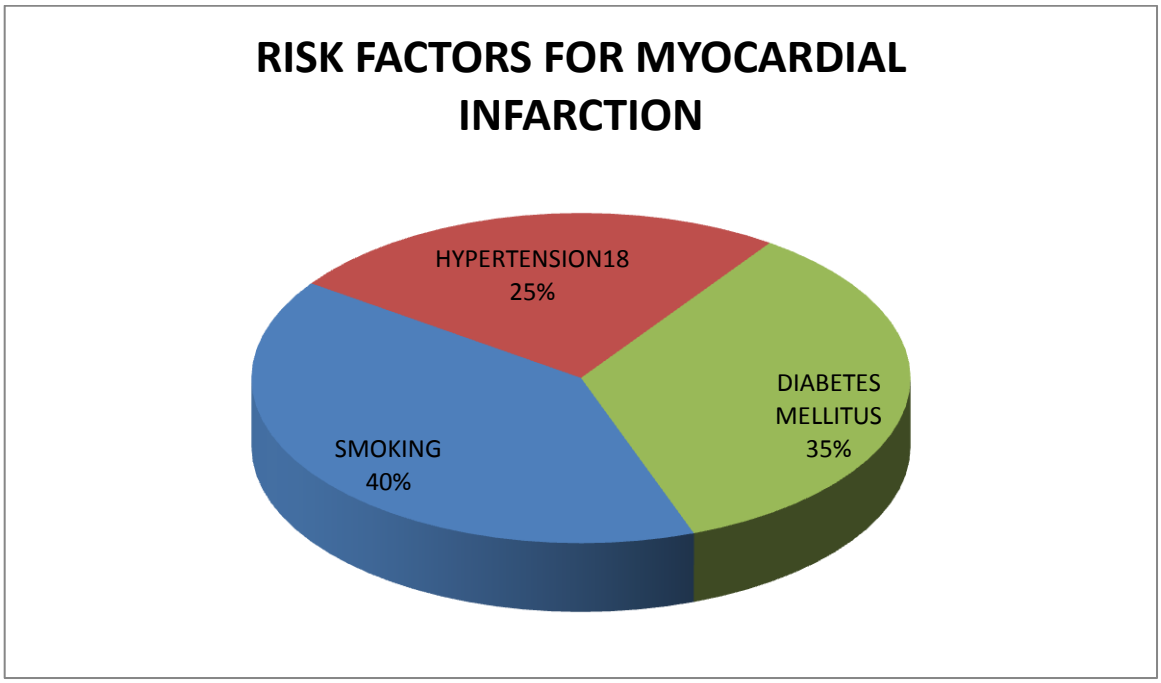
The above graph represents the mean age of presentation of acute myocardial infarction in early reperfused group (55.8 years) and late reperfused group (57.9) in our study.

TABLE 3: RISK FACTORS FOR MYOCARDIAL INFARCTION

RISK FACTORS	NUMBER OF CASES
SMOKING	29
HYPERTENSION	18
DIABETES MELLITUS	25

In our study involving 60 cases of acute myocardial infarction predominant risk factor for causation is smoking seen 29 cases followed by diabetes mellitus (25 cases) and hypertension (18 cases). All the three risk factors were present in 5 cases.

CHART 4: RISK FACTOR FOR MYOCARDIAL INFARCTION

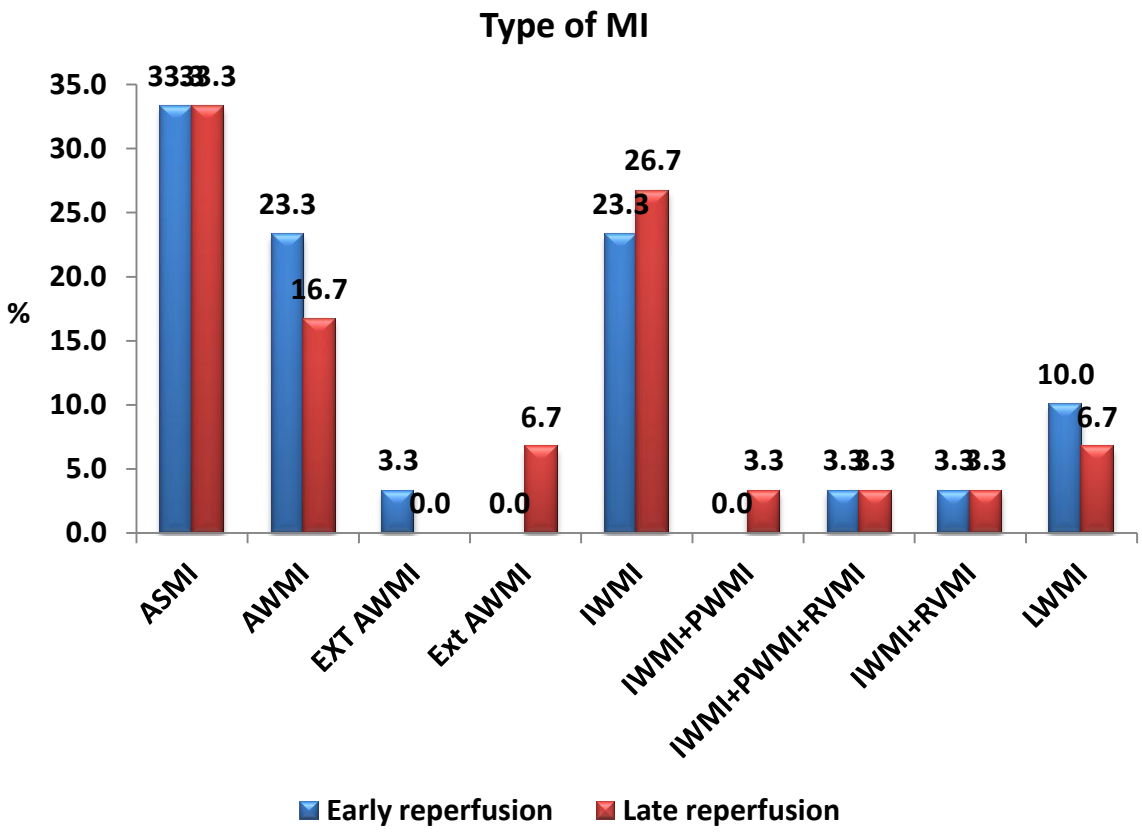


The above pie diagram represents the risk factors in percentage contributing for the acute myocardial infarction in our study group.

TABLE 4: TYPES OF MYOCARDIAL INFARCTION INCLUDED IN THE STUDY GROUP.

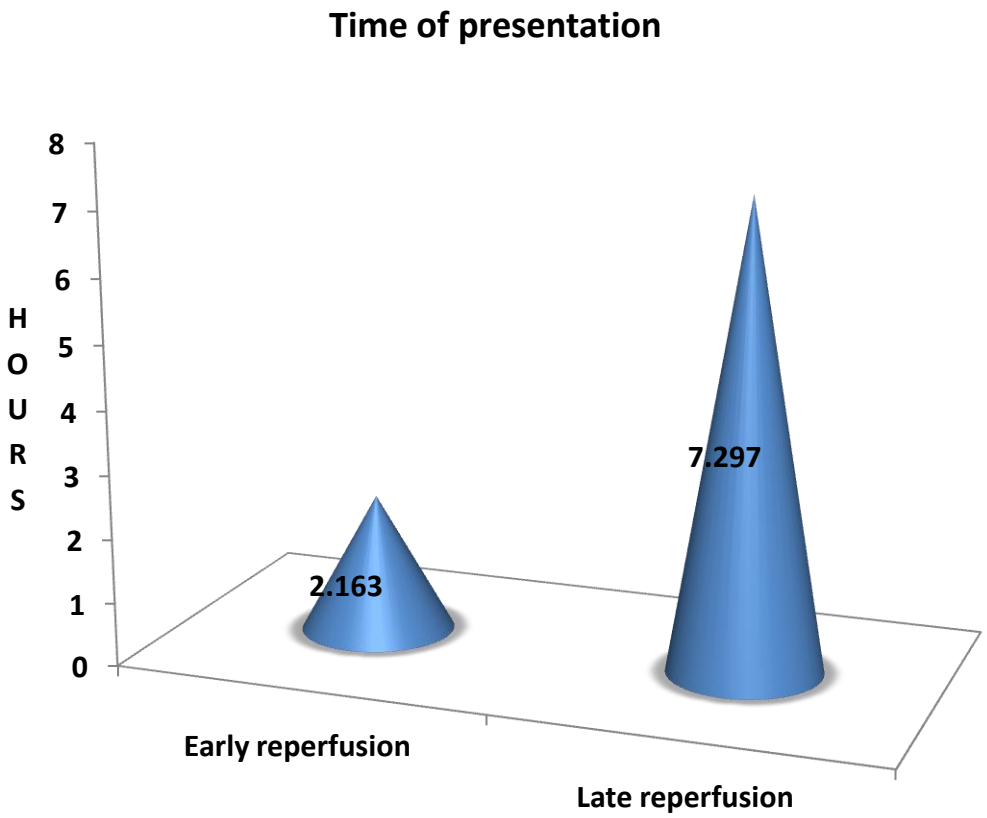
Type of MI		Group		Total
		Early	**Late**	
ASMI	Count	10	10	20
	% within Group	33.3%	33.3%	33.3%
AWMI	Count	7	5	12
	% within Group	23.3%	16.7%	20.0%
EXT AWMI	Count	1	0	1
	% within Group	3.3%	.0%	1.7%
Ext AWMI	Count	0	2	2
	% within Group	.0%	6.7%	3.3%
IWMI	Count	7	8	15
	% within Group	23.3%	26.7%	25.0%
IWMI+PWMI	Count	0	1	1
	% within Group	.0%	3.3%	1.7%
IWMI+PWMI+RVMI	Count	1	1	2
	% within Group	3.3%	3.3%	3.3%
IWMI+RVMI	Count	1	1	2
	% within Group	3.3%	3.3%	3.3%
LWMI	Count	3	2	5
	% within Group	10.0%	6.7%	8.3%
Total	Count	30	30	60
	% within Group	100.0%	100.0%	100.0%

CHART 5: TYPES OF MI INCLUDED IN THE STUDY GROUP.



In the present study group, the number of various myocardial infarction studied in both early and late reperfused group were almost similar indicating no selection bias was done in recruiting the cases for the analysis of QT dispersion.

CHART 6: MEAN TIME OF PRESENTATION IN BOTH EARLY AND LATE GROUP



The above graph represents the mean time of presentation of cases in both early and late group.

TABLE 5: QT PAREMETERS IN BOTH STUDY GROUPS BEFORE AND AFTER THROMBOLYSIS.

	Group	N	Mean QT	Std. Deviation	P-value
Before thrombolysis max	Early	30	.4727	.02377	0.000
	Late	30	.5153	.04911	
Before thrombolysis min	Early	30	.4220	.02524	0.631
	Late	30	.4183	.03312	
After thrombolysis max	Early	30	.4710	.02784	0.000
	Late	30	.5413	.04876	
After thrombolysis min	Early	30	.4230	.02615	0.064
	Late	30	.4370	.03109	

The above observation shows the mean maximum & minimum QT interval between early and late groups.

The correlation between Maximum QT before and after reperfusion in the two groups is statistically significant ($p<0.05$) indicating significant prolongation of maximum Q-T interval in late group on comparing with early group.

CHART 7: MEAN QT MAXIMUM AND QT MINIMUM IN BOTH STUDY GROUPS

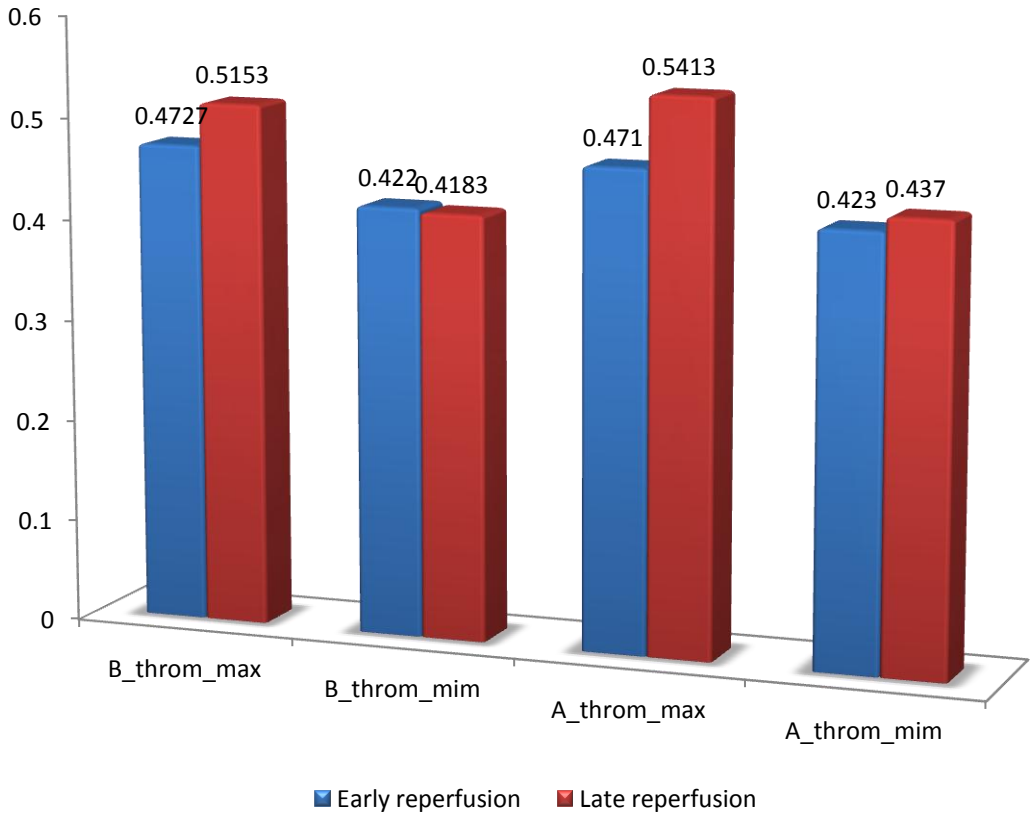


TABLE 6: TABLE SHOWING MEAN QT DISPERSION BETWEEN EARLY AND LATE THROMBOLYSED CASES

	Group	N	Mean QTd	Std. Deviation	P-value
Before thrombolysis	Early	30	50.33	19.025	0.000
	Late	30	97.67	38.118	
After thrombolysis	Early	30	48.33	21.509	0.000
	Late	30	103.00	49.071	
Second day	Early	30	112.00	40.802	0.000
	Late	30	167.67	51.574	
Fifth day	Early	30	44.33	19.945	0.111
	Late	30	54.00	25.944	
Six weeks	Early	30	48.00	18.458	0.228
	Late	30	41.67	21.669	

The above observation represents the mean QTd in the early and late thrombolysis groups, with the QT dispersion being higher in acute myocardial infarction among late reperfused group than early reperfused group.

The correlation between the QTd and time of reperfusion is statistically significant ($p<0.05$) in those taken before & after thrombolysis and on 2nd day.

CHART 8: MEAN QT DISPERSION BETWEEN EARLY AND LATE THROMBOLYSED CASES.

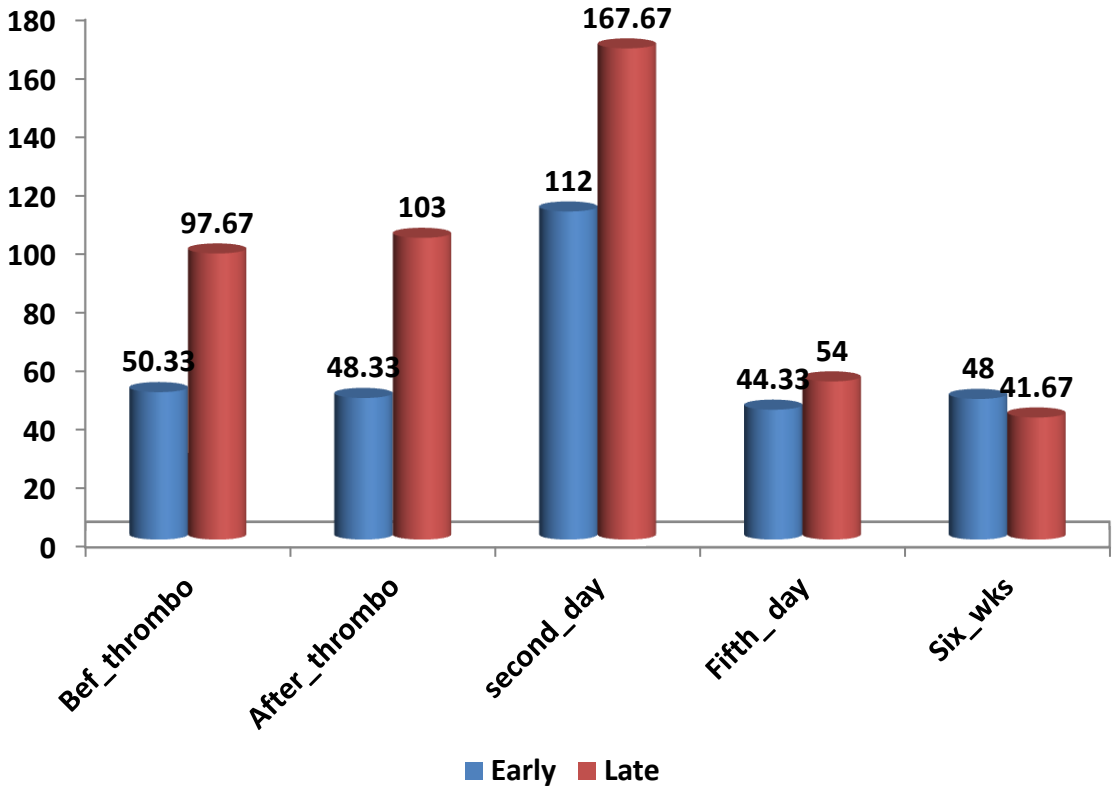


TABLE 7: QT DISPERSION BEFORE AND AFTER THROMBOLYSIS IN EARLY REPERFUSION.

Early Reperfusion	Mean QTd	N	Std. Deviation	P-value
Before thrombolysis	50.33	30	19.025	0.000
Second day after thrombolysis	112.00	30	40.802	

In the above observation mean QT dispersion before thrombolysis (50.33ms) and 2^{nd} day after thrombolysis (112ms) is compared in the early thrombolysed group and is found to be significantly increased on 2^{nd} day ($p<0.05$) .

CHART 9: GRAPH SHOWING QT DISPERSION BETWEEN BEFORE AND 2ND DAY AFTER THROMBOLYSIS IN EARLY REPERFUSION

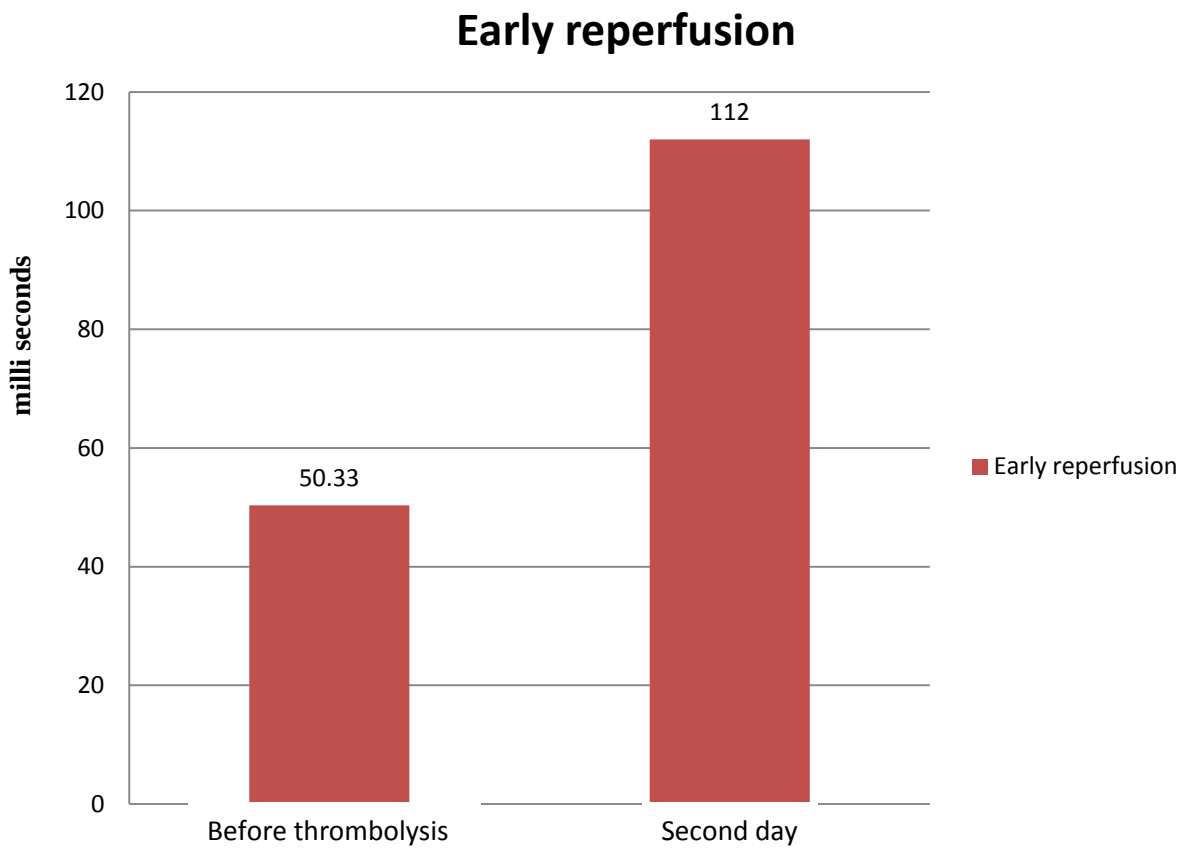


TABLE 8: QT DISPERSION BETWEEN BEFORE THROMBOLYSIS AND 2ND DAY AFTER THROMBOLYSIS IN LATE REPERFUSION

Late reperfusion	Mean QTd
Before thrombolysis	97.67
Second day after thrombolysis	167.67

In the above observation mean QT dispersion, before thrombolysis (97.67ms) and 2nd day after thrombolysis (167.67) in late reperfusion group is compared and is found to be significantly increased on 2nd day ($p<0.05$).

CHART 10: GRAPH SHOWING QT DISPERSION BEFORE THROMBOLYSIS AND 2ND DAY AFTER THROMBOLYSIS IN LATE REPERFUSION

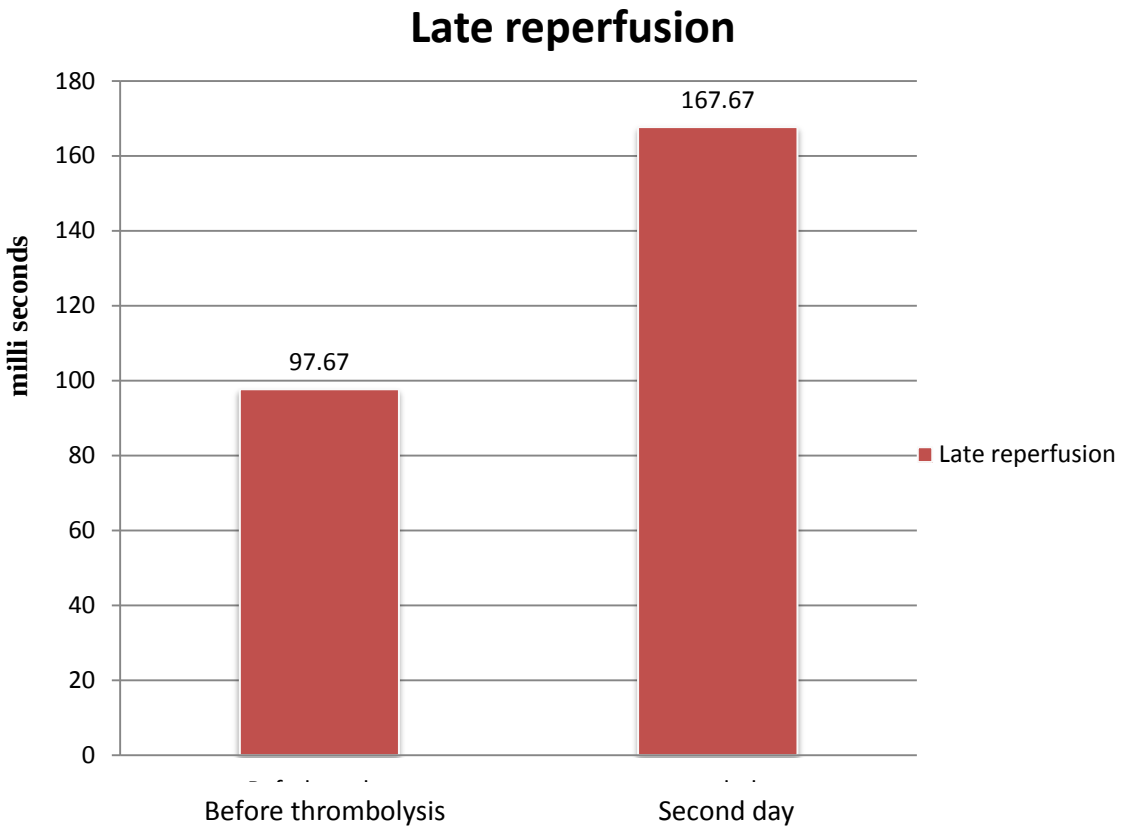


TABLE 9: PREVALENCE OF VENTRICULAR ARRYTHMIAS IN EARLY AND LATE GROUP

Group		Ventricular arrythmias		Total
		Yes	No	
Early	Count	2	28	30
	% within	6.7%	93.3%	100.0%
Late	Count	6	24	30
	% within	20.0%	80.0%	100.0%
Total	Count	8	52	60
	% within	13.3%	86.7%	100.0%

In our study we had two cases of ventricular arrhythmias in early reperfused group and six cases in late reperfused group.

CHART 11: PREVALENCE OF ARRYTHMIAS IN EARLY AND LATE REPERFUSION GROUP.

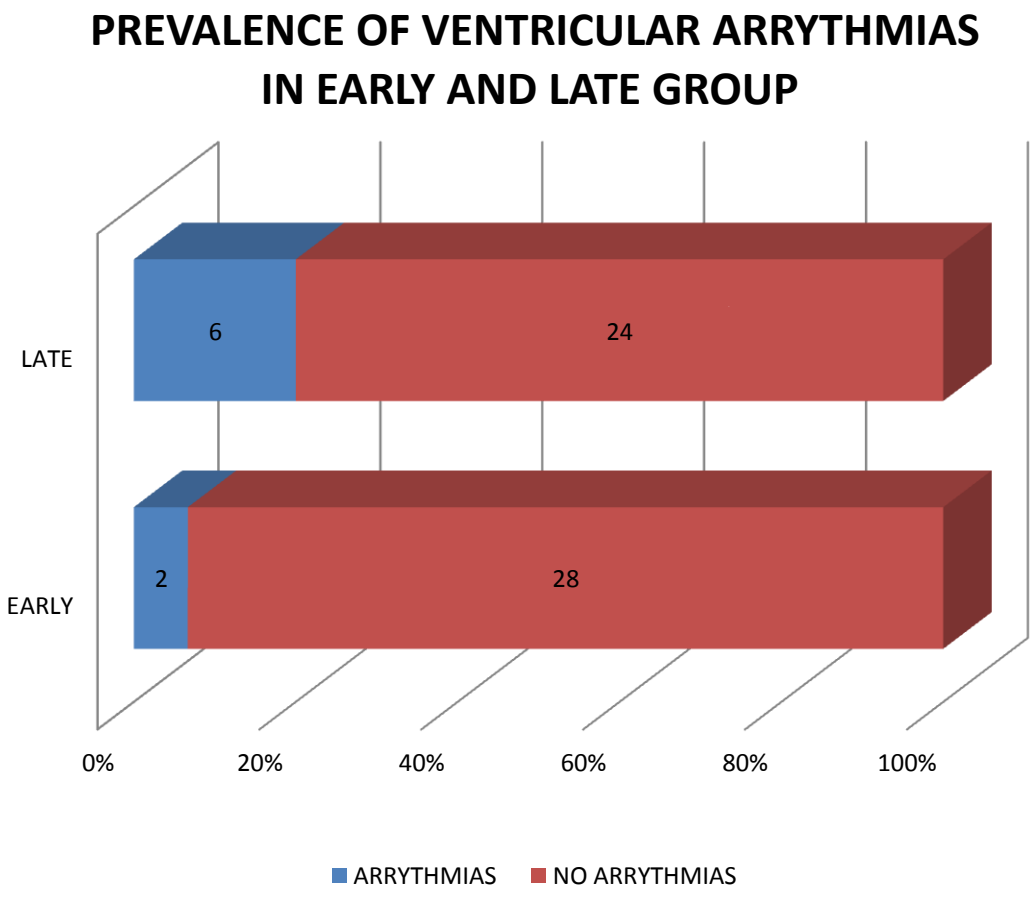


TABLE 10: MEAN QTd IN LATE REPERFUSION GROUP WITH VENTRICULAR ARRYTHMIAS.

Early group	Ventricular arrythmias	N	Mean QTd	Std. deviation	p- value
Before thrombolysis	Yes	2	85	7.071	0.005
	No	28	47.86	17.074	
After thrombolysis	Yes	2	100.00	14.142	0.000
	No	28	44.64	16.603	
Second day	Yes	2	160.00	14.142	0.085
	No	28	108.57	39.974	
Fifth day	Yes	2	40.00	000	0.244
	no	28	44.64	20.635	

The above observation shows mean QT dispersion values of arrhythmias group and no arrhythmias group before and after thrombolysis, 2nd day and fifth day post thrombolysis. There is a significant increase in QTd in those with arrhythmias compared to those without. This correlation is statistically significant in the ECGs taken before and after thrombolysis, and not in the ones taken on 2nd & 5th day. This indicates significant QT dispersion prolongation among those who had arrhythmias in the earlier phase of thrombolysis.

TABLE 11: MEAN QTd IN LATE REPERFUSION GROUP WITH VENTRICULAR ARRYTHMIAS.

Late group	Ventricular arrythmias	N	Mean QTd	Std. deviation	P-value
Before thrombolysis	yes	6	81.67	26.394	0.257
	no	24	101.67	39.964	
After thrombolysis	Yes	6	120.00	61.319	0.312
	no	24	98.75	46.092	
Second day	Yes	6	186.67	75.542	0.322
	no	24	162.92	44.671	
Fifth day	Yes	6	56.67	20.656	0.784
	no	24	53.33	27.452	

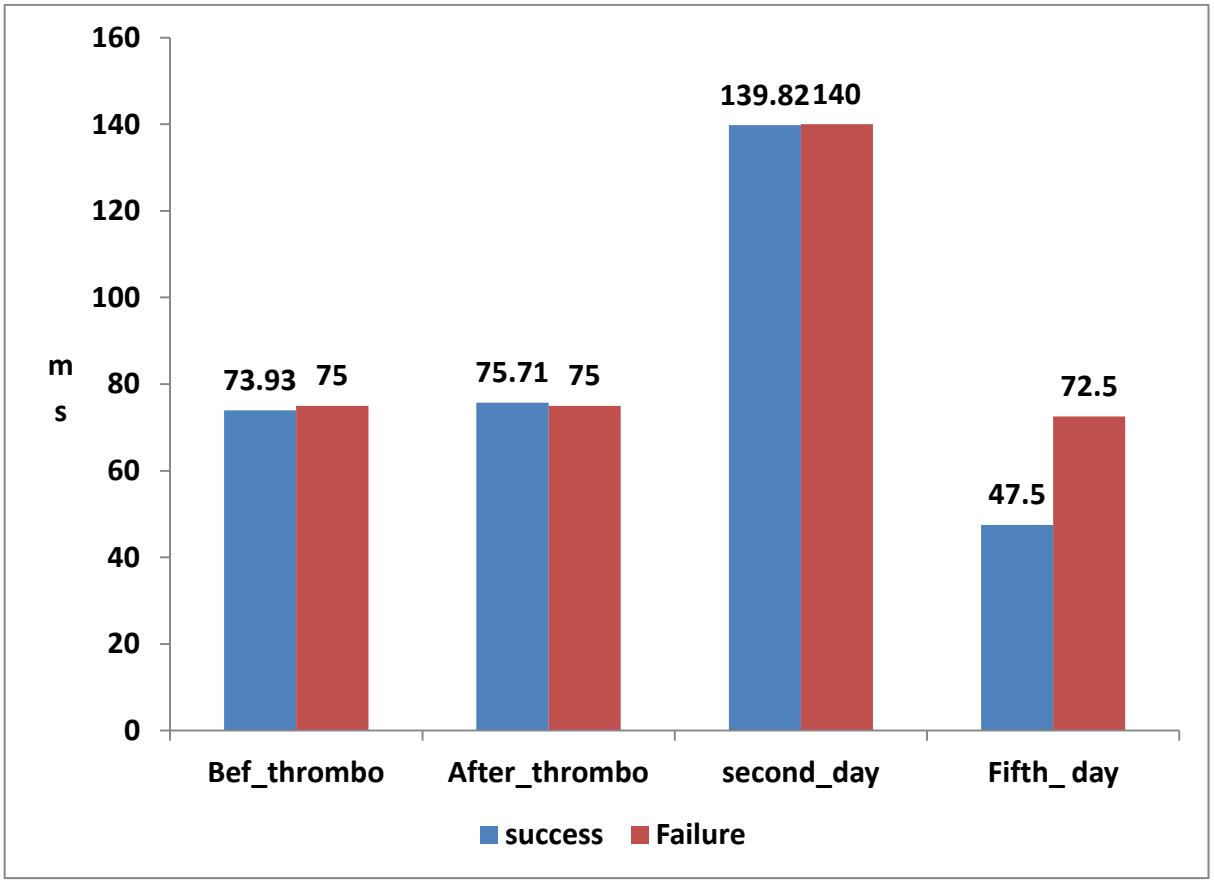
The above table shows mean QT dispersion values in late reperfusion group in those with and without arrhythmias. There mean QT dispersion was more in those with arrhythmias compared to those without in the ECGs taken after thrombolysis but this correlation was statistically not significant ($p>0.05$)

TABLE 12: MEAN QTd IN SUCCESSFUL AND FAILED THROMBOLYSIS.

	Status of the treatment	N	Mean QTd	Std. Deviation	p-value
Before thrombolysis	success	56	73.93	38.408	0.957
	Failure	4	75.00	41.231	
After thrombolysis	success	56	75.71	44.716	0.977
	Failure	4	75.00	77.675	
Second day	success	56	139.82	54.689	0.995
	Failure	4	140.00	49.666	
Fifth day	success	56	47.50	21.847	0.038
	Failure	4	72.50	35.940	

In the above observation QT dispersion is compared between those with successful and failed thrombolysis, before and after treatment till 5 days and lower QTd is seen in the recovery phase among those with successful thrombolysis which is statistically significant in the one taken on 5^{th} day post thrombolysis.

CHART 12: MEAN QTd IN SUCCESSFUL AND FAILED THROMBOLYSIS



DISCUSSION

Acute myocardial infarction in the spectrum of ischemic heart disease is the most common cause of death and is seen to be increasing in the present scenario both in rural and urban area in both developed and developing nations. Due to high prevalence of risk factors like smoking, hypertension, diabetes mellitus, alcoholism together with adverse life style changes it is increasing day by day. Ventricular arrhythmias occurring in the acute setting adds to its high mortality though preventable [(3)]. Recognition of patients developing high risk arrhythmias is a challenging job in coronary care units especially in setting of acute myocardial infarction. Increased QT prolongation and vulnerability of ventricular myocardium has been well studied and documented in earlier studies, nevertheless there is a need to develop more sophisticated and sensitive measures to identify it [(35,118,119)]. However there is a strong evidence for correlation between prolongation of QT interval and sudden cardiac death. Previous studies have proved beyond doubt that successful reperfusion decreases the QT dispersion and hence incidence of ventricular arrhythmias and mortality [(104, 105)]. Analysing QT dispersion will definitely be helpful as a simple, non invasive tool in predicting the arrythmogenecity of the heart and aid in the treatment particularly in rural areas.

The present study evaluates QT dispersion in patients with acute myocardial infarction treated with early thrombolytic therapy (< 3hrs) and those with a delay (>3 hrs) after the onset of chest pain.

QT dispersion and myocardial infarction

In acute myocardial infarction ventricular repolarisation is inhomogeneous, a complex interaction exists between an ischemic myocardium and depolarised dying tissue affecting QT interval and thereby QT dispersion.

In our study, mean QT dispersion ranged from 40 milliseconds to 170 milliseconds with lower mean QT dispersion in early thrombolysed group than in the late thrombolysed group and the correlation was statistically significant which is very much in accordance with the previous studies. The maximum mean QT interval is high before and after thrombolysis & on the second day in the late group when compared to early group and is statistically significant ($p < 0.05$).

In our study QT dispersion was high at the time of admission before and after thrombolysis & on day 2, QT dispersion was highest. Thereafter, it was found to decrease in course of time and stabilising by 5^{th} day. The correlation between the QTd and time of reperfusion is statistically significant ($p<0.05$) in those taken before & after thrombolysis and on 2^{nd}

day. There was no increase in the QT dispersion after 5th day and not much of difference in the QT dispersion between 5th day and 6th week ECG.

TABLE 11: COMPARING MEAN QT MAXIMUM AND QT MINIMUM IN EARLY AND LATE GROUP AT DIFFERENT TIME.

Time of ECG	Early reperfused group		Late reperfused group	
	QT max	QT min	QT max	QT min
Before thrombolysis	0.4727	0.4220	0.5153	0.4163
After thrombolysis	0.4710	0.4230	0.5143	0.4370
2nd day	o.5233	0.4080	0.5930	0.4213
5th day	0.4647	0.419	0.4790	0.425

Yashuhiro Endoh, Hiroshi Kasanuki et al studied ***Influence of early coronary reperfusion on QT dispersion in acute myocardial infarction.*** Similar to our study they studied in 51 patients out of which 28 were re-canalised early and rest 23 were re-canalised late, study revealed significant QT dispersion reduction between acute and recovery phase. The incidence of PVC and arrhythmias were also reduced in the early group.

In summary they opined early coronary reperfusion reduces electrophysiological instability by reducing QT dispersion.

STUDIES	No. OF CASES	EARLY REPERFUSION QTd	LATE REPERFUSION QTd
Yashuhiro Endoh, Hiroshi Kasanuki et al	51 cases	28 cases 38 ± 12 ms	23 cases 50 ± 15 ms
Our study	60 cases	30 cases 43.3 ± 19 ms	30 cases 54 ± 25.9 ms

Yet another study on ***QT dispersion and thrombolytic therapy in acute myocardial infarction*** by Prabhu Shankar. S et al showed benefit of early successful thrombolysis in decreasing QTd and ventricular arrhythmias. They showed reduction in QTd among early thrombolysed cases.

Comparison study of QT dispersion between primary coronary angioplasty and thrombolysis by Cavusoglu Y, Gorenek B involving 42 patients showed better reduction of QT dispersion in those who underwent primary coronary angioplasty compared to those who were thrombolysed, and higher QTd was seen among those with anterior wall MI compared to inferior wall MI.

In summary QT dispersion is higher in AMI and successful reperfusion either by thrombolysis or PCI reduces QT dispersion, ventricular inhomogenecity and occurrence of arrhythmias.

In our study, ventricular arrhythmias occurred in 2 cases in early reperfused group and in six cases of late reperfused group. There was higher QT dispersion in cases with arrhythmias comparing to those without in early reperfused group which was also statistically significant in those taken before and after thrombolysis, but there was no significant QT dispersion prolongation in late group. A higher incidence of ventricular arrhythmias among late reperfused group is seen, indicating earlier successful thrombolysis results in decreasing the occurrence of arrhythmias.

QT dispersion among successful and failed cases

In our study there were totally 4 cases which were considered as treatment failure based on clinical grounds and ECG criteria i,e with persistent chest pain and reduction of ST segment elevation not more than 50% at 90^{th} minute after thrombolysis. Even though mean QT dispersion was higher among failed cases it was not statistically significant ($p>0.05$) in the earlier period post thrombolysis. However with recovery (i.e. on 5^{th} day post thrombolysis), the QTd was significantly lower in those with successful thrombolysis.

LIMITATIONS OF THE STUDY

1. Sample size was small so further studies with bigger sample size has to be done to further verify the results.
2. Our study has excluded AMI with bundle branch block and atrial fibrillation and this may have produced an underestimation of arrhythmias and mortality.

CONCLUSION

1. QT dispersion was higher among those who were reperfused later than 3 hours than those who were reperfused earlier.
2. Incidence of arrhythmias was high among late group than early group indicating early successful thrombolysis reduces occurrence of arrhythmias.
3. QT dispersion was lower in those with successful thrombolysis during the recovery phase

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LIST OF ABBREVIATIONS USED

STEMI	-	ST segment elevation myocardial infarction
AMI	-	Acute myocardial infarction
MI	-	Myocardial infarction
QTd	-	QT dispersion
PCI	-	Percutaneous coronary intervention
ACS	-	Acute coronary syndrome
ECG	-	Electrocardiogram
CABG	-	Coronary artery bypass graft
NSTEMI	-	Non ST segment elevation myocardial infarction
WHO	-	World health organisation
CVD	-	Cerebrovascular disease
CHD	-	Coronary artery disease
HDL-c	-	High density lipoprotein cholesterol
hs CRP	-	High sensitivity C–reactive protein
LDL-c	-	Low density lipoprotein cholesterol
TGL	-	triglycerides
AHA	-	American heart disease
LVH	-	Left ventricular hypertrophy
ADP	-	Adenosine diphosphate
LBBB	-	Left bundle branch block
UA	-	Unstable angina
TIMI	-	Thrombolysis in myocardial infarction
JVP	-	Jugular venous pressure
RVMI	-	Right ventricular myocardial infarction
RVI	-	Right ventricular infarction
IABP	-	Intra aortic balloon pump

VSR	-	ventricular septal rupture
ACE	-	Angiotensin converting enzyme
aPTT	-	Activated partial thromboplastin time
EAD	-	Early after depolarisation
Tdp	-	torsades de pointes
AP	-	Action potential
IRA	-	Infarct related artery
CPK	-	Creatine phosphokinase
MAP	-	Monophasic action potential
INR	-	International normalised ratio
VT	-	Ventricular tachycardia
PVC	-	Premature ventricular complex
H-FABP	-	Heart type fatty acid binding protein
IMA	-	Ischemic modified protein
trop-t	-	Troponin t

PROFORMA

NAME:

AGE:

SEX:

ADDRESS: O.P/I.P No.:

D.O.A: D.O.D.:

PRESENTING COMPLAINTS:

Duration of chest pain –

Past history –

Drug history -

SIGNS:

GENERAL

Built:	Pallor:	Icterus:
Pedal oedema:	Fever:	Hydration:
Clubbing	PR:	BP:

CVS:

RS:

PER ABDOMEN:

CNS:

INVESTIGATIONS

Blood:

Complete haemogram - Hb,TC, DC, ESR

Blood – Sugar urea and Serum creatinine

Serum electrolytes - Na, K

Blood CPKMB

Urine: Routine - alb, sugar, deposits.

Imaging:

X-ray chest P.A.

ECG: Rate –

Rhythm –

Axis –

Abnormality in waves –

QT max, QTmin –

QTc max, QTc min –

QT dispersion in milliseconds –

ECG impression – before thrombolysis –

After thrombolysis –

2^{nd} day –

5^{th} day –

6^{th} week –

Treatment:

1. Whether thrombolysed or not
2. Treatment outcome – successful/ failed
3. Occurance of arrhythmias – yes/no.

EARLY REPERFUSION GROUP

Sl.no	Name	Age	Sex	smoking	SHT	DM	Previous MI	Time of presentation	Type of MI	Thrmbolysis<3hrs	Successful thrombolysis	Failed thrombolysis	Occurance of arrythmias
1	Murugan	55	Male	yes	no	yes	no	2.5	AWMI	yes	yes	no	no
2	Ellappan	62	Male	yes	yes	no	no	1.8	IWMI	YES	yes	no	no
3	Muniammal	60	female	no	no	yes	no	2	ASMI	yes	yes	no	no
4	Joseph	45	male	no	no	yes	no	2.3	ASMI	yes	yes	no	no
5	Devi	52	female	no	yes	no	no	1.2	LWMI	yes	yes	no	no
6	Anbalagan	48	male	yes	no	no	no	2.9	AWMI	yes	no	yes	no
7	Dhanalakshmi	68	female	no	yes	yes	no	1.9	IWMI+RVMI	yes	yes	no	no
8	Bhuvaneshwari	60	female	no	no	no	no	2.3	ASMI	yes	yes	no	no
9	Tangappan	50	male	yes	no	no	no	2	IWMI	yes	yes	no	no
10	Saravanan	32	Male	yes	no	no	no	2.8	AWMI	yes	yes	no	no
11	Parthasarathy	67	male	no	yes	no	no	1.5	ASMI	yes	yes	no	no
12	Dhanabhakiyam	72	female	no	yes	yes	no	3	EXT AWMI	yes	yes	no	no
13	Kumaran	42	male	yes	no	no	no	2.1	AWMI	yes	no	yes	no
14	Gandhimathi	65	female	no	no	no	no	3	ASMI	yes	yes	no	no
15	Kodandaraman	62	male	yes	yes	no	yes	2.2	IWMI	yes	yes	no	no
16	sheriff ahmed	56	male	yes	no	yes	no	1.6	AWMI	yes	yes	no	no
17	Tamilselvi	67	female	no	no	no	no	2.3	IWMI	yes	yes	no	no
18	Rajan	44	Male	yes	yes	yes	no	2	AWMI	yes	yes	no	yes
19	Selvam	52	Male	yes	yes	no	no	2.5	ASMI	yes	yes	no	no
20	Vishwanathan	56	male	yes	no	no	no	2.5	LWMI	yes	yes	no	no
21	Ammakannu	66	female	no	no	yes	no	2	ASMI	yes	yes	no	no
22	Rajakumar	27	male	no	no	no	no	1.5	IWMI	yes	yes	no	no
23	Ibrahim	65	male	yes	no	no	no	2.9	IWMI+PWMI+RVMI	yes	yes	no	no
24	Masilamani	60	female	no	no	yes	no	3	ASMI	yes	yes	no	no
25	Rajendran	68	Male	yes	no	no	no	1	ASMI	yes	yes	no	no
26	Vijayan	52	Male	no	yes	no	no	1.8	IWMI	yes	yes	no	no
27	Sridaran	50	Male	yes	no	yes	no	1.5	ASMI	yes	yes	no	yes
28	Meenakhi	48	female	no	no	no	no	2.5	LWMI	yes	yes	no	no
29	Rajeshwari	57	female	no	no	no	no	2.3	IWMI	yes	yes	no	no
30	Pachayammal	66	female	no	no	yes	no	2	AWMI	yes	yes	no	no

QTc INTERVAL AND QT DISPERSION VALUES OF EARLY REPERFUSION GROUP

SL:NO	CORRECTED QT INTERVAL=QTc (IN SECONDS)										QT DISPERSION (IN MILLI SECONDS)				
	BEFORE THROMBOLYSIS		AFTER THROMBOLYSIS		2^{ND} DAY		5^{TH} DAY		6^{TH} WEEK		BEFORE THROMBOLYSIS	AFTER THROMBOLYSIS	2^{ND} DAY	5^{TH} DAY	6^{TH} WEEK
	MAX	MIN	MAX	MIN	MAX	MIN	MAX	MIN	MAX	MIN					
1	0.45	0.38	0.41	0.35	0.53	0.37	0.43	0.4	0.45	0.42	70	60	160	30	30
2	0.44	0.41	0.47	0.42	0.53	0.4	0.47	0.43	0.46	0.42	30	50	130	40	40
3	0.48	0.43	0.45	0.4	0.47	0.38	0.43	0.36	0.47	0.41	50	50	90	70	60
4	0.49	0.47	0.46	0.41	0.53	0.37	0.46	0.42	0.47	0.41	20	50	160	40	60
5	0.47	0.41	0.46	0.43	0.54	0.41	0.47	0.41	0.45	0.42	60	30	130	60	30
6	0.45	0.43	0.44	0.41	0.54	0.42	0.53	0.45	0.45	0.4	20	40	120	80	50
7	0.47	0.43	0.49	0.43	0.45	0.36	0.49	0.41	0.47	0.42	40	60	90	80	50
8	0.49	0.45	0.46	0.43	0.51	0.43	0.47	0.42	0.45	0.41	40	30	80	50	40
9	0.47	0.41	0.48	0.43	0.5	0.41	0.48	0.42	0.47	0.44	60	50	90	60	30
10	0.43	0.35	0.42	0.33	0.36	0.35	0.41	0.35	0.45	0.42	80	90	10	60	30
11	0.51	0.45	0.48	0.45	0.53	0.46	0.44	0.4	0.49	0.43	60	30	70	40	60
12	0.49	0.45	0.46	0.43	0.47	0.41	0.47	0.4	0.45	0.43	30	30	60	30	20
13	0.49	0.41	0.45	0.43	0.51	0.43	0.52	0.43	0.49	0.43	80	20	80	90	60
14	0.43	0.4	0.47	0.43	0.53	0.38	0.45	0.43	0.42	0.36	30	40	150	20	60
15	0.47	0.44	0.49	0.46	0.54	0.41	0.47	0.41	0.47	0.39	30	30	130	60	80
16	0.5	0.45	0.51	0.43	0.53	0.4	0.47	0.44	0.45	0.43	50	80	130	30	20
17	0.47	0.43	0.45	0.43	0.51	0.43	0.45	0.41	0.46	0.39	40	30	80	30	70
18	0.5	0.41	0.52	0.43	0.66	0.49	0.52	0.48	0.47	0.4	90	90	170	40	70
19	0.53	0.45	0.48	0.45	0.55	0.41	0.46	0.41	0.45	0.38	80	30	60	50	70
20	0.47	0.43	0.47	0.41	0.53	0.43	0.47	0.43	0.48	0.43	40	60	100	40	50
21	0.45	0.4	0.49	0.44	0.56	0.43	0.45	0.43	0.44	0.39	50	40	130	20	50
22	0.47	0.41	0.49	0.44	0.46	0.35	0.45	0.44	0.47	0.43	60	50	110	10	40
23	0.49	0.45	0.48	0.42	0.54	0.42	0.47	0.42	0.47	0.45	40	60	120	50	20
24	0.47	0.43	0.46	0.43	0.51	0.42	0.46	0.44	0.49	0.42	40	30	90	20	70
25	0.47	0.41	0.49	0.44	0.53	0.4	0.47	0.41	0.47	0.39	60	50	130	60	80
26	0.44	0.39	0.45	0.43	0.51	0.43	0.49	0.44	0.49	0.43	50	20	80	50	60
27	0.5	0.42	0.55	0.44	0.64	0.49	0.45	0.41	0.46	0.44	80	110	150	40	20
28	0.45	0.4	0.47	0.42	0.53	0.42	0.45	0.42	0.44	0.41	50	50	90	30	30
29	0.47	0.44	0.48	0.43	0.56	0.36	0.44	0.41	0.47	0.43	30	50	200	30	40
30	0.47	0.42	0.45	0.41	0.54	0.37	0.45	0.43	0.47	0.42	50	40	170	20	50

LATE REPERFUSION GROUP

Sl.no	Name	Age	Sex	Smoking	SHT	DM	Previous MI	Time of presentation	Type of MI	Thrombolysed	Successful	Failed	Arrythmias
1	Ibrahim	57	male	yes	no	no	no	7	IWMI	yes	yes	no	no
2	Dakshinamoorthy	66	male	yes	yes	no	no	6.2	AWMI	yes	yes	no	yes
3	Murthy	48	male	no	yes	yes	no	5.6	AWMI	yes	yes	no	no
4	Meenakshi	54	female	no	no	yes	no	9.4	ASMI	yes	yes	no	no
5	Babu	38	male	yes	no	no	no	5	Ext AWMI	yes	yes	no	no
6	Jayanthi	66	female	no	no	yes	no	10	IWMI	yes	yes	no	no
7	Durai	52	male	yes	yes	no	yes	8.8	LWMI	yes	yes	no	no
8	Rajammal	45	female	no	no	no	no	7.6	ASMI	yes	yes	no	yes
9	Nagraj	67	male	yes	no	yes	no	6	IWMI+PWMI	yes	yes	no	no
10	Saroja	69	female	no	no	no	no	8.8	IWMI	yes	yes	no	no
11	Yashodha	63	female	no	no	yes	no	7.2	ASMI	yes	yes	no	no
12	Kamalakannan	58	male	yes	no	no	no	8.8	ASMI	yes	yes	no	no
13	Kantharaj	62	male	yes	no	yes	no	9.2	IWMI	yes	yes	no	no
14	Dhanam	57	female	no	yes	yes	yes	6.3	AWMI	yes	no	yes	yes
15	Robert	43	male	yes	yes	no	no	7.3	ASMI	yes	yes	no	no
16	Elumalai	65	male	yes	no	yes	no	5	IWMI+RVMI	yes	yes	no	no
17	Valli	54	female	no	no	yes	no	6.7	AWMI	yes	yes	no	no
18	Chelldurai	62	male	no	yes	no	no	8.3	IWMI	yes	yes	no	no
19	Somasundaram	55	male	no	no	yes	no	9.2	ASMI	yes	yes	no	yes
20	Kuppammal	67	female	no	no	no	no	5.3	IWMI+PWMI+RVMI	yes	yes	no	no
21	Jayakumar	47	male	yes	no	no	no	6.7	ASMI	yes	yes	no	no
22	Gunasundari	56	female	no	no	no	no	7	IWMI	yes	yes	no	yes
23	Appan raj	62	male	yes	no	yes	no	4.5	ASMI	yes	no	yes	no
24	Mayilvahanan	60	male	yes	yes	no	no	6.3	Ext AWMI	yes	yes	no	no
25	Senthamari	65	female	no	no	yes	no	7	IWMI	yes	yes	no	no
26	Shanmugasundaram	51	male	yes	no	no	no	9.5	ASMI	yes	yes	no	no
27	Kannan	67	male	yes	no	yes	no	5.6	AWMI	yes	yes	no	yes
28	Dhakshayini	58	female	no	yes	yes	no	7.2	ASMI	yes	yes	no	no
29	Malliga	63	female	no	yes	no	no	6.4	IWMI	yes	yes	no	no
30	Jayaraman	60	male	yes	no	no	no	11	LWMI	yes	yes	no	no

QTc INTERVAL AND QT DISPERSION VALUES OF LATE REPERFUSION GROUP

S:NO	CORRECTED QT INTERVAL= QTc (IN SEONDS)										QT DISPERSION (IN MILLI SECONDS)				
	BEFORE THROMBOLYSIS		AFTER THROMBOLYSIS		2^{ND} DAY		5^{TH} DAY		6^{TH} WEEK		BEFORE THROMBOLYSIS	AFTER THROMBOLYSIS	2^{ND} DAY	5^{TH} DAY	6^{TH} WEEK
	MAX	MIN	MAX	MIN	MAX	MIN	MAX	MIN	MAX	MIN					
1	0.47	0.43	0.49	0.43	0.56	0.43	0.47	0.43	0.48	0.43	40	60	130	40	50
2	0.53	0.47	0.5	0.46	0.62	0.4	0.47	0.39	0.48	0.39	60	40	220	80	90
3	0.59	0.47	0.57	0.49	0.63	0.49	0.56	0.43	0.45	0.45	120	80	140	130	0
4	0.42	0.38	0.57	0.44	0.66	0.5	0.45	0.39	0.46	0.42	40	100	190	60	40
5	0.52	0.38	0.51	0.41	0.57	0.4	0.46	0.42	0.45	0.41	140	100	170	40	40
6	0.55	0.44	0.56	0.47	0.6	0.43	0.47	0.43	0.49	0.45	110	90	170	40	40
7	0.54	0.39	0.56	0.44	0.59	0.31	0.51	0.42	0.49	0.43	150	120	280	90	60
8	0.56	0.45	0.63	0.47	0.49	0.45	0.51	0.45	0.47	0.43	90	160	40	60	40
9	0.55	0.39	0.56	0.41	0.66	0.41	0.47	0.41	0.47	0.43	160	150	150	60	40
10	0.57	0.43	0.57	0.45	0.6	0.43	0.48	0.45	0.46	0.42	140	120	170	30	40
11	0.5	0.43	0.59	0.42	0.57	0.35	0.47	0.43	0.45	0.39	70	170	220	40	60
12	0.42	0.35	0.47	0.43	0.56	0.43	0.45	0.41	0.47	0.4	70	40	130	40	30
13	0.51	0.44	0.56	0.42	0.66	0.49	0.48	0.4	0.49	0.41	70	140	170	80	80
14	0.57	0.45	0.66	0.47	0.61	0.42	0.46	0.44	0.47	0.42	120	190	190	20	50
15	0.5	0.43	0.54	0.47	0.66	0.47	0.51	0.46	0.49	0.45	70	70	190	50	40
16	0.51	0.43	0.57	0.43	0.63	0.41	0.45	0.43	0.47	0.42	120	130	180	20	40
17	0.51	0.43	0.53	0.45	0.61	0.39	0.49	0.45	0.47	0.47	80	80	120	40	0
18	0.56	0.44	0.6	0.46	0.64	0.46	0.49	0.43	0.46	0.43	120	140	180	60	30
19	0.45	0.4	0.47	0.42	0.57	0.34	0.45	0.39	0.46	0.43	50	50	230	60	30
20	0.48	0.39	0.5	0.42	0.52	0.4	0.45	0.36	0.46	0.41	90	80	120	90	50
21	0.42	0.38	0.45	0.45	0.48	0.42	0.45	0.41	0.47	0.43	40	0	60	40	40
22	0.5	0.43	0.55	0.4	0.6	0.41	0.51	0.46	0.47	0.45	70	150	190	50	20
23	0.56	0.48	0.52	0.47	0.56	0.39	0.53	0.43	0.5	0.41	80	50	170	100	90
24	0.52	0.42	0.56	0.48	0.65	0.56	0.47	0.43	0.45	0.41	100	80	180	70	40
25	0.54	0.4	0.52	0.4	0.57	0.39	0.47	0.4	0.45	0.41	140	120	180	30	40
26	0.52	0.4	0.54	0.45	0.58	0.35	0.45	0.41	0.49	0.42	120	90	230	40	70
27	0.53	0.43	0.52	0.39	0.6	0.35	0.5	0.43	0.46	0.42	100	130	250	70	40
28	0.58	0.45	0.56	0.45	0.6	0.46	0.49	0.45	0.48	0.46	130	110	140	40	20
29	0.55	0.37	0.56	0.35	0.6	0.49	0.52	0.49	0.49	0.47	180	210	110	30	20
30	0.43	0.37	0.45	0.41	0.54	0.41	0.43	0.41	0.45	0.43	60	40	130	20	20

ETHICAL COMMITTEE
GOVT. KILPAUK MEDICAL COLLEGE, KILPAUK,
CHENNAI- 10.
Venue: PANAGAL HALL, KMC
Dt: 01.02.2011

CHAIRPERSON
Prof. Dr.V.KANAGASABAI, MD.,
Dean
Govt. Kilpauk Medical College, Chennai-10
Sub: Ethical Committee project work - approved – regarding.
Ref: Lr.No.3944/Audit/E1/09 Dt. 30.11.2010

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With above reference, the Institutional Ethical committee meeting for the following students was conducted at our Institution on 01.02.2011.

S.NO.	Name	Topic
1.	Dr.Navin Kumar, MS(Ortho), PG., Govt. Royapettah Hospital, Chennai.	1.To Identify a Safe Zone to approach proximal Humerus 2.To study Anatomical relations of Axillary nerve, its course & its Variations
2.	Dr.T.Satheesh Kumar, D.Ortho., PG., Govt. Royapettah Hospital, Chennai	Heriditary Multiple Exostosis
3.	Dr.J. Jeya Shambavi, MD(Pathology), PG., Govt. Kilpauk Medical College, Chennai-10	Clinicopathological Histomorphological and Immunohistochemical Study of Neuroendocrine Tumors of GIT
4.	Dr.L. R. Saranya. MD., (Paed.)PG., Govt. Kilpauk Medical College, Chennai-10	Cord Blood Zinc Level in Term-Small for Gestational Age Neonates
5.	Dr.A.Satheesh Kumar, MS(ENT), PG., Kilpauk Medical College, Chennai	Study on Cases of Chronic Suppurative Otitis Media in Tubo Tympanic Type Due to Sinusitis as Focal Sepsis
6.	R.Prathiban (Msc.,Physiology), PG., Student, The TN. Dr.MGR Medical University,Chennai-32	Prevalence of Cardiac Dysautonomia in Type I Diabetes mellitus
7.	B. Manikandan, (Msc., Physiology), PG., Student, The TN Dr.M.G.R. Medical University, Chennai-32.	A Comparative Study of Left Ventricular Structure and Function in Obese and Non Obese Subjects
8.	G. Selvakumar, (MSc., Physiology), PG., Student, The TN Dr.M.G.R. Medical University, Chennai-32.	A Study of the Intraocular Pressure In Patients with Diabetic Normotensive, Diabetic Hypertensive and Normal Subjects

9.	R. Ragulji, (Msc.,Physiology), PG., The TN Dr.MGR Medical University, Chennai-32.	A Study of Pulmonary function in insulin dependent diabetes mellitus
10.	V.M. Jenila Veny,(MscPhysiology), PG. The TN Dr.MGR Medical University, Chennai-32	Cardiovascular Autonomic Dysfunction in Chronic Kidney Disease
11.	Dr.G. Lakshmi, MD(O&G), PG., Govt. Kilpauk Medical College,Chennai-10	A Study of Association of Thyroid Disorders in Abnormal Uterine Bleeding
12.	Dr.R. Harini, MD(O&G), PG., Kilpauk Medical College, Chennai	Single Dose Antibacterial treatment for Asymptomatic Bacteriuria in Pregnancy
13.	Dr.E.Geetha, MD(O&G), PG., Govt. Kilpauk Medical College, Chennai-10	A Study of the incidence course of Pregnancy and Pregnancy outcome in Obstetric Cholestasis and to evaluate the efficiency of UDCA in relieving the Symptoms and Improving the Perinatal outcome in these Patients
14.	Dr.S. Nithya, MD(O&G), PG., Govt. Kilpauk Medical College, Chennai-10	Prospective Study of Prevalence of diabetes Mellitus, Thyroid Dysfunction and Hyperprolactinemia in Recurrent Pregnancy loss
15.	Dr.Mohideen Fathima, MD(O&G), PG., Govt. Kilpauk Medical College, Chennai-10	A Study of evaluation of multi system changes in Gestational hypertension / severe pre-eclamptic/eclampasia patients
16.	Dr.M.Padma Priya, MD(O&G), PG., Kilpauk Medical College, Chennai	Dyslipidemia as a Predictor of PIH
17.	Mrs.G. Savitha, (Msc.,Medical Bio Chemistry), TN Dr.M.G.R.Medical University, Chennai-32.	Association of subclinical hypothyroidism in metabolic syndrome patients
18.	Dr.K. Bharadhwaj, MD(G.M.), PG., Kilpauk Medical College, Ch-10	A Study on Peripheral Vascular Disease in Type 2 Diabetes Mellitus
19.	Dr.B.Priya, MD(G.M.), PG	Study of Serum Bilirubin Concentration in Established Coronary Artery Disease
20.	Dr.R.Hema, MD(G.M.), PG.,	Study of Troponin I level in Supraventricular Tachycardia in Non Cad Patients
21.	Dr.P.Manoj Kumar, MD(G.M.), PG., Kilpauk Medical College, Ch-10	A Study on Pulmonary Functions in Type 2 Diabetes Mellitus
22.	Dr.M.Dhanasekar, MD(G.M.), PG.,	Prognostic Risk Stratification of Acute Coronary Syndrome – Role of Highly Sencitive – Reactive Protien
23.	Dr.N. Karthik, MD(G.M.), PG., Govt.Kilpauk Medical College, Chennai-10	A Study of Comparison of QT Dispersion in Acute Myocardial Infraction Between Early Reperfusion and Late Reperfusion Therapy

24.	Dr.H. Anuradha, MD(G.M.), PG., Kilpauk Medical College, Ch-10	A Study of Stress Hyperglycemia in Moderate Degree Burns
25	Dr. V. Nandakumar, MD(G.M.), PG.,	A Prospective Study of Clinical Profile of Emphysematous Pyelonephritis in Type Two Diabetes Mellitus
26.	Dr.S.Sasikumar, MS(G.S.), PG., Govt. Royapettah Hospital, Chennai	A Study of Unusual Presentations of Appendicitis.
27.	Dr.S.R.Padmanabhan, MS(GS), PG., Govt. Royapettah Hospital, Chennai	A Comparative Study Between Autologous Platelet Rich Plasma and Saline Dressing for Diabetic Ulcer
28.	Dr.C.Rose, Scientist-G and Head, Biotechnology, Central Leather Institute, Chennai.	Wound healing efficacy of the chitosan – containing collagenous biomaterial, on burn wound
29.	E.K. Lavanya,B.Tech, Biotechnology, PG., Prathyusha Institute of Technology and Management, Tiruvallur.	Isolation and Characterization of Bacterial Pathogens from Eye Infection

We are glad to inform you that at the Ethical Committee meeting, the documents were discussed and the above short term projects are Ethically approved.

4/2/11

CHAIRPERSON
DEAN
Govt. Kilpauk Medical College.
Chennai-10.

To: The Individuals