A STUDY OF FRONTAL QRST ANGLE IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION

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LIST OF ABBREVIATIONS USED

ACS	:	ACUTE CORONARY SYNDROME
CS	:	CARDIOGENIC SHOCK
ECG	:	ELECTROCARDIOGRAPHY
FQRST	:	FRONTAL QRST ANGLE
HF	:	HEART FAILURE
LA	:	LEFT ATRIUM
LAD	:	LEFT ANTERIOR DESCENDING ARTERY
LCX	:	LEFT CIRCUMFLEX ARTERY
LV	:	LEFT VENTRICLE
LVEF	:	LEFT VENTRICULAR EJECTION FRACTION
MI	:	MYOCARDIAL INFARCTION
MACE	:	MAJOR ADVERSE CARDIAC EVENTS
NSTEMI	:	NON ST ELEVATION MYOCARDIAL INFARCTION
PE	:	PULMONARY EDEMA
RCA	:	RIGHT CORONARY ARTERY
RWMA	:	REGIONAL WALL MOTION ABNORMALITY
STEMI	:	ST ELEVATION MYOCARDIAL INFARCTION
VPC	:	VENTRICULAR PREMATURE COMPLEXES
VT	:	VENTRICULAR TACHYCARDIA
UA	:	UNSTABLE ANGINA

ABSTRACT

BACKGROUND-

Acute myocardial infarction has reached an epidemic proportion in the Indian population. Premature onset of acute myocardial infarction has shifted the focus on research. The ECG is a highly effective tool for determining a patient's level of risk for heart morbidity and mortality. Recent research has determined frontal QRST angle as a helpful indicator of dispersion between depolarization and repolarization. It can be easily calculated from a 12 lead ECG. Studies have shown the frontal QRST angle is a parameter to identify patients at high risk during acute myocardial infarction.

AIMS AND OBJECTIVES-

To study the Frontal QRST angle as a predictor of the in-hospital outcome of adverse cardiac events in patients with acute myocardial infarction.

MATERIALS AND METHOD-

A prospective observational study was carried out on 95 patients admitted in BLDE (Deemed to be university), Shri B.M. Patil Medical College Hospital and Research Centre, with a diagnosis of acute myocardial infarction considering the inclusion and exclusion criteria. Patients with acute coronary syndrome had their clinical, electrocardiographic, laboratory, and in-hospital outcomes evaluated as part of the work up. ECG on admission was used to calculate the Frontal QRST angle. Patients were classified into two groups, one with angle of <100 degrees as group A (n=68), and the other with an angle of >100 degrees as group B (n=27).The

patients were monitored for the emergence of serious adverse cardiac events like heart failure, pulmonary edema, cardiogenic shock, arrhythmias, and death while they were hospitalized.

RESULTS-

Total of 102 patients of acute coronary syndrome were studied out of which seven patients were excluded based on the exclusion criteria of which four patients had Left bundle branch block (LBBB), two had unstable angina and one patient had Right bundle branch block (RBBB). Hence total of 95 patients were included in the study. Out of 95 patients with acute coronary syndrome, 68 patients who had FQRST angle <100 degrees were in group A and 27 patients with FQRST angle >100 degrees were in group B.

The most common age group in group A was 60-70 years and group B was 50-60 years and both groups had male preponderance. The most common occupation in both groups was found to be Farmer. In both group A and group B the most common symptom was chest pain (97.1% vs 100%). In group B reduced left ventricular ejection fraction was seen.

When major adverse cardiac events were examined, it was shown that there was a significant difference between the two groups with a P value of < 0.05. Heart failure was found in 8.8% patients in group A and 77.8% patients in Group B. 10.3% of patients in group A and 77.8% of patients in group B had pulmonary edema, respectively. Compared to 40.7% of patients in group B, cardiogenic shock was discovered in 7.4% of patients in group A. Therefore, in patients with acute myocardial infarction, a frontal QRST angle of greater than 100 degrees was associated with the development of significant adverse outcomes.

CONCLUSION:

A strong predictor of in-hospital major adverse cardiac outcomes such heart failure, pulmonary edema, and cardiogenic shock was shown to be a frontal qrst angle of >100 degrees in this study. Hence a Frontal QRST angle of >100 degrees on a 12 lead ECG is cost-effective, reliable, and non-invasive predictor of major adverse cardiac events in patients with acute myocardial infarction.

KEYWORDS-

frontal qrst, acute myocardial infarction, major adverse cardiac events, acute coronary syndrome, heart failure, cardiogenic shock

INTRODUCTION

I. INTRODUCTION

The top cause of death in the world is ischemic heart disease. Ischemic heart disease was responsible for 7.1 million fatalities globally in 2001.[1]

In developing nations, acute myocardial infarction is a substantial risk factor for morbidity and mortality. The diagnosis and prognosis of acute myocardial infarction are directly connected to ECG alterations. India, the second most populous nation in the world, is a place of extreme diversity. Factors such as geography, race, culture, literacy, infrastructure and economy cause serious challenge in the management of acute myocardial infarction. According to data from the World Health Organization, the incidence of acute myocardial infarction is rapidly changing in India. [2]

As the leading cause of death in India, acute myocardial infarction has already surpassed communicable diseases. That around three million cases of ST- segment elevation myocardial infarction (STEMI) alone are burdensome each year. [3]

Acute coronary syndrome is term used to denote three conditions, which are, Unstable Angina (UA), Non-ST-segment elevation myocardial infarction (NSTEMI) and ST segment elevation myocardial infarction (STEMI) [4]. Unstable angina is similar to NSTEMI but cardiac enzymes are not elevated. Although all the three conditions have similar pathophysiology leading to oxygen demand and supply mismatch, but in STEMI there is total occlusion by intra coronary thrombus compared to unstable angina and NSTEMI, where there is partial occlusion.[5]

Many predisposing risk factors have been proven for Acute myocardial infarction, which are non-modifiable risk factors like age, sex, ethnicity, family history, genetic factors and modifiable factors such as hypertension, diabetes mellitus, smoking/tobacco use, obesity

and diet.[6] The complications such as heart failure, cardiogenic shock, pulmonary edema, arrhythmias and re-infarction are considered as major adverse cardiac events.[7] Hence to avoid such complications it is very much essential to predict them well in advance in patients at risk and manage accordingly.

Predicting the likelihood of serious adverse cardiovascular events and mortality in patients who have experienced an acute myocardial infarction aids in developing both immediate and short-term treatment plans. Population based studies have shown that certain electrocardiographic variables can be used for clinical risk stratification for major adverse cardiac events. [8]

An electrocardiography machine is a valuable tool in the diagnosis of acute myocardial infarction. The ECG, which is affordable, non-invasive, quick to do, and rapid to get results, has shown to be highly effective in categorising patients to a greater or lesser degree of risk for a range of cardiac morbidity and overall mortality. [9]

In the past, many literatures has shown that regional myocardial lesion is associated with changes in QRS complex configuration that can cause change in frontal QRST angle on 12 lead ECG. A relevant electrocardiographic measure of the dispersion between depolarization and repolarization, according to recent research, is the frontal QRST angle [10].

Additionally, it has been demonstrated that a higher spatial QRS-T angle is linked to higher mortality in the general population[10].A specialized software is required for measuring the spatial QRST angle as it is not routinely measured on 12 lead electrocardiographic machines. On the other hand, Frontal QRST is easy to calculate from a routine 12 lead ECG and no specialized software is needed. Studies have revealed a substantial link between the frontal and spatial QRST angles [11].

Therefore, an abnormal Frontal QRST angle can be used as a marker of acute myocardial infarction and as well as to predict major adverse cardiac events like heart failure, complex ventricular arrhythmias (sustained ventricular tachycardia or ventricular fibrillation), early post infarction angina, or mechanical complication, and cardiac death as a consequence of acute myocardial infarction.

AIMS AND OBJECTIVES

II. AIMS AND OBJECTIVES

To study the Frontal QRST angle as a predictor of the in-hospital outcome of the adverse cardiac events in patients with acute myocardial infarction.

REVIEW OF LITERATURE

III. REVIEW OF LITERATURE

The world's biggest factor in morbidity and mortality is myocardial infarction. Coronary atherosclerosis is a chronic condition with phases of stability and instability. Patients may experience a myocardial infarction when there are unstable periods of activated inflammation in the arterial wall. Myocardial infarction can be a little, undiscovered incident in a lifetime chronic condition, or it can be a huge catastrophe resulting in abrupt death or severe hemodynamic decline.[12]

More than 3 million patients each year experience an acute ST-elevation myocardial infarction (STEMI), while more than 4 million get a non-ST-elevation myocardial infarction (NSTEMI). Myocardial infarction has historically been more prevalent in affluent countries but is now increasingly prevalent in underdeveloped countries. [12]

Willem Einthoven first invented electrocardiogram (ECG) in 1902. The electrocardiograph is the major diagnostic instrument of cardiac electrophysiology. An ECG is important for the first assessment of a patient who may be experiencing cardiac-related issues. ECG is a non-invasive, surface-based recording that shows an electrical tracing of the heart.[13] Given that cardiovascular illness is the leading cause of mortality, it is essential for healthcare professionals to get proficiency in reading ECGs in order to give the best care possible as soon as possible.

Anatomy and Physiology of the heart

A basic understanding of cardiac anatomy and coronary distribution is essential for understanding electrocardiographic findings. The heart is a muscular organ comprised of four chambers with two atria (right and left) opening into right and left ventricles via tricuspid and mitral valves, respectively. Septum separates all four chambers. The basic functioning is as follows

Deoxygenated blood from the body → Superior and inferior vena cava → Right atrium → Right ventricle → Left and right pulmonary artery → lungs → oxygenated blood → Right and left pulmonary veins → Left atrium → Left ventricle → aorta → Rest of the body.

The arrangement of the normal coronary arteries

The heart derives its blood supply from the coronary arteries that branch from the aorta. The left main coronary artery, which separates into left anterior descending and circumflex branches, and the right main coronary artery are the principal vessels of the coronary circulation. Behind the aortic valve leaflets are apertures known as the coronary ostia from which the left and right coronary arteries emerge at the base of the aorta. [14]

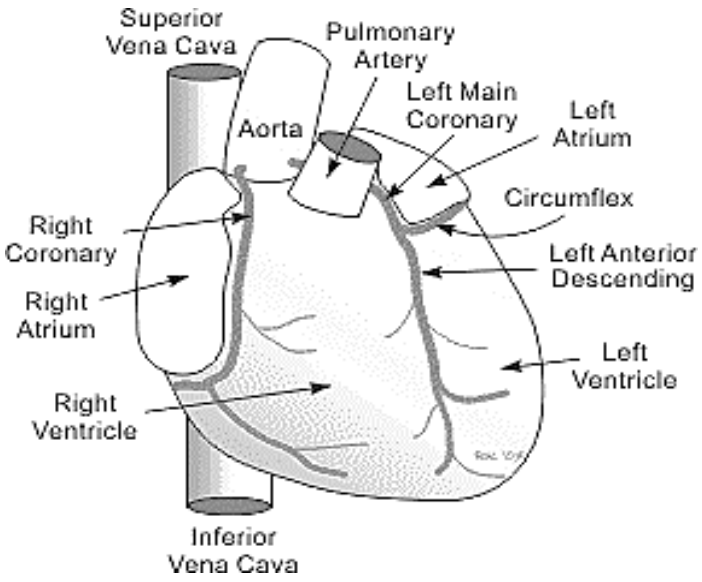


Figure 1 – The arrangement of the normal coronary arteries

These vessels distribute blood flow to different regions of the heart muscle. The key sources of vascular resistance in the microcirculation, like in all vascular beds, are the small arteries and arterioles, which also serve as the main sites for controlling blood flow. The cardiac myocytes are located close to the arterioles, which divide into many capillaries. Short diffusion

distances and a high capillary-to-cardiomyocyte ratio ensure that the myocytes receive adequate oxygen and that metabolic waste products like CO^2 and H+ are removed from the cells. The coronary sinus, which is on the posterior of the heart and drains into the right atrium, is where capillary blood flow joins venules that combine to produce cardiac veins. Thesbesian veins and anterior cardiac veins both enter the heart chambers directly.[14]

Coronary artery disease may cause coronary blood flow to be decreased. As a result, there will be a greater extraction of oxygen from the coronary circulation and less oxygen in the veins. Angina and tissue hypoxia result from this.

ACUTE MYOCARDIAL INFARCTION

Definition of Myocardial Infarction

Acute myocardial cell necrosis caused by substantial and prolonged ischemia serves as the hallmark of acute myocardial infarction. Usually, but not always, it is an indication of coronary heart disease brought on by atherosclerosis. Acute myocardial infarction is brought on by coronary heart disease, which restricts blood flow as a result of plaque build-up in the coronary arteries or by other obstructive factors. [15]

Clinical criteria for Acute Myocardial Infarction

The presence of acute myocardial damage as shown by abnormal cardiac biomarkers in the presence of acute myocardial ischemia is acute myocardial infarction. [16]

The phrase "risk factors" for the start of cardiovascular disease (CVD) was originally used by American researchers working on the Framingham Heart Study in the 1960s [17, 18] They discovered that the risk of CVD was raised by smoking, high cholesterol, high blood pressure, and irregular electrocardiograms.

“The risk factors” for development of AMI are both modifiable and non-modifiable.

Modifiable risk factors

Regardless of gender, ethnicity, or location, the INTERHEART study published in 2004, found nine key modifiable risk variables that together account for greater than 90% of the chance of suffering an acute myocardial infarction. [19]

The risk factors included:

1. Smoking

The INTERHEART study demonstrated that there is no upper limit to the risk increase, which is exactly proportionate to cigarette consumption.

2. Abnormal blood lipids

The "ApoB/ApoB1" ratio in particular appears to be a strong predictor of risk in various populations.

3. Hypertension

The majority of cardiovascular deaths are caused by hypertension.

4. Diabetes

People with diabetes have a higher risk of cardiovascular illness even when their blood sugar levels are under control.

5. Abdominal obesity

The risk is more strongly linked to the waist to hip ratio than it is to the body mass index. In addition to increasing the chance of developing heart disease, obesity increases the likelihood of developing diabetes, high cholesterol, high blood pressure, and other illnesses.

6. Psychosocial factors

A high risk of acute myocardial infarction is linked to psychological factors like depression, stress (particularly financial stress), and life events.

7. Low amount of physical activity

8. Poor eating of fruits and vegetables

9. Drinking alcohol

Non-Modifiable risk factors

1. Increasing age

Compared to younger patients, older adults have a higher risk of dying from cardiac disease. Prior to the age of 50, the incidence of CHD is significantly lower in women than in men; after that, the incidence of CHD rises and approaches that seen in men by the eighth decade. The greater levels of several risk variables in men at younger ages can explain the earlier age of acute MI in men. [20]

2. Gender

Coronary heart disease (CHD) is the leading cause of death in both men and women around the world. Since 1950, the incidence of coronary heart disease has increased in women while decreasing in males. The average age of death from a cardiac arrest is lower in men. However, by the age of 65, and above, the number of deaths due to ischemic heart disease is 11% more in females as compared to males.[20, 21, 22]

3. Racial and ethnic background

In comparison to White people, South Asians and American Blacks have a higher risk of cardiovascular disease. Severe hypertension, which is linked to heart disease, is more common in African Americans. The prevalence of obesity and diabetes, which are linked to heart disease, is higher in the other ethnic groups that are at higher risk.[22]

4. Genetic factors

If an immediate relative had a coronary heart disease or stroke before the age of 55 (for men) or 65 (for women), the chance of developing cardiovascular disease is enhanced (for females). [22]

Additionally to increasing the risk of obesity, diabetes, hypertension, and smoking addiction, genetic differences can influence lipid metabolism, cardiac function, blood coagulation, and heart rate.[23]

Complications of myocardial infarction

Despite advances in the management of acute myocardial infarction, complications can and do occur [24, 25]

TYPE OF MI	MANIFESTATION
Ischaemic	Reinfarction Infarct extension Angina
Mechanical	Cardiac failure Cardiogenic shock Mitral regurgitation Ventricular aneurysm Cardiac rupture (ventricular septum, papillary septum or cardiac wall)
Arrhythmic	Atrial or ventricular arrhythmia

	Sinus or atrioventricular node dysfunction
Embolic	Central nervous system embolus Peripheral embolus Left ventricular mural thrombus
Inflammatory	Pericarditits

Table 1 – Complications of Myocardial infarction

1. Cardiogenic shock

A severe decline in forward cardiac output causes cardiogenic shock, which is results in hypotension, decreased organ perfusion, and high left ventricular filling pressures, which lead to congestive heart failure. This could result from a severe left ventricular infarction or one of the mechanical problems outlined above.

The first step in treating cardiogenic shock is to rule out any potentially treatable reasons, such as RV infarction (which is managed with fluid infusions) or mechanical issues that would require surgical repair.[24]

2. Pericarditis

As a result of an inflammatory exudate in the pericardium, post-infarction pericarditis frequently starts a few days after the infarct. The pleuritic character, radiation to the left trapezius ridge, low-grade fever, and pericardial friction rub distinguish pericarditis pain from infarct pain.[24]

Non-steroidal anti-inflammatory drugs are used in the treatment of post-MI pericarditis to lessen pain and inflammation in the delicate pericardial tissue.

3. Arrhythmias / Heart block

Almost all cardiac arrhythmia types can develop as a result of an acute MI. The His-Purkinje system is mostly supplied by the RCA, while the LAD is primarily responsible for the conduction system at and above the A-V node (including the S-A node and the A-V nodes themselves).

Any type of infarct may result in an abnormal conduction interface (where healthy tissue is present next to damaged tissue), which may result in re-entry rhythms like ventricular tachycardia and atrial flutter. As with any infarct, aberrant atrial hypertrophy, atrial arrhythmias such atrial fibrillation, and insufficient LV filling can all occur.[24]

4. Heart failure

Heart failure is a complex clinical condition occurring as a result of any functional or anatomical defect of ventricular filling or blood ejection. It is one of the most common cause of late morbidity and mortality and myocardial infarction remains the most common cause of heart failure. A low volume pulse, low blood pressure, cold and sweaty extremities, a third heart sound (S3 gallop), pulmonary rales, decreased urine production, and sweating are clinical signs of circulatory failure.[25,26]

When 20% to 25% of the left ventricular myocardium is damaged, a reduction in cardiac output begins to occur. Cardiogenic shock often occurs when there is 40% or greater left ventricular myocardial damage. S3 and S4 gallops, together with pulmonary congestion, are the most typical clinical symptoms.

Both the patients' short-term and long-term prognoses are related to left cardiac dysfunction. Breathlessness, easy fatigue, sinus tachycardia, a third heart sound (S3 gallop), systolic or

diastolic murmurs, and pulmonary rales that can be detected in the lung bases are the first symptoms of the syndrome of heart failure. As the disease advances, however, it also affects the entire lungs.[25]

5. Ventricular septal rupture

Due to the necrotic section of the septum softening, acute ventricular septal rupture can happen several days after the acute infarction. Both an anterior and inferoposterior myocardial infarction may experience this. Congestive heart failure, an acute left-to-right shunt, and generally cardiogenic shock are present. There is also typically a loud systolic ejection murmur. The diagnosis is made when a new systolic ejection murmur is heard, which is frequently accompanied by a precordial thrill. [25]

6. Left ventricular free wall rupture

A sudden cardiogenic shock from "cardiac tamponade" is frequently the result of a rupture of the left ventricular free wall, which affects the left ventricle's free wall similarly to a ventricular septal defect. Rarely, a partial rupture of the free wall causes a "pseudoaneurysm" of the left ventricle, which might not be clinically recognised until the patient's health starts to deteriorate quickly.[25]

Clinical features

A sudden, severe onset of symptoms is not usually present in myocardial infarction (MI). In contrast, it usually starts off gradually and with only a little pain or discomfort.

The most important signs that can indicate an acute MI are [27, 28, 29]

- Chest discomfort, which commonly starts in the centre of the chest but can also radiate to the neck, jaw, stomach, shoulder, back, and arms (typically left arm).
- Light-headedness
- Arrhythmia or severe hypotension resulting in syncope.
- Sympathetic nerve activation leading to tachycardia
- Bradycardia in patients with inferior STEMI caused by inactivation of the vagus nerve
- Cardiogenic shock brought on by weakened myocardial performance
- Exhaustion or shortness of breath
- Sweating profusely, feeling ill or nauseous

Although the symptoms of each MI can vary, there is no connection between the intensity of the symptoms and the patients' eventual prognosis. The MI may be labelled as "silent" if the patient exhibits no symptoms or unusual signs. Silent MI may occasionally (but not always) be later discovered and referred to as "unrecognised MI." Undiagnosed MI is a frequent and clinically important occurrence.

Pathophysiology of acute myocardial infarction

The result of a severe and prolonged imbalance between myocardial oxygen supply and demand is myocardial infarction. Luminal thrombus overlying coronary atherosclerosis is usually often the culprit in myocardial infarction. [30]

1. High-risk plaques and prothrombotic tendencies in the genesis of myocardial infarction.

The two main mechanisms that cause thrombosis to form in acute myocardial infarction patients are physical disruption of the plaque, which encourages thrombin generation, and platelet activation. When a high-risk plaque ruptures, thrombosis happens because the circulating blood comes into contact with the highly thrombogenic necrotic core. [31, 32]

People with acute myocardial infarction who die at autopsy have plaque rupture as their main cause of death. The primary reason for instability and thrombosis, however, is superficial plaque erosion in some patient subpopulations, such as diabetics and women [31, 33]

In addition, proteolytic enzymes (such matrix metalloproteinases) that are highly expressed and liberated, destroy the plaque extracellular matrix, weakening the fibrous cap. Blood flow-induced shear stress elevates mechanosensitive gene expression in endothelial cells, escalating oxidative stress and promoting the production of cytokines and chemokines. [34]

2. Metabolic & ionic consequences of severe & prolonged myocardial Ischemia

Supply ischemia, which results from the stoppage of myocardial perfusion brought on by an occlusive coronary thrombus, results in contractile malfunction and change in the electrical activity. An ischemic insult causes death of the cardiac tissue if it is sustained.

After the onset of acute ischemia, the myocardium's capacity to produce energy sufficient to meet its needs is rapidly lost as a result of a shortage of oxygen. The myocardium switches to glycolysis in the absence of oxygen, in which glycogen present inside the cell is employed substrate and quickly transformed into glucose-6-phosphate and lactic acid. The formation of lactate lowers ph within the cell, which inhibits a number of glycolytic enzymes. Therefore, even if some glycogen may still be present in cardiac muscle after 10 to 20 minutes, glycolysis gradually slows and then stops.

Ischemic cardiac tissue experiences significant ionic dysregulation as a result of intracellular acidosis. The pathophysiology of arrhythmias in the infarcted heart depends on decreased membrane potentials brought on by ischemia-induced ionic alterations. [30]

3. Functional consequences of myocardial infarction

60 seconds after the total stoppage of blood flow, contractile function is lost. High-energy phosphates are preserved by this significant decrease in metabolic activity, which prolongs the survival of ischemic cardiomyocytes compared to what would happen if energy utilization remained at normal levels. Inspite the availability of high-energy phosphates, a number of factors contribute to the myocardium's quick loss of contraction in ischemia patients.

Oldest known mechanism causing ischemia systolic dysfunction may be the inhibition of contractile proteins by the production of inorganic phosphate. Additionally, minutes after coronary blockage, intracellular acidosis develops and prevents calcium from binding to contractile proteins, decreasing contractility. When blood flow is restored, the ischemic dysfunction that was present during the first five minutes of ischemia is completely reversed. The phenomenon of reversible, persistent, post ischemic dysfunction in the absence of irreversible injury is known as myocardial stunning[35]

Contractile function does not recover for many hours even after reperfusion with prolonged ischemic intervals (5–20 min). Several key mechanisms implicated in the pathophysiology of stunning include the production of reactive oxygen species and the proteolysis of contractile proteins through the activation of calcium-dependent enzymes.

4. Pathology of myocardial infarction

Cell death does not begin right away even though detectable hypocontractility manifests within 60 seconds of the beginning of an acute event of ischemia, these functional abnormalities in the beginning are entirely reversible if the flow is restored. In study models of myocardial ischemia, irreversible cardiomyocyte damage requires a sustained period of acute ischemia lasting at least 20 to 40 minutes. [36]

The appearance of tiny amorphous concentrations in the mitochondria and sarcolemmal rupture are the initial ultrastructural signs of irreparable injury. Within 3 hours of coronary occlusion, the earliest necrotic alterations identifiable under optical microscopy can be found. The histologic abnormalities which are first seen in the infarcted heart are "wavy" (lengthened and thin) fibres, odema of the interstitium , hypereosinophilia, caryolysis, and vacuolar degeneration.

Apoptosis or necrosis causes cardiomyocyte mortality in the infarcted myocardium; [37] The necrotic area gradually encompasses a bigger and larger portion of the ischemic territory as a "wavefront" of cell death which travels from the subendocardium to the less vulnerable midmyocardial and subepicardial areas with longer ischemia intervals.

As subendocardial cardiomyocytes are more susceptible to ischemic injury, cell death begins after 20-30 minutes of coronary occlusion beneath the endocardial surface.With more prolonged ischemia, cardiomyocyte necrosis extends towards the midmyocardium and subepicardium, eventually involves all the ischemic territory as seen in the figure below.

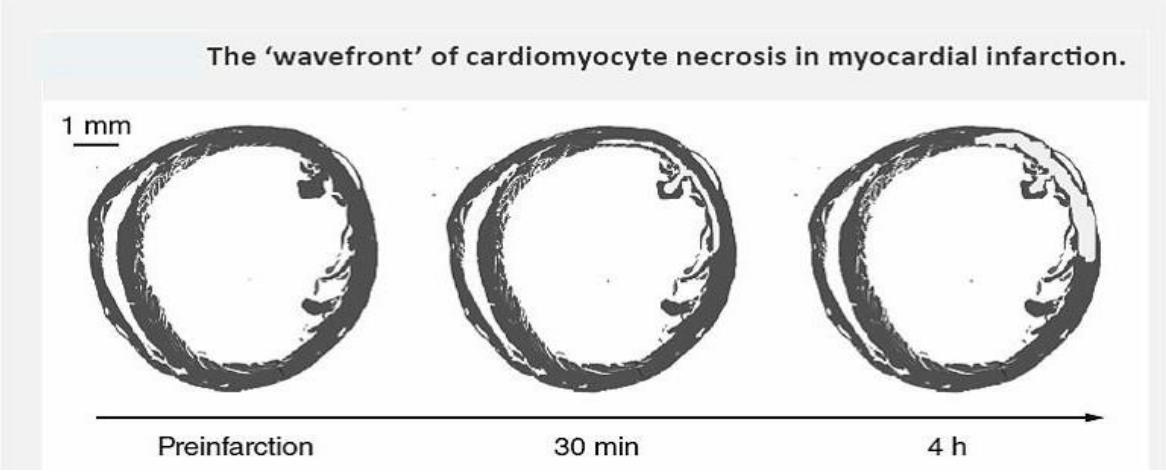


Figure 2- The wavefront of cardiomyocyte necrosis in myocardial infarction

Universal criteria for acute myocardial infarction [38]

A surge or fall in troponin levels detected with at least one result over the upper reference limit's 99th percentile and myocardial ischaemia evidenced by at least one of:

• Ischemia-related symptoms

• ECG alterations indicating fresh ischaemia (new ST-T changes or new left bundle branch block)

• Emergence of abnormal Q waves

• Imaging proof of a new myocardial viability loss or a new regional wall motion anomaly

• Sudden, unexpected cardiac death with cardiac arrest, frequently accompanied by symptoms of myocardial ischemia, and possibly new ST elevation, new left bundle branch block, and evidence of fresh thrombus by coronary angiography or at autopsy, but death occurring before blood samples could be obtained, or at a time before troponins appeared in the blood

• Biomarker increases over the 3x99th percentile upper reference limit for percutaneous coronary intervention (PCI).

• For coronary artery bypass grafting, increases in biomarkers greater than the upper reference limit of the 5x99th percentile along with either new Q waves or new left bundle branch block, as well as newly documented graft or native coronary artery occlusion or newly detected imaging evidence of viable myocardial myocardium loss, are all relevant.

• Post-mortem pathological evidence of acute myocardial infarction[38]

Classification of myocardial infarction

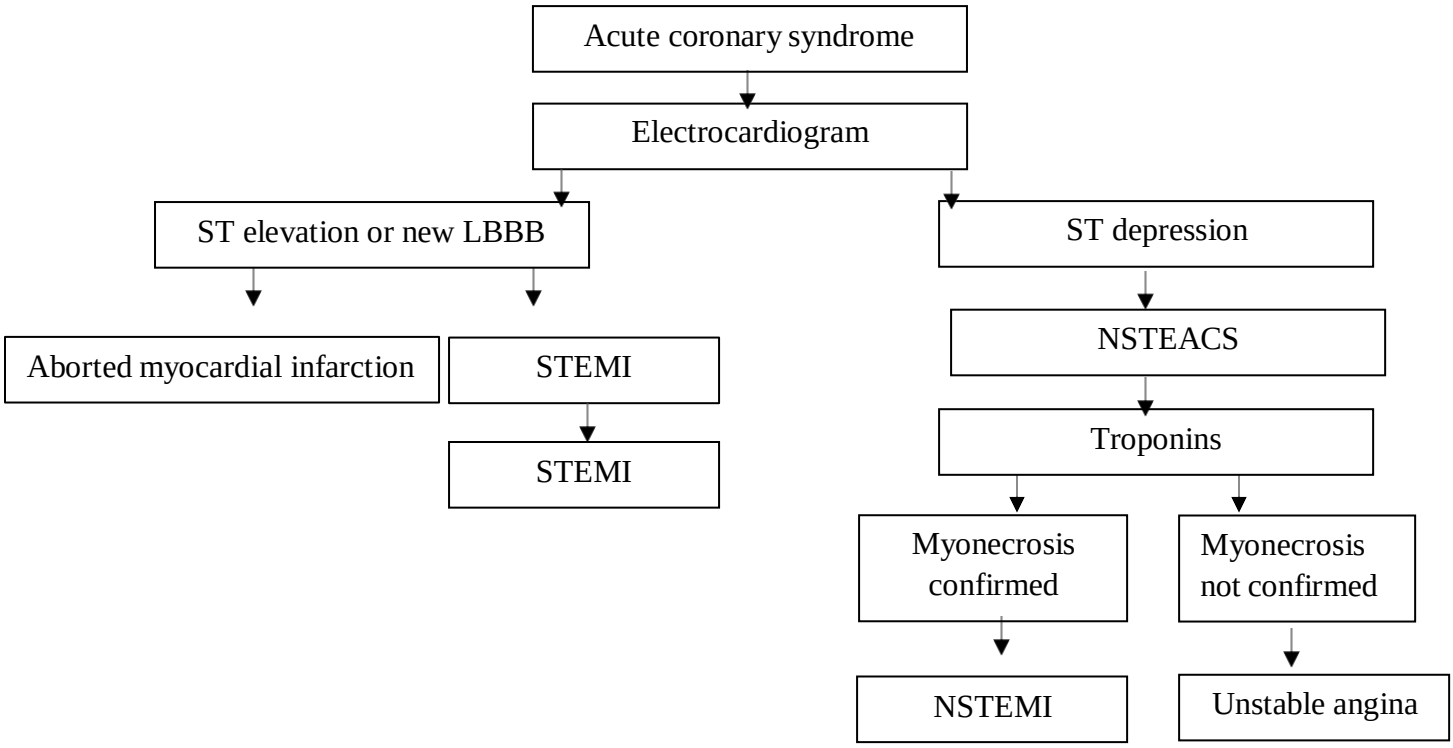


Figure 3- Classification of myocardial infarction

1. ANATOMICAL CLASSIFICATION: [16]

Transmural and nontransmural MI are the two forms of MI from an anatomical standpoint.

- Ischemic necrosis that extends the complete thickness of the afflicted muscle segment(s) from the endocardium through the myocardium to the epicardium characterises a transmural MI.
- A nontransmural MI is described as an area of ischemic necrosis that is either restricted to the endocardium or to the endocardium and the myocardium and does not extend across the complete thickness of the myocardial wall segment(s).

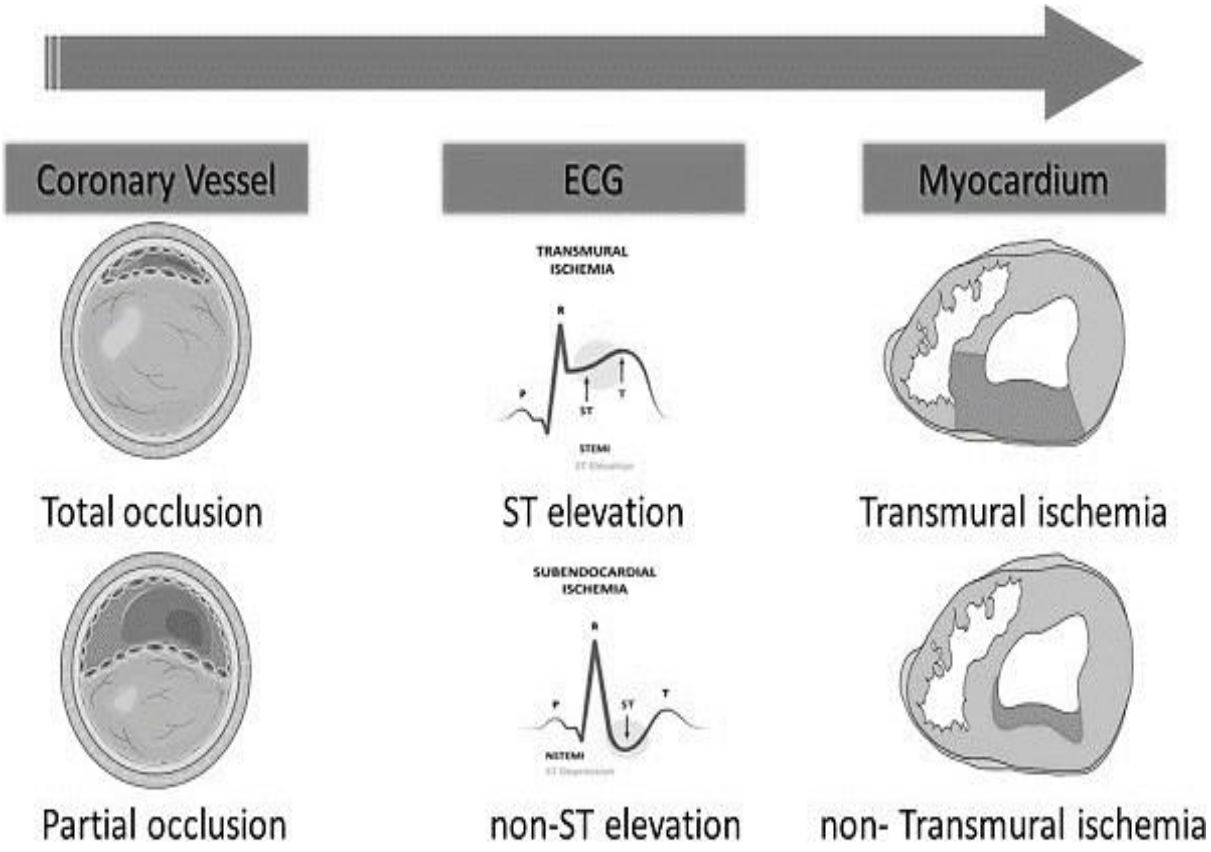


Figure 4- Anatomical classification of myocardial infarction

- **Anterior Myocardial Infarction**

The left ventricular free wall between the interventricular groove and the lateral edge of the anterior papillary muscle is where the anterior MI is located.

- **Lateral Myocardial Infarction**

Between the lateral margins of the anterior papillary muscle and the posterior papillary muscle is where the lateral MI is localised.

- **Septal Myocardial Infarction**

The interventricular septum is the site of this type of MI.

- **Inferior Myocardial Infarction**

The inferior MI is concentrated between the posterior papillary muscle's lateral border and the posterior septum.

2. CLINICAL CLASSIFICATION [16]

i) TYPE 1: Spontaneous MI due to coronary thrombosis

ii) TYPE 2: Supply / demand mismatch by secondary process other than coronary artery disease.

iii) TYPE 3: Suspected MI-related death

iv) TYPE 4:

TYPE 4a: Percutaneous coronary intervention-related death

TYPE 4b: Stent thrombosis

v) TYPE 5: Coronary artery bypass grafting- related MI.

3. ELECTROCARDIOGRAPHIC CLASSIFICATION[16]

i) ST-segment elevation myocardial infarction.

ii) Non-ST-segment elevation myocardial infarction.

The diagnostic criteria for MI comprise of the following [39, 40]

CARDIAC MARKERS	Troponin is the preferred biomarker for diagnosis
FULL BLOOD COUNT	Elevation of white blood cell count is usual Erythrocyte sedimentation rate (ESR) & C-reactive protein (CRP) may be elevated
CHEST X-RAY	For assessing pulmonary oedema
ECHOCARDIOGRAPHY	Not essential, but helpful if ECG is inconclusive

Table 2- Diagnostic criteria of acute myocardial infarction

PHYSICAL EXAMINATION

The symptoms of myocardial infarction overlaps with many other conditions, therefore it is important to differentiate between them to so that we can reach a definitive diagnosis. [41]

1. Vital signs

The patient frequently experiences an increase in heart rate as a result of sympathoadrenal discharge. The patient typically experiences peripheral artery vasoconstriction, an adrenergic reaction to pain, and ventricular dysfunction, which together cause the patient's blood pressure to initially rise. Anxiety or lung congestion may cause the respiratory rate to increase. There may be wheezing, coughing, and the creation of foamy sputum. Fever typically appears within 24 to 48 hours, and the temperature curve normally

follows the time period of rising blood levels of creatine kinase (CK). Sometimes a person's body temperature might go above 102°F.

2. Arterial pulsations

Pulsus alternans, which is characterised by strong and weak alternating pulse waves and signals poor left ventricular function, may be seen in arterial pulsations (the difference in systolic pressure is >20 mm Hg). The carotid pulsation may be thin (pulsus parvus) due to the pulse's decreased length and amplitude as a result of its smaller stroke volume.

3. Venous pulsations

Right ventricular myocardial infarction or right ventricular failure caused by severe left ventricular dysfunction and pulmonary hypertension may be accompanied with jugular venous distention.

4. Chest

Auscultation may reveal wheezes or rales; these are caused by pulmonary venous hypertension, which is linked to severe acute left ventricular myocardial infarction. At the lung bases, unilateral or bilateral pleural effusions may cause egophony.

5. Heart sounds

A mild S1 sound, dyskinesis, a perceptible S4 gallop, and lateral displacement of the apical impulse can all be felt during palpation. These point to the impaired LV's decreased contractility.

6. Extremities

It's possible that vasoconstriction, decreased cardiac output, and right ventricular dysfunction or failure are to blame for peripheral cyanosis, oedema, pallor, lowered pulse volume, delayed rise, and delayed capillary refill.

ELECTROCADIOGRAM IN ACUTE MYOCARDIAL INFARCTION

An essential component in making a diagnosis of patients with suspected MI is the ECG. The secret to effective care of ST-elevation myocardial infarction (STEMI) is early detection. For all patients with suspected STEMI, a 12-lead ECG should be obtained and analysed as soon as practically practicable at the time of initial medical contact, with a target delay of 10 minutes, to look for potentially fatal arrhythmias. The ECG is rarely normal, even at an early stage.[42]

Multiple ECGs must frequently be acquired during acute MI due to the dynamic changes in the ECG waveforms, especially if the initial ECG is non-diagnostic. “In symptomatic patients having an initial non-diagnostic ECG, if possible, a continuous computer-assisted 12-lead ECG recording or serial recordings should be made at intervals of 15 to 30 minutes”. [42, 43]

According to the ESC/ACCF/AHA/WHF Task Force for the Universal Definition of Myocardial Infarction, STEMI in the absence of left ventricular (LV) hypertrophy or left bundle-branch block (LBBB), measured at the J point, should be found in at least two contiguous leads of ≥0.2 mV in men ≥40 years, ≥0.25 mV in men <40 Years, or ≥0.15 mV in women in leads V2-V3 and/or ≥0.1 mV in other contiguous chest leads or ECG diagnosis may be more difficult in patients with atypical ECG presentations, which nevertheless deserve prompt management:[45]

- LBBB

- Ventricular paced rhythm
- Patients without ST-segment elevation but with persistent ischaemic symptoms
- Isolated posterior myocardial infarction
- Left main coronary obstruction the limb leads.

Diagnosis of STEMI: ECG changes

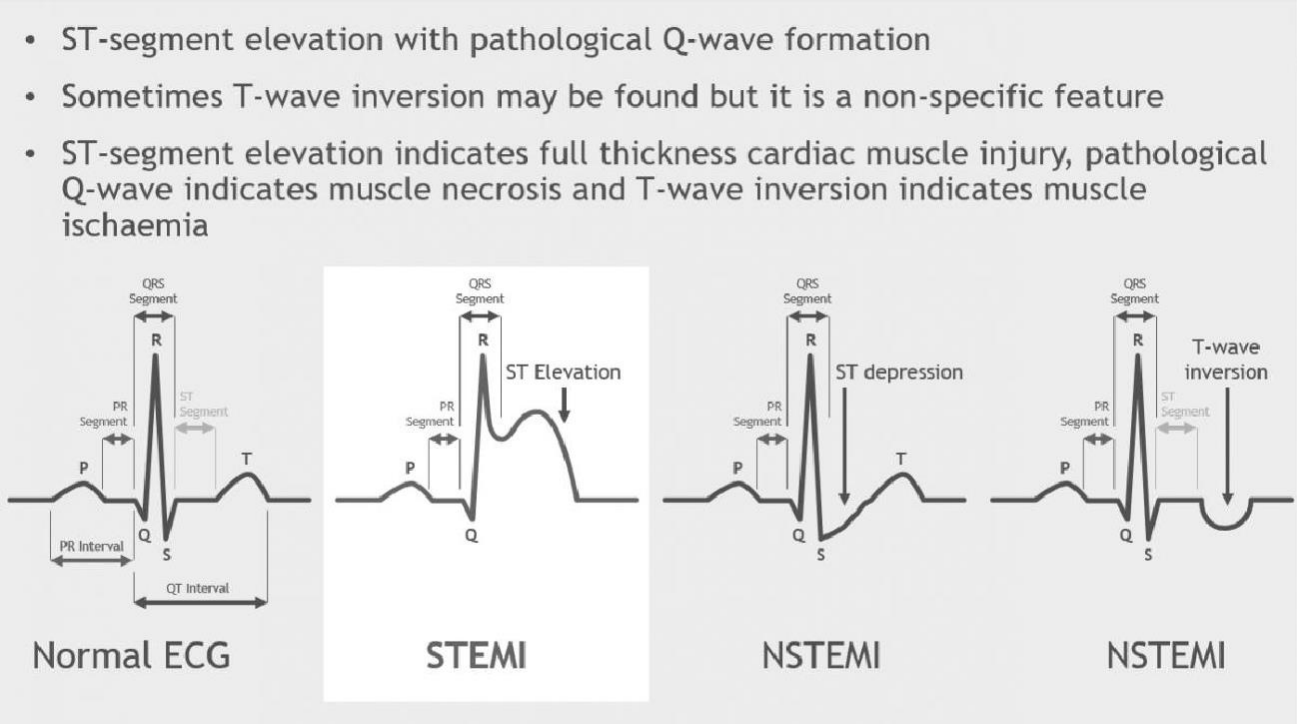


Figure 5- Diagnosis of STEMI: ECG changes

BIOCHEMICAL MARKERS

Sensitive and specific biomarkers with elevated blood levels can signal myocardial injury. Blood tests are routinely taken during the acute period, but reperfusion therapy shouldn't wait for the results

Cardiac troponin (T or I), a protein involved in the contractile process of myocardial cells, is almost exclusively expressed in the heart. Due to its strong cardiac tissue selectivity

and great clinical sensitivity, it is the suggested biomarker. The level for the diagnosis of MI, known as the upper reference limit (URL), is a value larger than the 99th percentile of a normal reference population for an increased troponin concentration. The appropriate level of quality control must be used to determine the outcomes of each individual assay.

If a troponin assay is not available, blood levels of the enzyme creatine kinase-MB isoenzyme (CK-MB) might be determined as the best fall-back. Similar to troponin, a result above the 99th percentile upper reference limit is referred to as a high CK-MB value. [43]

Non ischemic conditions can cause cardiac troponin elevation.[44]

CARDIAC	**NONCARDIAC**
Congestive heart failure	Drug toxicity
Infiltrative diseases	Pulmonary embolism
Malignancy	Renal failure
Myocarditis	Sepsis
Pericarditis	Stroke
Trauma (surgery or electric shock)	Subarachnoid haemorrhage
Viral cardiomyopathy	

Table 3- Non ischemic conditions causing raised troponin

FRONTAL QRST ANGLE

As a novel marker of myocardial depolarization and repolarization heterogeneity, the frontal QRS-T angle (QRSTa) was initially defined as the absolute value of the difference between ventricular depolarization (QRS axis) and repolarization (T axis). [46] Particularly in individuals with CAD, QRSTa abnormalities, which represent the electrical instability of the ventricular myocardium, predict poor cardiovascular outcomes and total mortality.[47] It has been shown that ventricular axis modifications are brought on by obstructive coronary artery stenosis associated with transient ischemia attacks.[48] However, the functional significance of coronary lesions related to QRSTa changes has not been studied until now.

Electrocardiogram

The electrical activity that is occurring within the heart is traced on an ECG. An electrical impulse will typically move from the sinoatrial node through the atrium, the atrioventricular node, and the heart's ventricular septum to the atrioventricular node. The four chambers of the heart contract and relax in synchronization as a result of this electrical impulse. Understanding these electrical impulses' nature helps us better understand how the heart is functioning.

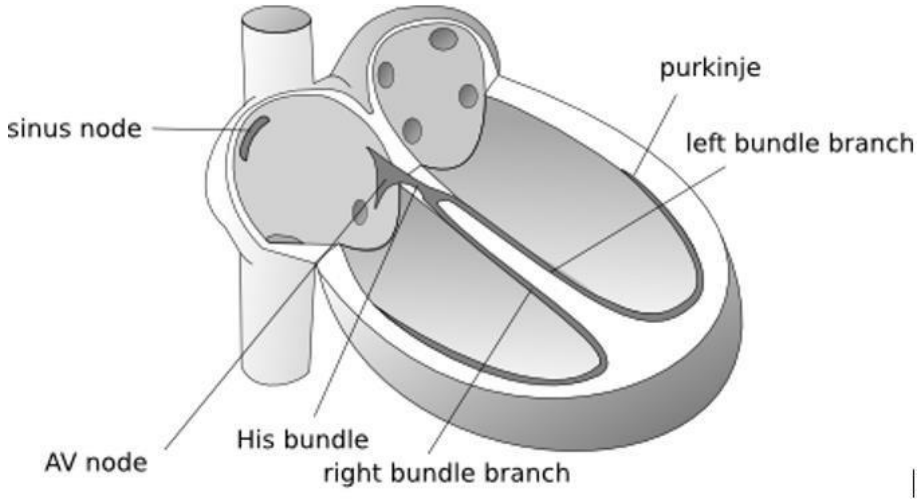


Figure 6: Conduction system of heart

ECG ELECTRODES

External (skin) electrodes can be used to measure the heart's electrical activity. These actions are recorded by the electrocardiogram (ECG) using electrodes that are attached to various body parts. Ten electrodes are used to compute a total of twelve leads. [51]

The ten electrodes are:

1. The four extremity electrodes:

- LA - left arm
- RA - right arm
- N - neutral, on the right leg (= electrical earth, or point zero, to which the electrical current is measured)
- F - foot, on the left leg

2. The six chest electrodes are as follows:

V1 is positioned in the fourth intercostal space to the right of the sternum;

V2 is positioned in the fourth intercostal space to the left of the sternum.

Position V3 between V2 and V4.

V4 is located in the nipple line at the fifth intercostal space. According to official instructions, in females position of V4 is under the breast.

V5 is positioned between V4 and V6, while V6 is positioned at the same height in the midaxillary line as V4 (horizontal line from V4, so not necessarily in the 5th intercostal space)

These 10 electrodes can be used to create 12 leads. There are six precordial leads and six extremities leads.

The Extremity Leads

- The leads for the extremities are as follows:

I from the right to the left arm

II from the right arm to the left leg

III from the left arm to the left leg

Other extremity leads are:

- AVL points to the left arm
- AVR points to the right arm
- AVF points to the feet

The capital A stands for "augmented" and V for voltage.

The Chest Leads: The precordial, or chest leads, (V1, V2, V3, V4, V5 and V6) 'observe' the depolarization wave in the frontal plane.

Ventricular depolarization and repolarization[50]

1. Cardiac cells are polarised while they are at rest, which means there is no electrical activity.

2. Different ion concentrations, including sodium, potassium, and calcium, are separated by the cell membrane of the heart muscle cell. The term for this is the resting potential.

3. Automaticity of specialized cardiac cells help to generate electrical impulses.

4. The action potential, also known as depolarization, is produced when an electrical cell generates an electrical impulse. This electrical impulse induces the ions to cross the cell membrane.

5. The force behind the contraction of cardiac muscle is the passage of ions across the cell membrane via sodium, potassium, and calcium channels.

6. The wave of depolarization causes the myocardium to contract.

7. Repolarization is the process of the ions coming back to their previous resting condition, which coincides with the cardiac muscle relaxing.

8. Electrical processes called depolarization and repolarization create cardiac muscle activation.

9. The depolarization-repolarization cycle are the electrical changes in the cardiac cell which are depicted by the action potential curve.

10. It is the electrical activity and not the muscle activity which is detected on the ECG.

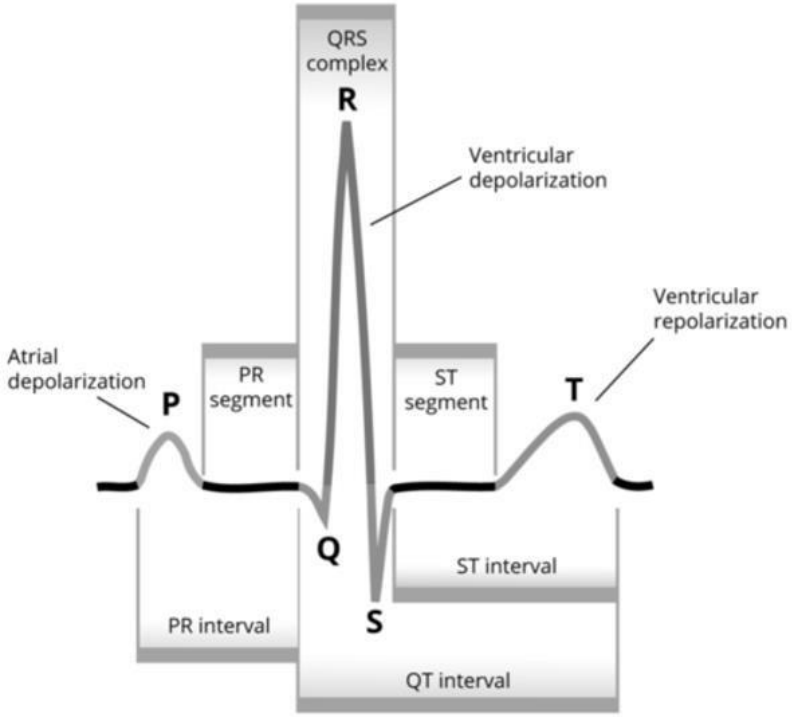


Figure 7- Electrocardiogram depicting ventricular depolarization and repolarization

Basics of electrocardiogram[51]

P Wave

The P wave correlates to both atrial contraction and the depolarization of the left and right atrium. In actuality, the atria begin to compress just before the P wave starts. Because it is so tiny, atrial repolarization is usually not detectable on ECG. The P wave will normally be smooth and rounded, no taller than 2.5 mm, and lasting no longer than 0.11 seconds. In leads I, II, aVF, and V1 through V6, it will be positive.

QRS Complex

The QRS complex includes the Q wave, R wave, and S wave. These three waves occur in rapid succession. The QRS complex indicates ventricular depolarization. An adult patient's QRS complex will last for 0.06 to 0.10 seconds on average. Typically, leads I, aVL, V5, V6, and II, III, and aVF have positive QRS complexes. Leads aVR, V1, and V2 frequently have a negative QRS complex.

J-point

The junction of the ST segment and the QRS complex is known as the J-point. It can also be considered the beginning of the ST segment. The J-point, often referred to as Junction, is significant because a ST segment elevation myocardial infarction can be identified using it. A STEMI is likely when the J-point is at least 2 mm raised above baseline.

T Wave

A T wave follows the QRS complex and indicates ventricular repolarization. T waves are normally positive in leads I, II, and V2 through V6 and negative in Avr. Typically, a T wave

that travels in the opposite direction from the QRS complex indicates some form of heart disease.

Electrical axis of heart [49]

The heart axis displays the normal direction of the depolarization wave. The normal cardiac axis is between -30 and +90 degrees.

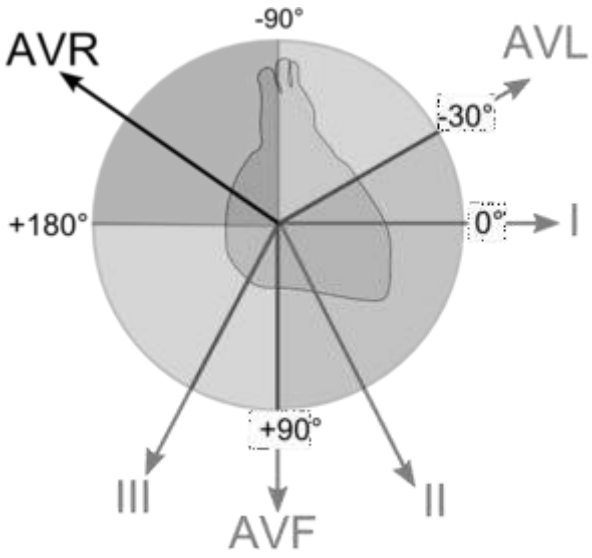


Figure 8- Electrical axis of heart according to frontal leads

The average of the heart's electrical signals is shown by an arrow (vector) pointing in the direction of electrical depolarization. This is the axis of the heart. An excessive deviation or alteration in the heart's axis may signal disease.

Electricity moving towards a lead causes a positive deflection in the lead's electric recording, is the concept used in establishing the heart axis. Leads I, II, and AVF help in calculating the heart axis.

- Positive (the average of the QRS surface above the baseline) QRS deflection in lead I: the electrical activity is directed to the left (of the patient)
- Positive QRS deflection in lead AVF: the electrical activity is directed down.

This indicates a normal heart axis. Normally the heart axis is between -30 and +90 degrees.

- A left heart axis is present when the QRS in lead I is positive and negative in II and AVF. (between -30 and -90 degrees)
- A right heart axis is present when lead I is negative and AVF positive. (between +90 and +180)
- An extreme heart axis is present when both I and AVF are negative. (axis between +180 and -90 degrees). This is a rare finding.

An iso-electric lead can help estimate the heart axis more precisely:

Iso-electric

When the depolarization is perpendicular on the lead, this is called iso-electric. The QRS is neither positive nor negative.

Undetermined axis

When all extremity leads are biphasic, the axis is directed to the front or back, in a transverse plane. The axis is than undetermined.

QRS AXIS

By analysing the vectors produced under the electrodes, the ventricular (QRS) axis is inferred. To do this, the electrical signal (QRS complex) recorded at each electrode is classified as positive, negative, or isoelectric before their interrelationship is taken into account.[52]

Typically, a positive QRS complex in a lead has a ventricular axis that is roughly parallel to that lead's positive QRS complex. A lead with a negative QRS complex has a

ventricular axis that is roughly perpendicular to that lead. The ventricular axis is perpendicular (90 degrees) to a lead if the QRS complex is isoelectric in that lead.

T AXIS

In an electrocardiogram T wave's axis is a sign of ventricular repolarization. The normal T axis ranges from 15° to 75°. When deviated from normal by more than 60 degrees in either direction in the frontal plane, has been strongly linked to an increased risk of fatal and non-fatal cardiac events in adults. When deviated by more than 60 degrees in either direction in the frontal plane is said to be associated with cardiac events. [53]

Approach to Determining QRS Axis [52]

The leads I, II, and aVF's QRS complexes are the primary ones to analyse. These three leads' positive ends all fit into the normal axis zone. Leads I, II, and aVF have respective positive ends that are 0 degrees, +60 degrees, and +90 degrees. As a result, the axis is normal if all three of these leads exhibit positive QRS complexes.

- **Method 1- Quadrant Approach Or Two-Lead Method**

I and avf leads are examined. Leads I and aVF must both be positive to indicate that the axis is within the normal range. There is LAD if lead I is positive and lead aVF is negative. There is RAD if lead I is negative and lead aVF is positive. Additionally, if leads I and aVF are both negative, the axis is in the extreme range of the axis range.

- **Method 2- Three-Lead Method**

Leads I, Avf as well as lead II is taken into account. If the net QRS deflection is positive in both leads I and II, the QRS axis is normal. If the net QRS deflection is positive in lead I but negative lead in II, then there is LAD.

- **Method 3**

The frontal plane is where the most isoelectric limb lead is situated. The lead with the least overall amplitude and a net amplitude of zero is known as the isoelectric (equiphasic) lead. The positive pole of that lead is almost perpendicular (90 degrees) to the QRS axis.

The electrical axis in the frontal plane is determined using these three techniques. A horizontal plane with a horizontal axis is also present. By looking at the heart under the diaphragm, one can identify the axis along this plane.

Frontal QRS-T angle

QRS-T angle consists of QRS axis and T-wave axis. The frontal QRS-T angle, defined as the absolute value of the difference between QRS axis and T-wave axis on 12 lead electrocardiogram (ECG), is the difference in orientation between ventricular depolarization and repolarization.

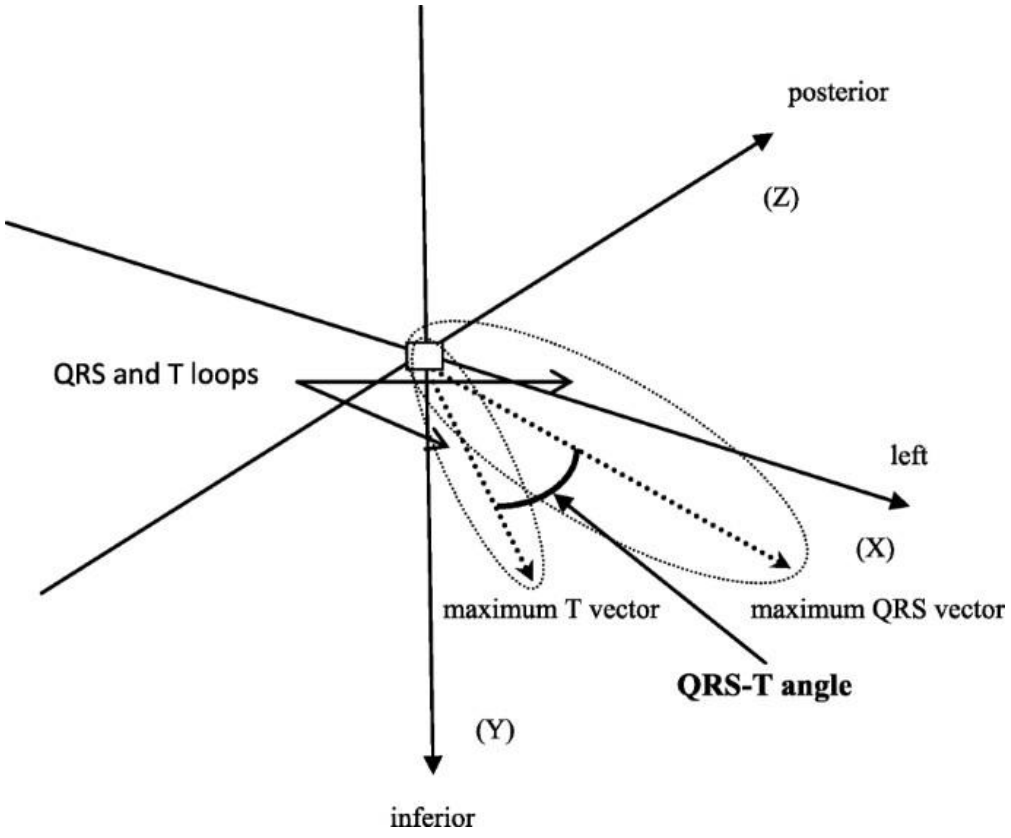


Figure 9- Frontal QRS-T angle

The QRS-T angle can be evaluated in three dimensions or as a projection on the frontal plane in a conventional ECG. An ordinary ECG can be used to read the frontal QRS-T angle.

An abnormal QRS-T angle suggests altered ventricular repolarization, which may be linked to underlying structural or functional cardiac conditions. Recent research has demonstrated that an aberrant QRS-T angle is a potent and reliable predictor of cardiac morbidity and mortality. [54]

Relation between Frontal QRS-T angle and adverse cardiac events

Patients can be followed up during their in-hospital stay for their occurrence of major adverse cardiac events like death, heart failure, complex ventricular arrhythmias (sustained ventricular tachycardia or ventricular fibrillation), early post infarction angina, or mechanical complications. Following few studies have been compiled to understand the relation between Frontal QRST angle and occurrence of adverse cardiac events.

- Sawant, A.et al in 2019 studied the Prognostic value of frontal QRS-T angle in predicting survival after primary percutaneous coronary revascularization/coronary artery bypass grafting for ST-elevation myocardial infarction. Indian Heart Journal, 71(6), pp.481-487. In the study, they concluded that FQRST represents a novel independent predictor of one-year mortality in STEMI patients undergoing reperfusion. A high FQRST based risk score was associated with more significant mortality and longer length of stay. [55]
- Dogan A, Kahraman S in 2018 studied that Frontal QRS-T angle predicts coronary atherosclerotic burden in patients with ST-segment elevation myocardial infarction. Journal of Electrocardiology. 2020;58:155-159. The researchers came to the conclusion that, as shown by the SYNTAX score, the FQRST angle accurately predicts the amount and severity of coronary artery disease. Additionally, it is linked to the atherosclerotic burden following percutaneous intervention. [56]
- Zhang Z et al in 2017 studied Electrocardiographic QRS-T angle and the risk of incident silent myocardial infarction in the Atherosclerosis Risk in Communities study. Journal of Electrocardiology. 2017;50(5):661-666. In the study, they concluded that both frontal and spatial QRS/T angles are predictive of silent myocardial infarction suggesting a potential use for these markers in identifying individuals at risk. [10]
- Zulkif Tanriverdi et al. in 2017 studied the relationship between Frontal QRS-T angle and hypertension. The study consisted of 122 hypertensive patients. It was found that the frontal

QRS-T angle was significantly higher in patients with nondipper hypertension than in patients with dipper hypertension. Also, the higher QRS-T angle was associated with higher sleeping systolic and diastolic BPs. [57]

- Jaroszyński A et al in 2019 studied the improvement of QRS-T angle as a manifestation of reverse electrical remodelling following renal transplantation in end-stage kidney disease patients on haemodialysis. BMC nephrology. 2019 Dec;20(1):1-8. It was found that Renal trasnplant induce biphasic reverse electrical remodeling as assessed by the narrowing of QRS-T angle. Early decrease of QRS-T angle is mainly due to the normalization of volume status, whereas late decrease is associated predominantly with the improvement of cardiac contractile function. [58]
- Chua KC et al in 2016 studied Wide QRS-T angle on the 12-lead ECG as a Predictor of Sudden Death beyond the Left ventricular Ejection Fraction Journal of cardiovascular electrophysiology. 2016 Jul;27(7):833-9. In the study, they concluded that A wide frontal QRS-T angle greater than 90° was associated with an increased risk of sudden cardiac arrest independent of demographic characteristics, baseline comorbidities, prolonged intraventricular conduction, ECG LVH and left ventricular ejection fraction in this population. [59]
- Li YH.et al in 2013 studied the Value of the frontal planar QRS-T angle on cardiac dysfunction in patients with old myocardial infarction. . International journal of clinical and experimental medicine. 2013;6(8):688. In the study, they concluded that In patients who have been prior diagnosed with myocardial infarction, there is a negative association between the planar QRS-T angle and LVEF; the larger the planar QRS-T angle, the lower the LVEF. For patients with a history of myocardial infarction, the planar QRS-T angle may be a simpler and more practical index to measure cardiac dysfunction. [60]
- Whang W.et al in 2012 studied the Relations between QRS|T Angle, Cardiac Risk Factors, and Mortality in the Third National Health and Nutrition Examination Survey (NHANES III). . The

American journal of cardiology. 2012 Apr 1;109(7):981-7. In the study, they came to the conclusion that in this population-based sample of individuals without a history of heart disease, an aberrant QRS|T angle as assessed by a 12-lead ECG was linked to an elevated risk for cardiovascular and all-cause mortality. [61]

- Borleffs CJ.et al in 2009 studied the QRS-T angle Predicting ventricular arrhythmias in patients with ischemic heart disease.Circulation: Arrhythmia and Electrophysiology. 2009 Oct 1;2(5):548-54. In the study, they concluded that e high incidence of ventricular arrhythmias in patients with a wide QRS-T angle and the low incidence in patients with a narrow QRS-T angle. [62]

Frontal QRS-T angle and Prognosis of MI

Acute ST-segment elevation myocardial infarction (STEMI) still has a high mortality rate even though primary percutaneous coronary intervention (PCI) reduces cardiovascular mortality and is acknowledged as the preferred revascularization technique. A number of electrocardiogram (ECG) parameters have been used to identify high-risk patients during an acute STEMI. Frontal QRS -T (fQRST) angle is one of these parameters.

Increased cardiovascular mortality has been found to be associated with an abnormal fQRST angle. In individuals with acute coronary syndromes (ACS), it is related to prognosis. Additionally, heart failure patients with reduced or retained ejection fraction can benefit from the prognostic significance of the fQRST angle.

In 2017, a study was conducted by May O.et al. The study included 178 diabetic patients. In the diabetic population, frontal QRST angle was investigated as a potent predictor of the long-term risk of myocardial infarction and all-cause mortality. A QRS-T angle greater than 90 degrees was discovered to be a potent independent prognostic factor for future all-

cause mortality as well as MI and the composite endpoint MI or all-cause death. In comparison to patients with a QRS-T angle over 90°, the group with a QRS-T angle below 90° had an estimated overall survival time that was 2.4 times longer. [63]

Investigations have been done into the connection between fQRST angle and CAD severity. According to Palaniswamy et al., patients with fQRST angle greater than 90 degrees were considerably more likely to have 2 or 3 vascular CAD. According to Colluoglu et al., STEMI patients who presented with a baseline fQRST angle of more than or equal to 95 degree were more likely to have three-vessel disease. [64]

A narrow fQRST angle is the result of similar directions of the ventricular depolarization and repolarization axes. Research has shown that the ventricular depolarization and repolarization axes can alter under more severe ischemia conditions, and this abnormal repolarization causes the fQRST angle to widen. [64]

MATERIALS AND METHODS

IV. MATERIALS AND METHODS

Source of data- Patients admitted with "Acute Myocardial Infarction'' in the ICCU of BLDE(DEEMED TO BE UNIVERSITY), Shri B.M.Patil Medical College Hospital and Research Centre, Vijayapura between January 2021 to June 2022.

Study Design- Cross-sectional study

Study Period- 18 months

Sample size calculation-

With anticipated Proportion of mortality among higher FQRST angle in patients with Acute Myocardial Infarction 28%, the study would require a sample size of 71 patients with 95% level of confidence and 10% absolute precision.[55]

Formula used

- $n=\frac{z^2\ p*q}{d^2}$

Where Z= Z statistic at α level of significance

d^2= Absolute error

P= Proportion rate

q = 100-p

PATIENT SELECTION

A. INCLUSION CRITERIA

1. Patients admitted with acute myocardial infarction (STEMI and NSTEM

B. EXCLUSION CRITERIA

1. Patients with unstable angina.
2. Patients having bundle branch block on ECG (LBBB or RBBB),
3. Patient on a temporary or permanent pacemaker.
4. Patients with old ischemic heart disease.

INVESTIGATIONS.

1. Blood

-Heamogram

-RBS

-Kidney function test

-Serum electrolytes

-Troponin I/ Troponin T

-CPK MB

2. Urine Examination

3. Electrocardiogram.

4. Chest X-ray

5. 2D Echocardiography and color Doppler study

METHODOLOGY:

1. INITIAL ASSESSEMENT

The study was conducted on patients who are admitted in BLDE (DU), Shri B M Patil Medical College Hospital and Research Centre, Vijayapura with prolonged chest discomfort typical of myocardial ischemia, who underwent a standardized assessment with history and examination, electrocardiogram at admission, cardiac enzymes – Troponin I / Troponin T, CPK-MB, and other necessary laboratory investigations like complete blood count, renal function test, serum electrolytes and 2d echo study.

2. ELECTROCARDIOGRAPHY-

12 Lead surface electrocardiography, with 25mm/sec paper speed and 10mm/sec voltage, was done using BPL CARDIART 6108T or VESTA 301i. ECG machine was used for diagnosis of acute myocardial infarction and was assessed for the Frontal QRST angle. Heart rate, PR interval, QRS duration, QT interval, corrected QT interval, QRS axis, and Taxis were all collected from each patient's initial ECG recording. The Frontal QRST angle is defined as the absolute value of the difference between the frontal plane QRS axis and T axis. If the difference came out to be more than 180 degrees then the Frontal QRST angle was adjusted to a minimum angle as 360 degrees minus the absolute value of the difference between the frontal plane QRS axis and T axis to obtain a continuous variable ranging from 0 to 180 degree. Based on relative risk associated with increasing values of FQRST angle, they were classified into two groups <100 degrees and >100 degrees. For QRS axis examine the frontal leads I, II, III, avl,avf, and avr determine the most equiphasic QRS deflection. The vector will be perpendicular to that particular lead where it has come equiphasic. Then the lead perpendicular to that equiphasic lead will be parallel to QRS vector, which is the axis of the QRS wave. For T axis the mean manifest frontal plane T wave axis is similarly directed to the mean manifest frontal plane QRS axis.

3. OUTCOME

Patients were followed up during their in-hospital course for the occurrence of adverse cardiac events namely: death, heart failure, complex ventricular arrhythmias (sustained ventricular tachycardia or ventricular fibrillation), early post-infarction angina, or mechanical complications. Heart failure was diagnosed clinically according to the standard criteria. Complex ventricular arrhythmia by monitoring ECG strip or by 12 lead ECG recording. Early post-infarction angina- recurrent typical chest discomfort during hospital admission following relief of that of the index myocardial infarction. Mechanical complications (recorded by echocardiography) including acute mitral regurgitation, rupture of the interventricular septum, left ventricle pseudoaneurysm formation, and rupture of the left ventricle free wall.

4. STATISCAL ANALYSIS

- Using the statistical package for the social sciences (Version 20) the data obtained was entered in a Microsoft excel sheet and statistical analysis was performed.
- The results are shown as counts, percentages, graphs, and Mean (Median) ± SD.
- Categorical variables were compared using the Chi-square test.
- $p<0.05$ was considered statistically significant. All statistical tests were perform two-tailed.

Formula used

- $n = \frac{(z\alpha + z\beta)2\ 2\ p*q}{MD2}$

Where Z= Z statistic at a level of significance

MD= Anticipated difference between two proportions

P=Common Proportion

q= 100-p

Statistical Analysis

• Using the statistical package for the social sciences (Version 20) the data obtained was entered in a Microsoft excel sheet and statistical analysis was perform. The results are shown as counts, percentages, graphs, and Mean (Median) ± SD.

• The Independent t-test was used to compare two groups' normally distributed continuous variables. Mann Whitney U test was employed for variables that weren't normally distributed. Categorical variables between the two groups were compared using the Chi-square test.

• $p<0.05$ was considered statistically significant. All statistical tests were performed two-tailed

RESULTS

V. RESULTS

Total of 102 patients of acute coronary syndrome admitted in the ICCU of BLDE (Deemed to be university), Shri B.M.Patil Medical College Hospital and Research Centre, Vijayapura between January 2021 to June 2022 were enrolled in the study. Seven patients were excluded from the study based on exclusion criteria, of which four patients had Left bundle branch block (LBBB), two had unstable angina and one patient had Right bundle branch block (RBBB). Hence total of 95 patients were included in the study.

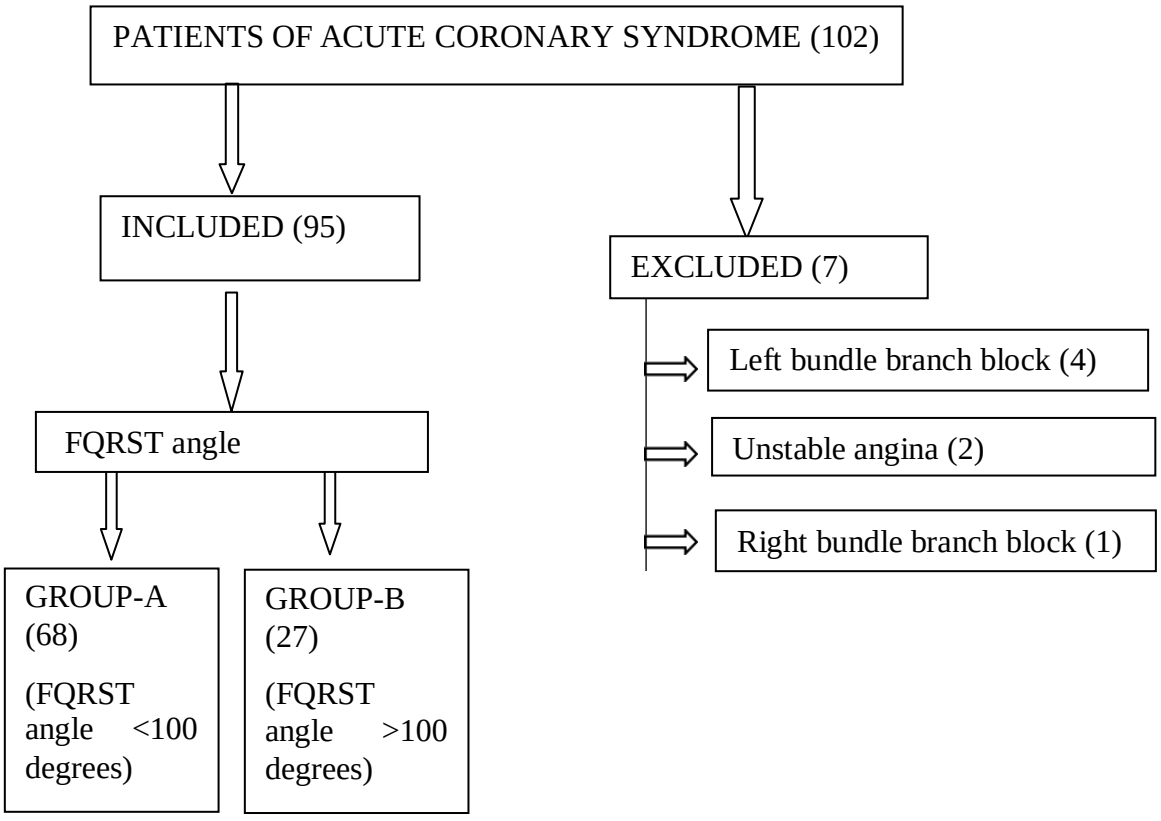


Figure 10: Flowchart showing included and excluded cases in the study**.**

Note: - $p < 0.05$ - statistically significant

$P<0.001$ – highly significant

Out of 95 patients with Acute coronary syndrome, 68 patients who had FQRST angle <100 degrees were in group A and 27 patients with FQRST angle >100 degrees were in group B as shown in Table 4.

Table 4: Grouping of patients with Acute Myocardial Infarction into Group A and Group B

Classification		No. of cases	%
Group A	FQRST angle <100 degrees	68	71.58
Group B	FQRST angle >100 degrees	27	28.42
Total		95	100.0

AGE DISTRIBUTION

The 95 patients were grouped with an age frequency of 10 years.

In group A patients aged between 30-40 yrs. were two (2.9%), patients aged between 40-50 years were 11(16.2%), patients aged between 50-60 years were 22(32.4%), patients aged between 60-70 years were 27 (39.7%) and patients aged between 70-80 years were six (8.8 %).

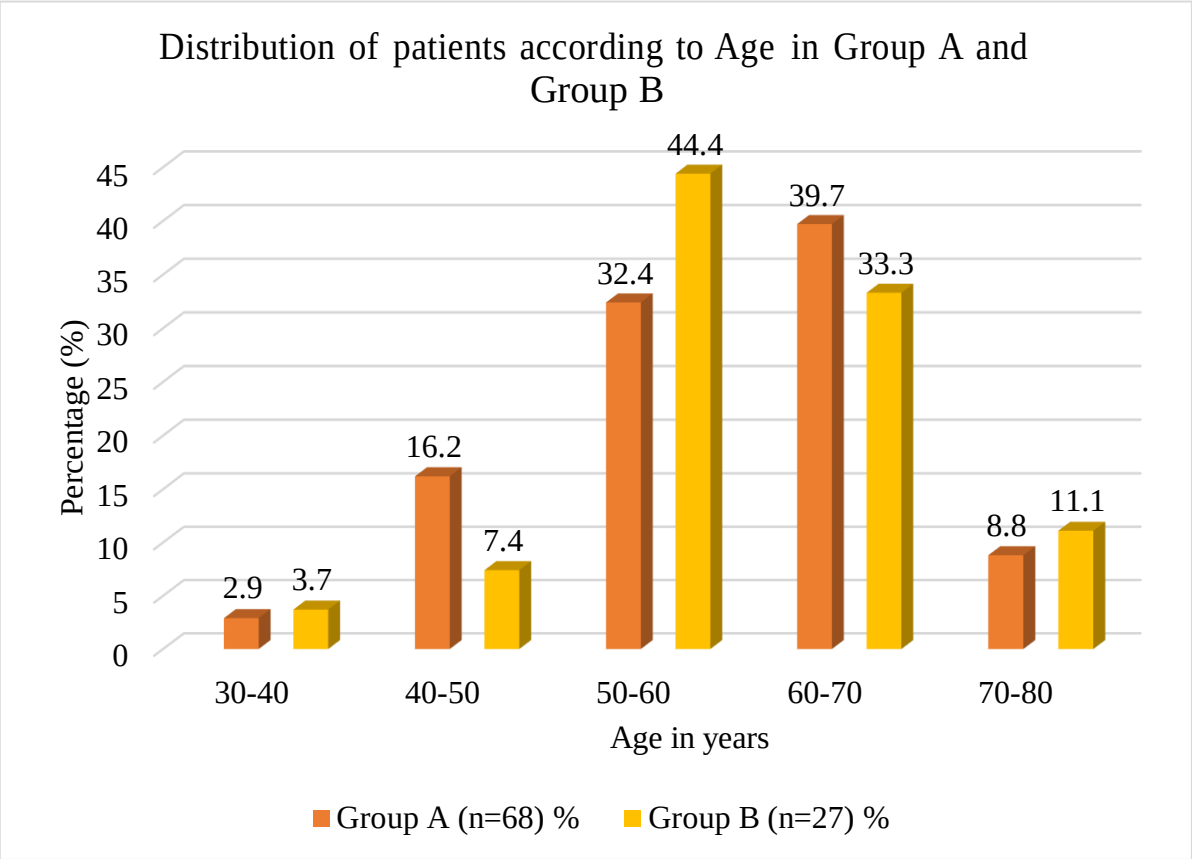
In group B patients aged between 30-40 yrs. were one (3.7%), patients aged between 40-50 years were two (7.4%), patients aged between 50-60 years were 12(44.4%), patients aged between 60-70 years were nine (33.3%) and patients aged between 70-80 years were three (11.1%).

The most common age group in group A was 60-70 years and group B was 50-60 years as described in table:5, Graph:1

Table 5: Distribution of patients according to Age in Group A and Group B						
Age (yrs.)	**Group A (n=68)**		**Group B (n=27)**		**Total (n=95)**	
	No.	**%**	**No.**	**%**	**No.**	**%**
30-40	2	2.9	1	3.7	3	3.2
40-50	11	16.2	2	7.4	13	13.7
50-60	22	32.4	12	44.4	34	35.8
60-70	27	39.7	9	33.3	36	37.9
70-80	6	8.8	3	11.1	9	9.5
Total	68	100.0	27	100.0	95	100.0

$X^2 = 2.225$,

$p = 0.694$, Not significant



Graph 1: Distribution of patients according to age in Group A and Group B

SEX DISTRIBUTION

32 patients (33.7%) and 63 patients (66.3%) out of 95 study participants were female and male, respectively. In the study male patients were more than female patients as depicted in Table: 6 In Group A 46 (67.6%) patients were male and 22 (32.4%) females, while in group B 17 (63%) were male and 10 (37%) were females as shown in table:7, Graph:2

Table 6: Distribution of Sex Among Cases in study.

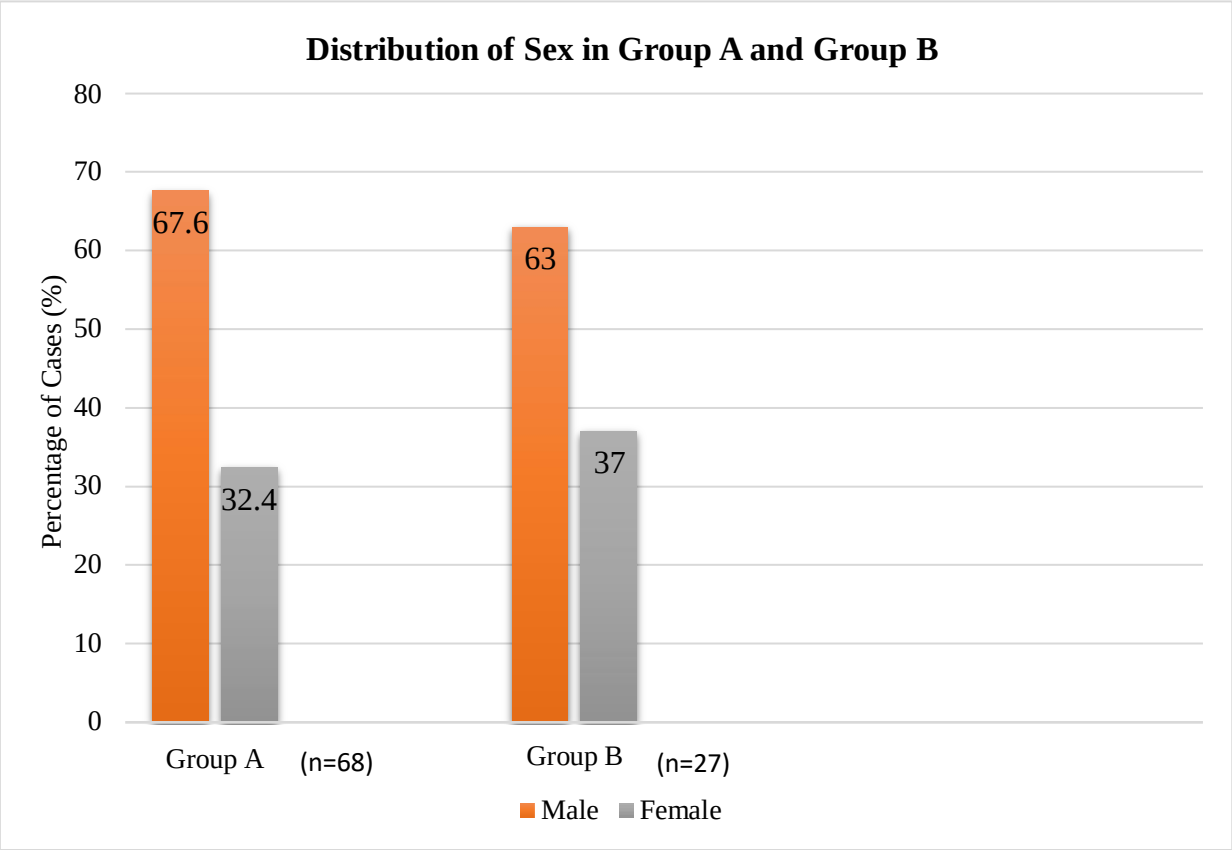
Sex	No.	%
Male	63	66.3
Female	32	33.7
Total	95	100

Table 7: Distribution of Sex in Group A and Group B.

Gender	Group A (n=68)		Group B (n=27)		Total (n=95)	
	No.	%	No.	%	No.	%
Male	46	67.6	17	63.0	63	66.3
Female	22	32.4	10	37.0	32	33.7
Total	68	100.0	27	100.0	95	100.0

$X^2 = 0.190$,

$p = 0.663$, Not significant.



Graph 2: Distribution of sex in group A and B

DISTRIBUTION OF PATIENTS ACCORDING TO OCCUPATION:

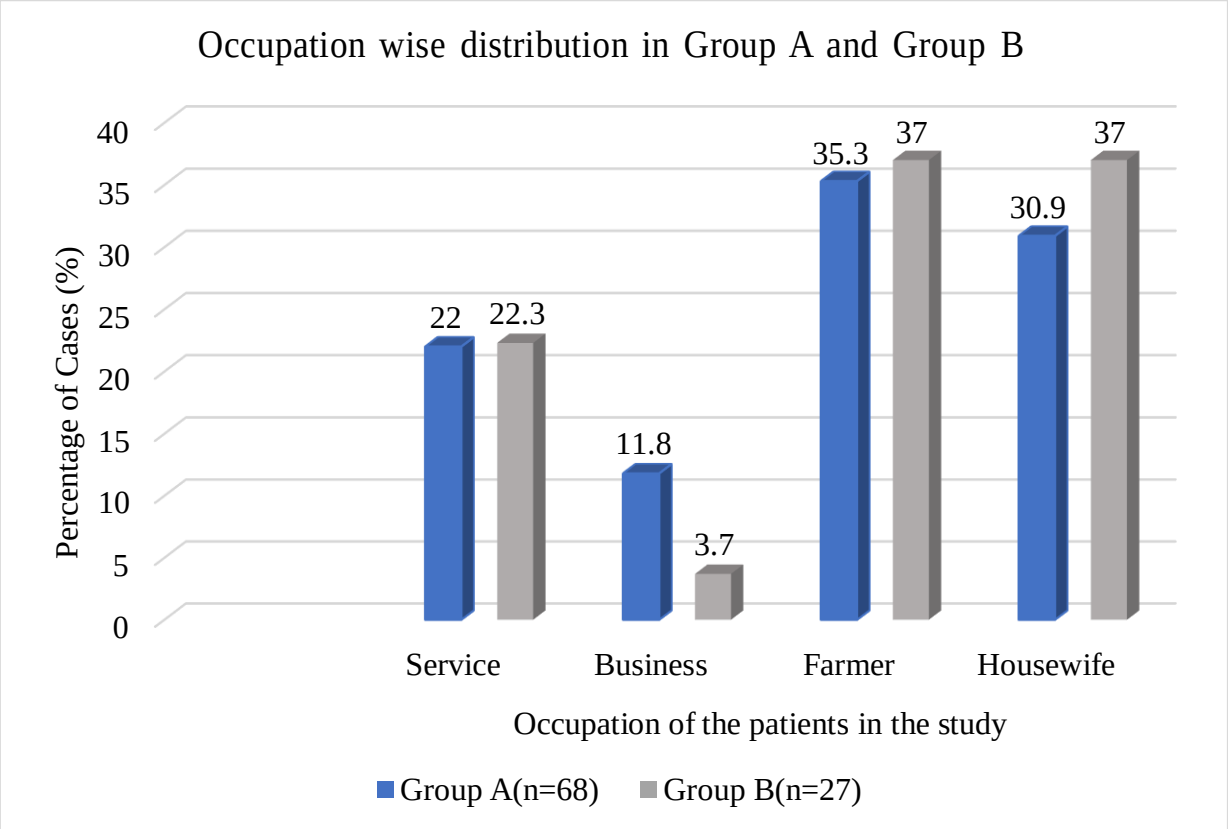
Depending on the nature of work, the patients were subclassified in to 4, Business, service, housewife and farmer.

Group A has the most common occupation as farmer 24(35.3%), followed by Housewife 21(30.9%), Service 15 (22%) and Business 8 (11.8%).

In group B the most common occupation was farmer and housewife both of which were 10 (37%), followed by service 6 (22.3%) and business 1 (3.7%).

The most common occupation in both groups was found to be farmer 34(35.8%) followed by housewife 31(32.6%).

Table 8: Occupation wise distribution in Group A and Group B						
	Group A		Group B		Total	
Occupation	**No.**	**%**	**No.**	**%**	**No.**	**%**
Service	15	22	6	22.3	21	22.1
Business	8	11.8	1	3.7	9	9.5
Farmer	24	35.3	10	37	34	35.8
Housewife	21	30.9	10	37	31	32.6
TOTAL	68	100	27	100	95	100



Graph 3: Occupation wise distribution of patients in Group A and Group B

DISTRIBUTION OF PATIENTS ACCORDING TO RISK FACTORS.

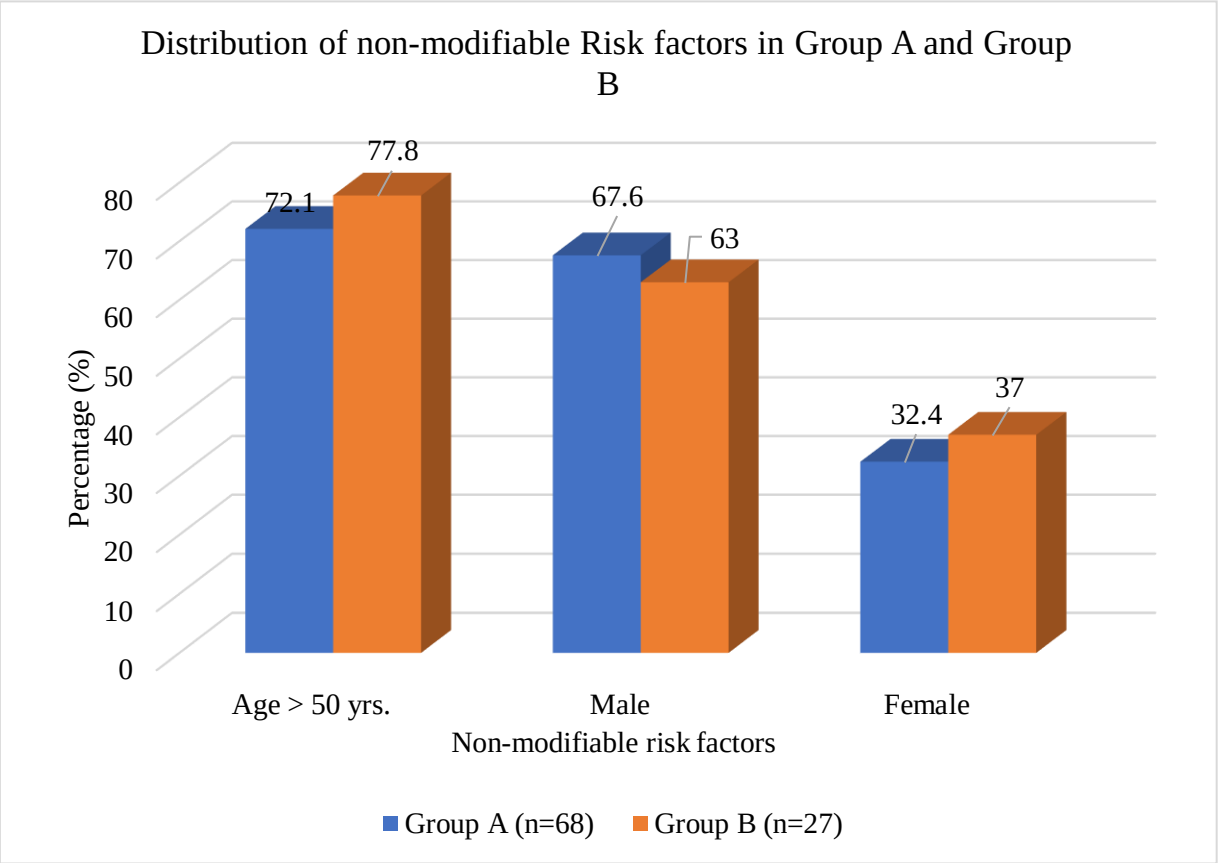
Out of 95 patients in the study, 49 patients (72.1%) in group A and 21 patients (77.8%) in group B were over the age of 50, which was one of the risk factors. Male sex was seen in 46 patients (67.6 %) compared to 17 patients (63 %) in group B.

Smoking was seen in 33 patients of which 24 patients (35.3%) were in group A and nine (33.3%) were in Group B. Hypertension was seen in 21 patients of which 13 patients (19.1%) were in group A and eight patients (29.6%) in group B. Diabetes was seen in six patients of which four patients (5.9%) were in group A and two patients (7.4%) in group B. Among tobacco chewing was seen in 39 patients of which 27 patients (39.7 %) were in group A and 12 patients (44.4%) were in group B. Alcohol consumption was seen in 12 patients who were in group A as seen in Table :9, Graph: 5. There was a noteworthy comparison in alcohol consumption with P value of 0.020.

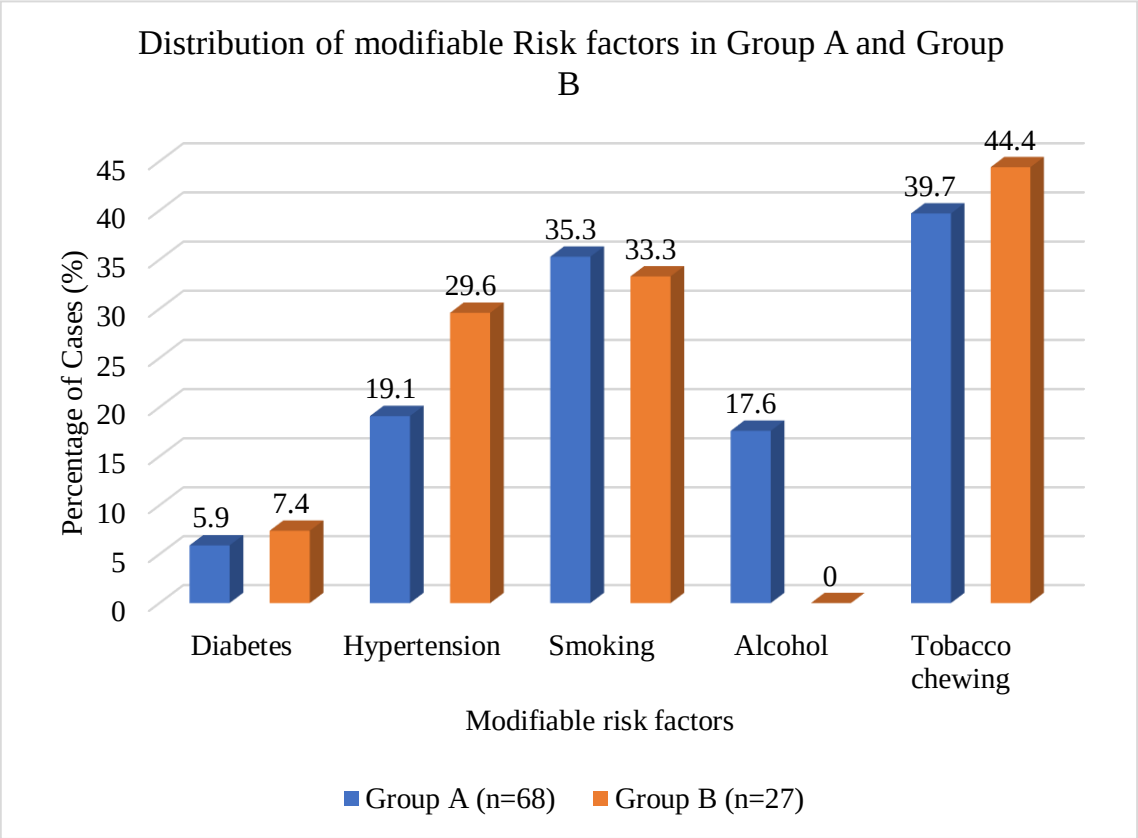
Table 9: Distribution of Risk factors in Group A and Group B.								
Risk factors			**Group A**		**Group B**		**Group A vs Group B**	
			No.	**%**	**No.**	**%**	**X^2**	**P value**
Non-modifiable	**Age**	**> 50 yrs.**	49	72.1	21	77.8	0.326	0.568
	Sex	**Male**	46	67.6	17	63	0.190	0.663
		Female	22	32.4	10	37.0		
Modifiable	**Diabetes**		4	5.9	2	7.4	0.076	0.783
	Hypertension		13	19.1	8	29.6	1.240	0.265
	Smoking		24	35.3	9	33.3	0.033	0.856
	Alcohol		12	17.6	0	0	5.454	0.020*
	Tobacco chewing		27	39.7	12	44.4	0.179	0.672

X^2: Chi-square test

*P < 0.05, significant



Graph 4: Distribution of non-modifiable Risk factors in Group A and Group B



Graph 5: Distribution of modifiable Risk factors in Group A and Group B

DISTRIBUTION OF PATIENTS ACCORDING TO SYMPTOMS

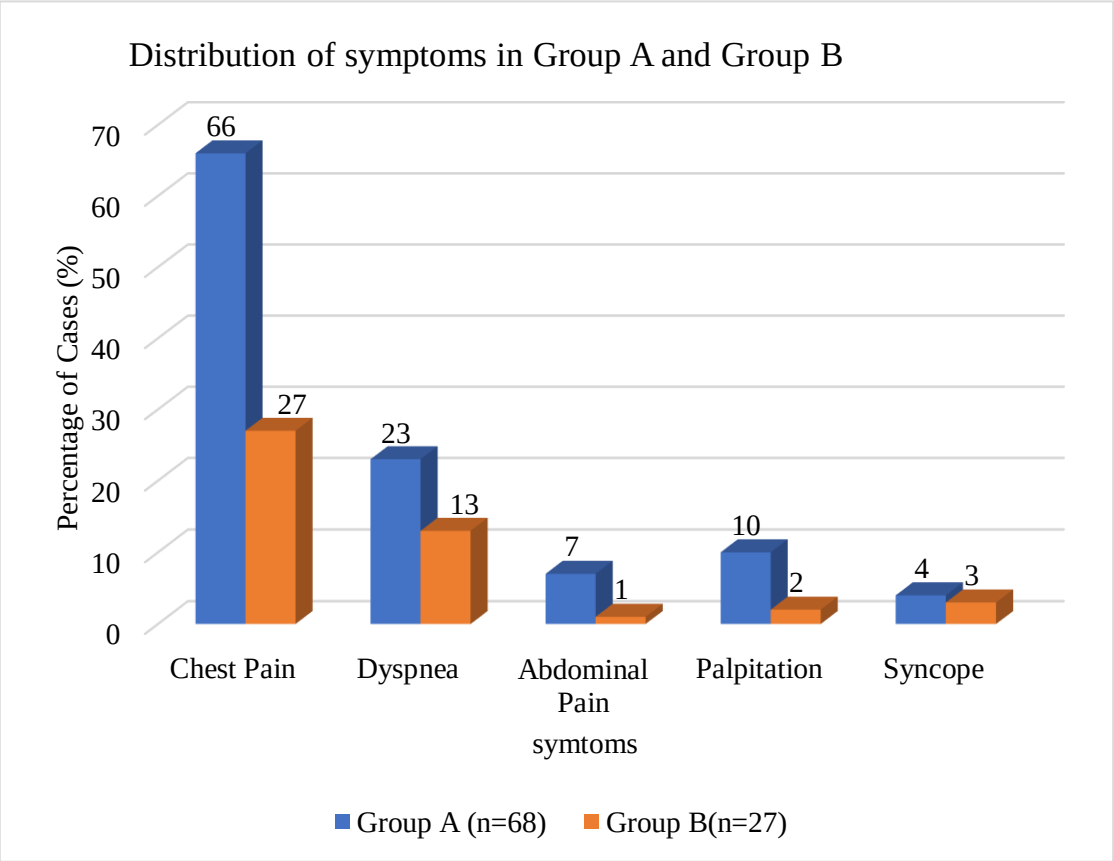
In this study as shown in Table: 10, Graph: 6.

In both group A and group B the most common symptom was chest pain (97.1% vs 100%), followed by dyspnoea (33.8% vs 48.1%), palpitation (14.7% vs 7.4%), abdominal pain (10.3% vs 3.7%) and syncope (5.9% vs 11.1%).

Table 10: Distribution of symptoms in Group A and Group B						
Symptoms	**Group A (n=68)**		**Group B (n=27)**		**Group A vs Group B**	
	No.	**%**	**No.**	**%**	**X^2**	**P value**
Chest Pain	66	97.1	27	100	0.811	0.368, ns
Dyspnoea	23	33.8	13	48.1	1.685	0.194, ns
Abdominal Pain	7	10.3	1	3.7	1.088	0.297, ns
Palpitation	10	14.7	2	7.4	0.933	0.334, ns
Syncope	4	5.9	3	11.1	0.774	0.379, ns

X^2: Chi-square test

P > 0.05, Not Significant.



Graph 6: Distribution of symptoms in Group A and Group B

DISTRIBUTION OF PATIENTS ACCORDING TO ELECTROCARDIOGRAM (ECG) FINDINGS.

Out of 68 patients enrolled in Group A, NSTEMI was seen in 23(33.8%), STEMI-Inferior wall seen in 18(26.5%), STEMI-Antero lateral wall seen in 10 (14.7%) and STEMI-Anterolateral septal wall was seen in 17 (25%).

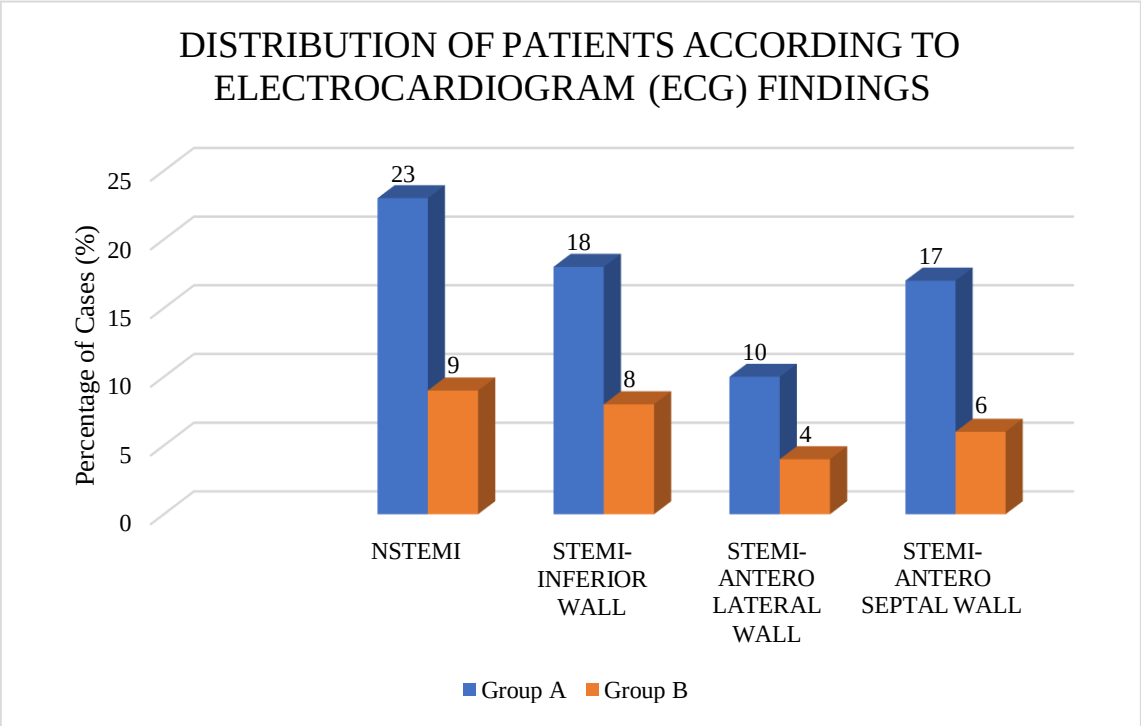
Out of 27 patients enrolled in Group B, NSTEMI was seen in nine (33.3%), STEMI-Inferior wall seen in eight (269.6%), STEMI-Antero lateral wall seen in four (14.8%) and STEMI-Anterolateral septal wall was seen in six (22.2%).

In both the groups, NSTEMI was seen as the most common type of myocardial infarction in patients of acute coronary syndrome.

Table 11: DISTRIBUTION OF PATIENTS ACCORDING TO ELECTROCARDIOGRAM (ECG) FINDINGS IN Group A and Group B

MI	Group A (n=68)		Group B (n=27)		Total (n=95)	
	No.	%	No.	%	No.	%
NSTEMI	23	33.8	9	33.3	32	33.7
STEMI- INFERIOR WALL	18	26.5	8	29.6	26	27.4
STEMI-ANTERO LATERAL WALL	10	14.7	4	14.8	14	14.7
STEMI-ANTERO SEPTAL WALL	17	25.0	6	22.2	23	24.2
Total	68	100.0	27	100.0	96	100.0

$X^2 = 0.134$, $P = 0.988$, NS

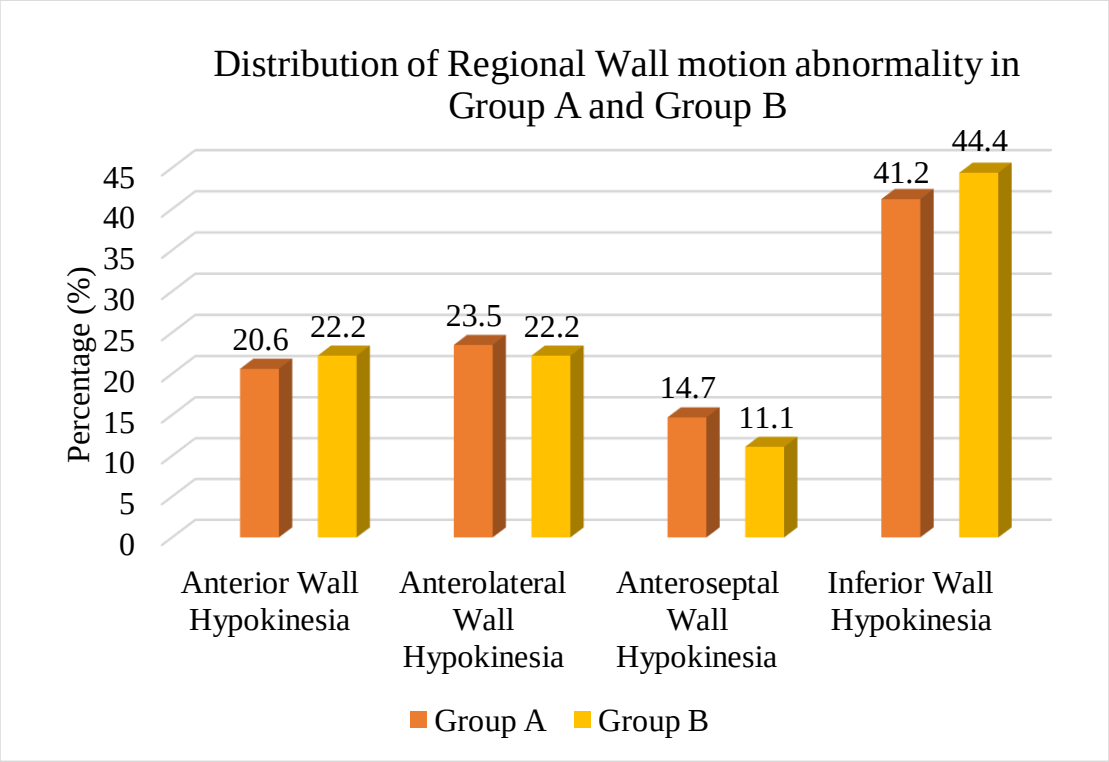


Graph 7: Distribution of patients according to electrocardiogram (ECG) findings in group A and group B

DISTRIBUTION OF PATIENTS ACCORDING TO ECHOCARDIOGRAPHIC VARIABLES:

In this study of 95 patients, echocardiographic parameters were analysed. Out of 68 patients in Group A, 16 patients (23.5%) had antero-lateral wall hypokinesia, 14 patients (20.6%) had anterior wall hypokinesia, ten patients (14.7%) had antero-septal wall hypokinesia and 28 patients (41.2%) had inferior wall hypokinesia. Out of 27 patients in Group B, six patients (22.2%) had antero-lateral wall hypokinesia, six patients (22.2%) had anterior wall hypokinesia, three patients (11.1%) had anteroseptal wall hypokinesia and 12 patients (44.4%) had inferior wall hypokinesia. In this study most commonly, there was hypokinesia of anterolateral wall in group A and inferior wall hypokinesia in group B as shown in Table: 12, Graph:8

Table 12: Distribution of Regional Wall motion abnormality in Group A and Group B						
Regional Wall Motion Abnormality	**Group A (n=68)**		**Group B (n=27)**		**Total (n=95)**	
	No.	**%**	**No.**	**%**	**No.**	**%**
Anterior Wall Hypokinesia	14	20.6	6	22.2	20	21.1
Anterolateral Wall Hypokinesia	16	23.5	6	22.2	22	23.2
Anteroseptal Wall Hypokinesia	10	14.7	3	11.1	13	13.7
Inferior Wall Hypokinesia	28	41.2	12	44.4	40	42.1
Total	68	100	27	100	95	100



Graph 8: Distribution of Regional Wall motion abnormality in Group A and Group B

DISTRIBUTION OF PATIENTS ACCORDING TO LEFT VENTRICULAR EJECTION FRACTION

In this study of 95 patients, they were divided into group A and group B, and distribution of left ventricular ejection fraction was studied as shown in Table:13, Graph :9

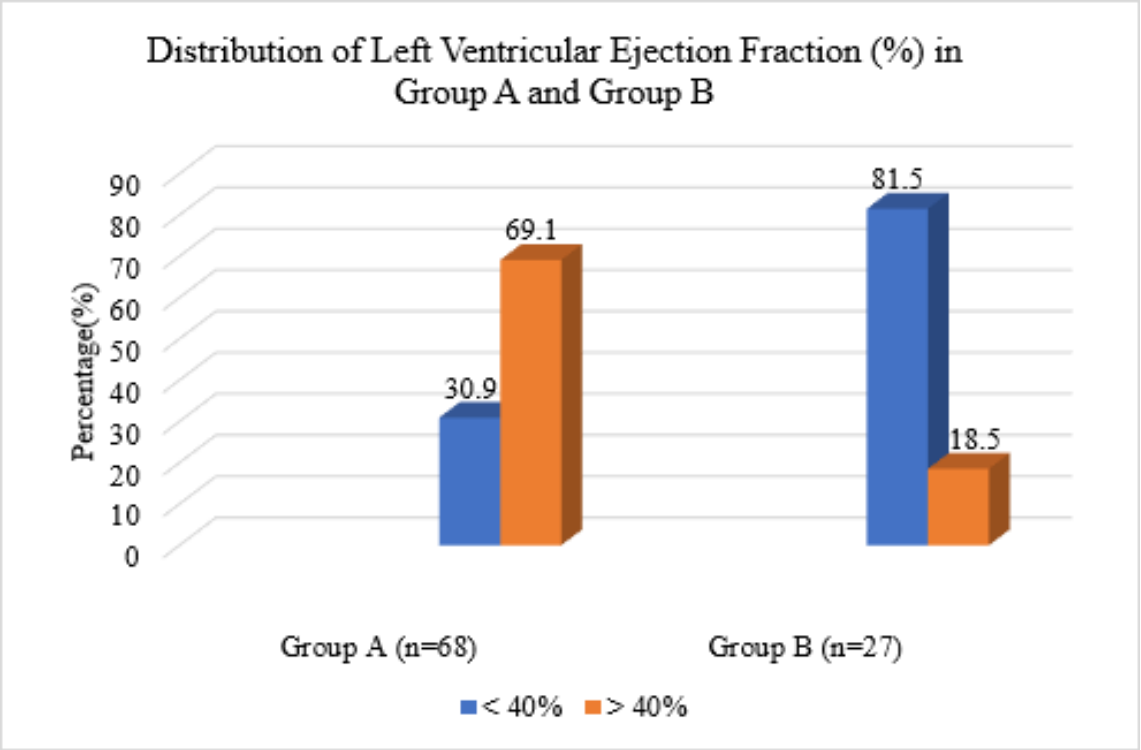
In group A with 68 cases distribution of left ventricular ejection fraction according to regional wall motion abnormality showed LVEF of < 40% in 21 patients (30.9%) and >40% in 47 patients (69.1%). While in group B with 27 cases distribution of left ventricular ejection fraction according to regional wall motion abnormality showed LVEF of <40% in 22 patients (81.5%) and >40% in 5 patients (18.5%).

Table 13: Distribution of Left Ventricular Ejection Fraction (%) in Group A and Group B

LVEF	Group A (n=68)		Group B (n=27)		Group A vs Group B	
	No.	%	No.	%	X^2	P value
< 40%	21	30.9	22	81.5	19.971	0.00*, S
> 40%	47	69.1	5	18.5		
Total	68	100.0	27	100.0	-	

X^2: Chi-square test

$P < 0.05$, S



Graph 9: Distribution of LVEF (%) in Group A and Group B

BACKGROUND PARAMETERS IN STUDY GROUPS

Table 14: Background parameters in Group A and Group B

Parameter			Group A		Group B		Group A vs Group B	
			Mean	SD	Mean	SD	T	P value
Vitals	PR (Beats per minute)		85.118	15.67	87.556	15.215	753	0.173
	BP (mmHg)	Systolic	124.441	21.656	114.815	21.904	1176.000	0.031*
		Diastolic	77.735	12.197	72.222	12.81	1160.500	0.038*
	RR (Cycles per minute)		18.882	5.637	19.37	2.151	693.000	0.057
	Temperature (Degree Celsius)		37.209	0.388	37.189	0.492	971.000	0.626
Hemoglobin(gm%)			12.879	1.996	13.104	2.434	840.500	0.525
Total count(cells/cu.mm)			11018.6	3587.45	12239.1	3723.6	744.500	0.153
ESR (mm/hr.)			17.206	10.202	20	14.085	801.500	0.336
RBS (mg/dl)			120.985	29.61	126.444	59.161	986.500	0.574
Blood Urea (mg/dl)			29.779	10.074	31.519	22.379	996.000	0.522
Serum Creatinine (mg/dl)			0.868	0.219	1.03	0.74	838.500	0.510
Serum Sodium (meq/l)			136.574	3.495	136.741	4.129	916.000	0.990
Serum Potassium (meq/l)			4.188	0.509	4.111	0.455	1030.000	0.356

t: Unpaired t test

* $P < 0.05$, Sig.

$P > 0.05$, Not Sig.

DISTRIBUTION OF PATIENTS ACCORDING MAJOR ADVERSE CARDIAC EVENTS

Heart failure was found in 8.8% patients in group A and 77.8% patients in Group B. It was found statistically significant with a P value <0.05. Pulmonary oedema was found in 10.3% patients among group A compared to 77.8% patients among group B. It was found statistically significant with a P value <0.05. Cardiogenic shock was found in 7.4% patients among group A compared to 40.7% patients among group B, with a p value of < 0.05, statistical significance is determined.

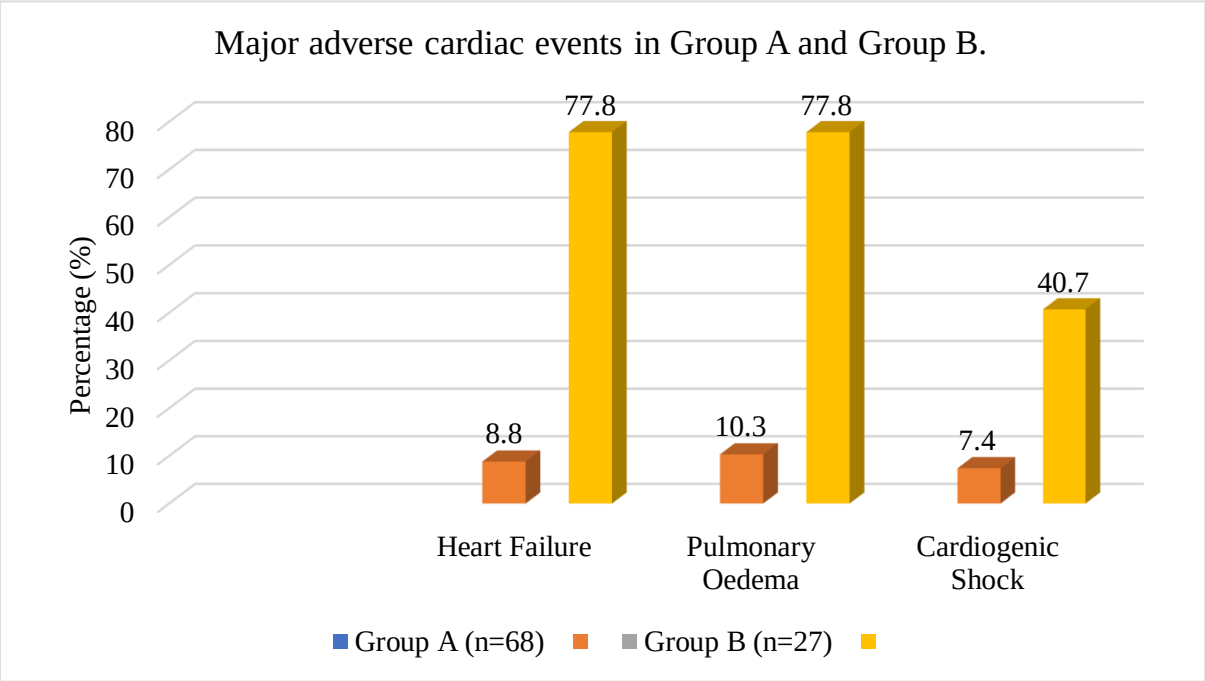
Thus, Frontal QRST angle >100 degrees was related to the occurrence of major adverse events in the patients of Acute myocardial infarction.

Table 15: Major adverse cardiac events in Group A and Group B.

Events	Group A (n=68)		Group B (n=27)		Group A vs. Group B	
	No.	%	No.	%	X^2	P value
Heart Failure	6	8.8	21	77.8	45.170	0.00, S
Pulmonary Oedema	7	10.3	21	77.8	42.341	0.00, S
Cardiogenic Shock	5	7.4	11	40.7	15.382	0.00, S

* P < 0.05, Sig.

P > 0.05, not sig.



Graph 10: Major adverse cardiac events in Group A and Group B

DISCUSSION

VI. DISCUSSION

This study is a prospective observational study conducted from January 2021 to June 2022.The aim was to study the Frontal QRST angle as a predictor of the in-hospital outcome of the adverse cardiac events in patients with acute myocardial infarction.

95 patients included in this study were analysed to predict in-hospital major adverse cardiac events like heart failure, pulmonary oedema, cardiogenic shock, arrhythmias (ventricular tachycardia, supraventricular tachycardia, atrial fibrillation) and death.

1. AGE

In this study the most common age group was 50-70 yrs.

Similarly, in a study done by Abhishek C. Sawant *et al.*[65] in 2019, it was found that among the 267 patients enrolled for the study the mean age was between 50 to 70 years.

In a similar study done by Mark T. Lown *et al.*[66] in 2012, of the 1843 patients the mean age was 70.1+/- 13.1 years. Our results are in line with similar studies conducted before, which implies that increasing age is an important non-modifiable risk factor for the development of myocardial infarction. Also, increased age has an impact on the diagnosis, as it results in decreased sensitivity and specificity of troponins among the patients [72]. So, other diagnostic modalities like ECG should be used for diagnosis and frontal QRST angle measurement, can be used to predict the outcome.

2. SEX

In this study there was male predominance as 63 patients (66.3%) were males and 32 patients (33.7%) were female patients which was similar to a study done by Abhishek C. Sawant *et al.*[65] in a year 2019 on 267 patients where male patients were 187 (70%).

In another study done by B. Zadeh *et al.*[67] from February 2015 to March 2017. Out of 169 patients, 125 (73.96%) were male and 44 (26.03%) females. This shows that there is male

predominance for the development of myocardial infarction. This can be attributed to modifiable risk factors like smoking and alcohol consumption, which are more common in males as compared to women.

3. OCCUPATION

Depending on the nature of work, the patients were subclassified in to 4, Business, service, housewife and farmer. Group A has the most common occupation as farmer 24 (35.3%), followed by Housewife 21(30.9%), Service 15 (22%) and Business 8 (11.8%). In group B the most common occupation was farmer and housewife both of which were 10 (37%), followed by service 6 (22.3%) and business 1 (3.7%). The most common occupation in both groups was found to be farmer 34(35.8%) followed by housewife 31(32.6%).

The reason could be lack of education about disease, risk factors, inability to afford for treatment, lack of compliance to medication, inability to modify risk factors and lack of regular follow up.

4. RISK FACTORS

Non- modifiable risk factors like age and gender are been discussed above.

Modifiable risk factors are those, whose effect on the development of disease can be changed if timely measures are taken to control them.

In this study, modifiable risk factors like alcohol consumption were present in 12 patients (17.6%) in group A and in 0 patients in group B. Hence with a significant p value of 0.02, alcohol and Frontal QRST angle <100 degrees show strong association. Hypertension was seen in 13 patients (19.1%) in group A and 8 patients (29.6%) in group B. Diabetes mellitus was found in 4 patients (5.9%) in group A and 2 patients (7.4%) in group B. Other risk factors like

tobacco chewing in patients (39.7% vs 44.4%), smoking in (35.3% vs 33.3%) was seen in both group A and group B respectively.

There is a significant variation in various risk factors and their association with acute coronary syndrome in different studies.

In a study done by Abhishek Sawant *et al.*[65] in 2019, on 267 patients with acute coronary syndrome, the patients were divided in three groups of FQRST 1-50^0 (Group A,n=118), FQRST 51-100^0 (Group B, n=65) and FQRST 100-180^0 (Group C, n=84). In these groups Diabetes Mellitus was found in 9.4% vs 6.4% vs 21.7%, Smoking was present in 20.6% vs 11.2% vs 44.6%, Hypertension was seen in 28.1% vs 18% vs 26.2%. It was concluded in this study that patients with abnormal FQRST angle, were more likely to have diabetes, hypertension and other minor electrographically diagnosed abnormalities like ST-T wave abnormalities.

In a study done by Joseph A. Walsh III *et al.*[68] in 2013, on 6814 patients with CVD diabetes mellitus was present in 17.7% patients, hypertension in 46.8% patients and smoking in 13.1% (n=329) of patients with FQRST > 78 degrees.

Li YH.*et al.* [60] in 2013 studied the value of the frontal planar QRS-T angle on cardiac dysfunction in patients with old myocardial infarction. In this study the patients were divided into 2 groups based on Frontal QRST angle <90 degrees (n=388) and >90 degrees (n=612). It was found that hypertension was found in 53.4% vs 56.7%, Diabetes in 33.4% vs 29.2%, Smoking in 57.2% vs 58.5%.

Thus, it can be seen that in patients who had modifiable risk factors like smoking, alcohol consumption, hypertension, diabetes, tobacco chewing there is an increased risk of development of MI as well was increased risk of major adverse cardiac events post MI. With timely management of these risk factors by early diagnosis, treatment, lifestyle modifications we can prevent not only the development of MI, but also the risk of major cardiac adverse events.

5. SYMPTOMS

In this study, in both group A and group B, the most common symptom was chest pain (97.1% vs 100 followed by dyspnea (33.8% vs 48.1%), palpitation (14.7% vs 7.4%), abdominal pain (10.3% vs 3.7%) and syncope (5.9% vs 11.1%).

Similarly, in a study done by Harshida Patel *et al.*[69] in 2004, 15 patients admitted with ACS, they found that the most common symptom in patients with acute coronary syndrome was chest pain, followed by dyspnea which was similar to our study. Chest pain can be often described as burning sensation, shooting pain which radiates to the left arm, shoulder or hand. It can often be misdiagnosed as a case of hyperacidity if care is not taken. Early diagnosis of MI is possible, if timely diagnostics are carried when the symptoms occur. This can also help in improving the prognosis and make timely treatment facilities available to the patient.

6. ELECTROCARDIOGRAM

ECG is the most common non-invasive diagnostic test performed for the diagnosis and monitoring of acute myocardial infarction. It is one of the simplest and the fastest test used for the evaluation of the heart.

Out of 68 patients enrolled in Group A, NSTEMI was seen in 23(33.8%), STEMI-Inferior wall seen in 18(26.5%), STEMI-Antero lateral wall seen in 10 (14.7%) and STEMI-Anterolateral septal wall was seen in 17 (25%).

Out of 27 patients enrolled in Group B, NSTEMI was seen in 9(33.3%), STEMI-Inferior wall seen in 8(269.6%), STEMI-Antero lateral wall seen in 4 (14.8%) and STEMI-Anterolateral septal wall was seen in 6 (22.2%).

In a study done by Felice Gragnano *et al.*[70] in 2019, they reported that electrocardiogram plays a major role in diagnosis and stratification of risks in patients with acute coronary syndrome.

A study was conducted by M W Millar-Craig I *et al* [73]. in 1997 to see if there was any change in the outcome of treatment of MI, if the patients were directly admitted to the CCU rather than in-hospital admission, on the basis of their initial ECG. It was concluded in this study that by training the paramedics to diagnose MI by ECG, potentially reduces the time interval of thrombolytic treatment and there by improves the prognosis.

Diagnostics with the ECG can either be carried out in isolation or with other diagnostic tests for myocardial infarction. It gives an idea about the type of MI along with the heart wall involved. Since it is non-invasive, it can be easily performed even at primary health centres. This can further ensure early diagnosis and management of MI, thereby improving the survival and prognostic outcomes.

7. ECHOCARDIOGRAPHY

In this study most commonly, there was hypokinesia of anterolateral wall in group A and inferior wall hypokinesia in group B.

In this study of 95 patients, echocardiographic parameters were analyzed. Out of 68 patients in Group A, 16 patients (23.5%) had antero-lateral wall hypokinesia, 14 patients (20.6%) had anterior wall hypokinesia, 10 patients (14.7%) had antero-septal wall hypokinesia and 28 patients (41.2%) had inferior wall hypokinesia. Out of 27 patients in Group B, 6 patients (22.2%) had antero-lateral wall hypokinesia, 6 patients (22.2%) had anterior wall hypokinesia, 3 patients (11.1%) had anteroseptal wall hypokinesia and 12 patients (44.4%) had inferior wall hypokinesia.

8. LEFT VENTRICULAR EJECTION FRACTION

LVEF helps in assessing the systolic function of the heart. LVEF is an important predictor of cardiac mortality.

In this study of 95 patients, they were divided into group A and group B, and distribution of left ventricular ejection fraction was studied

In group A with 68 cases distribution of left ventricular ejection fraction according to regional wall motion abnormality showed LVEF of < 40% in 21 patients (30.9%) and >40% in 47 patients (69.1%). While in group B with 27 cases distribution of left ventricular ejection fraction according to regional wall motion abnormality showed LVEF of <40% in 22 patients (81.5%) and >40% in 5 patients (18.5%).

Therefore, in patients with Frontal QRST angle >100 degrees there is higher incidence of reduced ejection fraction and depressed left ventricular systolic function.

In a study done by Olga Perelshtein Brezinov *et al.*[71] in 2017, LVEF was studied as an independent factor to predict the prognosis of ACS. It was concluded in this study that there is a strong co-relation between LVEF and prognosis of ACS. In a study done by Li YH *et al* [60]. it was concluded that the planar QRS-T angle and LVEF of patients with old myocardial infarction are negatively correlated, with a bigger planar QRS-T angle indicating a lower LVEF, according to the partial correlation analysis results and when LVEF is less than 50%, the planar QRS-T angle rises every 13.8° and LVEF drops by 5%.

9. MAJOR ADVERSE CARDIAC EVENTS

Heart failure was found in 8.8% patients in group A and 77.8% patients in Group B. It was found statistically significant with a P value <0.05. Pulmonary oedema was found in 10.3% patients among group A compared to 77.8% patients among group B. It was found statistically significant with a P value <0.05. Cardiogenic shock was found in 7.4% patients among group A compared to 40.7% patients among group B. It was found statistically significant with a P value <0.05.

Thus, Frontal QRST angle >100 degrees was related to the occurrence of major adverse events in the patients of Acute myocardial infarction. Our results are in line with a similar study done by Abhishek C Sawant *et al.*[65] in which FQRST was found to be strong predictor of mortality among patients of acute myocardial infarction.

In a study done by Israel Gotsman *et al.* [74] in 2018, on 2929 patients of heart failure it was concluded that Frontal QRST angle has a strong predictive value for outcome and is an ominous sign.

MACE remains one of the strongest adverse outcomes of MI, as concluded in a study done by Poudel I *et al.* [75] done in 2019. A positive relation between an increase in Frontal QRST angle and MACE was found in our study. Frontal QRST angle can thus be used to predict the treatment outcomes of the patients presenting with MI.

CONCLUSION

VII. CONCLUSION

As we are all aware, electrocardiography is a quick, low-cost, and non-invasive method to evaluate heart abnormalities. QRS and T axes can easily be reported from ECG and Frontal QRST angle can be easily calculated from them.

The importance of a Frontal QRST angle in cardiology is far more than is currently recognized We conducted this study to evaluate the relation between Frontal QRST angle and prognosis of AMI. It was found that there was an elevated risk of in-hospital major adverse cardiac outcomes like heart failure, pulmonary oedema, cardiogenic shock in patients with Frontal QRST angle >100 degrees on electrocardiograph in the current study of patients with Acute myocardial infarction. Also, a decrease in LVEF was found with increase in the Frontal QRST angle. As a result, Frontal QRST angle can be utilized to predict in-hospital major adverse cardiac events in patients with acute myocardial infarction.

SUMMARY

VIII. SUMMARY

Ninety-five patients with Acute coronary syndrome admitted in BLDE (Deemed to be University), Shri B M Patil Medical College Hospital and Research Centre, Vijayapura between January 2021 to June 2022were studied.

This study was conducted to know Frontal QRST angle on an electrocardiograph as a predictor of in-hospital outcome of the major adverse cardiac events in patients with Acute myocardial infarction. Patients with both STEMI and NSTEMI were included in the study.

A total of 102 patients were enrolled out of which 7 were excluded based on exclusion criteria. Patients with frontal QRST angle of <100 degrees were classified as group A (68 patients) and patients with frontal QRST angle of >100 degrees as group B (27).

1. In this study Group A 46 (67.6%) patients were male and 22 (32.4%) females, while in group B 17 (63%) were male and 10 (37%) were females.
2. The most common age group in group A was 60-70 years and group B was 50-60 years. There was no significant difference between the age distributions among both the groups.
3. The most common occupation associated with acute myocardial infarction in both group A and group B was farming 34(35.8%). In group A farmers were followed by housewife 21 (30.9%), service 15(22%) and business 8(11.8%). In group B the most common occupation was farmer and housewife both which were 10 (37%), followed by service 6(22.3%) and business 1 (3.7%).
4. The most common risk factors in both groups was Smoking seen in 33 patients of which 24 patients (35.3%) were in group A and 9 (33.3%) were in Group B followed by diabetes mellitus (52.9%), hypertension (41.7%1), alcohol (20.5%) and tobacco chewing (5.8%). Alcohol consumption was seen in 12 patients who were in group A and there was a noteworthy difference in alcohol consumption with p value of 0.020.

5. The most common symptom in both group A and group B was chest pain (97.1% vs 100%), followed by dyspnoea, palpitations, abdominal pain and syncope.
6. In this study most commonly, there was inferior wall hypokinesis in both the groups (41.2% vs 44.4%).
7. In group A around 30.9% patients had ejection fraction less than 40% compared to 81.5% in group B. Therefore, in patients with Frontal QRST angle >100 degrees there is higher incidence of reduced ejection fraction and depressed left ventricular systolic function.
8. In group A, the in-hospital major adverse cardiac events like, pulmonary oedema (10.3%) with significant p value <0.005 followed by heart failure (8.8%) with significant p value <0.005 and cardiogenic shock (7.4%) with significant p value <0.005 were less common when compared to group B, where it was pulmonary oedema and heart failure (77.8%) and cardiogenic shock(40.7%).

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IX. BIBLIOGRAPHY

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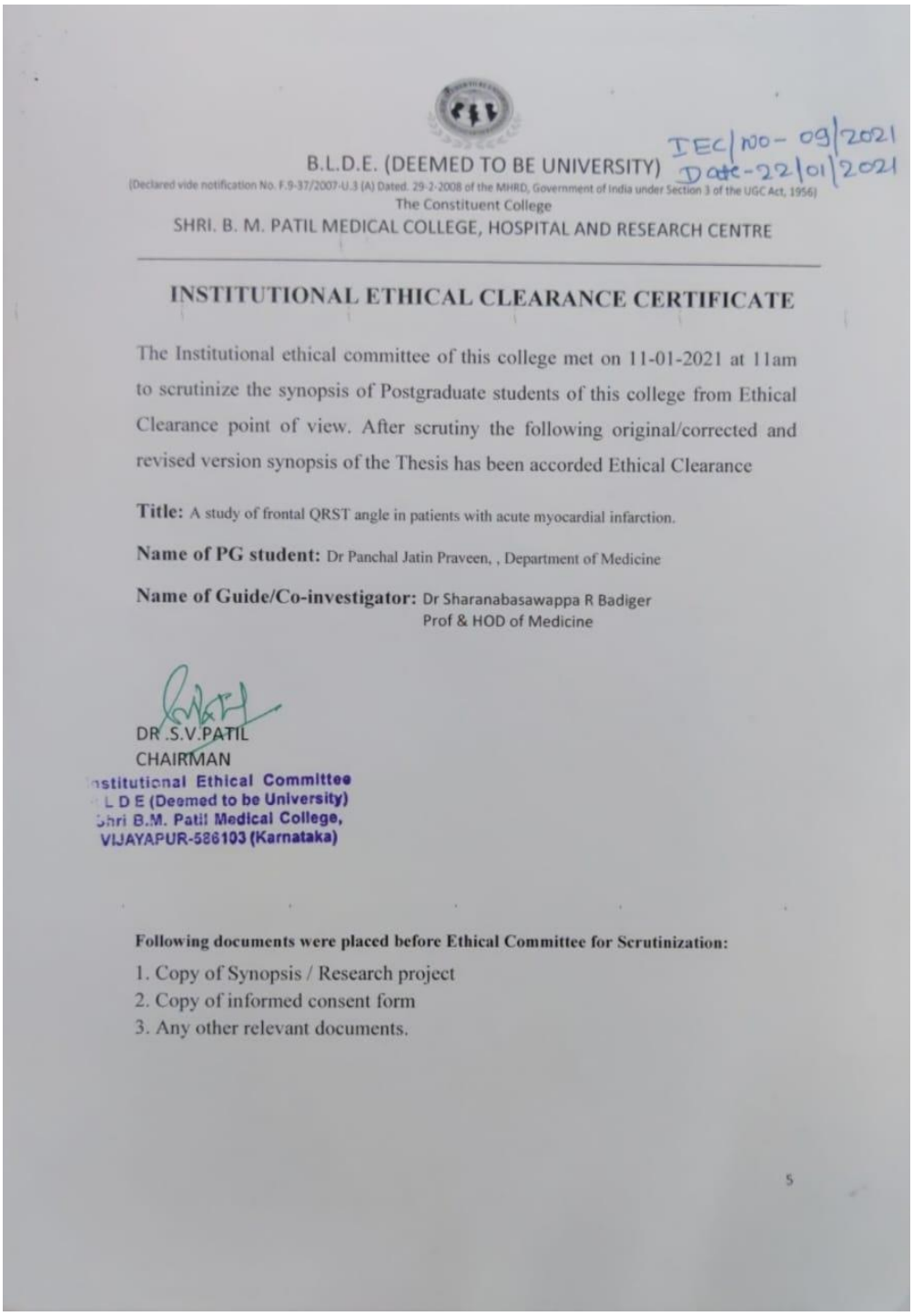
ANNEXURES

ANNEXURE I

INSTITUTIONAL ETHICAL CLEARENCE CERTIFICATE.



B.L.D.E. (DEEMED TO BE UNIVERSITY) IEC/NO- 09/2021 Date-22/01/2021

(Declared vide notification No. F.9-37/2007-U.3 (A) Dated. 29-2-2008 of the MHRD, Government of India under Section 3 of the UGC Act, 1956)

The Constituent College

SHRI. B. M. PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Institutional ethical committee of this college met on 11-01-2021 at 11am to scrutinize the synopsis of Postgraduate students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected and revised version synopsis of the Thesis has been accorded Ethical Clearance

Title: A study of frontal QRST angle in patients with acute myocardial infarction.

Name of PG student: Dr Panchal Jatin Praveen, , Department of Medicine

Name of Guide/Co-investigator: Dr Sharanabasawappa R Badiger
Prof & HOD of Medicine

DR .S.V.PATIL
CHAIRMAN
Institutional Ethical Committee
L D E (Deemed to be University)
Shri B.M. Patil Medical College,
VIJAYAPUR-586103 (Karnataka)

Following documents were placed before Ethical Committee for Scrutinization:

1. Copy of Synopsis / Research project
2. Copy of informed consent form
3. Any other relevant documents.

5

ANNEXURE – II

CONSENT FORM

B.L.D.E. (DEEMED TO BE UNIVERSITY) SHRI B.M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTER, VIJAYAPURA-586103

INFORMED CONSENT FOR PARTICIPATION IN DISSERTATION / RESEARCH.

I, the undersigned,_____,S/O D/O W/O_____, aged ____years, ordinarily resident of_____do hereby state/declare that Dr PANCHAL JATIN PRAVEEN of BLDE (DU), Shri. B. M. Patil Medical College Hospital and Research Centre has examined me thoroughly on_____at_____(place) and it has been explained to me in my own language that I am suffering from _____ disease (condition) and this disease/condition mimic following diseases. Further Doctor Dr PANCHAL JATIN PRAVEEN informed me that he/she is conducting dissertation/research titled "**A STUDY OF FRONTAL QRST ANGLE IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION**" under the guidance of Dr. Badiger Sharanabasawappa requesting my participation in the study. Apart from routine treatment procedure, the pre-operative, operative, post-operative and follow-up observations will be utilized for the study as reference data. Doctor has also informed me that during conduct of this procedure like adverse results may be encountered. Among the above complications most of them are treatable but are not anticipated hence there is chance of aggravation of my condition and in rare circumstances it may prove fatal in spite of anticipated diagnosis and best treatment made available. Further Doctor has informed me that my participation in this study help in evaluation of the results of the study which is useful reference to treatment of other similar cases in near future, and also, I may be benefited in getting relieved of suffering or cure of the disease I am suffering.

The Doctor has also informed me that information given by me, observations made photographs video graphs taken upon me by the investigator will be kept secret and not assessed by the person other than me or my legal hirer except for academic purposes.

The Doctor did inform me that though my participation is purely voluntary, based on information given by me, I can ask any clarification during the course of treatment / study related to diagnosis, procedure of treatment, result of treatment or prognosis. At the same time, I have been informed that I can withdraw from my participation in this study at any time if I want or the investigator can terminate me from the study at any time from the study but not the procedure of treatment and follow-up unless I request to be discharged.

After understanding the nature of dissertation or research, diagnosis made,

mode of treatment, I the undersigned Shri/Smt_____under my full conscious state of mind agree to participate in the said research/dissertation.

Signature of patient:

Signature of doctor:

Witness:

Date:

Place

ANNEXURE – III: SCHEME OF CASE TAKING PROFORMA

BLDE (DEEMED TO BE UNIVERSITY)

SHRI B.M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYAPUR.

"A STUDY OF FRONTAL QRST ANGLE IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION"

Name: CASE NO:

Age: IP NO:

Sex: DOA:

Religion: DOD:

Occupation:

Residence:

Presenting complaints:

History of present illness:

Past History:

Family History:

Personal History:

Diet/appetite

Sleep

Bladder and bowel habits:

Smoking/Tobacco chewing/Alcohol

General Physical Examination:

Vitals

PR:

BP:

RR:

Temp:

Hair:

Eyes:

Pupils:

Nose:

Ears:

Oral Cavity:

Upper Limbs:

Chest:

Abdomen:

Genitalia:

Lower Limbs:

Skin:

SYSTEMIC EXAMINATION

Cardiovascular System

Arterial system:

Pulse

Rate

Rhythm

Volume

Character

Condition of the vessel wall

Radio radial

Radio femoral delay

Other peripheral pulses

Venous system:

Engorged veins in the neck

Blood Pressure

Precordial examination:

Inspection:

Palpation:

Auscultation:

Respiratory System:

Per abdomen:

Central Nervous System:

INVESTIGATIONS

HAEMATOLOGY –

Hemoglobin	gm %
Total WBC counts	Cells/mm^3
Differential counts -	
Neutrophils	%
Lymphocytes	%
Eosinophils	%
Monocytes	%
Basophils	%
ESR	mm after 1 hour

BIOCHEMISTRY–

Random blood sugar	mg/dl
Blood urea	mg/dl
Serum creatinine	mg/dl
Serum sodium	meq/l
Serum potassium	meq/l

URINE EXAMINATION -

Albumin	
Sugar	
Microscopy	

LIPID PROFILE

Total cholesterol	mg/dl
Triglycerides	mg/dl
HDL	mg/dl
LDL	mg/dl

TROPONIN I:

CPK MB:

2D-ECHO/DOPPLER STUDY:

ECG-

	ECG
Standardization	
Rate	
Rhythm	
P wave	

PR interval	
QRS duration	
QRS axis	
QRS duration	
ST-Segment	
T wave	
T axis	
QT	
QTc	
Arrhythmias	
Ectopics	
Heart blocks	

Ecg diagnosis:

FRONTAL QRST ANGLE :

ADVERSE CARDIAC EVENTS :

ANNEXURE IV: MASTER CHART

KEY TO MASTER CHART:

A: ABSENT

AF: ATRIAL FIBRILLATION

B: BUSINESSMAN

DE: DEATH

D: DEPRESSION

E: ELEVATION

EM: EMPLOYEE

FA: FARMER

F: FEMALE

H: HOUSEWIFE

HB-HEART BLOCK

L: LABOUR

LBBB: LEFT BUNDLE BRANCH BLOCK

LVEF: LEFT VENTRICULAR EJECTION FRACTION

M: MALE

MR: MITRAL REGURGITATION

P-PRESENT

SR: SINUS RHYTHM

VT-VENTRICULAR TACHYCARDIA

SL NO	Patient Name	AGE	age	hospital stay in days	SEX	OCCUPATION	D.O.A	D.O.D	CHESTPAIN	DYSPNEA	ABDOMINAL PAIN	PALPITATION	SYNCOPE	DIABETES	HTN	FAMILY HISTORY	SMOKING	ALCOHOL	TOBACCO CHEWING	PR	SYSTOLIC	DIASTOLIC	TEMPERATUR	RR	HEMOGLOBIN	TOTAL COUNT	ESR	RBS	BLOOD UREA	Sr.CREATININE	Sr.SODIUM	Sr.POTASSIUM	TOTAL CHOLESTROL	TRIGLYCERIDES	HDL	LDL	TROPONIN I	CPK MB	ECG FINDINGS	RHYTHEM	FQRST	2D ECHO	LVEF	LVEF	HEART FAILURE	PULMONARY EDEMA	CARDIOGENIC SHOCK	OTHERS	INCLUSION/ EXCLUSION
1	LALITHA SADUGOL	60	>50	7	F	HOUSEWIFE	9-1-21	15-1-21	P	P	A	A	A	A	P	NA	A	A	P	90	180	110	37.5	20	14	12450	10	123	31	0.7	143	4.4	130	90	40	74	P	P	LBBB	Regula	60	GLOBAL HYPOKINESIA	25%	≤40	P	P	A	LBB	P
2	MAHABUBBE CHIMALAGI	45	≤50	7	F	HOUSEWIFE	22-3-21	28-3-21	P	A	A	P	A	A	A	NA	A	A	A	90	110	70	38	18	11	10400	15	110	16	0.5	135	3.8	200	240	40	100	P	P	NSTEMI	Regula	90	HYPOKINESIA OF INFERIOR WALL	45%	>40	A	A	A		
3	MAHANANDA EMM	55	>50	5	F	HOUSEWIFE	31-3-21	4-4-21	P	A	A	A	A	A	A	NA	A	A	P	84	120	80	36.7	18	11	15500	10	135	27	1	141	4.1	150	136	30	96	P	P	NSTEMI	Regula	120	HYPOKINESIA OF ANTERIOR WALL	45%	>40	P	P	A		
4	ANANDKUMAR LONARMATH	58	>50	6	M	DESK JOB	3-4-21	8-4-21	P	A	A	P	A	A	P	NA	P	A	A	98	170	100	38	20	17	10000	11	138	20	0.9	138	5	160	140	36	90	P	P	NSTEMI	Regula	90	HYPOKINESIA OF INFERIOR WALL	45%	>40	A	A	A		
5	RENUKA	51	>50	8	F	HOUSEWIFE	21-4-21	28-4-21	P	A	A	A	A	A	A	NA	A	A	A	84	130	90	37	18	12	7000	15	118	30	0.9	132	3.7	158	110	34	88	P	P	NSTEMI	Regula	60	HYPOKINESIA OF INFERIOR WALL	61%	>40	A	A	A		
6	SHANKAR	70	>50	7	M	FARMER	23-4-21	29-4-21	P	P	A	P	A	A	A	NA	P	A	P	70	120	80	37	18	12	9000	15	120	25	0.9	132	4	164	200	30	80	P	P	STEMI-INFERIOR WALL	Regula	90	HYPOKINESIA OF INFERIOR WALL	45%	>40	A	A	A		
7	SUKHDEV GALAVE	60	>50	6	M	FARMER	23-4-21	28-4-21	P	P	A	P	A	A	A	NA	P	A	P	70	118	70	38	16	14	9600	14	118	38	1.5	139	4.6	190	150	34	130	P	P	STEMI-INFERIOR WALL	Regula	60	HYPOKINESIA OF INFERIOR WALL	45%	>40	A	A	A		
8	LAXMIBAI PATIL	55	>50	7	F	HOUSEWIFE	24-4-21	30-4-21	P	A	A	A	A	A	A	NA	A	A	A	76	140	90	37	16	12	7200	11	162	25	1.1	138	3.9	174	180	30	78	P	P	NSTEMI	Regula	30	HYPOKINESIA OF ANTERO LATERAL	45%	>40	A	A	A		
9	SHIVALINGAPPA	48	≤50	6	M	TEACHER	10-5-21	15-5-21	P	P	A	A	A	A	A	NA	P	A	A	90	100	60	38	18	15	11640	20	110	27	1.3	132	3.7	190	146	38	88	P	P	STEMI-ANTERO SEPTAL WALL	Regula	180	HYPOKINESIA OF ANTERO LATERAL	25%	≤40	P	P	P		
10	CHINAWWA SAVADATTI	58	>50	7	F	HOUSEWIFE	15-5-21	21-5-21	P	P	A	P	A	A	A	NA	A	A	A	110	110	70	37	24	8.7	12500	13	100	52	0.9	134	4	131	215	27	61	P	P	STEMI-ANTERO SEPTAL WALL	Regula	90	HYPOKINESIA OF ANTERO LATERAL	35%	≤40	A	A	A		
11	MHD.RAFIQ WALIKAR	54	>50	8	M	BUSINESSMAN	15-5-21	23-5-21	P	P	A	A	A	P	A	NA	A	A	P	86	100	70	37	24	15	10200	21	206	19	1	138	3.7	140	200	28	70	P	P	STEMI-ANTERO SEPTAL WALL	Regula	90	HYPOKINESIA OF ANTERO LATERAL	30%	≤40	P	P	P		
12	BHAGIRATHI GULED	45	≤50	8	F	HOUSEWIFE	18-5-21	25-5-21	P	A	A	P	A	A	P	NA	A	A	A	90	90	70	37	18	16	8310	20	130	25	0.7	132	4	220	240	42	88	P	P	LBBB	Regula	60	GLOBAL HYPOKINESIA	25%	≤40	P	P	A	LBB	P
13	SHANTABAI TADALGI	60	>50	6	F	HOUSEWIFE	27-5-21	1-6-21	A	P	A	P	A	A	A	NA	A	A	A	84	100	60	37	18	15	14690	10	116	32	0.9	139	3.4	180	110	40	90	P	P	NSTEMI	Regula	90	HYPOKINESIA OF ANTERIOR WALL	45%	>40	A	A	A		
14	SITARAM RAJPUT	60	>50	5	M	SHOP KEEPER	4-6-21	8-6-21	P	A	A	A	A	A	P	NA	A	A	A	90	130	80	37.5	18	13	20580	5	110	40	0.6	142	4.6	190	145	30	130	P	P	STEMI-ANTERO SEPTAL WALL	Regula	120	HYPOKINESIA OF ANTERO LATERAL	55%	>40	A	A	A		
15	SANGAPPA	49	≤50	6	M	FARMER	12-6-21	17-6-21	P	A	A	A	A	A	P	NA	A	A	P	88	130	70	37	18	11	15000	13	110	60	1	135	4.8	170	130	30	90	P	P	STEMI-ANTERO SEPTAL WALL	Regula	90	HYPOKINESIA OF ANTERIOR WALL	45%	>40	A	A	A		
16	DASTAGIRSAB NADAF	60	>50	7	M	BUSINESSMAN	16-6-21	22-6-21	P	A	P	A	A	A	P	NA	P	A	P	94	110	70	37	20	16	12150	10	116	16	0.9	140	4.2	179	154	49	99	P	P	STEMI-ANTERO SEPTAL WALL	Regula	90	HYPOKINESIA OF ANTERO SEPTAL	45%	>40	A	A	A		
17	SHANTABAI BALI	80	>50	6	F	HOUSEWIFE	21-6-21	26-6-21	P	A	A	A	A	A	A	NA	A	A	A	80	110	70	37	18	12	12000	14	124	14	1.2	139	4.2	190	147	33	128	P	P	NSTEMI	Regula	30	HYPOKINESIA OF ANTERO LATERAL	40%	≤40	A	A	A		
18	BASAPPA LONI	70	>50	9	M	FARMER	22-6-21	1-7-21	P	P	A	A	A	A	A	NA	P	P	P	96	90	60	38	24	10	2500	30	89	31	1.2	137	4.3	88	59	30	60	P	P	STEMI-INFERIOR WALL	Regula	30	HYPOKINESIA OF ANTERO LATERAL	30%	≤40	P	P	P		
19	RAJSHEKHAR SINDAGI	65	>50	8	M	FARMER	23-6-21	1-7-21	P	P	A	A	A	A	A	NA	P	A	P	100	100	60	37	18	15	3600	5	125	29	0.9	139	3.8	136	188	29	69	P	P	STEMI-INFERIOR WALL	Regula	120	HYPOKINESIA OF INFERIOR WALL	40%	≤40	P	P	P		
20	YASHUBAI	60	>50	5	F	HOUSEWIFE	24-6-21	28-6-21	P	P	A	P	A	A	P	NA	A	A	P	90	90	60	38	24	14	10140	15	120	22	0.7	136	4.3	303	184	48	200	P	P	STEMI-ANTERO LATERAL WALL	Regula	120	HYPOKINESIA OF ANTERIOR WALL	30%	≤40	P	P	P		
21	SAHEBAGOUDA IRAPPA	39	≤50	11	M	CLERK	6-7-21	16-7-21	P	A	A	A	A	A	A	NA	A	A	A	90	130	80	37	18	16	10056	20	121	24	0.9	138	3.5	144	92	38	88	P	P	STEMI-ANTERO LATERAL WALL	Regula	120	HYPOKINESIA OF ANTERO SEPTAL	25%	≤40	P	P	P		
22	GOURAMMA NUCHCHI	75	>50	7	F	HOUSEWIFE	9-7-21	15-7-21	P	P	A	A	A	A	A	NA	A	A	A	98	120	70	37	60	13	13000	13	140	34	0.6	137	4.6	177	57	43	123	P	P	STEMI-ANTERO SEPTAL WALL	Regula	60	HYPOKINESIA OF ANTERIOR WALL	45%	>40	A	A	A		
23	SUSHILBHAI CHAVAN	55	>50	6	F	HOUSEWIFE	11-7-21	16-7-21	P	A	A	A	A	A	A	NA	A	A	A	86	140	90	37	18	10	6400	15	126	16	0.8	137	3.5	166	333	28	71	P	P	NSTEMI	Regula	30	HYPOKINESIA OF INFERIOR WALL	45%	>40	A	A	A		
24	ASHOK HADA PAD	65	>50	11	M	FARMER	12-7-21	23-7-21	P	P	A	A	A	A	A	NA	A	A	P	60	100	60	37	18	12	10000	15	96	35	1.2	136	4.2	59	58	16	40	P	P	STEMI-INFERIOR WALL	Regula	120	HYPOKINESIA OF INFERIOR WALL	35%	≤40	P	P	A		
25	NIRMALA PATTAR	38	≤50	11	F	HOUSEWIFE	13-7-21	24-7-21	P	P	A	P	A	A	A	NA	A	A	A	88	110	70	37.8	15	15	7740	26	100	26	0.7	139	4.2	190	56	38	140	P	P	NSTEMI	Regula	0	HYPOKINESIA OF INFERIOR WALL	50%	>40	A	A	A		

SL NO	Patient Name	AGE	age	hospital stay in days	SEX	OCCUPATION	D.O.A	D.O.D	CHESTPAIN	DYSPNEA	ABDOMINAL PAIN	PALPITATION	SYNCOPE	DIABETES	HTN	FAMILY HISTORY	SMOKING	ALCOHOL	TOBACCO CHEWING	PR	SYSTOLIC	DIASTOLIC	TEMPERATUR	RR	HEMOGLOBIN	TOTAL COUNT	ESR	RBS	BLOOD UREA	Sr.CREATININE	Sr.SODIUM	Sr.POTASSIUM	TOTAL CHOLESTROL	TRIGLYCERIDES	HDL	LDL	TROPONIN I	CPK MB	ECG FINDINGS	RHYTHEM	FQRST	2D ECHO	LVEF	LVEF	HEART FAILURE	PULMONARY EDEMA	CARDIOGENIC SHOCK	OTHERS	INCLUSION/ EXCLUSION
26	KANTABAI BELAGE	70	>50	13	F	HOUSEWIFE	13-7-21	25-7-21	P	P	A	A	A	A	A	NA	A	A	A	90	90	60	37.5	24	11	13570	23	126	38	1	135	4.3	269	164	52	184	P	P	STEMI-ANTERO LATERAL WALL	Regula	90	HYPOKINESIA OF ANTERO LATERAL	25%	≤40	P	P	P		I
27	BASAVANAGOUDA NAGAPPA	68	>50	6	M	FARMER	13-7-21	18-7-21	P	P	A	A	A	A	A	NA	P	P	P	64	110	80	36	16	11	9000	14	124	24	1	135	4.2	154	80	26	112	P	P	STEMI-ANTERO LATERAL WALL	Regula	60	HYPOKINESIA OF ANTERIOR WALL	45%	>40	A	A	A		I
28	CHANDRAVVA	58	>50	10	F	HOUSEWIFE	15-7-21	24-7-21	P	A	A	A	A	A	A	NA	A	A	A	110	110	70	37.5	18	8.7	12500	16	145	52	0.9	120	4	110	68	50	72	P	P	STEMI-INFERIOR WALL	Regula	90	HYPOKINESIA OF ANTERO SEPTAL	35%	≤40	P	P	A		I
29	SUREKHA	45	≤50	6	F	HOUSEWIFE	16-7-21	21-7-21	P	A	A	A	A	A	A	NA	A	A	A	90	120	80	37	18	8.2	14300	35	134	28	0.6	139	4	140	180	32	60	P	P	NSTEMI	Regula	30	HYPOKINESIA OF ANTERO SEPTAL	45%	>40	A	A	A		I
30	SANGANGOUDA	65	>50	3	M	BUS DRIVER	17-7-21	19-7-21	P	P	A	A	A	A	A	NA	A	A	A	92	120	70	36	20	15	10450	29	104	23	0.9	129	4.6	240	124	46	180	P	P	NSTEMI	Regula	120	HYPOKINESIA OF ANTERO SEPTAL	35%	≤40	P	P	A		I
31	SURESH SHRISHAIL	41	≤50	5	M	CLERK	17-7-21	21-7-21	P	P	A	A	A	A	A	NA	P	A	A	80	100	70	37	18	14	8040	3	100	11	0.6	128	4.7	235	161	53	150	P	P	STEMI-INFERIOR WALL	Regula	0	HYPOKINESIA OF INFERIOR WALL	45%	>40	A	A	A		I
32	IRAYYA hiremath	58	>50	13	M	FARMER	20-7-21	2-8-21	P	A	A	A	A	A	P	NA	P	A	P	98	160	90	38	16	16	14860	15	100	32	1.6	140	3.7	140	116	40	77	P	P	STEMI-INFERIOR WALL	Regula	120	HYPOKINESIA OF ANTERO LATERAL	35%	≤40	P	P	A		I
33	LAXMIBAI SIDAREDDY	50	≤50	11	F	HOUSEWIFE	23-7-21	2-8-21	P	P	A	A	A	P	A	NA	A	A	A	116	90	60	38	22	15	18720	25	209	23	1.2	134	4.2	190	150	28	138	P	P	STEMI-ANTERO SEPTAL WALL	Regula	180	HYPOKINESIA OF ANTERIOR WALL	25%	≤40	P	P	P		I
34	MAHADEVI GOUDAPPANAV	66	>50	5	F	HOUSEWIFE	30-7-21	4-8-21	P	P	A	A	A	A	A	NA	A	A	A	84	150	90	37	20	10	11600	5	108	20	1	139	3.2	140	200	28	74	P	P	NSTEMI	Regula	60	HYPOKINESIA OF ANTERIOR WALL	45%	>40	A	A	A		I
35	MALLAPPA MIRAGI	52	>50	11	M	FARMER	31-7-21	10-8-21	P	P	A	A	P	A	A	NA	P	A	A	66	120	80	37	20	17	11600	15	109	18	1.2	134	4.3	180	140	30	92	P	P	STEMI-ANTERO SEPTAL WALL	Regula	180	HYPOKINESIA OF ANTERO SEPTAL	35%	≤40	P	P	A		I
36	MALLIKARJUN KALLAPA	63	>50	7	M	FARMER	31-7-21	5-8-21	P	P	A	A	A	A	A	NA	P	A	P	64	110	70	37	16	9.7	4460	15	99	40	1	137	3.6	184	130	34	90	P	P	STEMI-INFERIOR WALL	Regula	90	HYPOKINESIA OF INFERIOR WALL	50%	>40	A	A	A		I
37	CHANNAYYA MATHAD	70	>50	8	M	FARMER	2-8-21	10-8-21	P	P	A	A	P	A	P	NA	P	A	A	86	110	70	38	22	17	14420	25	116	17	1	136	4.3	210	140	30	98	P	P	STEMI-INFERIOR WALL	Regula	120	HYPOKINESIA OF INFERIOR WALL	30%	≤40	P	P	P		I
38	SADASHIV SHIDHARAY	47	≤50	5	M	FARMER	2-8-21	7-8-21	P	A	A	A	A	A	A	NA	A	A	P	84	120	70	37	16	15	8000	11	120	16	1.1	136	3.6	150	148	26	74	N	P	UNSTABLE ANGINA	Regula	30	HYPOKINESIA OF ANTERIOR WALL	63%	>40	A	A	A	ua	E
39	CHANABASAYYA NANDIKOL	42	≤50	5	M	BUSINESSMAN	2-8-21	7-8-21	P	P	A	A	A	A	A	NA	P	P	A	100	130	90	37	16	15	12506	30	112	32	0.9	140	4.3	130	80	30	66	P	P	STEMI-ANTERO SEPTAL WALL	Regula	0	HYPOKINESIA OF ANTERIOR WALL	45%	>40	A	A	A		I
40	RAJASHEKAR AMBIGAR	59	>50	7	M	BANK EMPLOYEE	4-8-21	10-8-21	P	P	A	A	A	A	P	NA	A	A	A	90	180	120	37	20	14	15890	13	108	27	1.2	134	4.9	114	150	36	90	P	P	STEMI-INFERIOR WALL	Regula	60	HYPOKINESIA OF INFERIOR WALL	40%	≤40	A	A	A		I
41	IRAGONDAPPA	67	>50	5	M	FARMER	12-8-21	16-8-21	A	A	A	P	A	A	P	NA	P	P	A	84	170	80	37	20	13	10910	45	114	26	0.8	134	4.4	113	72	33	66	P	P	NSTEMI	Regula	60	HYPOKINESIA OF INFERIOR WALL	30%	≤40	A	A	A		I
42	MAHADEV JAMBAGI	57	>50	8	M	CLERK	13-8-21	19-8-21	P	A	A	A	A	A	A	NA	A	A	A	66	130	80	37	16	12	8720	40	120	25	0.7	136	3.9	192	264	31	108	P	P	NSTEMI	Regula	30	HYPOKINESIA OF INFERIOR WALL	60%	>40	A	A	A		I
43	NINGAPPA BHEEMAPPA	66	>50	7	M	FARMER	19-8-21	25-8-21	P	A	P	P	A	A	A	NA	A	A	A	140	110	70	38	20	15	18000	25	78	29	0.8	136	4.1	130	92	32	80	P	P	NSTEMI	Regula	30	HYPOKINESIA OF ANTERIOR WALL	50%	>40	A	A	A		I
44	SHANTABAI	80	>50	10	F	HOUSEWIFE	21-8-21	30-8-21	P	P	A	A	A	A	A	NA	A	A	P	80	110	70	36.5	18	12	15400	25	116	25	0.7	138	3.8	120	100	38	70	P	P	STEMI-ANTERO SEPTAL WALL	Regula	120	HYPOKINESIA OF ANTERIOR WALL	35%	≤40	P	P	A		I
45	PRALAD	55	>50	6	M	CLERK	25-8-21	30-8-21	P	A	A	A	P	A	A	NA	P	P	A	76	110	70	37.5	22	13	6570	5	110	25	0.7	134	4.6	150	110	38	60	P	P	STEMI-ANTERO SEPTAL WALL	Regula	60	HYPOKINESIA OF ANTERIOR WALL	45%	>40	A	A	A		I
46	AKSHAY KUMAR	31	≤50	6	M	CLERK	25-8-21	1-9-21	P	A	A	A	A	A	A	NA	P	P	A	100	110	80	37.5	22	12	10400	5	116	56	1.2	140	4.6	130	90	34	80	P	P	STEMI-INFERIOR WALL	Regula	60	HYPOKINESIA OF INFERIOR WALL	45%	>40	A	A	A		I
47	BHIMRAY PADAGANUR	61	>50	6	M	BUSINESSMAN	26-8-21	1-9-21	P	A	A	A	A	P	A	NA	A	A	P	78	126	90	37	14	15	10250	5	186	32	0.8	137	4.4	118	110	43	82	P	P	NSTEMI	Regula	30	NO RWMA	60%	>40	A	A	A		I
48	RUDRAPPA	60	>50	7	M	FARMER	26-8-21	2-9-21	P	A	A	A	A	A	A	NA	P	P	A	120	110	80	37.5	22	12	15460	15	120	46	1.1	140	3.8	160	120	30	70	P	P	STEMI-ANTERO LATERAL WALL	Regula	90	HYPOKINESIA OF ANTERO LATERAL	35%	≤40	A	A	A		I
49	ROZUSAB	56	>50	6	M	MECHANIC	9-9-21	14-9-21	P	A	A	A	A	A	A	NA	P	A	P	78	120	80	37.5	18	14	7170	25	90	16	0.7	140	4.6	185	298	53	73	P	P	STEMI-ANTERO SEPTAL WALL	Regula	30	HYPOKINESIA OF ANTERO SEPTAL	35%	≤40	A	A	A		I
50	AMBARAYYA	60	>50	13	M	FARMER	10-9-21	22-9-21	P	P	A	P	A	A	A	NA	P	A	P	90	80	50	37	22	13	10250	36	113	140	4.5	141	5.2	108	157	14	63	P	P	STEMI-ANTERO LATERAL WALL	Regula	150	HYPOKINESIA OF ANTERO LATERAL	30%	≤40	P	P	P		I
51	MANSINGH NAIK	82	>50	8	M	FARMER	4-10-21	11-10-21	P	A	A	A	A	A	A	NA	P	A	P	90	110	70	36.5	18	13	10500	10	130	26	1.2	136	3.1	174	200	28	60	P	P	LBBB	Regula	120	HYPOKINESIA OF ANTERO SEPTAL	45%	>40	A	A	A	LBBB	E
52	ASHOK	50	≤50	7	M	BUSINESSMAN	30-11-21	5-12-21	P	P	A	A	A	A	A	NA	P	A	A	90	110	70	38	22	12	19700	10	120	34	0.7	138	5	170	90	32	84	P	P	STEMI-INFERIOR WALL	Regula	0	HYPOKINESIA OF ANTERIOR WALL	30%	≤40	A	A	A		I

SL NO	Patient Name	AGE	age	hospital stay in days	SEX	OCCUPATION	D.O.A	D.O.D	CHESTPAIN	DYSPNEA	ABDOMINAL PAIN	PALPITATION	SYNCOPE	DIABETES	HTN	FAMILY HISTORY	SMOKING	ALCOHOL	TOBACCO CHEWING	PR	SYSTOLIC	DIASTOLIC	TEMPERATUR	RR	HEMOGLOBIN	TOTAL COUNT	ESR	RBS	BLOOD UREA	Sr.CREATININE	Sr.SODIUM	Sr.POTASSIUM	TOTAL CHOLESTROL	TRIGLYCERIDES	HDL	LDL	TROPONIN I	CPK MB	ECG FINDINGS	RHYTHEM	FQRST	2D ECHO	LVEF	LVEF	HEART FAILURE	PULMONARY EDEMA	CARDIOGENIC SHOCK	OTHERS	INCLUSION/ EXCLUSION
53	VISHWANATH	57	>50	9	M	FARMER	2-12-21	10-12-21	P	P	A	A	A	A	P	NA	A	A	A	64	110	70	37	19	13	9000	16	108	32	0.9	140	3.8	174	110	36	84	P	P	NSTEMI	Regular	150	HYPOKINESIA OF INFERIOR WALL	35%	≤40	P	P	A		I
54	KULAPPA BANIKOL	43	≤50	11	M	SHOP KEEPER	2-12-21	12-12-21	P	A	A	A	A	A	A	NA	P	A	P	90	120	70	37	16	15	12300	14	99	28	0.6	130	4.4	190	150	26	94	P	P	NSTEMI	Regular	150	HYPOKINESIA OF ANTERO LATERAL	30%	≤40	P	P	A		I
55	DUNDAPPA KATNALLI	50	≤50	12	M	BUSINESSMAN	2-12-21	13-12-21	P	A	A	A	A	A	A	NA	A	A	P	108	140	90	37	17	12	12000	12	112	34	1.1	143	3.7	144	82	32	76	P	P	NSTEMI	Regular	180	HYPOKINESIA OF ANTERIOR WALL	35%	≤40	P	P	P		I
56	MANOHAR NISTANE	59	>50	6	M	EMPLOYEE	7-12-21	13-12-21	P	A	A	A	A	A	A	NA	A	A	A	72	122	76	37	16	14	8900	12	109	23	0.9	136	4.2	140	92	30	86	P	P	NSTEMI	Regular	30	HYPOKINESIA OF INFERIOR WALL	45%	>40	A	A	A		I
57	NEELAGANGA SOMAPPA	55	>50	6	F	HOUSEWIFE	7-12-21	13-12-21	P	P	A	A	A	A	A	NA	A	A	A	80	110	70	37	18	15	10400	11	98	24	0.6	137	4	158	140	36	84	P	P	STEMI-ANTERO LATERAL WALL	Regular	60	HYPOKINESIA OF ANTERO LATERAL	55%	>40	A	A	A		I
58	SADIK RAJASAHEB	54	>50	7	M	MECHANIC	8-12-21	14-12-21	P	P	A	A	A	A	A	NA	P	A	P	82	140	90	37	18	12	7500	5	110	18	1.1	141	3.6	130	110	40	74	N	P	UNSTABLE ANGINA	Regular	30	NO RWMA	60%	>40	A	A	A	ua	E
59	HANAMANTRA I JATTEPPA	65	>50	6	M	FARMER	8-12-21	13-12-21	P	A	A	A	A	A	A	NA	A	A	P	70	94	60	37	20	13	11600	12	98	34	1.2	132	4.2	180	150	34	90	P	P	STEMI-ANTERO LATERAL WALL	Regular	30	HYPOKINESIA OF ANTERO LATERAL	45%	>40	A	A	A		I
60	KALLAPPA KOMAR	48	≤50	7	M	FARMER	9-12-21	15-12-21	P	A	A	A	A	A	A	NA	P	A	A	70	110	70	37	14	12	9400	15	117	28	1	140	3.7	130	200	30	60	P	P	STEMI-ANTERO SEPTAL WALL	Regular	30	HYPOKINESIA OF ANTERO LATERAL	45%	>40	A	A	A		I
61	SHANKERAPPA SIDAPPA	45	≤50	7	M	FARMER	10-12-21	16-12-21	P	P	A	A	A	A	A	NA	P	A	P	84	110	70	37	20	16	8460	23	110	30	0.8	139	4.2	140	90	30	82	P	P	STEMI-ANTERO LATERAL WALL	Regular	60	HYPOKINESIA OF ANTERO LATERAL	50%	>40	A	A	A		I
62	NINGANGONDA PPA	70	>50	7	M	FARMER	13-12-21	19-12-21	P	P	A	A	A	A	A	NA	A	A	P	86	110	70	37	17	12	14000	10	110	27	0.9	137	5	190	160	28	90	P	P	STEMI-INFERIOR WALL	Regular	30	HYPOKINESIA OF INFERIOR WALL	45%	>40	A	A	A		I
63	HANUMANTAP PA	40	≤50	6	M	SHOP KEEPER	16-12-21	21-12-21	P	A	A	A	A	A	A	NA	A	A	A	56	110	80	37	12	15	16740	14	156	27	0.9	137	4.3	190	140	36	74	P	P	STEMI-ANTERO LATERAL WALL	Regular	0	HYPOKINESIA OF ANTERO LATERAL	45%	>40	A	A	A		I
64	SHABANA	35	≤50	8	F	HOUSEWIFE	17-12-21	24-12-21	P	P	A	A	A	A	A	NA	A	A	A	100	110	70	37	16	13	8600	12	117	21	0.8	136	3.8	174	150	28	94	P	P	LBBB	Regular	180	HYPOKINESIA OF ANTERO SEPTAL	45%	>40	A	A	A	LBBB	E
65	GANGABAI MASHYAL	65	>50	6	F	HOUSEWIFE	20-12-21	25-12-21	P	A	A	A	A	A	A	NA	A	A	A	76	132	70	37	16	12	12000	12	90	22	0.7	134	3.6	150	100	32	78	P	P	NSTEMI	Regular	90	HYPOKINESIA OF ANTERO LATERAL	55%	>40	A	A	A		I
66	RAFIQ MHD MUTTAWALI	60	>50	7	M	SHOP KEEPER	20-12-21	26-12-21	P	P	A	A	A	A	A	NA	P	A	A	80	120	70	37	16	14	11000	15	112	25	0.9	137	3.4	160	200	28	70	P	P	STEMI-ANTERO SEPTAL WALL	Regular	90	HYPOKINESIA OF INFERIOR WALL	35%	≤40	A	A	A		I
67	SHRIRANGA KADAM	60	>50	8	M	FARMER	20-12-21	27-12-21	P	A	A	A	P	A	A	NA	P	A	P	70	120	80	37	17	15	7540	23	114	23	1	138	4.1	170	200	30	70	P	P	STEMI-ANTERO SEPTAL WALL	Regular	90	HYPOKINESIA OF ANTERO SEPTAL	35%	≤40	A	P	A		I
68	BASAVARAJ SAHEBGOUDA	60	>50	7	M	FARMER	24-12-21	30-12-21	P	A	A	A	A	A	A	NA	A	A	P	76	140	90	37	18	14	10170	25	123	14	0.6	134	4.2	160	120	38	90	P	P	STEMI-ANTERO SEPTAL WALL	Regular	30	HYPOKINESIA OF ANTERIOR WALL	45%	>40	A	A	A		I
69	SONABHAI SHIVAPPA	70	>50	6	F	HOUSEWIFE	25-12-21	30-12-21	P	A	P	P	A	A	P	NA	A	A	A	84	190	100	37	20	9.7	5970	15	110	20	0.8	138	3.7	164	140	34	70	P	P	STEMI-INFERIOR WALL	Regular	30	HYPOKINESIA OF INFERIOR WALL	45%	>40	A	A	A		I
70	GUNDAWWA HEBBAL	65	>50	6	F	HOUSEWIFE	26-12-21	31-12-21	P	A	A	A	A	A	A	NA	A	A	A	80	150	90	37	16	12	6700	15	98	23	0.5	141	2.7	135	98	37	78	P	P	STEMI-ANTERO SEPTAL WALL	Regular	0	HYPOKINESIA OF ANTERIOR WALL	55%	>40	A	A	A		I
71	NAGARAJ KASE	40	≤50	6	M	EMPLOYEE	10-1-22	15-1-22	P	P	A	A	A	A	P	NA	A	A	A	82	140	90	37.2	18	14	6530	15	123	32	0.8	143	4.3	257	267	53	151	P	P	STEMI-ANTERO SEPTAL WALL	Regular	0	HYPOKINESIA OF ANTERO LATERAL	45%	>40	A	A	A		I
72	ABDUL REHMAN	75	>50	5	M	BUSINESSMAN	11-1-22	15-1-22	P	A	A	A	A	A	A	NA	A	A	P	60	120	60	37.2	18	12	9570	24	114	34	0.9	140	4.9	170	150	40	90	P	P	STEMI-ANTERO SEPTAL WALL	Regular	60	HYPOKINESIA OF ANTERIOR WALL	50%	>40	A	A	A		I
73	RUKUMABAI RAMCHANDRA	50	≤50	7	F	HOUSEWIFE	29-1-22	4-2-22	P	A	P	A	A	A	P	NA	A	A	A	82	150	80	37	20	13	9700	15	105	32	0.5	136	4.3	150	180	26	90	P	P	STEMI-INFERIOR WALL	Regular	0	HYPOKINESIA OF INFERIOR WALL	50%	>40	A	A	A		I
74	SIDDALINGAPP A ADALLI	61	>50	7	M	FARMER	31-1-22	5-2-22	P	A	P	A	P	P	A	NA	P	P	P	64	120	70	37	18	15	11000	26	209	35	0.9	136	3.5	162	94	38	96	P	P	STEMI-INFERIOR WALL	Regular	30	HYPOKINESIA OF INFERIOR WALL	45%	>40	A	A	A		I
75	AHMMADASHA MAKANDAR	71	>50	10	M	BUSINESSMAN	1-2-22	10-2-22	P	A	A	A	A	A	A	NA	A	A	P	82	100	60	37	22	14	7830	25	102	22	0.8	140	3.8	112	74	30	50	P	P	RBBB	Irregular	90	RMWA	35%	≤40	P	P	A	rbbb	E
76	VIRUPAKSHAYYA	51	>50	9	M	EMPLOYEE	3-2-22	11-2-22	P	A	A	A	A	A	A	NA	A	A	A	80	110	70	37	18	12	9400	23	109	21	1	135	3.7	190	147	33	28	P	P	NSTEMI	Regular	120	HYPOKINESIA OF INFERIOR WALL	40%	≤40	P	P	A		I
77	CHANDRAWWA SIDARAYYA	68	>50	7	F	HOUSEWIFE	4-2-22	10-2-22	P	A	A	A	P	A	A	NA	A	A	A	68	110	70	37	19	10	10480	25	110	28	0.8	136	5	100	90	40	60	P	P	STEMI-INFERIOR WALL	Regular	120	HYPOKINESIA OF INFERIOR WALL	40%	≤40	A	A	A		I

SL NO	Patient Name	AGE	age	hospital stay in days	SEX	OCCUPATION	D.O.A	D.O.D	CHESTPAIN	DYSPNEA	ABDOMINAL PAIN	PALPITATION	SYNCOPE	DIABETES	HTN	FAMILY HISTORY	SMOKING	ALCOHOL	TOBACCO CHEWING	PR	SYSTOLIC	DIASTOLIC	TEMPERATUR	RR	HEMOGLOBIN	TOTAL COUNT	ESR	RBS	BLOOD UREA	Sr.CREATININE	Sr.SODIUM	Sr.POTASSIUM	TOTAL CHOLESTROL	TRIGLYCERIDES	HDL	LDL	TROPONIN I	CPK MB	ECG FINDINGS	RHYTHEM	FQRST	2D ECHO	LVEF	LVEF	HEART FAILURE	PULMONARY EDEMA	CARDIOGENIC SHOCK	OTHERS	INCLUSION/ EXCLUSION
78	INDIRA GUDDANAVAR	53	>50	7	F	HOUSEWIFE	7-2-22	13-2-22	P	A	A	A	A	A	A	NA	A	A	A	90	120	70	37	16	15	17120	15	118	15	0.7	138	3	158	140	36	80	P	P	NSTEMI	Regula	30	HYPOKINESIA OF ANTERO SEPTAL	40%	≤40	A	A	A		
79	HAJILAL MUJAWAR	80	>50	8	M	SHOP KEEPER	7-2-22	14-2-22	P	A	A	A	A	A	A	NA	A	A	P	70	130	70	37	16	12	9660	21	109	36	0.8	136	5.1	194	200	26	88	P	P	NSTEMI	Regula	0	HYPOKINESIA OF INFERIOR WALL	40%	≤40	A	A	A		
80	PHAKIRAPPA MSHAPPA	56	>50	12	M	FARMER	7-2-22	18-2-22	P	A	A	A	A	A	A	NA	P	A	P	70	110	70	37	18	14	9600	11	109	32	0.7	132	4.2	156	200	34	92	P	P	NSTEMI	Regula	120	HYPOKINESIA OF INFERIOR WALL	40%	≤40	P	P	A		
81	SIDDAWWA	70	>50	6	F	HOUSEWIFE	7-2-22	12-2-22	P	A	A	A	A	A	A	NA	A	A	A	60	120	70	37	22	10	12000	36	80	36	0.8	145	3.5	97	65	35	49	P	P	STEMI-INFERIOR WALL	Regula	120	HYPOKINESIA OF INFERIOR WALL	50%	>40	A	A	A		
82	VIKAS	45	≤50	7	M	CLERK	8-2-22	14-2-22	P	A	A	A	A	A	A	NA	A	A	A	102	140	90	37.5	18	15	13240	5	100	46	1.5	141	4.5	128	84	36	60	P	P	STEMI-INFERIOR WALL	Regula	60	HYPOKINESIA OF INFERIOR WALL	45%	>40	A	A	A		
83	JUMAPPA SIDDAPPA	65	>50	11	M	FARMER	9-2-22	19-2-22	P	P	A	A	A	A	P	NA	P	A	P	88	140	90	37	22	12	14000	23	110	30	0.8	130	4	150	140	34	74	P	P	NSTEMI	Regula	180	HYPOKINESIA OF INFERIOR WALL	30%	≤40	P	P	A		
84	MUTTAMMA MYAKERI	57	>50	5	F	HOUSEWIFE	12-2-22	16-2-22	P	P	A	A	A	P	P	NA	A	A	P	110	180	110	37.5	22	11	18600	75	401	20	0.7	139	4	164	112	53	88	P	P	NSTEMI	Regula	120	HYPOKINESIA OF INFERIOR WALL	60%	>40	A	A	A		
85	LALITHA	60	>50	6	F	HOUSEWIFE	4-3-22	9-3-22	P	A	A	A	A	A	P	NA	A	A	A	100	150	90	37.2	20	11	12600	40	101	30	1	134	4.1	130	90	40	74	P	P	STEMI-ANTERO LATERAL WALL	Regula	30	HYPOKINESIA OF ANTERO SEPTAL	35%	≤40	A	A	A		
86	DYANAVVA	60	>50	7	F	FARMER	8-3-22	14-3-22	P	A	P	A	A	A	P	NA	A	A	P	76	110	70	37.5	20	12	10360	40	138	21	0.6	140	5	110	70	40	64	P	P	NSTEMI	Regula	30	HYPOKINESIA OF INFERIOR WALL	35%	≤40	A	A	A		
87	LATA	50	≤50	12	F	HOUSEWIFE	12-3-22	23-3-22	P	A	P	A	A	A	A	NA	A	A	A	106	90	60	37	22	10	8500	5	140	26	0.4	135	3.8	100	74	40	50	P	P	STEMI-INFERIOR WALL	Regula	120	HYPOKINESIA OF ANTERO LATERAL	30%	≤40	P	P	P		
88	IRANGOUDA	60	>50	12	M	FARMER	4-4-22	15-4-22	P	A	A	A	A	A	A	NA	A	A	A	90	100	70	37.5	18	16	11300	10	112	25	1	140	3.5	120	84	34	60	P	P	STEMI-ANTERO LATERAL WALL	Regula	180	HYPOKINESIA OF ANTERIOR WALL	35%	≤40	P	P	P		
89	SANGAPPA	50	≤50	7	M	FARMER	6-4-22	12-4-22	P	A	A	A	A	A	A	NA	A	A	P	76	110	70	37.5	16	13	10400	5	150	25	0.9	137	4.2	100	150	50	70	P	P	STEMI-INFERIOR WALL	Regula	60	HYPOKINESIA OF INFERIOR WALL	50%	>40	A	A	A		
90	NEELAPPA	65	>50	6	M	BUSINESSMAN	7-4-22	12-4-22	P	A	A	A	A	A	P	NA	A	A	P	80	160	90	37.5	19	11	7950	5	112	36	0.7	140	4.2	224	425	38	101	P	P	NSTEMI	Regula	60	HYPOKINESIA OF INFERIOR WALL	35%	≤40	A	A	A		
91	KALAVATHI	57	>50	7	F	HOUSEWIFE	10-4-22	16-4-22	P	A	A	A	A	A	P	NA	A	A	A	98	110	80	37.4	20	13	9060	5	110	30	0.6	134	4.8	110	90	36	52	P	P	STEMI-ANTERO SEPTAL WALL	Regula	120	HYPOKINESIA OF INFERIOR WALL	45%	>40	A	A	A		
92	IRANNA	59	>50	12	M	FARMER	14-4-22	25-4-22	P	A	P	A	P	A	A	NA	A	P	A	106	100	60	38.2	20	13	6160	5	112	40	0.7	142	3.6	175	75	70	90	P	P	STEMI-ANTERO SEPTAL WALL	Regula	60	HYPOKINESIA OF ANTERO SEPTAL	45%	>40	P	P	P		
93	PARVATHI	60	>50	6	F	HOUSEWIFE	22-4-22	27-4-22	P	A	A	A	A	A	A	NA	A	A	A	80	140	90	37.5	18	10	10650	50	115	28	0.6	132	4.6	165	112	53	89	P	P	NSTEMI	Regula	60	HYPOKINESIA OF INFERIOR WALL	50%	>40	A	A	A		
94	SAHNKAR TOLL	50	≤50	5	M	CLERK	24-4-22	29-4-22	P	P	A	A	A	A	A	NA	P	P	A	90	140	90	37.5	18	16	13110	10	98	36	0.8	136	4.8	146	103	38	88	P	P	STEMI-ANTERO LATERAL WALL	Regula	0	HYPOKINESIA OF ANTERO SEPTAL	60%	>40	A	A	A		
95	MUDERYAPPA	60	>50	11	M	FARMER	1-5-22	11-5-22	P	A	A	A	A	A	A	NA	P	A	P	140	90	60	37.6	20	11	16600	5	140	40	0.6	132	4.6	117	96	53	45	P	P	STEMI-INFERIOR WALL	Regula	60	HYPOKINESIA OF INFERIOR WALL	30%	≤40	P	P	P		
96	BASALINGAPPA	46	≤50	7	M	SHOP KEEPER	4-5-22	10-5-22	P	A	A	A	A	A	P	NA	A	P	P	80	150	90	37.5	16	15	9720	10	148	32	0.8	136	4.2	190	240	38	101	P	P	STEMI-ANTERO LATERAL WALL	Regula	0	HYPOKINESIA OF ANTERO SEPTAL	45%	>40	A	A	A		
97	BASAVARAJ SHEVATI	63	>50	6	M	FARMER	6-5-22	11-5-22	P	A	A	A	A	A	P	NA	A	A	P	70	130	80	37	17	15	14300	11	110	27	1.1	138	4.4	140	150	40	68	P	P	NSTEMI	Regula	30	HYPOKINESIA OF INFERIOR WALL	50%	>40	A	A	A		
98	SUSHILBHAI BIRADAR	55	>50	8	F	HOUSEWIFE	8-6-22	15-6-22	P	A	A	A	A	A	A	NA	A	A	A	100	100	60	37	18	6.3	17000	25	130	27	0.7	137	4.3	166	333	28	71	P	P	STEMI-INFERIOR WALL	Regula	120	HYPOKINESIA OF INFERIOR WALL	40%	≤40	A	A	P		
99	BHIMASHI MURAL	58	>50	6	M	FARMER	9-6-22	14-6-22	P	A	A	A	A	A	A	NA	P	A	P	84	120	70	36.5	15	15	15400	20	89	31	0.5	131	5.1	130	200	27	61	P	P	STEMI-INFERIOR WALL	Regula	30	HYPOKINESIA OF INFERIOR WALL	45%	>40	A	A	A		
100	CHANDSAB JUMANAL	60	>50	10	M	BUSINESSMAN	18-6-22	27-6-22	P	A	A	A	A	A	A	NA	A	P	P	88	130	80	37	16	13	12600	21	112	36	0.9	136	4.4	148	170	28	70	P	P	NSTEMI	Regula	0	HYPOKINESIA OF ANTERIOR WALL	50%	>40	A	A	A		
101	CHANNAYYA	50	≤50	8	M	FARMER	23-6-22	30-6-22	P	P	A	A	A	A	A	NA	A	A	A	92	170	110	37	20	15	21290	25	114	39	1.1	136	4.1	200	140	36	94	P	P	STEMI-INFERIOR WALL	Regula	0	HYPOKINESIA OF ANTERIOR WALL	30%	≤40	A	A	A		
102	NINGAPPA YALLAPPA	50	≤50	8	M	FARMER	23-6-22	29-6-22	P	A	A	A	A	P	A	NA	P	A	P	90	120	80	37	18	15	10000	25	262	39	1.1	136	4.1	180	200	26	70	P	P	STEMI-ANTERO SEPTAL WALL	Regula	60	HYPOKINESIA OF ANTERO LATERAL	40%	≤40	A	A	A		