"POSITIVE T-WAVE IN LEAD aVR AS A PREDICTOR OF INHOSPITAL OUTCOME OF THE MAJOR ADVERSE CARDIAC EVENTS IN PATIENTS WITH ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION"

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

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

DOCTOR OF MEDICINE

IN

GENERAL MEDICINE

Under the guidance of

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Professor and HOD Department of GENERAL MEDICINE

BLDE (DEEMED TO BE UNIVERSITY)

SHRI B.M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH

CENTRE, VIJAYAPURA, KARNATAKA.

2019

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TABLE OF CONTENTS

S.NO	TOPIC	P.NO

LIST OF FIGURES

LIST OF TABLES

LIST OF GRAPHS

LIST OF ABBREVIATIONS USED

ACS	:	ACUTE CORONARY SYNDROME
CS	:	CARDIOGENIC SHOCK
ECG	:	ELECTROCARDIOGRAPHY
HF	:	HEART FAILURE
LA	:	LEFT ATRIUM
LAD	:	LEFT ANTERIOR DESCENDING ARTERY
LCX	:	LEFT CIRCUMFLEX ARTERY
LCA	:	LEFT CORONARY 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
VT	:	VENTRICULAR TACHYCARDIA
UA	:	UNSTABLE ANGINA
IHD	:	ISCHEMIC HEART DISEASE
PCI	:	PERCUTANEOUS CORONARY INTERVENTION
CABG	:	CORONARY ARTERY BYPASS GRAFT
MACE	:	MAJOR ADVERSE CARDIAC EVENTS

ABSTRACT

BACKGROUND:

Acute coronary syndrome (ACS), in particular, continues to be the leading cause of death worldwide. ST-segment elevation myocardial infarction (STEMI) is a serious and morbid ischemic heart condition that puts the patient's lives in danger. In-hospital significant major adverse cardiac events (MACE) and in-hospital mortality are more common in STEMI patients. Electrocardiography (ECG) is a standard aspect of any cardiac patient's examination.

For these reasons, early detection of in hospital major adverse cardiac events using ECG and starting prevention strategies is an important aspect in the treatment of STEMI.

AIMS AND OBJECTIVE

This study is done to analyze positive T wave in lead aVR 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 98 patients admitted in ICCU of BLDE (Deemed to be university), Shri B.M. Patil Medical College Hospital and Research Centre, with a diagnosis of ST-segment elevation myocardial infarction considering the inclusion and exclusion criteria. 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 positive T wave in lead aVR as Group A (n=34), and Group B (n=62) with negative T wave in lead aVR. 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 98 patients were studied out of which two were excluded based on exclusion criteria where one patient had previous history of ischemic heart disease and the other patient had left bundle branch block. Hence 96 patients were included in this study and were classified into group A and group B. In group A most common age group was 51-60 years with male predominance. Risk factor like smoking had strong association with group A. In group A, major adverse cardiac events like pulmonary edema were (67.6%, p value of 0.01), heart failure (58.8%, p value of 0.02), cardiogenic shock (52.9%, p value of 0.65), arrhythmia (20%, p value of 0.04) and death (14.7%, p value of 0.46) were seen compared to group B where pulmonary edema (38.7%), heart failure (33.8%), cardiogenic shock (46.7%), arrhythmias (4.9%) and death of (9.6%) was seen. The incidence of major adverse cardiac events was higher in group A compared to group B. The incidence of reduced ejection fraction in group A was (67.7%) which was higher when compared to group B (59.6%). The most common major adverse cardiac event was pulmonary edema followed by heart failure, cardiogenic shock, arrhythmias and death.

CONCLUSION:

In this study Positive T wave in lead aVR 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 positive T wave in lead aVR on a 12 lead ECG is cost-effective, reliable, and non-invasive predictor of major adverse cardiac events in patients with ST-segment elevation myocardial infarction.

KEYWORDS:

Lead aVR, Major adverse cardiac events, Positive T wave, ST-Segment elevation myocardial infarction.

INTRODUCTION

INTRODUCTION

Acute coronary syndrome is associated with extremely high death rates. Therefore, it is critical to identify people with this condition as early as possible. Only 22% of individuals with chest discomfort who visit an emergency cardiology clinic have coronary artery disease.[1] Acute coronary syndrome (ACS), is the leading cause of disability and death in both developed and developing countries, as well as a substantial economic burden. A wide range of risk factors are linked to the formation and progression of ACS, the most common, serious, chronic, and life-threatening disease.[2]

India is home to the world's second-largest population and is a land diverse in geography, race, culture, literacy, infrastructure, and economic variety. All of these issues make ACS management extremely difficult. According to World health organization (WHO) data, the frequency of ACS continues to rise in India, indicating a rapid epidemiological transition.[3] ACS has already surpassed communicable disease as the leading cause of death in India, with over three million STEMIs reported each year.[4]

Unstable Angina (UA), Non-ST-segment elevation myocardial infarction (NSTEMI), and ST segment elevation myocardial infarction (STEMI) are all types of acute chest pain caused by myocardial ischemia.[5] The only difference between unstable angina and NSTEMI is that cardiac enzymes are not elevated. Although the pathophysiology of all three disorders leads to an oxygen demand and supply imbalance, STEMI involves total occlusion by intra coronary thrombus, whereas unstable angina and NSTEMI only have partial occlusion.[6]

Many predisposing risk factors for ACS have been identified, including non-modifiable risk factors like age, sex, ethnicity, family history, genetic factors, and

modifiable risk factors including hypertension, diabetes, smoking/tobacco use, obesity, and diet.[7] Heart failure, cardiogenic shock, pulmonary edema, arrhythmias, and re-infarction are all examples of significant adverse cardiac events.[8] To avoid such consequences, it is critical to predict them in patients who are at risk and manage them effectively.

Electrocardiography, introduced in 1902 by Dutch physician, William Einthoven provided information about the electrophysiology of the heart. Electrocardiography marked the beginning of a new era in which numerous machines and technical methods gradually replaced the physician's unassisted senses and the stethoscope as the principal tool for cardiac diagnosis. Electrocardiography is now considered an important and common aspect of first assessment of patients with cardiac symptoms. It is a non-invasive, low-cost, and easily accessible technique for assessing ACS.[9]

Among the twelve-lead electrocardiogram, lead aVR has been the forgotten lead until recently where it has been shown to provide useful diagnostic and predictive information in various myocardial diseases. [10]

T wave is generally negative in lead aVR and is in the same direction as the QRS complex.[11] But in recent studies positive T wave in lead aVR has been shown to be an important marker to speculate major adverse cardiac events like heart failure, pulmonary oedema, sustained ventricular tachycardia or ventricular fibrillation, cardiogenic shock and cardiac death as a consequence of STEMI.[10]

AIMS
AND
OBJECTIVES

AIMS AND OBJECTIVES

To study positive T wave in lead aVR as a predictor of in-hospital outcome of the major adverse cardiac events in patients with ST-segment elevation myocardial infarction.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

In developing countries, acute myocardial infarction is one of the leading causes of illness and mortality.[12] The diagnosis and prognosis of acute myocardial infarction are tightly linked by electrocardiographic alterations.

Acute Coronary Syndrome occurs when a coronary artery is suddenly blocked completely or partially, causing myocardial ischemia and its consequences. The clinical entities such as unstable angina, non-ST-elevation MI, and ST-elevation MI are thus included in the syndrome. Acute coronary syndrome is caused by the thrombotic effects of an atherosclerotic plaque rupture or erosion in the vast majority of cases [5]

HISTORICAL REVIEW

In the Ebers Papyrus, Egyptians gave a realistic description of heart ischemia circa 1550 BC: if thou examine a man for illness in his cardia, implies if he has pain in his arms, breast, and one side of his cardia...it is death threatening him.[13] However, William Heberden's essay from 1772 is credited as being the first clinically acceptable description of angina pectoris. The term angina was coined by Heberden from the Latin word meaning strangling. His seminal work laid the groundwork for our understanding of coronary artery disease and myocardial ischemia.[14]

Nonetheless, until the late 19th century, a coronary origin of angina pectoris was not widely accepted[15]. Adam Hammer was the first to describe the pre-mortem diagnosis of myocardial infarction (1878).[16]

John Warren gave a detailed description of angina pectoris in the first edition of the New England Journal of Medicine and Surgery in 1812, in which the symptoms were connected to the existence of coronary disease.[17] This description still has a lot of importance. Several decades later, in 1889, Ludwig Hektoen demonstrated that

myocardial infarction (MI) is caused by coronary thrombosis secondary to sclerotic changes in the coronaries.[18]

In 2008, Aygul N *et al* investigated the role of lead aVR in predicting acute occlusion of the proximal left anterior descending coronary artery and hospital outcomes in ST segment elevation myocardial infarction. They found that lead aVR positivity was linked to a higher heart rate, lower systolic blood pressure and ejection fraction, and a worse Killip class at hospital admission.[19]

In 2011, Shinozaki K *et al* studied the associations of positive T waves in lead aVR with hemodynamic, coronary, and left ventricular angiographic findings in anterior wall myocardial infarction and found that positive T waves in lead aVR are linked to severely reduced cardiac function with left anterior descending wrapping the apex in anterior wall myocardial infarction.[20]

In a study published in 2013, Ayan *et al* looked at the prognostic significance of T wave amplitude in lead aVR in patients with anterior wall myocardial infarction treated with primary percutaneous intervention. They found that patients with positive T waves were older, had significantly more multivessel disease, had a longer hospital stay, and had higher in-hospital mortality than T wave negative patients.[21]

In a study published in 2019, Aysenur F *et al* investigated the prognostic value of a positive T wave in lead aVR as a novel marker of adverse cardiac outcome in peripartum cardiomyopathy patients, finding that patients with a positive T wave in lead aVR had higher rates of persistent left ventricular systolic dysfunction, arrhythmic events, and cardiac death than patients without it. [22]

In 2017, Akihiro K *et al* investigated the role of positive T wave in lead aVR as an independent predictor of 1-year major adverse cardiac events in patients with anterior wall ST- segment elevation myocardial infarction. They found no statistically

significant difference in major adverse cardiac events between patients with positive and negative T waves, but there was a trend toward a higher rate of in-hospital major adverse cardiac events in patients with positive T waves.[10]

INTRODUCTION

Acute myocardial infarction (AMI) is defined as cardiomyocyte death caused by sustained ischemia caused by a sudden oxygen supply and demand imbalance. Despite the fact that a multitude of risk factors have a role in disease progression, atheroma formation and vascular occlusion are the end results. [6]

ANATOMY OF CORONARY CIRCULATION

Since antiquity, the heart has played a vital role in comprehending the body. The Greek philosopher Aristotle designated the heart as the most essential organ of the body in the fourth century B.C. [23]

The word coronary comes from the Latin word corona, which meaning ring or circle. By virtue of their coursing on the entire atrioventricular (AV) sulcus or coronary sulcus, the right and left coronary arteries form a complete arterial ring.

The left main coronary artery (LMCA) branches off from the left coronary sinus of Valsalva to form the left anterior descending coronary artery (LAD) and the left circumflex coronary artery (LCX). The LAD runs through the anterior epicardial ventricular septum, giving rise to a number of diagonals and septal perforators. The LAD is divided into three segments: proximal, middle, and distal. The first septal perforator separates the LAD's proximal and mid-segments.[24]

The LCX originates in the sulcus between the left atrium and the ventricle and gives rise to obtuse marginal branches (OM). The artery's origin portion is referred to as the ostium.

Area of distribution

1. Left atrium

2. Greater part of left ventricle except area adjoining the posterior
   interventricular groove.

3. A small part of right ventricle adjoining the anterior interventricular groove.

4. Anterior part of interventricular septum.

5. SA node (in 40 percent people).

6. A part of left branch of AV bundle.

7. Anterior papillary muscle.

LEFT CORONARY ARTERY (Right Oblique)

Left Main

Proximal Circumflex
Proximal LAD
1st Diagonal

Atrial Branch
1st Septal Perforator
Mid LAD

2nd Diagonal

Obtuse Marginal
Distal Circumflex

Distal LAD

Posterior Lateral
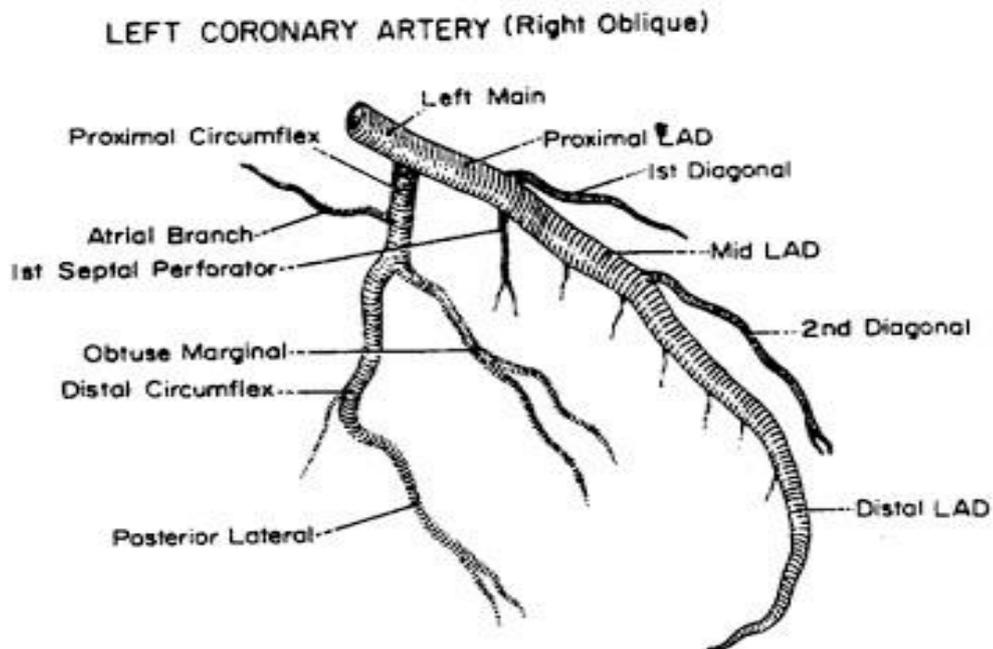
Figure 1: Branches of Left coronary artery.[25]

The right coronary artery (RCA) is divided into three segments: proximal, middle, and distal. It starts from the right coronary sinus. The RCA's proximal portion runs from the ostium to the first acute marginal artery's origin. The conus artery, which arises from the ostium of the RCA or independently from the right coronary sinus in the majority

of cases, is the first visible branch. The conus artery has a superior and anterior course. The sinoatrial (SA) artery, which originates from the proximal RCA and runs posteriorly, is usually the second artery to be seen. Acute marginal (AM) branches arise from the RCA and vary in size. [24]

AREA OF DISTRIBUTION

1. Right atrium.

2. Right ventricle except area adjoining the anterior interventricular groove.

3. A small part of left ventricle adjoining the posterior interventricular groove.

4. SA node (in 60% people).

5. Posterior part of interventricular septum.

6. Posterior papillary muscle.

7. Whole of the conduction of system of the heart except a part of the left branch of the AV bundle.
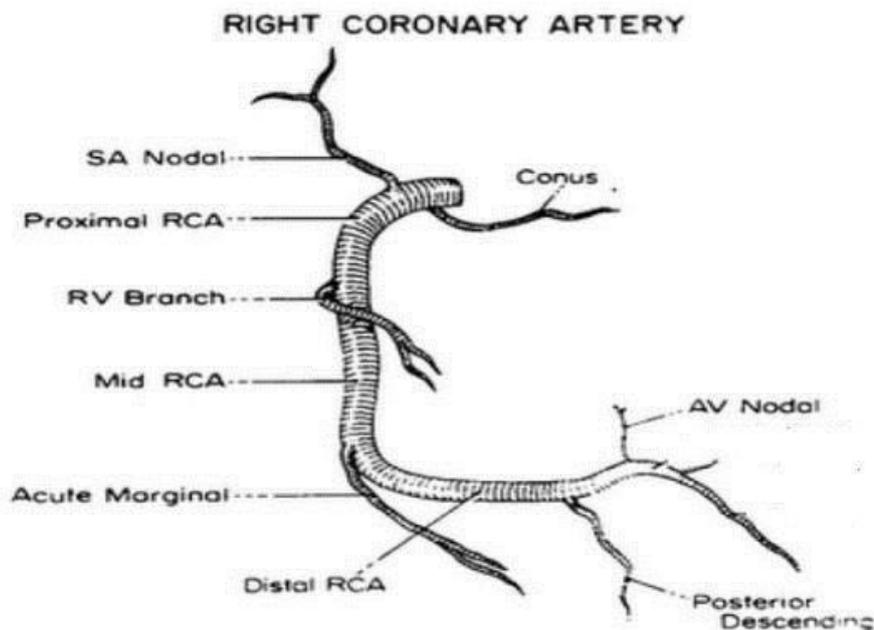
Figure 2: Branches of Right coronary artery[25]

If the right coronary artery (RCA) supplies the posterior descending artery, the coronary circulation is said to be right-dominant. If the circumflex artery (LCX), a branch of the left artery, supplies the posterior descending artery, the coronary circulation is said to be left-dominant. The coronary circulation is described as co-dominant if the right coronary artery and the circumflex artery both supply the posterior descending artery. Right-dominant people make up about 80% of the population, whereas co-dominant people make up 5-10% and left-dominant people make for 10% .[26]

VENOUS DRAINAGE OF HEART

1. Blood from the myocardial capillaries is returned to the right atrium via the coronary vein.

2. The great cardiac vein is located in the anterior interventricular sulcus and drains the coronary sinus from the base of the heart. The anterior interventricular septum, the walls of both ventricles, and the left atrium all flow into it.

3. The coronary sinus receives tributaries from the posterior interventricular septum and ventricular walls, and the middle cardiac vein drains into it.

4. 3-12 anterior cardiac veins drain the right ventricle's wall and empty into the right atrium directly.

5. The coronary sinus is a continuation of the great cardiac vein that is 2-5 cm long and receives veins from the left atrium and posterior left ventricle. It drains into the right atrium directly.

DEFINITION

According to Fourth Universal Definition of Myocardial Infarction (MI)[5] 2018, the term acute myocardial infarction is defined as an acute myocardial damage with clinical evidence of: acute myocardial ischemia and detection of a rise and/or decline in cTn values with at least one value over the 99th percentile upper reference limit and at least one of the following:

1. Symptoms of myocardial ischemia;

2. New ischemic ECG changes;

3. Development of pathological Q waves;

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

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

Acute atherothrombosis in the artery supplying the infarcted myocardium met postmortem criteria for type 1 MI. Type 2 MI is defined as an imbalance between myocardial oxygen supply and demand that is not produced by acute atherothrombosis. Cardiac mortality in individuals with symptoms of myocardial ischemia and new ischemic ECG changes before cTn values are available or aberrant meets type 3 MI criteria.

CLASSIFICATION

A. ANATOMICAL CLASSIFICATION: [5]

- 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.

B. CLINICAL CLASSIFICATION:

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.

ECG CRITERIA FOR DIAGNOSIS OF MYOCARDIAL INFARCTION

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

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. [27]

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.

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 [5]

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

- ≥ 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 [28,29]

TABLE NO.1 LOCALIZATION OF MYOCARDIAL INFARCTION ON ELECTROCARDIOGRAPHY:

Septal	V1, V2
Anterior	V3 and V4
Lateral	V5 and V6
Anteroseptal	V1-V4
Anterolateral	V3-V6
Extensive anterior	V1-V6
Inferior	II, III, aVF
High Lateral	I, aVL
Posterior	Tall R wave in V1 and ST depression in V1-V2

RISK FACTORS[30,31,32]:

Coronary heart disease has a multifactorial etiology. Several major "Risk factors" have been identified through studies.

(A) NON-MODIFIABLE RISK FACTORS

1. Age - Men over 45, women over 55, and anybody with a first-degree male or female relative who had coronary artery disease before the age of 55 or 65.

2. Gender - Men are more at risk than women.

3. Positive family history.

4. Genetic predisposition.

(B) MODIFIABLE RISK FACTORS

1. Obesity

2. Physical inactivity

3. Smoking

4. Alcohol consumption

5. Hypertension

6. Diabetes mellitus

7. Hyperlipidemia

TABLE NO.2 RISK FACTORS FOR CORONARY ARTERY DISEASE[33]

NON-MODIFIABLE	MODIFIABLE/LIFESTYLE	MODIFIABLE
Age	Diet and nutrition (lifestyle)	Hypertension
Gender(male)	Physical activity	Hyperglycemia
Hereditary (including ethnicity)	Stress	Atherogenic dyslipidemia
Family history of cardiovascular disease	Smoking/alcohol	Microalbuminuria
Inflammation	Obesity/overweight	Diabetes mellitus

ETIOLOGY OF ISCHEMIC HEART DISEASE (IHD) [34,35]

Myocardial ischemia result when there is imbalance between oxygen supply and demand. Ischemia is characterized by a lack of oxygen, a reduction in the availability of nutritional substrates, and insufficient metabolite clearance.

(A) Conditions which diminish oxygen supply are:

i. Decreased coronary blood flow

a) Intraluminal variables include atheroma, thrombosis, embolism, and other conditions.

b) Arteriosclerosis, arteritis, and arterial spasm in the wall.

c) Outside the wall of the coronary artery congenital abnormality of the coronaries, coronary steal, left ventricular hypertrophy (LVH) or local myocardial muscle pathology constricting coronary arteries.

ii. Oxygen carrying capacity reduction - severe anemia, hemoglobinopathies

iii. Others - Polycythemia vera, DIC, thrombocytosis, and aortic stenosis.

B. Conditions which increase demand for oxygen

Heavy exercise, heavy meal, increased heart rate.

MECHANISM

1) Coronary Vasospasm: This is defined as a local reversible reduction in coronary artery diameter, resulting in reduced blood supply and ischemia, which can lead to infarction if left untreated.

2) Coronary Steal: When both arterial beds share a single source of perfusion, vascular steal is defined as preferential blood flow to one at the expense of the other.

Causes of coronary artery steal are:

a) Congenital malformation

b) Atherosclerotic artery disease

c) Vasoactive drugs

d) Coronary stenosis

Coronary stenosis is the progressive deposition of atherosclerotic material in the arterial wall, diminishing vascular cross-sectional area and producing luminal narrowing and eventually full obstruction of the coronary artery, which is the underlying cause of ischemic heart disease.

Almost all myocardial infarctions are caused by coronary atherosclerosis, which is usually accompanied by coronary thrombosis.

Clinically significant stenosing plaque can be found anywhere, but it is most common in the first few centimeters of the LAD and LCX, as well as across the entire length of the RCA.

PATHOPHYSIOLOGY

Atherosclerosis is a systemic disease characterized by intimal thickening induced by the accumulation of cells and lipids in the intima of large and medium-sized arteries, such as the aorta, carotids, coronaries, and peripheral arteries. The development of atherosclerotic plaque starts early in life and progresses slowly over decades.[36] Because of compensatory vascular expansion (Glagovian remodeling), early atherosclerotic lesions may grow without affecting the lumen.[37] Inflammation and hemostatic processes are linked by an increasing number of molecular and cellular pathways. Inflammation and possibly persistent infection may play a role in the onset and progression of atherosclerosis to its terminal stages.[38] A large lipid core, a thin fibrous cap, and a high density of inflammatory cells characterize these high-risk, rupture-prone lesions (particularly at the shoulder region, where disruptions most often occur). Platelet aggregation and thrombus development occur when plaque erosion or disruption occurs. White thrombi contain platelets, fibrin, or both, while red thrombi contain erythrocytes, fibrin, platelets, and leukocytes.

Acute coronary syndromes are caused by the formation of an acute thrombus on a ruptured coronary atherosclerotic lesion, which obstructs coronary blood flow and reduces oxygen supply to the myocardial (ACS).

Atherosclerotic plaques according to their structure and relation with prognosis are divided into:

(A) Stable plaque:

Clinical research suggests that stable plaque shows higher luminal narrowing detectable by angiography than vulnerable plaque because they are less vulnerable to fissure and rupture. They feature a thin fibrous covering that separates the lipid core from the blood.

(B) Unstable plaque:

Stable plaque or vulnerable plaque refers to atherosclerotic plaque in coronary arteries that is more susceptible to fissure and rupture. They feature a lipid core and a thin fibrous top. Because the fibrous cap that separates the lipid core from the blood is so thin, it is more prone to fissure and rupture.

(C) Complicated plaque:

Fibrocalcific degeneration, lipid and calcium deposition, fibrous tissue, necrotic debris, extravascular blood, and a fibrous cap are all characteristics of complicated plaques. Coronary thrombi are usually superimposed on or adjacent to atherosclerotic plaque in individuals with myocardial infarction and adhere to the luminal surface of an artery in the majority of cases.
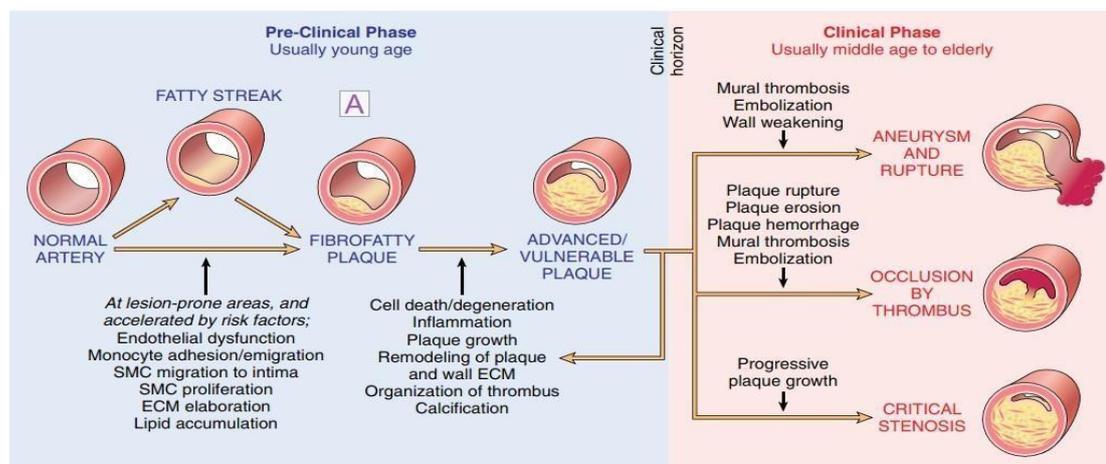
Figure 3: Summary of the natural history, morphologic features, main pathogenic events, and clinical complications of atherosclerosis.[39]

Fresh platelet rich thrombi are frequently associated with unstable angina and acute MI, showing that platelet aggregation and adhesion, as well as fibrin deposition, are pathogenetic components in ischemic heart disease.

The duration, severity, and treatment of unstable angina, non–ST-segment myocardial infarction (NSTEMI), and ST-segment myocardial infarction (STEMI) are all different.

Unstable Angina Caused due to thrombus partially or intermittently occludes the coronary Artery, Signs and Symptoms involves pain with or without radiation to arm, neck, back, or epigastric region, Shortness of breath, diaphoresis, nausea, light headedness, tachycardia, tachypnea, hypotension or hypertension, decreased arterial oxygen saturation (SaO2) and rhythm abnormalities occurs at rest or with exertion; limits activity [37]. Electrocardiography findings include ST-segment depression or T-wave inversion, as well as cardiac biomarkers that are not increased.

NSTEMI (Non–ST-Segment Elevation Myocardial Infarction) is caused by thrombus occluding the coronary artery partially or intermittently. The symptoms are the same as in unstable angina. On electrocardiography, there is ST-segment depression or T-wave inversion, and cardiac biomarkers are increased.

STEMI occurs when a thrombus completely obstructs a coronary artery.

2. CLINICAL PRESENTATION:

Acute myocardial infarction can present in a variety of ways. Patients with AMI may present with atypical symptoms such as dyspnea, abdominal or epigastric discomfort, nausea, vomiting, and syncope, in addition to the hallmark sign of chest pain.[40]

A. Chest pain:

The most prevalent symptom is chest pain, which is described as a feeling of tightness, pressure, or squeezing that is not relieved by rest, position change, or the administration of analgesics or antacids. The pain usually radiates to the left arm, but it can also radiate

to the lower jaw, neck, right arm, back, and upper abdomen, mimicking heartburn. Levine's sign is when a person clenches their fists over their sternum to localize chest pain. Silent acute myocardial infarction is characterized by the absence of chest pain, which is common in people with diabetes, hypertension, and the elderly.[40]

B. Shortness of breath (dyspnea):

The injury to the heart reduces the left ventricle's output, resulting in left ventricular failure and pulmonary edema.

C. Nausea and Vomiting:

Vomiting is a response that occurs as a result of extreme pain triggered by vasovagal reflexes triggered by ischemia.

D. Diaphoresis:

Diaphoresis is caused by sympathetic nervous system excitation and catecholamine release.

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.

INVESTIGATIONS

A) CARDIAC BIO MARKERS

1) Creatine kinase (CK):

It is found in the cell's cytoplasm. Between 14 and 36 hours after myocardial infarction, 15% of the CK in the myocardium is in the form of MB-CK, which returns to normal after 48-72 hours. Within 12-16 hours of the onset of symptoms, the plasma level reaches diagnostic sensitivity. [41]

2) Cardiac troponins:

Two of the three troponin subunits (troponin I and troponin T) are derived from genes expressed uniquely in the myocardium.

Cardiac troponin (cTn) became popular in the 1990s and has since become the gold standard for MI diagnosis. An increase and/or decrease in cTn with at least one value over the 99th percentile of the upper reference limit is currently used as diagnostic criteria for MI.[42] Biomarkers are prognostic for STEMI patients and can be used to confirm a diagnosis. Biochemical indicators, on the other hand, play a critical role in NSTEMI diagnosis, separation from unstable angina, and therapy [41].

3) 2D-ECHO (ECHOCARDIOGRAPHY)

They are useful for evaluating patients with nondiagnostic ECGs. The existence of a localized wall motion anomaly is strong indication of acute coronary artery occlusion. The function of the ventricles can be tested. It aids in the diagnosis of right ventricular infarction and the prediction of prognosis. It can be used to detect mechanical issues, ventricular thrombus, and pericardial fluid. Small infarctions can be undetected, and a wall motion abnormality does not always mean it is severe. Its importance in detecting complications and determining the extent of myocardial injury cannot be overstated. It also aids in determining a patient's prognosis.[43]

TREATMENT

The primary goal of STEMI is to restore blood flow to the ischemic myocardium as soon as possible, as delaying this may result in increased myocardial necrosis and a worse prognosis.

Primary Percutaneous coronary intervention (PCI) is the treatment of choice, assuming that the point of initial contact has the necessary manpower and equipment.

In patients with acute myocardial infarction and ST-segment elevation, early coronary revascularization is the best treatment option (STEMI). This is reflected in the current European and American guidelines for the treatment of STEMI.[44,45]

Effective reperfusion therapy has been linked to a lower death rate and better outcomes.[46]

The American college of cardiology (ACC) and American Heart Association (AHA) guidelines recommend categorizing patients with possible ACS into four categories based on information from the history, physical examination, 12-lead ECG, and initial cardiac marker tests. These categories are non-cardiac-related diagnosis, chronic stable angina, possible ACS, and definite ACS.[47]

TABLE NO.3 ACC/AHA GUIDELINES CATEGORIZING ACS INTO

FOUR CATERGORIES

FEATURE	HIGH LIKELIHOOD Any of the following	INTERMEDIATE LIKELIHOOD Absence of High-likelihood features and presence of any of the following	LOW LIKELIHOOD Absence of high or intermediate likelihood features but may have any of the following
History	Chest or left arm pain or discomfort as the chief symptom reproducing documented previous angina. Known history of coronary artery disease, including MI.	Chest or left arm pain or discomfort as the chief symptom: Age >70yrs Male sex Diabetes mellitus	Probable ischemic symptoms in the absence of any of the intermediate-likelihood characteristics. Recent cocaine use.
Examination	Transient mitral regurgitation murmur, hypotension, diaphoresis, pulmonary edema or rales	Extracardiac vascular disease	Chest discomfort reproduced by palpitation
ECG	New transient ST-segment deviation (>0.1mV) or T wave inversion (>0.2mV) in multiple precordial leads.	Fixed Q waves. ST-segment depression of 0.05-0.1 mv or T wave inversion >0.1mV	T wave flattening or inversion <0.1mV in leads with dominant R waves.
Cardiac markers	Elevated cardiac cTnI , cTnT, or CK-MB	Normal	Normal
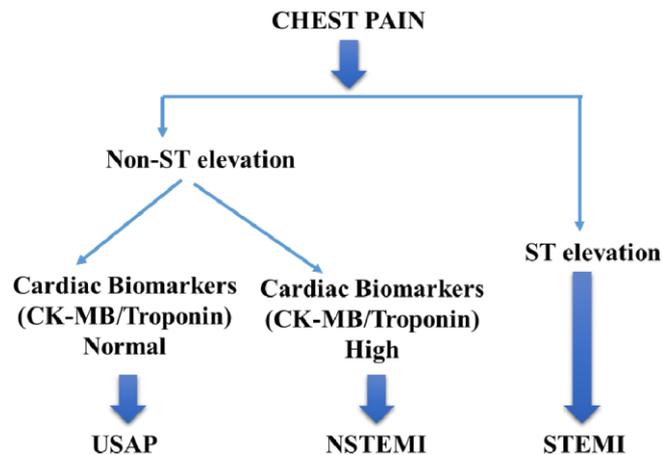
Figure 4: Classification of ACS based on ECG findings and Cardiac Troponins[48]

After confirming the diagnosis, the following measures should be taken,

GENERAL TREATMENT MEASURES

1) **ASPIRIN:** Aspirin is effective across the entire ACS spectrum and is the initial management strategy for patients with suspected STEMI. Because low doses take several days to achieve a full antiplatelet effect, 162 to 325 mg should be administered at the first opportunity after initial medical contact.[12] To achieve therapeutic blood levels rapidly, the patient should chew the tablet to promote buccal absorption rather than absorption through the gastric mucosa.

2) **CARDIAC PAIN MANAGEMENT:** Control of cardiac pain is usually done using a combination of analgesics (such as morphine) and therapies to improve the balance of myocardial oxygen supply and demand, such as oxygen, nitrates, and, in appropriately selected patients, beta-adrenergic receptor–blocking medicines (beta blockers).

3) **ANALGESICS:** Except in individuals with well-documented morphine hypersensitivity, morphine remains the drug of choice. Intravenous dosages of 4 to 8 mg and doses of 2 to 8 mg repeated every 5 to 15 minutes have been recommended until the pain is resolved or side symptoms such as hypotension, depression of respiration, or severe vomiting occur, preventing further drug delivery. With successful analgesia, the patient's restlessness and autonomic nervous system activity are reduced, lowering the heart's metabolic needs. Patients with pulmonary edema induced by peripheral arterial and venous dilatation (especially those with high sympathoadrenal activity) benefit from morphine.

4) **NITRATES:** Sublingual nitrates are recommended for most patients with an ACS because of their capacity to increase coronary blood flow through coronary vasodilation and decrease ventricular preload through increased venous capacitance. Those with suspected right ventricular infarction or marked hypotension (e.g., systolic pressure 90 mm Hg), especially if accompanied by bradycardia, should not be given sublingual nitroglycerin. Intravenous nitroglycerin may help manage symptoms and correct ischemia in patients with a lengthy history of waxing and waning chest discomfort, but blood pressure must be monitored often.

5) **BETA-ADRENERGIC BLOCKING AGENTS:** These medications help to relieve ischemic pain, minimize the need for analgesics in many patients, and lower the size of infarcts and life-threatening arrhythmias. It's critical to avoid early intravenous blockage in patients with Killip's class II or above.

The following is a practical protocol for using a beta blocker in this case.

- Patients with heart failure, hypotension (systolic blood pressure less than 90 mm Hg), bradycardia (heart rate less than 60 beats per minute), or substantial atrioventricular (AV) block should be excluded from the study.

- Give three 5-mg intravenous boluses of metoprolol.

- After each bolus, observe the patient for 2 to 5 minutes, and if the heart rate drops below 60 beats per minute or the systolic blood pressure drops below 100 mm Hg, do not administer any more medication.

- If hemodynamic stability persists 15 minutes after the last IV dosage, start oral metoprolol tartrate at 25 to 50 mg every 6 hours for 2 to 3 days, then switch to 100 mg twice daily if tolerated.

6) **OXYGEN** If the oximetric findings are normal, oxygen therapy can be skipped. Patients experiencing STEMI and arterial hypoxemia, on the other hand, should be given oxygen. Endotracheal intubation and mechanical ventilation may be required in individuals with severe pulmonary edema to correct hypoxemia and reduce work of breathing.

7) **REPERFUSION THERAPY:**

Patients treated with fibrinolysis or PCI (Percutaneous coronary intervention) have myocardial salvage that is dependent on the time passed before treatment. The relative risk (RR) of 1-year death increases by 8% for every 30-minute delay between symptom onset and PCI. Early reperfusion reduces the risk of pump failure or malignant ventricular tachyarrhythmias in STEMI patients by reducing the duration of coronary occlusion, the degree of ultimate left ventricular dysfunction and dilation, and the likelihood of pump failure or malignant ventricular tachyarrhythmias.

Thrombolytic therapy as discussed below:

- Heparin.

- Unfractionated or fractionated heparin intravenously or subcutaneously for 3-5 days.

- GP II / III, receptor agonists (Abciximab, Tirofiban, eptifibatide) due to high cost and occasional bleeding complication limits their use.

- PTCA (Percutaneous transluminal coronary angioplasty)

THROMBOLYTIC THERAPY

In the early stages of treatment for acute myocardial infarction, reperfusion techniques strive to quickly restore and sustain tissue perfusion.

Primary angioplasty is the best current treatment, although it can only be used in a small percentage of patients and has its own set of issues. The most widely utilized treatment is thrombolysis.[49]

Patients who present relatively early (60-90 minutes after symptom onset) had great outcomes with fibrinolytic treatment alone.[50]

INDICATIONS FOR FIBRINOLYSIS

- Ischemic type chest discomfort or equivalent for 30 minutes - 12 hours with new or presumed new ST segment elevation in two contiguous leads of ≥2 mm (≥0.2 mV) in leads V1, V2, or V3 or ≥1 mm in other leads.

- New or presumed new left bundle branch block with symptoms consistent with myocardial infarction.

- Absence of contraindications.

1) **AGENTS WITH FIBRIN SPECIFICITY**

- Alteplase (tPA) - IV bolus 15 mg, followed by 0.75 mg per kg by iv infusion over 30min and then 0.5 mg per kg over 60 min.

- Reteplase (r-PA) - IV bolus of 10 units over 2 min, followed by another bolus of 10units after 30 min.

- Tenecteplase - 30-50mg bolus based on weight.

2) **AGENTS WITHOUT FIBRIN SPECIFICITY**

- Streptokinase -IV infusion of 1.5 million over 60 min.

- Anistreplase - IV bolus of 30 units over 2 min.

- Urokinase - IV infusion of 3 million units over 60 min.

These all-drugs act by promoting the conversion of plasma fibrinogen to plasmin which subsequently lyses fibrin thrombi.

ABSOLUTE CONTRAINDICATION FOR THROMBOLYSIS

- Active bleeding or bleeding diathesis (menses excluded).

- Prior hemorrhagic stroke.

- Ischemic stroke within 3 months, except acute ischemic stroke within 3-4.5hours

- Intracranial or spinal cord neoplasm or arteriovenous malformation.

- Suspected or known aortic dissection.

- Closed head or facial trauma within 3 months.

RELATIVE CONTRAINDICATIONS FOR THROMBOLYSIS

- Severe, uncontrolled hypertension by history or on presentation (BP>180/110 mm Hg).

- Anticoagulation with therapeutic or elevated international normalized ratio (INR >2-3).

- Old ischemic stroke (>3 months ago), intracerebral disease other than above.

- Recent (<3 weeks) major trauma/surgery or prolonged (>10minutes) cardiopulmonary resuscitation or internal bleeding.

- Active peptic ulcer.

- Recent non compressible vascular punctures.

- Pregnancy

- For streptokinase/anistreplase: prior exposure (especially if >5 days ago) or allergic reaction.

COMPLICATIONS OF FIBRINOLYTIC THERAPY

The most common complication of fibrinolytic therapy is bleeding, and cerebral hemorrhage is the most serious; its incidence is typically less than 1%.

According to reports, fibrinolytic therapy has an early hazard - that is, an excess of mortality in the first 24 hours in fibrinolytic-treated patients when compared to control subjects (especially in elderly patients treated more than 12 hours after symptom onset).

If a patient has been treated with a streptokinase product within the last 6 months, they should not receive streptokinase for STEMI.

CATHETER-BASED REPERFUSION STRATEGIES

PCI is referred to as direct or primary PCI when it is utilized as the primary reperfusion treatment in patients with STEMI.

Rescue PCI can be used after fibrinolysis has failed to re-perfuse the infarct vessel or if the infarct vessel has a severe stenosis.

After effective fibrinolytic therapy, a strategy of routine delayed angiography and PCI may be considered.

Indications for Coronary Angiography in Patients Who Were Managed with Fibrinolytic Therapy or Who Did Not Receive Reperfusion Therapy

1) Cardiogenic shock or acute severe HF that develops after initial evaluation

2) Intermediate- or high-risk findings on predischarge noninvasive ischemia testing.

3) Spontaneous or easily provoked myocardial ischemia

4) Failed reperfusion or re occlusion after fibrinolytic therapy

5) Patients after successful fibrinolysis - before discharge and ideally between 3 and 24 hr.

Patients Not Eligible for Reperfusion Therapy

Patients who are not candidates for acute reperfusion due to a lack of PCI or contraindications to fibrinolysis can be offered aspirin and antithrombin treatment. In the absence of access to PCI facilities and absolute contraindications to fibrinolysis.

INDICATIONS FOR PRIMARY ANGIOPLASTY

- Using the femoral or, increasingly, the radial artery route, prompt PCI is possible. And radial route is the preferred reperfusion technique at PCI-capable facilities for patients with STEMI and ischemic symptoms lasting less than 12 hours.[51]

- Cardiogenic shock emerging within 36 hours following ST segment elevation/Q wave acute MI or LBBB acute MI in patients under 75 years of age who can be revascularized within 18 hours of the commencement of the shock.

- Only for centers that do >200 PCIs per year with backup cardiac surgery and for operators who execute >75 PCIs per year.

Coronary Artery Bypass Graft (CABG) is recommended for:

- Patients who have a disease of the left main coronary artery.

- Individuals with multivessel disease (including all three major epicardial vessels or the proximal left anterior descending artery plus a second artery) and/or diabetes mellitus.

- Left ventricular ejection fraction lower than 40% and/or diabetes mellitus.

COMPLICATIONS OF ACUTE MYOCARDIAL INFARCTION

The majority of these patients' deaths are caused by pathophysiologic abnormalities that occur as a result of the AMI. Many more people are affected by AMI problems. To avoid unnecessary morbidity and mortality, these individuals require fast and early detection of their conditions, as well as vigorous management. AMI complications can be divided into three categories:

A. Heart failure and Cardiogenic shock.

B. Ischemic Complication

   Reinfarction

C. Mechanical Complications

   i. Left ventricular aneurysm

   ii. Myocardial rupture

   iii. Rupture of the ventricular septum

   iv. Pseudoaneurysm

D. Conduction Abnormalities

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

E. Embolic Complications

Stroke

F. Inflammatory complications

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

A. HEART FAILURE:

After a myocardial infarction, heart failure (HF) is a leading cause of late morbidity and mortality. The most common cause of heart failure (HF) worldwide is myocardial infarction (MI). HF is a clinical syndrome caused by any structural or functional cardiac problem that affects the ventricle's ability to fill or eject blood. A low volume pulse, low blood pressure, cool and clammy extremities, a third heart sound (S3 gallop), pulmonary rales, reduced urine production, and sweating are all signs of circulatory failure.

When the left ventricular myocardium is damaged by 20% to 25%, there is a reduction in cardiac output. When the left ventricular myocardium is injured by 40% or more, cardiogenic shock occurs. Pulmonary congestion and S3 and S4 gallops are the most typical clinical findings.

The presence of left cardiac dysfunction is linked to the patients' short- and long-term prognosis. Breathlessness, easy fatiguability, sinus tachycardia, a third heart sound (S3 gallop), systolic or diastolic murmurs, pulmonary rales that can be identified in the lung bases, and systolic or diastolic murmurs are all symptoms of heart failure. The developed pulmonary edema does not correlate with the auscultatory signs.[52]

Killip's classification is one method used to access the severity of heart failure following a myocardial infarction,[53]

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.

SYSTOLIC DYSFUNCTION OF MYOCARDIUM:

In non-valvular HF, the major problem is poor left ventricular (LV) function, which results in decreased cardiac output. The activation of several compensatory neurohormonal processes occurs as a result of the decrease in cardiac output. Through endothelial dysfunction, this stimulation of neurohormonal pathways will enhance myocardial contractility while decreasing coronary perfusion.

Increased heart rate, myocardial contractile function, peripheral vasoconstriction, and activation of the Renin-Angiotensin-Aldosterone system (RAAS) are all benefits of sympathetic nervous system activation in maintaining appropriate cardiac output. With salt and water retention, RAAS causes vasoconstriction and a rise in blood volume. Vasopressin and natriuretic peptides like Brain natriuretic peptide (BNP) and NT pro-BNP concentrations are also rising. The heart may dilate gradually, or the structure of the heart may alter, or both.[54]

DIASTOLIC DYSFUNCTION:

Diastolic dysfunction is defined as a "clinical syndrome of HF with intact Left ventricle ejection fraction, i.e. ejection fraction > 40% with no significant valvular heart disease. Because of the increased LV mass in diastolic HF, the LV cavity is stiff. The myocardium progressively relaxes during diastole in the early phase, and there is increased resistance to left ventricular filling in late diastole, resulting in an increase in diastolic pressure. Reduced cardiac output manifests as fatigue, and elevated end diastolic pressure is transmitted back to pulmonary capillaries via valveless pulmonary veins, resulting in exertional breathlessness.[54]

B. CARDIOGENIC SHOCK:

Cardiogenic shock (CS) is a severe form of left ventricular failure characterized by hypotension (systolic blood pressure less than 80 mm Hg) and a low cardiac index (1.8

L/min/m2) despite a high left ventricular filling pressure. The cause for this is the loss of functional myocardial tissue, which accounts for more than 40% of the left ventricle. Despite early medical treatment, cardiogenic shock has a significant fatality rate of more than 70%.[55]

C. ARRHYTHMIAS: The earliest signs of ischemia are often life-threatening arrhythmias such ventricular tachycardia, ventricular fibrillation, and complete AV block. Up to 20% of individuals have been documented to have ventricular fibrillation or prolonged ventricular tachycardia. In individuals who have survived an acute myocardial infarction, the risk of death due to an arrhythmic episode is highest in the first six months and stays high over the next two years. Arrhythmias can occur as a result of myocardial infarction, reperfusion, an irritable myocardium, toxic metabolites, or even metabolism (especially potassium and magnesium imbalance).[56]

LEAD aVR – THE NEGLECTED LEAD

Clinicians frequently overlook Lead aVR, one of the 12 electrocardiographic leads.[10]

It's a unipolar lead aimed at the superior right surface. All depolarizations in a typical sinus rhythm go away from the lead aVR, hence all the waves in aVR are negative (P, QRS, T). Lead aVR has been demonstrated to be significant in locating coronary artery disease in recent years.

Electrocardiographic significance of lead aVR.

1) **Coronary artery disease:**

a) Localizing the level of obstruction in acute coronary syndrome - Lead aVR can be used to determine the degree of obstruction in the left main coronary artery (LMCA). Ischemia in this location causes the ST segment vector in the frontal plane to point in a superior direction, resulting in ST elevation in lead aVR and ST depression in inferior leads due to the dominance of basal ventricular mass.

LMCA and proximal left anterior descending artery (LAD) blockage are also distinguished by lead aVR. ST elevation in lead aVR > lead V1 indicates LMCA disease, while ST elevation in lead aVR > lead V1 indicates LAD disease.[57]
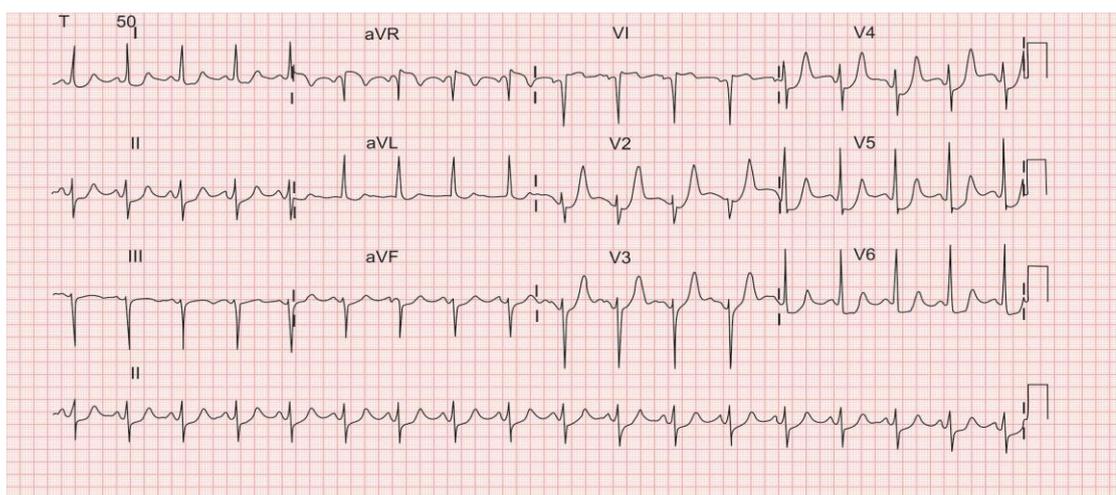
Figure 5: Left main coronary artery disease showing ST elevation in lead aVR more than V1.[57]

b) Atrial infarction - PR segment elevation in inferior leads and PR segment depression in lead aVR are both signs of atrial infarction in inferior wall MI.

c) Left Ventricular Aneurysm (Goldberger's sign)- An anterior wall MI with sustained ST elevation in chest leads and a tall R wave in lead aVR may indicate ventricular aneurysm (Goldberger's sign).

2) **Arrhythmias:**

a) Ventricular Tachycardia (VT) - A tall R wave in lead aVR is present in VT (due to caudo-cranial activation).

b) Supraventricular Tachycardia (SVT) - Any SVT with caudo-cranial atrial activation, such as AV nodal tachycardia (AVRT), left atrial tachycardia, or circus movement tachycardia, will display positive P waves in lead aVR.

3) **Acute Pericarditis**- PR, ST segment discordance indicates acute pericarditis, whereas PR, ST segment concordance indicates acute MI.

4) **Tension Pneumothorax**- ECG changes with or without tension are more likely in left pneumothorax. There is anterior T wave inversion, right QRS axis deviation, low QRS voltage, reduced precordial R wave voltage, and right QRS axis deviation. With left tension pneumothorax, marked PR segment elevation in inferior leads and reciprocal PR segment depression in lead aVR has been recorded.

5) **Acute Pulmonary Embolism**- Acute right ventricular overload might manifest as ST segment elevation in lead aVR and terminal r wave, in addition to sinus tachycardia, normal ECG findings, and the characteristic S1Q3T3 pattern. [57]

ST elevation in lead aVR has predictive value:

When there is global ST depression with symptoms of myocardial ischemia:

1) If the STE in aVR is greater than 1 mm, it implies proximal LAD/LMCA blockage or significant 3 vessel disease.

2) CABG is predicted by STE in aVR greater than 1 mm.

3) Absence of ST elevation in lead aVR precludes a severe LMCA lesion.

4) STE in aVR > V1 differentiates LMCA from proximal LAD blockage.

In anterior STEMI

For LAD blockage proximal to the first septal branch, STE in aVR > 1 mm is specific.

In patients undergoing exercise stress test

STE in aVR >1 mm predicts LMCA or ostial LAD stenosis in patients undergoing an exercise stress test. [57]

T WAVE

INTRODUCTION

The positive deflection after each QRS complex is the T wave on the ECG. It is a symbol for ventricular repolarization.[27] The explanation for the positive deflection of the T wave on the ECG is because the last cells in the ventricles to depolarize are the first to repolarize, i.e., from epicardium to endocardium. This is due to the fact that the final cells to depolarize are found in the subepicardial area of the ventricles, where their action potential is shorter than that of subendocardial cells. As a result, the subepicardial cells repolarize (phase 3) before the subendocardial cells.[58]

DEFINITION

Positive T wave in lead aVR is defined as T wave amplitude >0mV and negative T wave < 0mV in lead aVR. [10]
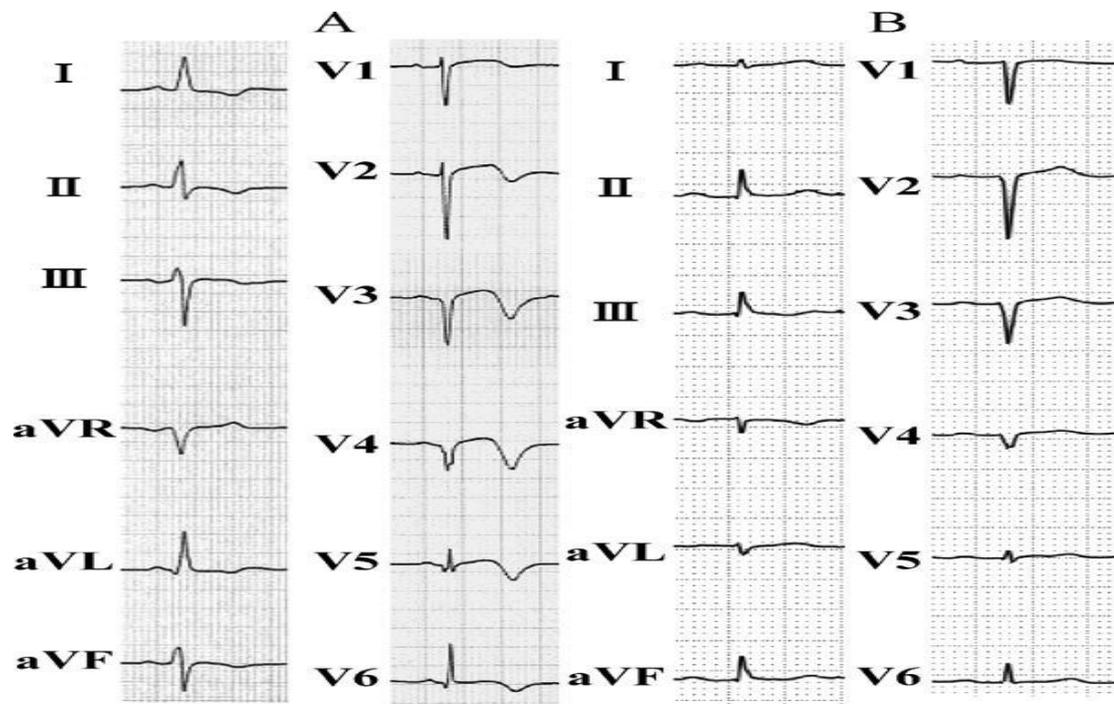
Figure 6: Representative electrocardiograms with a positive T wave in lead aVR (A) and without it (B) [20]

MECHANISM OF POSITIVE T WAVE IN LEAD aVR

The spatial-temporal properties of ventricular repolarization, particularly the asynchrony of phase 3 of the ventricular action potential, determine T wave configuration.[59]

The repolarization of injured cardiac cells is delayed compared to normal cells, and as a result, the T wave vector changes direction towards the wounded myocardium. The wounded myocardium of the left ventricle's apical, inferior, and lower lateral portions causes a normal negative T wave to become a positive T wave in lead aVR. [10]

MATERIALS
AND
METHODS

MATERIALS AND METHODS

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 2019 to June 2021 on 98 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.

Study Design: Prospective cross-sectional study

Study Period: One and half year from November 2019 to June 2021.

Sample size calculation

With 95% confidence level and margin of error of ±10%, a sample size of 92 (100), on the basis of the study, subject will allow the study to determine the "POSITIVE T WAVE IN LEAD aVR AS A PREDICTOR OF IN HOSPITAL OUTCOME OF THE MAJOR ADVERSE CARDIAC EVENTS IN PATIENTS WITH ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION" with finite population correction (N=200).[10]

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%)

PATIENT SELECTION

A. INCLUSION CRITERIA:

      I) Patients admitted with ST segment elevation myocardial infarction.

B. EXCLUSION CRITERIA

1) Patients with Non-STEMI.

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

3) Previous history of myocardial infarction

4) Arrythmias.

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

INITIAL ASSESSEMENT

The study was conducted on patients who were admitted in BLDE (Deemed to be University), 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.

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 positive T wave in lead aVR. Patients were grouped according to the presence of positive T wave as Group A, and negative T wave as Group B. The positive T wave is defined as T wave amplitude more than 0Mv and negative T wave was defined as T wave amplitude less than 0mv in lead aVR.

ST- segment elevations was measured at the J point. ST-segment elevation > 2mm in lead V2 - V3, ST-segment elevation >1mm in other leads (except for lead aVR) will be recorded.

In addition, ST-segment elevation >0.5 mm in lead aVR was recorded. Patients were divided into those with positive T wave and those with negative T wave in lead aVR and clinical variables were compared between the two groups.[10]

OUTCOME

Patients were followed up during their in-hospital course for the occurrence of major adverse cardiac events namely: pulmonary edema, cardiogenic shock, heart failure, post -infarction angina, complex ventricular arrhythmias (sustained ventricular tachycardia

or ventricular fibrillation) and death. Heart failure was diagnosed clinically according to the standard criteria. Complex ventricular arrhythmia by monitoring ECG strip or by 12 lead ECG recording. Early post-infarction angina- recurrent typical chest discomfort during hospital admission following relief of that of the index myocardial infarction. Mechanical complications (recorded by echocardiography) including: acute mitral regurgitation, rupture of the interventricular septum, LV pseudo-aneurysm formation, and rupture of the LV free wall.

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:

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

where $\overline{x_1}$ = mean of sample 1

$\overline{x_2}$ = 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 = $\dfrac{\Sigma(x_1 - \overline{x_1})^2}{n_1}$

s_2^2 = variance of sample 2 = $\dfrac{\Sigma(x_2 - \overline{x_2})^2}{n_2}$

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 98 patients admitted with acute coronary syndrome, two patients were excluded

from the study based on exclusion criteria, of which the one patient had previous history

of ischemic heart disease and the other patient had left bundle branch block. Hence total

of 96 patients were included in the study.

```
                 ┌──────────────────────────────────────────────┐
                 │ PATIENTS OF ACUTE CORONARY SYNDROME (98)     │
                 └──────────────────────────────────────────────┘
                      │                              │
                      ▼                              ▼
              ┌──────────────┐              ┌──────────────┐
              │  INCLUDED    │              │  EXCLUDED    │
              └──────────────┘              └──────────────┘
                      │                    ┌───────────────────────────────┐
                      ▼                  → │ Old Ischemic heart disease (1)│
              ┌──────────────┐            └───────────────────────────────┘
              │   STEMI      │            ┌───────────────────────────────┐
              └──────────────┘          → │ Left bundle branch block (1)  │
               │          │              └───────────────────────────────┘
               ▼          ▼
    ┌────────────┐  ┌────────────┐
    │ GROUP-A    │  │ GROUP-B    │
    │ (34)       │  │ (62)       │
    │            │  │            │
    │ (With      │  │ (With      │
    │ positive-T │  │ negative-T │
    │ wave)      │  │ wave)      │
    └────────────┘  └────────────┘
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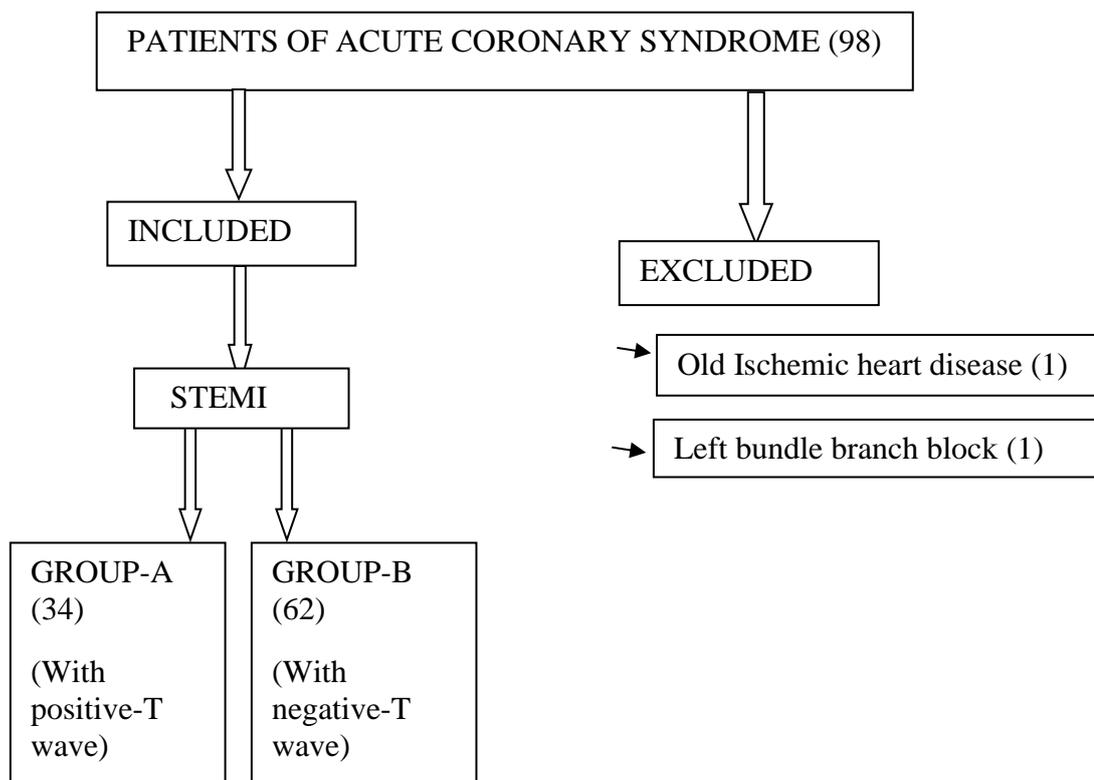
Figure 7: Flowchart showing included and excluded cases in the study.

Note: - p < 0.05 - statistically significant

   P<0.001 – highly significant

Out of 96 patients with STEMI, 34 patients who had positive T wave in lead aVR

were in group A and 62 patients with negative T wave in lead aVR were in group B as

shown in Table 4.

Table 4: Grouping of patients with STEMI into Group A and Group B			
Classification		No. of cases	%
Group A	Positive T wave in lead aVR	34	35.4
Group B	Negative T wave in lead aVR	62	64.5
Total		96	100.0

AGE DISTRIBUTION

The 96 patients were grouped with an age frequency of 10 years.

In group A patients aged between 21-30 yrs. were 0 (0%), patients aged between 31 - 40 years were 2(5.8%), patients aged between 41-50 years were 2(5.8%), patients aged between 51 -60 years were 20 (58.8%), patients aged between 61-70 years were 5 (14.8%) patients aged between 71-80 years were 5(14.8%), patients aged between 81-90years were 0(0%).

In group B patients aged between 21-30 years were 2 (3.3%), patients aged between 31-40 years were 3(4.9%), patients aged between 41-50 years were 15(24.1%), patients aged between 51-60years were 25 (40.3%), patients aged between 61-70 years were 11 (17.7%), patients aged between 71 -80 years were 5 (8%) patients aged between 81-90 years were 1 (1.6%).

The most common age group in both group A and group B was 51-60 years as described in table:5, Graph:1

Table 5: Distribution of patients according to Age in Group A and Group B						
Age (yrs.)	Group A		Group B		Total	
	No.	%	No.	%	No.	%
21-30	0	0	2	3.3	2	2.08
31-40	2	5.8	3	4.9	5	5.2
41-50	2	5.8	15	24.1	17	17.7
51-60	20	58.8	25	40.3	45	46.8
61-70	5	14.8	11	17.7	16	16.6
71-80	5	14.8	5	8.0	10	10.4
81-90	0	0.0	1	1.6	1	1.04
Total	34	100.0	62	100.0	96	100.0

$X^2 = 9.50,$

$p = 0.15$, Not significant
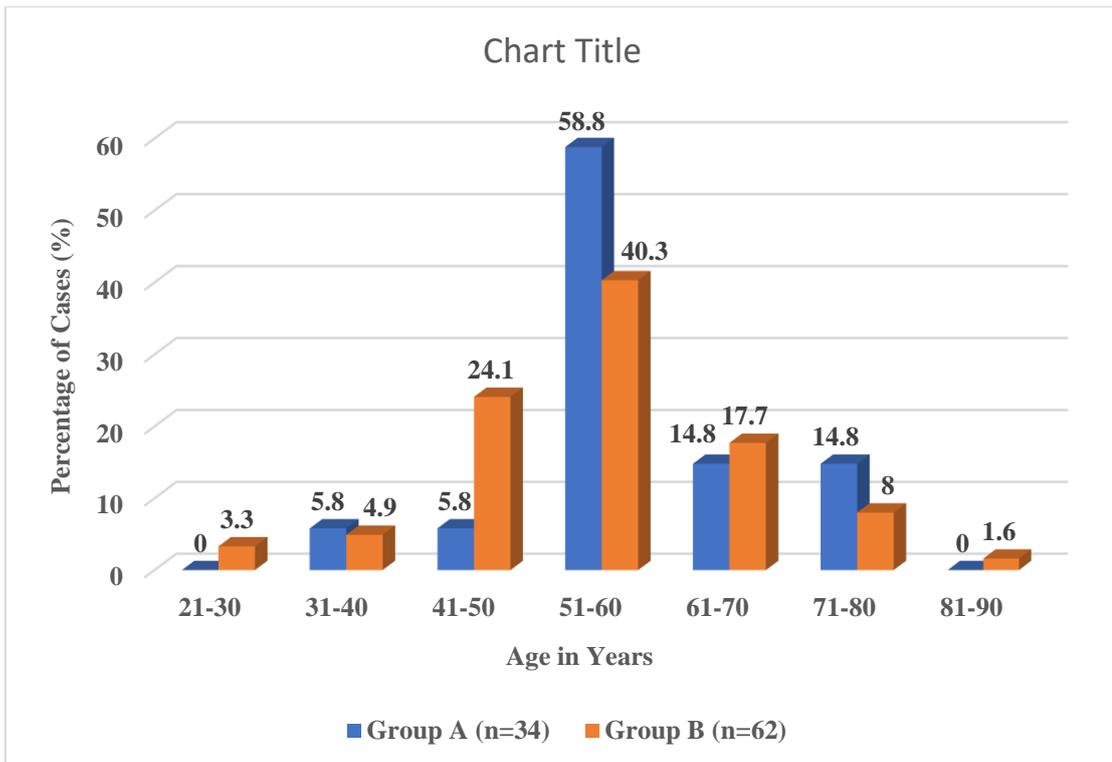
Graph 1: Distribution of patients according to age in Group A and Group B

54

SEX DISTRIBUTION

Out of 96 patients in the study,71 patients (73.9%) were male and 25 patients (26.1%) were female. In the study male patients were more than female patients as depicted in Table:6

In Group A 27 (79.4%) patients were male and 7 (20.5%) females, while in group B 44(70.9%) were male and 18(29%) were females as shown in table:7, Graph:2

Table 6: Distribution of Sex Among All Cases.		
Sex	No.	%
Male	71	73.9
Female	25	26.1
Total	96	100

Gender	Group A		Group B		Total	
	No.	%	No.	%	No.	%
Male	27	79.4	44	70.9	71	73.9
Female	7	20.5	18	29.0	25	26.04
Total	34	100.0	62	100.0	96	100.0

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

$X^2 = 0.81$,

$p = 0.37$, Not significant.
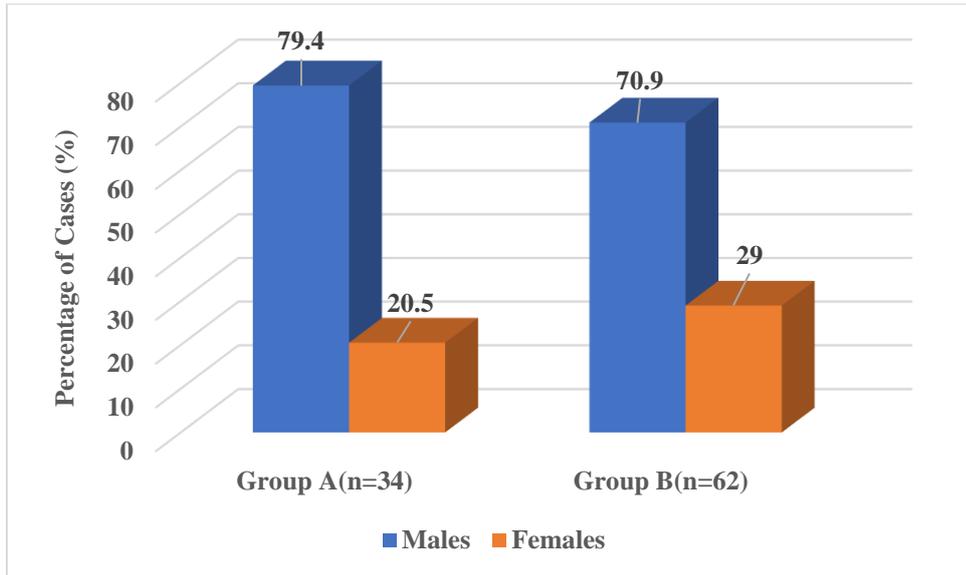
Graph 2: Distribution of patients according to gender wise

DISTRIBUTION OF PATIENTS ACCORDING TO OCCUPATION:

In group A there were 16 (47.1%) farmers followed by business 9 (26.4%), housewife 7(20.5%), labourers were 2 (5.8%), while in group B there were 24 (38.7%) farmers followed by housewife 18(29.03%), business 13(20.9%), labourers 5 (8.06%), teacher 1(1.6%), tailor 1 (1.6%).

`The most common occupation associated with positive T wave in this study was farming followed by business, housewife and labourers as depicted by table:8, 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 (47.1%) and (38.7%) respectively.

Table 8: Occupation wise distribution in Group A and Group B						
	Group A		**Group B**		**Total**	
Occupation	**No.**	**%**	**No.**	**%**	**No.**	**%**
Business	9	26.4	13	20.9	21	22.5
Farmer	16	47.1	24	38.7	39	40.6
Housewife	7	20.5	18	29.03	27	28.1
Labourer	2	5.8	5	8.06	7	7.2
Teacher	0	0.0	1	1.61	1	1.04
Tailor	0	0.0	1	1.61	1	1.04
Total	34	100.0	62	100.0	96	100.0

$X^2 = 2.50$, P = 0.87, NS
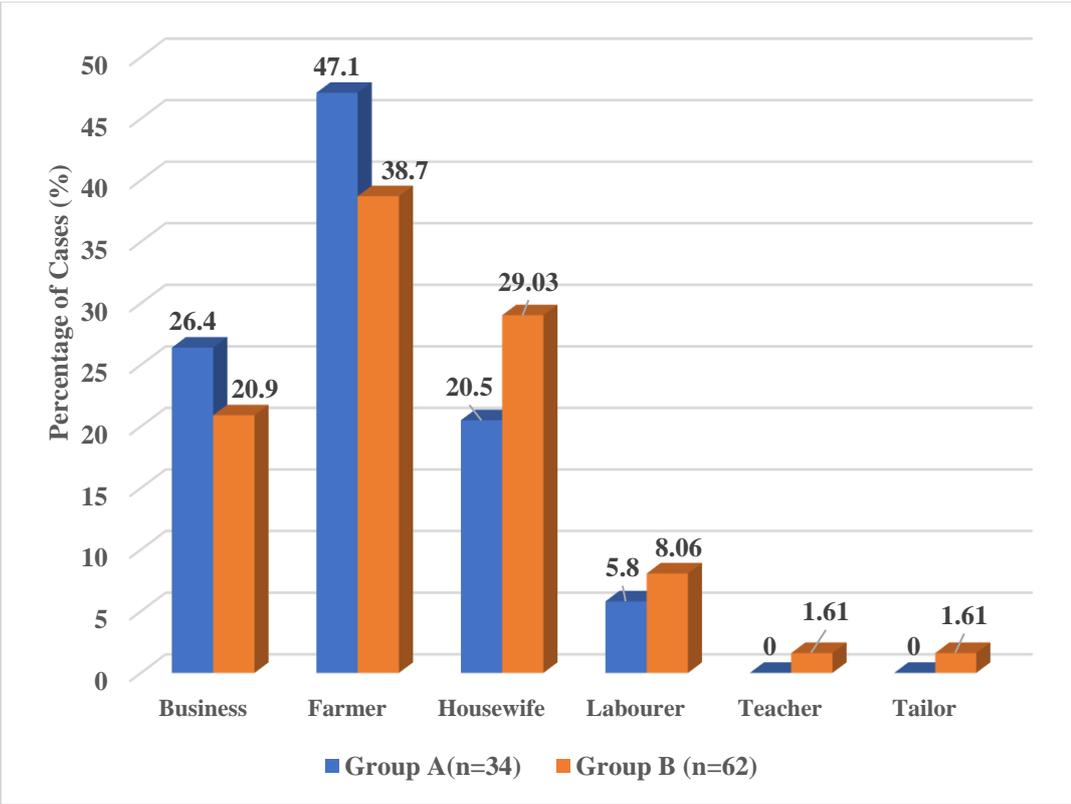
Graph 3: Occupation wise distribution of patients in Group A and Group B

DISTRIBUTION OF PATIENTS ACCORDING TO RISK FACTORS.

Among risk factors, out of 96 patients in the study, 29 patients (85.2%) in group A were compared to 42 patients (67.7%) in group B were aged more than 50 years. Male sex was seen in 27 patients (79.4%) compared to 44 patients (70.9%) in group B. Smoking was seen in 42 patients of which 22 patients (35.4%) were in group B who had negative T wave in lead aVR and 20 patient (58.8%) in group A who had positive T wave in lead aVR. Hence the incidence of positive T wave in lead aVR was more in smokers with statistically significant P value of 0.03. Hypertension was seen in 35 patients of which 14patients (41.7%) were in group A and 21 patients (33.8%) in group B. Diabetes was seen in 42 patients of which 18 patients (52.9%) were in group A and 24 patients (38%) in group B Among tobacco chewing was seen in 13 patients of which 2 patients (5.8%) were in group A and 11 patients (17.7%) were in group B. Alcohol consumption was seen in 22 patients of which 7 patients (20.5%) were in group A and 15 patients (24.1 %) in group B as shown in Table :9, Graph : 4, 5.

Table 9: Distribution of Risk factors in Group A and Group B.							
Risk factors			**Group A**		**Group B**		**Group A vs Group B**	
			No.	**%**	**No.**	**%**	**X²**	**P value**
Non-modifiable	**Age**	**> 50 yrs.**	29	85.2	42	67.7	3.51	0.06
	Sex	**Male**	27	79.4	44	70.9	0.81	0.37
		Female	7	20.5	18	29		
	Family H/o		5	14.7	13	20.9	0.57	0.45
Modifiable	**Diabetes**		18	52.9	24	38	1.81	0.18
	Hypertension		14	41.7	21	33.8	0.51	0.48
	Smoking		20	58.8	22	35.4	4.86	0.03*
	Alcohol		7	20.5	15	24.1	0.16	0.69
	Tobacco chewing		2	5.8	11	17.7	2.64	0.10

X^2: Chi-square test

*P < 0.05, significant
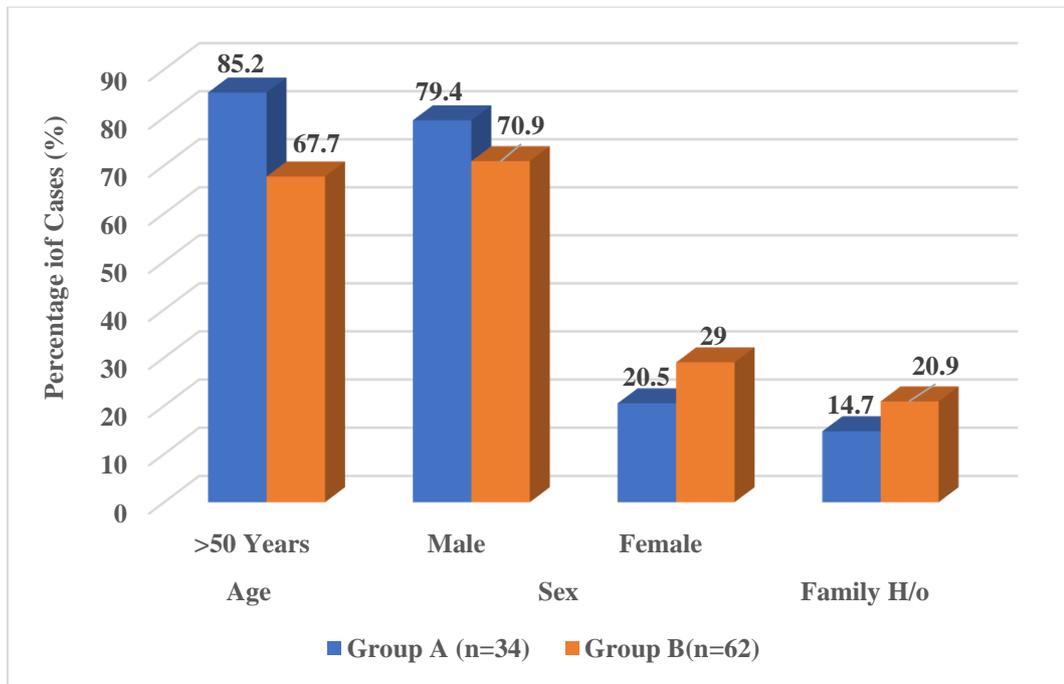
Graph 4: Distribution of non-modifiable Risk factors in Group A and Group B
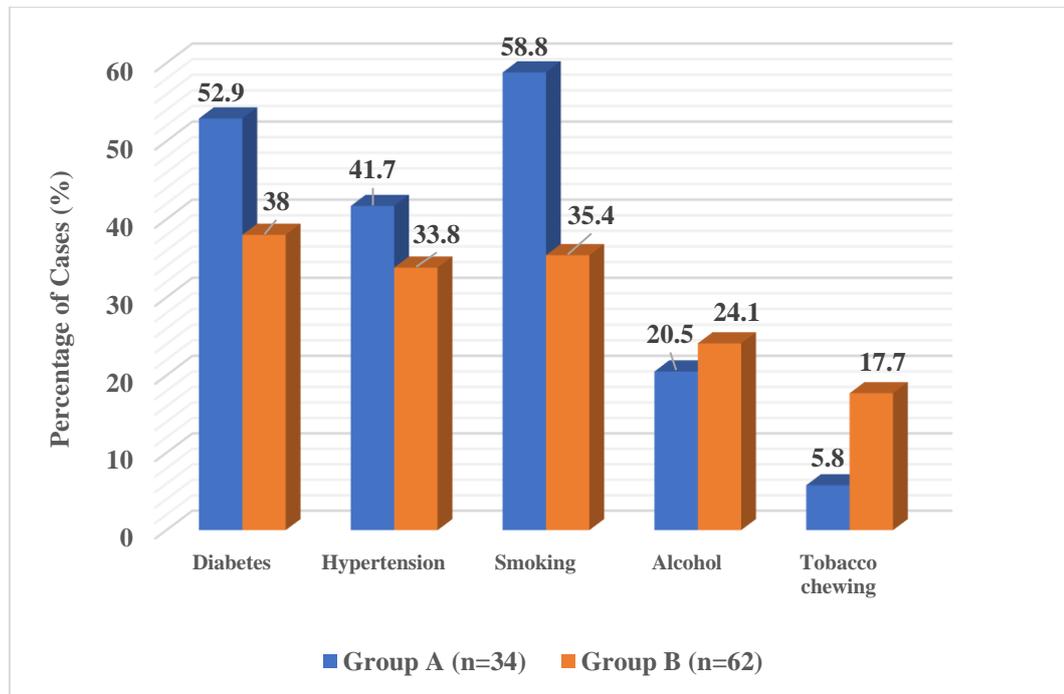
Graph 5: Distribution of modifiable Risk factors in Group A and Group B

DISTRIBUTION OF PATIENTS ACCORDING TO SYMPTOMS

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

In both group A and group B the most common symptom was chest pain (85.2% vs 88.7%), followed by dyspnea (73.5% vs 61.2%), syncope (41.1% vs 17.7%), palpitation (26.4% vs 12.9%), abdominal pain (11.7% vs 12.9%).

Table 10: Distribution of symptoms in Group A and Group B					
Symptoms	**Group A**		**Group B**		**Group A vs Group B**	
	No.	**%**	**No.**	**%**	**X²**	**P value**
Chest Pain	29	85.2	55	88.7	0.23	0.63, ns
Dyspnea	25	73.5	38	61.2	1.46	0.23, ns
Abdominal Pain	4	11.7	8	12.9	0.03	0.87, ns
Palpitation	9	26.4	8	12.9	2.77	0.10, ns
Syncope	14	41.1	11	17.7	6.26	0.06, ns

X^2: Chi-square test

$P > 0.05$, Not Significant.
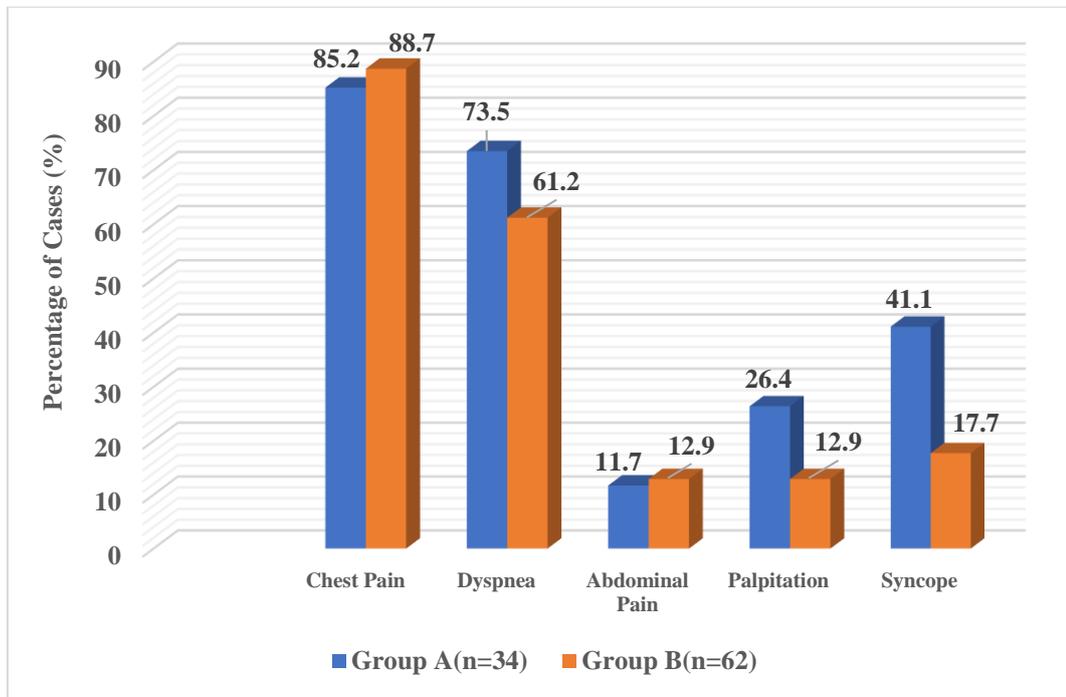
Graph 6: Distribution of symptoms in Group A and Group B

DISTRIBUTION OF PATIENTS ACCORDING TO ECHOCARDIOGRAPHIC(ECG) FINDINGS.

Out of 34 patients with ST-segment elevation in group A, anterior leads (V3-V4) ST-segment elevation was seen in 11 patients (32.3%), antero-septal leads (V1-V4) ST-segment elevation was seen in 9 patients (26.4%), inferior leads (II, III, aVF) ST-segment elevation was seen in 14 patients (41.1%). The most common ST- segment elevation was seen in inferior leads (II, III, aVF) in group A and group B as depicted in Table:11, Graph :7

Table 11: Distribution of ST Segment elevation in Group A and Group B						
ST-segment	**Group A**		**Group B**		**Total**	
	No.	**%**	**No.**	**%**	**No.**	**%**
V3, V4 (Anterior wall STEMI)	11	32.3	19	30.6	30	31.2
V3, V4, I, AVL, V5, V6 (Antero-lateral wall STEMI)	0	0.0	7	11.2	7	7.3
V1, V2, V3, V4 (Antero-septal wall STEMI)	9	26.4	12	19.3	21	21.9
II, III, AVF (Inferior wall STEMI)	14	41.1	24	38.7	38	39.6
Total	34	100.0	62	100.0	96	100.0

$X^2 = 4.40$, P = 0.22, NS
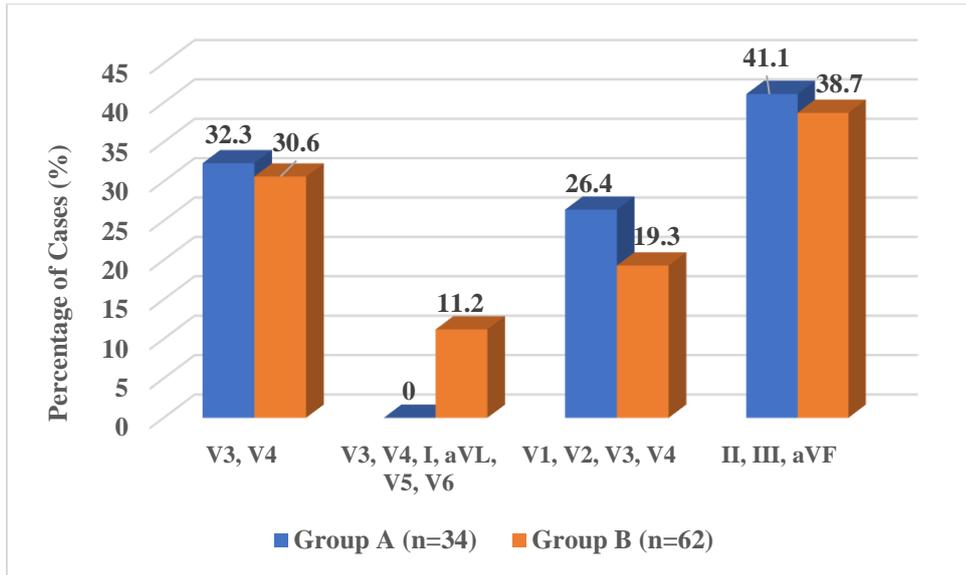
Graph 7: Distribution of ST-Segment elevation in Group A and Group B

DISTRIBUTION OF PATIENTS ACCORDING TO ECHOCARDIOGRAPHIC VARIABLES:

In this study of 96 patients, echocardiographic parameters were analyzed. Out of 34 patients in Group A, 2 patients (5.8%) had antero-lateral wall hypokinesia, 5 patients (14.7%) had anterior wall hypokinesia, 16 patients (47%) had antero-septal wall hypokinesia and 11 patients (32.3%) had inferior wall hypokinesia. Out of 62 patients in Group B, 8 patients (12.9%) had antero-lateral wall hypokinesia, 11 patients (17.7%) had anterior wall hypokinesia, 19 patients (30.6%) had anteroseptal wall hypokinesia and 24 patients (38.7%) had inferior wall hypokinesia. In this study most commonly, there was hypokinesia of antero-septal wall   in group A and inferior wall hypokinesia in group B as shown in Table: 12, Graph:8

Table 12: Distribution of Regional Wall motion abnormality in Group A and Group B						
Regional Wall Motion Abnormality	**Group A**		**Group B**		**Total**	
	No.	**%**	**No.**	**%**	**No.**	**%**
Anterolateral Wall Hypokinesia	2	5.8	8	12.9	10	10.5
Anterior Wall Hypokinesia	5	14.7	11	17.7	16	16.7
Anteroseptal Wall Hypokinesia	16	47.0	19	30.6	35	36.4
Inferior Wall Hypokinesia	11	32.3	24	38.7	35	36.4
Total	34	100	62	100	96	100
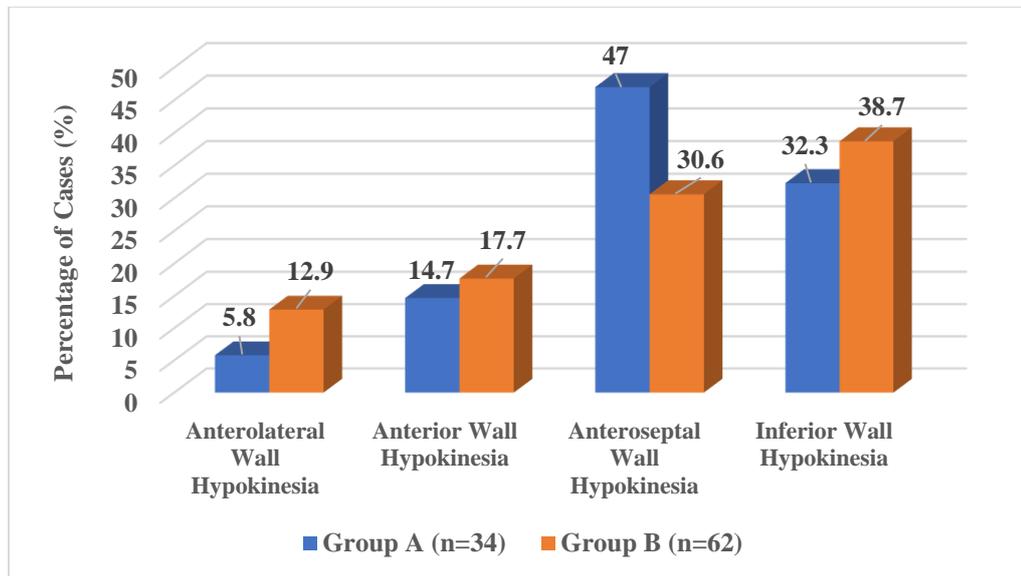
Graph 8: Distribution of Regional Wall motion abnormality in Group A and Group B

DISTRIBUTION OF PATIENTS ACCORDING TO LEFT VENTRICULAR EJECTION FRACTION

In this study of 96 patients, they were divided into group A and group B, and distribution of left ventricular ejection fraction was studied as shown in Table:13, Graph :9

In group A (positive T wave in lead aVR) with 34 cases distribution of left ventricular ejection fraction according to regional wall motion abnormality showed LVEF of < 40% in 23 patients (67.6%) and >40% in 11 patients (32.3%). While in group B (positive T wave absent cases) with 62 cases distribution of left ventricular ejection fraction according to regional wall motion abnormality showed LVEF of <40% in 37 patients (59.6%) and >40% in 25 patients (40.3%).

Table 13: Distribution of Left Ventricular Ejection Fraction (%) in Group A and Group B						
LVEF	**Group A**		**Group B**		**Group A vs Group B**	
	No.	**%**	**No.**	**%**	**X²**	**P value**
< 40%	23	67.6	37	59.6	0.60	0.44, ns
> 40%	11	32.3	25	40.3		
Total	34	100.0	62	100.0	-	

X^2: Chi-square test

$P > 0.05$, NS
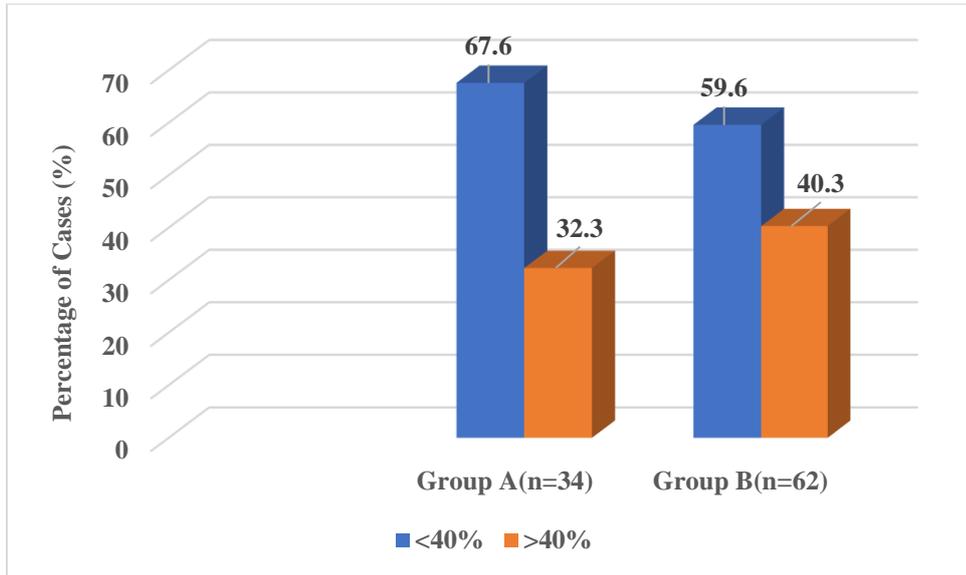
Graph 9: Distribution of LVEF (%) in Group A and Group B

Parameter		Group A		Group B		Group A vs Group B	
		Mean	SD	Mean	SD	T	P value
Vitals	PR (Beats per minute)	86.7	27.3	87.4	19.1	-0.12	0.9
	BP (mmHg)	110.5	30.3	108.2	25.9	0.32	0.75
	RR (Cycles per minute)	25.6	6.6	21.9	5.8	2.24	0.027* S
	Temperature (Degree Celsius)	37.5	0.7	37.6	0.8	-0.29	0.78
Hemoglobin(gm%)		13.9	2.1	13.4	2.5	0.71	0.48
Total count(cells/cu.mm)		13953	3494.6	11946	5175.5	1.44	0.15
ESR (mm/hr.)		21.3	14.4	25.3	25.2	-0.57	0.57
RBS (mg/dl)		209.5	82.3	205.6	103.1	0.14	0.89
Blood Urea (mg/dl)		35.5	21.5	28.9	13.1	1.61	0.11
Serum Creatinine (mg/dl)		1.12	0.38	0.89	0.3	2.65	0.009* S
Serum Sodium (mmol/l)		133.9	5.2	136	4.4	-1.66	0.1
Serum Potassium (mmol/l)		4.39	0.43	4.18	0.6	1.27	0.21

Table 14: Background parameters in Group A and Group B

t: Unpaired t test

* P < 0.05, Sig.

P > 0.05, Not Sig.

DISTRIBUTION OF PATIENTS ACCORDING TO POSITIVE T WAVE IN LEAD aVR AND MAJOR ADVERSE CARDIAC EVENTS

Patients are grouped according to the positive and negative T wave in lead aVR. Out of 96 patients in this study 34 patients had positive T wave in lead aVR (group A) on ECG, the remaining 62 had negative T wave in lead aVR (group B). Among these groups, as depicted in Table:15, Graph:10

Heart failure was found in 20 patients (58.8%) with positive T wave in lead aVR as compared to 21 patients (33.8%) in group B with negative T wave. Pulmonary oedema was present in 23 patients (67.6%) in group A compared to 24 patients (38.7%) in group B. Cardiogenic shock was present in 18 patients (52.9 %) in group A compared to 29 patients (46.7%) in group B. In total 5 patients (14.7%) died in group A compared to 6 patients (9.6%) in group B.

In this study major adverse cardiac events like heart failure (p value of 0.02), pulmonary oedema (p value of 0.01) and arrhythmia (p value of 0.04) were higher in patients with positive T wave in lead aVR (group A) with p value < 0.05 which is statistically significant.

Table 15: Major adverse cardiac events in Group A and Group B.						
Events	**Group A**		**Group B**		**Group A vs. Group B**	
	No.	**%**	**No.**	**%**	**X²**	**P value**
Heart Failure	20	58.8	21	33.8	5.59	0.02*, S
Pulmonary Oedema	23	67.6	24	38.7	7.36	0.01*, S
Cardiogenic Shock	18	52.9	29	46.7	0.34	0.56, ns
Arrhythmia	7	20	3	4.9	5.84	0.02*, S
Deaths	5	14.7	6	9.6	0.55	0.46, ns

* P < 0.05, Sig.

P > 0.05, not sig.
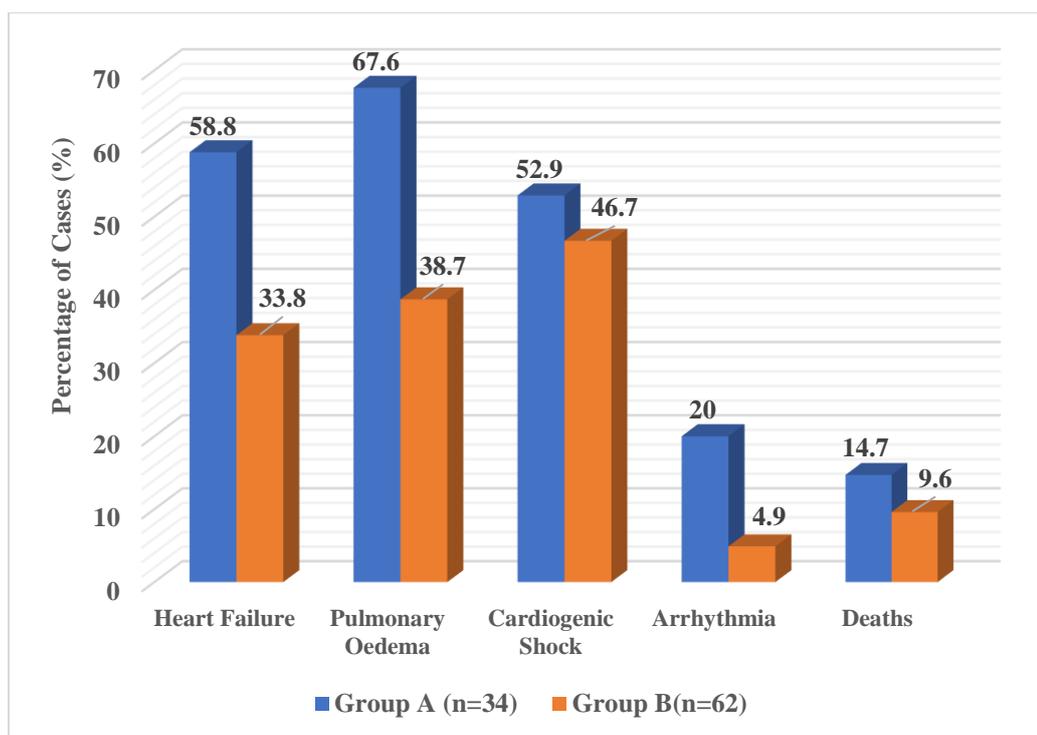
Graph 10: Major adverse cardiac events in Group A and Group B

Statistical analysis: Results are expressed as mean, SD and range values for continuous measurements and frequencies as number & percentages. Unpaired t test was used to compare the means of two groups. Categorical data was analyzed by Chi-square test. A p value of 0.05 or less was considered for statistical significance. SPSS (Version, 17) software was used for data analysis.

DISCUSSION

DISCUSSION

This study is a prospective observational study conducted from November 2019 to June 2021.The aim was to study positive T wave in lead aVR as a predictor of major adverse cardiac events in patients with ST-Segment elevation myocardial infarction.

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

AGE

In this study the most common age group was 51-60 yrs. Similarly in a study done by Ayhan E *et al*, in 2013, on 169 patients, they found that common presenting age group was 50-60yrs, which was similar to this study and mean age was 58% .[21]

Another study done by Kobayashi A *et al* on 190 patients hospitalized with acute coronary syndrome between years January 2007- December 2013, they observed that common age group was 60-70yrs.[10]

In a study done by Aygul N *et al*, mean age group was found to be 60-70 yrs.[19]

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[60]. The reason could be lack of education about disease and in risk factors, evidence-based treatment, lack of compliance of medications.

SEX

In this study there was male predominance as 71 patients (73.9%) were males and 25 patients (26.1%) were female patients which was similar to a study done by Shinozaki K *et al*, in a year 2011 on 122 patients where male patients were 96 and 26 were

women patients where there was male predominance[.20]   In another study done by Aygul N *et al*, on patients with 950 where 742 were men and 208 were women which was significantly higher than this study.[19]

OCCUPATION

In this study the most common occupation associated with ST- segment elevation myocardial infarction in both Group A and Group B was farmer 16 (47.1%) and 24 (38.7%) respectively.

In group A farmers were followed by businessmen 9(26.4%), housewife 7(20.5%), labourer was 2 (5.8%) while in group B, farmers were followed by housewife 18(29.03), businessmen 13 (20.9%), labourer 5(8.06%). Most of these patients belong to low and middle socio-economic 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.

RISK FACTORS

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

In this study, modifiable risk factors like smoking were present in 20 patients (58.8%) in group A and 22 (35.4%) in group B. Hence with a significant p value of 0.03, smoking and positive T Wave in lead aVR shows strong association. Hypertension was seen in 14 patients (41.7%) in group A and 21 patients (33.8%) in group B. Diabetes mellitus was found in 18 patients (52.9%) in group A and 24 patients (38%) in group B. Other risk factors like tobacco chewing in patients (5.8% vs 17.7%), alcohol consumption in (20.5% vs 24.1%) was seen in both group A and group B respectively. There is a significant variation in various risk factors and their association with acute coronary syndrome in different studies. In a study done by Ayhan E *et al* on 169 patients

with acute coronary syndrome, diabetes mellitus was present in 16.9% (n=53), smoker 60.3%(n=53), hypertension in 50.9%(n=53).[21]

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[61]. 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[62]. This study had 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[63]. 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.

SYMPTOMS

In this study, in both group A and group B, the most common symptom was chest pain (85.2% vs 88.7%) followed by dyspnea (73.5% vs 61.2%), syncope (41.1% vs 17.7%), palpitations (26.4% vs 12.9%), abdominal pain (11.7% vs 12.9%). 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 was similar to our study[64].

In other study done by J G Conto *et al.* on 4,34,877 patients admitted with acute myocardial infarction from June 1994 to March 1998 in the National Registry of Myocardial Infarction 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[65].

ELECTROCARDIOGRAM

In this study out of 96 patients showing ST- segment elevation on electrocardiogram, anterior leads (V2-V5) ST-segment elevation was seen in 30 patients, antero-septal leads (V1-V4) ST-segment elevation was seen in 21 patients, antero-lateral (V3-V6, I, aVL) leads ST segment elevation was seen in 7 patients, inferior leads (II, III, aVF) ST-segment elevation was seen in 38 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[66].

In this study positive T wave in lead aVR was present in 34 patients with ST segment elevation which is 35.4% of total patients.

ECHOCARDIOGRAPHY

In this study of 96 patients, echocardiographic parameters like regional wall motion abnormality and ejection fraction had been studied. In this study most commonly, there was hypokinesia of antero-septal wall in group A (47%) and inferior wall hypokinesis in group B (38.7%). while in group A around 67.6 % patients had ejection fraction less than 40% compared to 59.6% in group B. Therefore, in patients with positive T wave

in lead aVR there is higher incidence of reduced ejection fraction and depressed left ventricular systolic function.

MAJOR ADVERSE CARDIAC EVENTS

In this study it was observed that positive T wave in leads aVR (Group A) in patients with ST-segment elevation myocardial infarction as a predictor of in-hospital outcome of the major adverse cardiac events like heart failure was seen in 20 patients (58.8%) which was statistically significant with p value (0.02), pulmonary edema in 23 patients (67.6%) which was also statistically significant of p value (0.01),cardiogenic shock in 18 patients (52.9%), arrhythmia in 7 patients (20%) was again statistically significant with p value of (0.02) and deaths in 5 patients (14.7%) with p value of 0.46 was seen. Whereas in patients with negative T wave in lead aVR (Group B) heart failure was seen in 21 patients (33.8%), pulmonary edema was seen in 24 patients (38.7%), cardiogenic shock in 29 patients (46.7%), arrhythmias in 3 patients (4.9%) and deaths in 6 patients (9.6%). Patients with positive T wave in lead aVR (Group A) on 12 lead electrocardiographs had worse prognosis in terms of major adverse cardiac events like heart failure, pulmonary edema, cardiogenic shock, arrhythmias and death. So, in general, patients with negative T wave in lead aVR (Group B) had good outcome.

CONCLUSION

CONCLUSION

The importance of a positive T wave in a lead aVR in cardiology is far more than is currently recognized. And, as we all know, electrocardiography is a simple, inexpensive, and non-invasive way to assess cardiac issues. There was an elevated risk of in-hospital major adverse cardiac outcomes like heart failure, pulmonary edema, cardiogenic shock, and mortality in patients with positive T wave in lead aVR on electrocardiograph in the current study of patients with ST-segment elevation myocardial infarction. As a result, a positive T wave in lead aVR can be utilized to predict in-hospital major adverse cardiac events in patients with ST-segment elevation myocardial infarction.

SUMMARY

SUMMARY

Ninety-Eight patients with ST-segment elevation myocardial infarction admitted in BLDE (Deemed to be University), Shri B M Patil Medical College Hospital and Research Centre, Vijayapura between November 2019 to June 2021 were studied. This study was conducted to know positive T wave in lead aVR 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 98 patients were studied out of which two were excluded based on exclusion criteria. Rest 96 patients were classified into group A, with positive T wave in lead aVR (34 patients) and group B, with negative T wave (62 patients).

2. In this study male patients (73.9 %) were more than females (26.1%), whereas in group A, 27 male (79.4%) and 7 females (20.5%) compared to 44 male (70.9%) and 18 females (29%) in group B.

3. The most common age group in both group A and group B was between 51-60 years. 20 (58.8%) patients in group A, and 25 (40.3%) in group B were more than 50 years age.

4. The most common risk factors in group A, was smoking (58.8%) with p value of 0.03 which was statistically significant followed by diabetes mellitus (52.9%), hypertension (41.7%1), alcohol (20.5%) and tobacco chewing (5.8%).

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 businessmen, housewife, labourer.

6. The most common symptom in both group A and group B was chest pain followed by dyspnea, syncope, palpitations and abdominal pain.

7. Positive T wave was present in 34 patients with ST- segment elevation that is around 35.5%, while it was most commonly seen in, 41.1% inferior leads (II, III, aVF), followed by 32.3% in anterior (V2-V5), 26.4% in antero-septal (V1-V4).

8. In this study most commonly, there was hypokinesia of antero-septal wall in group A (47%) and inferior wall hypokinesis in group B (38.7%).

9. In group A around 67.6% patients had ejection fraction less than 40% compared to 59.6% in group B. Therefore, in patients with positive T wave in lead aVR 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, pulmonary edema (67.6%) with significant p value <0.005 followed by heart failure (58.8%) with significant p value <0.005, cardiogenic shock (52.9%), arrhythmias (20%) with significant p value <0.005 and death (14.7%) were more common when compared to group B, where it was, cardiogenic shock (46.7 %) followed by pulmonary edema (38.7%), heart failure (33.8%), arrhythmias (4.9%) and death (9.6%).

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).

12. The patients with positive T wave in lead aVR had high incidence of major adverse cardiac events and mortality. Therefore, positive T wave in lead aVR 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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ANNEXURES

ANNEXURE I

INSTITUTIONAL ETHICAL CLEARENCE CERTIFICATE.

B.L.D.E. (DEEMED TO BE UNIVERSITY)

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

The Constituent College

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

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The ethical committee of this college met on 13-11-2019 at 3-15 pm to scrutinize the synopsis of Postgraduate students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected and revised version synopsis of the Thesis has been accorded Ethical Clearance

Title: Positive T wave in lead aVR as a predictor of in-hospital outcome of adverse cardiac events in patients with ST-segment elevationmyocardial infarction

Name of PG student: Dr Hiremath Shruti Basayya, Department of General Medicine,

Name of Guide/Co-investigator: Dr Sharanabasawappa Badiger, Prof & HOD Department of General Medicine

DR RAGHVENDRA KULKARNI

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

Following documents were placed before Ethical Committee for Scrutinization:

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

2

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. HIREMATH SHRUTI BASAYYA of BLDE (Deemed to be University), 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 HIREMATH SHRUTI BASAYYA** informed me that he/she is conducting dissertation/research titled **"POSITIVE T WAVE IN LEAD AVR AS A PREDICTOR OF IN-HOSPITAL OUTCOME OF THE MAJOR 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

99

reference to treatment of other similar cases in near future, and also, I may be benefited in getting relieved of suffering or cure of the disease I am suffering.

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

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

After understanding the nature of dissertation or research, diagnosis made, mode of treatment, I the undersigned Shri/Smt _____ under my full conscious state of mind agree to participate in the said research/dissertation.

Signature of patient:

Signature of doctor:

Witness:

Date:

Place

SCHEME OF CASE TAKING PROFORMA

BLDE (DEEMED TO BE UNIVERSITY)

SHRI B.M. PATIL MEDICAL COLLEGE

HOSPITAL AND RESEARCH CENTRE, VIJAYAPUR.

"POSITIVE T WAVE IN LEAD aVR AS A PREDICTOR OF IN-HOSPITAL

OUTCOME OF THE MAJOR 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:

Genitalia:

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	
Heart blocks	

Lead aVR: ST-segment elevation

T wave – Positive /Negative

ECG Diagnosis:

MAJOR ADVERSE CARDIAC EVENTS: Heart Failure

Arrhythmias

Pulmonary edema

Cardiogenic shock

Death

KEY TO MASTER CHART

AB	:	ABSENT
B	:	BUSINESSMAN
D	:	DEATH
E	:	ELEVATION
EM	:	EMPLOYEE
FA	:	FARMER
F	:	FEMALE
H	:	HOUSEWIFE
HB	:	HEART BLOCK
L	:	LABOUR
LVEF	:	LEFT VENTRICULAR EJECTION FRACTION
M	:	MALE
P	:	PRESENT
SR	:	SINUS RHYTHM
VT	:	VENTRICULAR TACHYCARDIA

MASTER CHART

SL. N.	PATIENT NAME	AGE	SEX	OCCUPATION	IP N.	D.O.A	D.O.D	CHESTPAIN	DYSPNEA	ABDOMINAL PAIN	PALPITATION	SYNCOPE	DIABETES	HYPERTENSION	FAMILY HISTORY	SMOKING	ALCOHOL	TOBACCO CHEWING	PR	BP	RR	TEMPERATURE	HEMOGLOBIN	TOTAL COUNT	ESR	RBS	BLOOD UREA	Sr.CREATININE	Sr.SODIUM	Sr.POTASSIUM	TROPONIN T	CPK MB	ST-SEGMENT ELEVATION	RHYTHEM	POSITIVE T WAVE IN LEAD AVr	NEGATIVE T WAVE IN LEAD AVr	REGIONAL WALL MOTION ABNRMALITY	LVEF	HEART FAILURE	PULMONARY EDEMA	CARDIOGENIC SHOCK	OTHERS	INCLUSION/ EXCLUSION
1	PARASSAPA HONAKERA	52	M	LABOUR	4599	2/11/2019	15/11/2019	P	P	A	A	A	P	P	P	P	A	A	80	110/70	18	38	16	11300	10	243	88	2	136	5	P	P	ANTEROSEPTAL STEMI	SR	yes	No	ANTEROLATERAL WALL HYPOKINESIA	30%	P	P	A		I
2	NINGANAGOUDA PATIL	57	M	Farmer	36705	2/11/2019	4/11/2019	P	P	A	A	P	P	A	A	P	A	A	100	90/70	30	37	15	15450	34	96	24	1	136	4	P	P	ANTEROSEPTAL STEMI	SR	yes	No	ANTEROSEPTAL WALL HYPOKINESIA	40%	P	P	P	D	I
3	SUNANDA NAVADAG	48	F	HOUSE WIFE	30179	9/11/2019	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	P	P	INFERIOR WALL STEMI	SR	Yes	No	ANTERIOR WALL HYPOKINESIA	30%	P	P	A		I
4	ANNASAHAB HOSATI	52	M	FARMER	37723	11/11/2019	16/11/2019	P	P	A	P	P	P	P	P	P	A	A	80	90/70	12	38	14	16220	40	208	27	1	135	4	P	P	ANTEROSEPTAL STEMI	SR	yes	No	ANTEROSEPTAL WALL HYPOKINESIA	25%	P	P	P		I
5	IRAPPA CHALAWADI	65	M	FARMER	3599	1/2/2020	6/2/2020	P	P	A	A	A	A	P	A	P	A	A	102	140/80	21	38	16	13850	15	81	45	1	131	4	P	P	ANTEROSEPTAL STEMI	SR	No	Yes	ANTEROSEPTAL WALL HYPOKINESIA	40%	A	A	A		I
6	PRAKASH SHARANAPPA	60	M	BUISENESS	4443	5/2/2020	10/2/2020	P	P	A	A	A	A	A	A	P	A	A	90	80/60	22	38	12	14270	29	134	32	1	139	5	P	P	ANTEROSEPTAL STEMI	SR	No	Yes	ANTEROSEPTAL WALL HYPOKINESIA	30%	P	P	P		I
7	NAGAPPA AVADI	73	M	BUSENESS	8350	6/3/2020	9/3/2020	P	A	A	A	A	P	P	A	P	A	A	80	80/60	18	38	14	9950	10	279	29	1	134	4	P	P	ANTEROSEPTAL STEMI	SR	No	Yes	ANTEROSEPTAL WALL HYPOKINESIA	25%	P	P	P	D	I
8	NINGAMMA HATTI	52	F	HOUSE WIFE	9951	19/3/2020	23/3/2020	P	P	A	A	A	P	A	P	A	A	A	79	100/70	22	38	11	10410	50	279	16	1	129	4	P	P	INFERIOR WALL STEMI	SR	Yes	No	INFERIOR WALL HYPOKINESIA	35%	P	A	A		I
9	RAMESH ANKALGI	28	M	FARMER	9397	16/3/2020	20/3/2020	P	P	A	P	P	P	P	P	P	A	A	98	80/60	23	38	12	12345	60	145	56	2	134	4	P	P	ANTEROSEPTAL STEMI	SR	No	Yes	ANTEROSEPTAL WALL HYPOKINESIA	30%	P	P	P		I
10	BASAPPA PUJARI	45	M	BUISENESS	10204	22/3/2020	27/3/2020	P	P	A	A	P	P	P	P	P	A	A	78	80/60	18	38	19	15480	10	251	22	1	136	4	P	P	ANTERIOR WALL STEMI	SR	Yes	No	ANTERIOR WALL HYPOKINESIA	30%	P	P	P		I
11	LAGAMANNA BASAPPA	55	M	FARMER	10511	29/3/2020	2/4/2020	P	A	A	A	A	A	A	A	A	A	P	102	80/60	19	38	13	9300	25	149	34	1	124	4	P	P	ANTERIOR WALL STEMI	SR	No	Yes	ANTERIOR WALL HYPOKINESIA	37%	A	A	A		I
12	BASAPPA KOKATNUR	48	M	FARMER	10778	3/4/2020	4/4/2020	A	A	P	P	P	P	P	P	P	A	A	76	100/70	20	38	15	14270	10	265	14	1	133	4	P	P	INFERIOR WALL STEMI	SR	No	Yes	INFERIOR WALL HYPOKINESIA	40%	A	A	A		I
13	BHIMARAYA SIDDAPPA UKKALI	56	M	LABOUR	12391	5/4/2020	7/4/2020	P	P	P	P	P	P	P	P	P	A	A	102	80/40	22	38	10	5630	#	143	34	1	136	5	P	P	ANTERIOR WALL STEMI	SR	No	Yes	ANTERIOR WALL HYPOKINESIA	30%	P	P	P	D	I
14	DANAYYA	52	M	FARMER	11244	12/4/2020	17/4/2020	A	A	P	A	P	P	A	A	P	A	A	65	90/70	16	38	13	12010	10	234	34	1	135	5	P	P	INFERIOR WALL STEMI	SR	Yes	No	INFERIOR WALL HYPOKINESIA	40%	P	P	P	Arrhythmia	I
15	YUNUS HUSSAINSAB MORTUR	25	M	LABOUR	11238	12/4/2020	18/4/2020	P	P	A	A	A	A	P	A	P	A	A	65	110/70	15	38	15	8110	36	213	32	1	134	5	P	P	ANTEROSEPTAL WALL STEMI	SR	Yes	No	ANTEROSEPTAL WALL HYPOKINESIA	45%	A	A	A		I
16	ALLAMMA NADAF	65	F	HOUSE WIFE	11613	17/4/2020	22/4/2020	P	P	A	A	P	P	P	P	A	A	A	89	80/60	22	38	14	34260	10	375	34	1	138	4	P	P	ANTERIOR WALL STEMI	SR	No	Yes	ANTEROSEPTAL HYPOKINESIA	25%	P	P	P		I
17	NINGU YALAMELI	50	M	BUISENESS	11675	18/4/2020	24/4/2020	A	A	A	A	P	P	P	P	P	A	A	120	70/30	19	39	16	17100	5	132	23	1	138	5	P	P	ANTEROSEPTAL AND LATERAL STEMI	SR	No	Yes	INFERIOR WALL HYPOKINESIA	20%	P	P	P	D	I
18	IRAPPA SARWARD	54	M	LABOUR	11669	18/4/2020	25/4/2020	P	P	A	A	A	A	A	A	A	P	A	76	110/70	14	38	14	13040	30	211	34	1	136	4	P	P	ANTERIOR WALL STEMI	SR	No	Yes	ANTERIOR WALL HYPOKINESIA	45%	A	A	A		I
19	RUDRAYYA HIREMATH	50	M	FARMER	11805	20/4/2020	23/4/2020	P	P	P	P	A	P	P	P	P	A	A	78	110/60	14	38	12	10730	10	270	29	1	136	4	P	P	ANTERIOR WALL STEMI	SR	No	Yes	ANTERIOR WALL HYPOKINESIA	30%	P	P	P		I
20	DUSANGAPPA KARIGAR	67	M	FARMER	12184	28/4/2020	1/5/2020	P	P	A	A	P	A	P	A	P	P	A	98	80/30	12	38	15	9880	60	115	38	1	144	4	P	P	ANTERIOR WALL STEMI	SR	Yes	No	ANTERIOR WALL HYPOKINESIA	35%	P	P	P	D	I
21	Mr. LALATH JADHAV	60	F	HOUSE WIFE	12217	29/4/2020	29/4/2020	P	P	A	A	A	P	A	A	A	A	A	118	80/60	19	39	14	12456	70	167	54	1	126	4	P	P	ANTERIOR WALL STEMI	SR	No	Yes	ANTERIOR WALL HYPOKINESIA	30%	A	A	A		I
22	MALLESHAPPA	58	M	FARMER	13493	30/4/2020	2/5/2020	P	P	A	P	A	P	A	A	P	A	A	98	90/70	13	38	12	14356	54	176	34	1	143	4	P	P	ANTERIOR WALL STEMI	SR	Yes	No	ANTERIOR WALL HYPOKINESIA	40%	A	A	A	Arrhythmia	I
23	KAMALABAI PATIL	88	F	HOUSE WIFE	12262	30/4/2020	5/5/2020	P	P	P	P	P	A	A	A	A	A	A	78	110/80	18	39	14	10987	15	156	25	1	135	4	P	P	ANTEROSEPTAL WALL STEMI	SR	No	Yes	ANTEROSEPTAL WALL HYPOKINESIA	50%	A	A	A		I
24	CHINNAMMA HADAPAD #	41	F	HOUSE WIFE	12300	1/5/2020	2/5/2020	P	P	A	A	P	P	P	A	P	A	A	100	90/60	20	38	11	16650	40	560	14	1	133	2	P	P	INFERIOR AND POSTERIOR STEMI	SR	No	Yes	INFERIOR WALL HYPOKINESIA	40%	A	A	P		I
25	SARADAR IMAMSAB MOGAL	68	M	BUISENESS	12308	1/5/2020	4/5/2020	A	A	A	A	A	A	P	A	A	A	A	108	100/70	17	38	13	16012	10	221	21	1	137	4	P	P	INFERIOR WALL STEMI	SR	No	Yes	INFERIOR WALL HYPOKINESIA	35%	A	A	A		I
26	MALLIKARJUN SANGAPPA	56	M	LABOUR	12303	1/5/2020	6/5/2020	P	P	A	A	A	A	A	A	A	A	A	89	100/70	14	38	11	6660	60	118	25	1	147	3	P	P	ANTERIOR WALL STEMI	SR	No	Yes	LATERAL WALL HYPOKINESIA	45%	A	A	A		I
27	BANGAREWWA GUNDAL	60	F	FARMER	12389	3/5/2020	7/5/2020	P	P	A	A	A	P	P	A	P	A	A	84	110/70	18	38	13	6900	20	167	34	1	145	3	P	P	ANTERIOR WALL STEMI	SR	Yes	No	ANTEROSEPTAL WALL HYPOKINESIA	50%	A	A	A		I
28	PARASHURAM SOMANING PUJARI	32	M	BUISENESS	12998	15/5/2020	19/5/2020	P	P	A	A	A	A	A	A	A	A	A	79	110/80	17	39	9.2	9040	#	143	20	1	137	5	P	P	ANTERIOR WALL STEMI	SR	No	Yes	ANTERIOR WALL HYPOKINESIA	35%	A	A	A		I
29	HANUMANTRAY JALAWADI	56	M	FARMER	13003	15/5/2020	18/5/2020	A	P	P	P	P	A	P	A	P	A	A	107	90/60	15	38	16	16520	40	188	34	1	134	4	P	P	ANTERIOR WALL STEMI	SR	Yes	No	ANTEROSEPTAL WALL HYPOKINESIA	35%	A	P	A		I
30	MAHADEVI MALLADI	56	F	HOUSE WIFE	13014	16/5/2020	19/5/2020	P	A	A	A	A	A	A	A	A	A	A	76	100/76	15	38	6.4	9970	75	140	34	1	137	5	P	P	INFERIOR WALL STEMI	SR	No	Yes	INFERIOR WALL HYPOKINESIA	45%	A	A	A		I
31	HUSANSAB KAJASAB	34	M	BUISENESS	13372	22/5/2020	28/5/2020	P	A	A	A	P	A	A	P	P	A	A	98	104/68	16	38	14	10550	40	350	34	1	145	4	P	P	ANTEROSEPTAL STEMI	SR	Yes	No	ANTERIOR WALL HYPOKINESIA	50%	A	P	A		I
32	BABU NATIKAR	45	M	FARMER	13678	27/5/2020	29/5/2020	P	P	A	A	A	A	A	A	P	A	A	87	90/60	16	38	17	21010	5	154	34	1	135	6	P	P	INFERIOR WALL STEMI	SR	No	Yes	INFERIOR WALL HYPOKINESIA	35%	P	P	A	D	I
33	SHASHIDHAR SHETTI	54	M	FARMER	13784	28/5/2020	2/6/2020	P	P	P	P	A	P	P	A	P	A	A	98	100/70	14	38	17	16570	5	625	29	2	125	6	P	P	INFERIOR WALL STEMI	SR	No	Yes	INFERIOR WALL HYPOKINESIA	40%	A	A	A		I
34	NIJLINGAPPA	54	M	BUISENESS	13788	29/5/2020	5/6/2020	P	P	A	A	A	A	A	A	A	A	A	79	110/80	17	39	9.2	9040	#	143	20	1	137	5	P	P	ANTERIOR WALL STEMI	SR	No	Yes	ANTERIOR WALL HYPOKINESIA	35%	P	P	A		I
35	BHAIRAVA MANAGI	56	M	FARMER	13798	29/5/2020	7/6/2020	P	P	A	P	P	P	A	P	A	A	A	107	90/60	15	38	16	16520	40	188	34	1	134	4	P	P	ANTERIOR WALL STEMI	SR	Yes	No	ANTEROSEPTAL WALL HYPOKINESIA	35%	A	P	A		I
36	MAHADEVI	56	F	HOUSE WIFE	13829	2/6/2020	10/6/2020	P	A	A	A	A	A	A	A	A	A	A	76	100/76	15	38	6.4	9970	75	140	34	1	137	5	P	P	INFERIOR WALL STEMI	SR	No	Yes	INFERIOR WALL HYPOKINESIA	45%	A	A	P		I
37	RAMAYYA YALAMELI	50	M	BUISENESS	13849	4/6/2020	12/6/2020	A	P	A	A	A	P	P	P	P	A	A	120	70/30	19	39	16	17100	5	132	23	1	138	5	P	P	ANTEROLATERAL STEMI	SR	No	Yes	ANTERIOR WALL HYPOKINESIA	20%	P	P	P		I
38	DEVAKI MODAGI	45	F	HOUSE WIFE	14293	5/6/2020	10/6/2020	P	P	A	A	A	A	A	A	A	A	A	82	90/60	18	38	12	12600	10	321	23	1	135	3	P	P	ANTEROLATERAL STEMI	SR	No	yes	ANTEROLATERAL WALL HYPOKINESIA	30%	A	A	A		I
39	SANGAPPA #	56	M	FARMER	11021	10/8/2020	16/8/2020	P	A	A	A	A	A	A	A	P	A	A	110	100/70	30	39	17	20100	20	200	26	1	130	4	P	P	INFERIOR WALL STEMI	SR	yes	No	INFERIOR WALL HYPOKINESIA	##	P	P	A	A	I

No	Name	Age	Sex	Occupation	Reg No	Date 1	Date 2	Clinical (A/P)		V1	BP	V2	V3	V4	V5	V6	V7	V8	V9	V10	V11	P1	P2	Diagnosis	Rhythm	Q1	Q2	Echo Findings	EF	P3	P4	P5	Complication	I
40	ALLABHAKSH	63	M	LABOUR	11661	10/10/2020	10/10/2020	A P A A P A A A A A P		48	60/40	34	36	13	15000	20	280	40	1	138	4	P	P	INFERIOR WALL STEMI	SR	No	Yes	INFERIOR WALL HYPOKINESIA	40%	P	P	P	D	I
41	JILANI INAMDAR	55	M	BUISENESS	12199	13-10-2020	20/10/2020	P A A A A A A P A A A		100	150/100	24	36	14	9800	12	157	20	1	136	4	P	P	INFERIOR WALL STEMI	SR	No	Yes	INFERIOR WALL HYPOKINESIA	45%	A	A	A		I
42	DHARU KESHU	51	M	BUISENESS	34586	29/10/2020	5/11/2020	P P A A A A A P P A A		68	130/90	20	37	16	13500		131	42	1	136	4	P	P	ANTERIOR WALL STEMI	SR	Yes	No	ANTERIOR WALL HYPOKINESIA	40%	A	A	A		I
43	ARUN SHARANU DEABERI	27	M	TAILOR	21133	30/11/2020	1/12/2020	P A P A A A A A A A A		76	134/86	22	37	15	10660	10	108	24	1	135	5	P	P	LATERAL WALL STEMI	SR	No	Yes	LATERAL AND APICAL HYPOKINESIA	48%	A	A	A		I
44	BABU GANGARAM	48	M	FARMER	23651	2/12/2020	8/12/2020	P A A A P A A A A A A		98	170/90	20	37	14	9130	10	140	30	1	140	5	P	P	INFERIOR WALL STEMI	SR	No	yes	INFERIOR WALL HYPOKINESIA	48%	A	A	A		I
45	GOMABI	60	F	HOUSE WIFE	24605	6/12/2020	10/12/2020	P A A A A A A A A A A		90	110/70	22	37	13	14250		200	36	1	143	4	P	P	INFERIOR WALL STEMI	SR	No	yes	INFERIOR WALL HYPOKINESIA	45%	A	A	A		I
46	NEELAMMA NAIKODI	60	F	HOUSE WIFE	45327	7/1/2021	10/1/2021	P A A P A P P A A A A		102	160/90	22	37	11	12650	18	200	20	1	140	4	P	P	INFERIOR WALL STEMI	SR	Yes	No	INFERIOR WALL HYPOKINESIA	40%	P	P	A		I
47	SHAMAWWA MANTRI	62	F	HOUSE WIFE	50169	11/1/2021	16/1/2021	P P A A A A A A A A A		96	140/90	18	37	9.6	30400	12	126	28	1	135	4	P	P	LATERAL WALL STEMI	SR	No	Yes	ANTEROLATERAL WALL HYPOKINESIA	40%	P	P	A		I
48	NINGANAGOUDA LINGALEDD	55	M	FARMER	47613	8/1/2021	12/1/2021	P P A A A A A A A A A		60	120/80	18	37	13	8600	10	180	20	1	130	4	P	P	INFERIOR WALL STEMI	SR	Yes	No	INFEROLATERAL WALL HYPOKINESIA	48%	A	A	A		I
49	RAVI ANNAJI KULAKARNI	35	M	TEACHER	50129	10/1/2021	14/1/2021	P P A A A A A A A A A		110	130/90	20	37	15	10060	8	186	12	1	134	4	P	P	ANTEROLATERAL STEMI	SR	No	Yes	ANTEROLATERAL WALL HYPOKINESIA	40%	P	P	A		I
50	SAVITHRI	65	F	HOUSE WIFE	53993	13/1/2021	20/1/2021	P A A A A A A A A A A		78	140/90	18	37	10	12600	14	250	22	1	130	4	P	P	ANTERIOR WALL STEMI	SR	No	Yes	ANTEROLATERAL WALL HYPOKINESIA	30%	A	A	A		I
51	SHABBIR DASTAGIRSAB	48	M	LABOUR	66279	22/1/2021	30/1/2021	P P A A P P A A	A	106	80/60	32	37	15	9800	20	200	70	1	136	4	P	P	ANTERIOR WALL STEMI	SR	No	Yes	ANTEROSEPTAL HYPOKINESIA	20%	P	P	P	A	I
52	SIDDAPPA ANNAPPA INDI	45	M	FARMER	67898	22/1/2021	26/1/2021	P P A A A A A	A	64	130/80	20	37	16	11500	18	92	20	1	139	4	P	P	INFERIOR AND POSTERIOR STEMI	SR	No	Yes	INFERIOR WALL HYPOKINESIA	45%	A	A	A		I
53	HANUMANTH	79	M	FARMER	69224	23/1/2021	23/1/2021	P P A A P A A	A P A	110	90/60	28	37	13	10600	14	260	18	1	140	4	P	P	INFERIOR WALL STEMI	SR	yes	No	ANTEROSEPTAL AND INFERIOR WALL HYPOKINESIA	40%	P	P	P	D	I
54	CHANDAPPA TALWAR	50	M	FARMER	71391	26/1/2021	30/1/2021	P P A A A A A	A	62	90/60	24	37	14	8240	8	180	24	1	138	4	P	P	INFERIOR AND POSTERIOR STEMI	SR	No	Yes	INFERIOR AND POSTERIOR WALL HYPOKINESIA	45%	A	A	A		I
55	NOORJAHA INAMDAR	65	F	HOUSE WIFE	75967	28/1/2021	3/2/2021	P P A A A P A	A	110	120/80	32	37	9.2	10000	18	402	26	2	128	5	P	P	INFERIOR AND POSTERIOR STEMI	SR	No	Yes	INFERIOR WALL HYPOKINESIA	45%	P	P	A		I
56	NEELAKANTA KALLAPPA	69	M	FARMER	74970	28/1/2021	4/2/2021	P P A A A A A	A	130	100/60	30	37	13	8100	6	150	23	1	140	4	P	P	ANTERIOR WALL STEMI	SR	No	Yes	ANTERIOR AND INFEROLATERAL WALL HYPOKINESIA	40%	A	A	A		I
57	RUDRAPPA BHIMAPPA KUMBAR	47	M	FARMER	76681	29/1/2021	3/2/2021	P P A A A P A	A	60	120/80	24	37	14	6800	10	180	12	1	134	5	P	P	INFERIOR WALL STEMI	SR	No	yes	ANTEROSEPTAL, INFERIOR WALL HYPOKINESIA,	45%	A	A	A		I
58	MALLIKARJUN MASALI	55	M	FARMER	77655	29/1/2021	5/2/2021	A P A P A A A	A P A	72	100/60	24	37	14	17180	6	136	31	1	134	4	P	P	ANTERIOR WALL STEMI	IRREGULAR	Yes	No	ANTEROSEPTAL WALL HYPOKINESIA	40%	P	P	P	arrhythmias	I
59	KALLAPPA	48	M	BUISENESS	79103	30/01/2021	3/2/2021	P P A A A P A	A	90	140/90	22	37	14	16330	8	180	20	1	138	4	P	P	ANTERIOR WALL STEMI	SR	No	yes	ANTEROSEPTAL WALL HYPOKINESIA	50%	A	A	A		I
60	D k RAMESH	64	M	BUISENESS	79334	31/01/2021	5/2/2021	P P A A P A P P	A	130	190/120	36	37	12	11790	10	200	30	1	141	4	P	P	ANTERIOR WALL STEMI	SR	yes	No	ANTEROSEPTAL WALL HYPOKINESIA	30%	P	P	A		I
61	SAKREWWA MAHADEV	60	F	HOUSE WIFE	81248	1/2/2021	6/2/2021	P P A A A P P	A	73	128/80	18	38	11	14500	15	240	14	1	132	4	P	P	INFERIOR WALL STEMI	SR	yes	No	INFERIOR WALL HYPOKINESIA	45%	A	A	A		I
62	MALLIKARJUN JAIPAL	33	M	BUISENESS	79496	1/2/2021	4/2/2021	P P A A A A A	A	110	160/90	26	38	11	11940	16	180	59	2	129	5	P	P	INFERIOR WALL STEMI	SR	yes	No	INFERIOR WALL HYPOKINESIA	40%	P	P	A		I
63	RATNABAI	60	F	HOUSE WIFE	85781	4/2/2021	10/2/2021	P P A A P A A	A	76	110/80	26	38	9.7	6690	20	550	102	1	131	6	P	P	ANTERIOR WALL STEMI	SR	No	yes	ANTEROSEPTAL, INFERIOR WALL HYPOKINESIA	20%	P	P	P		I
64	MANGALABAI RATHOD	55	F	HOUSE WIFE	85784	4/2/2021	9/2/2021	P A A A P A A	A	56	80/60	22	38	12	15900	10	260	30	0	133	5	P	P	INFERIOR WALL STEMI	SR	No	yes	INFEROLATERAL WALL HYPOKINESIA	45%	A	A	A		I
65	BHIMASENRAO PATIL	85	M	FARMER	87194	5/2/2021	10/2/2021	P P A A A A A	A	62	130/90	22	38	12	10170	10	320	24	1	132	4	P	P	INFERIOR WALL STEMI	SR	No	yes	INFEROLATERAL WALL HYPOKINESIA	40%	A	A	A		I
66	HANUMANTH PUJARI	51	M	FARMER	90294	8/2/2021	14/2/2021	P P A A P A A	A	90	160/100	20	38	18	8400	10	240	20	1	134	4	P	P	ANTEROSEPTAL STEMI	SR	No	yes	ANTEROSEPTAL HYPOKINESIA	45%	A	A	A		I
67	JAYASINGH MULIMANI	46	M	BUISENESS	90491	8/2/2021	16/2/2021	P P A A A A A	A	86	90/60	24	38	18	9970	15	243	13	1	130	4	P	P	ANTEROSEPTAL STEMI	SR	yes	No	ANTEROSEPTAL WALL HYPOKINESIA	45%	A	A	A		I
68	RAMANNA BIRADAR	55	M	FARMER	95131	12/2/2021	22/2/2021	P P A A P A P A	A	43	80/60	34	38	13	10540	20	410	35	1	136	5	P	P	INFERIOR WALL STEMI	IRREGULAR	yes	No	INFERIOR WALL HYPOKINESIA	20%	P	P	P	arrhythmias	I
69	HUSENABI NABISAB	73	F	HOUSE WIFE	73904	12/2/2021	20/2/2021	P P A A P P A	A	120	80/60	30	38	13	14800	22	280	18	1	131	5	P	P	ANTEROSEPTAL STEMI	SR	No	yes	ANTEROSEPTAL WALL HYPOKINESIA	35%	P	P	P		I
70	MAHANANDA SANGAPPA	63	F	HOUSE WIFE	105613	19/2/2021	23/2/2021	P A A A P P A	A	68	130/80	22	38	12	7200	60	106	14	1	130	5	P	P	LATERAL WALL STEMI	SR	No	yes	INFEROLATERAL WALL HYPOKINESIA	60%	A	A	A		I
71	KALLAPPA PAWAR	75	M	FARMER	105606	19/2/2021	26/2/2021	P A A P A A A	A	40	60/40	26	38	14	6500	30	167	40	1	133	3	P	P	INFERIOR WALL STEMI	SR	No	yes	INFEROLATERAL WALL HYPOKINESIA	45%	A	A	A		I
72	ANNAPURNA HUGAR	75	F	HOUSE WIFE	109429	21/2/2021	1/3/2021	P A P P A A A	A	130	90/60	24	38	13	20600	60	116	72	2	120	5	P	P	ANTERIOR WALL STEMI	IRREGULAR	yes	No	ANTEROSEPTAL WALL HYPOKINESIA	20%	P	P	P	Arrhythmia	I
73	SUSHILA BAI PATTAR	80	F	HOUSE WIFE	116123	25/2/2021	1/3/2021	P P A A A A A	A	110	90/60	26	38	11	16500	15	200	20	1	137	4	P	P	INFERIOR WALL STEMI	SR	No	yes	INFEROLATERAL WALL HYPOKINESIA AND RV FREE WALL	40%	A	A	A		I
74	AMBANNA HUGAR	65	M	FARMER	129039	6/3/2021	11/3/2021	P P A A A A P P	A	90	94/70	22	38	13	9700	30	180	33	1	140	5	P	P	INFERIOR WALL STEMI	SR	No	yes	INFERIOR AND POSTERIOR WALL HYPOKINESIA	45%	A	A	A		I
75	MAHANTESH MADIWALAPPA	40	M	FARMER	143662	16/3/2021	20/3/2021	P P A A A A P	A	86	94/70	24	38	15	11960	10	132	15	1	137	4	P	P	ANTEROSEPTAL STEMI	SR	No	yes	ANTEROSEPTAL WALL AND RV FREE WALL HYPOKINESIA	40%	A	A	A		I
76	SHASHIKALA KISANSING	75	F	HOUSE WIFE	145646	17/3/2021	27/3/2021	P P A A P A P	A	45	80/60	36	37	13	25250	15	186	41	1	139	5	P	P	INFERIOR AND POSTERIOR STEMI	IRREGULR	No	yes	INFEROLATERAL WALL HYPOKINESIA	45%	P	P	P	Arrhythmia	I
77	SHIVANAND	42	M	BUISENESS	2989	2/4/2021	6/4/2021	P A A A A P A P	A	100	160/80	28	37	18	12200	12	150	20	1	138	3	P	P	INFERIOR WALL STEMI	SR	Yes	No	INFERIOR WALL HYPOKINESIA	60%	A	A	A		I
78	ANNAND KUMAR	58	M	BUISENESS	4487	3/4/2021	7/4/2021	P A A A P A P	A	63	170/110	22	37	17	10600	10	138	20	1	138	5	P	P	ANTERIOR WALL STEMI	SR	No	yes	ANTEROSEPTAL HYPOKINESIA	50%	A	A	A		I
79	KASHINATH ROMANNA	60	M	FARMER	22574	15/4/2021	20/4/2021	P A A A P A A P		70	110/70	22	36	14	9100	10	200	33	1	139	4	P	P	INFERIOR WALL STEMI	SR	NO	Yes	INFERIOR WALL HYPOKINESIA	45%	A	A	A		I
80	DASHARAM BABURAO	44	M	BUISENESS	21548	15/4/2021	24/4/2021	P P A A A A A P		108	80/60	24	37	17	6400	4	93	28	1	134	4	p	p	ANTEROSEPTAL STEMI	SR	Yes	No	ANTEROSEPTAL WALL HYPOKINESIA	35%	P	P	P	arrhythmia	I
81	SHANKAREPPA	70	M	FARMER	34178	23/4/2021	27/4/2021	P A A A A A A		72	120/80	24	38	14	7800	24	96	22	1	136	4	P	P	INFERIOR WALL STEMI	SR	No	Yes	INFERIOR WALL HYPOKINESIA	45%	A	A	A		I
82	SUKHADEV RAVJI	60	M	BUISENESS	34104	23/4/2021	30/4/2021	P A A A P P P P		74	110/70	26	37	14	9600	10	250	38	2	139	4	P	P	INFERIOR WALL STEMI	SR	No	Yes	INFERIOR WALL HYPOKINESIA	40%	A	A	A		I
83	LAKSHMIBAI PATIL	55	F	HOUSEWIFE	35468	24/4/2021	1/5/2021	P A A A A A A		86	120/80	24	38	12	7200	12	180	24	1	138	4	P	P	INFERIOR WALL STEMI	SR	Yes	No	ANTEROSEPTAL WALL HYPOKINESIA	45%	A	A	A		I
84	YALLAPPA KATAPPA	55	M	FARMER	42765	30/4/2021	4/5/2021	P A A A A P A		90	100/60	28	38	14	9800	18	240	18	1	140	4	P	P	ANTEROSEPTAL STEMI	SR	No	Yes	ANTEROSEPTAL WALL HYPOKINESIA	45%	A	A	A		I
85	MD RAFIQ WALIKAR	54	M	BUISENESS	58255	15/5/2021	20/5/2021	P A A A P A P A		96	100/70	34	37	15	10200	20	206	19	1	138	4	P	P	LATERAL WALL STEMI	SR	NO	Yes	ANTEROSEPTAL WALL HYPOKINESIA	35%	P	P	A		I
86	BHAGIRATHI GULED	45	F	HOUSEWIFE	60650	18/5/2021	30/5/2021	P A A A A A A		90	90/60	28	36	16	8300	10	130	30	1	130	5	P	P	ANTEROSEPTAL STEMI	SR	NO	Yes	ANTEROSEPTAL WALL HYPOKINESIA	20%	P	P	P	Arrhythmia	I
87	BAMINI SHUVAPUTRAPPA	65	F	HOUSEWIFE	69323	1/6/2021	5/6/2021	P A A P P A A		98	170/100	32	36	15	14900	18	255	21	1	135	4	P	P	ANTEROSEPTAL STEMI	SR	NO	Yes	ANTEROLATERAL WALL HYPOKINESIA	35%	P	P	A	Arrhythmia and death	I
88	KASTURIBAI PEERAPPA	65	F	HOUSEWIFE	72923	5/6/2021	12/6/2021	A A A A P P A		54	100/70	30	36	12	15400	22	160	24	1	138	4	P	P	INTERIOR WALL STEMI	CHB	yes	No	INFEROLATERAL WALL HYPOKINESIA	25%	P	P	P	Death and Arrhythmia	I

89	GURAPPA MANVI	60	M	FARMER	72897	5/6/2021	10/6/2021	P	P	P	A	A	A	A	A	P	A	A	130	140/90	32	37	16	11600	6	110	25	1	140	5	P	P	ANTEROSEPTAL STEMI	SR	NO	yes	ANTEROLATERAL WALL HYPOKINESIA	40%	A	P	A	A	I
90	KALAVATHI CHANDUKAR	42	F	HOUSEWIFE	73178	6/6/2021	13/6/2021	P	A	A	A	A	A	A	A	A	A	A	80	126/80	24	37	13	6200	20	132	18	1	138	4	P	P	ANTERIOR WALL STEMI	SR	NO	yes	ANTEROSEPTAL WALL HYPOKINESIA	35%	A	A	A	A	I
91	DASTAGIR SAB NADAF	60	M	FARMER	80882	16/6/2021	25/6/2021	P	P	A	A	A	P	P	A	A	A	A	92	100/70	28	37	16	12100	15	200	16	1	140	5	P	P	ANTEROSEPTAL STEMI	SR	Yes	No	ANTEROSEPTAL WALL HYPOKINESIA	30%	P	P	P	A	I
92	VITTAL HANAMANTH	59	M	FARMER	83464	19/6/2021	25/6/2021	P	P	A	A	A	A	A	A	P	P	A	90	120/80	26	37	17	9380	12	150	51	1	134	3	P	P	ANTERIOR WALL STEMI	SR	NO	Yes	ANTEROSEPTAL WALL HYPOKINESIA	45%	A	A	A	A	I
93	RAM GUNDAWAN	60	M	FARMER	84543	21/6/2021	26/6/2021	P	P	A	A	A	P	P	A	A	A	A	54	90/60	30	37	15	10200	16	320	30	1	135	5	P	P	ANTEROSEPTAL STEMI	SR	Yes	NO	ANTEROSEPTAL WALL HYPOKINESIA	45%	A	A	P	A	I
94	BASAPPA LONI	79	M	FARMER	85323	22/6/2021	28/6/2021	P	P	A	A	A	A	P	A	P	P	A	58	100/60	28	38	10	2100	14	88	31	1	137	4	P	P	INFERIOR WALL STEMI	SR	NO	Yes	INFERIOR WALL HYPOKINESIA	40%	A	A	A	A	I
95	SHRISHAIL MASALI	60	M	BUISENESS	84598	21/6/2021	24/6/2021	P	P	P	P	P	A	A	A	A	A	A	110	90/60	36	35	17	22100	20	300	32	2	143	4	P	P	ANTERIOR WALL STEMI	SR	Yes	No	ANTEROSEPTAL WALL HYPOKINESIA	30%	P	P	P	D	I
96	RAJASEKHAR	65	M	FARMER	86249	23/6/2021	27/6/2021	P	A	P	A	A	A	A	A	A	A	A	85	120/80	24	38	15	3630	5	125	29	1	139	4	P	P	INFERIOR WALL STEMI	SR	Yes	No	INFERIOR WALL HYPOKINESIA	45%	A	A	A	A	I
97	IRAGONDAPPA UMADI	67	M	FARMER	40719	24/6/2021	28/6/2021	A	A	A	A	P	P	P	A	P	A		84	170/80	26	37	13	10.9	45	116	26	1	137	4	P	P	INFERIOR WALL STEMI	SR	No	Yes	INFERIOR WALL HYPOKINESIA	35%	A	A	A	A	E
98	SADASHIV MADAR	47	M	BUISENESS	30539	25/6/2021	29/6/2021	P	A	A	A	A	A	P	A	P	P	A	90	126/70	24	38	15	8000	10	94	15	1	139	4	P	P	ANTEROIR WALL STEMI	SR	No	Yes	ANTERIOR WALL HYPOKINESIA	63%	A	A	A	A	E