FRAGMENTED QRS COMPLEX AS A PREDICTOR OF IN-HOSPITAL OUTCOME OF THE MAJOR ADVERSE CARDIAC EVENTS IN PATIENTS WITH ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION

By

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Dissertation submitted to BLDE (Deemed to be University), Vijayapura.

In partial fulfillment of the requirements for the award of the degree of

DOCTOR OF MEDICINE

IN

GENERAL MEDICINE

Under the guidance of

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Each waveform is a sign of life and has vital information in itself. And a wise one will analyze each deflection and a fragment of it to rewrite the tale of life. This is my first baby step in understanding the human heart and its electrical activity to aid the prognosis of ST-segment elevation myocardial infarction.

On completion of this contribution of scientific document, it gives me deep pleasure to acknowledge the guidance provided by my distinguished mentors.

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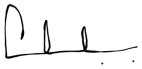

Dr. SIDDARTHKUMAR CHAWATH

Each wave formed on electrocardiograph, has a story to be told, a story to be read, a story to be understood; and one who learns to analyze each fragment of this story, has the potential to change lives.

Dr. SIDDARTHKUMAR CHAWATH

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LIST OF ABBREVIATIONS USED

ACS	:	ACUTE CORONARY SYNDROME
CS	:	CARDIOGENIC SHOCK
ECG	:	ELECTROCARDIOGRAPHY
fQRS	:	FRAGMENTED QRS COMPLEX
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:

The ST- segment elevation myocardial infarction is a critical and morbid disease that risks the life of the patient. STEMI patients always have a higher incidence of in-hospital mortality and in-hospital major adverse cardiac events. And as we know electrocardiography is a routine part of evaluation in any cardiac patients.

AIMS AND OBJECTIVE

This study is done to analyze the fragmented QRS complex as a predictor of in-hospital outcome of the major adverse cardiac events in patients with ST-segment elevation myocardial infarction.

MATERIALS AND METHODS:

A Prospective observational study was carried out on patients admitted in ICCU of BLDE (DU), Shri B.M. Patil Medical College Hospital and Research Centre, with a diagnosis of ST-segment elevation myocardial infarction. Clinical, electrocardiographic and laboratory profile and in-hospital outcome of patients with STEMI were assessed as a part of work up. Electrocardiography was done within 24 hours of admission. Patients were grouped according to the presence of fQRS as Group A (n=86), and Group B (n=58) with absence of fQRS. The patients during in-hospital stay, were observed for development of major adverse cardiac events like heart failure, pulmonary edema, cardiogenic shock, arrhythmias and death.

RESULTS: Total of 152 patients were studied out of which 8 were excluded based on exclusion criteria, while rest 144 patients were classified into group A and group B. In group A most common age group was 51-60 years with male predominance. Risk factors like diabetes mellitus, family history of cardiac problems and smoking had strong association

with group A. In group A, major adverse cardiac events like heart failure (55.8% vs 12.1%, p<0.01), pulmonary edema (46.5% vs. 6.9%, p<0.01), cardiogenic shock (41.9% vs 12.1%, p<0.01), death (27.9% vs. 3.4%, p<0.01), a higher incidence of reduced ejection fraction was seen when compared with Group B. The most common major adverse cardiac event was heart failure followed by pulmonary edema, cardiogenic shock and death.

CONCLUSION: Fragmented QRS complex was found to be an important predictor of in-hospital major adverse cardiac events like heart failure, pulmonary edema, cardiogenic shock, arrhythmias and death. Hence fQRS on a 12 lead ECG is a cheap, reliable and non-invasive prognostic marker of major adverse cardiac events and in-hospital mortality in patients with ST-segment elevation myocardial infarction.

KEYWORDS: Fragmented QRS complex, Major adverse cardiac events, ST-Segment elevation myocardial infarction.

INTRODUCTION

I. INTRODUCTION

Acute coronary syndrome (ACS) causes more deaths and disability and attracts greater economic burden than any other illness in the modern world. ACS is affected by wide spectrum of risk factors which are associated with emergence and progression of this most common, serious, chronic, life-threatening disease[1].

India, the world's second largest populated country, is a land with utmost diversity in terms of geography, race, culture, literacy, infrastructure and economy. All these factors cause serious challenge in the management of ACS. As per World Health Organization data, the ACS prevalence continues to rise in India with rapid epidemiological transition[2]. ACS has already surpassed communicable disease as the major cause of mortality in India. So much so that ST- segment elevation myocardial infarction (STEMI) alone incurs a burden of nearly three million cases per 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 ACS, 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.

Electrocardiography, introduced in 1902 by Dutch physician, William Einthoven provided information about the electrophysiology of the heart[8]. Electrocardiography inaugurated a new era in which various machine and technical procedures gradually replaced the physician's unaided senses and the stethoscope as the primary tool of cardiac diagnosis. Today electrocardiography is an essential part of initial evaluation for the patients presenting with cardiac complaints, specifically it plays an important role as a non-invasive, cost-effective tool to evaluate ACS and arrhythmias.

In the past, many literatures has shown that regional myocardial lesion is associated with changes in QRS complex configuration that can cause terminal conduction delay or fragmented QRS (fQRS) complexes on 12 lead ECG[9]. The fragmented QRS complex includes various RSR patterns with or without the Q wave and was defined by the presence of an additional R wave (R' prime), or notching in nadir of the S wave, notching of R wave, or the presence of more than one R prime (fragmentation) in two contiguous leads corresponding to major coronary artery territory[10]. Slow and non-homogenous conduction through ischemic or infarcted myocardium and conduction system has been postulated to be the mechanism of fQRS[11].

The presence of fQRS in electrocardiogram has been used to predict myocardial lesions, poor outcomes and ventricular arrhythmias. Hence fragmented QRS complexes can be used as a marker of major adverse cardiac events like heart failure, sustained ventricular tachycardia or ventricular fibrillation, early post-infarction angina, or mechanical complications and cardiac death as a consequence of STEMI[9].

AIMS
AND OBJECTIVES

II. AIMS AND OBJECTIVES

To study the fragmented QRS complex as a predictor of in-hospital outcome of the major adverse cardiac events in patients with ST-segment elevation myocardial infarction.

REVIEW OF LITERATURE

III. REVIEW OF LITERATURE

Acute myocardial infarction is one of the major risk factors causing morbidity and mortality in developing countries. Electrocardiographic changes are closely related to diagnosis and prognosis of acute myocardial infarction.

Electrocardiogram is an invaluable and easily available bedside non-invasive too l in assessing acute myocardial infarction. Fragmented QRS complex on 12 lead ECG has been shown to be valuable predictor of cardiac-linked events.

Patients can be followed up during their in-hospital course for the occurrence of adverse cardiac events namely: death, heart failure, sustained ventricular tachycardia or ventricular fibrillation, early post infarction angina, or mechanical complication.

In 1966, Langner, P. H., & Lauer, J. A. discussed about the relative significance of high-frequency and low-frequency notching in the electrocardiogram and stated that low-frequency notching was more prevalent in persons with coronary heart disease than in normal control subjects, it was less prevalent and less informative than high frequency notching in abnormal subjects[12].

In 2008, Mithilesh Kumar Das et al studied Fragmented Wide QRS on a 12-lead ECG a sign of myocardial scar and poor prognosis and presented Fragmented Wide QRS as an independent predictor of coronary arterydisease[13].

In 2012, Mustafa Cetin et al studied absence and resolution of fragmented QRS predict reversible myocardial ischemia with higher probability of ST Segment resolution in patients with ST-Segment Elevation Myocardial infarction and concluded that Successful myocardial reperfusion by primary percutaneous coronary intervention caused the reduction in number of fQRS and QRS time with higher ST resolution[14].

In 2015, Zuber Suleman et al. studied usefulness of fragmented QRS in predicting the successful reperfusion by using noninvasive criteria of reperfusion in STEMI after thrombolytic therapy[15].

In 2017, Di Liang et al. studied the difference on features of fragmented QRS complex and influence on mortality in patients with acute coronary syndrome and concluded that presence of fQRS complexes within 48 hours of presentation may be used to differentiate NSTEMI patients from UA patients. fQRS may also be used as a survival predictor for patients with AMI[11].

1. ACUTE MYOCARDIAL INFARCTION

1.1. INTRODUCTION

Acute myocardial infarction can be broadly termed as cardiomyocyte death secondary to prolonged ischemia resulting from a sudden imbalance between oxygen supply and demand. Though there are multiple risk factors that play a pivotal role in progression of disease but ultimate pathogenesis leads to atheroma formation and vascular occlusion[5].

1.2. DEFINITION

According to Fourth Universal Definition of Myocardial Infarction (2018), the term acute myocardial infarction is defined as acute myocardial injury with clinical evidence of acute myocardial ischemia and with detection of a rise and/or fall of cTn values with at least one value above the 99th percentile upper reference limit and at least one of the following:

• Symptoms of myocardial ischemia;

• New ischemic ECG changes;

• Development of pathological Q waves;

• Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality in a pattern consistent with an ischemic etiology;

• Identification of a coronary thrombus by angiography or autopsy (not for type 2 or 3 MIs).

Postmortem demonstration of acute atherothrombosis in the artery supplying the infarcted myocardium meets criteria for type 1 MI. Evidence of an imbalance between myocardial oxygen supply and demand unrelated to acute atherothrombosis meets criteria for type 2 MI. Cardiac death in patients with symptoms suggestive of myocardial ischemia

and presumed new ischemic ECG changes before cTn values become available or abnormal meets criteria for type 3 MI[4].

1.3. CLASSIFICATION

A. ANATOMICAL CLASSIFICATION:

- Transmural infarction: involvement of all the three layers. Endocardium, myocardium and epicardium.
- Subendocardial infarction: involvement of small area in the subendocardial wall of the left ventricle, ventricular septum or papillary muscles.

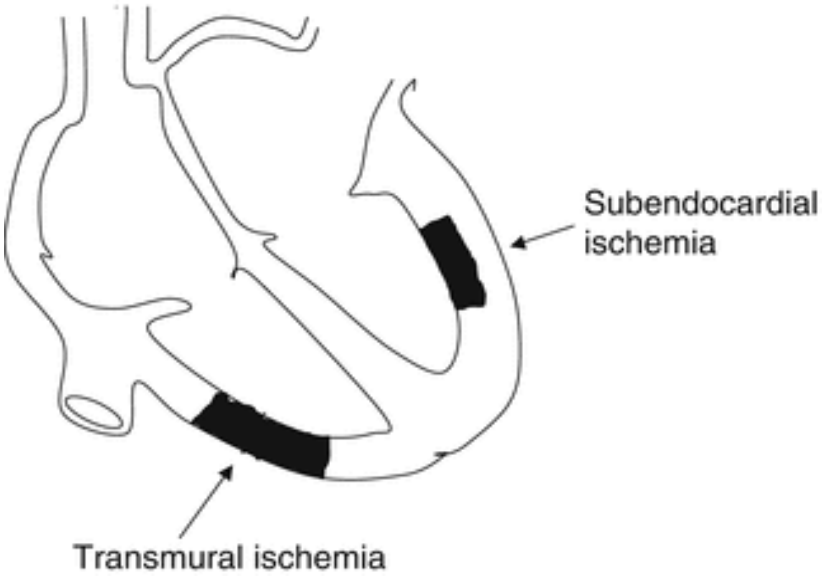


Figure 1: Anatomical classification of myocardial infarction.

B. CLINICAL CLASSIFICATION[4]:

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 4a: Percutaneous coronary intervention-related death

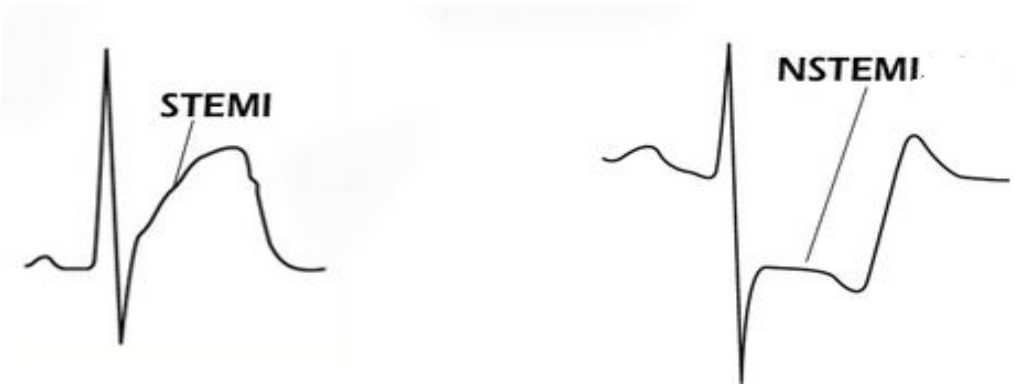
TYPE 4b: Stent thrombosis

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

C. ELECTROCARDIOGRAPHIC CLASSIFICATION:

i) ST-segment elevation myocardial infarction.

ii) Non-ST-segment elevation myocardial infarction.



1.4. ECG CRITERIA FOR DIAGNOSIS OF MYOCARDIAL INFARCTION[4]:

A. IN THE ABSENCE OF LEFT VENTRICULAR HYPERTROPHY AND BUNDLE BRANCH BLOCK:

i) ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION:

New ST-elevation at the J-point in 2 contiguous leads with the cut-point: ≥1 mm in all leads other than leads V2–V3 where the following cut-points apply: ≥2 mm in men ≥40 years; ≥2.5 mm in men <40 years, or ≥1.5 mm in women regardless of age.

When the magnitudes of J-point elevation in leads V2 and V3 are registered from a prior electrocardiogram, new J-point elevation ≥1 mm (as compared with the earlier electrocardiogram) should be considered an ischemic response.

ii) NON-ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION:

New horizontal or down sloping ST-depression ≥0.5 mm in 2 contiguous leads and/or T inversion >1 mm in 2 contiguous leads with prominent R wave or R/S ratio >1.

iii) CHANGES ASSOCIATED WITH PRIOR MYOCARDIAL INFARCTION:

Any Q wave in leads V2–V3 >0.02 s or QS complex in leads V2–V3. Q wave ≥0.03 s and ≥1 mm deep or QS complex in leads I, II, aVL, aVF or V4–V6 in any 2 leads of a contiguous lead grouping (I, aVL; V1–V6; II, III, aVF). R wave >0.04 s in V1–V2 and R/S >1 with a concordant positive T wave in absence of conduction defect[4].

B. IN THE PRESENCE OF LEFT BUNDLE BRANCH BLOCK (LBBB) OR VENTRICULAR PACED RHYTHM, DIAGNOSIS IS BASED ON MODIFIED SGARBOSSA CRITERIA:

- ≥ 1 lead with ≥1 mm of concordant ST elevation
- ≥ 1 lead of V1-V3 with ≥ 1 mm of concordant ST depression
- ≥ 1 lead anywhere with ≥ 1 mm ST elevation and proportionally excessive discordant ST elevation, as defined by ≥ 25% of the depth of the preceding S-wave[16].

1.5. LOCALIZATION OF MYOCARDIAL INFARCTION ON ELECTROCARDIOGRAPHY:

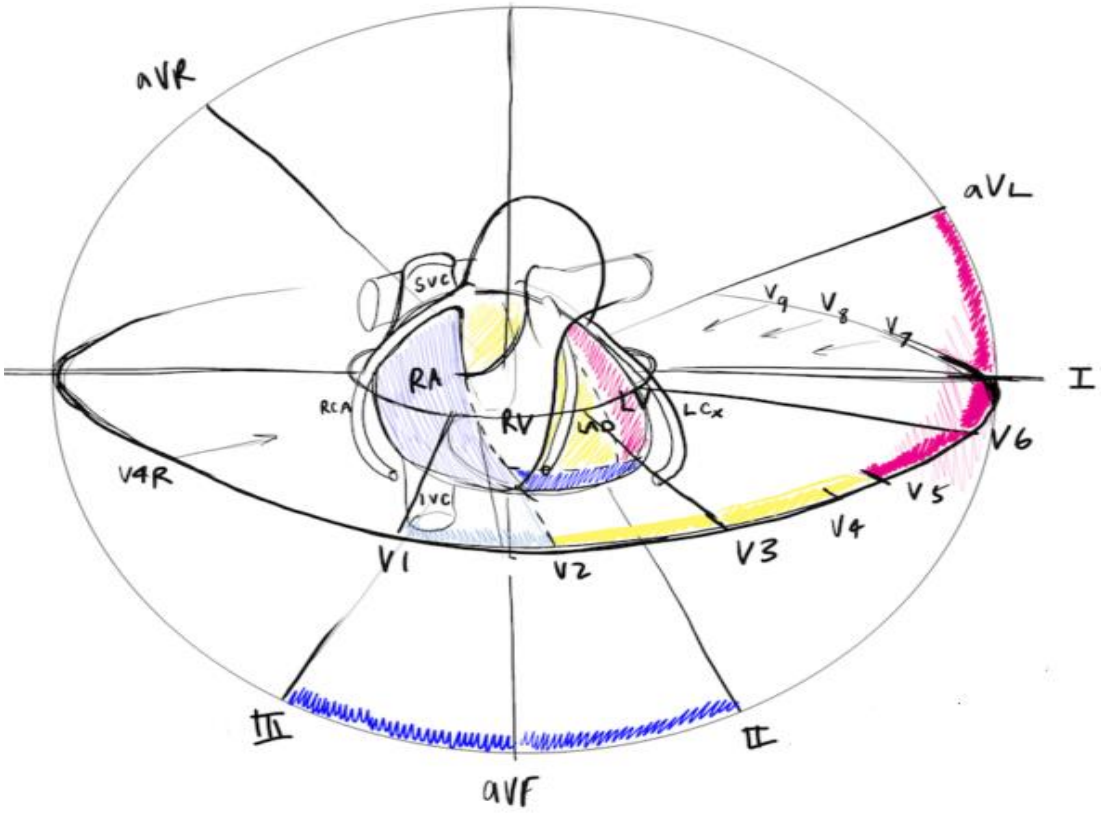


Figure 2: Localization of myocardial infarction on electrocardiograph.

SITE	ARTERY	ECG LEADS
Anterior	LAD	V3, V4
Anterolateral	LAD	V5, V6
Anteroseptal	LAD	V1, V2
Inferior	RCA	II, III, aVF
Posterior	RCA, LCX	V7-V9

Table1: Electrocardiographic localization of myocardial infarction and coronary artery territories.

1.6. PATHOGENESIS OF ACUTE MYOCARDIAL INFARCTION

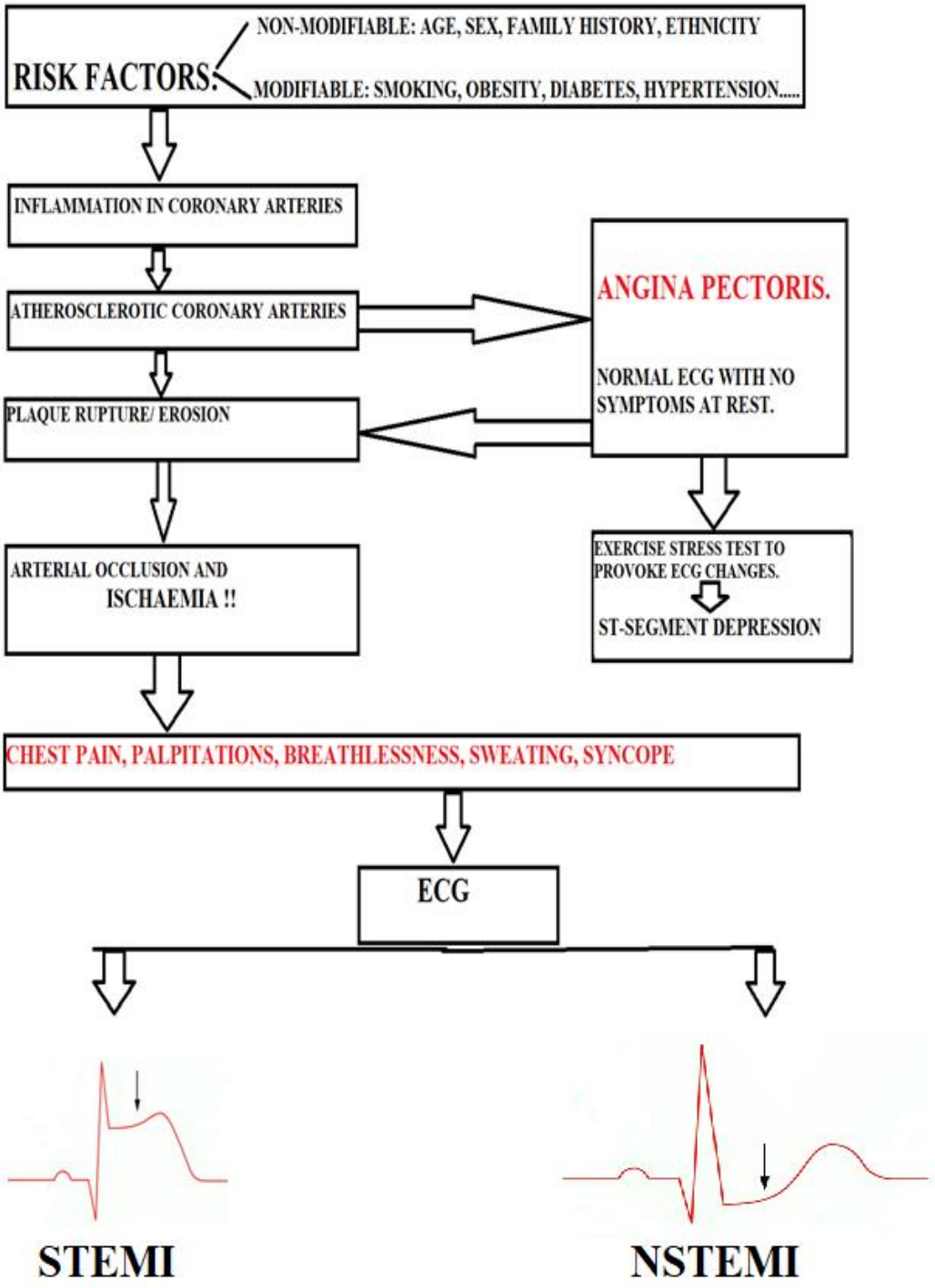


Figure 3: Pathogenesis of myocardial infarction

Acute coronary syndrome occurs due to sudden reduction of coronary arterial blood flow. This reduction is because of atherosclerosis with addition of thrombus over it, with or without associated vasoconstriction. The complaints with which patient is admitted to hospital and outcome of the patients will depend on the location of the coronary occlusion and the duration and severity of myocardial ischemia. In acute myocardial infarction patients with elevation of ST segment, thrombosis usually is due to persistent or occlusive. The precipitating factor for most of the fatal coronary thrombi is sudden rupture of the vulnerable plaque. Around 3/4 of all infarct related thrombi will grow over plaques which leads to mild-to-moderate stenosis before the infarction and after the thrombolysis[4].

Thrombosis over plaques occurs because of two somewhat different processes. One is caused by an extension of the process of endothelial denudation so that large areas of the surface of the subendothelial connective tissue of the plaque are exposed. Thrombus forms which is adherent to the plaque surface. This process has become known as endothelial erosion. Observational studies have linked endothelial cell loss to the proximity of macrophages. These macrophages are highly activated and cause endothelial cell death by apoptosis, and also by the production of proteases which cut loose the endothelial cells from their adhesion to the vessel wall.

The second mechanism for thrombus formation is plaque disruption (synonyms rupture, fissuring). Here the plaque cap tears to expose the lipid core to blood in the arterial lumen. The core area is highly thrombogenic, containing tissue factor, fragments of collagen, and crystalline surfaces to accelerate coagulation. Thrombus forms initially in the plaque itself which is expanded and distorted from within; thrombus may then extend into the arterial lumen.

When compared to mild stenosis of coronary artery, severe stenosis has more chances of having plaque events. Myocardial infarction due to total coronary artery obstruction starts

to occur after 15–30 min of massive ischemia. Its progression starts in the sub endocardium and occurs towards sub epicardium in a slow time-dependent manner [3].

Non-ST elevation myocardial infarction is due to a sudden imbalance between myocardial cell demand of oxygen and its supply. It is very commonly due to a reduced myocardial perfusion. It very oftenly occurs due to a non-occlusive thrombus that had developed on a dislodged atherosclerotic plaque, and ultimately leading to nonocclusive or completely obstructive thrombosis of a coronary vessel supplying the myocardial cell[4].

Various events that may lead to an NSTEMI:

- Rupture of the plaque with imposed nonocclusive thrombus or embolic events causing coronary artery obstruction.
- Dynamic obstruction, as seen in coronary vasospasm
- Slow narrowness of lumen
- Inflammatory mechanisms like vasculitis
- Extrinsic factors causing impaired coronary artery perfusion are hypotension, hypoxemia and hypovolemia.

The most common cause is plaque rupture or obstructive atherosclerotic disease. Plaque rupture usually occurs at the weakest and thinnest part, that is shoulder region of the atherosclerotic cap. Ruptured plaques have high numbers of inflammatory cells like T lymphocytes, monocytes, macrophages. It is known that one third of obstructions usually occur at the site of highest stenosis, mostly arise from lesions with less than 50% of stenosis, and in less than 5% of the patients it arises from lesions showing more than 70% of stenosis. The absence of ST elevation is due to the reason that the infarct will not involve the complete thickness of the myocardium[4].

1.7. CLINICAL PRESENTATION:

Presentation of AMI may have many variants. The classic symptom of chest pain is by no means pathognomic and patients with AMI may have atypical presentations like dyspnoea, abdominal or epigastric pain, nausea, vomiting, and syncope.

A. Chest pain:

Chest pain is the most common symptom, described as a sensation of tightness, pressure, or squeezing and is not relieved by rest, position change or analgesic or antacid administration. Pain radiates most often to the left arm, but may also radiate to the lower jaw, neck, right arm, back, and upper abdomen, where it may mimic heartburn. Levine's sign, in which a person localizes the chest pain by clenching their fists over their sternum. In Silent acute myocardial infarction 20-30% subjects don't have chest pain, common in patients with diabetes mellitus, hypertension, and in elderly patients.

B. Shortness of breath (dyspnea):

The damage to the heart limits the output of the left ventricle, causing left ventricular failure and consequent pulmonary edema.

C. Nausea and Vomiting:

Vomiting results as a reflex from severe pain that is vasovagal reflexes initiated from area of ischemia.

D. Diaphoresis:

Diaphoresis is due to stimulation of sympathetic nervous system and release of catecholamines.

E. Palpitations:

Palpitation is associated with change in heart rate and rhythm.

F. Loss of consciousness: Due to inadequate blood flow to the brain and cardiogenic shock.

G. Sudden death: Due to the development of ventricular fibrillation

1.8 COMPLICATIONS OF ACUTE MYOCARDIAL INFARCTION

Most of the deaths in these patients are the direct result of pathophysiologic changes which occur as a result of the AMI. Many more patients suffer from complications of AMI. These patients require prompt and early recognition of these condition and aggressive management in order to prevent unnecessary morbidity and mortality. Complications of AMI can be broadly classified into:

A. Heart failure and Cardiogenic shock.

B. Ischemic Complication

i. Reinfarction

C. Mechanical Complications

i. Left ventricular aneurysm

ii. Myocardial rupture

iii. Rupture of the ventricular septum

iv. Pseudoaneurysm

D. Conduction Abnormalities

Post infarction conduction abnormalities like tachyarrhythmias and bradyarrhythmia's leading to sudden cardiac deaths.

E. Embolic Complications

Stroke

F. Inflammatory complications

Dressler's syndrome and Post myocardial infarction pericarditis: occurs after one week up to several weeks of myocardial infarction, presenting as fever and chest pain.

A. HEART FAILURE:

Heart failure (HF) is a major cause of late morbidity and mortality after myocardial infarction. Myocardial infarction (MI) remains the most common cause of heart failure (HF) worldwide. HF is defined as a clinical syndrome resulting from any structural or functional cardiac disorder that impairs the ability of the ventricle to fill or eject blood. Clinical features of circulatory failure are a low volume pulse, reduced blood pressure, cool and clammy extremities, a third heart sound (S3 gallop), pulmonary rales, reduced urine output, and sweating.

Reduction in cardiac output starts occurring when damage of left ventricular myocardium is 20% to 25%. Cardiogenic shock usually occurs when damaged left ventricular myocardium is 40% or more. The most common clinical findings include Pulmonary congestion and S3 and S4 gallops.

Left heart dysfunction is correlated with both short-term as well as long-term prognosis of the patients. Syndrome of heart failure begin with breathlessness, easy fatiguability, sinus tachycardia, a third heart sound (S3 gallop) and systolic or diastolic murmurs, pulmonary rales which can be identified in the lung bases but then as disease progresses it involve whole lungs. The developed pulmonary edema does not correlate with the auscultatory signs[17].

Killip's classification is one method used to access the severity of heart failure following a myocardial infarction,

Killip class I: No crackles and no third heart sound.

Killip class II: crackles in fewer than 50% of lung fields or a third heart sound .

Killip class III: crackles in over 50% of lung fields.

Killip class IV: cardiogenic shock[18].

PATHO-PHYSIOLOGY:

Heart failure is characterized by the abnormalities of function of skeletal muscle, heart and kidney with activation of the sympathetic nervous system and various neurohormonal changes[17].

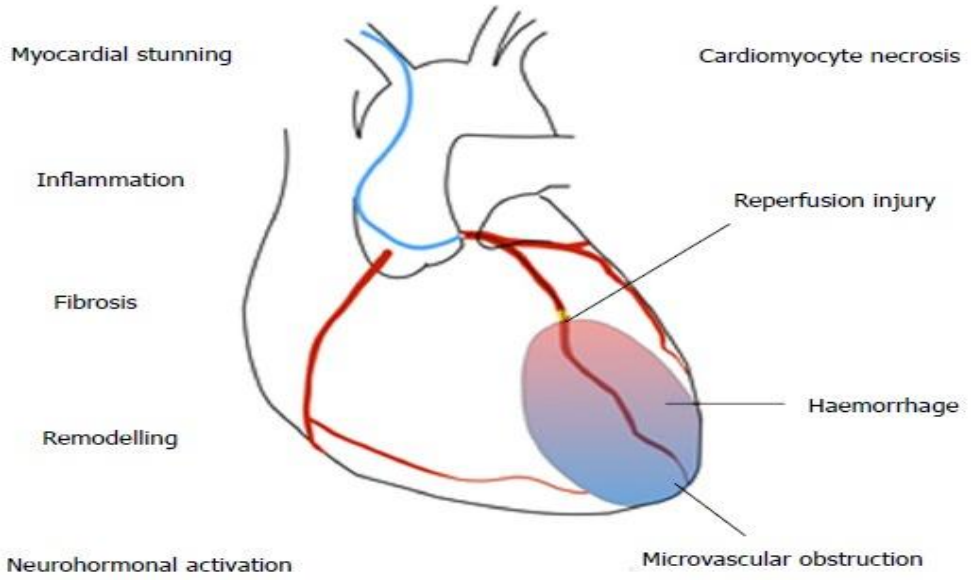


Figure 4: Mechanisms of heart failure after myocardial infarction.

SYSTOLIC DYSFUNCTION OF MYOCARDIUM:

The main abnormality in non-valvular HF is an impaired left ventricular (LV) function which leads to reduced cardiac output. This reduction in cardiac output leads to activation of various compensatory neurohormonal mechanisms. This activation of neurohormonal mechanisms will increase myocardial contractility and reduce coronary perfusion through endothelial dysfunction.

Activation of sympathetic nervous system helps to maintain normal cardiac output by increasing heart rate, myocardial contractile function, peripheral vasoconstriction and also activation of Renin-Angiotensin-Aldosterone system (RAAS). RAAS leads to vasoconstriction and increase volume of the blood with salt and water retention. The Concentration of vasopressin and natriuretic peptides like BNP and NT pro-BNP are also increase. There may be gradual dilatation of the heart or changes in structure of the heart or both[19].

DIASTOLIC DYSFUNCTION:

Diastolic dysfunction refers to a "clinical syndrome of HF with preserved Left Ventricle Ejection Fraction i.e. ejection fraction >40% with no major valvular heart disease". In diastolic HF, LV cavity is stiff due to increased LV mass. During diastole in the early phase myocardium slowly relaxes and in the late diastole there is increased resistance to left ventricular filling and hence, diastolic pressure is increased. The reduced cardiac output is evident as fatigue and raised end diastolic pressure is transferred back via valve less pulmonary veins to pulmonary capillaries which results in exertional breathlessness[19].

B. CARDIOGENIC SHOCK:

Cardiogenic shock (CS) is a "severe form of left ventricular failure which manifests as hypotension where systolic blood pressure is less than 80mm Hg and reduced cardiac index that is, <1.8 L/min/m^2in spite of raised left ventricular filling pressure. The reason being loss of functional myocardial tissue i.e., more than 40% of the left ventricle. Cardiogenic shock has very high mortality rates , more than 70% in spite of immediate medical therapy[20].

C. ARRHYTHMIAS:

Many a times life threatening arrhythmias like ventricular tachycardia, ventricular fibrillation and total AV block can be the first manifestation of ischaemia. Ventricular fibrillation or sustained ventricular tachycardia has been reported in up to 20% of patients.

The risk of death secondary to arrhythmic event in patients survived of acute myocardial infarction is highest in the first six months and remains susceptible for next two years[21].

PATHOPHYSIOLOGY:

Arrhythmias may be due to infarction itself, reperfusion, irritable myocardium, toxic metabolites or even due to metabolism (especially potassium and magnesium imbalance).

2.FRAGMENTED QRS COMPLEX:

2.1. INTRODUCTION

Appearance of additional spikes in the QRS complexes hasgained interest in recent years. These are different from the standard rSR'pattern seen in right bundle branch block (RBBB) and thenotched R waves seen in left bundle branch block (LBBB). Subtle abnormalities within the QRS complex like wide complex or slurring can represent conduction disturbance and myocardial scar. Fragmented QRS complex being one such representation has been a topic of discussion and research since past decade.

2.2. DEFINITION

The fragmented QRS complex includes various RSR' patterns with or without the Q wave and was defined by the presence of an additional R wave (R' prime), or notching in nadir of the R wave or the S wave, or the presence of more than one R prime (fragmentation) in two contiguous leads corresponding to major coronary artery territory[10].

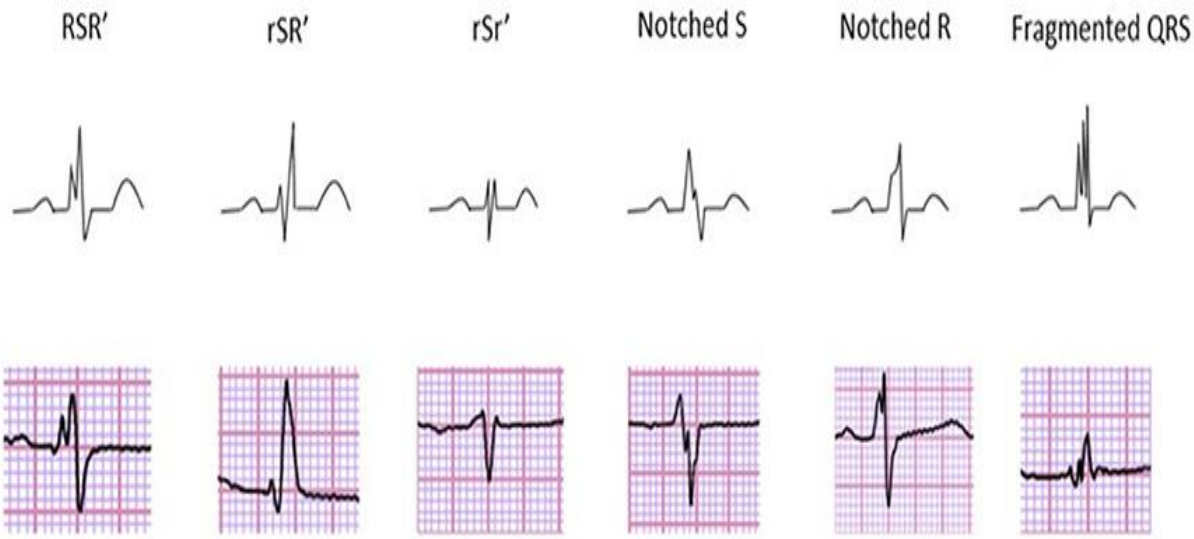


Figure 5: Different morphologies of fragmented QRS complex on 12 lead ECG.

2.3. INTER AND INTRA-OBSERVER VARIABILITY

Inter and intra observer variability of fQRS has been studied by Vandenberk B et al. Hundred ECGs with fQRS were evaluated by two experienced and 3 novel observers. Fleiss and Cohen's Kappawas calculated among subgroups. There was a significant inter-observer variability with a Kappa of 0.651. Experienced observers had a better agreement with a Kappa of 0.823. Inter-observer variability was much higher in paced-rhythm compared with normal rhythm, with Kappa 0.493 vs 0.664 ($p<0.001$). Intra-observer variability had a Kappa between 0.736 and 0.880. So visual assessment variability will depend on experience as well as the underlying rhythm. Hence To overcome this bias automation of detection by computer based algorithms have been attempted[22].

2.4. MECHANISM

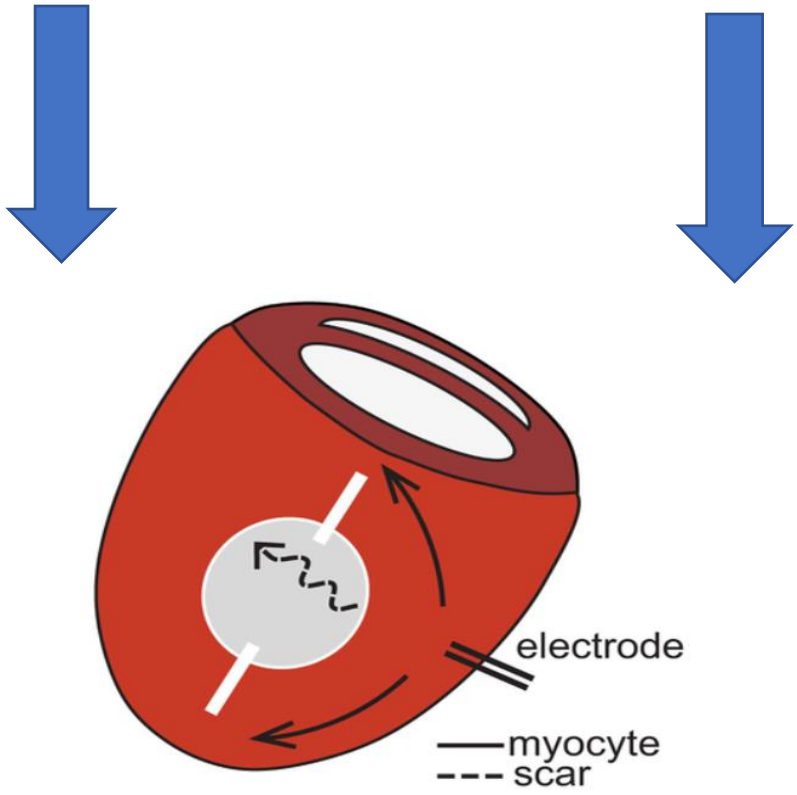
Broadly fQRS is explained as unexpected deviation in the QRS morphology, the exact cause of QRS complex fractionation on surface ECG is not yet completely understood. Myocardial scar or fibrosis or ischaemia leads to formation of electrical window, and does not let conduction of electrical current through it. Hence there is non-homogenous activation and heterogeneous depolarization of ventricular myocardium followed by zig-zag conduction of electrical activity around the scarred myocardium resulting in multiple spikes with in the QRS complex[9].

Myocardial single photon emission tomography can identify regional perfusion abnormalities from a scar by a prior myocardial infarction. Studies in which the diagnostic values of Q wave and fQRS for a myocardial scar detected by single photon emission

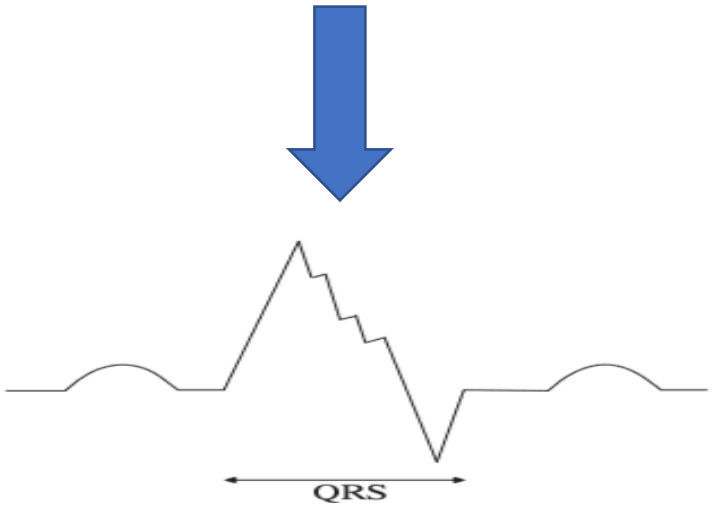
tomography were compared showed that fQRS was associated with significantly greater perfusion and functional abnormalities than was the Q wave[9].

Therefore pathologies that can cause myocardial scar, fibrosis, ischaemia and many cahnnel dysfunction that can cause significant interventricular systolic desynchrony can lead to fragmentation of QRS comlex[23].

MYOCARDIAL FIBROSIS/ SCARS **MYOCARDIAL ISCHAEMIA**



NON-HOMOGENOUSACTIVATION AND ZIG-ZAG CONDUCTION OF ELECTRICAL ACTIVITY AROUND THE SCARRED MYOCARDIUM



FRAGMENTED QRS COMPLEX

Figure 6: Mechanism of fragmented QRS complex formation.

2.5. SIGNIFICANCE OF FQRS IN VARIOUS CARDIAC AND SYSTEMIC CONDITIONS

Over the past few years, literature on fQRS has evolved, with studies in a wide variety of cardiac conditions ranging from acute coronary syndrome, cardiomyopathies, valvular heart disease, aortic dissection, pulmonary embolism, congenital heart disease and cardiac channelopathies. Studies of fQRS in various primarily non-cardiac conditions like obstructive sleep apnea, renal disease, cirrhosis of the liver, radiotherapy in breast cancer, autoimmune disorders and beta thalassemia have also been reported.

A. Acute coronary syndrome:

In 2016 Dr. Sita Ram Mittal have reported that fQRS complex represents myocardial scar in patients with suspected or known coronary artery disease. Hence in patients presenting acute coronary syndrome the possibility of myocardial necrosis over mere unstable angina is very high if fQRS complex developed within 48 hours of presentation[24].

B. Cardiac channelopathies:

i) Brugada syndrome:

Brugada syndrome is a rare genetic condition associated with increased risk of ventricular tachyarrhythmias and sudden cardiac death. It is characterized by cove-type ST elevation in right precordial leads and episode of ventricular tachyarrhythmias. fQRS complex is often present in these patients and more frequently in Ventricular fibrillation group. In 2012 with prospective Italian registry of 308 patients, the PRELUDE (Programmed Electrical stimulation predictive valuE) study has shown that the presence of fQRS in patient with

Brugada syndrome was useful to identify candidates for prophylactic implantable cardioverter-defibrillator (ICD) implantation[25].

ii) Acquired long QT syndrome:

It is a disorder of cardiac repolarization most often due to some drug like Quinidine, Amiodarone, Sotalol, Thioridazine, Fluoroquinolone and hypokalemia or hypomagnesemia that may precipitate torsade de pointes and cause sudden cardiac death. It was found that fQRS was present in a large proportion of patients with acquired long QT syndrome with syncope/torsades de pointes[26].

C. Left ventricular hypertrophy:

Presence of fQRS correlates with increased arterial stiffness in asymptomatic patients with hypertension. Fragmented QRS complex is also associated with more severe diastolic dysfunction in patients with hypertension, which is attributed to an excessive myocardial fibrosis and collagen deposits[27].

D. Aortic stenosis:

The presence of fQRS independently correlates with severity of aortic stenosis. As it is clearly associated with myocardial fibrosis[28].

E. Congenital heart disease:

In adult patients with repaired Tetralogy of Fallot presence of fQRS is associated with right ventricular fibrosis. QRS duration and fragmentation are also associated with right ventricular dysfunction and atrialization in patients with Ebsteins anomaly[29].

F. Rheumatic heart disease:

Fragmentation of QRS complex is frequently seen in patients with mitral stenosis caused by rheumatic fever. Mitral stenosis in rheumatic heart disease is attributed to inflammation and degeneration of cardiac valves secondary to rheumatic fever and hence fQRS was associated with poor NYHA function class, low ejection fraction, pulmonary hypertension and decreased mitral valve area[30].

G. Dilated cardiomyopathy:

In patients with non-ischaemic dilated cardiomyopathy fQRS was associated with cardiac fibrosis and significant intraventricular dyssynchrony and was found to be useful for identifying patients who benefit from cardiac resynchronization therapy[31].

H. Cardiac sarcoidosis:

Sarcoidosis is a chronic inflammatory granulomatous disease that more frequently involves the lung, although multiorgan involvement is common. Cardiac involvement usually manifests with atrio-ventricular block, right bundle branch block, ventricular tachyarrhythmias and heart failure. It was reported that the sensitivity and specificity for

detecting abnormal gadolinium delayed enhancement in cardiac magnetic resonance imaging were 100% and 80% respectively in the presence of fQRS[32].

I. Other conditions:

As major pathogenesis for fragmentation of QRS complex being cardiac fibrosis, fQRS suggests cardiac involvement in patients with rheumatoid arthritis, systemic lupus erythematosus, systemic sclerosis, familial Mediterranean fever, obstructive sleep apnea, iron overload in beta-thalassemia major and metabolic syndrome. Though exact clinical significance of fQRS is not yet determined due to small number of patients with theses disease conditions[24].

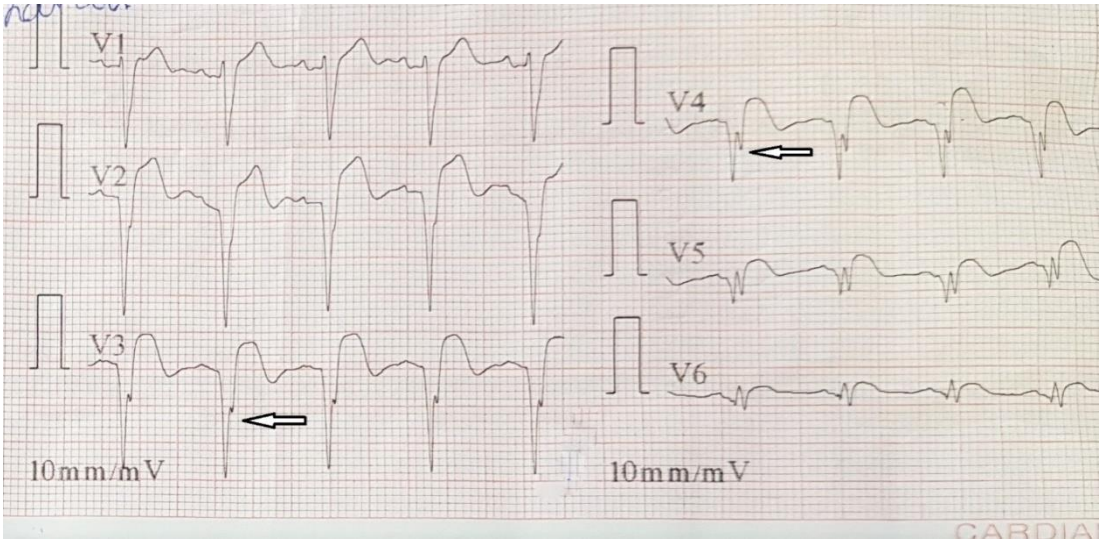


Figure 7: Anterior wall ST- segment elevation myocardial infarction with fragmented QRS complex of **Notched S type in V2, V3, V4 and rSr' type in V5, V6.**

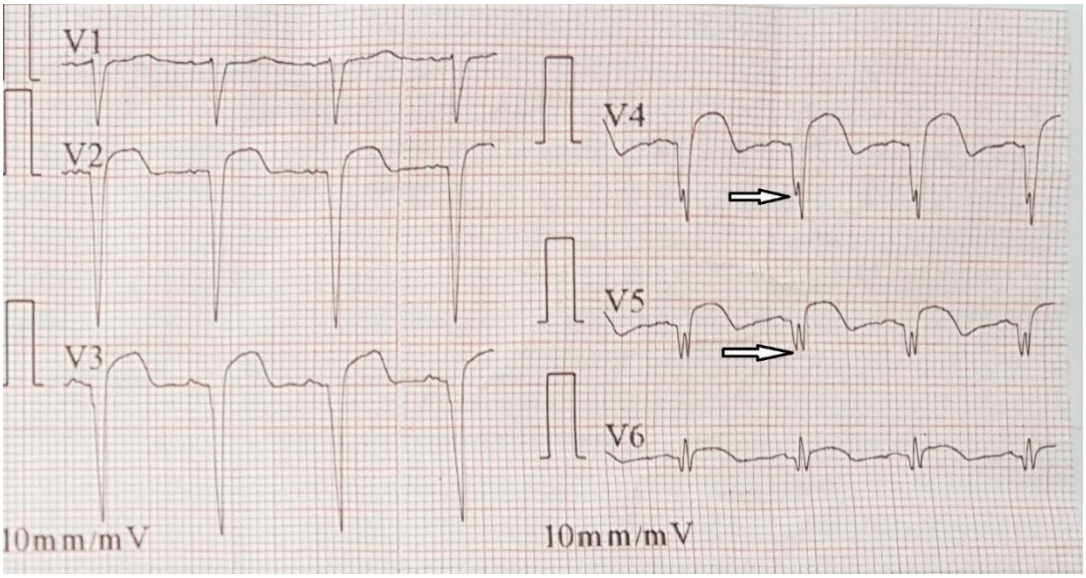


Figure 8: Anterior wall ST- segment elevation myocardial infarction with fragmented QRS complex of **Notched R type in V4and rSr' type in V5**

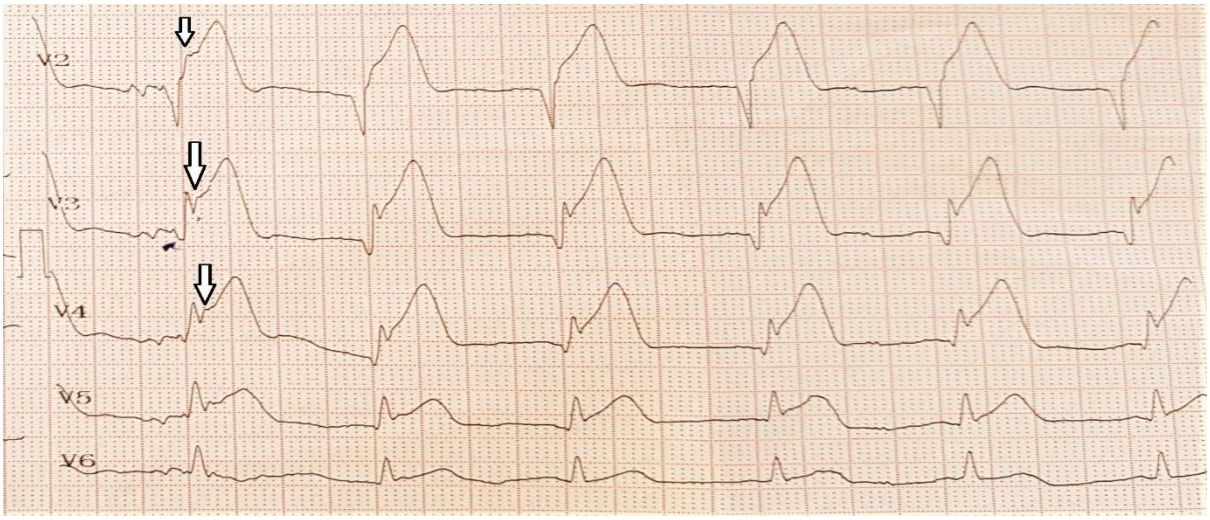


Figure 9: Anteroseptal wall ST- segment elevation myocardial infarction with fragmented QRS complex of **RSR' type in V2, V3 and V4.**

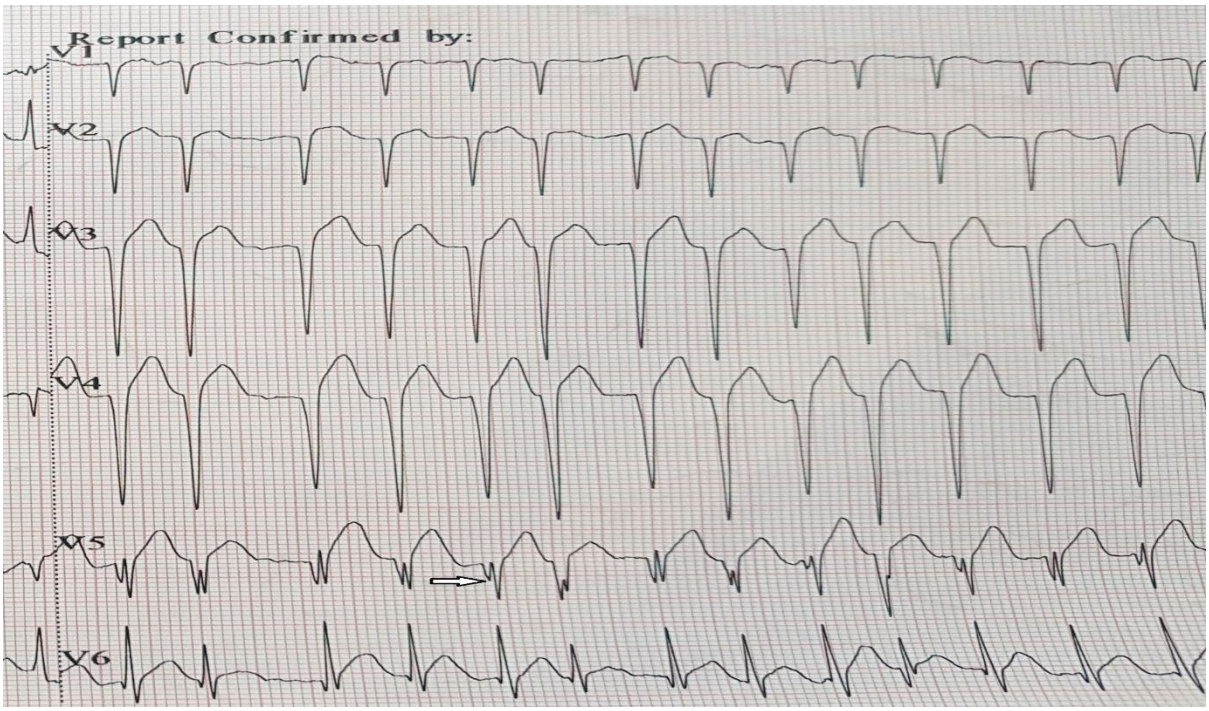


Figure 10: Anterolateral wall ST- segment elevation myocardial infarction with fragmented QRS complex of **rSr' type inV5.**

2.6. FRAGMENTED QRS COMPLEX AND NT -pro-BNP

ECG is the most widely available tool to diagnose and manage cardiac events. In recent years number of researches have been done to predict major adverse cardiac events at the earliest. One such biomarkers being NT-pro-BNP, which have been proven to be elevated in many major adverse cardiac events, especially in heart failure. The natriuretic peptides comprise several structurally related molecules, such as atrial natriuretic peptide (ANP) and brain natriuretic peptide (BNP), which play important roles in cardiovascular homeostasis. Myocardial ischemia is a strong trigger of NT-pro-BNP. As ischemia precedes necrosis in acute myocardial infarction, NT-pro-BNP also might increase in myocardial infarction. In a study done on 216 patients from June 2014 to February 2015 by Qi Zhao et al. reported that presence of fQRS is significantly associated with NT-pro-BNP and left ventricular ejection fraction, which can predict LV function in patients with STEMI[33].

MATERIALS AND METHODS

IV. MATERIALS AND METHODS

1.1. SOURCE OF DATA

This study was carried out in the Department of General Medicine, BLDE (Deemed to be university) Shri B M Patil Medical College, Hospital and Research Centre, Vijayapura. Study was conducted from November 2018 to April 2020 on 152 patients admitted to ICCU with ST-Segment elevation myocardial infarction. This study was conducted after obtaining approval from the institutional ethical committee. Patients were explained about the procedure in detail and consent was obtained for the same.

1.2. Study Design: Prospective cross-sectional study

1.3. Study Period: One and half year from November 2018 to April 2020.

1.4. Sample size calculation

With 95% confidence level and margin of error of ±10%, a sample size of 81 (≈90), on the basis of the study, subject will allow the study to determine the "FRAGMENTED QRS COMPLEX AS A PREDICTOR OF IN HOSPITAL OUTCOME OF ADVERSE CARDIAC EVENTS IN ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION" with finite population correction (N=500).

By using the formula:

$$n = \frac{z^2 p(1-p)}{d^2}$$

where

Z= z statistic at 5% level of significance

d is margin of error

p is anticipated prevalence rate (50%)

1.5. PATIENT SELECTION

A. INCLUSION CRITERIA:

i) Patients admitted with ST segment elevation myocardial infarction.

B. EXCLUSION CRITERIA

i) Patients with Non-STEMI.

ii) Patients with valvular heart disorder.

iii) Patients having bundle branch block on ECG (LBBB or RBBB).

iv) Patient on temporary or permanent pacemaker.

1.6. INVESTIGATIONS.

Investigations required in this study are standardized procedures. Baseline investigations like, Complete blood count, Blood glucose, Renal function test, Serum electrolytes and Urine Examination were done. In addition, cardiac specific investigations like Troponin I/ Troponin T, CPK MB, Electrocardiogram, Chest X ray, 2D Echocardiography study was done.

METHODOLOGY:

2.1. INITIAL ASSESSEMENT

The study was conducted on patients who were admitted in BLDE (DU), Shri B M Patil Medical College Hospital and Research Centre, Vijayapura with prolonged chest discomfort typical of myocardial ischemia, underwent standardized assessment with clinical history and examination, electrocardiogram at admission, cardiac enzymes – Troponin I / Troponin T, CPK-MB and other necessary laboratory investigations.

2.2. ELECTROCARDIOGRAPHY

Electrocardiography was done using BPL CARDIART 6108T or VESTA 301i ECG machine for diagnosis of myocardial infarction, to localize the territory and will be assessed for fragmented QRS complexes. Patients were grouped according to the presence of fQRS as Group A, and Group B without fQRS.

2.3. OUTCOME

Patients were followed up during their in-hospital course for the occurrence of major 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 will be 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, LV pseudo-aneurysm formation, and rupture of the LV free wall.

2.4 STATISTICAL ANALYSIS

All characteristics were summarized descriptively. For continuous variables, the summary statistics of mean±standard deviation (SD) were used. For categorical data, the number and percentage were used in the data summaries and diagrammatic presentation. Chi-square (χ^2) test was used for association between two categorical variables.

The formula for the chi-square statistic used in the chi square test is:

$$\chi_c^2 = \sum \frac{(O_i - E_i)^2}{E_i}$$

The subscript “c” are the degrees of freedom. “O” is observed value and E is expected value. C= (number of rows-1)*(number of columns-1)

The difference of the means of analysis variables between two independent groups was tested by unpaired t test.

The t statistic to test whether the means are different can be calculated as follows:

$$t = \frac{(\overline{x_1} - \overline{x_2}) - (\mu_1 - \mu_2)}{\sqrt{\frac{s_1^2}{n_1} + \frac{s_2^2}{n_2}}}$$

where $\bar{x}_1$ = mean of sample 1

$\bar{x}_2$ = mean of sample 2

n_1 = number of subjects in sample 1

n_2 = number of subjects in sample 2

s_1^2 = variance of sample 1 = $\frac{\Sigma(x_1 - \bar{x}_1)^2}{n_1}$

s_2^2 = variance of sample 2 = $\frac{\Sigma(x_2 - \bar{x}_2)^2}{n_2}$

The difference of the means of analysis variables between more than two independent groups was tested by ANOVA and F test of testing of equality of Variance

RESULTS

V. RESULTS

Total of 152 patients were admitted with acute coronary syndrome. Eight patients were excluded from the study based on exclusion criteria, of which three patients had non-ST-segment elevation myocardial infarction, one patient had unstable angina, two patients had valvular heart disease while two patients had Left bundle branch block. Hence total of 144 patients were included in the study.

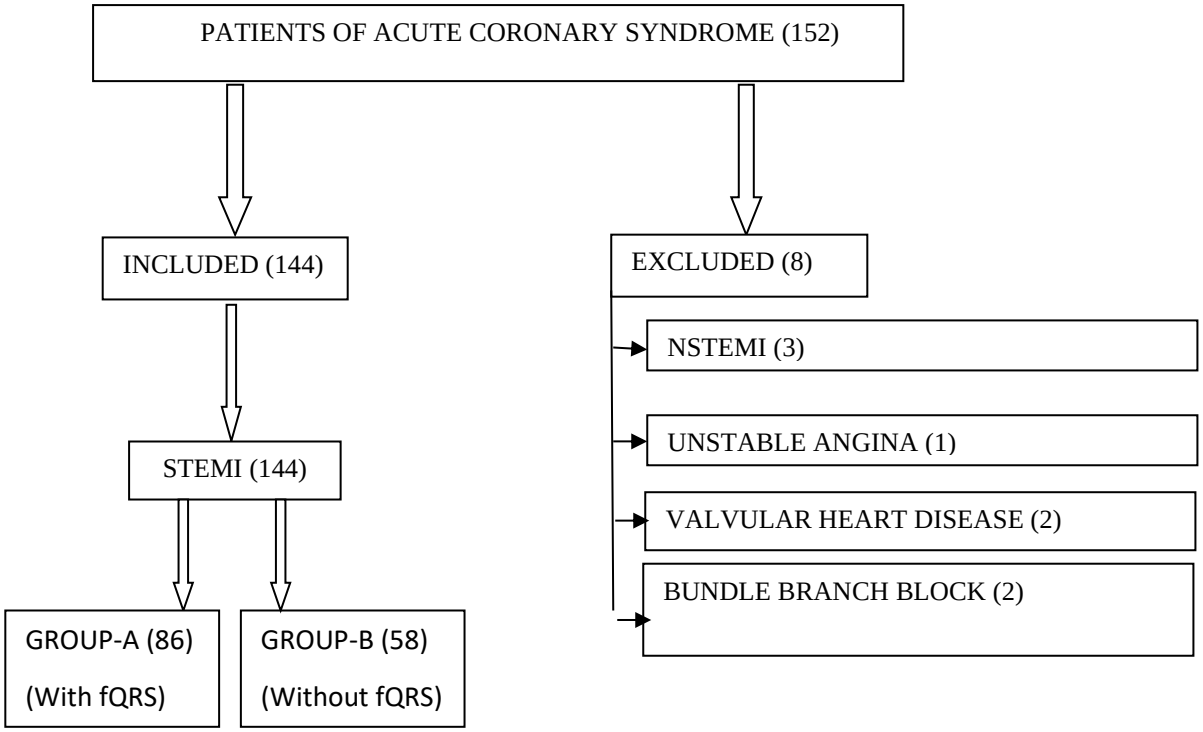


Figure 11: Flowchart showing included and excluded cases in the study.

Note: - $p < 0.05$ – statistically significant

$P < 0.001$ – highly significant

Data were analyzed using SPSS software v.23 (IBM Statistics, Chicago, USA).

Out of 144 patients with STEMI, 86 patients with presence of fQRS on ECG are in group A, and 58 patients without fQRS are in group B as shown in Table 2, Figure 11.

Table 2: Grouping of patients with STEMI.

CLASSIFICATION	NUMBER OF PATIENTS
GROUP A	86
GROUP B	58

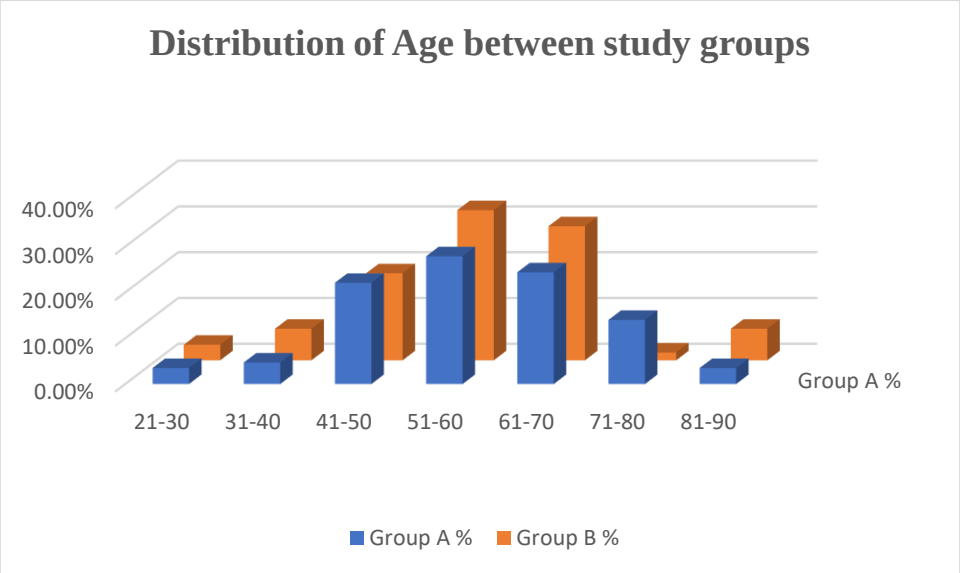
1.1. AGE DISTRIBUTION

The 144 patients were grouped with an age frequency of 10 years. In group A patients aged between 21-30 yrs. were 3 (3.5%), patients aged between 31-40 yrs. were 4 (4.7%), patients between the age 41-50 yrs. were 19 (22.1%), patients between the age 51-60 were 24 (27.9%), patients between the age 61-70 years were 21 (24.4%), patients between the age 71-80 yrs. were 12 (14.0%) and patients aged more than 80 yrs. were 3 (3.5%). In group B patients aged between 21-30 yrs. were 2 (3.4%), patients aged between 31-40 yrs. were 4(6.9%), patients between the age 41-50 yrs. were 11(19.0%), patients between the age 51-60 were 19(32.8%), patients between the age 61-70 yrs. were 17(29.3%), patients between the age 71-80 yrs. were 1(1.7%), patients aged more than 80 yrs. were 4(6.9%). The most common age group in both group A and group B was 51-60 years as described in Table 3, Graph 1.

TABLE 3: DISTRIBUTION OF PATIENTS ACCORDING TO AGE

Age(yrs.)	Group A		Group B		p value
	N	%	N	%	
21-30	3	3.5%	2	3.4%	0.266
31-40	4	4.7%	4	6.9%	
41-50	19	22.1%	11	19.0%	
51-60	24	27.9%	19	32.8%	
61-70	21	24.4%	17	29.3%	
71-80	12	14.0%	1	1.7%	
81-90	3	3.5%	4	6.9%	
Total	86	100.0%	58	100.0%	

Graph 1: DISTRIBUTION OF PATIENTS ACCORDING TO AGE



1.2. SEX DISTRIBUTION

Out of 144 patients in the study, 113 patients (78.5%) were male and 31 patients (21.5%) were female. In this study male patients were more than females as depicted in Table 4. In group A 69 (80.2%) patients were male and 17 (19.8%) females; while 44(75.9%) patients were male and 14(24.1%) were female in group B as shown in Table 5, Graph 2.

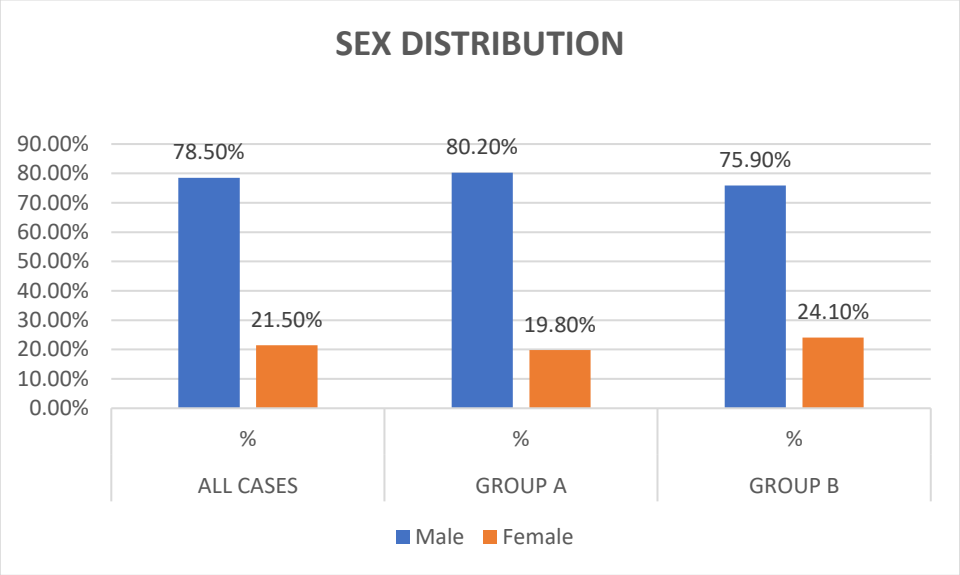
Table 4: Distribution of Sex among all cases

Sex	N	%
Male	113	78.5
Female	31	21.5
Total	144	100

Table 5: Distribution of Sex between study groups

Sex	Group A		Group B		p value
	N	%	N	%	
Male	69	80.2%	44	75.9%	0.531
Female	17	19.8%	14	24.1%	
Total	86	100.0%	58	100.0%	

Graph 2: Distribution of Sex among all cases and between study groups



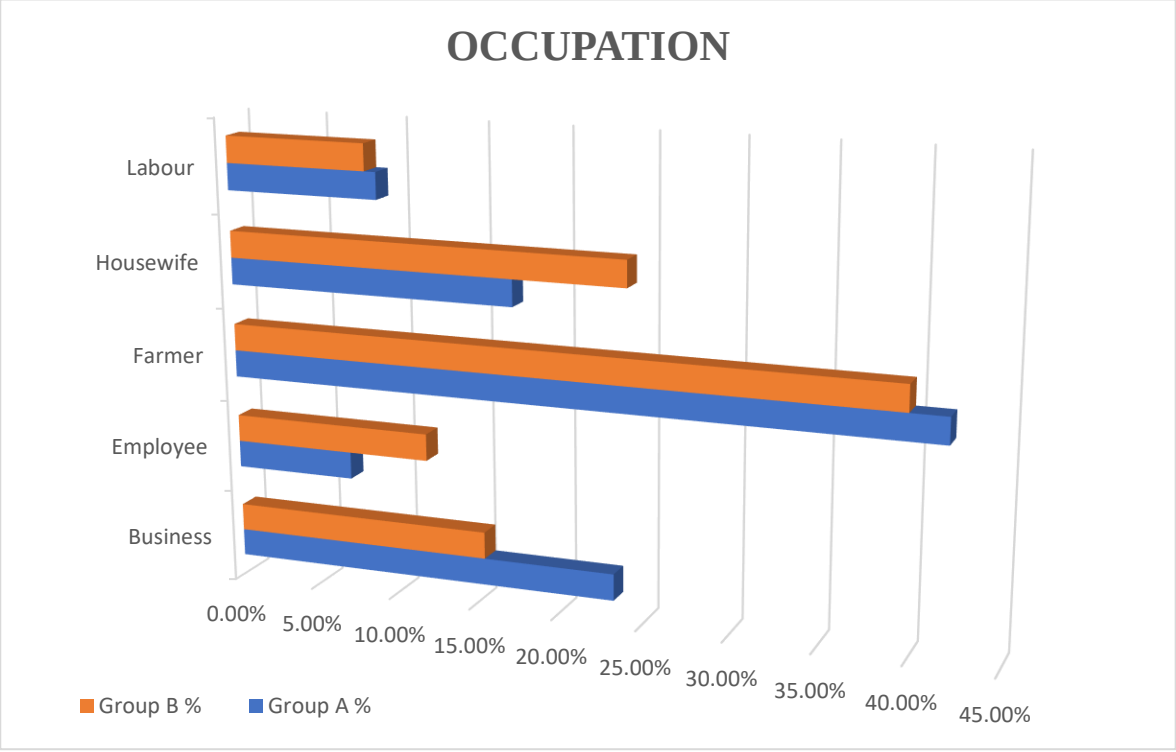
1.3. DISTRIBUTION OF PATIENTS ACCORDING TO OCCUPATION

In group A there were 36 (41.9%) farmers followed by business- 20 (23.3%), housewife- 15 (17.4%), labourers- 8 (9.3%) and 7 (7.2%) were service employee. while in group B, housewife- 14 (24.1%), business- 9 (15.5%), service employee- 7 (12.0%) and laborer were 5 (8.6%). The most common occupation associated with fragmented QRS complex in this study was Farming followed by business, housewife, laborer, service employee as depicted in Table 6, Graph 3. In this study the most common occupation associated with ST-segment elevation myocardial infarction in both group A and group B was farmers i.e., 41.5% and 39.7% respectively.

Table 6: Distribution of Occupation between study groups

Occupation	Group A		Group B		p value
	N	%	N	%	
Business	20	23.3%	9	15.5%	0.652
Employee	7	7.2%	7	12.0%	
Farmer	36	41.9%	23	39.7%	
Housewife	15	17.4%	14	24.1%	
Labor	8	9.3%	5	8.6%	
Total	86	100.0%	58	100.0%	

Graph 3: Distribution of Occupation between study groups



1.4. DISTRIBUTION OF PATIENTS ACCORDING TO RISK FACTORS:

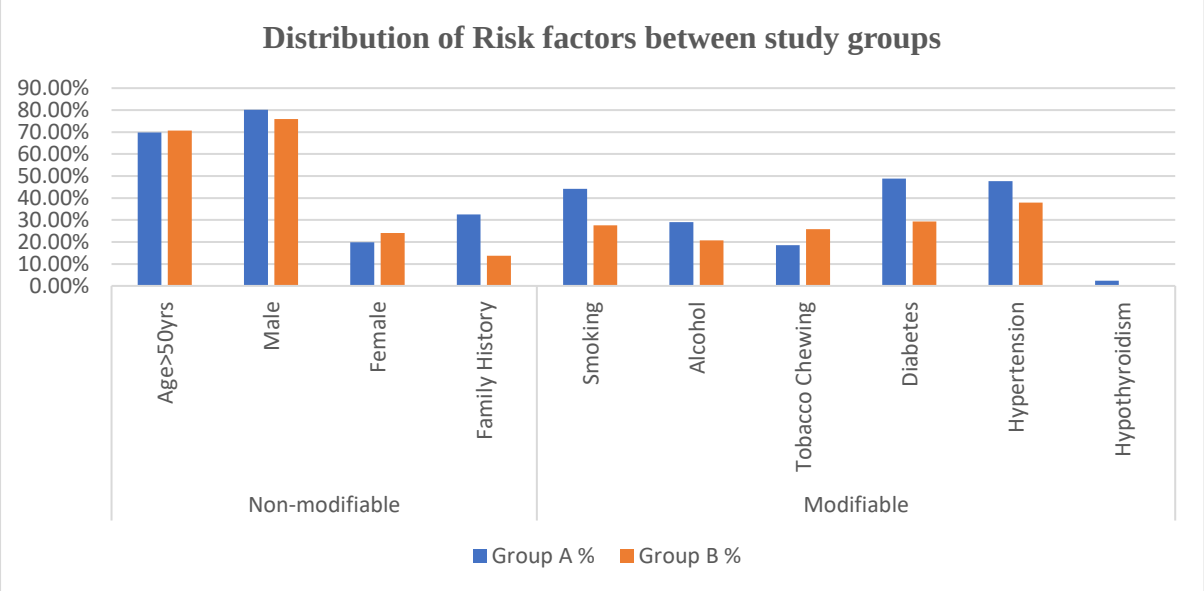
Among risk factors, out of 144 patients in the study, 60 patients (69.8%) in group A compared to 41 patients (70.7%) in group B were aged more than 50 years. Male sex was seen in 69 patients (80.2%) compared to 44 patients (75.9%) in group B. Diabetes mellitus was seen in 59 patients (40.97%) of which 42 patients (48.8%) had fragmented QRS complex and 17 patients (29.3%) were without fragmented QRS complex. Hence the incidence of fragmented QRS complex was more in diabetic with a statistically significant p value of 0.019. Hypertension was seen in 63 patients (43.75%) of which 41 patients in group A and 22 patients in group B. Family history of Acute coronary syndrome was seen in 36 patients, of which 28 patients (32.6%) had fragmented QRS complex and 8 patients (13.8%) were without fragmented QRS complex. Hence the incidence of fragmented QRS complex was more in patients with family history of acute coronary syndrome with a statistically significant p value of 0.011. Smoking habit was seen in 54 patients of which 38 patients (44.2%) are in group A and 16 patients (27.6%) in group B. Alcohol consumption was present in 37 patients of which 25 patients (29.1%) from group A and 12 patient (20.7%) in group B. Tobacco chewing was seen in 31 patients , of which 16 patients (18.6%) from group A and 15 patients (25.9%) in group B as shown in Table 7, Graph 4.

Table 7: Distribution of Risk factors between study groups

Risk factors		Group A		Group B		p value
		N	%	N	%	
Non-modifiable	Age>50yrs	60	69.8%	41	70.7%	0.906
	Sex					
	Male	69	80.2%	44	75.9%	0.531
	Female	17	19.8%	14	24.1%	
	Family History	28	32.6%	8	13.8%	0.011*
Modifiable	Smoking	38	44.2%	16	27.6%	0.044*
	Alcohol	25	29.1%	12	20.7%	0.259
	Tobacco Chewing	16	18.6%	15	25.9%	0.299
	Diabetes	42	48.8%	17	29.3%	0.019*
	Hypertension	41	47.7%	22	37.9%	0.248
	Hypothyroidism	2	2.3%	0	0.0%	0.125

Note: * significant at 5% level of significance ($p<0.05$)

Graph 4: Distribution of Risk factors between study groups



1.5. DISTRIBUTION OF PATIENTS ACCORDING TO SYMPTOMS:

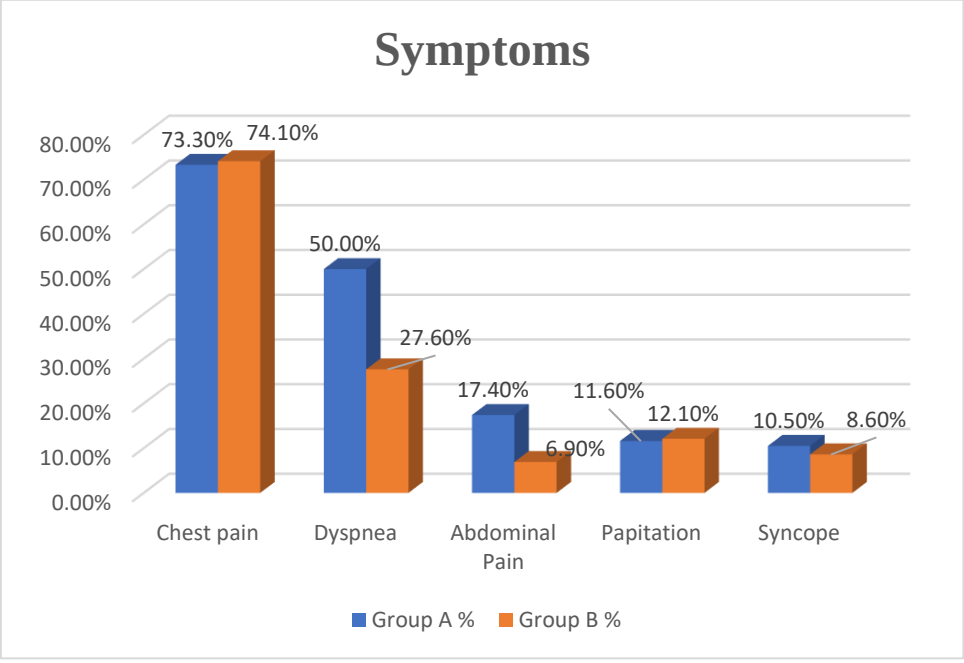
In this study, as shown in Table 8, Graph 5, in both group A and group B the most common symptom was chest pain (73.3% vs 74.1%), followed by dyspnea (50.0% vs 27.6%), abdominal pain (17.4% VS 6.9%), palpitations (11.6% VS 12.1%%) and syncope (10.5% vs 8.6%).

Table 8: Distribution of Symptoms between study groups

Symptoms	Group A		Group B		p value
	N	%	N	%	
Chest pain	63	73.3%	43	74.1%	0.906
Dyspnea	43	50.0%	16	27.6%	0.007*
Abdominal Pain	15	17.4%	4	6.9%	0.067
Palpitation	10	11.6%	7	12.1%	0.936
Syncope	9	10.5%	5	8.6%	0.714

Note: * significant at 5% level of significance (p<0.05)

Graph 5: Distribution of Symptoms between study groups



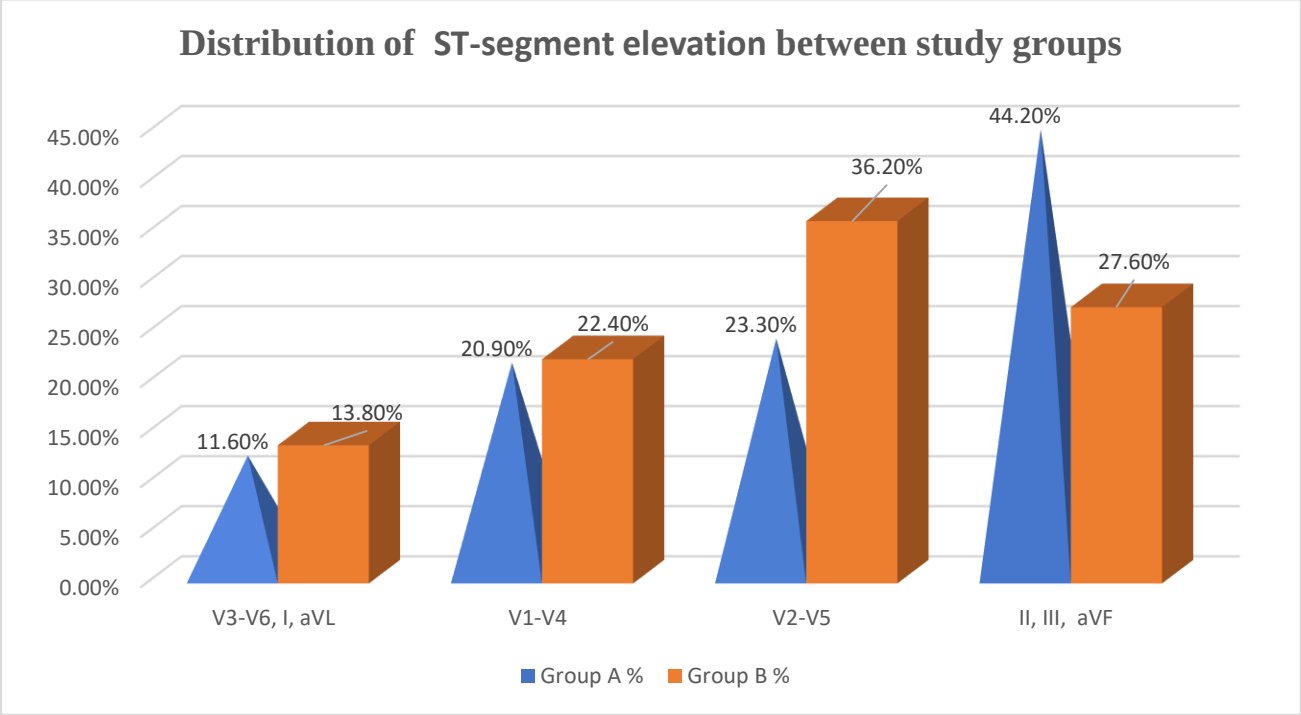
1.6. DISTRIBUTION OF PATIENTS ACCORDING TO ELECTROCARDIOGRAPHIC FINDINGS:

Out of 86 patients with ST segment elevation in group A, anterior leads (V2-V5) ST elevation was seen in 20 patients, antero-septal leads (V1-V4) ST elevation was seen in 18 patients, antero-lateral (V3-V6, I, aVL) leads ST segment elevation was seen in 10 patients, inferior leads (II, III, aVF) ST elevation was seen in 38 patients. The most common ST segment elevation was seen in inferior leads (II, III, aVF) in group A compared to anterior leads in group B as depicted in Table 9, Graph 6.

Table 9: Distribution of ST-segment elevation between study groups

ST-segment elevation	Group A		Group B		p value
	N	%	N	%	
V3-V6, I, aVL	10	11.6%	8	13.8%	0.191
V1-V4	18	20.9%	13	22.4%	
V2-V5	20	23.3%	21	36.2%	
II, III, Avf	38	44.2%	16	27.6%	
Total	86	100.0%	58	100.0%	

Graph 6: Distribution of ST-segment elevation between study groups



1.7. DISTRIBUTION OF PATIENTS ACCORDING TO ECHOCARDIOGRAPHIC VARIABLES:

In this study of 144 patients, echocardiographic parameters were analyzed. Out of 86 patients in group A, 10 patients (11.6%) had antero-lateral wall hypokinesia, 18 patients (20.9%) had antero-septal wall hypokinesia, 20 patients (23.3%) had anterior wall hypokinesia and 38 patients (44.2%) had inferior wall hypokinesia. While out of 58 patients in group B, 8 patients (13.8%) had antero-lateral wall hypokinesia, 13 patients (22.4%) had antero-septal wall hypokinesia, 21 patients (36.2%) had anterior wall hypokinesia and 16 patients (27.6%) had inferior wall hypokinesia. In this study most commonly, there was hypokinesia of inferior wall in both group A and anterior wall hypokinesis in group B as shown in Table 10, Graph 7.

In our study of 144 patients were divided into group A and group B, distribution of left ventricular ejection fraction was studied as shown in table 11, Graph 8.In group A (fQRS Present cases) with 86 cases distribution of left ventricular ejection fraction according to regional wall motion abnormality showed LVEF of < 40% in 53 patients (62%) and > 40% in 26 patients(44.82%). While in group B (fQRS absent cases) with 58 cases distribution of left ventricular ejection fraction according to regional wall motion abnormality showed LVEF of < 40% in 33 patients (38.37%) and >40% in 32 patients(55.17%), with significant p value of 0.047.

Table 10: Distribution of regional wall motion abnormality between study groups

Regional wall motion abnormality	Group A		Group B		p value
	N	%	N	%	
Antero-Lateral	10	11.6%	8	13.8%	0.191
Antero-Septal	18	20.9%	13	22.4%	
Anterior	20	23.3%	21	36.2%	
Inferior	38	44.2%	16	27.6%	
Total	86	100.0%	58	100.0%	

Graph 7: Distribution of regional wall motion abnormality between study groups

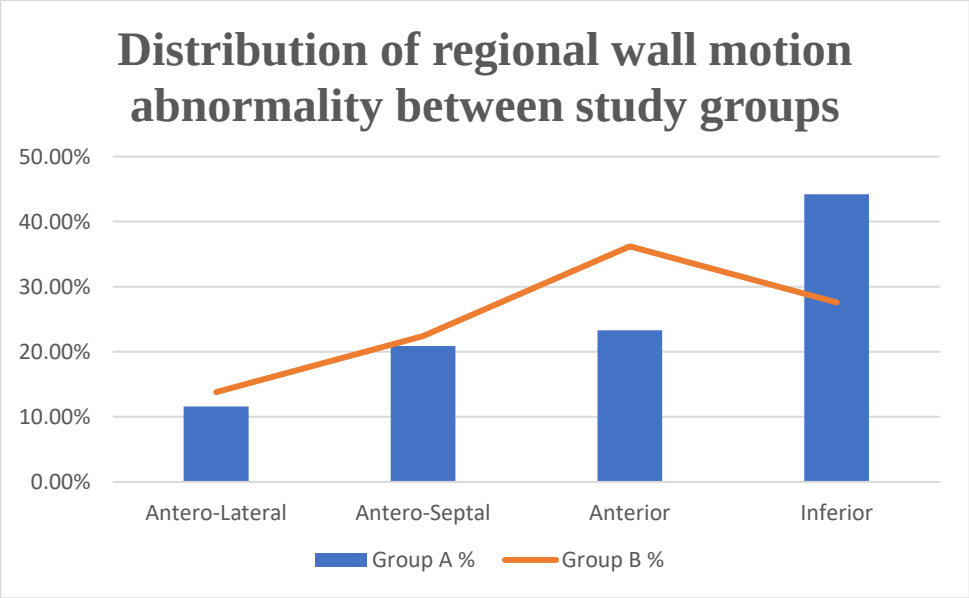


Table 11: Distribution of left ventricular ejection fraction between study groups

LVEF	Group A		Group B		P VALUE
	N	%	N	%	
<40%	53	62%	26	44.82%	0.047
>40%	33	38.37%	32	55.17%	
TOTAL	86	100.00%	58	100.00%	

Graph 8: Distribution of left ventricular ejection fraction between study groups

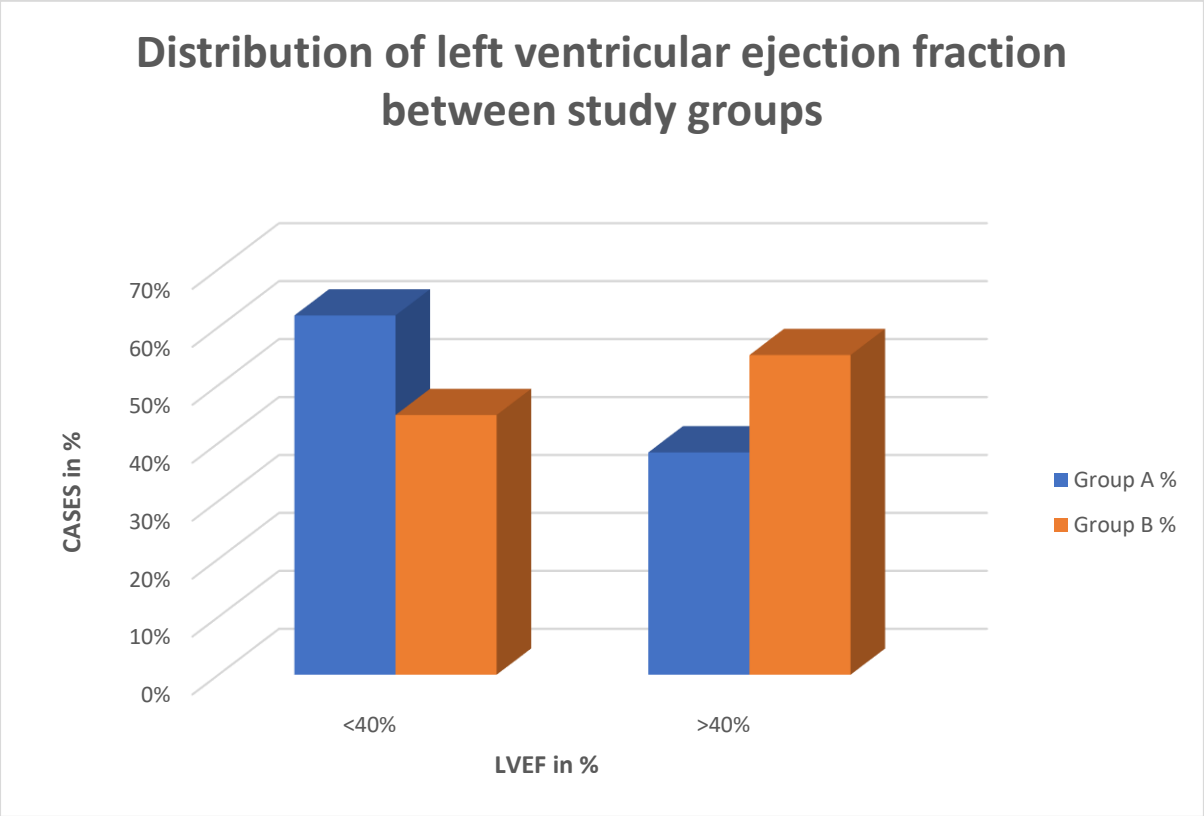


Table 12: Background Parameters between study groups

Parameters	fQRS Present		fQRS Absent		p value
	Mean	SD	Mean	SD	
PR (beats per minute)	87.2	20.4	87.4	20.3	0.953
RR (cycles per minute)	17.2	4.0	15.8	3.0	0.028*
Temperature (degree Celsius)	38.4	6.4	38.6	8.0	0.913
Hemoglobin (gm%)	13.1	5.0	13.1	2.7	0.962
Total Count (cells/cu.mm)	13342.3	4940.2	12257.3	4607.7	0.186
ESR (mm/hr)	31.1	26.8	34.0	27.0	0.531
RBS (mg/dl)	212.2	113.9	174.0	92.3	0.035*
Blood Urea (mg/dl)	29.9	16.1	29.8	15.3	0.97
Sr. Creatinine (mg/dl)	1.0	0.4	1.2	1.2	0.122
Sr. Sodium (mmol/l)	134.7	4.9	132.6	18.2	0.322
Sr. Potassium (mmol/l)	4.6	3.5	5.0	5.8	0.662
Troponin T (ng/l)	42.9	148.2	6.7	9.8	0.202
CPK MB (ng/ml)	80.2	72.0	71.6	74.8	0.493

Note: * significant at 5% level of significance ($p<0.05$)

1.8. DISTRIBUTION OF PATIENTS ACCORDING TO FRAGMENTED QRS COMPLEX AND MAJOR ADVERSE CARDIAC EVENTS:

Patients are grouped according to presence and absence of fragmented QRS complex. Out of 144 patients in study, 86 patients had fragmented QRS (group A) complex on ECG, whereas fragmented QRS complex was absent in rest 58 patients (group B). Among these groups, as depicted in Table 13, Graph 9, Heart failure was found in 48 patients (55.8%) with fragmented QRS as compared to 7 patients (12.1%) without fragmented QRS. Pulmonary edema was found in 40 patients (46.5%) with fragmented QRS as compared to 4 patients(6.9%) without fragmented QRS. Cardiogenic shock was present in 36 patients (41.9%) with fragmented QRS as compared to 7 patients(12.1%) without fragmented QRS. In fragmented QRS present group one patient (1.2%) had heart block, three patients had monomorphic ventricular tachycardia. In fragmented QRS absent group one patient (1.7%) had heart block, one patient (1.7%) had atrial fibrillation and one patient (1.7%) had ventricular premature contraction. In total 24 patients (27.9%) died in group A compared to 2 patients (3.4%) in group B. In this study, major adverse cardiac events like heart failure (p value <0.001), pulmonary edema (p value <0.001), cardiogenic shock (p value <0.001), death (p value <0.001) were higher in patients with fragmented QRS complex, with p value <0.001which is statistically very significant.

Table 13: Major adverse cardiac events between study groups

Major adverse cardiac events	Group A		Group B		p value
	N	%	N	%	
Arrhythmia	4	4.7%	2	3.4%	0.347
Heart Failure	48	55.8%	7	12.1%	<0.001*
Pulmonary Edema	40	46.5%	4	6.9%	<0.001*
Cardiogenic Shock	36	41.9%	7	12.1%	<0.001*
Death	24	27.9%	2	3.4%	0.001*

Note: * significant at 5% level of significance (p<0.05)

Graph 9: Major adverse cardiac events between study groups

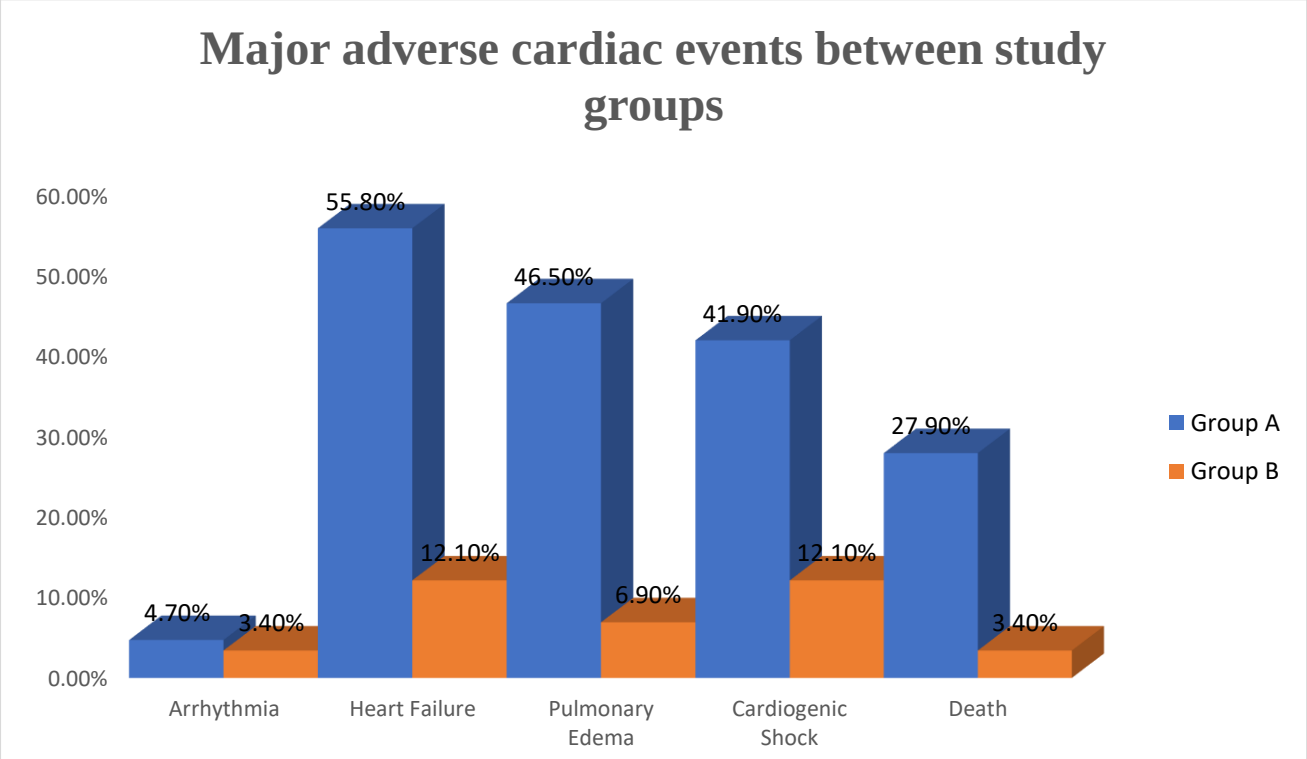
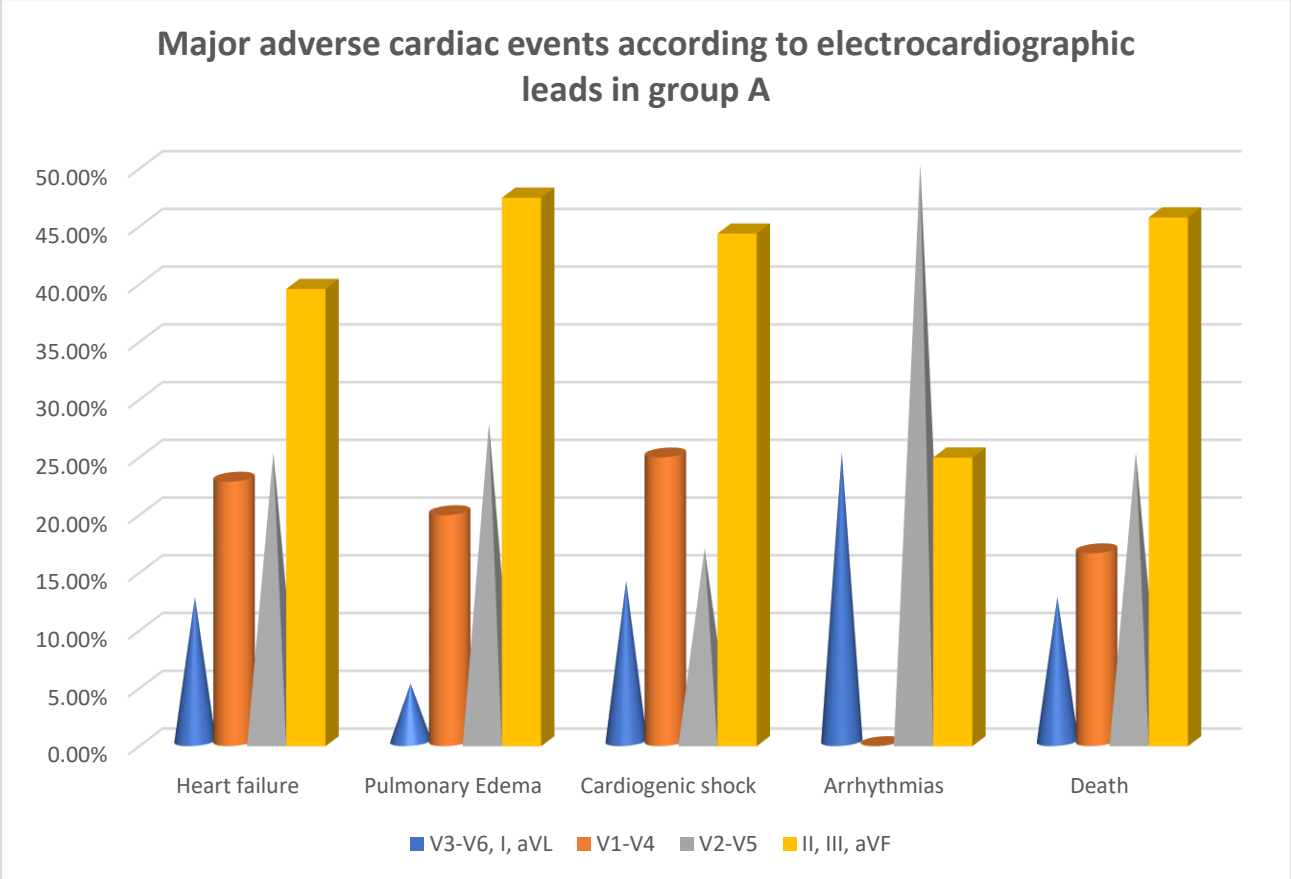


Table 14: Distribution of major adverse cardiac events according to electrocardiographic leads in group A (fQRS Present cases).

Major adverse cardiac events	V3-V6, I, aVL		V1-V4		V2-V5		II, III, aVF		Total fQRS Present cases	p value
	N	%	N	%	N	%	N	%		
Heart failure	6	12.5%	11	22.9%	12	25.0%	19	39.6%	48	0.816
Pulmonary Edema	2	5.0%	8	20.0%	11	27.5%	19	47.5%	40	0.305
Cardiogenic shock	5	13.9%	9	25.0%	6	16.7%	16	44.4%	36	0.589
Arrhythmias	1	25%	0	0.0%	2	50%	1	25.0%	4	0.367
Death	3	12.5%	4	16.7%	6	25.0%	11	45.8%	24	0.945

Graph 10: Distribution of major adverse cardiac events according to electrocardiographic leads in group A (fQRS Present cases).



DISCUSSION

VI. DISCUSSION

This study is a prospective observational study conducted from November 2018 to April 2020. The aim was to study fragmented QRS complex as a predictor of in-hospital outcome of the major adverse cardiac events in patients with ST-segment elevation myocardial infarction. 144 patients included in this study were analyzed to predict in-hospital major adverse cardiac events, like heart failure, pulmonary edema, cardiogenic shock, conduction abnormalities like sinus bradycardia, bradyarrhythmia (conduction blocks), tachyarrhythmias (ventricular tachycardia, supraventricular tachycardia, atrial fibrillation), ventricular ectopic and death.

1.1. AGE

In this study the most common age group was 51-60 years. Similarly, in a study done by Rosengren et al, in 2006 on 10253 patients, they found that, common presenting age group was 65-74 years[34].

Another study done by Nedkoff L J et al. in 29421 patients hospitalized with acute coronary syndrome between years 1996 to 2007 in Western Australia, they observed that, common age group was 60-70 years, which was similar to this study and mean age group was 68.2% which was significantly higher than that absorbed in this study[35]. In a study done by Vamadevan et al. it was found than mean age group has changed since early 70's, where it was equal in both Indian and western countries, now it is found that acute coronary syndrome occurs a decade earlier in Indian population compared to western countries[36]. The reason could be lack of education about disease and risk factors, evidence-based treatment, lack of compliance of medications.

1.2. SEX

In this study there was male predominance as 60.7% of patients were males and female patients were 39.3%, which was similar to study done by Nedkoff L J et al. in year 1996 to 2007 on 29421 patients where male patients were 19601(66.6%) and female patients were 9820 (33.4%)[35].

In a study done by Jonathan D Newman et al. between February 1, 2009 and June 30, 2010 out of 476 subjects, male patients were 68.7% and female were 31.3%[37]. In another study done by Sharma R et al. in 2014 on 1562 South Indian patients, Majority were male 1242 (79.5%) and rest were females320 (20.5%) which was significantly higher than this study[38].

1.3. OCCUPATION

In this study the most common occupation associated with ST-segment elevation myocardial infarction in both group A and group B was farmers, 36 (41.5%) and 23 (39.7%) respectively. In group A farmers were followed by business-23.3%, housewife-17.4%, labourers-9.3% and service employee were 7.2%. While in group B, housewife-24.1%, business- 15.5%, service employee- 12.0% and laborer were 8.6%. Most of these patients belong to low and middle socioeconomic status. 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.

1.4. RISK FACTORS:

Non- modifiable risk factors like age and gender is been discussed above. Whereas family history of cardiac events was present in 28 patients (32.6%) in group A and while only 8 patients (13.8%) in group B, and p value of 0.011 suggestive of fragmented QRS complex being more common in patients with family history.

In this study, modifiable risk factors like Diabetes mellitus was present in 42 patients (48.8%) in group A while only 17 patients (29.3%) were diabetic in group B. Hence with a statistically significant p value of 0.019, diabetes mellitus and fragmented QRS complex shows strong association. Hypertension was present in 41 patients (47.7%) in group A and 22 patients (37.9%) in group B. Two patients (2.3%) were known case of hypothyroidism in group A. Smoking was present in 38 patients (44.2%) in group A while 16 (27.6%) in group B, with a statistically significant p value of 0.044. Other risk factors like tobacco chewing in patients (18.6% vs 25.9%), alcohol consumption in (29.1% vs 20.7%) was seen in both group A and group B respectively. There is significant variation in various risk factors and their association with acute coronary syndrome in different studies. In this study diabetes mellitus was observed in 26.2% which is higher than that is reported in INTERHEART study done in 2004, where 15152 cases and 14820 controls were enrolled from 52 other countries[41]. In a study done by M N Krishnan et al. in 2016, on 5167 patients with acute coronary syndrome, diabetes mellitus was present in 15% (n=775) patients, hypertension in 28% (n=1446) patients and smoking in 28% (n=1446) of patients[42]. Another study done by Vinay Rao et al. in 2017, in 100 patients with acute coronary syndrome, it was observed that diabetes was present in 67% of patients, hypertension in 52% of patients, smoking was present in 61% of patients, alcohol consumption in 21% of patients[43]. This study has high incidence of risk factors like diabetes mellitus, hypertension, smoking and alcohol consumption for acute coronary syndrome compared to this study. In a study done by Unal et al. between 1981 to 2000, they concluded that, life expectancy of patients with ACS can be increased four times than that is increased by modern cardiological treatment by modest reduction in major risk factors like smoking, hypertension, diabetes mellitus[44]. Therefore, there is need for policies to control tobacco use, promote healthy diet and educate

patients regarding control of diabetes mellitus which help in improving life expectancy of patients with ACS.

1.5. SYMPTOMS

In this study, in both group A and group B the most common symptom was chest pain (73.3% vs 74.1%), followed by dyspnea (50.0% vs 27.6%), abdominal pain (17.4% VS 6.9%), palpitations (11.6% VS 12.1 %%) and syncope (10.5% vs 8.6%). Similarly in a study done by Pravin K Goel et al. from January, 2008–December, 2008 on 609 patients admitted with ACS, they found that the most common symptom in patients with acute coronary syndrome was chest pain(n=510, 84%), followed by dyspnea(n=53, 8.7%) and epigastric pain(n=16, 2.6%) which is similar to our study[39].

In other study done by J G Conto et al. on 434877 patients admitted with acute myocardial infarction from June 1994 to March 1998 in the National Registry of Myocardial Infarction 2, which includes 1674 hospitals in the United States, they found that chest pain was present in 67% (n=291367) of patients which is less than that observed in this study[40].

1.6. ELECTROCARDIOGRAM

In this study of 144 patients, electrocardiogram shows ST segment elevation, of which, anterior leads (V2-V5) ST elevation was seen in 41 patients, antero-septal leads (V1-V4) ST elevation was seen in 31 patients, antero-lateral (V3-V6, I, aVL) leads ST segment elevation was seen in 18 patients, inferior leads (II, III, aVF) ST elevation was seen in 54 patients. In our study ST segment elevation was commonly seen in inferior leads. In a study done by Reddy CST et al in 2013, they reported that electrocardiogram identification of culprit artery helps in not only localization of proximal or distal occlusion but also to predict severity of myocardial infarction and plan emergency management[45].

In this study fragmented QRS complex was present in 86 patients with ST segment elevation which is 59% of total patients. In a study done by Uslu N et al. on 241 consecutive patients with STEMI being treated with primary PCI between June 2014 and February 2015the frequency of fragmented QRS complex on standard 12 lead electrocardiograph has been reported to range from 34.9% to 60.1% in acute coronary syndrome[46]. Incidence of fragmented QRS complex in anterior leads (V2-V5) ST elevation was seen in 20 patients, antero-septal leads (V1-V4) ST elevation was seen in 18 patients, antero-lateral (V3-V6, I, aVL) leads ST segment elevation was seen in 10 patients, inferior leads (II, III, aVF) ST elevation was seen in 38 patients. Therefore, in this study fragmented QRS complex was most commonly seen with inferior lead ST segment elevation. In a study done by Di Liang et al, in 2017 on 302 patients the inferior wall leads showed a significantly higher incidence of fragmented QRS complex among patients with acute myocardial infarction, which is similar to this study[11]. Brenyo et al. in 2012, analyzed 1040 electrocardiograph and reported that fragmented QRS complexes was located in 21% (n=218) patients in inferior leads and were an appropriate predictor of sudden cardiac linked death(hazard ratio 1.46, P = 0.032) [47]. Das et al. in 2009 showed that the location of the leads with fragmented QRS complex on standard 12 lead electrocardiograph reflected the location of ischemia, infarct size and scar tissue in the ventricles[48].

1.6. ECHOCARDIOGRAPHY

In this study of 144 patients, echocardiographic parameters like regional wall motion abnormality and ejection fraction had been studied. In this study most commonly, there was hypokinesia of inferior wall in group A (44.2%) and anterior wall hypokinesis in group B (36.2%). while in group A around 61.7% patients had ejection fraction less than 40% compared to 44.8% in group B with significant p value of 0.047. Therefore, in patients with

fragmented QRS complex there is higher incidence of reduced ejection fraction and depressed left ventricular systolic function.

In a study done by Yan et al., in 2012 on 176 patients showed a strong relation between fragmented QRS complex and depression of left ventricular function h even in sub-clinical situation which is been shown even in this study[49].Another study done by Cheem A et al, in 2010 on 842 patients supports high sensitivity of fragmented QRS complex to the presence of symptomatic left ventricular failure in patients with coronary artery disease[50]. While one more study done by Tarek M Abdelrahman in 2013 on 220 patients out of which 74 patients had fragmented QRS complex showed left ventricular ejection fraction of 49 ± 4 when compared to patients with absence of fragmented QRS complex 61 ± 4.4 with significant p value of 0.001and proved that patients with fragmented QRS complex showed a lower left ventricular function in the form of low ejection fraction which is similar to our study[51].

1.7. MAJOR ADVERSE CARDIAC EVENTS

In this study it was observed that presence of fragmented QRS complex in patients with ST-segment elevation myocardial infarction as a predictor of in-hospital outcome of the major adverse cardiac events like heart failure (48 patients,55.8%), pulmonary edema (40 patients,46.5%), cardiogenic shock (36 patients,41.9%), arrhythmias (4 patients, 4.7%) and death (24 patients,27.9%). Patients with fragmented QRS complex on 12 lead electrocardiograph had worse prognosis in terms of major adverse cardiac events like heart failure, pulmonary edema, cardiogenic shock, arrhythmias and death. So, in general, patients without fragmented QRS complex has good outcome.

The observations in this present study are similar to study done by Tarek M Abdelrahman in 2013, on 220 patients with acute coronary syndrome there where they 74

patients with fragmented QRS complex and 146 patients without fragmented QRS complex, the incidence of major adverse cardiac events like heart failure (n=26 vs n=8), re-infarction (n=8 vs n=2), ventricular arrhythmias (n=18 vs n=6) , and cardiac deaths (n=6 vs n=0) were significantly higher compared to patients without fragmented QRS complex which is similar to this study[51]. In a study by Stavileci et al. in 2014, on 296 patients, fragmented QRS complex was present in 80 (27%) patients, and demonstrated an association between persistent fragmented QRS complex on electrocardiograph at hospitalization with a poor prognosis in acute ST-segment elevation myocardial infarction[52].

Diagnosis of STEMI and assessment of reperfusion may be performed through dynamic changes in standard ECG, which includes pathological Q-waves, T- waves and ST-segment. As previously demonstrated by Das et al. in 2008, Fragmentation of QRS complexes on a routine 12-lead ECG signifies myocardial scar detected by myocardial single photon emission computed tomography (SPECT) imaging in patients with or suspected coronary artery disease. They have also reported fQRS as an independent predictor of mortality in patients with coronary artery disease[48]. Even our study showed increased incidence of mortality in patients with presence of fQRS (n=24, 27.9%) compared to 2 death (3.4%) in patients with absence of fQRS. Zhang et al. in 2017 examined the prognostic value of the complex QRS (fQRS) for microvascular reperfusion and changes in left ventricular function in patients with ST elevation myocardial infarction who underwent primary percutaneous coronary intervention. Total of 216 patients were examined. The patients were divided into two groups of 126 and 90 based on the presence or absence of fQRS. In both groups, troponin levels, NT-pro-BNP and creatine kinase-MB were studied. Zhang stated that in the analysis of logistic regression, LVEF, NT-pro-BNP, Troponin I and microvascular reperfusion were associated with fQRS. Moreover, that the presence of fQRS was not only significantly associated with the myocardial microvascular reperfusion and left

ventricular function, but also with the prognosis of STEMI[53]. Even our study demonstrates high incidence of major adverse cardiac events in patients with fragmented QRS complex.

CONCLUSION

VIII. CONCLUSION

The value of fQRS in cardiology is much higher than what is being understood currently. And as we know electrocardiography is an easily available, inexpensive and non- invasive parameter to evaluate cardiac problems. In the present study of patients with ST-segment elevation myocardial infarction, there was increased incidence of in-hospital major adverse cardiac events like heart failure, pulmonary edema, cardiogenic shock and death in patients with fragmented QRS complexes on electrocardiograph. Hence fQRS can be used as a cheap and reliable marker to predict in-hospital major adverse cardiac events in patients with ST-segment elevation myocardial infarction.

SUMMARY

VII. SUMMARY

One hundred and forty-four patients with ST-segment elevation myocardial infarction admitted at BLDE (Deemed to be University), Shri B M Patil Medical College Hospital and Research Centre, Vijayapura between November 2018to April 2020 were studied.

This study was conducted to know fragmented QRS complex on electrocardiograph as a predictor of in-hospital outcome of the major adverse cardiac events in patients with ST-segment elevation myocardial infarction.

1. Total of 152 patients were studied out of which 8 were excluded based on exclusion criteria. Rest 144 patients were classified into group A, with presence of fragmented QRS complex (86 patients) and group B, with absence of fragmented QRS complex (58 patients).
2. In this study male patients (78.5%) were more than females (21.5%), whereas in group A 69 male (80.2%) and 17 females (19.8%) compared to 44 male (75.9%) and 14 females (24.1%) in group B.
3. The most common age group in both group A and group B was between 51-60 years. 60 (69.8%) patients in group A, and 41 (70.7%) in group B were more than 50 years age.
4. The most common risk factors in group A, were diabetes (48.8%, p value 0.019), hypertension (47.7%), family history (32.6%, p value 0.011) and smoking (44.2%, p value 0.044).
5. The most common occupation associated with ST-segment elevation myocardial infarction in both group A and group B was farming. In group A farmers were followed by business, housewife, labourers and service employee.
6. The most common symptom in both group A and group B was chest pain followed by dyspnea, abdominal pain, palpitations and syncope.

7. Fragmented QRS complex was present in 86 patients with ST segment elevation that is around 59%, while it was most commonly seen in, 44.2% inferior leads (II, III, aVF), followed by 23.3% in anterior (V2-V5), 20.9% in antero-septal (V1-V4) and 11.6% in antero-lateral (V3-V6, I, aVL).
8. In this study most commonly, there was hypokinesia of inferior wall in group A (44.2%) and anterior wall hypokinesis in group B (36.2%).
9. In group A around 61.7% patients had ejection fraction less than 40% compared to 44.8% in group B with significant p value of 0.047. Therefore, in patients with fragmented QRS complex there is higher incidence of reduced ejection fraction and depressed left ventricular systolic function.
10. In group A, the in-hospital major adverse cardiac events like, heart failure (55.8%), pulmonary edema (46.5%), cardiogenic shock (41.9%) and death (27.9%) were more common when compared to group B, were it was heart failure (12.1%),cardiogenic shock (12.1%), pulmonary edema (6.9%), and death (3.4%)with significant p value<0.001.
11. In group A, it is observed that in-hospital major adverse cardiac events are more common with inferior leads (II, III, aVF), followed by anterior (V2-V5), antero-septal (V1-V4) and antero-lateral (V3-V6, I, aVL).
12. The patients with fragmented QRS complex had high incidence of major adverse cardiac events and mortality. Therefore, fQRS can be used as a predictor of in-hospital major adverse cardiac events in patients with ST-segment elevation myocardial infarction.

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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)
SHRI.B.M.PATIL MEDICAL COLLEGE HOSPITAL & RESEARCH CENTRE
VIJAYAPUR – 586103 IEC/NO: 286/2018
17-11-2018

INSTITUTIONAL ETHICAL COMMITTEE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 13-11-2018 at 03-15 PM 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 accorded Ethical Clearance.

Title : Fragmented QRS complex as a predictor of in-hospital outcome of adverse cardiac events in patients with ST-Segment elevation myocardial infarction.

Name of P.G. Student : Dr Siddarthkumar Chawath.
Department of General Medicine.

Name of Guide/Co-investigator: Dr. Badiger Sharanabasawappa, Professor of General Medicine.

DR,RAGHAVENDRA KULKARNI
CHAIRMAN
Institutional Ethical Committee
BLDEU's Shri B.M. Patil
Medical College,BIJAPUR-586103.

Following documents were placed before E.C. for Scrutinization:

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

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 SIDDARTHKUMAR CHAWATH 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 SIDDARTHKUMAR CHAWATH informed me that he/she is conducting dissertation/research titled "FRAGMENTED QRS COMPLEXES AS A PREDICTOR OF IN-HOSPITAL OUTCOME OF ADVERSE CARDIAC EVENTS IN PATIENTS WITH ST-SEGMENT ELEVATION 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: 1.

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.

"FRAGMENTED QRS COMPLEX AS A PREDICTOR OF IN-HOSPITAL OUTCOME OF ADVERSE CARDIAC EVENTS IN PATIENTS WITH ST-SEGMENT ELEVATION 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 Limb:

Chest:

Abdomen:

Genetilia:

Lower Limbs:

Skin:

SYSTEMIC EXAMINATION

Cardiovascular System

Arterial system:

Pulse

Rate

Rhythm

Volume

Character

Condition of vessel wall

Radio radial

Radio femoral delay

Other peripheral pulses

Venous system:

Engorged veins in 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	
Blood urea	
Serum creatinine	
Serum sodium	
Serum potassium	

URINE EXAMINATION -

Albumin	
Sugar	
Microscopy	

TROPONIN I:

CPK MB:

2D-ECHO DOPPLER:

ELECTROCARDIOGRAM

	ECG
Standardization	
Rate	
Rhythm	
P wave	
PR interval	
QRS complex	
QRS configuration	
QRS duration	
QRS Axis	
ST Segment	
T wave	
QT	
QTc	
Arrythmias	
Ectopics	
Heart blocks	

ECG Diagnosis:

FRAGMENTED QRS COMPLEX:

MAJOR 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 N.	PATIENT NAME	AGE	SEX	OCCUPATION	IP N.	D.O.A	D.O.D	SYMPTOMS					DIABETES	HYPERTENSION	FAMILY HISTORY	HABITS			VITALS				HEMOGLOBIN	TOTAL COUNT	ESR	RBS	BLOOD UREA	Sr.CREATININE	Sr.SODIUM	Sr.POTASSIUM	TROPONIN T	CPK MB	ECG			2D-ECHO		MAJOR ADVERSE				INCLUSION/ EXCLUSION
								CHESTPAIN	DYSPNEA	ABDOMINAL PAIN	P	SYNCOPE				SMOKING	ALCOHOL	TOBACCO CHEWING	PR	BP	RR	TEMPERATURE												ST-SEGMENT	RHYTHEM	Fragmented QRS (Qrs)	REGIONALWALL MOTION ABNRMALITY	LVEF	HEART FAILURE	PULMONARY EDEMA	CARDIOGENIC SHOCK	OTHERS	
1	ALISAB	65	M	L	7989/17	11-03-2018	3/16/2018	P	A	A	A	A	A	P	A	P	A	A	88	140/90	16	37	13	11530	40	120	33	1	140	4	P	95	E V1-3	SR	A	ANTERIOSEPTAL	45%	A	A	A	A	I
2	BALA SAHEB	60	M	Fa	8854/17	03/19/2018	3/16/2018	P	A	A	A	A	A	A	A	P	A	A	64	120/80	16	37	14	11840	20	153	20	3	137	4	P	112	E II, III, AVF	SR	A	ANTERIOLATERAL	50%	A	A	A	A	I
3	GANGA	60	F	Fa	9056/17	03/21/2018	3/29/2018	P	A	A	P	A	P	A	A	A	A	A	102	190/100	16	37	11	13600	20	422	26	1	141	5	P	53	E II, III, AVF	SR	P	ANTERIOLATERAL	55%	A	A	A	A	I
4	BAHUBALI	30	M	Em	10063/17	03/30/2018	06-04-2018	A	P	A	A	A	A	A	A	A	A	A	120	96/50	32	37	10	17110	40	85	18	1	140	5	P	72	E II, III, AVF	SR	P	ANTERIOLATERAL	55%	A	A	P	A	I
5	KAMALA BAI	60	F	Fa	10754/17	05-04-2018	06-04-2018	A	A	P	A	A	P	A	A	A	A	A	80	130/80	14	37	12	12100	70	269	34	2	142	3	P	35	E V2-5	SR	P	ANTERIOR	40%	A	P	A	Z	I
6	YELLAPPA	50	M	Fa	11078/17	07-04-2018	12-04-2018	P	A	A	P	A	A	A	A	P	P	A	70	120/70	16	37	12	3830	65	122	22	1	133	4	P	69	E II,III.AVF	SR	A	INFERIOR	45%	A	A	A	A	I
7	GOLLAPPA	87	M	Fa	11399/17	10-04-2018	11-04-2018	P	A	A	A	A	A	P	A	A	A	P	86	90/50	26	37	11	12200	45	104	31	1	137	5	P	114	E V2-6	SR	P	ANTERIOSEPTAL	45%	P	P	P	Z	I
8	GANGARAM	52	M	Em	11400/17	01-10-2018	4/16/2018	P	A	A	A	P	P	A	A	A	A	A	88	130/80	14	37	14	21150	5	93	30	1	141	4	P	206	E V1-4	SR	A	ANTERIOSEPTAL	45%	A	A	A	A	I
9	MACHINDRA	50	M	Fa	11608/17	12-04-2018	4/18/2018	P	A	A	A	A	A	A	A	A	A	P	60	90/60	14	37	14	11620	15	112	24	1	137	5	P	225	E V1-4	SR	A	ANTERIOSEPTAL	45%	A	A	P	A	I
10	MALLIKARJUNA	65	M	Em	13708/17	04/30/2018	05-05-2018	P	A	A	A	A	P	P	A	A	A	A	80	120/80	14	37	13	10140	40	312	22	1	132	5	P	55	E V2-5	SR	A	ANTERIOSEPTAL	50%	A	A	A	B	I
11	BHEERAPPA	65	M	Fa	14012/17	03-05-2018	09-05-2018	P	A	A	A	A	A	A	A	P	A	A	80	1224/80	14	37	14	10200	10	176	22	1	130	4	N	34	E II,III,AVF	SR	A	ANTERIOLATERAL	40%	A	A	A	A	I
12	MAHABOOB	48	M	L	16147/17	5/21/2018	5/26/2018	P	A	A	A	P	P	A	A	A	A	A	40	90/60	14	37	11	16430	5	458	79	3	130	5	P	129	E III, AVF	3:1 HB	P	INFERIOR	60%	A	A	P	A	I
13	PARVATHI	77	F	H	16198/17	5/22/2018	5/28/2018	P	A	A	A	A	P	A	A	A	A	A	80	140/80	15	37	11	7620	15	204	20	1	135	4	N	16	E V5-6, I, AVL	SR	P	ANTERIOLATERAL	45%	A	A	A	A	I
14	YEMKAWWA	80	F	H	17831/17	03-06-2018	07-06-2018	P	A	A	A	A	A	A	A	A	A	A	76	90/60	14	37	13	17650	20	121	34	1	134	3	P	234	E II, III, AVF	SR	P	INFERIOR	45%	A	A	P	A	I
15	RADHA	38	F	H	18253/17	07-06-2018	6/14/2018	P	A	A	A	A	A	A	A	A	A	A	90	110/80	15	37	12	28030	10	185	26	1	133	4	N	15	E V2-4	SR	A	ANTERIOSEPTAL	60%	A	A	A	A	I
16	DEVAPPANNAGOUDA	56	M	BU	18534/17	09-06-2018	6/15/2018	P	A	A	A	A	A	A	A	P	P	A	80	120/90	16	37	15	8950	10	199	14	1	131	4	N	28	E V2-5, I AVL	SR	A	ANTERIOSEPTAL	35%	A	A	A	A	I
17	NANAGOUDA	55	M	Fa	19443/17	6/16/2018	6/22/2018	P	A	A	A	A	A	P	A	A	A	P	130	160/106	15	37	16	16190	5	166	31	1	144	4	P	85	E V2-6, I, AVL	SR	A	ANTERIOR	40%	A	A	A	Z	I
18	SANGANNA GOUDA	41	M	Fa	19796/17	6/19/2018	6/26/2018	P	A	A	A	A	A	A	A	P	P	A	80	136/90	16	37	20	18270	5	77	20	1	140	3	P	20	E II, II, AVF	SR	A	INFERIOR	60%	A	A	A	A	I
19	KAMALA BAI	55	F	H	20558/17	6/24/2018	30-60-17	P	P	A	A	A	A	A	A	A	A	A	84	130/70	16	37	12	20970	10	180	12	1	136	3	N	33	E II, III, AVF	SR	P	INFERIOR	50%	A	A	P	A	I
20	MAHAVEER	45	M	L	20668/17	6/25/2018	02-07-2018	P	A	A	A	A	A	A	A	A	A	A	98	150/80	14	37	14	14700	20	124	20	1	140	4	P	78	E V2-6, II, III, AVF	SR	A	ANTERIOR	50%	A	A	A	A	I
21	RAYAWWA	65	F	H	25005/17	7/30/2018	04-08-2018	P	A	A	A	A	A	A	A	A	A	A	60	130/100	20	37	11	11810	20	159	21	1	132	4	P	72	E II, III, AVF	HB	A	INFERIOR	50%	A	A	A	A	I
22	PRAKASH	58	M	L	25001/17	7/31/2018	05-08-2018	P	A	A	A	A	A	A	A	P	A	A	90	140/90	16	37	7	12650	60	127	22	1	132	4	N	82	E V2-5	SR	A	ANTERIOR	45%	A	A	A	A	I
23	HANAMANTH	65	M	Fa	27519/17	8/19/2018	8/25/2018	P	A	A	A	A	A	A	A	A	A	P	84	130/80	16	37	12	11400	40	122	22	1	136	5	P	45	E V1-4, I AVL	SR	A	ANTERIOSEPTAL	40%	A	A	A	VT	I
24	NEELAKANTAYYA	78	M	Fa	27516/17	8/19/2018	8/28/2018	A	P	A	A	A	A	P	A	A	A	A	130	150/110	16	37	10	1000	90	332	30	1	139	5	P	30	E V1-3	VT	P	ANTERIOR	40%	A	P	A	VT	I
25	MALAKAWWA	50	F	H	27774/17	8/21/2018	8/28/2018	P	A	A	A	A	P	A	A	A	A	A	72	130/80	14	37	12	17710	20	169	27	1	136	0	P	300	E V2-4, I AVL	SR	A	ANTERIOR	40%	A	A	A	A	I

26	SHIVANGOUDA	64	M	Fa	11901	08-04-2018	11-04-2018	P	A	A	A	A	A	A	A	A	P	114	110/70	16	37	12	10850	80	190	20	1	129	3	P	45	E V1-V4	SR	A	ANTERIOR	50%	P	A	A	A	I
27	RAYAPPA	52	M	Fa	14773	02-05-2018	08-05-2018	A	A	P	A	A	P	P	A	A	P	A	110	80/60	22	37	12	20970	10	180	12	1	136	3	N	33	E II,III,AVF	SR	P	INFERIOR	60%	P	A	A	A	I
28	MALLAPPA	58	M	Fa	16000	12-05-2018	18-05-2018	A	A	A	A	P	A	A	A	A	A	A	70	130/80	16	37	14	9640	35	447	20	1	135	5	N	64	E V1-V4	SR	A	ANTERIOR	30%	A	A	A	A	I
29	MALLIKARJUNA	53	M	L	17016	20-05-2018	28-05-2018	A	A	A	A	P	A	P	A	A	A	A	108	130/80	12	38	13	9460	15	180	21	1	136	4	P	79	E II,III,AVF	SR	P	INFERIOR	40%	P	A	P	Z	I
30	YALLAPA	50	M	Fa	17976	28-05-2018	01-06-2018	A	P	A	A	A	P	P	A	A	A	A	82	112/80	11	37	14	14398	5	189	27	1	131	4	P	74	E V1-V4	SR	P	ANTERIOR	60%	A	A	A	A	I
31	HANAMANTH	72	M	L	18795	03-06-2018	09-06-2018	P	A	A	A	A	A	A	A	A	A	A	56	110/70	14	37	13	24770	5	500	_	1	128	4	P	113	E II,III,AVF	SR	P	INFERIOR	50%	A	P	P	A	I
32	RAJASAEB	45	M	Fa	18986	05-06-2018	05-06-2018	P	A	A	A	A	A	A	A	A	A	A	114	110/70	16	37	12	10850	80	190	20	1	129	3	P	45	E V1-V4	SR	P	ANTERIOR	45%	P	A	A	Z	I
33	MAHADEVI	60	F	H	19040	05-06-2018	11-06-2018	A	A	A	A	A	A	A	A	A	A	A	110	80/60	22	37	12	20970	10	180	12	1	136	3	N	33	E II,III,AVF	SR	A	INFERIOR	50%	A	A	P	A	I
34	MALLIKARJUNA	68	M	BU	19415	08-06-2018	13-06-2018	P	A	A	A	A	A	A	A	A	A	A	56	110/70	14	37	12	20970	80	190	20	1	137	4	P	28	E V1-V4	SR	A	ANTERIOR	40%	A	P	A	A	I
35	GANGADHAR	78	M	Fa	19674	10-06-2018	11-06-2018	P	A	A	A	A	P	P	A	A	A	A	114	110/70	16	37	14	9640	10	180	12	1	128	4	P	113	E II,III,AVF	SR	P	INFERIOR	50%	P	A	A	Z	I
36	RUKAMAWWA	85	F	H	22858	06-07-2018	14-07-2018	P	A	A	A	A	P	A	A	A	A	P	80	120/80	14	37	14	14398	15	180	21	1	135	5	N	64	E II,III,AVF	SR	A	INFERIOR	60%	A	A	A	A	I
37	NAGRAJ	22	M	Em	23519	11-07-2018	17-07-2018	A	A	A	A	A	P	A	P	A	A	A	98	140/80	14	37	16	5530	5	500	_	1	140	4	P	78	E II,III,AVF	SR	P	INFERIOR	30%	P	A	A	A	I
38	BHIMGOUDA	47	M	Em	24054	16-07-2018	19-07-2018	A	P	A	A	A	P	P	A	A	A	A	80	1224/80	14	37	20	18270	80	190	20	1	136	4	P	79	E V2-5	SR	A	ANTERIOR	35%	A	P	P	A	I
39	SUTAN	50	M	Fa	24130	17-07-2018	22-07-2018	P	A	A	A	A	A	A	A	P	P	A	80	120/70	16	37	12	10850	10	180	12	1	131	4	P	74	E II,III,AVF	SR	P	INFERIOR	40%	A	P	P	A	I
40	CHIDANAND	65	M	Fa	24481	20-07-2018	26-07-2018	P	A	A	A	A	A	A	A	A	A	A	80	140/90	16	37	12	20970	80	190	20	1	137	4	P	28	E V2-5	SR	P	ANTERIOR	60%	A	A	A	A	I
41	TUKARAM	65	M	BU	24676	21-07-2018	24-07-2018	P	A	A	A	A	A	A	A	A	A	A	40	90/60	14	37	14	9640	10	180	12	1	128	4	P	113	E V1-V4	SR	P	ANTERIOR	40%	P	P	A	Z	I
42	TULJABAI	60	F	H	26565	06-08-2018	10-08-2018	A	A	A	A	A	A	A	A	A	A	A	80	140/80	15	37	14	14700	35	447	20	2	134	6	P	53	E II,III,AVF	SR	A	INFERIOR	50%	P	A	A	A	I
43	SHIVSHANKEPPA	70	M	Fa	29189	27-08-2018	02-09-2018	A	A	P	A	A	A	A	A	A	A	A	70	120/70	16	37	7	12650	5	500	_	3	127	5	P	109	E II,III,AVF	SR	P	INFERIOR	60%	P	A	A	A	I
44	LAXMAN	45	M	Fa	29276	28-08-2018	30-08-2018	A	A	A	A	P	A	P	A	A	A	A	86	90/50	26	37	12	11400	80	190	20	1	131	4	P	74	E II,III,AVF	SR	A	INFERIOR	40%	A	A	P	A	I
45	SUBAS	70	M	Fa	29454	29-08-2018	03-09-2018	A	P	A	A	A	P	P	A	A	A	A	88	130/80	14	37	10	1000	10	180	12	1	137	4	P	28	E II,III,AVF	SR	P	INFERIOR	50%	A	P	A	A	I
46	SHANKAR	80	M	Fa	29790	31-08-2018	07-09-2018	A	P	A	A	A	A	A	A	P	A	A	60	130/100	20	37	12	17710	20	159	21	1	128	4	P	113	E V1-V4	SR	P	ANTERIOR	45%	A	A	A	A	I
47	AMBADAS	63	M	Fa	29923	02-09-2018	03-09-2018	P	A	A	A	A	A	A	A	P	P	A	90	140/90	16	37	12	20970	60	127	22	2	134	6	P	53	E II,III,AVF	SR	P	INFERIOR	40%	P	P	A	Z	I
48	SAGAR	24	M	BU	29996	03-09-2018	07-09-2018	P	A	A	A	A	A	A	A	A	A	A	130	150/110	16	37	14	14700	90	332	30	2	134	6	P	53	E V1-V4	SR	A	ANTERIOR	40%	A	A	A	A	I
49	BABU	60	M	BU	30058	03-09-2018	08-09-2018	A	A	A	A	A	A	A	A	A	A	A	100	170/90	14	37	13	9460	20	169	27	1	138	5	P	50	E II,III,AVF	SR	P	INFERIOR	50%	P	P	A	Z	I
50	KHUBA	68	M	Fa	30315	05-09-2018	09-09-2018	P	A	A	A	A	A	A	A	A	A	A	80	130/80	14	37	14	14398	10	169	20	7	136	5	P	22	E V2-5	SR	A	ANTERIOR	45%	A	A	A	A	I
51	MALLINATH	65	M	Fa	31569	14-09-2018	19-09-2018	P	A	A	A	A	P	P	A	A	A	A	70	120/70	16	37	16	5530	5	333	36	3	127	5	P	109	E V1-V4	SR	A	ANTERIOR	50%	A	A	A	A	I
52	TULISIRAM	42	M	Fa	31741	17-09-2018	18-09-2018	A	A	P	A	A	A	A	A	A	A	A	86	90/50	26	37	20	18270	15	180	21	1	131	4	P	74	E II,III,AVF	SR	P	INFERIOR	40%	P	P	P	Z	I
53	POOLANBAI	50	F	H	35214	14-10-2018	20-10-2018	P	A	A	A	A	A	A	A	A	A	A	50	110/70	18	37	12	15290	40	160	29	1	131	4	P	61	E II,III,AVF	VPC	A	INFERIOR	40%	A	A	P	A	I
54	KAJAPPA	55	M	Fa	35208	14-10-2018	15-10-2018	A	P	A	A	A	A	A	A	A	A	A	100	150/100	20	38	16	12480	10	116	35	1	132	5	P	267	E V2-V6	SR	A	ANTERIOR, SEPTUM AND APEX	40%	P	A	A	A	I
55	BHIMAPPA	65	M	Fa	35894	19-10-2018	24-10-2018	P	A	A	A	A	A	A	P	A	A	70	90/60	23	38	15	16710	25	113	73	1	131	4	6	63	E V2-V5	SR	P	ANTERIOR SEPTUM	55%	A	A	P	A	I
56	LAXMIBAI	85	F	H	508	04-01-2019	08-01-2019	P	A	A	P	A	A	A	A	A	A	A	79	180/90	16	38	16	13090	21	205	37	1	136	4	1	20	E V1-V6	SR	A	ANTERIOR WALL, ANTERIOR SEPTUM, APEX	40%	A	A	A	A	I
57	CHANDRAKANTH	61	M	BU	518	04-01-2019	12-01-2019	P	A	P	P	A	P	P	P	A	A	A	96	150/90	18	37	14	11700	37	202	52	2	133	5	18	170	E V1-V4	SR	A	ANTERIOR	35%	A	A	A	Z	I
58	CHANDRASHEKAR	46	M	Em	556	05-01-2019	10-01-2019	P	A	A	A	A	P	P	A	A	A	A	100	210/100	18	37	15	9720	10	309	20	1	131	4	0	10	E V2-V4	SR	P	ANTERIOR WALL AND SEPTUM	45%	P	A	A	A	I
59	NINGANGOUDA	52	M	Fa	1543	14-01-2019	20-01-2019	A	A	A	P	A	A	A	A	A	A	A	84	120/80	18	38	14	9660	25	124	28	1	135	5	1	30	E II, III, AVF	SR	A	INFERIOR	40%	P	A	A	A	I
60	SUBHASHGOWDA	65	M	BU	1662	15-01-2019	20-01-2019	P	A	A	A	A	A	A	A	P	P	A	62	140/100	19	37	16	11200	5	112	40	1	140	4	1	54	E II, III, AVF	SR	P	INFERIOLATERAL AND LATERAL	55%	A	A	A	A	I
61	SITARAM	62	M	Em	3065	28-01-2019	04-02-2019	A	P	A	A	A	P	P	P	P	A	P	78	130/80	18	38	11	16320	15	319	25	1	136	4	##	68	E V3-V5	SR	P	APICAL SEPTUM AND ANTERION SEPTU	45%	A	A	A	A	I

61	SITARAM	62	M	Em	3065	28-01-2019	04-02-2019	A	P	A	A	A	P	P	P	A	P	78	130/80	18	38	11	16320	15	319	25	1	136	4	##	68	E V3-V5	SR	P	APICAL SEPTUM AND ANTERION SEPTU	45%	A	A	A	A	I
62	IRAPPA	55	M	Fa	3599	01-02-2019	06-02-2019	P	A	A	A	A	A	A	P	P	A	82	110/84	14	37	16	13850	15	81	30	1	131	4	10	36	E V2-V6	SR	A	ANTERIO-LATERAL	60%	A	A	A	A	I
63	SUKHADEV BHIMANNA HONAKATTI	63	M	BU	5336	07-02-2019	12-02-2019	P	A	A	P	A	A	A	A	P	P	A	68	100/70	18	38	13	12347	25	124	23	1	132	4, 8	20	75	E 11, 111, AVF	SR	A	INFERIOR AND INFERIO-LATERAL WALL WITH GRADE I MR	30%	P	A	A	A	I
64	SAIBANNA	70	M	Fa	4701	11-02-2019	12-02-2019	P	P	A	A	A	P	A	A	P	P	A	80	120/80	18	38	12	11860	25	329	45	2	133	6	19	104	E V2-V6	SR	P	ANTERIOR, SEPTAL ANDLATERAL	45%	P	A	P	Z	I
65	PARASAPPA	64	M	BU	4599	11-02-2019	16-02-2019	P	A	A	A	P	P	P	A	A	P	113	120/70	18	38	16	11300	10	243	40	1	136	5	62	105	E V2-V6	SR	P	ANTERIOR AND LATERAL	35%	P	A	P	Z	I
66	RAMAGOUDA	65	M	Fa	5289	16-02-2019	17-02-2019	P	A	A	A	A	A	A	A	A	A	84	110/70	20	37	14	20340	5	134	44	2	138	5	80	127	E II,III,AVF	SR	P	INFERIOR	40%	A	A	A	A	I
67	MALLAPPA	48	M	Fa	5334	17-02-2019	23-02-2019	P	A	A	A	P	A	A	A	A	A	100	140/100	22	38	15	12990	15	94	44	1	136	5	0	23	E II, III, AVF	SR	P	INFERIOR, LATERAL	40%	P	A	A	A	I
68	SAYAWWA	65	F	H	5311	17-02-2019	18-02-2019	P	A	A	A	A	A	P	A	A	A	A	120	170/100	24	38	13	13230	5	88	24	1	139	4	13	79	E II,III,AVF	SR	A	INFERIOR	40%	A	A	A	A	I
69	MUTTAPPA	50	M	BU	6158	25-02-2019	04-03-2019	P	P	A	P	A	A	A	P	P	P	A	90	120/90	22	39	15	16850	7	327	60	1	140	5	##	300	E II,III,AVF	SR	P	INFERIOR, LATERAL	45%	P	A	A	A	I
70	SHARANAWWA	72	F	H	8305	16-03-2019	21-03-2019	P	A	A	A	A	P	P	P	A	A	A	80	110/70	18	38	11	13450	55	143	36	1	137	5	24	65	E II,III,AVF	SR	P	INFERIOR, INFERIOLATERAL	50%	A	A	A	MR	I
71	S. CROSSWIN	50	M	BU	9660	28-03-2019	03-04-2019	P	A	A	A	A	P	P	P	A	A	A	60	90/70	19	37	14	18730	40	203	39	1	135	5	P	20	E V2-V5	SR	A	INFERIOR,INFERO-LATERAL, ANTERO-LATERAL	40%	A	A	A	A	I
72	BHIMANGOUDA	40	M	Fa	9761	29-03-2019	03-04-2019	P	A	A	A	A	A	A	A	A	A	84	110/70	22	39	14	10030	40	87	33	1	138	5	3	22	E V2-V6	SR	A	LATERAL	55%	A	A	A	A	I
73	BUDDAWWA	65	F	H	12913	25-04-2019	26-04-2019	P	A	P	A	A	P	A	A	A	A	P	90	130/80	14	97	10	13180	10	212	30	1	132	4	##	300	E II, III, AVF	SR	P	GLOBAL HYPOKINESIA	30%	A	P	P	Z	I
74	RUIDRAGOWDA SANGANGOWDA PATIL	50	M	Em	12848	25-04-2019	27-04-2019	A	P	A	P	A	A	P	A	A	P	P	90	170/90	14	98	15	7410	20	86	22	1	141	4	40	29	V2, V3, V4. V5	SR	A	APICAL SEPTUM AND ANTERION SEPTU	45%	A	A	A	A	I
75	MUDAKAPPA	55	M	Fa	14123	06-05-2019	09-05-2019	P	P	A	A	A	A	A	P	P	A	130	NR	30	38	51	14460	51	212	19	1	140	4	9	33	E II, III, AVF	SR	P	INFERIOR, INFERIOLATERAL, SEPTUM, APEX	35%	P	P	P	Z	I
76	CHANDRABAGA SHANKAR DESAI	78	M	BU	15050	13-05-2019	15-05-2019	P	A	A	P	A	A	P	P	P	P	A	72	150/90	16	38	11	8390	40	166	34	1	132	4	0	10	E II, III, AVF	SR	P	INERIOR AND INFERIOLATERAL WALL	50%	A	P	A	Z	I
77	JATINAGARAYA	86	M	Fa	15113	14-05-2019	15-05-2019	A	P	P	A	A	P	P	P	P	A	A	90	80/50	16	38	15	7900	35	119	45	1	141	3, 8	0	13	LBBB	SR	A	ASYNCHRONUS MOVEMENT OF LV IN VIEW OF LBBB	50%	A	P	P	A	E
78	SHIVAPPA	60	M	BU	15189	14-05-2019	17-05-2019	P	P	A	A	A	P	P	P	P	P	A	NR	NR	16	38	12	13340	40	148	23	1	124	3	24	120	E V2, V3, V4, V5, V6	SR	P	ANTERIOR WALL	30%	P	A	P	A	I
79	SADASHIVA DUNDAPPA	40	M	Fa	15265	15-05-2019	20-05-2019	A	P	P	A	A	A	A	A	A	A	A	78	110/80	14	37	14	15410	20	81	40	1	135	4	0	24	D IN V2-V4	SR	A	RHD, GLOBAL HYPOKINESIUA, LV CLOT	20%	P	A	A	RHD	E
80	BHILA SHENPADU	47	M	L	15332	15-05-2019	21-05-2019	P	A	A	A	A	A	A	P	A	P	82	160/90	15	38	16	14730	30	153	23	1	141	4	2	78	E IN V2, V3. V4	SR	P	ANTERIOR AWALL AND SEPTUM	40%	A	P	P	A	I
81	MEENAKSHI MADIWallappa	57	F	H	15619	17-05-2019	23-05-2019	P	A	A	A	A	P	A	P	A	A	A	70	170/90	14	39	13	21110	5	370	24	1	140	4	0	40	E V2, V3, V4, V5, V6	SR	P	GLOBAL HYPOKONESIA	35%	P	A	A	A	I
82	RAJUGOWDA BIRADAR	43	M	BU	15491	17-05-2019	23-05-2019	P	A	A	A	A	P	A	P	P	A	A	94	140/90	14	38	11	4970	30	229	30	1	136	3, 6	0	23	E V2, V3, V4, V5, V6	SR	P	APICAL SEPTUM AND ANTERION SEPTU	40%	P	A	A	A	I
83	VITTAL BHIMAPPA	70	M	L	15565	17-05-2019	22-05-2019	P	P	A	A	A	A	P	P	P	P	P	96	190/110	15	39	14	14240	25	94	36	1	132	5	2	48	E II, III, AVF	SR	P	INFERIOR AND INFERIO-LATERAL WALL WITH GRADE I MR	40%	P	A	A	A	I

84	BHIMASHANKAR	75	M	BU	15840	20-05-2019	26-05-2019	P	P	A	A	A	A	A	A	A	A	A	65	140/80	18	37	13	14790	10	99	52	2	136	4	25	190	E II, III, AVF	SR	P	INFERIOR AND INFERIO-LATERAL WALL WITH GRADE I MR	50%	A	A	P	VT	I
85	RAMACHANDRA SHIVU HAKKI	70	M	Fa	16239	23-05-2019	27-05-2019	A	P	A	A	A	A	A	A	A	A	P	90	150/90	14	38	12	8730	40	174	56	1	143	4, 1	1	8	LBBB WITH VPCS	IR	A	INFERIOR AND INFERIO-LATERAL WALL WITH GRADE I MR	30%	P	A	A	LBBB	E
86	SHIVAPPA SAGAGI	45	M	BU	16458	24-05-2019	27-05-2019	P	P	A	A	A	P	P	P	P	A	A	120	140/90	14	38	8	9830	95	59	45	1	140	5	0	21	E V2, V3, V4, V5, V6	SR	A	DCM WITH GLOBAL HYPOKINESIA	30%	P	A	A	A	I
87	RAMANNA UPPAR	65	M	Fa	16635	26-05-2019	30-05-2019	P	A	A	A	A	A	A	A	A	A	A	80	130/80	16	37	17	9680	10	124	42	1	145	4	1	35	E V1, V2, V3, V4, V5	SR	P	ANTERIOR WAL;L SEPTUM AND APEX	40%	P	P	A	A	
88	SHANKREPPA	63	M	Fa	16603	26-05-2019	30-05-2019	P	P	A	A	A	A	A	A	A	A	A	68	130/90	22	38	14	12040	10	106	21	1	143	4	0	19	V1, V2, V3, V4, V5, V6	VT	P	ANTERIOR, LATERAL AND APICAL WALL	30%	P	A	A	A	I
89	BASALINGAPPA GOWDA	87	M	Fa	17487	02-06-2019	06-06-2019	P	P	A	A	A	A	A	A	A	A	P	80	110/70	13	37	12	13880	##	71	88	3	134	6	1	20	E V1, V2, V3	SR	A	ANTERIOR WALL AND SEPTUM	40%	A	A	A	A	I
90	KALLAWWA	75	F	H	17714	04-06-2019	08-06-2019	P	P	A	A	A	P	P	A	A	A	A	116	110/80	12	38	8	11560	84	108	86	7	133	4	26	7	E II, III, AVF	SR	A	INFERIOR AND INFERIO-LATERAL WALL WITH GRADE I MR	40%	A	A	A	A	I
91	SURESH BIRADAR	40	M	BU	18117	07-06-2019	12-06-2019	P	P	A	A	A	P	P	A	A	A	P	82	180/110	14	38	13	7850	28	183	45	1	132	5	2	57	E V1, V2, V3, V4, V5	SR	P	ANTERIOR WALL AND SEPTUM	40%	P	P	A	A	I
92	BASAVANT AGWAR	82	M	Fa	18362	09-06-2019	14-06-2019	A	A	A	A	P	P	P	A	A	A	P	104	100/70	15	38	15	8470	15	120	30	1	132	5	0	30	E V3, V 4, V5	SR	A	SEVERE PAH	55%	A	A	A	A	I
93	GIRIMALLAYYA APPAYYA	70	M	BU	18330	09-06-2019	13-06-2019	A	A	P	A	A	P	P	A	P	P	A	50	70/50	22	39	12	13702	21	117	45	1	138	4	42	300	E II, III, AVF	SR	P	INFERIOR AND INFERIO-LATERAL WALL WITH GRADE I MR	45%	P	P	A	A	I
94	PANDURANG RAMANNA	52	M	Em	18352	09-06-2019	14-06-2019	P	P	A	A	A	A	P	A	P	P	A	100	140/100	18	39	5	9960	12	132	25	1	135	4	0	11	E V2, V3, V4, V5, V6	SR	A	ANTERIOR WALL	55%	A	A	A	A	E
95	SIDRAYA KOLI	70	M	L	18366	10-06-2019	15-06-2019	A	P	A	A	A	A	A	A	A	P	A	96	120/70	14	38	16	13780	20	109	17	1	135	3	0	19	E V2, V3	SR	A	INFERIOLATERAL	50%	A	A	A	A	I
96	GURUSANGAPPA SHIVAPPA	70	M	Fa	18369	10-06-2019	14-06-2019	A	P	A	A	A	P	P	A	P	P	P	54	230/60	15	38	10	`15830	56	97	34	1	137	5	0	6	E V2, V3, V4, V5, V6	SR	A	CONCENTRIC LVH WITH GRADE 111 AR	55%	A	A	A	A	E
97	BHEEMASHANKAR	55	M	Fa	18649	11-06-2019	17-06-2019	A	P	P	A	A	P	P	A	P	P	A	144	130/90	12	38	15	13370	40	163	82	2	135	6	0	14	E V3, V 4, V5	IR	A	GLOBAL HYPOKINESIA,RHD,GRADE II AR	50%	A	A	A	AF	I
98	GURUAPPA	85	M	BU	18798	13-06-2019	18-06-2019	P	P	A	A	A	A	P	A	P	A	A	90	140/80	14	38	10	18620	##	249	22	2	137	6	0	9	E V1,V2,V3,V4	SR	P	ANTERIOR WALL	40%	P	P	A	A	I
99	BASAYYA HIREMATH	40	M	Fa	18971	14-06-2019	17-06-2019	A	A	P	A	A	P	P	A	P	P	A	108	80/60	18	38	12	13730	35	512	40	2	123	4	7	124	E II,III,AVF	SR	P	INFERIOR WALL	30%	P	A	A	A	I
100	JAKKAWWA MARPANGI	67	F	H	19016	14-06-2019	19-06-2019	A	A	P	A	A	A	P	A	A	A	A	88	80/60	12	38	13	9020	6	130	20	1	134	3	5	70	E V2,V3,V4,V5	SR	A	GLOBAL HYPOKINESIA	30%	A	A	P	A	I
101	SANJAY BIRADAR	32	M	BU	19021	15-06-2019	20-06-2019	A	A	A	P	P	A	A	P	P	P	A	130	140/90	18	38	15	13760	6	137	32	1	134	3	0	40	E V2,V3,V4,V5	SR	A	NRMAL LV /30 FUNCTION	60%	A	A	A	A	I
102	JATAPPA HITTANDLI	60	M	Fa	19054	15-06-2019	20-06-2019	A	P	P	A	A	P	P	A	P	A	P	74	130/80	12	39	7	7010	56	98	22	1	138	5	2	48	E V2,V3,V4,V5	SR	A	ANTERIOR WALL	40%	A	P	A	A	I
103	BAPURAI DESHPANDE	56	M	L	19123	16-06-2019	22-06-2019	P	A	P	A	A	A	P	A	P	P	A	102	110/70	14	38	10	13340	31	126	13	1	129	5	80	292	E II,III,AVF	SR	P	INFERIOR	40%	A	P	A	A	I
104	DUNDAPPA CHOUDHAR	82	M	L	19165	16-06-2019	20-06-2019	A	A	P	P	A	P	P	P	P	P	A	120	80/60	18	38	13	9720	57	74	16	1	132	4	0	5	E V2,V3,V4,V5	SR	P	ANTERIOR	40%	A	A	P	A	I
105	BHEEMSINGH HAJERI	71	M	Fa	19168	16-06-2019	22-06-2019	P	P	A	A	A	A	A	A	A	A	A	90	116/80	14	38	10	8570	36	139	40	1	129	4	0	3	E V1,V2,V3,V4	SR	P	GLOBAL HYPOKINESIA	30%	P	A	A	A	I

106	BASANGAUDA SEVADHI	48	M	L	19130	16-06-2019	22-06-2019	P	P	A	A	A	P	P	P	P	P	A	94	130/90	18	38	14	12340	50	220	14	1	129	4	80	118	E V2,V3,V4,V5	SR	P	ANTERIOR WALL AND SEPTUM	40%	P	P	A	A	I
107	SAHIBGAUDA GUNAPUR	60	M	Fa	19181	17-06-2019	22-06-2019	P	P	A	A	A	P	P	A	P	P	A	80	110/70	12	38	18	13540	24	210	15	1	136	6	1	12	E V2,V3,V4,V5, V6	SR	P	ANTERIOR WALL SEPTUM AND APEX	45%	A	A	P	A	I
108	LOKKAMMA MANDEWALA	70	F	H	19205	17-06-2019	22-06-2019	P	A	A	A	A	A	A	A	A	A	A	90	130/80	12	38	12	6110	24	120	30	1	132	4	13	30	E V2,V3,V4	SR	A	ANTERIOR WALL	30%	P	A	A	A	I
109	SHABBIR AHMED	48	M	L	20857	01-07-2019	07-07-2019	P	P	A	A	A	P	P	P	A	A	120	80/60	14	38	12	12040	6	214	30	1	134	5	0	15	E II,III,AVF	SR	P	INFERIOR WALL	30%	P	P	P	A	I
110	LAKSHMI DESAI	50	F	H	21854	09-07-2019	12-07-2019	P	P	A	A	A	P	P	P	A	A	A	80	70/50	22	39	0	14680	37	242	34	1	133	4	12	9	E V1,V2,V3,V4, V5,V6	SR	P	ANTERIOR AND ANTERO LATERAL WALL	25%	P	P	P	V T	I
111	SHANTABAI PUJAR	68	F	H	21741	08-07-2019	14-07-2019	P	A	A	A	A	P	P	P	A	A	A	84	110/70	12	38	11	10610	79	383	44	1	133	5	0	70	E II,III,AVF	SR	A	INFERIOR WALL	45%	A	A	A	A	I
112	MALLIKAARJUN KUNTOJI	55	M	L	22600	14-06-2019	20-06-2019	P	P	A	A	A	P	P	A	A	P	P	80	210/110	12	38	16	14380	10	407	12	1	131	4	8	300	E V1,V2,V3	SR	A	ANTERIOR WALL,SEPTUM,APEX	40%	A	A	A	A	I
113	BANGAREWWA BIRADAR	80	F	H	22806	15-07-2019	20-07-2019	A	A	P	P	A	A	A	P	A	A	P	80	70/30	18	38	11	14780	45	135	72	1	133	5	10	51	E II,III,AVF	SR	P	INFERIOIR WALL,INFEROLATERAL WALL	25%	P	P	P	A	I
114	NELLAMA KATTIMANI	56	F	H	23083	17-07-2019	27-07-2019	P	P	A	A	A	P	P	P	A	A	P	100	110/80	12	38	10	7620	28	130	28	1	138	5	4	111	D II,II,AVR	SR	A	INFERIOIR WALL,INFEROLATERAL WALL	45%	A	A	A	A	E
115	RANABAI RATHOD	55	F	H	23151	17-07-2019	22-07-2019	P	P	A	A	A	P	P	A	A	A	A	80	110/80	12	37	9	14340	48	135	28	1	137	4	20	19	E II,III,AVF	SR	P	INFERIOER WALL	40%	P	P	A	A	I
116	SHARANAPPA MADAR	63	M	BU	232922	18-07-2019	26-07-2019	P	P	A	P	A	A	P	P	A	P	A	A	100	150/80	14	38	14	9750	45	104	20	1	135	4	22	73	E V1,V2,V3,V4, V5,V6	SR	P	HYPOKINESIA OF ANTERIOR WALL SEPTUM AND APEX	45%	A	A	A	A	I
117	SANGITA SHREDHAR	36	F	H	29013	02-09-2019	07-09-2019	p	p	A	A	A	A	A	P	A	A	A	88	120/70	14	38	11	8750	54	99	20	1	127	4	12	21	E II,II,AVF	SR	P	INFERIOR AND INFERIO-LATERAL WALL WITH GRADE I MR	30%	P	P	A	A	I
118	PARASSAPA HONAKERA	52	M	L	4599	02-11-2019	15-011-2019	P	P	A	A	A	P	P	P	P	A	A	80	110/70	18	38	16	11300	10	243	88	2	136	5	62	105	E V2,V3,V4,V5	SR	P	ANTERO LATERAL WALL	30%	P	P	A	A	I
119	SUNANDA NAVADAG	48	F	H	30179	09-11-2019	14-11-2019	P	P	A	A	A	P	P	P	A	A	P	80	110/70	12	37	12	13900	83	410	30	1	130	4	1	26	E V3,V4,V5,V6	SR	P	ANTERIOR WALL	30%	P	P	A	A	I
120	ANNASAHAB HOSATI	52	M	Fa	37723	11-11-2019	16-11-2019	P	P	A	P	P	P	P	P	P	A	A	80	110/70	12	38	14	16220	40	208	27	1	135	4	0	105	E V1,V2,V3,V4, V5,V6	SR	P	ANTERIOR WALL SEPTAL AND LATERAL WALL	25%	P	P	A	A	I
121	IRAPPA CHALAWADI	65	M	Fa	3599	01-02-2020	06-02-2020	P	P	A	A	A	A	A	P	A	A	102	140/80	21	38	16	13850	15	81	45	1	131	4	10	36	E V1, V2, V3, V4, V5	SR	P	ANTERIOR AND SEPTAL WALL	40%	A	A	A	A	I
122	PRAKASH SHARANAPPA	60	M	BU	4443	05-02-2020	10-02-2020	P	P	A	A	A	A	A	A	A	P	90	80/60	22	38	12	14270	29	134	32	1	139	5	0	13	E V1, V2, V3, V4, V5 , V6	SR	P	ANTERIOR AND SEPTAL WALL	30%	P	A	P	A	I
123	NAGAPPA AVADI	73	M	BU	8350	06-03-2020	09-03-2020	P	A	A	A	A	P	P	A	P	P	A	80	80/60	18	38	14	9950	10	279	29	1	134	4	23	70	E V1, V2, V3, V4, V5, V6	SR	P	ANTERIOR AND SEPTAL WALL	25%	P	A	P	Z	I
124	NINGAMMA HATTI	52	F	H	9951	19-03-2020	23-03-2020	P	P	A	A	A	P	A	P	A	A	P	79	100/70	22	38	11	10410	50	279	16	1	129	4	0	24	E II, III, AVF	SR	P	INFERIOR WALL	35%	A	P	A	A	I
125	RAMESH ANKALGI	28	M	Fa	9397	16-03-2020	20-03-2020	P	P	A	A	A	P	P	P	P	A	98	80/60	23	38	12	12345	60	145	56	2	134	4	6	76	E V1, V2, V3, V4, V5	SR	P	ANTERIOR AND SEPTAL WALL	30%	P	P	P	Z	I

126	BASAPPA PUJARI	45	M	BU	10204	22-03-2020	27-03-2020	P	P	A	A	A	P	P	P	P	A	A	78	150/10 0	18	38	19	15480	10	251	22	1	136	4	14	214	E V2, V3, V4, V5	SR	P	ANTERIOR WALL,SEPTAL RUPTURE	30%	P	A	A	A	I
127	LAGAMANNA BASAPPA	55	M	Fa	10511	29-03-2020	02-04-2020	P	A	A	A	A	A	A	A	A	A	A	102	80/60	19	38	13	9300	25	149	34	1	124	4	18	300	E V2, V3, V4, V5	SR	A	ANTERIOR ,SEPTUM AND DISTAL REGION	37%	A	A	P	A	I
128	BASAPPA KOKATNUR	48	M	Fa	10778	03-04-2020	04-04-2020	A	A	A P	P	P	A	P	P	A	P	A	76	100/70	20	38	15	14270	10	265	14	1	133	4	0	16	E II, III, AVF	SR	P	INFERIOR AND APICAL WALL	40%	A	A	A	A	I
129	BHIMARAYA SIDDAPPA UKKALI	56	M	L	12391	05-04-2020	07-04-2020	P	P	A	A	A	P	P	P	P	P	A	102	80/40	22	38	10	5630	##	143	34	1	136	5	4	56	E V2, V3, V4, V5, V6	SR	P	ANTERIOR AND SEPTAL WALL	30%	P	P	P	Z	I
130	DANAYYA	52	M	Fa	11244	12-04-2020	17-04-2020	A	A	P	A	A	P	A	A	A	A	P	65	100/70	16	38	13	12010	10	234	34	1	135	5	80	245	E II, III, AVF	SR	P	INFERIOR	45%	A	P	A	Z	I
131	YUNUS HUSSAINSAB MORTUR	25	M	L	11238	12-04-2020	18-04-2020	P	P	A	A	A	A	A	P	A	A	P	65	110/70	15	38	15	8110	36	213	32	1	134	5	1	35	E V4, V5, V6	SR	A	ANTERIOR	45%	A	A	A	A	I
132	ALLAMMA NADAF	65	F	H	11613	17-04-2020	22-04-2020	P	P	A	A	P	P	P	P	A	A	P	89	80/60	22	38	14	34260	10	375	34	1	138	4	5	76	E V1, V2, V3, V4, V5, V6	SR	P	GLOBAL	25%	P	P	P	Z	I
133	NINGU YALAMELI	50	M	BU	11675	18-04-2020	24-04-2020	A	P	A	A	A	P	P	P	P	P	A	120	70 /30	19	39	16	17100	5	132	23	1	138	5	16	221	E LEAD I, AVL, V1,V2,V3,V4,	SR	P	GLOBAL HYPOKINESIA	20%	P	P	P	Z	I
134	IRAPPA SARWARD	54	M	L	11669	18-04-2020	25-04-2020	P	P	A	A	A	A	A	A	A	A	P	76	110/70	14	38	14	13040	30	211	34	1	136	4	0	10	E V2, V3, V4	SR	A	SEPTAL AND ANTEROR	45%	A	A	A	A	I
135	RUDRAYYA HIREMATH	50	M	Fa	11805	20-04-2020	23-04-2020	P	P	P	P	A	P	P	P	P	P	A	78	110/60	14	38	12	10730	10	270	29	1	136	4	17	139	E V2, V3, V4, V5, V6	SR	P	ANTERIOR	30%	P	P	A	A	I
136	DUSANGAPPA KARIGAR	67	M	Fa	12184	28-04-2020	01-05-2020	P	A	A	A	A	A	A	P	P	A	A	98	80 /30	12	38	15	9880	60	115	38	1	144	4	2	54	E V2, V3, V4	SR	P	ANTERIOR	35%	P	P	P	Z	I
137	DRAKSHAYINI	60	F	H	12217	29-04-2020	29-04-2020	P	P	P	A	A	P	P	P	A	A	A	118	80/60	19	39	14	12456	70	167	54	1	126	4	7	67	E V2, V3, V4, V5, V6	SR	P	GLOBAL	30%	P	A	P	Z	I
138	MALLESHAPPA	58	M	Fa	13493	30-04-2020	02-05-2020	A	P	P	A	P	P	P	A	P	A	P	98	90/70	13	38	12	14356	54	176	34	1	143	4	6	45	E IN V2, V3, V4, V5	SR	P	ANTERIOR	45%	A	A	P	V T	I
139	KAMALABAI PATIL	88	F	H	12262	30-04-2020	05-05-2020	P	P	P	P	A	A	A	A	A	A	A	78	110/80	18	39	14	10987	15	156	25	1	135	4	1	35	D IN V2-V4	SR	A	NRMAL LV /30 FUNCTION	50%	A	A	A	A	E
140	SARADAR IMAMSAB MOGAL	68	M	BU	12308	01-05-2020	04-05-2020	A	P	A	A	A	P	A	A	P	A	A	108	100/70	17	38	13	16012	10	221	21	1	137	4	43	167	E II,III, AVF	SR	P	INFERIOR WALL	35%	A	A	P	A	I
141	MALLIKARJUN SANGAPPA	56	M	L	12303	01-05-2020	06-05-2020	P	A	A	A	A	P	A	A	A	A	P	89	100/70	14	38	11	6660	60	118	25	1	147	3	3	48	E V2, V3, V4	SR	P	LATERAL	45%	A	A	A	A	I
142	BANGAREWWA GUNDAL	60	M	Fa	12389	03-05-2020	07-03-2020	P	P	A	A	A	A	P	A	A	A	A	84	110/70	18	38	13	6900	20	167	34	1	145	3	5	34	E V2, V3. V4	SR	A	SEPTAL AND LATERAL	50%	A	A	A	A	I
143	PARASHURAM SOMANING PUJARI	32	M	BU	12998	15-05-2020	19-05-2020	P	P	A	A	A	A	A	A	A	P	A	79	110/80	17	39	9	9040	##	143	20	1	137	5	5	11	E V2, V3, BV4, V5	SR	P	ANTERIOR	35%	A	P	A	A	I
144	HANUMANTRAY JALAWADI	56	M	Fa	13003	15-05-2020	18-05-2020	A	P	P	P	A	A	P	A	P	P	A	107	90/60	15	38	16	16520	40	188	34	1	134	4	11	123	E V2, V3, V4, V5, V6	SR	P	ANTERIOR AND SEPTAL WALL	35%	A	P	P	A	I
145	MAHADEVI MALLADI	56	F	H	13014	16-05-2020	19-05-2020	P	A	A	A	A	A	A	A	A	A	A	76	100/76	15	38	6	9970	75	140	34	1	137	5	4	45	E II, III, AVF	SR	P	INFERIOR	45%	A	A	P	A	I
146	HU+B148:AI148S ANSAB KAJASAB	34	M	BU	13372	22-05-2020	28-05-2020	P	A	A	A	A	A	A	P	P	A	P	98	104/68	16	38	14	10550	40	350	34	1	145	4	0	23	E V1, V2, V3, V4, V5, V6	SR	A	ANTERIOR	50%	A	P	A	A	I
147	BABU NATIKAR	45	M	Fa	13678	27-05-2020	29-05-2020	P	P	A	A	A	A	A	A	P	A	P	87	90/60	16	38	17	21010	5	154	34	1	135	6	0	30	E II, III, AVF	SR	P	INFERIOR	35%	P	P	P	Z	I
148	SHASHIDHAR SHETTI	54	M	Fa	13784	28-05-2020	02-06-2020	P	P	A S	A	A	A	A	A	P	P	A	98	100/70	14	38	17	16570	5	625	29	2	125	6	2	65	E II, III, AVF	SR	P	INFERIOR	40%	A	P	P	Z	I
149	NIJLINGAPPA	54	M	BU	13788	29-05-2020	05-06-2020	P	P	A	A	A	A	A	A	A	P	A	79	110/80	17	39	9	9040	##	143	20	1	137	5	5	11	E V2, V3, BV4, V5	SR	P	ANTERIOR WALL	35%	A	P	A	M R	E
150	BHAIRAVA MANAGI	56	M	Fa	13798	29-05-2020	07-06-2020	A	P	P	P	A	A	P	A	P	P	A	107	90/60	15	38	16	16520	40	188	34	1	134	4	11	123	E V2, V3, V4, V5, V6	SR	P	ANTERIOR AND SEPTAL WALL	35%	A	P	P	A	I
151	MAHADEVI	56	F	H	13829	02-06-2020	10-06-2020	P	A	A	A	A	A	A	A	A	A	A	76	100/76	15	38	6	9970	75	140	34	1	137	5	4	45	E II, III, AVF	SR	P	INFERIOR	45%	A	A	P	A	I
152	RAMAYYA YALAMELI	50	M	BU	13849	04-06-2020	12-06-2020	A	P	A	A	A	P	P	P	P	P	A	120	70/30	19	39	16	17100	5	132	23	1	138	5	16	221	E LEAD I, AVL, V1,V2,V3,V4,	SR	P	GLOBAL HYPOKINESIA	20%	P	P	P	Z	I