"A STUDY ON QT DISPERSION IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION AND ITS CORRELATION WITH ECHO AND ANGIOGRAM FINDINGS"

BY

DR . V AMULYA REDDY

DISSERTATION SUBMITTED TO
SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION & RESEARCH , TAMAKA, KOLAR, KARNATAKA

IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF

DOCTOR OF MEDICINE
IN
GENERAL MEDICINE

UNDER THE GUIDANCE OF
Dr PRABHAKAR K
PRINCIPAL AND PROFESSOR
DEPT OF GENERAL MEDICINE

UNDER THE CO-GUIDANCE OF
Dr. YASWANTH A L
MBBS MD DM
INTERVENTIONAL CARDIOLOGIST

DEPARTMENT OF GENERAL MEDICINE
SRI DEVARAJ URS MEDICAL COLLEGE
TAMAKA, KOLAR - 563103
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INTERVENTIONAL CARDIOLOGY
Sri Devaraj Urs Medical College,
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DEPARTMENT OF MEDICINE
SDUMC, KOLAR

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PRIOR PERMISSION TO START OF STUDY

The Institutional Ethics Committee of Sri Devaraj Urs Medical College, Tamaka, Kolar has examined and unanimously approved the synopsis entitled **" A Study On Qt Dispersion In Patients With Acute Myocardial Infarction And Its Correlation With Echo And Angiogram Findings"** being investigated by **Dr.Vulavapalle Amulya, Dr Prabhakar K & Dr.Yashwanth L**[1] in the Departments of General Medicine & Narayana Health Heart Centre, R. L. Jalappa Hospital[1] at Sri Devaraj Urs Medical College, Tamaka, Kolar. **Permission is granted by the Ethics Committee to start the study.**

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DR. V AMULYA REDDY

Post graduate,
Department of General Medicine.

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION & RESEARCH
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A STUDY ON QT DISPERSION IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION AND ITS CORRELATION WITH ECHO AND ANGIOGRAM FINDINGS

ABSTRACT

Background: Myocardial infarction is an important public health concern that increases mortality rates and disability frequency. The most common cause of myocardial infarction is coronary atherosclerosis, a chronic disease that can evolve from stable to unstable. QT dispersion (QTD) assesses cardiac repolarization heterogeneity by comparing maximal and minimal QT intervals on a 12-lead electrocardiogram. The QTD lowers with reperfusion treatment. The ventricular action potential should be equal throughout the heart in order to treat infarcted arteries with fibrinolytic medicines and PPCI. Research findings on QTD reduction varied between the two perfusion strategies employed in STEMI patient therapy. This study was primarily designed to investigate QT dispersion throughout various follow-up durations.

Material and Methods: A hospital-based prospective analytical study was conducted on 50 patients admitted to the Cardiac Critical Care Unit with ACUTE MYOCARDIAL INFARCTION. Before completing a comprehensive physical examination using the specified proforma, all patients were informed and given written consent. All patients had various tests performed, including a complete blood count, renal and liver function tests, and an ECG evaluation. Patients had their 12-lead ECGs recorded at admission and again at 24 hours, with the measurement equipment set at 1mV=10mm and the recording speed set to 25mm/sec. QT measurement begins with the starting of the R wave and concludes when the T wave is completed; nevertheless, the tangent technique is the most effective tool for identifying T wave termination.

Results: The study included 45 percent of the individuals tested, who were between the ages of 61 and 70. Female participants accounted for 54.5% of the research dataset. Approximately 18.2% had previously smoked. Alcoholics accounted for around 11.4% of all patients in this study. This study found that 40.9% of participants had hypertension. According to the findings of this study, 9.1% of participants exhibited left ventricular hypertrophy on their ECG. This study found that unstable angina developed in 47.7% of heart attack patients, STEMI in 31.8%, and NSTEMI in 20.5%. Complications occurred in 31.8% of the cases studied. The most prevalent consequences were heart failure, hypotension, and ventricular arrhythmia. On day zero, the mean QT interval in NSTEMI was 160.3, in STEMI it was 153.43, and in unstable angina it was 121.5. Based on angiographic evaluation, single vessel disease was seen in 38.6%

of patients, while double and triple vessel disease were seen in 38.6% and 36.4% of cases, respectively. On days 0 and 1, there was a significant negative correlation between the ejection percent and QT dispersion. On days 2 and 7, it was negative but not really visible. The findings revealed that patients with right wall motion anomalies had higher QT dispersion. Right wall motion anomalies resulted in statistically significant differences on testing days 0, 1, and 7. Triple vessel disease caused larger QT dispersion than single and double vascular disease conditions. Triple vessel disease had larger QT dispersion than single or double vessel disease, with a significant statistical link between the two.

Conclusion: This study concludes that, STEMI and NSTEMI subjects had a larger QT dispersion than those with unstable angina. The QT dispersion reduced with follow-up and was lowest by day 7.

Keywords: Acute myocardial infarction, QT dispersion, Reperfusion Therapy, Ejection fraction,Coronaryangiography

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ABBREVIATIONS

Abbreviations (in Alphabetical order)	
ACS	Acute Coronary Syndrome
AIDS	Acquired Immunodeficiency Syndrome
ADP	Adenosine Diphosphate
AMI	Acute Myocardial Infarction
AST	Aspartate transaminase
AWMI	Anterior Wall Myocardial Infarction
CABG	Coronary Artery Bypass Grafting
CAD	Coronary Artery Disease
CHB	Complete Heart Block
CHF	Congestive Heart Failure
CK-MB	Creatinine Kinase - MB
CK-MM	Creatinine Kinase - MM
CPK	Creatinine Phosphokinase
CRP	C Reactive Protein
CT	Computed Tomography
CVD	Cardiovascular Diseases
ECG	Electroicardiogram
ECHO	Echocardiogram
HBDH	Hydroxybutyrate Dehydrogenase
HDL	High Density Lipoprotein
hFABP	Heart – Type of Fatty Acid – Binding Protein
IWMI	Inferior Wall Myocardial Infarction
IHD	Ischemic Heart Diseases
LAD	Left anterior Descending artery
LDH	Lactate Dehydrogenase
LDL	Low density Lipoprotein
LVEF	Left Ventricular Ejection Fraction
MI	Myocardial Infarction
NSTEMI	Non ST segment elevation myocardial infarction
PPCI	Primary Percutaneous Coronary Intervention
PCI	Percutaneous Coronary Intervention
PSVT	Paroxysmal Supra Ventricular Tachycardia
QTD	QT Dispersion
RTSD	Right Stem Depression
SGOT	Serum Glutamic – Oxaloacetic Transaminase
STEMI	ST segment elevation myocardial infarction
TnT	Troponin - T
TnI	Troponin – I
TnC	Troponin - C
UA	Unstable Angina
VPC	Ventricular Primary Complex
WHO	World Health Organization

INTRODUCTION

INTRODUCTION

Myocardial infarction functions as a major public health issue that increases mortality rates and disability frequencies. Coronary atherosclerosis stands as the dominant factor that trigger myocardial infarction., a chronic condition that can progress from stable to unstable. Myocardial infarction is caused by the vascular wall's triggered inflammation during the unstable phase. Myocardial infarction occurs because ischemia causes death of cardiac myocytes that stems from inadequate blood supply-demand ratio. The term "myocardial infarction" refers to myocardial necrosis caused by an abrupt halt of coronary blood flow.(1)

Chronic illness like myocardial infarction can be a modest occurrence or perhaps go unnoticed, but it can also be a big disaster that causes abrupt mortality or serious hemodynamic decline. Additionally, it could be the initial sign of Heart disease patients and those who already have coronary artery disease are more likely to experience another heart event or their disease recurs.(2)

During exercise or resting periods the symptoms primarily appear as pain in chest, jaw, upper extremities and epigastrium. Dyspnea, diaphoresis, nausea, and syncope may accompany the disease's discomfort, which is not positional, localized, or impacted by movement of the area. These symptoms can be mistakenly attributed to gastrointestinal, neurological, respiratory, or musculoskeletal conditions because they are not unique to myocardial ischemia. Myocardial infarction can be identified only by Electrocardiogram, biomarker elevations or cardiac imaging, and it can happen with or without unusual symptoms. In a clinical context, ischemia is typically detected by the ECG and the patient's medical history. An essential component of the diagnosis process for patients with suspected Dyspnea, diaphoresis, nausea, and syncope may accompany the disease's discomfort, which is not positional, localized, or impacted by movement of the area.(3,4)

The 1979 WHO guidelines specify myocardial infarction (MI) diagnosis through any two probable or three definite criteria presented below(5):

1.The patient needs to report persistent type chest pain statements which exceed a 20-minute time span.

2. Serial ECG tracing changes.

3. Both creatine kinase-MB fraction and troponin show changes in serum cardiac indicators.

The WHO released updated criteria in 2000 which identified cardiac biomarkers as the primary diagnostic tool for myocardial infarction diagnosis.(6) With rising cardiac troponin levels, doctors can diagnose an MI from either typical symptoms together with ST elevation or depression or pathological Q waves or coronary intervention.

The ECG is still a crucial test for identifying both acute and chronic cardiac syndromes. The length of the ischemic process, the degree of ischemia, the topography, including anterior versus inferior, and the existence of other underlying abnormalities (such as pacemaker patterns, WPW syndrome, or Left Bundle Branch block) that can obscure or change the classic pattern are some of the variables that affect the results. During acute ischemia the ST segment expansion serves as the first and most reliable ECG indicator that arises from the present damage mechanism. Electrocardiogram stands as the most straightforward tool for diagnosis of acute myocardial infarction.(7)

During acute ECG assessment, ST segment depression is typically observed in leads reflecting heart regions not directly affected by infarction. In cases of inferior wall myocardial infarction, reciprocal depression is seen in leads I, aVL, and V2–V6. For anterior wall infarctions, it appears in leads II, III, and aVF. This pattern of reciprocal ST changes helps distinguish acute MI from other conditions that also present with widespread ST elevation.(8)

ST-elevation myocardial infarction (STEMI) patients risk death mainly because of arrhythmic complications during their initial hospital period. Numerous studies show Dispersion of repolarization is the most common cause of deadly STEMI arrhythmias. QT dispersion (QTD) measures cardiac repolarization heterogeneity by comparing maximal and minimal QT intervals on a 12-lead electrocardiogram. STEMI and acute ischemia extend the QT interval and QTD outcomes, according to current studies. In the early phases of acute myocardial infarction, cardiac activity variations alter ventricular excitability healing processes.(9–12)

The use of fibrinolytic therapy remains critical as a coronary artery reperfusion method despite PPCI being the preferred STEMI treatment for patients when PPCI is not available in a timely fashion. The QTD decreases after reperfusion therapy The ventricular action potential should become equal across the entire heart to enable infarct-related artery treatments between fibrinolytic medications and PPCI. Research findings about QTD reduction differ for both perfusion methods used in STEMI patient treatment. Researchers have not revealed the effects that different reperfusion procedures have on the occurrence of in-hospital arrhythmias.(13)

The assessment of QT dispersion represents a straightforward and affordable method which identifies ventricular arrhythmias before they develop.

The present study aims to link QT dispersion with myocardial infarct location and it determines both QT dispersion extent and its connection with ECHO and Angiogram findings for AMI patients.

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AIMS & OBJECTIVES

AIMS & OBJECTIVES OF THE STUDY

Objectives

- The QT dispersion should be measured in patients suffering from ACUTE MYOCARDIAL INFARCTION.
- The study seeks to evaluate QT dispersion together with infarct locations in patients with ACUTE MYOCARDIAL INFARCTION.
- The research aims to evaluate QT dispersion relationships with arrhythmia risks observed in patients experiencing ACUTE MYOCARDIAL INFARCTION.
- The evaluation measures the connection between QT dispersion values and ECHO results and angiographic findings.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

HISTORICAL PERSPECTIVES

According to William Heberden in 1772 he first stated a standardized description of angina pectoris. One hundred years after this description, Ludvig Hetoen came to the conclusion that coronary thrombosis, which is a result of sclerotic alterations in coronary arteries, is what causes myocardial infarction. Obrastzov and Strashesko provided the first myocardial infarction description that did not result in death in 1910. Based on the Russian study, James B. Herrick told American doctors about cardiac infarction symptoms two years later. Additionally, he underlined the value of electrocardiograms as a diagnostic tool for myocardial infarction. For the first time, he stressed the importance of "total bed rest" as a treatment. These methods were the accepted treatment for patients with AMI till the start of study. One hundred years after this description, Ludvig Hetoen came to the conclusion that coronary thrombosis, which is a result of sclerotic alterations in the coronary arteries, is what causes myocardial infarction. Obrastzov and Strashesko provided the first description of a myocardial infarction that did not result in death in 1910.(14–16)

EPIDEMIOLOGY

Over the past three decades, there has been several noteworthy research on the diagnosis and treatment of acute MI. Nevertheless, illness remains significant health issue in the developed world and is becoming more significant in emerging nations.(17) Arrhythmias, most commonly ventricular fibrillation, Acute myocardial infarction leads to over 40% of prehospital deaths when occurrences happen within the first hour of the event.(18)

In 2002, the World Health Organization estimated that ischemic heart disease accounted for 12.6 percent of all fatalities globally, making it the top cause of death in industrialized nations.(19)

In underdeveloped nations, it was ranked third, followed by AIDS and lower respiratory infections.(20) Every year, around 3 million people. The global incidence of STEMIs amounts to 22 million cases per year while NSTEMIs affect another 4 million patients.(21) Hospital data show that coronary heart disease causes death in one out of twenty patients in the USA. India's CVD stands as the main reason for deceased population numbers making it a leading contributor to death statistics.(22) Trace data indicates that CVD contributed to 32 percent of total deaths observed in India during 2007. The predicted increase of CVD deaths will rise to 1.59 million in 2000 preceding a further increase to 2.03 million in 2010.(23) However, CVD represents a recent surge as an epidemic in India. CVD is predicted to increase in mortality between 1985 and 2015, making it a serious health concern.(24,25)

According to the percentage of all deaths, mortality rates from cardiovascular disease The percentage of cardiovascular deaths per year ranges from 10% in Meghalaya to 49% in Punjab across different Indian states. CVD-associated deaths demonstrate their maximum levels in states like Tamil Nadu (36%), Andhra Pradesh (31%) and Punjab (49%) as well as Goa (42%). Each state shows a unique prevalence pattern among its dietary risk factors because of their unique territorial differences. In India, moderate physical activity is linked to a lower incidence of CVD; individuals who exercise have a risk that is less than half that of those who do not.(26)

CLINICAL FEATURES

The world's leading deaths stem from myocardial infarctions in men and women combined.19 Multiple factors increase death risk including heart disease background, age and smoking but also high triglycerides, LDL and low HDL with obesity, diabetes, kidney failure, hypertension, heart failure, excessive drinking and drug use (including cocaine/methamphetamine and persistent mental stress).(27, 28)

Cardiovascular death among both men and women stands as the worldwide leader because of myocardial infarction. The most dangerous risk elements for heart attack stem from cardiovascular disease background alongside aging patients who use tobacco products and possess elevated triglyceride and low-density lipoprotein amounts and decreased HDL levels and diabetic patients with hypertension along with obesity and chronic kidney disease and heart failure and alcohol abuse (mainly cocaine or methamphetamine use) and enduring elevated stress.

Cardiac patients typically experience declining symptoms at first rather than immediate onset because the symptoms evolve during several minutes. The hallmark symptom of heart attack is chest distress identified as intense pressure that feels like constriction or a squeezing feeling. Angina pectoris describes heart discomfort which arises when inadequate blood supply and oxygen reach the heart muscle during the condition of ischemia. Left arm pain serves as the primary sign of heart attack but the symptoms may equally match those of heartburn and spread to right arm or upper body areas like back and neck along with lower jaw and stomach.(29)

Research showed that the chest pain localization test in which patients place their hand in a fist on their sternum as Levine's sign possesses a weak value to predict the accurate diagnosis. Medical experts maintain that this physical indicator traditionally indicates signs of heart-associated chest pain.(30)

The medical condition shortness of breath develops when heart disease lowers left ventricle output until it reaches left ventricular failure and leads to pulmonary edema formation. The additional manifestations causing concern are palpitations and nausea with vomiting followed by lightheadedness and weakness and diaphoresis along with excessive sweating. These symptoms develop mostly due to the massive catecholamine release from the sympathetic nervous system after pain onset and heart failure-related changes in blood pressure. Ventricular fibrillation coupled with

sudden death represents the main fatal outcomes of MI while loss of consciousness emerges from failed cerebral perfusion and cardiogenic shock.(31)

The frequency of atypical symptoms runs higher among female elderly patients rather than male younger patients. The average number of symptoms women experience amounts to 2.6 whereas men report an average of 1.8 symptoms.(32)

According to studies, women frequently experience weariness, weakness, and dyspnea. It has been documented that symptoms such as fatigue, dyspnea, and sleep difficulties might appear up to one month prior to the actual clinically evident ischemia stroke. Compared to men, women's chest pain may not be as indicative of coronary ischemia.(33)

About one-fourth of MI occur without any symptoms, such as chest discomfort. Without a history of linked complaints, these cases may be found at autopsy, later on electrocardiograms, or by blood enzyme testing. The lack of nerve connection between the donor heart and host body prevents suitable heart transplantation for elderly patients with diabetes. and those who have undergone heart transplantation are more prone to experience a calm course. Diabetics' lack of symptoms has been related to psychological difficulties, autonomic neuropathy, and varying pain thresholds. An acute coronary syndrome is a set of symptoms that occur in conjunction with an abrupt cessation of the heart's blood flow.(34–36)

Other severe causes of chest discomfort, including as The rising intracranial pressure activated two emergency programs which resulted in harmful These effects include aortic dissection that causes cardiac tamponade, tension pneumothorax and pulmonary embolism. are included in the differential diagnosis.(37)

Intense physical or psychological stress is linked to an increased risk of heart attacks, particularly if the exertion is more strenuous than the person typically does. For those who are physically very fit, the duration of hard activity and the recuperation that follows is quantitatively linked to a myocardial infarction rate that is roughly six times higher than other, more relaxed time periods. The rate difference is more than 35 times greater for people who are physically ill. The recent discovery relies on increased arterial pulse pressure because it extends and relaxes arteries during each heartbeat cycle. Ultrasonography images show that mechanical stress increases on atheromas due to this phenomenon therefore raising the risk of plaque rupture.(37)

Myocardial infarction can be brought on by an acute, serious infection, like pneumonia. (38,39) The association between atherosclerosis and Chlamydophila pneumoniae infection is more debatable. Research shows conflicting findings regarding the role of this intracellular organism as a cause agent in atherosclerotic plaques despite its detection. Proof reveals that antibiotics fail to reduce heart attack incidence or coronary artery complications during atherosclerosis.(40) Heart attacks usually occurs in the morning, specifically at approximately nine in the morning.(41–43) Although they haven't established a link, several researchers have observed that platelets' capacity to aggregate fluctuates in accordance with a circadian pattern.(44) According to some researchers, this higher prevalence can be caused by circadian fluctuations in cortisol production, which impact levels of several cytokines and other inflammatory mediators.(45)

RISK FACTORS

Complications may include rupture of the esophagus, collapsed lung under pressure, blood clots in the lungs, or fluid buildup around the heart due to a torn aorta, all of which can restrict cardiac output.

- Diabetes with or without insulin resistance is the key risk factor which is most significant.
- Tobacco smoking
- Hypercholesterolemia (high LDL and low HDL)
- High blood pressure
- Family history of IHD
- Obesity (BMI > 30 kg/m², or according to waist circumference or waist-hip ratio).(46,47)

The risk factor status of having experienced coronary vascular events reaches an independent status for adults whose father or brother aged 55 years or younger developed these conditions. The risk factor becomes independent for males who reach age 45 and for females who reach age 55. When a female relative such as mother or sister suffers from a coronary vascular incident before turning 65 years old it establishes an additional individual risk factor.

- Hyperhomocysteinemia
- Stress
- Prolonged drinking of large alcohol amounts leads to an elevated danger of heart attack according to studies.
- The risk of suffering a heart attack is higher among male patients in comparison to female patients.(32)

Following better lifestyle choices can enable prevention of numerous heart attacks because these risk variables can be modified. Physical activity reduces risk profile factors according to research studies.(47) Despite these findings age and sex together with familial predisposition toward early heart attack before age sixty represent non-alterable risk characteristics.(32)

It has been demonstrated that unmarried cohabitation, lower income, and less education all increase the risk of MI, particularly in women. Numerous risk variables that mitigate the risk through other factors are linked to MI, according to the details of epidemiological studies. For

instance, the impact of education is partly determined by how it affects marital status and income.(48) The risk of MI is slightly elevated for women who use combined oral contraceptive pills, particularly when additional risk factors like smoking are present.(49)

PATHOLOGY

An essential stage in development of atherosclerotic plaque is known to be inflammation.(50) CRP is a sensitive indicator of inflammation that lacks specificity.

Elevated CRP blood levels would show potential dangers of stroke or MI and new diabetes development. when assessed using high sensitivity tests. Furthermore, several MI medications may also lower CRP levels. Although it is not recommended to utilize high sensitivity CRP assays for screening the general public, doctors may choose to use them sometimes on patients who already have known coronary artery disease or other risk factors.(51) It is yet unknown if CRP directly contributes to atherosclerosis.(50, 51)

Given the prevalence of periodontitis, the association between inflammation in this condition and coronary heart disease may have serious public health repercussions.(52, 53) According to serological investigations that measure antibody levels against common bacteria that cause periodontitis, people with coronary heart disease had higher levels of these antibodies. Periodontitis increases Blood levels of cytokines and fibrinogen and CRP.(54) may contribute to MI risk through underlying risk factors according to the literature. (55–57) Preliminary expert opinions show that periodontal bacteria fuels platelet aggregation with foam cell development while specific bacteria roles remain supposition. Some evidence is available that acute MI could be caused by influenza. (58)

Blood levels of cytokines and fibrinogen and CRP may contribute to MI risk through underlying risk factors according to the literature.(54) Preliminary expert opinions show that periodontal bacteria fuels platelet aggregation with foam cell development while specific bacteria

roles remain supposition. function is still up for debate, although there is thought to be a shared factor between these symptoms and the risk of MI that may be inherited. (55)

Another aspect of the development of atherosclerotic plaque is calcium deposition. CT scans(59) can identify coronary arteries calcium deposits. According to a number of studies, coronary calcium can offer more predictive data than traditional risk markers. (60–62)

There exist two main categories of acute myocardial infarction which include ST-elevated myocardial infarction and non-ST-elevated myocardial infarction. The acute cardiac syndrome features both ST-elevated myocardial infarction and non-ST-elevated myocardial infarction. Coronary artery disease remains the leading cause of these conditions but other possible healthcare conditions exist. The breaking open of an atherosclerotic plaque inside a coronary artery results in a blood clot that causes complete blockage of the artery. The process of atherosclerosis occurs when cholesterol accumulates with fibrous tissue to form plaques that build up inside artery walls (here specifically within coronary arteries) during many long years. Long-term atherosclerosis results in reduction of the arterial channels. as evidenced by blood stream column abnormalities on angiography. Plaques can become unstable and burst in minutes, causing a thrombus, or blood clot, to form and clog the artery. Myocardial infarction, also known as downstream myocardial necrosis, occurs when a sufficiently severe plaque ruptures in the coronary artery.

Heart cells die through necrosis as they surround the blocked coronary artery due to disrupted blood supply unless the occlusion leads to an ischemic cascade. In its place, a collagen scar forms. Apoptosis, another type of cell death, has been shown in recent studies to influence tissue damage following myocardial infarction.(63)

DIAGNOSIS

By combining the physical examination and history of the presenting disease with the results of the EKG and cardiac markers, a myocardial infarction is diagnosed. (18)

Routine blood tests and a chest radiograph are typically performed when a patient first arrives at the emergency room, and they can indicate abnormalities or triggering factors. Echocardiography may potentially reveal new regional wall motion anomalies, indicating a myocardial infarction. A cardiologist may conduct echocardiography in circumstances that are unclear. (18)

Stable patients with resolved symptoms receive technetium (99mTc) sestamibi (MIBI scan) together with thallium-201 chloride examination at Nuclear medicine facilities to measure blood flow restrictions. These tests help determine if hibernated or stunned myocardial tissue is actually dead.(64)

The research conducted by Gibson et al. analyzed 48 urgent acute IMI cases from 1982. The ST segment decreased by at most 1.0 mm in 21 patients who formed group A in precordial leads V1-6 whereas 27 patients in group B experienced ST segment drops exceeding 1.0 mm. The research unveiled no substantial differences between individuals in groups A and B regarding their coronary heart disease history and left anterior descending coronary artery disease prevalence as well as ST-segment changes on exercise tests or angina development and anterior or septal wall motion findings from the 201Tl test. People with acute MI who demonstrate ST drops in their pericardial region develop broader inferior or inferoposterior wall myocardial infarctions that cause both widespread and localized dysfunction of the left ventricle when compared to those who exhibit anteroseptal ischemia.(65,66)

Cohen et al. examined ventricle-artery connections during acute illness. Thirty-two patients with acute transmural inferior wall MI who presented within 12 hours underwent cardiac catheterization and angiography. In twelve-lead ECGs performed before catheterization, 17 individuals had inferior wall anomalies and ST segment depression in anterior precordial leads. The anterior lead ECGs of 15 patients exhibited no ST segment alterations. Evaluation of both groups' clinical arteriographic and ventriculographic parameters found no significant differences.(67)

The research by Hlatky et al. across 20 years into acute inferior myocardial infarction revealed hospital mortality reached 13% among patients with ST depression but only 4% between those who did not show precordial ST depression. The correlation proved important because ST depression patients survived five years following hospital discharge at an 80% rate and patients without precordial ST depression reached 92% survival. During the initial ECG assessment for acute myocardial infarction the presence of ST depression in the precordial region indicates a larger heart attack size and higher hospital death risk along with diminished survival chances after discharge.(68) Patients with anterior infarcts displayed reciprocal abnormalities in 21 cases (62%) while inferior infarcts had reciprocal changes in 27 cases (59%). A constant association existed between infarct site and reciprocal ST segment changes alongside further coronary disease manifestation. At follow-up 51 patients demonstrated reciprocal ST segment alterations and among these 36 patients developed symptoms and 29 received coronary artery bypass surgery. Patients who experienced symptoms following coronary revascularization accounted for four out of the 27 individuals who did not show ST segment reciprocal abnormalities. Medical professionals can use the detection Using reciprocal ST segment depression to identify crucial coronary disease patients at risk of cardiac complications following acute myocardial infarction.(12)

Dewhurst et al. (1985) examined the ECG readings of 100 individuals who had serial radionuclide ventriculography and survived their first myocardial infarction. Anterior myocardial infarction occurred in 46 cases, while inferior wall myocardial infarction occurred in 54 patients. According to enzyme criteria, those with reciprocal ST segment depression experienced larger infarcts (CPK 2203 ± 1271 vs. 1544 ± 1197 IU) and a higher incidence of ventricular akinesia and dyskinesia. When comparing individuals with inferior infarction to those with anterior infarction, there was a noticeable preservation of left ventricular function with corresponding alterations. The patient group with modificatons in both right and left ventricles experienced the highest death count. Left ventricular dysfunction led to increased mortality in patients followed by the combination of large infarct regions with abnormal heart muscle motion as main factors producing ST change in the inferior leads. anterior infarction. Reciprocal change in inferior infarction patients who had pretty good ventricular function were likely at risk for anterior descending lesions or left main stem problems.(69)

Reciprocal ST-segment depression was observed in 57% of anterior MI patients (63 cases) and 65% of inferior MI patients (79 cases) in Yousif et al.'s 1989 study on acute myocardial infarction angiography in 142 patients. In 63% of 52 patients with reciprocal ST-segment depression and 67% of 46 patients with positive exercise tests, the coronary artery supplying the reciprocal ST-segment region was heavily stenotic. 35 patients with positive exercise tests (76%) and 41 patients with multivessel coronary artery disease (79%) had reciprocal ST-segment depression. In clinical investigations, four-fifths of patients had multivessel coronary artery disease, which resulted in reciprocal ST-segment depression, and two-thirds of patients had stenosis affecting their opposite coronaric artery supplying tissue. For multivessel coronary artery disease, reciprocal ST-segment depression was as reliable as a positive submaximal exercise test following myocardial infarction.(13)

Strasberg et al. classified 46 patients (41 men and 5 women, average age 56 years) into wide (V1-V6) or confined (V1-V4) groups based on the severity of pericardial ST segment depression. Patients with ST depression in V1 to V4 and ST elevation in V5 and V6 were not included in the study unless they did not exhibit reciprocal ST depression. Every patient admitted to the hospital for myocardial infarction received a catheter. Out of the 28 patients with ST segment depression extending in leads V1 through V6, 24 had substantial lesions in this coronary artery segment, while only 16 of the 18 patients with ST segment depression restricted to leads V1 through V4 had little to no structural damage in the left anterior descending artery. In order to identify patients who have inferior acute myocardial infarction and pericardial ST depression, medical practitioners consider the degree of ST depression to be significant. ST depression between V1 and V4 indicates the lack of left anterior descending coronary artery disease, whereas significant ST depression between V1 and V6 indicates the pathogenic status of the artery.(70)

Zoghi et al conducted research which investigated whether right stem depression (RSTD) shows any correlation with coronary artery disease severity. The study enrolled 188 patients who suffered from acute inferior myocardial infarction then received thrombolytic medication for treatment. A total of 63 out of 108 patients (58%) who showed RSTD presented with multivessel CAD. This rate was higher than the 32 patients (40%) from the group of patients without RSTD who had multivessel CAD. Patients with anterior RSTD had more multivessel CAD regardless of lateral ST segment depression. The occurrence of right ST segment depression during AIMI seems to have electrical as well as clinical implications for developing multivessel CAD according to their findings.(71)

Parale et al. evaluated reciprocal leads in 300 patients suffered acute MI. In addition to having strong LVF, acute AWMI patients with Q waves in their inferior leads also show less heart

damage and are more likely to experience distal LAD obstruction. When inferior lead reciprocal changes are absent, the LVEF in AWMI patients stays higher. Patients with acute IWMI who have apicolateral lead ST depression tend to present multivessel coronary artery disease in addition to substantial left ventricular functional impairment.(72)

ENZYMES CHANGES IN ACUTE MYOCARDIAL INFARCTION

When Karmen et al. discovered in 1954 that individuals with AMI had elevated SGOT, now known as aspartame transaminase or AST activity, diagnostic testing exploded. Later, in 1955, Wroblewski and Due discovered Patients diagnosed with acute MI showed elevated lactate dehydrogenase levels in their blood sample (LDH). AST was substituted by measuring LDH and then LDH isoenzymes for the confirmation of myocardial damage due to their higher specificity for the heart.(73)

In their groundbreaking research, La Due and his colleagues found that numerous other serum enzymes, such as malic dehydrogenase, phosphohexose isomerase, creatine kinase, aldolase, triosephosphate isomerase, enolase, pyruvate kinase, phosphoglucomutase, isocitrate dehydrogenase, and alpha hydroxybutyrate dehydrogenase, were elevated after AMI. However, further investigation and observation revealed that none of these enzymes were unique to the heart.(74)

Only a few number of these enzymes could be estimated for routinely diagnosing acute myocardial damage until the early 1970s. The identification and measurement of more cardio-specific isoenzymes became feasible with the development of electrophoresis and the separation of enzymes into different isoenzyme forms. The development of enzyme estimates for diagnosing acute MI experienced a major change because of this. Gambio identified the LDH1/LDH2 ratio >

1.0 pattern which effectively indicates acute myocardial necrosis. et al., who examined the various isoenzyme forms of LDH in patients with AMI, renal infarction, musculoskeletal trauma, stroke, shock, and neoplasia.(75)

In 1975, Roberts et al. identified three distinct types of creatine kinase that can be separated using electrophoretic techniques. Three specific isozymes of creatine kinase exist in different body tissues including CK-MM within skeletal muscle cells as well as CK-BB active in brain tissue and gastrointestinal tissues followed by CK-MB found only in cardiac muscle cells. The researchers demonstrated that cardiac enzyme CK-MB functions as a powerful tool for diagnosing acute myocardial infarction since it exhibits high test accuracy and precision.(76) Sobel et al. used periodic enzyme assays after AMI to explore the rate at which creatine kinase left the circulation. They discovered that the size of the infarct correlates closely with the pace at which the heart's enzymes are depleted.(77)

The serum levels of CK-MB earned rapid adoption by clinicians for acute myocardial infarction diagnosis because myocardial Ck elevation after AMI occurred early and MB isoenzyme assessment improved cardiac injury diagnosis. Clinical research is investigating myoglobin, heart muscle and myosin light chain Troponin-T, cardiac Troponin-I, and other structural proteins and enzymes are used to diagnose patients with ACS; however, these tests may have additional benefits and drawbacks. Along with other blood cardiac markers, the examination of hFABP, myosin heavy chain, and glycogen phosphorylase isoenzymes BB is a potential technique for diagnosing myocardial damage.(78–80)

Acute myocardial infarction diagnosis according to WHO recommendations includes three components where one diagnostic element is enzyme release patterns which match myocardial damage evidence. The enzymatic diagnosis remains essential for most acute myocardial infarction

cases in medical practice. Biochemical evidence of myocardial injury becomes the sole indicator of infarction because ECGs sometimes present benign or nondiagnostic results.. Because of this, some writers have proposed that, in the right clinical context, the normal rising and falling pattern of enzymes, specifically CK-MB, should be sufficient to confirm AMI.

If it is possible to ascertain these makers' release and clearance from the plasma, concentration in the myocardium, volume of distribution, and degree of depletion from the myocardium following myocardial injury, they can be used to estimate the size of infarction in addition to their diagnostic significance. It has been demonstrated that these cardiac enzyme-based infarct size estimations closely match morphologically-derived estimates, particularly when thrombolysis is not performed.(81)

Both the dynamics of macromolecule release from the heart and their diagnostic value are significantly altered in thrombolyzed AMI patients. The rate of release of these molecular markers is altered by recanalization or effective reperfusion after thrombolytic therapy. As a result, the pace at which their plasma changes can be utilized to assess the success of coronary revascularization induction. In patients whose reperfusion fails after thrombolytic therapy for AMI, this might help with the evaluation of further revascularization procedures.(82)

Markers Characteristics

The ideal indicator of myocardial damage would be highly concentrated in the myocardium, A unique combination of characteristics makes Creatine Kinase MM present only in heart tissue but absent in other tissues while promptly releasing its entire quantity after damage to the heart tissue in direct correlation with damage severity during a short diagnostic timeframe in plasma. Factors determining these attributes, as well as each marker's sensitivity and specificity, are determined by the following factors:

Size

Generally speaking, a molecular marker is delivered into the bloodstream more quickly the smaller it is.81 Therefore, it is expected that myoglobin and other low molecular weight indicators will have larger levels earlier than LDH and CK.(83)

Cellular localization

After sarcolemma damage, The speed of cytosolic protein diffusion into blood plasma surpasses structural proteins which results in plasma concentration increases.(84)

Solubility

Certain cardiac contractile proteins and other macromolecules with limited solubility exit the heart slowly.

Release ratio

Following release, certain macromolecules, like CK, may experience local degradation. As a result, the quantity of marker that is removed from the heart can be significantly more than what can be detected in plasma. Variations in blood flow can change the releaser ratio, for example. The amount of CK which drains from the heart into plasma reveals significant increased by reperfusion.(85)

Clearance

In general, smaller markers are eliminated faster than larger ones.(83) As a result, if multiple blood samples can be acquired, It is preferred to use markers which degrade quickly in plasma when defining critical time points similar to post-intervention reperfusion or reinfarction.. Longer half-lives make markers more effective in integrating events over time with sparse data, although they are frequently insensitive to new releases, for example from repeated damage to the heart.

Specificity for myocardium

The majority of the heart's macromolecules are also found in large quantities in skeletal muscle, particularly in diseased states. Following damage, cells change the molecular species they generate, reproducing the synthesis of proteins made during pregnancy or the early years of life.

Specificity for irreversible injury

The detection of plasma marker proteins represents a vital alternative to difficult cardiac necrosis diagnosis because of its association with normal plasma levels, and the assay's sensitivity, defining specificity for irreversible injury has proven challenging. Following myocardial damage, a prolonged release of structural proteins in plasma indicates persistent damage.

Detectability

Assays with high sensitivity, low variability, and ease of use are necessary for detectability. Generally speaking, the best diagnostic accuracy is offered by markers that are normally found in plasma at very low concentrations. The clinical context in which the test is conducted is extremely significant in addition to the previously mentioned factors. The patient population to which a test is administered determines both its specificity and diagnosis accuracy. Therefore, when ischemia-like symptoms are present, these indicators have a higher specificity for the acute myocardial infarction diagnosis

The table lists the several markers that are currently or may soon be used for diagnosing acute myocardial infarction along with the times it takes for them to rise, peak, and fall back to normal. Generally speaking, the best diagnostic accuracy is offered by markers that are normally found in plasma at very low concentrations. The clinical context in which the test is conducted is extremely significant in addition to the previously mentioned factors.

Molecular markers used the diagnosis of AMI

Creatinine kinase and its isoenzymes

In order to create creatine phosphate and adenosine diphosphate (ADP), Through the action of creatine kinase (CK) the enzyme transfers an energetic phosphate group from ATP to the molecule creatine. Creatine phosphate functions as quick storage of high energy utilization for the body. preventing the rapid depletion of ATP. The production of creatine phosphate from ATP and creatine is catalyzed by the mitochondrial CK. After diffusing into the cytosol, the high-energy phosphate is removed by cytosolic CK, which acts as an energy substrate and catalyzes the production of ATP.(86)

pH 9.0, mitochondria

Creatine+ATP ----------> Creatine phosphate+ ADP

Creatine phosphokinase

<----------

Ph6.7, cytosol

The majority of bodily tissues contain CK, which has a molecular weight of 86,000 D units. However, the brain, heart muscle, and skeletal muscle are the richest sources. Proteolysis in the lymphatic system deactivates it.(87)

Scientists used electrophoresis for identifying the three different creatine kinase isoenzyme forms. The isoenzymes consist of 39000 to 42000 D subunits that each is derived originating in the cytoplasm. Three distinct isoenzyme forms of creatine kinase are found: CK-MB, which consists of

one M and one B subunit; CK-MM, which consists of two M subunits; and CK-BB, which consists of two B subunits. Without enzymatic action, each distinct subunit comes after. Heart tissue contains the greatest CK-MB of the three isoenzymes, while skeletal muscle tissue contains CK-MM and brain tissue includes CK-BB. Since CK-MM and CK-BB have different isoelectric point isoforms but identical enzymatic activity, two CK-MB isoforms (MB1 and MB2) were identified by electrophoresis.(88) The early appearance of CK-MB and CK-BB subunits in plasma allows for the early diagnosis of both acute MI and successful reperfusion in patients. The tissue distribution pattern and ability to detect irreversible damage match exactly between CK.92 and its counterpart CK. (89)

The skeletal muscle contains predominantly CK-MM but traces (1-3%) of CK-MB also exist alongside minimal cardiac muscle quantities of the same enzyme. The small intestine along with the tongue as well as uterus and diaphragm and prostate carry the CK-MB isoenzymes at very low levels.(90) Increased serum activity of CK-MB indicates myocardial necrosis while minimal CK-MB levels in non-heart tissues can only suggest heart damage when those organs have not suffered trauma or surgical procedure. Evidence shows that mass assays of CK-MB deliver superior accuracy compared to CK-MM activity tests although clinical practice mostly relies on activity measurements for early acute myocardial infarction cases.(91)

CK-MB testing is currently the primary diagnostic for validating an AMI diagnosis. Following the onset of AMI, CK and CK-MB plasma levels often start to rise 4–8 hours later, peak 2–10 times the normal level 24 hours later, and then revert to normal within 72 hours. 86 The MB component declines a little quicker and the CK-MB levels peak a little earlier. than total CK. Lesser infarctions reached maximal enzyme concentration faster than severe infarctions.(92)

In addition to AMI, myocardial cell death from any of the following causes will cause CK-MB to rise and fall: myocarditis, electrical injury, cardiac catheterization, myocardial contusion following chest trauma, and cardiac surgery.(78) The majority of research shows a correlation between the production of CK-MB in plasma and further indicators of myocardial damage. Furthermore, CK-MB levels do not rise in individuals with severe unstable angina or following ischemia brought on by stress testing.(93)

The measurement of CK-MB is used to diagnose reperfusion early. Indicators of reperfusion have been identified as When treatment begins the level of CK-MB elevates quickly while the time it takes for peak CK-MB levels appears more rapidly. The measurements of CK-MB during the first two hours demonstrate a 2.2-fold elevation in patients having inferior myocardial infarction as well as a 2.5-fold elevation in patients who experienced anterior wall infarction. (85%) and specific (100%) for detecting recanalization.(94) However, CK-MB measurement one hour post-thrombolysis does not distinguish between patients who have successful recanalization and those who do not. Scientists investigated the association between the total CK or CK-MB release amount and necrosis size. at autopsy and with other methods for determining the size of an infarct in vivo during the prethrombotic era. However, Thrombolytic therapy during coronary artery perfusion modifies the kinetics of wash out for CK from the myocardium thus resulting in accelerated peak enzyme levels which are higher than normal. the use of infarct size.(95)

LACTATE DEHYDROGENASE

Enzyme lactate dehydrogenase (LDH), which is present in every cell in the body, is in charge of the last stage of glycolysis, which is the interconversion of pyruvate and lactate. LDH has a molecular weight of 135,000 and is found as a tetramer. Five isoenzymes are produced by the two subunits, M (muscle: MW 34000 D) and H (heart: MW 34000 D).

LDH

Despite its sensitivity, total LDH is not unique to cardiac tissue. LDH-2 is more abundant than LDH-1 in normal human plasma, and the ratio of LDH-1 to LDH-2 is often less than 0.76. More LDH-1 than LDH-2 is released when myocardial necrosis takes place, and the proportion of LDH-1 to LDH-2 rises. While a ratio >1.0 has a better specificity but poorer sensitivity, some writers consider a ratio >0.76 to be indicative of AMI.(73)

LDH and estimation of infarct size

Anatomical estimations of infarct size have been found to be well correlated with LDH [and alpha hydroxybutyrate dehydrogenase (HBDH) as defined by this test provides the measurement. LDH-1 plus LDH-2].

Limitation of LDH estimation in acute myocardial infarction

1. Total LDH, which is more widely measured than its isoenzymes, The myocardial markers show no exclusivity toward heart muscle tissue which leads to possible incorrect elevation diagnoses.n its plasma levels may occur in patients with the following conditions.

   i. Haemolysis.

   ii. Megaloblastic anaemia

   iii. Leukaemia and other neoplasms

   iv. Hepatic congestion due to cardiac failure or other cause

   v. Hepatic, renal, gastrointestinal or pancreatic disease

vi. Pulmonary embolism

vii. Myocarditis

viii. Skeletal muscle disease and

ix. Shock

2. LDH is not useful The test provides useful information for diagnosing acute myocardial infarction when patients show first symptoms between 8 to 12 hours after chest pain onset because of delayed myocardial release.

3. Troponin-T

Muscle contractile machinery in the thin filament contains a protein complex called troponin complex in skeletal and cardiac contraction.(96)

The troponin complex functions with three different protein subunits.

a. Troponin-T (TnT): TnT is in charge of The troponin complex becomes linked to the thin filament through tropomyosin attachment. Molecular weight of TNT is 39000 D. About 6% of TNT is dissolved in the cytosol of human cardiac cells, despite the fact that the majority of it is integrated into the troponin complex.
b. Troponin-I (TnI): TnI inhibits the interaction between actin and myosin by binding to actin. Its molecular weight is 26500 D, and its cytosolic pool contains roughly 2-3% of it.(78)
c. Troponin-C (TnC): This protein The protein acts as a calcium receptor while handling thin filament activation for contractions. of cardiac and skeletal muscles.(78)

Measurement of cardiac troponin T

Research by Katus et al. produced the inaugural enzyme immunoassay for cardiac troponin-T isoform during 1989. A one-step sandwich immunometric assay operates through troponin-T-specific polyclonal antibodies which are purified from polyvinyl chloride test tubes with cardinal cardiac protein specificity. The test tubes possess antibodies that have troponin-T attached to them and also contain both porous peroxidase-tagged anti-troponin-T monoclonal antibodies and troponin-T standards as well as blood samples. During incubation, both the liquid-phase monoclonal antibody-enzyme complex and the solid-phase polyclonal antibody fraction bind to troponin-T.

In 1995, Muller-Bardorff et al. created a quick test for cTnT determination at the bedside based on the same idea. This assay's benefit was that it produced accurate and dependable test results at the patient's bedside in less than 20 minutes.(78)

Role of cardiac troponin –T estimation in the diagnosis of AMI

If evaluated during the diagnostic window period, the cTnT is high in all The study included patients who received AMI diagnosis based on WHO standards. More than 100 percent of AMI cases can be identified using troponin-T assessment. The blood level of cTnT increases three hours from chest pain onset before remaining elevated for ten to fourteen days.

Troponin – T and the detection of reperfusion following thrombolysis

Troponin-T release kinetics in AMI patients are significantly changed by successful reperfusion after thrombolysis. Troponin-T levels in the serum peak early in individuals whose thrombolytic therapy has restored infarct-related artery patency.(97)

STREPTOKINASE(98)

An extracellular bacterial protein called streptokinase, the earliest known plasminogen activator, is generated by hemolytic streptococci that are actively developing and belong to human C, G, and Lance-field Group A. Group A and Group C strains have been the next most powerful producers of this chemical, after Human C. Nearly all experimental research in recent years has used STK generated by the Lancefield Group "C" bacterium. Christensen, Fletcher, and Johnson described several methods for purifying STK, including the continuous manufacture of 600 units per gamma nitrogen. STK has a molecular weight of roughly 47000 Dalton. It is stable across a broad pH range and easily soluble from pH 4.77 to 5.0. There are at least four ideas regarding how streptokinase activates plasminogen.

Coronary Angiography

Medical professionals identify coronary angiography as the sole procedure healthcare professionals use to determine if coronary artery disease causes blood vessel narrowing. This method provides the highest quality anatomical data needed to select proper treatments between medication and PCI or CABG surgery for ischemic CAD patients.

Hospital staff should use coronary angiography testing for both cardiogenic shock patients and PCI needs in individuals who need treatment for serious cardiovascular damage such as mitral regurgitation or ventricular septal ruptures.

Although a low threshold for coronary angiography is recommended as part of an early invasive strategy for patients with high risk indicators, such as recurrent ischemia symptoms despite adequate medical therapy, high risk non-invasive test results, depressed left ventricular systolic function, severe arrhythmia, and prior revascularization, coronary angiography is generally not advised as part of the routine evaluation for UA/NSTEMI.

Coronary angiography is not recommended as part of routine follow-up for patients whose clinical status has not changed, but it is recommended for patients who have significant functional status limitations in spite of the most effective medical treatment or who show signs of persistent ischemia following a revascularization procedure.

QT DISPERSION IN MYOCARDIAL INFARCTION

Targeted therapy stands as a possibility to minimize cardiac fatalities through successful identification of individuals who face high sudden cardiac death risks. An appropriate screening method for this condition remains out of reach at present. Medical practitioners face difficulty applying signal-averaged electrocardiography as well as T-wave alternans and heart rate variability due to their varying usefulness and dependence on specialized equipment. QT interval analysis stands as an alternative method for screening abrupt cardiac death risk because individuals with long QT syndromes are at a high risk of experiencing these sudden cardiac events.

Researchers have found potential to obtain data about future health outcome through examining QT interval variability patterns in standard ECG measurements. Experts are performing exhaustive tests on "QT interval dispersion" in present times. Campbell et al attributed this discovery the status of "electrophysiological Holy Grail" in their report three years ago. Medical research about QT dispersion conducted since 1990 demonstrated a 34 times increase in studies available in Medline. The research examines QT dispersion in Acute Myocardial Infarction patients alongside the analysis of ventricular arrhythmia rates related to QT dispersion values in these subjects.(99)

The length of ventricular myocardial depolarization and repolarization shows as the combined measurement of the QRS complex together with the J-T interval.

Wolff described in 1950 that the Q-T interval measures from QRS complex onset to end T wave duration yet expert consensus has eliminated its practical application.

QT Interval (100,101)

Between the beginning of the QRS complex and the end of the T wave is the QT interval. This is also known as the time between the first sign of ventricular depolarization and the last sign of ventricular repolarization.

Normal heart-rate correct QT (QTc) interval

- < 440 milliseconds in men.
- < 460 milliseconds in women.

QTc calculation formulas

QT interval calculation adjustments

- Correction of QT interval ought not to be done if RR interval has large variability (such as with atrial fibrillation) or if end of T wave cannot be reliably identified.
- Alter QT interval for sex.
- Adjust QRS duration if there are ventricular conduction abnormalities present.

QT dispersion(102)

QT dispersion is the gap between the QT intervals that are the longest and the shortest.

REVIEW OF RELATED LITERATURE

Chittora et al. found that the mean QTcd following acute coronary syndrome was highest upon admission and reduced till day seven as the patient improved. Complications like low blood pressure, congestive heart failure (CHF), PSVT, VPC, CHB, ventricular fibrillation, and mortality kept it high. Thrombolyzed STMI patients had considerably lower mean QTcd on day one and fewer hospital issues and mortality ($p<0.001$) compared to non-thrombolyzed patients. The QTcd interval may indicate life-threatening arrhythmias and hospital death. After a MI, timely thrombolysis lowers the mean QTcd, reducing death and sickness.(103) Acute MI patients had a statistically significant larger mean QTc dispersion than non-patients, according to Jatav et al. From day one until they were discharged home, heart patients had a larger mean QTc dispersion than non-heart patients. On the first day, the mean QTc spread was greater in deceased patients than in living patients. Finally, monitoring QTc dispersion may be a straightforward, low-cost, and painless technique to identify STEMI patients at risk for ventricular arrhythmias. It's also related to prediction, as a patient's future may reveal their death. (104) Valizadeh et al. found that the primary PCI group had a reduced QTD, not significant ($P > 0.05$). Main PCI patients had much higher mean EF values ($P = 0.022$). In fatal arrhythmia patients in the main PCI group, QTD was substantially lower ($P = 0.022$). This treatment works better than thrombolytic therapy, since the primary PCI group had considerably lower QTD of patients with lethal arrhythmias, and the QTD reduced overall. Healthy cardiac function is indicated by EF, which can increase myocardial function in the primary PCI group alone.(105) Eltahlawi et al. found that ischemia patients had prolonged QTd at rest and during exercise ($P = 0.000$). QTd difference between stress and rest was detected. QTd difference between normal and ischemia groups was much less ($P = 0.003$). A considerable positive correlation ($P = 0.04$) existed between defect size and QTd difference. QTd rises during ischemia, and the difference between rest and stress QTd is related to ischemia severity.(106) Khanna et al.

found that subsequent thrombolysis prolonged QTd. People with effective thrombolysis had reduced QTd. Late group had more arrhythmias than early. Fast, effective thrombolysis reduces arrhythmia risk. Early in an acute myocardial infarction, mean QT and QTc dispersions were wider. People with anterior acute myocardial infarctions exhibited longer QT intervals than those with lower ones. Streptokinase reduced QT and QTc dispersions more than before. QT and QTc dispersions peak during serious myocardial infarction. People with effective thrombolysis had reduced QTd. Late group had more arrhythmias than early. Fast, effective thrombolysis reduces arrhythmia risk.(107) In patients with arrhythmia, Apoorva et al. observed a QTcd of 0.07±0.06 sec at admission and 0.04±0.03 sec post-thrombolysis (p=0.0227). Those without arrhythmia had a QTcd of 0.04±0.03 seconds upon admission and 0.02±0.03 seconds following thrombolysis (p=0.0001). Thus, arrhythmia patients had a larger QT dispersion upon admission. This discrepancy persisted after thrombolysis, perhaps because reperfusion failed. There was a significant correlation ($p<0.0001$) between arrhythmic events and patient outcomes. QTcd median (IQR) was 0.01 (0-0.09) for released patients and 0 (-0.09-0.03) for deceased patients. The correlation between QTcd and MI outcomes after thrombolysis is substantial (p=0.0120). People without arrhythmia exhibited lower QT dispersion: 0.04±0.03 seconds at entrance and 0.02±0.03 seconds after thrombolysis (p=0.0001). Thus, arrhythmia patients had a larger QT dispersion upon admission. This discrepancy persisted after thrombolysis, perhaps because reperfusion failed. These findings support the concept that reperfusion condition alters QT dispersion after MI. Thrombolytics may reduce QT dispersion. QTcd predicts arrhythmogenicity by measuring damaged heart refractoriness.(108)

MATERIALS & METHODS

MATERIALS AND METHODS

A hospital based prospective analytical study was undertaken among 50 patients admitted in Cardiac Critical Care Unit of with ACUTE MYOCARDIAL INFARCTION. Clearance from Institutional ethics committee was obtained before the study was started. An All of the cases gave their full consent before they were added to the study. The following were the factors for inclusion and exclusion:

INCLUSION CRITERIA

1. Be at least 18 years old.

2. Have a ST segment rise at the J-point in two consecutive leads with ≥ 1 mm in all leads except leads V2 and V3, where ≥ 2 mm in men aged 40 and up, ≥ 2.5 mm in men aged 40 or younger, and ≥ 1.5 mm in women of any age.

3. High amounts of troponin-I.

4. Who signed the written permission form after being told about it?

EXCLUSION CRITERIA

1. People who have atrial fibrillation (AF) or flutter.
2. People who have a Left and/or Right Bundle Branch Block.
3. People who take long-term medicines that change the length of the QT interval were not included in the study.
4. Metabolic conditions leading to QT prolongation i.e., hypocalcemia, hypokalemia, and hypomagnesemia.

Method of collection of data

All patients received informed written consent before undergoing a thorough physical examination based on the prescribed proforma. All patients underwent multiple tests that included complete blood count along with tests for renal and liver function along with ECG evaluation. Patients underwent 12-lead ECG recordings at admission and again at 24 hours while adjusting the measuring device to 1mV=10mm and using 25mm/sec as the recording speed. The start of QT measurement occurs with R wave onset then ends when T wave completes; yet the tangent approach provides the most optimal methodology for T wave termination identification. The end of T wave identification occurs when a tangent drawn from maximum T wave downslope intersects with a vertical line produced from the isoelectric baseline. The physician considered the dip between T and U waves as marking the T wave termination when U waves showed prominence.

Statistical analysis

Microsoft Excel controlled the data that researchers analyzed with SPSS 22 version software. Statistical data representation consisted of both frequencies and proportional distributions. The Chi-square test and Fischer's exact test (only valid for 2x2 tables) functioned as the methods to determine statistical significance within qualitative data. Whenever rules for chi-square calculation were not possible the researchers applied Yates adjustment procedure. (for 2x2 tables only).

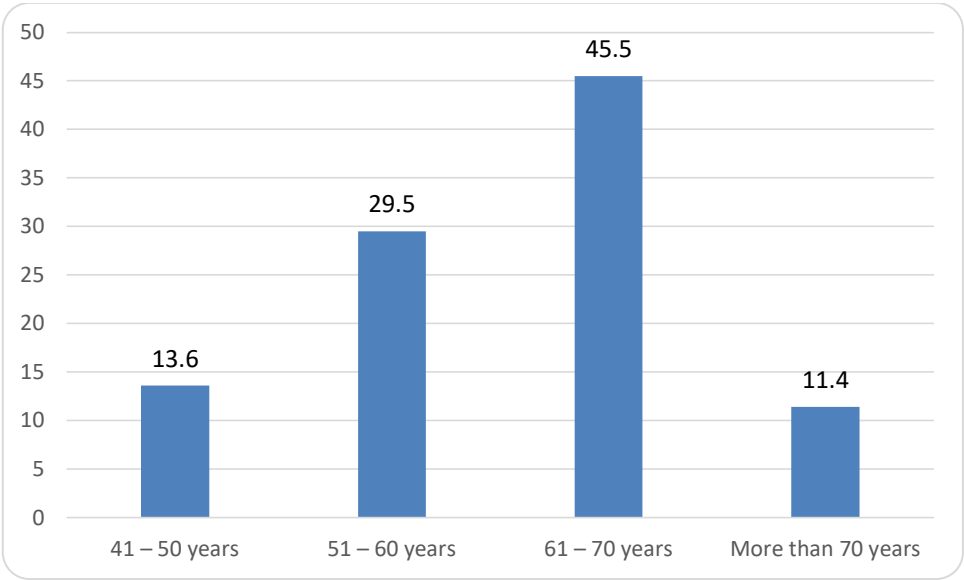
RESULTS

RESULTS

Table 1. Age group

Age group	Frequency	Percent
41 – 50 years	6	13.6
51 – 60 years	13	29.5
61 – 70 years	20	45.5
More than 70 years	5	11.4
Total	44	100

Char 1. Age group

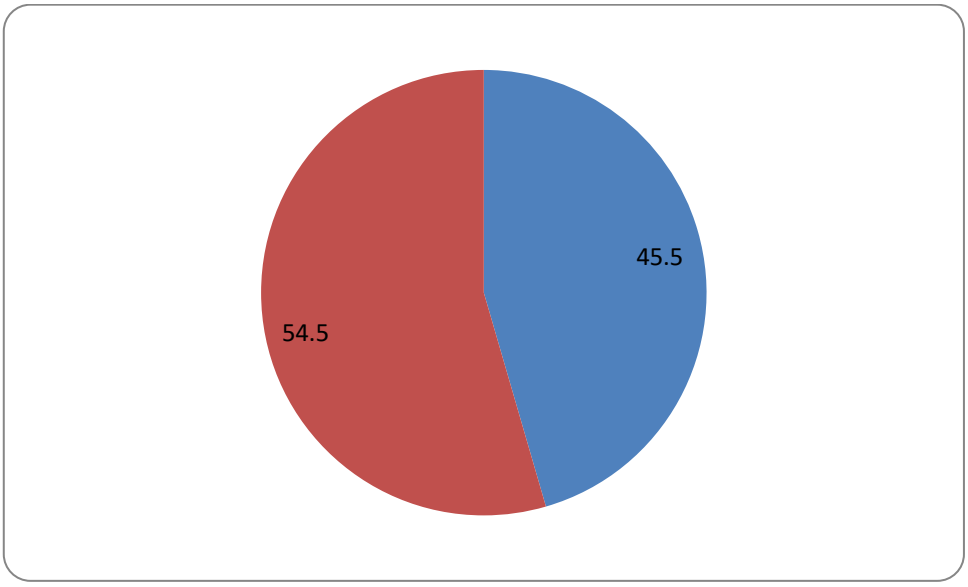


About 45.5% of the cases in this study were aged between 61 – 70 years.

Table 2. Sex

Sex	Frequency	Percent
Male	20	45.5
Female	24	54.5
Total	44	100

Chart 2. Sex

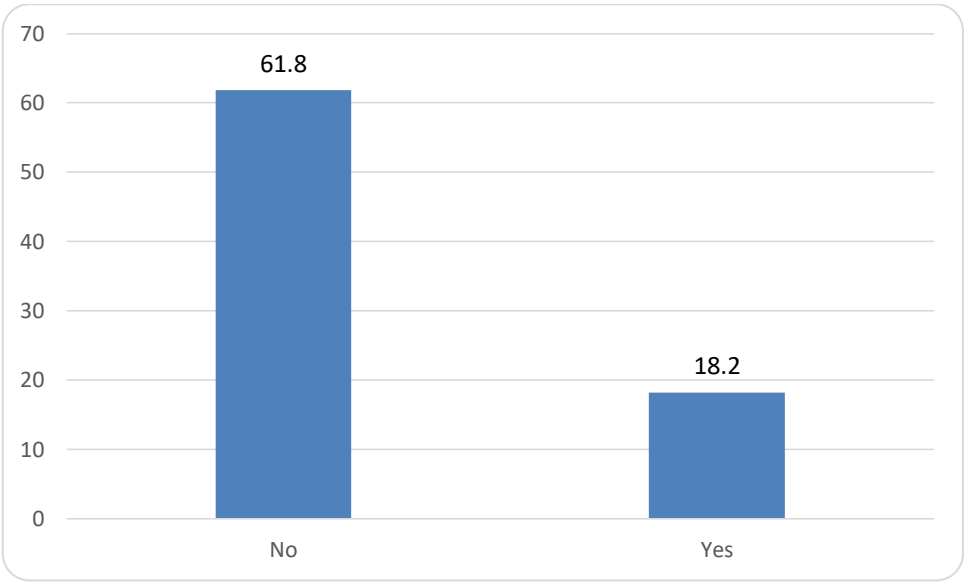


About 54.5% of the cases in this study were females.

Table 3. Smoking

Smoking	Frequency	Percent
No	36	61.8
Yes	8	18.2
Total	44	100

Chart 3. Smoking

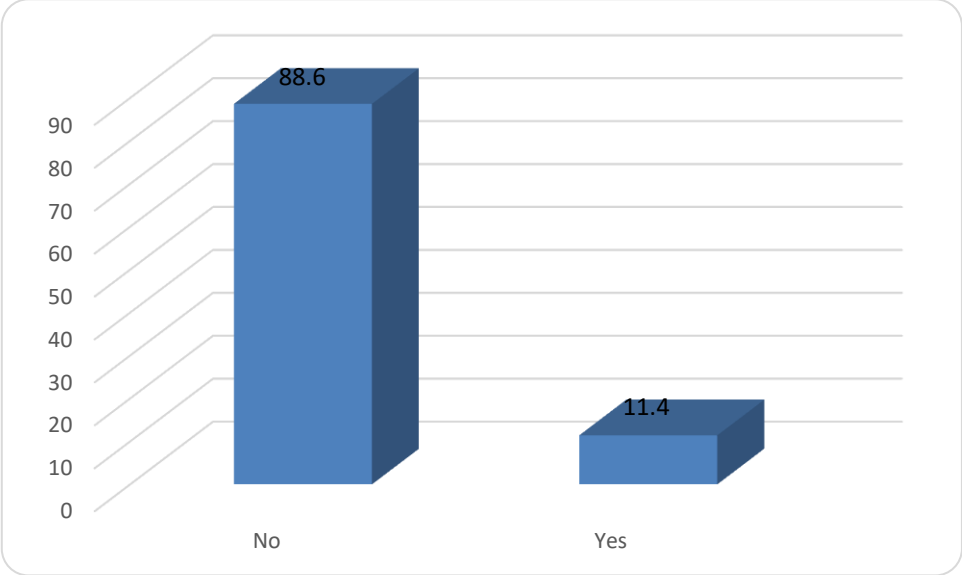


About 18.2% of the cases in this study had history of smoking.

Table 4. Alcohol

Alcohol	Frequency	Percent
No	39	88.6
Yes	5	11.4
Total	44	100

Chart 4. Alcohol

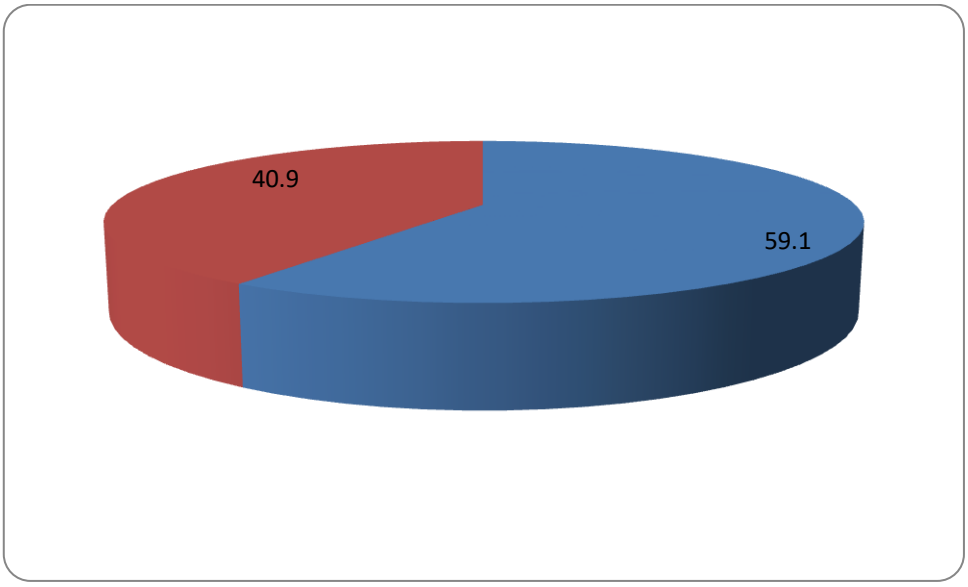


This study had shown that, about 11.4% of the cases were alcoholics.

Table 5. Hypertension

Hypertension	Frequency	Percent
No	26	59.1
Yes	18	40.9
Total	44	100

Chart 5. Hypertension

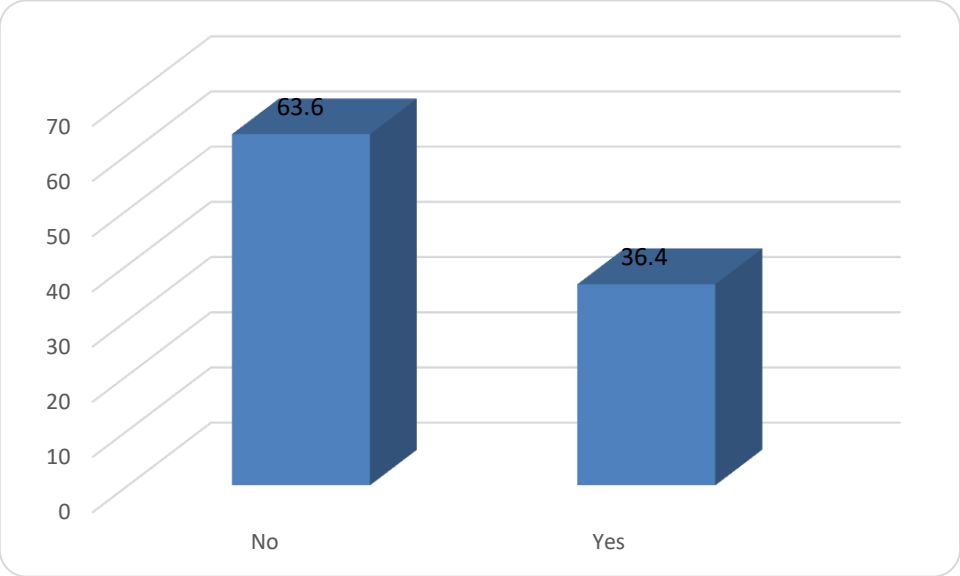


The occurrence of hypertension was reported in 40.9% of the study cases.

Table 6. Diabetes

Diabetes	Frequency	Percent
No	28	63.6
Yes	16	36.4
Total	44	100

Chart 6. Diabetes

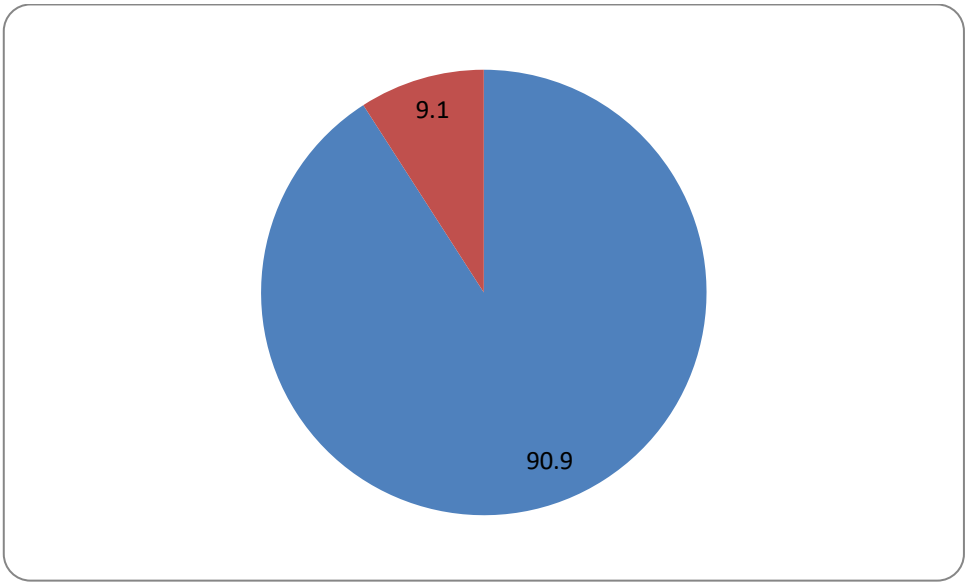


About 36.4% of the cases in this study had history of diabetes mellitus.

Table 7. ECG – LVH in study group

ECG - LVH	Frequency	Percent
No	40	90.9
Yes	4	9.1
Total	44	100

Chart 7. ECG – LVH in study group

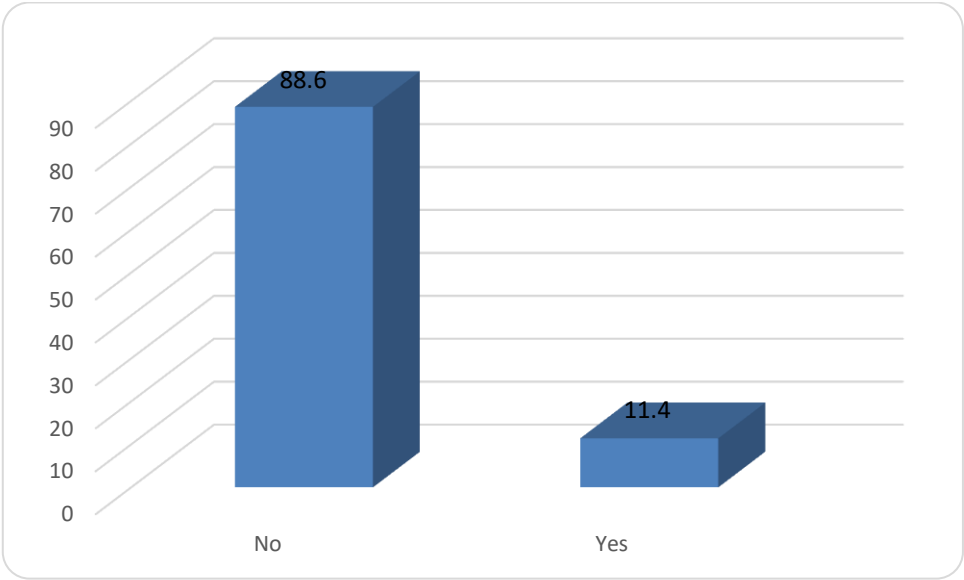


Left ventricular hypertrophy on Electrocardiogram was present in 9.1% of the cases in this study.

Table 8. Heart failure in the study group

Heart failure	Frequency	Percent
No	39	88.6
Yes	5	11.4
Total	44	100

Chart 8. Heart failure in the study group

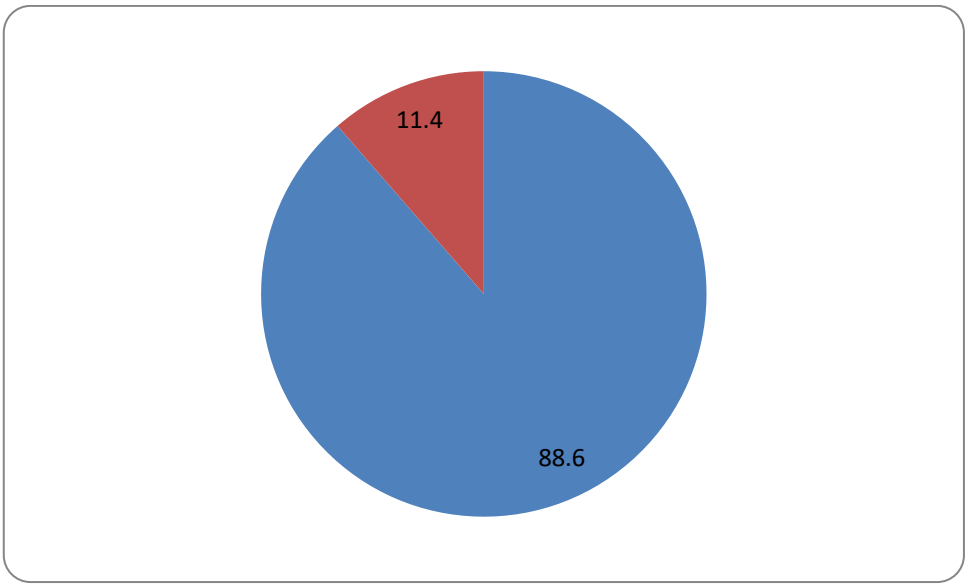


Heart failure was present in 11.4% of the cases in this study.

Table 9. Coronary artery syndrome in study group

Coronary artery syndrome	Frequency	Percent
No	39	88.6
Yes	5	11.4
Total	44	100

Chart 9. Coronary artery syndrome in study group

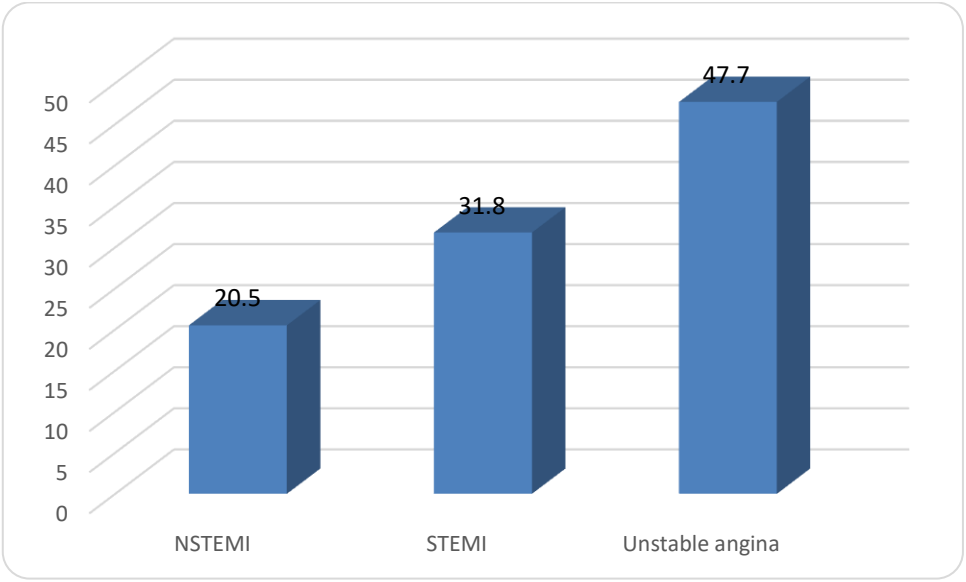


Coronary artery syndrome was present in 11.4% of the cases in this study.

Table 10. Type of ACS in study group

Type of ACS	Frequency	Percent
NSTEMI	9	20.5
STEMI	14	31.8
Unstable angina	21	47.7
Total	44	100

Chart 10. Type of ACS in study group

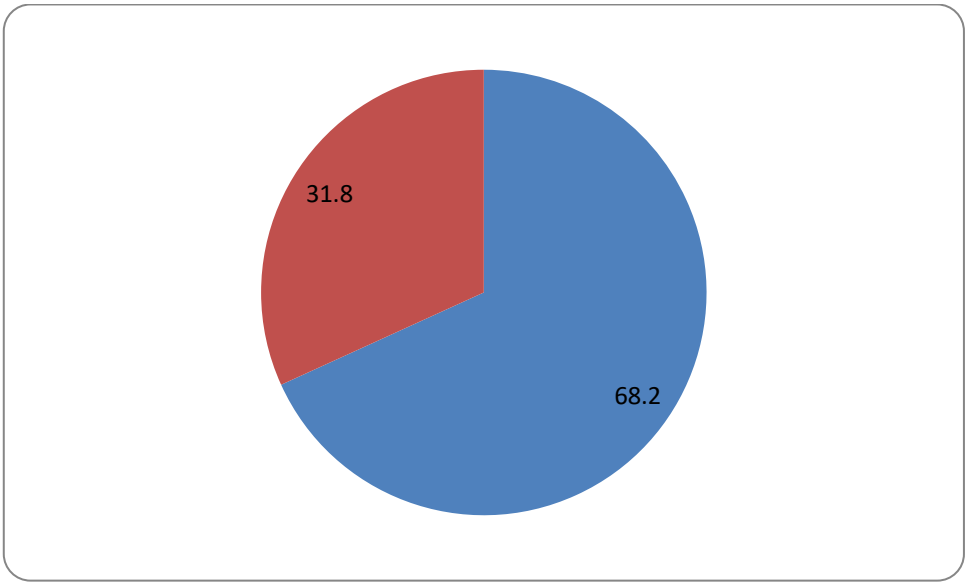


Unstable angina occurred in 47.7% of patient cases while STEMI cases amounted to 31.8% and NSTEMI accounted for 20.5% of the total patient population under investigation.

Table 11. Complications in study group

Complications	Frequency	Percent
No	30	68.2
Yes	14	31.8
Total	50	100

Chart 11. Complications in study group

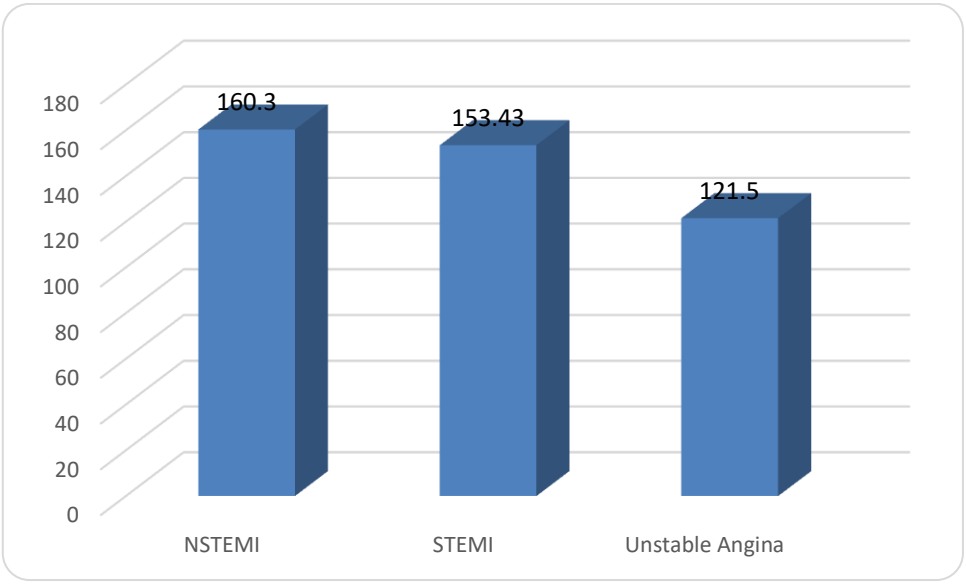


Complications were present in 31.8% of the cases in this study.

Table 12. Type of ACS and QT dispersion – Day - 0

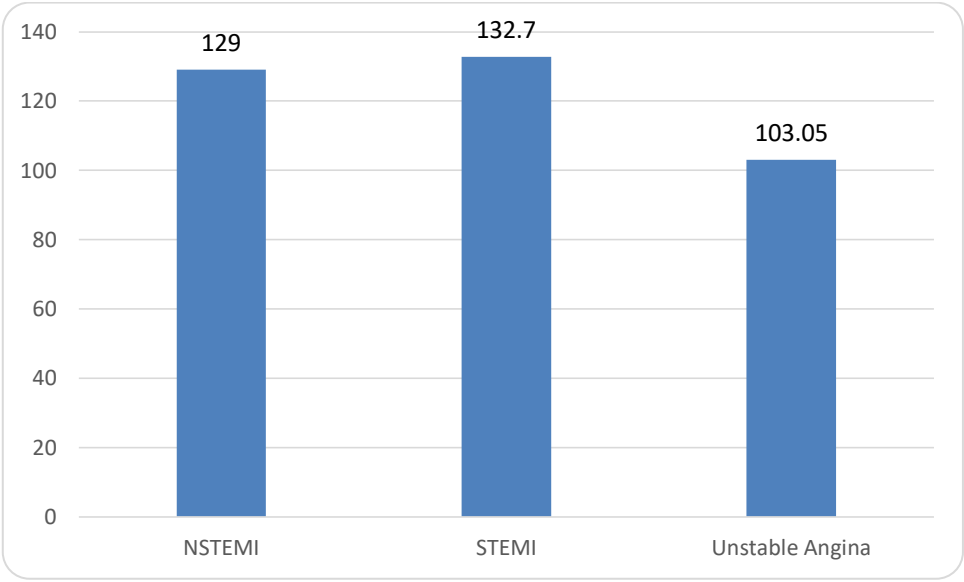
Type of ACS	QTd – Day 0	QTd – Day 1	QTd – Day 2	QTd – Day 7
	Mean ± SD	**Mean** ± SD	**Mean ± SD**	**Mean ± SD**
NSTEMI	160.3 ± 8.5	129.0 ± 6.8	115.4 ± 12.6	88.78 ± 11.0
STEMI	153.43 ± 6.3	132.7 ± 11.02	110.3 ± 8.36	85.3 ± 5.06
Unstable Angina	121.5 ± 9.35	103.05 ± 8.2	87.3 ± 13.2	79.62 ± 10.7
F value	96.68	54.587 ±	25.402	3.503
P value, Significance	0.000, Sig	0.000, Sig	0.000, Sig	0.039, Sig

Chart 12. Type of ACS and QT dispersion – Day – 0



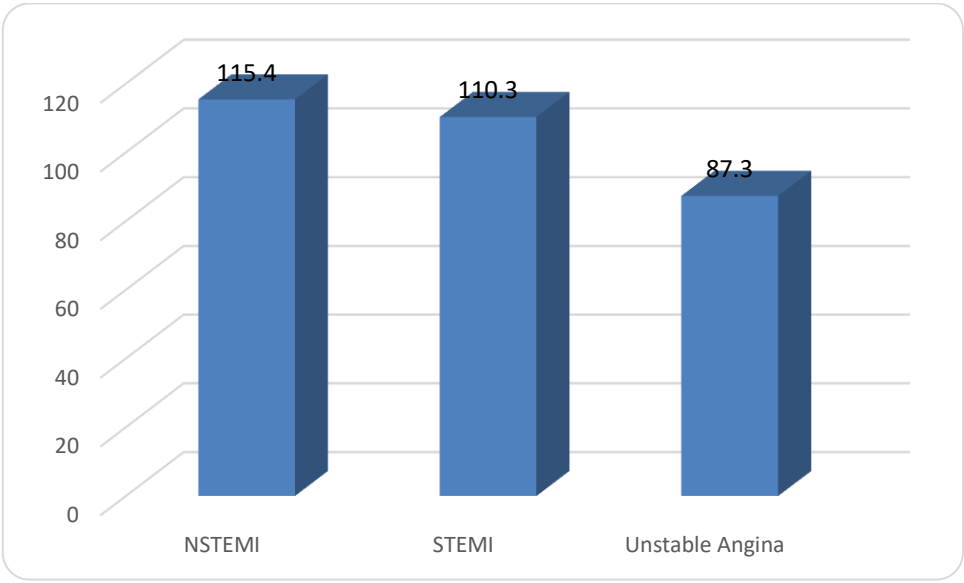
Mean QT dispersion in NSTEMI was 160.3, STEMI was 153.43 and unstable angina was 121.5 on Day 0. This difference was statistically significant between the three groups.

Chart 13. Type of ACS and QT dispersion – Day – 1



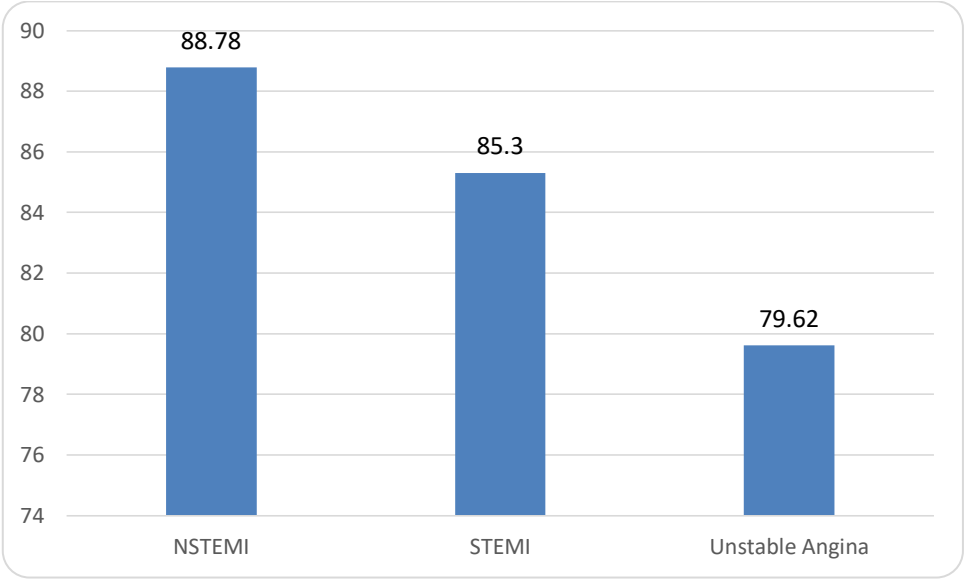
The QT dispersion was 129.0 in NSTEMI, 132.7 in STEMI and 103.05 in unstable angina cases in this study on Day 1. This difference was statistically significant.

Chart 14. Type of ACS and QT dispersion – Day – 2



Mean QT dispersion was 115.4in NSTEMI, 110.3 in STEMI and 87.3 in unstable angina cases in this study on Day 2. This difference was statistically significant.

Chart 15. Type of ACS and QT dispersion – Day – 7

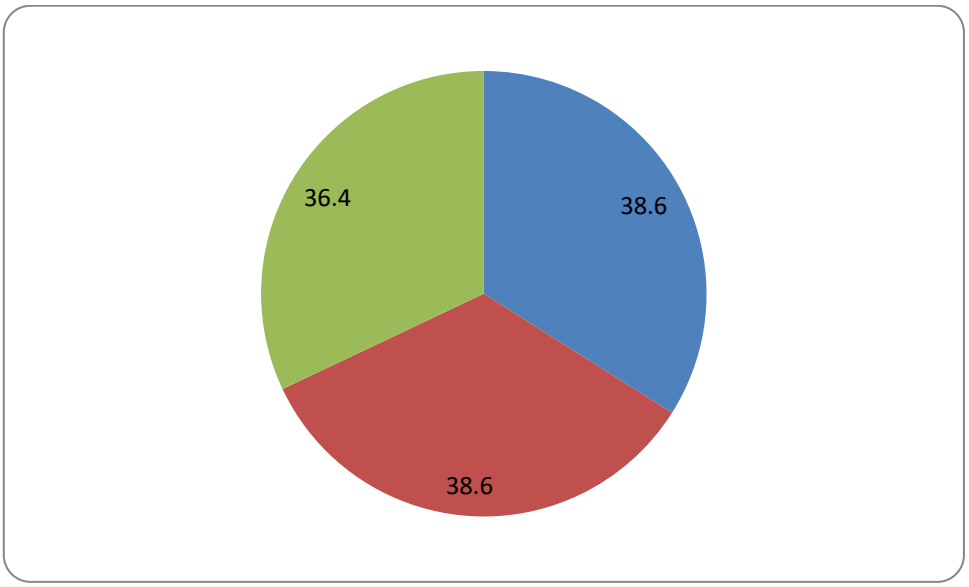


Mean QT dispersion on Day 7 was 88.78 in NSTEMI, 85.3 in STEMI and 79.62 in Unstable angina cases. This difference was statistically significant.

Table 13. Angiography findings

Angiography	Frequency	Percent
Single vessel disease	17	38.6
Double vessel disease	17	38.6
Triple vessel disease	16	36.4
Total	44	100

Chart 16. Angiography findings



Single vessel disease became evident on angiographic examinations across 38.6% of cases and double vessel disease occurred in 38.6% of cases but triple vessel disease appeared in 36.4% of cases from this study group.

Table 14. Correlation between Ejection fraction and QT dispersion

Correlations

		Ejection Fraction
Mean QTd - Day 0	Pearson Correlation	-.537**
	Sig. (2-tailed)	.000
	N	44
Mean QTd - Day 1	Pearson Correlation	-.308*
	Sig. (2-tailed)	.042
	N	44
Mean QTd - Day 2	Pearson Correlation	-.208
	Sig. (2-tailed)	.176
	N	44
Mean QTd - Day 7	Pearson Correlation	-.019
	Sig. (2-tailed)	.905
	N	44

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

The correlation between ejection fraction and QT dispersion was negative and significant on Day 0 and Day 1. It was negative and not significant on Day 2 and Day 7.

Table 15. Comparison of RWMA with QT dispersion

RWMA	QTd – Day 0	QTd – Day 1	QTd – Day 2	QTd – Day 7
	Mean ± SD	**Mean** ± SD	**Mean ± SD**	**Mean ± SD**
No	132.9 ± 19.1	112.8 ± 16.4	97.4 ± 17.1	80.9 ± 10.1
Yes	153.86 ± 10.7	128.5 ± 12.4	106.1 ± 16.5	88.4 ± 83.3
F value	14.487	10.114	2.552	6.138
P value, Significance	0.000, Sig	0.003, Sig	0.118, NS	0.017, Sig

The QT dispersion was higher in cases with right wall motion abnormalities in this study. This difference was statistically significant on Day 0, Day 1 and Day 7.

Table 15. Comparison of Angiogram findings with QT dispersion

Angiogram findings	**QTd – Day 0**	**QTd – Day 1**	**QTd – Day 2**	**QTd – Day 7**
	Mean ± SD	**Mean** ± SD	**Mean ± SD**	**Mean ± SD**
Single vessel disease	129.1 ± 16.1	110.5 ± 15.05	92.05 ± 16.3	80.3 ± 9.5
Double vessel disease	134.8 ± 17.4	116.9 ± 20.1	99.9 ± 16.8	82.3 ± 9.5
Triple vessel disease	159.8 ± 6.9	129.8 ± 8.3	112.9 ± 10.8	88.8 ± 9.1
F value	18.57	6.554	7.567	3.244
P value, Significance	0.000, Sig	0.003, Sig	0.002, Sig	0.049, Sig

People with coronary artery disease affecting all three major vessels displayed increased QT dispersion compared to patients with single or double coronary artery blockage. This difference was statistically significant.

DISCUSSION

DISCUSSION

The development of arrhythmia among ST-elevation myocardial infarction (STEMI) patients stands as the primary cause of mortality during their first hospital admission. Scientists established dispersion of repolarization as the leading trigger and principal substrate that initiates fatal arrhythmia in patients with ST elevation myocardial infarction.(107) QT dispersion (QTD) operates as an assessment tool to determine cardiac repolarization heterogeneity by computing maximal and minimal QT interval differences on standard 12-lead electrocardiograms. Studies have established that acute ischemia together with STEMI creates longer QT intervals and QTD measurements. The modifications likely represent ventricular excitability recovery patterns because this fundamental aspect becomes severely damaged during the first stages of an acute myocardial infarction (MI). The extension of QTD establishes an elevated risk for arrhythmia among patients who experience STEMI.(10)

The administration of fibrinolytic therapy remains essential for STEMI patients when PPCI is unavailable at the correct time despite its being the preferred treatment. The infusion of blood into an infarcted artery through both fibrinolytic therapy and PPCI procedure helps to minimize QTD by standardizing ventricular action potential duration. The efficacy of reperfusion procedures in STEMI patients to reduce QTD remains uncertain according to different research findings. Medical literature lacks data about how different reperfusion methods influence the occurrence of arrhythmias during hospital stay.(109)

The present research aims to measure QT dispersion while investigating its relationship to ECHO and angiographic results from patients suffering acute myocardial infarction (AMI). Another objective is to detect QT dispersion variability based on the myocardial infarction location.

Age group

The research involved forty-five percent five of the examined patients who fell within the age bracket of sixty-one to seventy. The mean patient age among patients receiving fibrinolytic treatment amounted to 59.1 years whereas PPCI recipients had an average age of 57.9 years according to Abdelmegid et al.(13) Chittora et al. also reported. women were 54 years old and men were 57 years old on average. Most of the instances were people between the ages of 51 and 60.106 The bulk of the cases in a research by Shashidhar et al. were over or equivalent to 50 years old.(110)

Gender

The research dataset included female participants at a level of 54.5%. The study conducted by Abdelmegid et al. revealed that male patients made up 80% of PPCI group admissions while the fibrinolytic group contained 77% male patients.(13) Thirteen Males dominated among PPCI group patients and male patients formed majority case numbers in research by Chittora et al. and Shashidhar et al.(110)

Smoking

Roughly 18.2% had previously smoked. According to Abdelmegid et al. patients treated with fibrinolytics displayed a history of smoking in 48.3% of cases while the rate was 40.8% among patients who underwent PPCI.(106)

Alcohol

About 11.4% of the cases in this study were alcoholics.

Hypertension

This study had shown that, 40.9% had hypertension. In a study by Abdelmegid et al, 29.2% of the cases in PPCI group and 35.8% of the cases in fibrinolytic group were hypertensives.(13)

Diabetes mellitus

Among patients, this research revealed diabetes mellitus diabetic status in 36.4% of participants. The research by Abdelmegid et al demonstrated that diabetes mellitus existed in 31.7% of PPCI patients and 33.3% of fibrinolytic group patients.(106)

Left ventricular hypertrophy

This study had shown that, 9.1% had left ventricular hypertrophy on electrocardiogram.

Heart failure

Heart failure occurred in 11.4% of the cases in this study.

Coronary artery syndrome

Coronary artery syndrome was detected in 11.4% of the cases in this investigation.

Type of ACS

This study demonstrated that unstable angina developed in 47.7% of patients with heart attack while STEMI occurred in 31.8% of the patients followed by NSTEMI in 20.5% of patients. The research by Chittora et al revealed that patients with unstable angina made up 30.8% of the cases while patients suffering from STEMI amounted to 44.9% and those with NSTEMI represented 24.3% of the cases.(103)

Complications

Complications occurred in 31.8% of the cases in this study. Most common complications included heart failure, hypotension and ventricular arrhythmia. A study by Chittora et al had shown that, congestive heart failure was common complication followed by hypotension, ventricular arrhythmia and death.(103)

QT dispersion with type of ACS

On day zero, the mean QT dispersion in NSTEMI was 160.3, STEMI was 153.43, and unstable angina was 121.5. This difference was statistically significant across the three groups. On day one of this investigation, the QT dispersion was 129.0 in NSTEMI, 132.7 in STEMI, and 103.05 in unstable angina cases. The difference was statistically significant. On Day 2, the mean QT dispersion was 115.4 in NSTEMI, 110.3 in STEMI, and 87.3 in unstable angina patients. The difference was statistically significant. On Day 7, the mean QT dispersion was 88.78 in NSTEMI, 85.3 in STEMI, and 79.62 in unstable angina cases. The difference was statistically significant. Normogram QT measurement reported 407.1 msec maximum value in PPCI patients but revealed 398.7 msec maximum in individuals undergoing fibrinolysis therapy according to Abdelmegid et al. The minimum QT measurements reached 325.4% in PPCI patients while the fibrinolytic group measured 341.3 msec. The study observed QTD results measured in msec at 81.8 for PPCI cases and 57.3 for fibrinolytic patients.(13) The mean QTcd measurement was 122.97 msec in STEMI cases with NSTEMI showing 158.44 msec. The research outcomes revealed that NSTEMI together with STEMI result in larger QTcd increases than unstable angina does. Results showed that thrombolysis administration caused a major reduction in QTcd duration compared to treatments without thrombolysis. Research by Shashidhar et al showed myocardial infarction presented with QTd of 80.71 ms above the normal upper range of 42 msec.(110)

Angiography

Single vessel disease appeared in 38.6% of patients and double vessel disease and triple vessel disease was seen in 38.6% and 36.4% of cases throughout this study based on angiographic evaluation.

QT dispersion with ejection fraction

On Days 0 and 1, there was a negative and substantial connection between the ejection fraction and QT dispersion. On Days 2 and 7, it was negative and not really noticeable. In a study by Pai et al, the QTc interval was negatively correlated with ejection fraction.(111)

QT dispersion with RWMA

The subjects with right wall motion abnormalities displayed increased QT dispersion as an investigation result. Right wall motion anomalies generated statistically significant differences on the testing dates of Day 0, Day 1 and Day 7.

QT dispersion with Angiography

The presence of triple vessel disease produced a greater QT dispersion than single and double vascular disease conditions. Triple vessel disease showed higher QT dispersion than single or double vascular diseases with a significant statistical relationship between them.

LIMITATIONS

LIMITATIONS

- Comparison group was not present in this study
- Sample size was not calculated and
- Sample chosen by convenience.

CONCLUSION

CONCLUSION

The purpose of this study was to research QT dispersion in patients with myocardial infarction. This study had shown that, majority of the cases belonged to were aged between 61 to 70 years and female sex. The QT dispersion in STEMI and NSTEMI cases was higher than the cases with unstable angina. The QT dispersion decreased with follow up and lower on day 7. But this study was able bring out important facts about QT dispersion and its importance in prognosis in cases with acute myocardial infarction. Further research on this path will reveal additional information about applying QT dispersion for acute myocardial infarction diagnosis.

BIBLIOGRAPHY

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ANNEXURE

PROFORMA

A STUDY ON QT DISPERSION IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION AND ITS CORRELATION WITH ECHO AND ANGIOGRAM FINDINGS

NAME		
AGE		
GENDER		
DATE OF ADMISSION		
PRESENTING COMPLIANTS		
If Yes then details about treatment history		
Treatment		
CORMORDBIDITES		
DURATION OF STAY IN HOSPITAL		
INVESTIGATIONS	**1.ECG** **2. 2D ECHO** **3. ANGIOGRAM** **4.TROPONIN I** **5.SERUM CALCIUM** **6.SERUM MAGNESIUM** **7.SERUM POTASSIUM**	

INFORMED CONSENT FORM

Date:

I, Mr/Mrs _____ ,have been explained in my own vernacular language that I will be included in "A Study On QT dispersion In patients with Acute Myocardial Infarction and its correlation with ECHO and angiogram findings", hereby I give my valid written informed consent without any force or prejudice for recording the observations of haematological and clinical parameters . The nature and risks involved have been explained to me, to my satisfaction. I have been explained in detail about the study being conducted. I have read the patient information sheet and I have had the opportunity to ask any question. Any question that I have asked, have been answered to my satisfaction. I provide consent voluntarily to allow myself / my relative as a participant in this research. I hereby give consent to provide history, undergo physical examination, undergo the procedure, undergo investigations and provide its results and documents etc to the doctor / institute etc. For academic and scientific purpose the operation / procedure, etc may be video graphed or photographed. All the data may be published or used for any academic purpose.

_____

Name of Patient/Guardian

(Relation with patient)

_____

(Signature of Patient / Attendant)

(Signature & Name of Research doctor)

ಮಾಹಿತಿಯುಕ್ತ ಒಪ್ಪಿಗೆ ನಮೂನೆ

ದಿನಾಂಕ:

ನನ್ನ ಸ್ವಂತ ಸ್ಥಳೀಯ ಭಾಷೆಯಲ್ಲಿ, ಶ್ರೀ/ಶ್ರೀಮತಿ _____, "ತೀವ್ರ ಮಯೋಕಾರ್ಡಿಯಲ್ ಇನ್ಫಾರ್ಕ್ಷನ್ ಹೊಂದಿರುವ ರೋಗಿಗಳಲ್ಲಿ QT ಪ್ರಸರಣ ಮತ್ತು ECHO ಮತ್ತು ಆಂಜಿಯೋಗ್ರಾಮ್ ಸಂಶೋಧನೆಗಳೊಂದಿಗೆ ಅದರ ಪರಸ್ಪರ ಸಂಬಂಧದ ಕುರಿತಾದ ಅಧ್ಯಯನ"ದಲ್ಲಿ ನನ್ನನ್ನು ಸೇರಿಸಲಾಗುವುದು ಎಂದು ವಿವರಿಸಲಾಗಿದೆ, ಈ ಮೂಲಕ ನಾನು ಹೆಮಟೊಲಾಜಿಕಲ್ ಮತ್ತು ಕ್ಲಿನಿಕಲ್ ನಿಯತಾಂಕಗಳ ಅವಲೋಕನಗಳನ್ನು ದಾಖಲಿಸಲು ಯಾವುದೇ ಬಲ ಅಥವಾ ಪೂರ್ವಾಗ್ರಹವಿಲ್ಲದೆ ನನ್ನ ಮಾನ್ಯ ಲಿಖಿತ ಮಾಹಿತಿಯುಕ್ತ ಒಪ್ಪಿಗೆಯನ್ನು ನೀಡುತ್ತೇನೆ. ಒಳಗೊಂಡಿರುವ ಸ್ವರೂಪ ಮತ್ತು ಅಪಾಯಗಳನ್ನು ನನಗೆ ತೃಪ್ತಿಪಡಿಸುವಂತೆ ವಿವರಿಸಲಾಗಿದೆ.

ನಡೆಸಲಾಗುತ್ತಿರುವ ಅಧ್ಯಯನದ ಬಗ್ಗೆ ನನಗೆ ವಿವರವಾಗಿ ವಿವರಿಸಲಾಗಿದೆ. ನಾನು ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆಯನ್ನು ಓದಿದ್ದೇನೆ ಮತ್ತು ಯಾವುದೇ ಪ್ರಶ್ನೆಯನ್ನು ಕೇಳಲು ನನಗೆ ಅವಕಾಶ ಸಿಕ್ಕಿದೆ. ನಾನು ಕೇಳಿದ ಯಾವುದೇ ಪ್ರಶ್ನೆಗೆ ನನ್ನ ತೃಪ್ತಿಗೆ ಉತ್ತರಿಸಲಾಗಿದೆ. ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ಭಾಗವಹಿಸಲು ನನ್ನನ್ನು / ನನ್ನ ಸಂಬಂಧಿಯನ್ನು ಅನುಮತಿಸಲು ನಾನು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಒಪ್ಪಿಗೆ ನೀಡುತ್ತೇನೆ.

ಇತಿಹಾಸವನ್ನು ಒದಗಿಸಲು, ದೈಹಿಕ ಪರೀಕ್ಷೆಗೆ ಒಳಗಾಗಲು, ಕಾರ್ಯವಿಧಾನಕ್ಕೆ ಒಳಗಾಗಲು, ತನಿಖೆಗಳಿಗೆ ಒಳಗಾಗಲು ಮತ್ತು ಅದರ ಫಲಿತಾಂಶಗಳು ಮತ್ತು ದಾಖಲೆಗಳನ್ನು ಇತ್ಯಾದಿಗಳನ್ನು ವೈದ್ಯರು / ಸಂಸ್ಥೆಗೆ ಒದಗಿಸಲು ನಾನು ಈ ಮೂಲಕ ಒಪ್ಪಿಗೆ ನೀಡುತ್ತೇನೆ. ಶೈಕ್ಷಣಿಕ ಮತ್ತು ವೈಜ್ಞಾನಿಕ ಉದ್ದೇಶಗಳಿಗಾಗಿ ಕಾರ್ಯಾಚರಣೆ / ಕಾರ್ಯವಿಧಾನ ಇತ್ಯಾದಿಗಳನ್ನು ವೀಡಿಯೊ ಗ್ರಾಫ್ ಮಾಡಬಹುದು ಅಥವಾ ಛಾಯಾಚಿತ್ರ ಮಾಡಬಹುದು. ಎಲ್ಲಾ ಡೇಟಾವನ್ನು ಯಾವುದೇ ಶೈಕ್ಷಣಿಕ ಉದ್ದೇಶಕ್ಕಾಗಿ ಪ್ರಕಟಿಸಬಹುದು ಅಥವಾ ಬಳಸಬಹುದು.

_____

ರೋಗಿಯ/ಪೋಷಕರ ಹೆಸರು

(ರೋಗಿಯೊಂದಿಗಿನ ಸಂಬಂಧ)

_____

(ರೋಗಿಯ/ಪರಿಚಾರಕರ ಸಹಿ)

(ಸಂಶೋಧನಾ ವೈದ್ಯರ ಸಹಿ ಮತ್ತು ಹೆಸರು)

PATIENT INFORMATION SHEET

Study title : A Study on QT dispersion in patients with Acute Myocardial Infarction and its correlation with ECHO and Angiogram findings

Principal investigator: Dr AMULYA /Dr.PRABHAKAR/Dr. YASWANTH

I Dr.Amulya , Post graduate student in Department of general medicine at Sri Devraj Urs Medical College, will be conducting a study titled "A Study on QT dispersion in patients with Acute Myocardial Infarction and its correlation with ECHO and Angiogram findings". This study will be useful for prognosis and management in Acute MI patients. The investigations done for the study are – ECG, 2D ECHO, Angiogram, Troponin I, Serum Calcium, Serum Magnesium, Serum Potassium. The funds needed for the investigations will be done at my own expense. All the data will be kept confidential and will be used for research and presentation purposes. You are free to provide consent for the participation of yourself in this study. You can also withdraw yourself from the study at any point of time without giving any reasons whatsoever. Your refusal to participate will not prejudice you to present or future care at this institution. The information collected will be used for research, presentation in conferences and presentation in medical journals.

In case of any clarifications are needed you are free to contact me on this mobile number - 6305682144

Name and Signature of the Principal Investigator

Date-

Patient or patient bystanders Signature

ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆ

ಅಧ್ಯಯನ ಶೀರ್ಷಿಕೆ: ತೀವ್ರವಾದ ಮಯೋಕಾರ್ಡಿಯಲ್ ಇನ್ಫಾರ್ಕ್ಷನ್ ಹೊಂದಿರುವ ರೋಗಿಗಳಲ್ಲಿ QT ಪ್ರಸರಣ ಮತ್ತು ECHO ಮತ್ತು ಆಂಜಿಯೋಗ್ರಾಮ್ ಸಂಶೋಧನೆಗಳೊಂದಿಗೆ ಅದರ ಸಂಬಂಧದ ಕುರಿತು ಅಧ್ಯಯನ

ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿ: ಡಾ. ಅಮೂಲ್ಯ / ಡಾ. ಪ್ರಭಾಕರ್ / ಡಾ. ಯಸ್ವಂತ್ ಶ್ರೀ ದೇವರಾಜ್ ಉರ್ಸ್ ವೈದ್ಯಕೀಯ ಕಾಲೇಜಿನಲ್ಲಿ ಜನರಲ್ ಮೆಡಿಸಿನ್ ವಿಭಾಗದಲ್ಲಿ ಸ್ನಾತಕೋತ್ತರ ವಿದ್ಯಾರ್ಥಿನಿ ಡಾ. ಅಮೂಲ್ಯ ಅವರು "ತೀವ್ರವಾದ ಮಯೋಕಾರ್ಡಿಯಲ್ ಇನ್ಫಾರ್ಕ್ಷನ್ ಹೊಂದಿರುವ ರೋಗಿಗಳಲ್ಲಿ QT ಪ್ರಸರಣ ಮತ್ತು ECHO ಮತ್ತು ಆಂಜಿಯೋಗ್ರಾಮ್ ಸಂಶೋಧನೆಗಳೊಂದಿಗೆ ಅದರ ಸಂಬಂಧದ ಕುರಿತು ಅಧ್ಯಯನ" ಎಂಬ ಶೀರ್ಷಿಕೆಯ ಅಧ್ಯಯನವನ್ನು ನಡೆಸಲಿದ್ದಾರೆ. ಈ ಅಧ್ಯಯನವು ತೀವ್ರವಾದ MI ರೋಗಿಗಳಲ್ಲಿ ಮುನ್ನರಿವು ಮತ್ತು ನಿರ್ವಹಣೆಗೆ ಉಪಯುಕ್ತವಾಗಿರುತ್ತದೆ.

ಅಧ್ಯಯನಕ್ಕಾಗಿ ಮಾಡಲಾದ ತನಿಖೆಗಳು - ECG, 2D ECHO, ಆಂಜಿಯೋಗ್ರಾಮ್, ಟ್ರೋಪೋನಿನ್ I, ಸೀರಮ್ ಕ್ಯಾಲ್ಸಿಯಂ, ಸೀರಮ್ ಮೆಗ್ನೀಸಿಯಮ್, ಸೀರಮ್ ಪೊಟ್ಯಾಸಿಯಮ್. ತನಿಖೆಗಳಿಗೆ ಅಗತ್ಯವಿರುವ ಹಣವನ್ನು ನನ್ನ ಸ್ವಂತ ಖರ್ಚಿನಲ್ಲಿ ಮಾಡಲಾಗುತ್ತದೆ. ಎಲ್ಲಾ ಡೇಟಾವನ್ನು ಗೌಪ್ಯವಾಗಿಡಲಾಗುತ್ತದೆ ಮತ್ತು ಸಂಶೋಧನೆ ಮತ್ತು ಪ್ರಸ್ತುತಿ ಉದ್ದೇಶಗಳಿಗಾಗಿ ಬಳಸಲಾಗುತ್ತದೆ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನಿಮ್ಮ ಭಾಗವಹಿಸುವಿಕೆಗೆ ನೀವು ಒಪ್ಪಿಗೆಯನ್ನು ನೀಡಲು ಸ್ವತಂತ್ರರು. ಯಾವುದೇ ಕಾರಣಗಳನ್ನು ನೀಡದೆ ನೀವು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಿಂದ ಹಿಂದೆ ಸರಿಯಬಹುದು. ಭಾಗವಹಿಸಲು ನಿಮ್ಮ ನಿರಾಕರಣೆಯು ಈ ಸಂಸ್ಥೆಯಲ್ಲಿ ಪ್ರಸ್ತುತ ಅಥವಾ ಭವಿಷ್ಯದ ಆರೈಕೆಗೆ ಯಾವುದೇ ಹಾನಿ ಮಾಡುವುದಿಲ್ಲ. ಸಂಗ್ರಹಿಸಿದ ಮಾಹಿತಿಯನ್ನು ಸಂಶೋಧನೆ, ಸಮ್ಮೇಳನಗಳಲ್ಲಿ ಪ್ರಸ್ತುತಿ ಮತ್ತು ವೈದ್ಯಕೀಯ ನಿಯತಕಾಲಿಕಗಳಲ್ಲಿ ಪ್ರಸ್ತುತಿಗಾಗಿ ಬಳಸಲಾಗುತ್ತದೆ.

ಯಾವುದೇ ಸ್ಪಷ್ಟೀಕರಣಗಳ ಅಗತ್ಯವಿದ್ದರೆ ನೀವು ಈ ಮೊಬೈಲ್ ಸಂಖ್ಯೆ - 6305682144 ನಲ್ಲಿ ನನ್ನನ್ನು ಸಂಪರ್ಕಿಸಬಹುದು.

ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿಯ ಹೆಸರು ಮತ್ತು ಸಹಿ

ದಿನಾಂಕ-

ರೋಗಿ ಅಥವಾ ರೋಗಿಯ ಪಕ್ಕದಲ್ಲಿರುವವರ ಸಹಿ

MASTER CHART

Sl no	Age	Sex	Smoking	Alcohol	Hypertension	Diabetes	ECG - LVH	Heart failure	Coronary artery disease	Type of ACS	Complications	Mean QTd - Day 0	Mean QTd - Day 1	Mean QTd - Day 2	Mean QTd - Day 7	Ejection Fraction	RWMA	Angiogram findings
1	45	F	No	No	No	No	No	No	No	STEMI	No	149	147	120	82	60	No	Double Vessel disease
2	46	M	Yes	No	No	Yes	No	No	No	NSTEMI	No	167	126	128	99	28	Yes	Triple vessel disease
3	64	M	No	No	Yes	No	No	No	Yes	Unstable Angina	Yes	118	116	107	79	55	No	Double Vessel disease
4	52	M	Yes	Yes	No	No	No	No	No	STEMI	No	162	118	96	91	25	Yes	Triple vessel disease
5	62	F	No	No	No	Yes	Yes	Yes	No	Unstable Angina	No	108	109	94	62	59	No	Single vessel disease
6	64	F	No	No	Yes	No	No	No	No	Unstable Angina	No	127	92	81	75	48	No	Single vessel disease
7	58	M	No	No	No	No	No	No	No	Unstable Angina	Yes	113	110	101	62	48	No	Double Vessel disease
8	69	F	No	No	No	No	No	No	No	Unstable Angina	No	129	99	76	91	55	No	Single vessel disease
9	64	M	No	Yes	No	Yes	No	No	No	STEMI	No	157	115	120	80	45	No	Double Vessel disease
10	55	M	Yes	No	Yes	No	No	No	No	NSTEMI	No	151	136	124	101	56	Yes	Triple vessel disease
11	63	F	No	No	No	No	No	No	No	Unstable Angina	Yes	123	99	90	81	45	No	Single vessel disease
12	66	F	No	No	No	Yes	No	No	No	STEMI	No	158	128	119	79	38	No	Triple vessel disease
13	65	F	No	No	Yes	No	Yes	No	No	Unstable Angina	No	135	109	76	92	54	Yes	Single vessel disease
14	64	F	No	No	No	No	No	No	No	NSTEMI	Yes	168	132	103	84	38	No	Triple vessel disease
15	55	M	No	No	No	Yes	No	No	Yes	STEMI	No	154	142	107	79	39	No	Single vessel disease
16	72	F	No	No	Yes	No	No	No	No	Unstable Angina	No	118	101	100	90	48	No	Single vessel disease
17	64	M	Yes	Yes	No	No	No	Yes	No	Unstable Angina	No	130	108	101	84	64	No	Single vessel disease
18	73	F	No	No	No	Yes	No	No	No	STEMI	Yes	148	123	107	84	64	No	Single vessel disease
19	65	M	No	No	Yes	No	No	No	No	NSTEMI	No	153	120	96	97	35	Yes	Double Vessel disease
20	52	F	No	No	No	Yes	No	No	No	STEMI	No	144	137	112	84	51	No	Single vessel disease
21	48	M	No	No	Yes	No	No	No	Yes	Unstable Angina	Yes	114	119	86	87	46	No	Single vessel disease
22	53	M	No	No	No	No	Yes	No	No	Unstable Angina	No	120	102	72	75	61	No	Single vessel disease

Sl no	Age	Sex	Smoking	Alcohol	Hypertension	Diabetes	ECG - LVH	Heart failure	Coronary artery disease	Type of ACS	Complications	Mean QTd - Day 0	Mean QTd - Day 1	Mean QTd - Day 2	Mean QTd - Day 7	Ejection Fraction	RWMA	Angiogram findings
23	57	F	No	No	No	Yes	No	No	No	NSTEMI	Yes	152	134	108	78	29	Yes	Triple vessel disease
24	56	M	No	No	Yes	No	No	No	No	STEMI	No	153	118	99	89	28	Yes	Triple vessel disease
25	85	F	No	No	No	Yes	No	No	No	Unstable Angina	No	125	99	84	91	61	No	Double Vessel disease
26	65	M	Yes	Yes	Yes	No	No	No	No	Unstable Angina	Yes	109	85	106	84	56	No	Double Vessel disease
27	48	M	No	No	No	Yes	Yes	No	No	STEMI	No	144	134	103	82	38	No	Single vessel disease
28	46	F	No	No	Yes	No	No	No	No	NSTEMI	No	166	128	131	81	25	Yes	Single vessel disease
29	85	F	No	No	No	Yes	No	Yes	No	Unstable Angina	Yes	111	101	71	91	58	No	Single vessel disease
30	75	M	No	No	Yes	No	No	No	No	STEMI	No	148	146	113	84	28	Yes	Double Vessel disease
31	63	F	No	No	No	Yes	No	No	No	Unstable Angina	No	130	108	75	71	54	No	Double Vessel disease
32	69	F	No	No	Yes	No	No	No	No	NSTEMI	Yes	170	117	110	73	35	No	Triple vessel disease
33	42	M	Yes	No	No	No	No	No	Yes	STEMI	No	162	129	122	96	36	No	Triple vessel disease
34	55	F	No	No	Yes	Yes	No	No	No	Unstable Angina	No	132	108	78	77	29	Yes	Single vessel disease
35	59	M	No	Yes	Yes	No	Yes	No	No	NSTEMI	Yes	150	133	128	102	55	No	Triple vessel disease
36	62	M	Yes	No	No	No	No	No	Yes	STEMI	No	156	145	104	87	44	Yes	Triple vessel disease
37	61	F	No	No	Yes	Yes	No	No	No	Unstable Angina	Yes	109	109	74	62	45	No	Single vessel disease
38	52	F	No	No	No	No	No	No	No	STEMI	No	162	136	116	91	45	Yes	Triple vessel disease
39	62	F	No	No	Yes	No	No	No	No	Unstable Angina	Yes	125	103	101	90	69	No	Single vessel disease
40	64	F	No	No	No	No	No	No	No	Unstable Angina	No	130	100	71	88	32	No	Double Vessel disease
41	58	M	No	No	Yes	Yes	No	Yes	No	Unstable Angina	No	109	91	78	78	41	No	Single vessel disease
42	69	F	No	No	No	No	No	No	No	STEMI	Yes	151	140	106	87	42	Yes	Double Vessel disease
43	64	M	Yes	No	Yes	No	No	No	No	Unstable Angina	No	136	96	103	62	42	No	Single vessel disease
44	55	F	No	No	No	Yes	Yes	No	No	NSTEMI	No	166	135	111	84	35	Yes	Triple vessel disease