
ROLE OF TROPONIN IN IDENTIFYING LEFT VENTRICULAR
EJECTION FRACTION OF $\leq 40\%$ IN ACUTE STELEVATED
MYOCARDIAL INFARCTION – A ONE YEAR CROSS
SECTIONAL STUDY IN KLE DR. PRABHAKAR KORE
HOSPITAL AND MRC, BELAGAVI.

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
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LIST OF ABBREVIATIONS

µg/L	-	Micro grams per litre
2D	-	2 dimensional
ACC	-	American College of Cardiology
ACEI	-	Angiotensin converting enzymeinhibitors
ACS	-	Acute Coronary Syndrome
AHA	-	American Stroke Association
AMI	-	Acute myocardial infarction
ARBs	-	Angiotensin II receptor blockers
AUC	-	Area under the curve
AV	-	Atrioventricular
AWMI	-	Anterior wall myocardial infarction
BMI	-	Body mass index
CAD	-	Coronary artery disease
CAG	-	Coronary angiography
cc	-	Cubic centimetre
CHD	-	Coronary heart disease
CI	-	Confidence interval
CKMB	-	Creatinine kinase MB
cTn	-	Cardiac troponin
cTnI	-	Cardiac Troponin I
cTnT	-	Cardiac Troponin T
CVD	-	Cardiovascular disease
DM	-	Diabetes mellitus
ECG	-	Electrocardiogram
ECHO	-	Echocardiography
EF	-	Ejection fraction
ESC	-	The European Society of Cardiology
HTN	-	Hypertension
I/R	-	Ischemia/reperfusion
ICD	-	implantable cardioverter-defibrillators
IHD	-	Ischemic heart disease
IP No.	-	In patient number

IQR	-	Interquartile range
IWMI	-	Inferior wall myocardial infarction
kg/m ²	-	Kilograms per square meter
LBBB	-	Left bundle branch block
LDH	-	Lactate dehydrogenase
LV	-	Left ventricular
LVD	-	Left ventricular dysfunction
LVED	-	Left ventricular end diastolic
LVEF	-	Left ventricular ejection fraction
LVMI	-	Left ventricular mass index
MI	-	Myocardial infarction
mm Hg	-	Millimetres of mercury
mm	-	Millimetres
mV	-	Milli volt
n	-	Total number
NPV	-	Negative predictive value
NRMI-4	-	National Registry of Myocardial Infarction-4
p	-	Probability
PAH	-	Pulmonary artery hypertension
PAMI	-	Primary Angioplasty in Myocardial Infarction
PCI	-	Percutaneous primary intervention
PPV	-	Positive predictive value
ROC	-	Receiver operating characteristic
RWMA	-	Regional Wall Motion Abnormality
SA	-	Sinoatrial
SD	-	Standard deviation
SMC	-	Smooth muscle cells
STEMI	-	ST-elevation myocardial infarction
TC	-	Total Cholesterol
Trop	-	Troponin
U/l	-	Units per litre
US	-	United States
WHO	-	World Health Organization

ABSTRACT

Background and objectives

Measurement of LVEF is important from both a prognostic and therapeutic perspective. The present study assessed relationship between Troponin T and LV dysfunction.

Methodology

The present one-year hospital based cross-sectional study was carried out under the Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum from January 2019 to December 2019. A total of 97 patients presenting with STEMI during the study were studied.

Results

Majority of the patients were males (69.07%) with male to female ratio of 2.23:1. Most of the patients (38.14%) of the patients were aged from 61 to 70 years. Majority of the patients (91.75%) presented with chest discomfort. Most of the patients reported history of diabetes mellitus (51.55%) and had BMI from 25.00 to 29.99 Kg/m²(44.33%).ECG findings revealed AAMI in 69.07% of the patients. On 2D echocardiography, mild PAH was noted in 22.68%. The mean and EF was 42.68±5.95% respectively. Further EF of 40% as noted in 45.36% of the patients. The mean troponin T levels were 2.41±3.22 and 1.31 (IQR 2.16) µg/L. Troponin T levels of > 0.010 was noted in all of the patients (100%). The median Troponin T (0.741 [IQR 1.15] vs 2.14 [IQR 6.22] years; p=0.328) levels in patients with normal and low EF were comparable. Moderate significant negative correlation between troponin T and EF ($r = -0.474$; $R^2 = 0.2056$; $p < 0.001$) was noted. Further a cut of value

of 1.28 μ g/L for troponin T has limited sensitivity and specificity in predicting LVEF 40% among the patients with STEMI.

Conclusion and interpretation

There is moderate negative correlation between Troponin T and LVEF as assessed by 2D echocardiography with EF in both AAMI and IWMI subset of STEMI patients.

CONTENTS

SL. NO.	TOPIC	PAGE NO.
1.	INTRODUCTION	1-3
2.	OBJECTIVES	4
3.	REVIEW OF LITERATURE	5-27
4.	METHODOLOGY	28-31
5.	RESULTS	32-49
6.	DISCUSSION	50-57
7.	CONCLUSION	58
8.	SUMMARY	59-61
9.	BIBLIOGRAPHY	62-73
10	APPENDICES	
	I - ETHICAL CLEARANCE CERTIFICATE	74
	II – CONSENT FORM	75-86
	III – PROFORMA	87-90
	IV – MASTER CHART	91

LIST OF TABLES

TABLE NO.	DESCRIPTION	PAGE NO.
1	Clinical characteristics of the study population	32
2	Distribution of patients according to the ejection fraction	33
3	Distribution of patients according to the sex and its association with EF	34
4	Distribution of patients according to the age and its association with EF	35
5	Distribution of patients according to the clinical presentation and its association with EF	36
6	Distribution of patients according to the risk factors and its association with EF	37
7	Distribution of patients according to the BMI and its association with EF	38
8	Distribution of patients according to the ECG findings and its association with EF	39
9	Distribution of patients according to the pulmonary artery pressure and its association with EF	40
10	Distribution of patients according to the pericardial effusion and its association with EF	41
11	Distribution of patients according to the troponin T levels	42
12	Comparison of clinical profile with EF	43
13	Descriptive data on ECG leads	48
14	Distribution of patients according to the management	49

LIST OF GRAPHS

GRAPH NO.	DESCRIPTION	PAGE NO.
1	Distribution of patients according to the ejection fraction	33
2	Distribution of patients according to the sex and its association with EF	34
3	Distribution of patients according to the age and its association with EF	35
4	Distribution of patients according to the clinical presentation and its association with EF	36
5	Distribution of patients according to the risk factors and its association with EF	37
6	Distribution of patients according to the BMI and its association with EF	38
7	Distribution of patients according to the ECG findings and its association with EF	39
8	Distribution of patients according to the pulmonary artery pressure and its association with EF	40
9	Distribution of patients according to the pericardial effusion and its association with EF	41
10	Distribution of patients according to the troponin T levels	42
11	Comparison of clinical profile with EF	43

12	Correlation between ejection fraction and troponin T	44
13	Correlation between ejection fraction and troponin T in patients with IWMI	45
14	Correlation between ejection fraction and troponin T in patients with AWTMI	46
15	Receiver operating curve for EF of 40%	47
16	Descriptive data on ECG leads	48
17	Distribution of patients according to the management	49

LIST OF FIGURES

FIGURE NO.	DESCRIPTION	PAGE NO.
1	Mortality per lakh of population by CVD and Type 2 Diabetes	9
2A	CVD mortality as a proportion of total mortality	10
3	Atherosclerotic plaque	14
4	Standard 12 lead ECG showing ST elevated MI (in leads V1-V4)	17
5	Cardiac Biomarkers and their trend in MI	19
6	Time course of blood levels of cardiac markers after acute coronary syndromes	24

INTRODUCTION

Coronary artery disease (CAD) is the leading cause of death worldwide.¹ The prevalence of this disease in the Indian population is estimated to be three crore.² When adjusted to age, the cardio vascular death rate was found to be 272 per 100000 in the population of our country as opposed to the global average of 235.³ Coronary Artery Disease (CAD) is the leading causes of heart failure and death worldwide. Acute cardiac events that may lead to acute myocardial infarction (AMI) and sudden cardiac death are unpredictable.⁴

Myocardial infarction remains a major clinical problem and the leading causes of mortality in the world. Myocardial infarction is “myocardial cell death due to prolonged ischemia”.⁵ In the United States (US) alone, approximately one million people suffer MI each year.⁶ The incidence of MI in India is 64.37/1000 people⁵ in men aged 29-69 years.⁷ The most common cause of MI is coronary atherosclerotic plaque rupture or erosion, resulting in the exposure of thrombogenic contents to the blood. This leads to thrombus formation and consequently MI.⁸

ST-elevation myocardial infarction (STEMI) continues to be a significant public health problem in industrialized countries and is becoming an increasingly significant problem in developing countries. Although the exact incidence is difficult to ascertain, using first-listed and secondary hospital discharge data, there were 1,680,000 unique discharges for ACS in 2001.¹¹ Applying the conservative estimate of 30% of the ACS patients who have STEMI from the National Registry of Myocardial Infarction-4 [NRMI-4],⁹ 500,000 STEMI events per year are estimated in the U.S. Inferior acute myocardial infarction (AMI) represents 40–50% of all AMIs.

The prevalence of STEMI is increasing in India¹⁰ as nearly thirty lakh ST segment elevated myocardial infarction cases happen annually in India.^{3,12}

Acute STEMI usually occurs when thrombus forms on a ruptured atheromatous plaque and occludes an epicardial coronary artery.¹³ Since a long time the 12-lead ECG is considered an essential part of the diagnosis and initial evaluation of patients with chest pain. It plays a crucial role in identifying the infarct-related artery and it is economical and universally available.¹⁴ The patients can be classified based on the wall affected by observing the ST segment deflection and a rough gauging of the risk can be made by looking at the extent of deflection and QRS complex width.¹⁵

Cardiac troponin (cTn) is released from cardiomyocytes exposed to ischemia severe enough to cause irreversible cell damage.¹⁶ Compared to old conventional markers of the heart, cardiac troponins (T and I) have greater sensitivity for damage to the myocardium.^{17,18} Troponins augment the diagnosis made on clinical and ECG findings and have usefulness in prognostication of the disease.^{19,20} Hence, various international bodies such as The European Society of Cardiology (ESC) and American College of Cardiology (ACC) now endorse troponin in diagnosing Acute Myocardial Infarction.²¹ cTn release has predictable kinetics in the setting of AMI, and peak levels correlate with both ischemic time and quantity of myocardium at risk.²² A previous investigation has shown that troponin kinetics can be used to estimate the onset of ischemia in STEMI patients, and this “biochemical ischemic time” correlates well with infarct size and long-term mortality.^{22,23}

Need for the study

After AMI, a patient's prognosis is closely related to the extent of irreversibly damaged myocardium.²⁴ Though cTn has high sensitivity and specificity for diagnosing AMI, we should also be aware of other states in which it might be raised, prior to arriving at a diagnosis.

Left ventricular (LV) systolic function is predictive of early and late complications and mortality in patients with STEMI. Echocardiography is valuable, non-invasive method for evaluating left ventricular wall motion. Despite the potential value of troponin as an indicator of prehospital ischemia and myocardial damage, there is a paucity of research to substantiate this claim. Furthermore, currently there is no WHO standardization for troponin due to the use of different antibodies in the test²⁵ raising further ambiguity. In this context, the present study was aimed to provide better understanding and quantification of the relation between troponin and ejection fraction of the left ventricle in patients with STEMI which is likely help in predicting the early, late complications and mortality.

Research question

Whether troponin levels in patients with STEMI predict LV dysfunction?

OBJECTIVES

The objective of this study was to study Troponin T as non-imaging modality in identifying LV dysfunction.

REVIEW OF LITERATURE

Since the present study is aimed to evaluate the role of troponin T as non-imaging modality in identifying LV dysfunction, literature pertaining to physiology of cardiac function, acute myocardial infarction, STEMI, LV function in STEMI and Troponin is reviewed.

The human heart is a vital organ that continuously circulates blood throughout the body in cycles. The heart rests on the diaphragm, which is near the thoracic cavity. It lies in the mediastinum which is an anatomical region that exit from the sternum to the vertebral column (rib to the diaphragm and between the lungs). Heart always lies to the left of the body's midline about two-thirds. Pericardium surrounds and protects the heart. It helps in confining the heart to its position in the mediastinum, as well as allows sufficient freedom of movement for vigorous and rapid contraction. Three layers of the wall of the heart are the epicardium (external layer), the myocardium (middle layer), and the endocardium (inner layer). The heart has the two superior receiving chambers (known as atria) and the two inferior pumping chambers (known as ventricles).²⁶

PHYSIOLOGY OF CARDIAC FUNCTION

The heart chambers normally contract with properly coordinated manner means they pump blood efficiently by a route determined by the valves. Coordination of contraction is by a specialized conducting system in which impulses arise from the sinoatrial (SA) node and are conducted in sequence through the atria, the atrioventricular (AV) node, bundle of His then Purkinje fibers and ventricles. Heart cells owe their electrical excitability to voltage-sensitive plasma membrane channels

selective for various ions, including Na⁺, K⁺, and Ca⁺. Electrophysiological features of cardiac muscle that distinguish it from other excitable tissues include pacemaker activity, absence of fast Na⁺ current in SA and AV nodes, where slow inward Ca²⁺ current initiates action potentials, long action potential (“plateau”) and refractory period, influx of Ca²⁺ during the plateau. Different phases of the action potential are shown in table below.²⁶

Different phases of the cardiac cycle²⁶

Phase	Name of the phase	Mechanism involved
Phase 0	Rapid depolarization	When the membrane potential reaches a critical firing threshold (about -60 mV), at which the inward current of Na ⁺ flowing through the voltage-dependent sodium channels becomes large enough to produce a regenerative (“all or nothing”) depolarization
Phase 1	Partial repolarization	Occurs as the Na ⁺ current is inactivated. There may also be a transient voltage-sensitive outward current
Phase 2	Plateau	Results from an inward Ca ²⁺ current. Calcium channels show a pattern of voltage-sensitive activation and inactivation qualitatively similar to sodium channels, but with a much slower time course. The plateau is assisted by a special property of the cardiac muscle membrane known as inward-going rectification, which means that the K ⁺ conductance falls to a low level when the membrane is depolarized
Phase 3	Repolarization	Occurs as the Ca ²⁺ current inactivates and a delayed outwardly rectifying K ⁺ current activates, causing outward K ⁺ current. During the plateau, and sometimes also by other K ⁺ currents, including one through channels activated by acetylcholine and another that is activated by arachidonic acid, which is liberated under pathological conditions such as myocardial infarction
Phase 4	Pacemaker potential	Gradual depolarization during diastole. Pacemaker activity is normally found only in nodal and conducting tissue. The pacemaker potential is caused by a combination of increasing inward currents and declining outward currents during diastole. It is usually most rapid in cells of the sinoatrial node, which therefore acts as a pacemaker for the whole heart

Myocardial Oxygen Consumption and Coronary Blood Flow

Relative to its large metabolic needs, the heart is one of the most poorly perfused tissues in the body. Coronary flow is, under normal circumstances, closely related to myocardial oxygen consumption, and both change over a nearly 10-fold range between conditions of rest and maximal exercise. The main physiological factors that regulate coronary flow are as follows:²⁶

- Physical factors
- Vascular control by metabolites
- Neural and humoral control.²⁶

Physical factors

During systole, the pressure exerted by the myocardium on vessels that pass through it equals or exceeds the perfusion pressure, so coronary flow occurs only during diastole. Diastole is shortened more than systole during tachycardia, reducing the period available for myocardial perfusion. During diastole, the effective perfusion pressure is equal to the difference between the aortic and ventricular pressures. If diastolic aortic pressure falls or diastolic ventricular pressure increases, perfusion pressure falls, and so (unless other control mechanisms can compensate) does coronary blood flow. Stenosis of the aortic valve reduces aortic pressure, but increases left ventricular (LV) pressure upstream of the narrowed valve, and often cause ischemic chest pain (angina) even in the absence of coronary artery disease.²⁰

Metabolic vasomotion

Metabolic vasomotion is “the response of the coronary vasculature to changes in myocardial metabolism, which in turn are almost entirely a consequence of changes in myocardial performance”. One of the metabolites – Adenosine, can raise the flow

of blood, oxygen and nutrients in the heart by increasing the caliber of the smooth muscle cells (SMC) on vessels supplying the heart. Similarly, the activation of K^+ channels of coronary SMC have similar effect.²⁶

Neural and humoral control

Neurohumoral regulation of blood flow occurs in large coronary arteries and in the microcirculation. However, neurohumoral factors can affect the endothelial, metabolic, and myogenic regulation of coronary blood flow. The large epicardial and intramyocardial coronary arteries are densely innervated by postganglionic sympathetic and parasympathetic nerve fibers. Therefore, norepinephrine and acetylcholine released from these fibers interact with coronary adrenergic and muscarinic receptors to control coronary vascular resistance.²⁶

Cardiovascular diseases

Cardiovascular diseases (CVDs) are the main causes of human disability and mortality worldwide.²⁶ Throughout the world, the primary cause of mortality remains CVD. The international organization WHO estimates that 74 lakh deaths were related to CVD in the year 2015. It was also seen that 82% of mortalities in low economic countries was due to CVD. Below is a pictorial depiction of deaths by CVD. Going by the trend, CVD will continue to be the chief cause of deaths even in the near future.²⁷

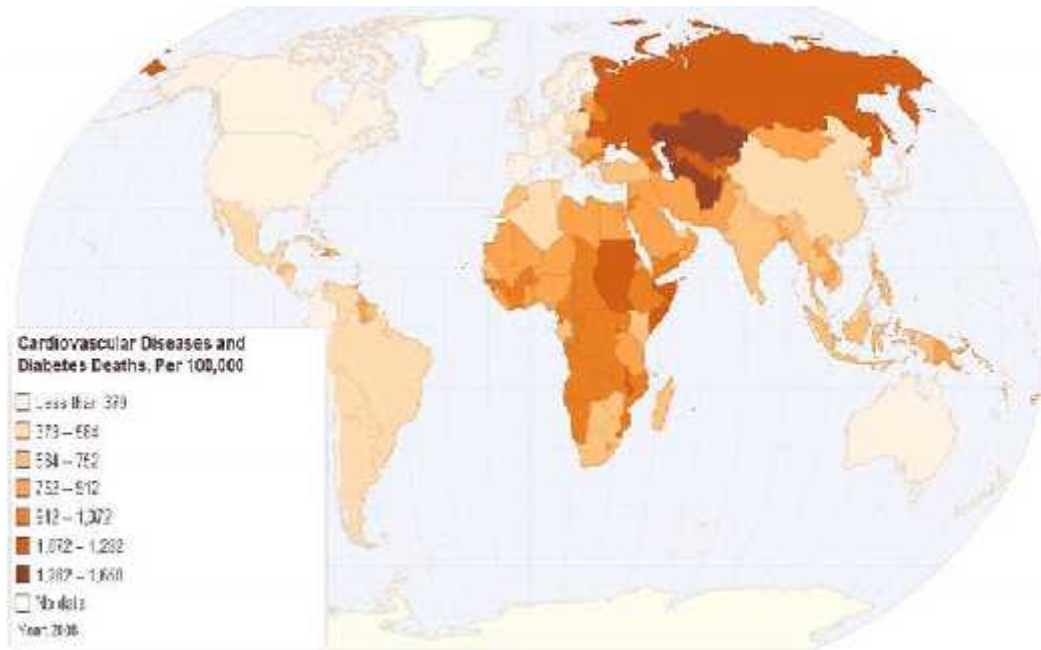


Figure 1. Mortality per lakh of population by CVD and Type 2 Diabetes. Source: World Health Organisation²⁷

Among several CVDs particularly MI has become a worldwide health problem affecting all economic groups of the society and it continues to be a major public health problem, not only in western and industrialized countries but also increasingly in developing countries, such as India and makes a significant contribution to the mortality statistics. It is the leading cause of mortality for both men and women in developed and developing countries. In developed countries, MI accounts for 10%–25% of all deaths.²⁶

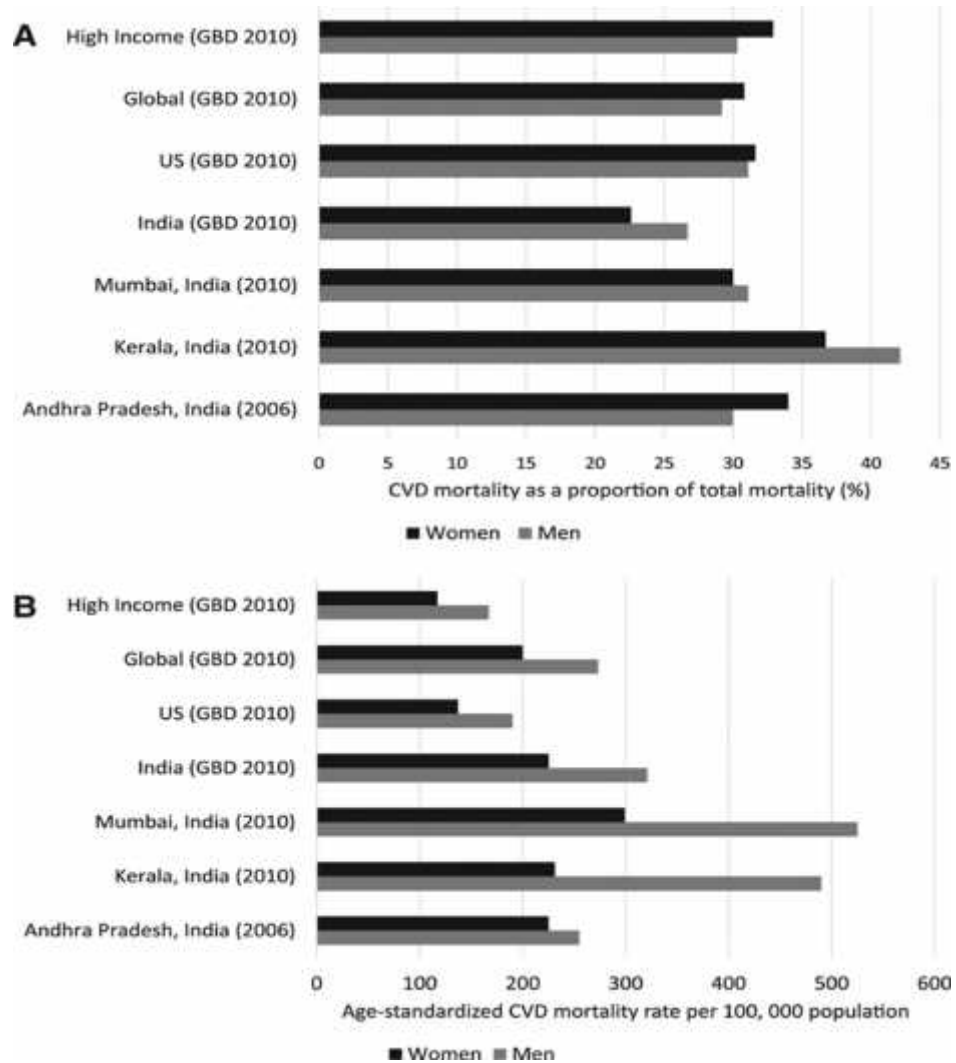


Figure 2A. CVD mortality as a proportion of total mortality. B, Age-standardized CVD mortality rate.²⁸

In our country, 24.8% of mortalities is from Cardiovascular Diseases.

Figure 2B shows that the age standardized mortality from Cardiovascular diseases is more in our country(272 / 1 lakh) than globally (235/ 1 lakh population)

The most common form of coronary heart disease (CHD) is the myocardial infarction (MI). MI occurs when a coronary artery is occluded or almost occluded, which creates a severe reduction in the blood flow, causing some of the heart muscle being supplied by that artery to become infarcted.²⁷

MYOCARDIAL INFARCTION

Myocardial infarction (MI) is commonly known as ischemic heart disease (IHD) or heart attack; it is an irreversible necrosis of tissue of a region of the myocardium caused by ischemia. Studies show that the pathophysiology mechanisms behind myocardial ischemia/reperfusion (I/R) injury are related to many factors, such as massive-free radical production, changes in hemorheology, intracellular calcium overload, increased inflammation, myocardial necrosis, and apoptosis. Therefore, in the clinical setting, myocardial I/R injury is considered a major factor that affects patient outcome.²⁶

The ischemia of the myocardium (leading to infarction) is most commonly caused by obstruction to the flow of blood in the epicardial artery. Hence the domain of the heart supplied by the corresponding vessel is affected. However, Acute Myocardial Infarction and ischemia can occur even in the absence of a blood clot. This occurs when there is a mismatch in “supply – demand” ratio. In conditions where there is reduced ‘supply’ such as hypotension or in conditions of excess ‘demand’ such as tachycardia during exertion, can also precipitate injury to the myocardium in the absence of a clot in the artery. To aid physicians in diagnosing Acute Myocardial Infarction, a globally accepted definition has been made which includes:²⁹

- Cardiac biomarkers (such as Trop T or I) of significant value
- Clinical Evidence

The evidence clinically comprises of clinical features and ECG findings (such as ST segment changes or new onset Left bundle branch block (LBBB) or new RWMA (“Regional Wall Motion Abnormality”))

The clinical spectrum of Acute Coronary Syndrome (ACS) encompasses Myocardial Infarction and Unstable Angina. Myocardial Infarction can further be differentiated based on the presence or absence of ST segment elevation into ST Elevated MI “STEMI” or non-ST elevated MI “NSTEMI”. In both the above-mentioned Myocardial Infarction, cardiac biomarkers are positive whereas in Unstable Angina it is absent.²⁷

Anatomically, Myocardial Infarction can be classified as :

- Transmural – Necrosis due to ischemic state involving the entire extent of the wall affected
- Non transmural – Involving the endocardium and sub endocardium.

Clinical Classification of Myocardial Infarction³⁰

Type	Description
Type 1	Spontaneous MI related to ischaemia due to a primary coronary event such as plaque erosion and/or rupture, fissuring or dissection
Type 2	MI secondary to ischaemia due to either increased oxygen demand or decreased supply for example, coronary artery spasm, coronary embolism, anaemia, arrhythmias, hypertension or hypotension
Type 3	Sudden unexpected cardiac death, including cardiac arrest, often with symptoms suggestive of myocardial ischaemia, accompanied by presumably new ST-elevation, or new left bundle branch block, or evidence of fresh thrombus in a coronary artery by angiography and/or at autopsy, but death occurring before blood samples could be obtained or at a time before the appearance of cardiac biomarkers in the blood
Type 4a	MI associated with percutaneous primary intervention (PCI)
Type 4b	MI associated with stent thrombosis as documented by angiography or at autopsy
Type 5	MI associated with coronary artery bypass grafting

Risk factors

The INTERHEART – South Asia study reported eight coronary risk factors – abnormal lipids, type 2 diabetes mellitus (DM), hypertension (HTN), smoking, psychosocial factors, low fruit and vegetable consumption and a lack of physical activity.³¹

Dyslipidemia

Increased amount of TC (Total Cholesterol), Low Density Lipoprotein or Triglycerides can have greater chance of causing atherosclerosis and Myocardial Infarction.

Low levels of High Density Lipoprotein is also a risk factor .³²

DM (Diabetes)

Patients with any type of diabetes are at an increased risk for myocardial infarction as it affects both micro and microvasculature and accelerates atherosclerosis in vasculature.³³

HTN

Patients with hypertension are more prone for Myocardial Infarction. Good blood pressure control decreases the chance of having an MI.³⁴

Tobacco

It causes direct injury to the vasculature and promotes formation of atherosclerotic plaque, hence elevating risk of infarction of the myocardium.³⁵

Sex

Men are more prone for MI but in post-menopausal women, with age the incidence rate difference between the two genders reduces.³⁶

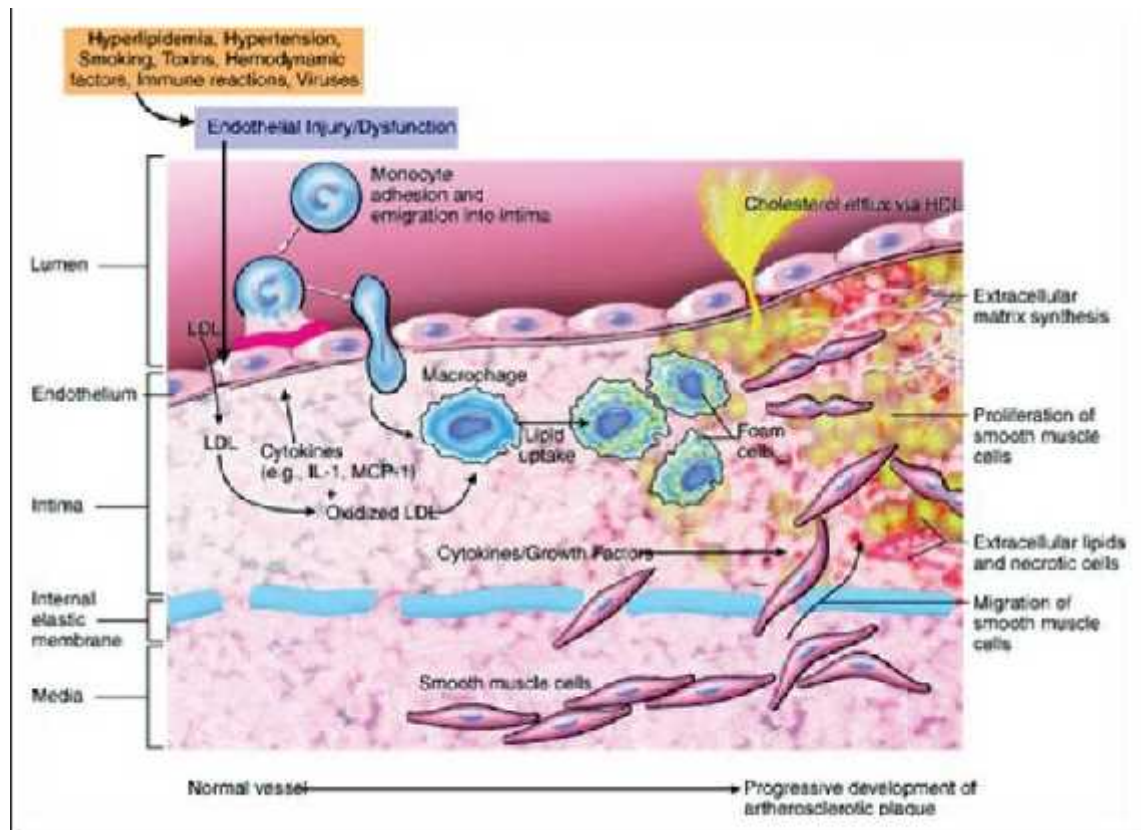
Atherosclerotic Plaque in Pathophysiology of MI:

Figure 3. Atherosclerotic plaque³⁷

The process of atherosclerosis involves the formation of a plaque in the vessels and this occurs over many years. In most cases of AMI, sudden interruption of endothelium with underlying plaque causes formation of a blood clot within the coronary vessels. Irreversible damage occurs if the obstruction lasts for over twenty minutes. The 2 key characteristics of a plaque are “Fibromuscular cap” which overlies a “Lipid Core”. The gradual wear and tear of the plaque happens because of enzyme

activity inside which consequently leads to reduction of the size of the cap. Coupled with this, the shear forces from the flow of blood in the artery can cause rupture of the overlying cap. This exposes the blood to the lipid core and a thrombus forms via activation of the coagulation cascade. If this causes significant occlusion, it results in Myocardial Infarction.³⁸

The layer of the heart most prone to ischemia is the endocardium as it is farthest from the supply from artery. With increasing time of obstruction of blood flow, the entire wall starts getting affected (starting from the endocardial layer to the myocardial and then finally the epicardial layer).³⁸

Clinical features

Sir Osler said, “Variability is the law of life, and as no two faces are the same, so no two bodies are alike, and no two individuals react alike and behave alike under the abnormal conditions which we know as disease.”³⁹

Symptoms of MI:

- Chest discomfort / pain – “Crescendo” in nature, vague, deep seated and might show radiation to arms, neck, jaw
- Breathlessness
- Severe sweating episodes
- Temporary loss of consciousness
- Unusual / Asymptomatic (in twenty five percent) seen in elderly, diabetics¹⁶

There are no characteristic findings which are exclusive to the diagnosis of AMI, but patient might have cold extremities, sweating. The heart sounds might be reduced in intensity and S4 might be heard. Blood pressure can be variable.

Hence, along with presentation we must confirm our suspicion with the help by use of electrocardiogram (ECG) and cardiac markers like cTn.³⁹

Arriving at a Diagnosis

Once a patient is suspected to have infarction of the heart based on clinical presentation, it should be further corroborated and confirmed with tests such as ECG, cardiac biomarkers and 2D ECHO (Echocardiography), to arrive at a diagnosis and differentiate between the types of ACS presentations.³⁸

Tests of Diagnosis

The initial investigation done is an ECG which helps determine if the infarction is ongoing or old and also helps to subcategorize if it STEMI or non -ST EMI. Typically in STEMI, along with ST Elevations, there are reciprocal ST depressions observed.³⁸

As recommended by the ACC in a paper published in the year 2000 and further reinforced in a subsequent year, serial ECG monitoring should be done if the initial ECG is normal^{5,21}

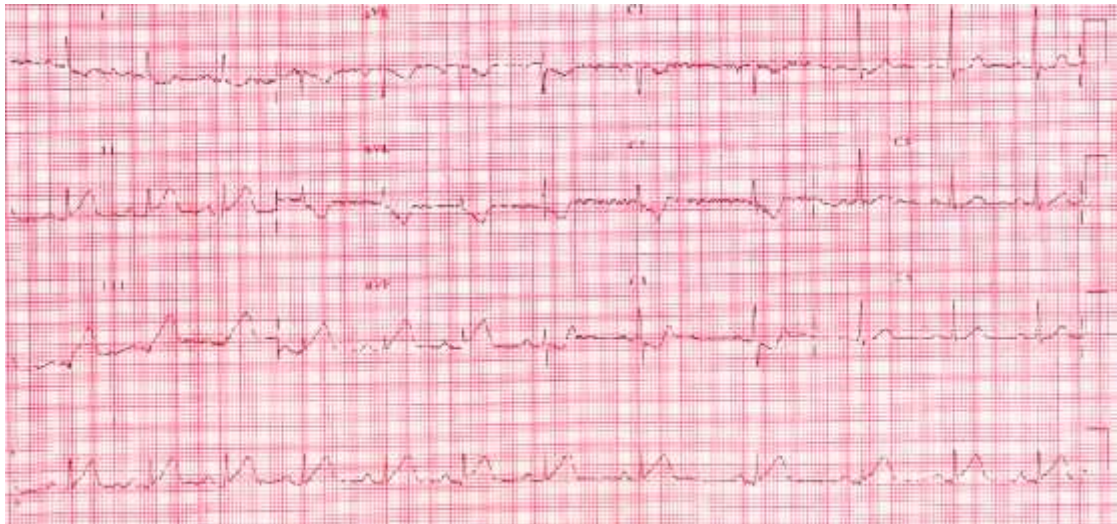


Figure4. ST-segment elevations in the inferior leads (II, III, aVF), and reciprocal ST-segment depressions in the lateral leads (I, aVL) and anterior leads (V1–V3)⁴⁰

By looking at the leads affected (in form of ST segment deviations), an ECG can help diagnose the type of MI based on the affected wall⁴¹ as shown by the diagram below.

Anatomic area	Leads
Inferior	II-III-aVF
Septal	V1-V2
Anterior	V3-V4
Low lateral	V5-V6
High lateral	I-aVL

Treatment in the form early reperfusion can be advised if ST elevations are present or if there is new onset LBBB and can be managed conservatively at first with

anticoagulation if only ST depressions are present. The ECG can also provide additional information such as the infarcted artery site, outcome expected however its significance has gone down with the rise of Primary PCI.¹⁵

It is important to understand that while coronary angiography (CAG) recognizes vessel anatomy, the Electro-cardiogram indicates the physiology of the heart.¹⁵

Though accurate, the ECG is not always definitive as there are other conditions which could affect the ST segment.⁴²

Limitations to ECG diagnosis⁵

“False-positive findings

- Benign early repolarization
- Brugada syndrome
- Cholecystitis
- Failure to recognize normal limits for J-point displacement
- Lead transposition or use of modified Mason-Likar configuration
- Left bundle branch block
- Metabolic disturbances such as hyperkalemia
- Pericarditis or myocarditis
- Pre-excitation
- Subarachnoid hemorrhage”

“False-negative findings

- Left bundle branch block
- Left ventricular hypertrophy
- Paced rhythm
- Prior myocardial infarction with Q waves, persistent ST elevation, or both”

Lab Investigations:

Cardiac biomarkers are enzymes/proteins which are primarily exclusively found in live cardiac cells. When there is irreversible injury or necrosis, they escape out of the cell and enter the blood circulation. These can then be detected with the use of various assays and are indicative of cardiac cell death. Cardiac markers are particularly of great use in differentiating NSTEMI from Unstable Angina (absent in the latter).³⁸

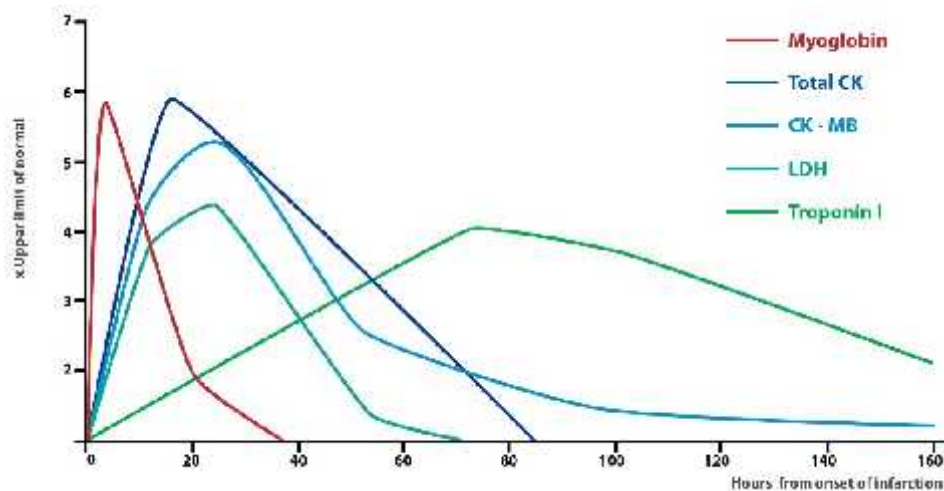


Figure 5. Cardiac Biomarkers and their trend in MI⁴³

Echocardiography

2D Echo can be used to detect regional wall motion abnormality (RWMA). RWMA occurs as a result of reduced contraction of the heart which is having reduced supply (such as during infarction) and hence the heart conserves energy. It can help detect which walls of the heart are diseased and hence which arteries might be affected. RWMA can however be present in both an acute episode of Myocardial Infarction or even Old MI and hence can't differentiate the two.³⁸

Coronary Angiogram

Selective coronary angiography is a definitive diagnostic procedure of coronary artery disease (CAD). It can be performed with low mortality (about 0.1%) and morbidity (1-5%). But cost is high.⁴⁴

ST-ELEVATION MYOCARDIAL INFARCTION

ST Elevated Myocardial Infarction is an acute coronary syndrome having clinical features of ischemia of the heart and electrocardiogram having ST segment heightened.⁴⁸

Unlike NSTEMI, in ST Elevated MI there occurs transmural ischemia which causes irreversible damage.²¹ The cardiac enzymes in the blood are raised in ST Elevated and NSTEMI.⁴⁶

Clinical Approach:

- A quick but thorough history should be obtained with information on the symptoms and associated risk factors present in the patient.
- Rapid and relevant examination of the body should be performed.
- ECG should be traced and Cardiac Troponins should be sent to the laboratory.

The ACC, American Stroke Association (AHA), ESC have defined ECG criteria for STEMI as:⁴⁷

- “New ST-segment elevation at the J point in 2 contiguous leads with the cutoff point as greater than 0.1 mV in all leads other than V2 or V3.”
- “In leads V2-V3 the cutoff point is greater than 0.2 mV in men older than 40 years old and greater than 0.25 in men younger than 40 years old, or greater than 0.15 mV in women.”

In the presence of LBBB, ‘Sgarbossa’s criteria’ is used:^{48,49}

- “ST-segment elevation of 1 mm or more that is concordant with (in the same direction as) the QRS complex.
- ST-segment depression of 1 mm or more in lead V1, V2, or V3.
- ST-segment elevation of 5 mm or more that is discordant with (in the opposite direction) the QRS complex”⁴⁶

Management of MI

Initial Management:

- Secure iv access
- Monitor vitals and cardiac electric activity
- Start oxygen only if patient has low oxygen saturation.⁴⁶

Percutaneous Coronary Intervention:

- Must be done within 1.5 hours (ninety minutes) of arriving at a center with PCI facility
- Within 2 hours if patient can be sent to a center with the above mentioned facility.⁴⁶

Thrombolysis must be initiated if patient is within window period and Percutaneous Coronary Intervention is not possible within half an hour of first contact with medical services.⁴⁶

For Analgesia – NTG and subsequently opioids (morphine) is used. All patients must be loaded with and then maintained on aspirin, statin and blocker Ticagrelor/Prasugrel is added if patient has undergone PCI. Clopidogrel is given if patient has received thrombolytic therapy.

To prevent further agents which act against coagulation cascade such as heparin (both low molecular and unfractionated) or direct thrombin antagonists must be initiated.^{46,50}

Outcome

The ‘TIMI Score’ is helpful in predicting death within 30 days and usually it is between two to ten percent.⁵¹

MI Complication:

The high mortality, complications from a structural defect following MI are:^{46,52}

- Ventricle Wall Rupture (Mortality >80%)
- IV Septal Rupture (Mortality >70%)
- Acute MR (Mortality 25%)

BIOMARKERS of the Heart:

A biomarker is defined as “a naturally occurring molecule, gene or characteristic by which a particular pathological or physiological process, disease can be identified”. So, biomarkers of the heart help in detecting damage to the cells of the heart.⁵³

Cardiac Troponin T and I (cTnT and cTnI)

- These have the highest diagnostic accuracy as
 - A. they are not routinely found in blood (as opposed to Creatine Kinase & lactate dehydrogenase [LDH]), hence increasing their specificity.⁵⁴
 - B. The rise in their level in MI is much greater than other markers (nearly twice as compared to CKMB).⁵⁵
- Kinetics after MI:

Start to appear after six hours from onset of infarction

Recognized by assays in the blood by twelve hours

Peak – 24-48 hours

Trop I and T differ in the duration to return to normal with T taking ten to fourteen days and I taking about seven to ten days.⁵⁵
- Differences between T and I Trop :
 - a. Troponin T has been standardized throughout the world due to the uniform availability of only a single assay, whereas Trop I is not standardized as there are many assays.⁵⁶
 - b. Based on site of binding in a muscle filament :T – Tropomyosin
I – Actin and Inhibin.⁵⁷
 - c. Structurally cTnT is larger and in higher concentration than cTnI in the heart.⁵⁷
 - d. cTnT is a bit less sensitive than Trop I
- The higher reference range has been set at “97.5 percentile of the values measured in the normal control population”
- According to ACC, ESC a troponin value greater than Ninety Ninth Percentile can be diagnosed of AMI.⁵⁶

CKMB:

It is not as sensitive as Troponin and can be affected by skeletal musculature and injury.⁵⁴ As it does not remain in blood as long as Troponin – it has a role to play in detecting reinfarction Best to diagnose fresh infarct (less than 6 hours).

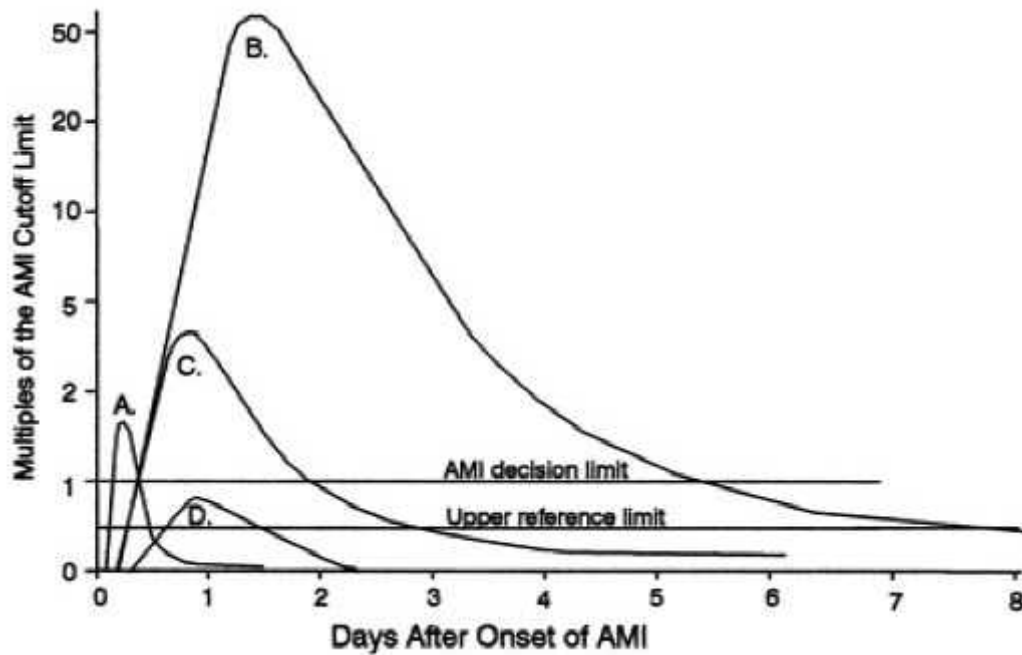


Figure 6. Time course of blood levels of cardiac markers after acute coronary syndromes. A Myoglobin or creatine kinase (CK)-MB iso-forms. B Troponin. C Total CK-MB after acute myocardial infarction (AMI). D Troponin after unstable angina pectoris (the cutoff for AMI is at 1)⁵⁷

LV FUNCTION IN PATIENTS WITH STEMI

Both the ventricles (left & right) are considered as a 'functional syncytium'. So, after an episode of heart attack, structural changes occur in both infarcted and also non infarcted parts of the ventricle and this causes dysfunction. Even if left ventricle is affected, it could progress and lead to right ventricular dysfunction by building up of back pressure. Further 'Cardiac Remodeling' occurs which is mediated by various molecule level and neurohormonal factors and resulting in irreversible damage and failure of both ventricles, which is associated with poor prognosis.^{58,59}

Even the ejection fraction (EF), as determined by echocardiography, is a well-documented strong predictor of mortality in patients with coronary disease including ST elevation (STEMI) and non-ST elevation acute myocardial infarction (NSTEMI).⁶⁰⁻⁶⁵

One of the predictors of mortality in patients with CAD is reduced Left Ventricular Ejection Fraction. Low Left ventricular ejection fraction (LVEF) is also associated with higher risk of sudden cardiac death. From a therapeutic perspective, knowing the LVEF can help in decision making for use of drugs dependent on it such as angiotensin converting enzymeinhibitors (ACEI), Angiotensin II receptor blockers (ARBs) and spironolactone. Similarly, need for ICD can be gauged from the LVEF.⁶⁶

The HORIZONS-AMI trial (2014) studied the impact of LVEF on outcomes of patients with STEMI and concluded that : among patients with STEMI undergoing primary percutaneous coronary intervention, adverse events are markedly increased in those with LVEF <40% during the index revascularization procedure. Nevertheless,

these high-risk patients experience substantial clinical benefits from bivalirudin and paclitaxel-eluting stents.⁶⁷

A study by El-Hadidy S. et al.⁵⁹ (2019) stated that “the incidence of right ventricular dysfunction in patients with acute left ventricular STEMI is higher than expected and has a negative impact on their outcome.”

Another study by Yajima K. et al.⁶⁸ (2019), observed that even diastole dysfunctionality can serve as a prognostic indicator post STEMI.

Nakamura H. et al.⁶⁹ in 2017 showed that, the cTnT levels in a stable state were associated with cardiac death in patients with LV dysfunction, even in those with non-ischemic diseases.

As seen above, the importance of LVEF is paramount and hence its assessment is recommended by international bodies such as AHA and ACC in all cases of myocardial infarction.^{70,71}

ROLE OF TROPONIN T IN IDENTIFYING LV DYSFUNCTION

Trop T is a relatively new biomarker of cardiac injury. The quantity released corresponds to the magnitude of the infarction. Hence it can be inferred that larger the infarct, more damage occurs to the myocardium and ejection fraction reduces. But the studies to substantiate this are only few.⁷²

A report by Rao A et al.⁷² in 1998 stated that there was a significant inverse relation between cTnT and LVEF and that troponin could also be used to identify ejection fraction of less than 40%. It also found that thrombolytic therapy did not alter the study conclusion.

A study conducted by Somani et al.⁷³ conducted in 50 patients presenting for the first time with ST elevated MI found that a level of 6.6 ng/ml provided a good indication for LVEF below 50% and thus can identify patients with higher risk.

In a study by Shaikh et al.⁷⁴ they concluded that Troponin I can assess the ejection fraction post treatment with STK or primary percutaneous coronary intervention following acute anterior myocardial infarction¹³.

Codolosa et al.⁷⁵ concluded that regarding cardiac biomarkers, patients whose LVEF recovered following reperfusion therapy had a lower peak troponin I, but the results were not statistically significant.

Overall, it is suggestive that though there is evidence of cardiac troponin being related to EF,⁷⁶ it is limited and more so for troponin T than troponin I.

MATERIALS AND METHODS

This one year cross-sectional study was undertaken at the Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum from January 2019 to December 2019.

Study design and duration

The study design was a one year hospital based cross-sectional study.

Study period

This study was carried out from January 2019 to December 2019.

Source of Data

Adult patients presenting with features of STEMI at Medicine Department, 'KLE Dr. Prabhakar Kore Hospital and MRC, Belagavi' were enrolled.

Sample size

A total of 97 patients presenting with STEMI during the study were enrolled.

The sample size was calculated based on the formula as mentioned below.

$$n = 4 \times p \times q / D^2$$

Where, 4 = Reliability coefficient @ 95% Confidence interval (CI)

P = Prevalence of the disease was considered as 6.47% based on the study by Rao AC et al.⁷² (1998) showing proportion of patient with left ventricular dysfunction (LVD) in patients with MI.

$$q = 100 - p = 100 - 6.47 = 93.43$$

$$d = \text{Absolute error } 5\%$$

$$\text{Therefore, } n = 4 \times 6.47 \times 93.43 / 5^2$$

$$n = 2417.96 / 25$$

$$n = 96.71 \quad 97$$

The minimum sample size required to fulfil the objective was 97. Hence the sample size of 97 was considered for the study.

Selection criteria

Inclusion Criteria

- patients with 18 years of age with ECG showing ST segment elevation of 'at least 1mm in two contiguous leads'
- First episode of MI (presenting within 2 days of onset of symptom)

Exclusion Criteria

- Heart failure patients
- Patients with renal failure
- Patients on treatment with cardiotoxic drugs.

Ethical clearance

It was obtained by the respective committee at JNMC, Belagavi

Informed consent

Patients fulfilling above said criteria were included in the study after obtaining an informed consent (Annexure-I).

Data collection

The patients diagnosed to have STEMI based on ECG were questioned on their symptoms and information regarding associated co-morbidities, habits and other demographical data was obtained. These findings were noted on a predesigned and pretested proforma (Annexure-II).

Investigations

Patients were subjected to following investigations

- Electrocardiography
- Troponin T
- 2 dimensional (2D) echocardiography

Outcome parameters

Electrocardiographic and enzymatic analysis

The included patients were subjected for ECG in order to diagnose the type of myocardial infarction, followed by systemic examination. A standard 12-lead ECG with maximal ST-segment elevation was chosen for measurements. The ECG was recorded at a paper speed of 25mm/sec at a calibration of 1 mV=10 mm. STEMI was diagnosed according to the ESC and ACC criteria^{30,77} as constrictive chest pain lasting longer than 30 min and an increased Creatinine kinase MB (CK MB) (fraction >200 U/l) and/or increased cardiac Troponin and/ or new ST elevation at the J point in two contiguous leads >0.2 mv leads v2-v3 or >0.1 mv in other leads).⁷⁷

Echocardiography

All included patients were assessed with echocardiography at the time of index hospitalization. The left ventricular end-diastolic diameter was measured from the long axis

of the left ventricle, and the left ventricular ejection fraction was calculated using biplane Simpson's method using two-dimensional echocardiography (ECHO). The echocardiographic techniques and calculations of different cardiac dimensions was performed according to the recommendations of the American Society of Echocardiography. The ejection fraction was obtained from apical two chamber and four chamber view. Measurements were made from three consecutive beats, and the average of three beats was used for analysis. Ejection fraction of 40% was regarded as presence of LVD.

Troponin T

Under aseptic precautions, 2 cc of blood was drawn into a plain bulb and sent for Troponin T estimation (within 12 to 48 hours of onset of symptom). This was measured using Elecsys® Troponin T-high sensitive assay from Roche.⁷⁸

Statistical methods

Microsoft Excel Worksheet was used to log the collected information (Annexure III). The data was analysed using statistical software SPSS version 20.0. Continuous variables were presented as mean±standard deviation (SD) and interquartile range (IQR) analyzed for normality by the Shapiro-Wilk test. Categorical variables were compared using the Chi-square or Fisher's exact test while continuous variables with normal distribution were compared using Independent t-test and those did not follow normal distribution were tested using Mann-Whitney U tests. Receiver operating characteristic (ROC) curve with Area under the curve (AUC) analysis was conducted to determine optimal cut-off value for cardiac troponin T in predicting EF of 40%. The accuracy of troponin T in discriminating EF of 40% was determined in terms of sensitivity, specificity, positive predictive value (PPV) negative predictive value (NPV). The correlation between troponin T and ejection fraction was determined by Spearman's correlation coefficient (). All tests were two-tailed and a p-value of less than 0.05 was considered significant.

RESULTS

This one year hospital based cross-sectional study was conducted in the Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum from January 2019 to December 2019. A total of 97 patients presenting with STEMI during the study were studied.

The data was analyzed and the final results were tabulated and interpreted as below.

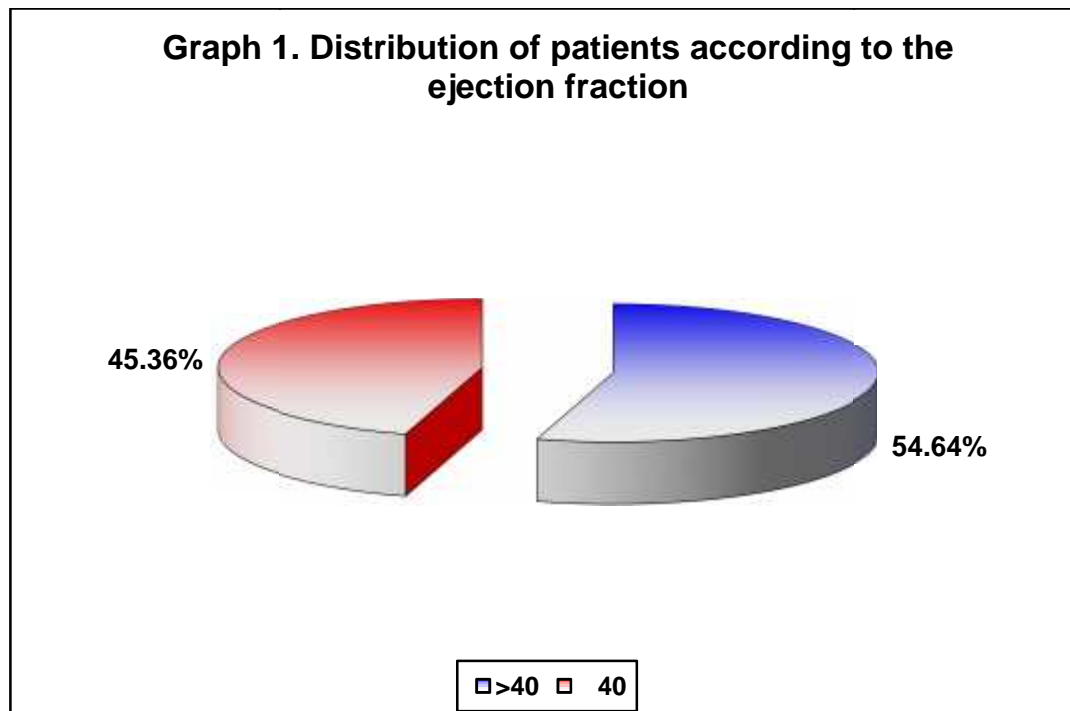
Table 1. Clinical characteristics of the study population

Parameters	Mean (n=72)		Median	IQR	Range		p value
	Mean	SD			Min	Max	
	Age (Years)	61.47			12.38	63.00	
Body mass index (kg/m ²)	25.15	3.42	25.00	5.20	18.80	32.90	0.070
Ejection fraction (%)	42.68	5.95	45.00	5.00	30.00	55.00	<0.001
Troponin T	2.41	3.22	1.31	2.16	0.05	17.11	<0.001

The clinical characteristics are as shown in table 1.

Table 2. Distribution of patients according to the ejection fraction

Ejection fraction (%)	Distribution (n=97)	
	Number	Percentage
>40	53	54.64
40	44	45.36
Total	97	100.00

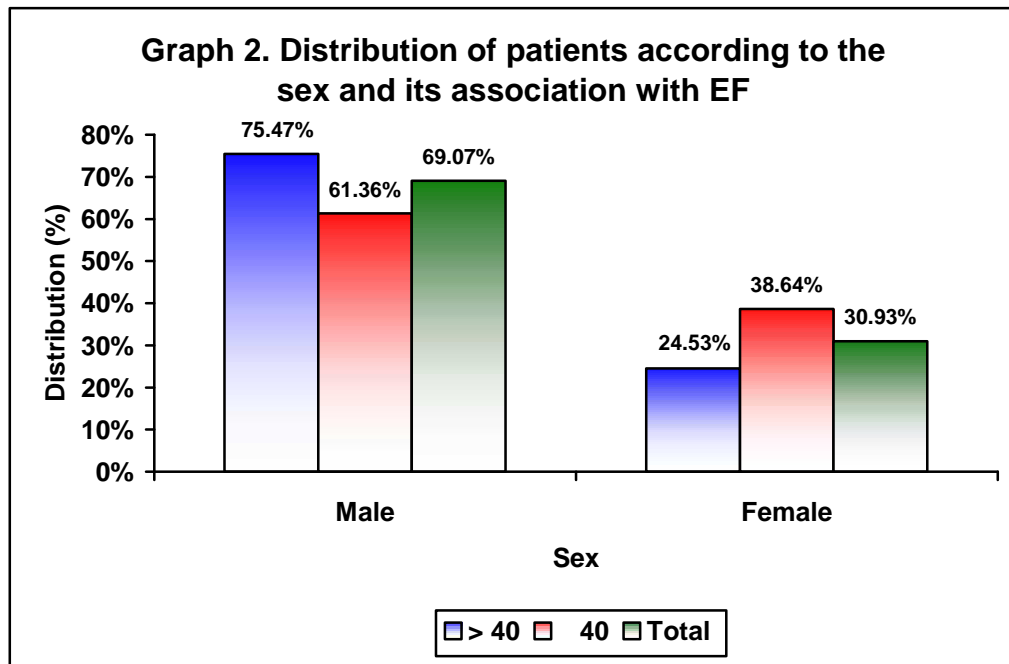


In the present study ejection fraction of 40% as noted in 45.36% of the patients.

Table 3. Distribution of patients according to the sex and its association with EF

Sex	Ejection fraction (%)				Total	
	>40		40		No.	%
	No.	%	No.	%		
Male	40	75.47	27	61.36	67	69.07
Female	13	24.53	17	38.64	30	30.93
Total	53	100.00	44	100.00	97	100.00

p=0.186

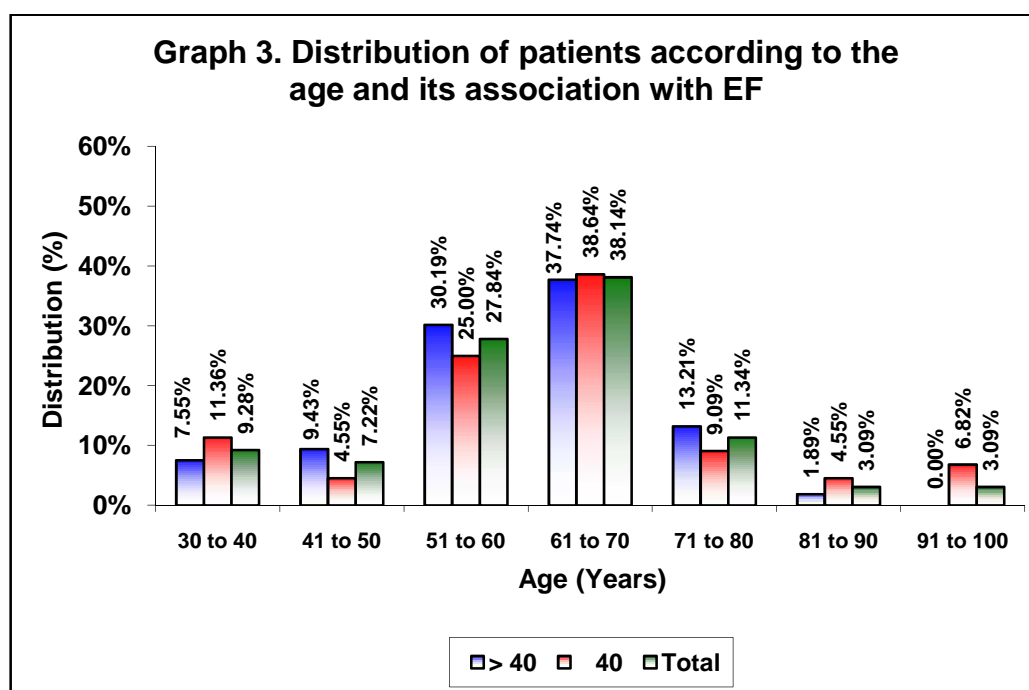


In the present study 69.07% of the patients were males and 30.93% were females with male to female ratio of 2.23:1. No association was found between sex and EF (p=0.186).

Table 4. Distribution of patients according to the age and its association with EF

Age (years)	Ejection fraction (%)				Total	
	>40		40			
	No.	%	No.	%	No.	%
30 to 40	4	7.55	5	11.36	9	9.28
41 to 50	5	9.43	2	4.55	7	7.22
51 to 60	16	30.19	11	25.00	27	27.84
61 to 70	20	37.74	17	38.64	37	38.14
71 to 80	7	13.21	4	9.09	11	11.34
81 to 90	1	1.89	2	4.55	3	3.09
91 to 100	0	0.00	3	6.82	3	3.09
Total	53	100.00	44	100.00	97	100.00

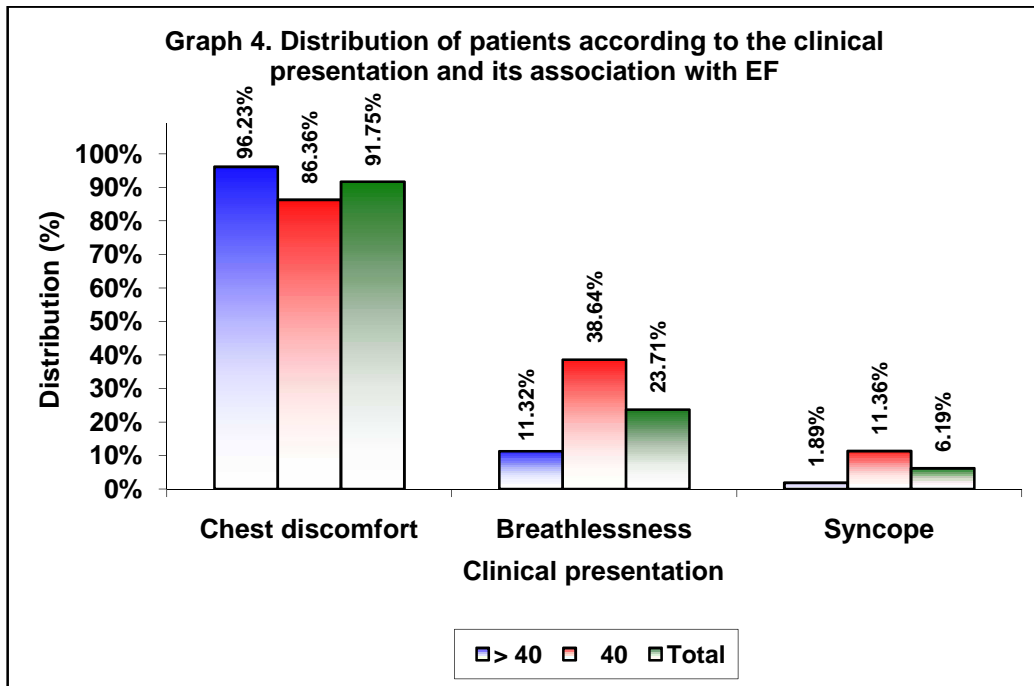
p=0.486



In this study 38.14% of the patients were aged from 61 to 70 years. No association was found between age and EF (p=0.486).

Table 5. Distribution of patients according to the clinical presentation and its association with EF

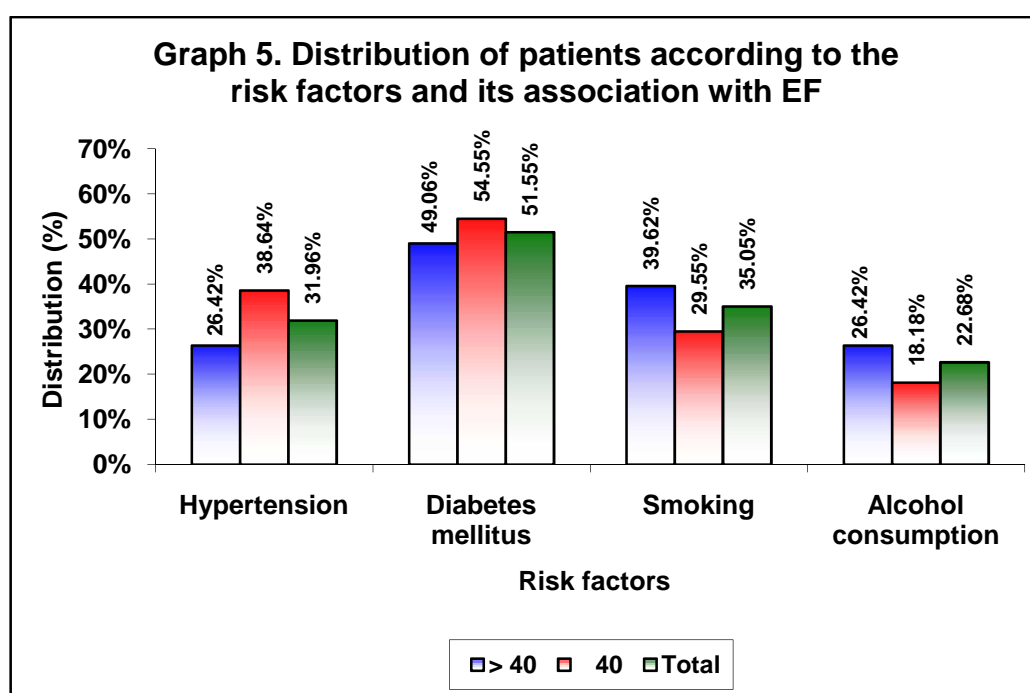
Clinical presentation	Ejection fraction (%)				Total		p value
	>40		40		No.	%	
	No.	%	No.	%			
Chest discomfort	51	96.23	38	86.36	89	91.75	0.083
Breathlessness	6	11.32	17	38.64	23	23.71	0.002
Syncope	1	1.89	5	11.36	6	6.19	0.065



In the present study 91.75% of the patients presented with chest discomfort, 23.71% has breathlessness and 6.19% of the patients had syncope. However, significant association was noted between breathlessness and EF (p=0.002).

Table 6. Distribution of patients according to the risk factors and its association with EF

Risk factors	Ejection fraction (%)				Total		p value
	>40		40		No.	%	
	No.	%	No.	%			
Hypertension	14	26.42	17	38.64	31	31.96	0.274
Diabetes mellitus	26	49.06	24	54.55	50	51.55	0.684
Smoking	21	39.62	13	29.55	34	35.05	0.393
Alcohol consumption	14	26.42	8	18.18	22	22.68	0.466

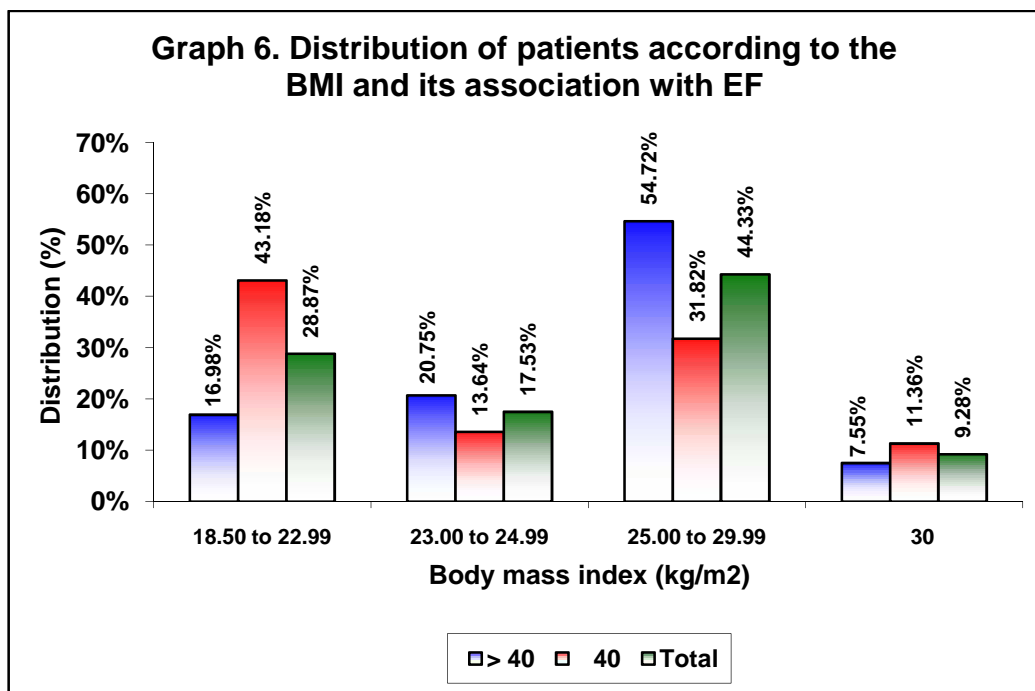


In this study 51.55% of the patients reported history of diabetes mellitus, 35.05% reported smoking, 31.96% reported hypertension and 22.68% reported history of alcohol consumption. However, no association was found between these risk factors with EF ($p > 0.050$).

Table 7. Distribution of patients according to the BMI and its association with EF

Body mass index (Kg/m ²)	Ejection fraction (%)				Total	
	>40		40		No.	%
	No.	%	No.	%		
18.50 to 22.99	9	16.98	19	43.18	28	28.87
23.00 to 24.99	11	20.75	6	13.64	17	17.53
25.00 to 29.99	29	54.72	14	31.82	43	44.33
30	4	7.55	5	11.36	9	9.28
Total	53	100.00	44	100.00	97	100.00

p=0.022

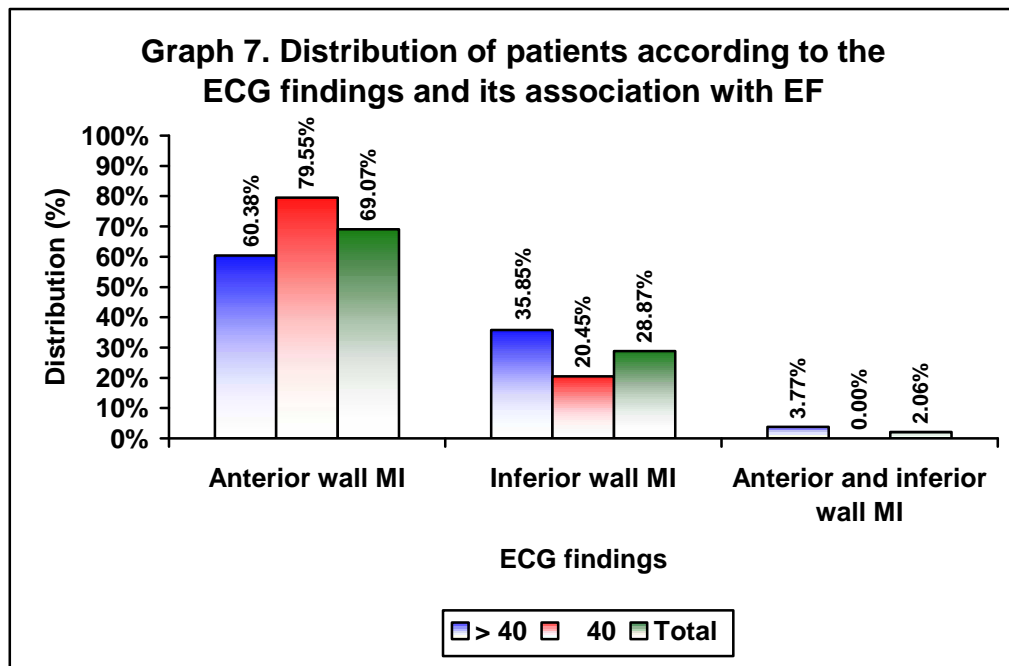


In the present study 44.33% of the patients had BMI from 25.00 to 29.99 Kg/m² and 9.28% had BMI of 30 Kg/m². Significant association was noted between BMI and EF (p=0.022).

Table 8. Distribution of patients according to the ECG findings and its association with EF

ECG findings	Ejection fraction (%)				Total	
	>40		40		No.	%
	No.	%	No.	%		
Anterior wall MI	32	60.38	35	79.55	67	69.07
Inferior wall MI	19	35.85	9	20.45	28	28.87
Anterior and inferior wall MI	2	3.77	0	0.00	2	2.06
Total	53	100.00	44	100.00	97	100.00

p=0.079

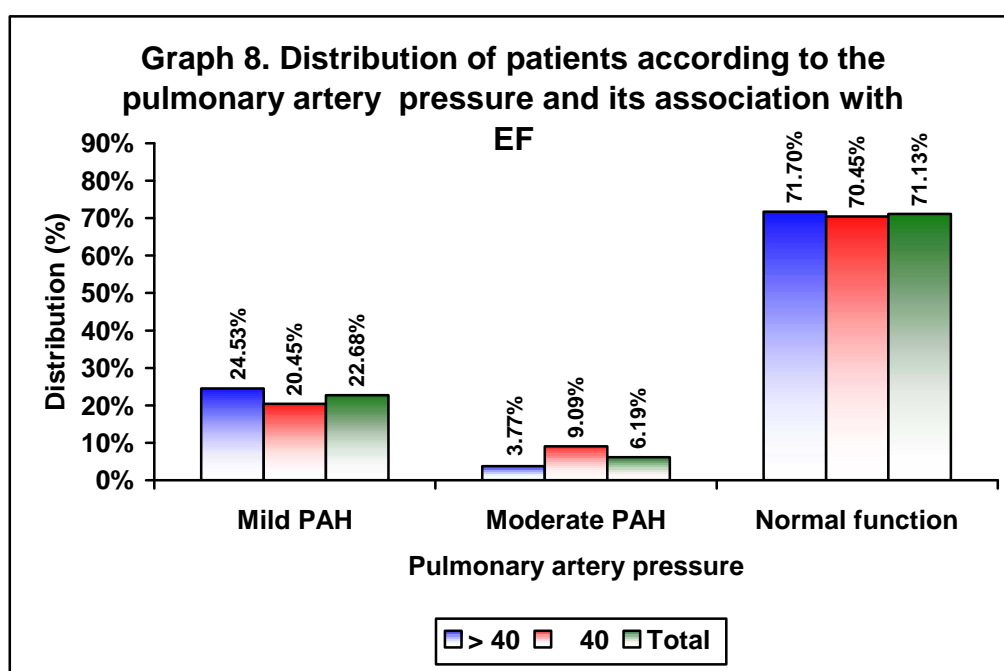


In this study ECG findings revealed Anterior wall / Extensive anterior myocardial infarction (AWMI) in 69.07% of the patients, IWMI in 28.87% and both in 2.06% of the patients. However no association was found between ECG findings and EF (p=0.079).

Table 9. Distribution of patients according to the pulmonary artery pressure and its association with EF

Pulmonary artery pressure	Ejection fraction (%)				Total	
	>40		40		No.	%
	No.	%	No.	%		
Mild pulmonary artery hypertension (PAH)	13	24.53	9	20.45	22	22.68
Moderate PAH	2	3.77	4	9.09	6	6.19
Normal function	38	71.70	31	70.45	69	71.13
Total	53	100.00	44	100.00	97	100.00

p=0.532

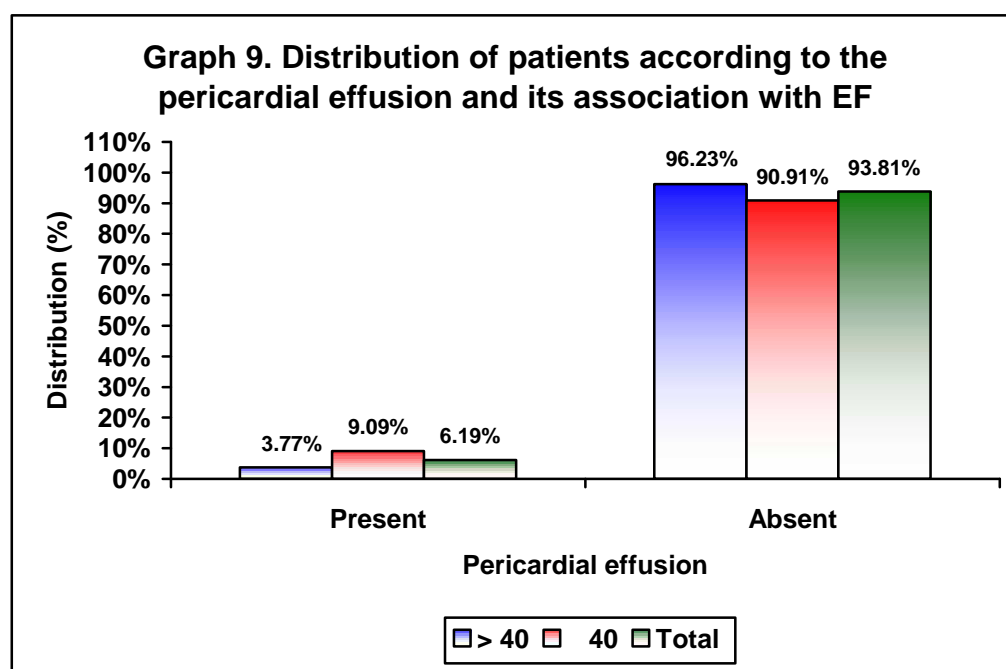


In the present study on 2D echocardiography, normal function was noted in 71.13% of the patients, mild PAH in 22.68% while moderate PAH was diagnosed in 6.19% of the patients. Further no association was found between pulmonary artery pressure and EF (p=0.532).

Table 10. Distribution of patients according to the pericardial effusion and its association with EF

Pericardial effusion	Ejection fraction (%)				Total	
	>40		40		No.	%
	No.	%	No.	%		
Present	2	3.77	4	9.09	6	6.19
Absent	51	96.23	40	90.91	91	93.81
Total	53	100.00	44	100.00	97	100.00

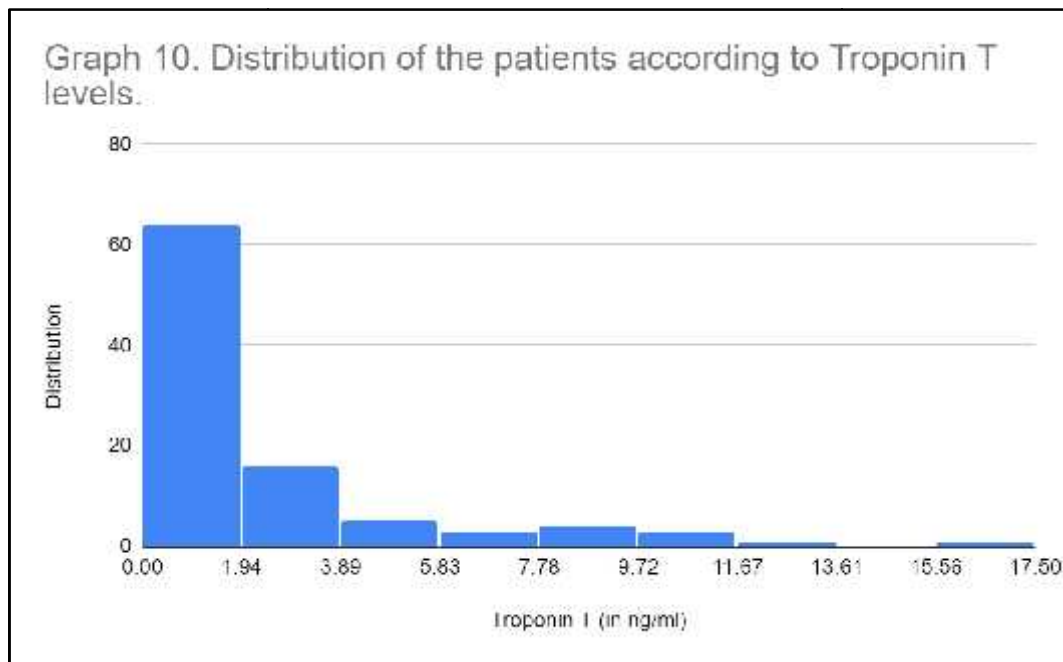
p=0.255



In this study diagnosis of PE was noted in 6.19% of the patients and no significant association was found between PE and EF (p=0.255).

Table 11. Distribution of patients according to the troponin T levels

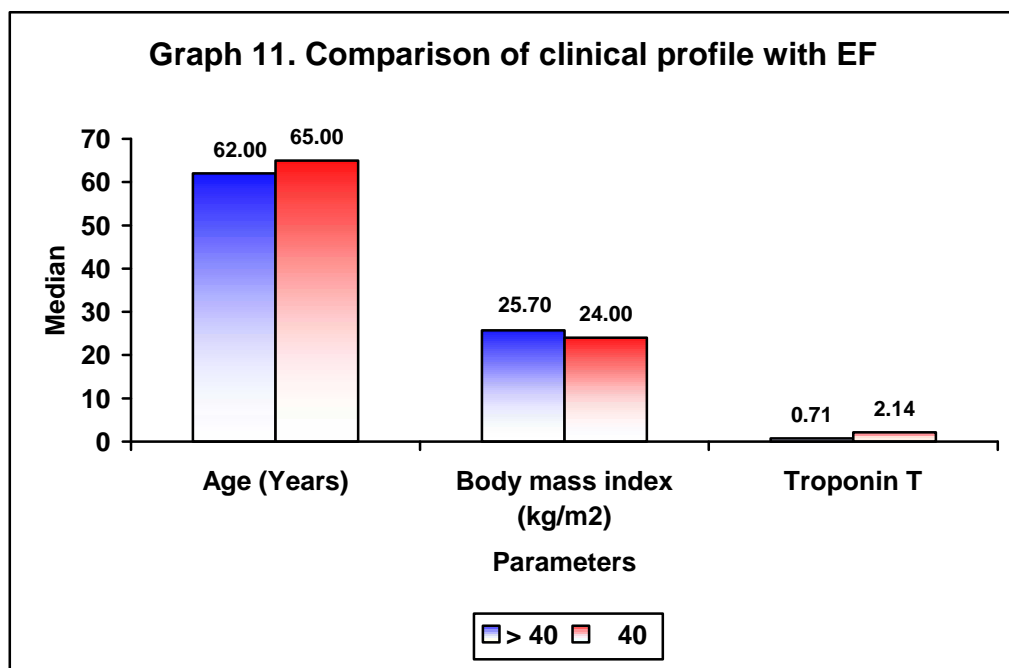
Troponin T levels	Distribution	
	Number	Percentage
0.010	0	0.00
>0.010	97	100.00
Total	97	100.00



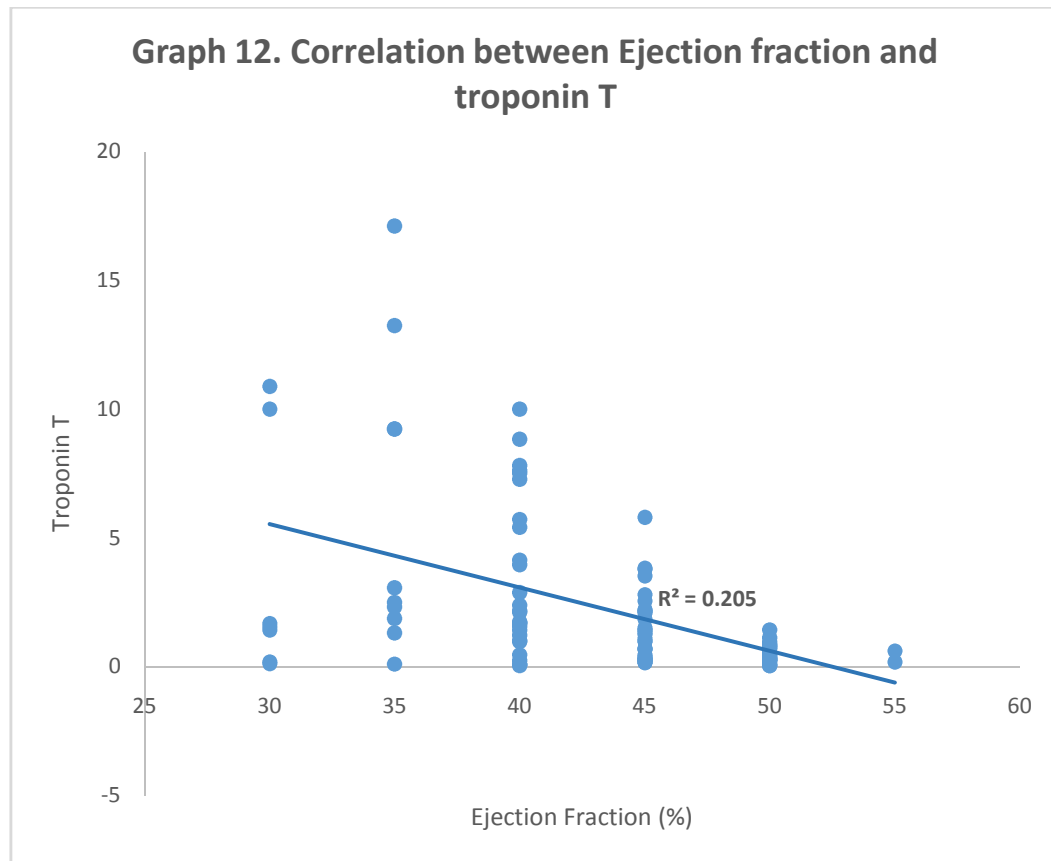
In the present study troponin T levels of > 0.010 (greater than reference range) were noted in all the patients (100%). The serum troponin levels ranged from 0.05 to 17.11 ng/ml.

Table 12. Comparison of clinical profile with EF

Parameters	Ejection fraction (%)				p value
	>40		40		
	Median	IQR	Median	IQR	
Age (Years)	62.00	12.00	65.00	15.50	0.226
Body mass index (Kg/m ²)	25.70	4.15	24.00	6.30	0.693
Troponin T	0.71	1.15	2.14	6.22	0.328

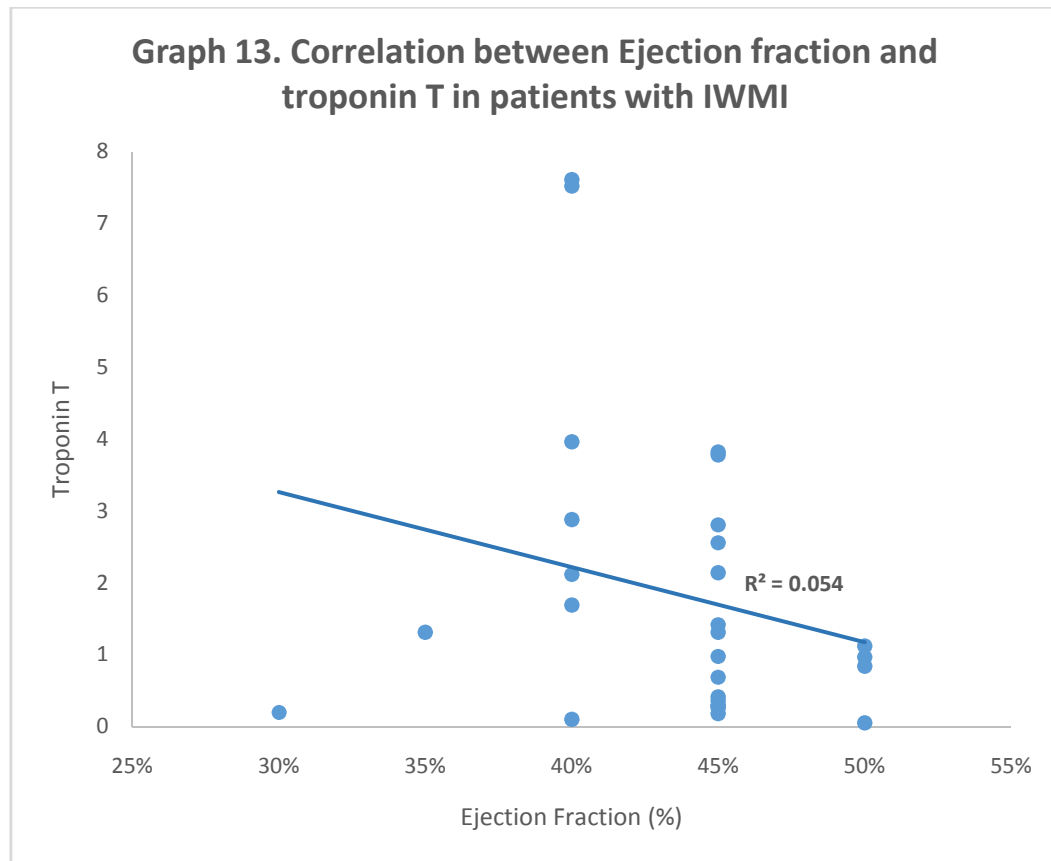


In this study the median age (62.00 [IQR 12.00] vs 65.00 [IQR 15.50] years; $p=0.226$), body mass index (25.70 [IQR 4.15] vs 24.00 [IQR 6.30] Kg/m²; $p=0.693$) and Troponin T (0.741 [IQR 1.15] vs 2.14 [IQR 6.22] years; $p=0.328$) in patients with normal and low EF were comparable.



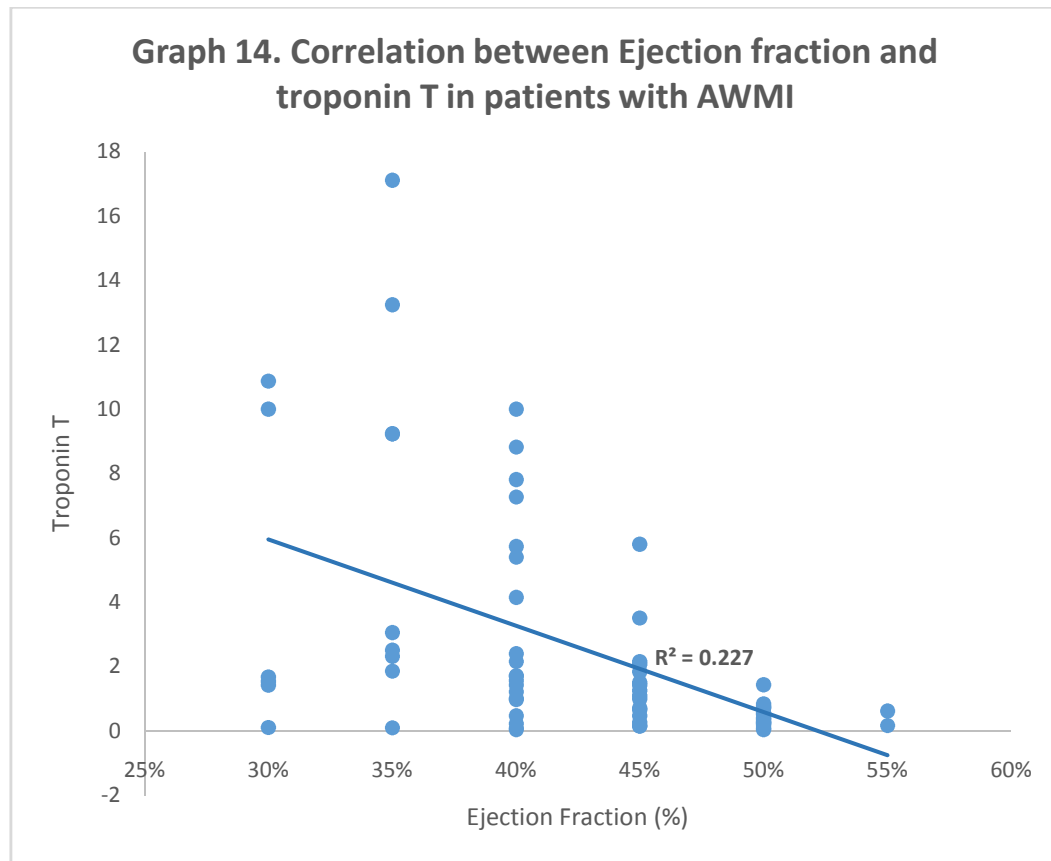
=-0.474; R2=0.2056; p<0.001

The correlation between troponin T and EF is as shown in graph 12. There was moderate significant negative correlation between troponin T and EF (=-0.474; R2=0.2056; p<0.001).



=-0.297; R2=0.0546; p=0.125

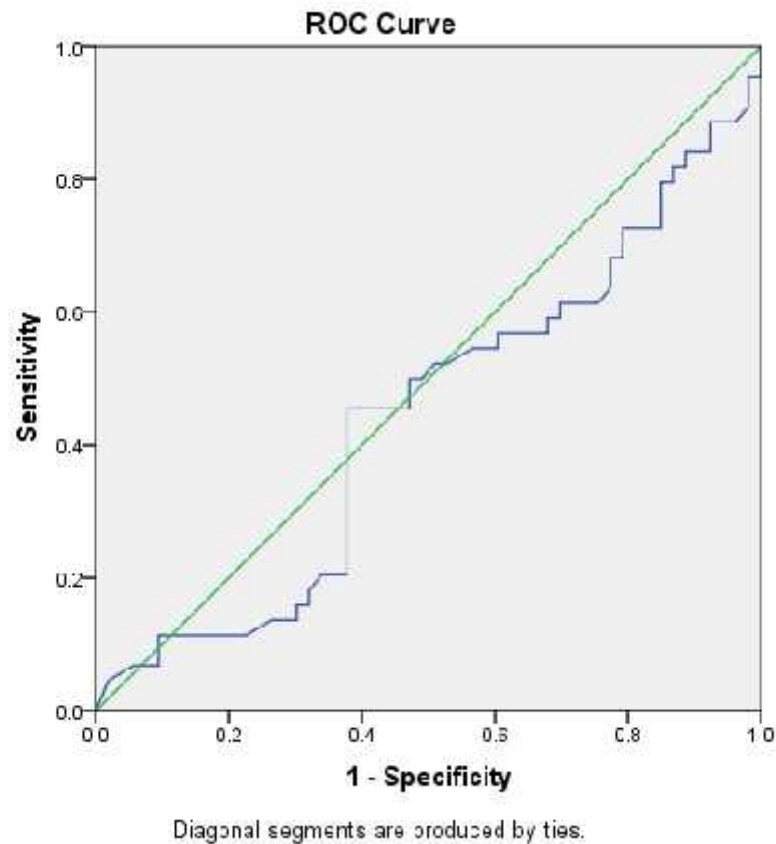
The correlation between troponin T and EF in patients with IWMI is as shown in graph 13. There was weak negative correlation between troponin T and EF but the correlation was not significant (-0.474 ; $R^2=0.0546$; $p=0.125$).



=-0.534; R2=0.2272; p<0.001

The correlation between troponin T and EF in patients with AWMI is as shown in graph 14. There was moderate negative significant correlation between troponin T and EF (=-0.534; R2=0.2272; p<0.001).

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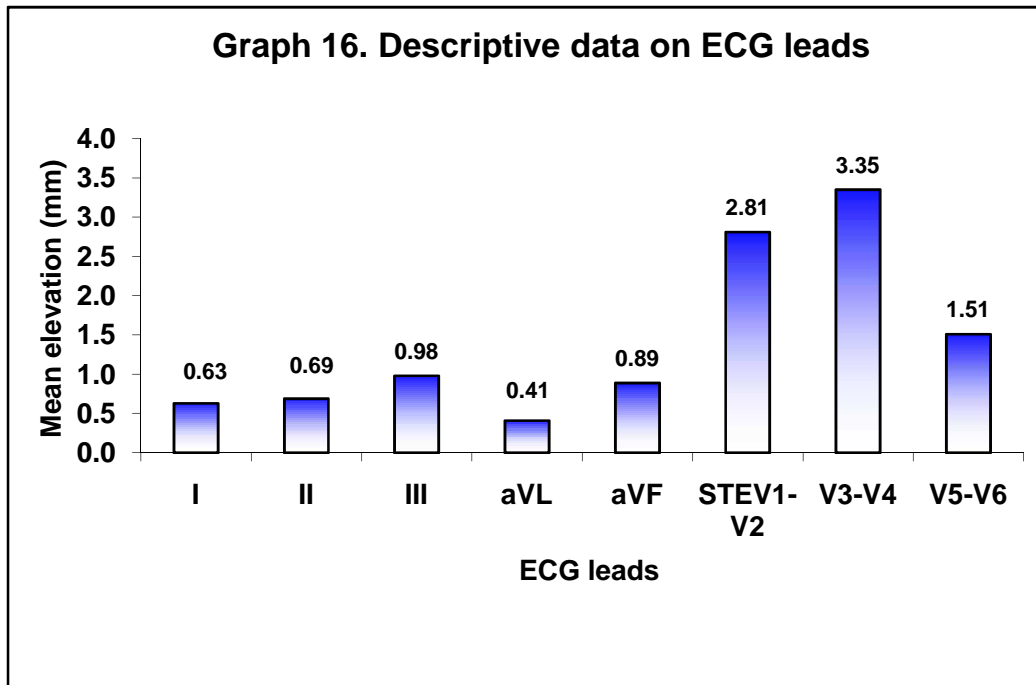
Graph 15. Receiver operating curve for EF of 40%

AUC=0.442; SE=0.048; p=0.328; (95% CI 0.326 to 0.558)

The ROC in the prediction of EF 40% using troponin T is as shown in graph 13. The cut-off value of troponin T in the diagnosis of EF 40% based ROC was 1.285 with AUC of 0.442 and not significant (p=0.328) with sensitivity and specificity of 50% and 50.94% respectively.

Table 13. Descriptive data on ECG leads

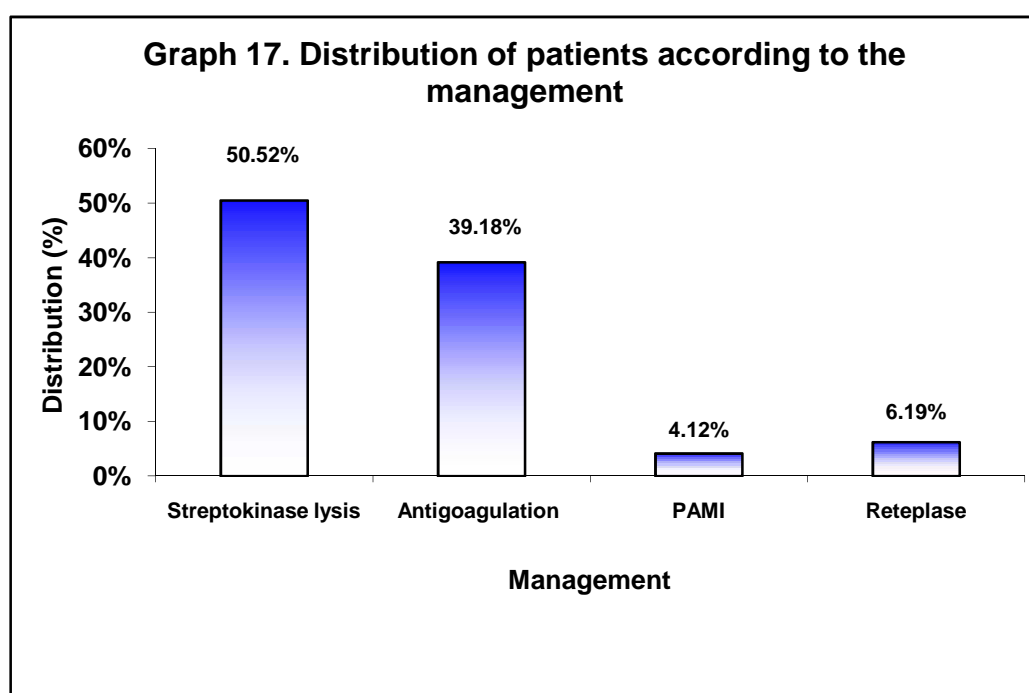
ECG lead	Mean (n=72)		Median	IQR	Range		p value
	Mean	SD			Min	Max	
I	0.63	1.08	1.00	0.00	0.00	5.00	<0.001
II	0.69	1.59	1.00	0.00	0.00	13.00	<0.001
III	0.98	1.97	2.00	0.00	0.00	14.00	<0.001
aVL	0.41	0.86	1.00	0.00	0.00	4.00	<0.001
avF	0.89	1.84	2.00	0.00	0.00	14.00	<0.001
STE V1-V2	2.81	2.81	4.00	3.00	0.00	10.00	<0.001
V3-V4	3.35	3.25	5.00	2.00	0.00	13.00	<0.001
V5-V6	1.51	1.99	2.00	0.00	0.00	10.00	<0.001



The descriptive data on elevation of ECG leads is as shown in table 13 and graph 14.

Table 14. Distribution of patients according to the management

Management	Distribution (n=97)	
	Number	Percentage
Streptokinase lysis	49	50.52
Anticoagulation	38	39.18
PAMI	4	4.12
Retepase	6	6.19
Total	97	100.00



In the present study 50.52% of the patients were treated with streptokinase lysis, 39.18% with anticoagulation, 6.19% with reteplase and 4.12% with PAMI.

DISCUSSION

Measurement of LV systolic function has been widely accepted as a primary method of risk stratifying patients after STEMI.⁷⁵ With the help of imaging modalities such as 2D Echo or nuclear scans, the LVEF can be estimated.

Old conventional markers of myocardial injury, such as CKMB and Myoglobin were known to roughly calculate the size of infarction and hence outcome. But these markers need to be recurrently recorded for the same and get affected by management instituted (such as thrombolysis) as it would change the kinetics. Hence their usefulness was limited especially in cases of reperfusion.

Among the relatively newer cardiac biomarkers, Troponins (cTn) have shown promise, owing to their greater diagnostic value in AMI. Similar to CKMB, it can correlate to the size of infarction, but unlike the former, it isn't affected by reperfusion therapy.⁷²

Although generally understudied, the void is however greater in cTnT than cTnI. Considering these facts, the present study was designed to assess relationship between Troponin T and LV dysfunction so as to identify its role as a non-imaging modality in identifying LV dysfunction.

The present one year hospital based cross-sectional study was carried out in the Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum from January 2019 to December 2019. A total of 97 patients presenting with STEMI during the study were evaluated for troponin T and LV dysfunction.

LVEF is well known risk factor for prognosis in patients with IHD, according to the data provided by Brezinov OP et al.⁶⁵ (2017) who demonstrated that the predictors of survival are different according the degree of LV dysfunction at admission. Another study by Yajima K et al.⁶⁸ (2019) also showed that, “diastolic dysfunction is an independent and powerful predictor for long-term outcome after STEMI”. Measurement of LVEF is needed from a therapeutic perspective to initiate lifelong ACEI in patients with ejection fraction <40%.⁷² Hence in the present study LV dysfunction was assessed by 2D echocardiography considering EF of 40%. Accordingly, the EF of the population studies ranged from 30% to 55%. The mean and median EF was $42.68 \pm 5.95\%$ and 45% (IQR 5%) respectively. Further EF of 40% as noted in 45.36% of the patients. These observations suggest that, every second patient presenting with STEMI is likely to have LVD as measured by EF of 40%. In our study 45.36% of patients with STEMI had LV dysfunction. This value was higher compared to Bodi V et al.⁷⁹ (2003) (33.3%) but comparable with the study of Somani D et al.⁷⁴ (2005) (48%). However very higher rate of LVD is reported by Khilar SM et al.⁸⁰ (2018) where as high as 82.25% of patients with STEMI had LV dysfunction.

In the present study on 2D echocardiography, the other important findings noted were that the majority of the patients had normal function (71.13%) followed by mild PAH (22.68%) and moderate PAH (6.19%). However, no association was found between pulmonary artery pressure and EF ($p=0.532$). Similarly, Dew patients were diagnosed to have PE (6.19%) but again no significant association was found between PE and EF ($p=0.255$).

In the present study serum troponin T levels ranged from 0.05 to 17.11 $\mu\text{g/L}$. The mean and median troponin T levels were 2.41 ± 3.22 and 1.31 (IQR 2.16)

$\mu\text{g/L}$. Further, troponin T levels of > 0.010 was noted in all the patients (100%). Also, the median Troponin T (0.741 [IQR 1.15] vs 2.14 [IQR 6.22] years; $p=0.328$) levels in patients with EF greater than or less than or equal to 40% were comparable. Moderate significant negative correlation between troponin T and EF ($=-0.474$; $R^2=0.2056$; $p<0.001$) was noted in the present study. This was also true in patients with AWMI as moderate negative significant correlation between troponin T and EF ($=-0.534$; $R^2=0.2272$; $p<0.001$). However in patients with IWMI, although, there was weak negative correlation between troponin T and EF, but, the correlation was not significant ($=-0.474$; $R^2=0.0546$; $p=0.125$). Taken together, these observations suggest a significant direct inverse relationship between troponin T and EF in patients presenting with STEMI. However these observations require further validation due to lack of similar data in the literature as the relationship between cTnT and LV dysfunction after IWMI specifically in patients with STEMI has not been examined previously. It is however known that RV dysfunction is more noted with IWMI but this is not in the scope of our study. However the findings from the present study corroborate with the observations reported by Rao ACR et al.⁷² (1998) who reported strong negative correlation between serum troponin T concentration measured 12 to 48 hours post MI and angiographic LVEF ($p<0.0001$). However one important difference between the present study and the study by Rao ACR et al.⁷² (1998) needs to be emphasized is that the strong correlation reported in the latter study and the moderate correlation in the present although both were negative and statistically significant. The exact reason for this difference cannot be elucidated but one important reason may be the method of estimation of LVEF which was 2D echocardiography in the present study while the latter study ascertained LVEF by ventriculography which is gold standard.

The findings of the present study were partly in agreement with another recent study by Khan MH et al.⁸¹ (2019) who stated that, cTnI has application in recognizing patients with LV Ejection Fraction \leq 40%, in whom treatment must be more vigorous. Waxman DA et al.⁸² (2006) also showed that cTnI measured at 12 and 48 hours had inverse relationship ($p < 0.001$) to LVEF assessed both early and at 3 months after STEMI. However these studies by Khan MH et al.⁸¹ (2019) and Waxman DA et al.⁸² (2006) considered troponin I but not troponin T.

In the present study, the ROC in the prediction of EF \leq 40% using troponin T yielded cut-off value for the troponin T 1.285 $\mu\text{g/L}$ in the diagnosis of EF \leq 40% with AUC of 0.442 and was not significant ($p=0.328$) The sensitivity and specificity was 50% and 50.94% respectively suggesting that, troponin T is not sensitive in predicting LVEF of \leq 40%. On the contrary, Rao ACR et al.⁷² (1998) showed that “troponin-T concentration of $> 2.8 \mu\text{g/l}$ predicted a LVEF \leq 40% in patients with STEMI who had received thrombolytic therapy with a sensitivity of 95.5% and specificity of 88.1%”. Again the disparity observed between the present study and the study by Rao ACR et al.⁷² (1998) might be by the method of estimation of LVEF in the present study which was 2D echocardiography in the present study while the latter study ascertained LVEF by ventriculography.

A large community-based cohort very recently by Lyngbakken MN et al.⁸³ (2020) showed that, extensive echocardiography, concentrations of cTnI and cTnT are associated with subclinical LV hypertrophy and dysfunction. Concentrations of cTnI appear superior to cTnT in predicting subclinical LV hypertrophy. The study also commented that, the associations with Left ventricular mass index (LVMI) were stronger for cTnI.

It is reported that, the risk for atherosclerosis and myocardial infarction is greater in males than females. However this gap reduces as the age increases in both group.^{38,84} the results of this study were strongly in agreement with the previous reports as in our study, most of the patients were males (69.07%) with men to women ratio of 2.23:1 suggesting male preponderance but no association was found between sex and EF (p=0.186). the male preponderance noted in the present study was also consistent with the previous reports by Rao ACR et al.⁷² (1998) and Khan MH et al.⁸¹ (2019). In this study age ranged between 30 to 93 years. The mean and median age was 61.47±12.38 years and 63 (IQR 13.00) years. Most of the patients (38.14%) were aged from 61 to 70 years. However, no association was found between age and EF (p=0.486). Also the median age (62.00 [IQR 12.00] vs 65.00 [IQR 15.50] years; p=0.226) was comparable in patients with EF 40% and >40%. The mean of the age in this study was consistent with the previous study by Rao ACR et al.⁷² (1998) who reported mean age as 62 years.

The clinical spectrum of ACS and MI encompasses a wide range of presentations from being asymptomatic to chest pain, sweating, syncope and dyspnea.³⁹In the present study majority of the patients (91.75%) presented with chest discomfort followed by breathlessness (23.71%) and syncope (6.19%). However, significant association was noted between breathlessness and EF (p=0.002).

In this study 51.55% of the patients reported history of diabetes mellitus, 35.05% reported smoking, 31.96% reported hypertension and 22.68% reported history of alcohol consumption. However, no association was found between these risk factors with EF (p>0.050). these observations were comparable with the observations reported in the INTERHEART study by Yusuf S. et al.³¹ (2004) which described nine

risk factors which account for >95% of acute MI. Eight risk factors including dyslipidemia, smoking, hypertension, DM, abdominal obesity, psychosocial factors, lack of consumption of fruits, vegetables and of regular physical activity were significantly associated with MI ($p < 0.0001$), whereas alcohol consumption had a weaker association ($p = 0.03$).

Another important finding of the present study needs to be discussed is significant association of BMI with LVEF 40%. In the present study BMI ranged between 18.80 to 32.90 Kg/m². The mean and median BMI was 25.15±3.42 and 25 (IQR 5.20) Kg/m². Most of the patients (44.33%) had BMI from 25.00 to 29.99 Kg/m² and 9.28% had BMI of 30 Kg/m² suggesting overweight and obesity. Further 31.82% of the patients with overweight and 11.36% of the patients with obesity had EF of 40% and this observation was statistically significant ($p=0.022$). However, body mass index (25.70 [IQR 4.15] vs 24.00 [IQR 6.30] Kg/m²; $p=0.693$) in patients with normal and low EF were comparable. These observations pose high risk of LVD in patients with STEMI with overweight and obesity. This observation was partly in agreement with the observations reported in Dorbala S. et al.⁸⁵ (2006) who found that, mild obesity was associated with LV dilatation, but the LVEF was preserved even with severe obesity.⁸⁵ Similar observations were documented by Seo JS et al.⁸⁶ (2017) recently who demonstrated that, obesity and overweight independently predict diastolic dysfunction and an optimal body weight lower than the universal cut-off is reasonable for preventing LV heart failure in Asians. Although the studies by Dorbala S. et al.⁸⁴ (2006) and Seo JS et al.⁸⁶ (2017) were not aimed to find the relation between troponin T and LVD in patients with STEMI the findings of the present study in principle are in agreement with the observations reported by Dorbala S. et al.⁸⁵ (2006) and Seo JS et al.⁸⁶ (2017).

In the present study most of the patients (50.52%) were treated with streptokinase lysis, followed by anticoagulation (39.18%) and reteplase (6.19%) and PAMI (4.12%).

Overall the present study highlights the relationship between Troponin T and Left ventricular end diastolic (LVED) so as to test the feasibility of Troponin T as non-imaging modality in identifying LV dysfunction and demonstrated moderate negative correlation between Troponin T and LVEF as assessed by 2D echocardiography with EF in both AWTMI and IWTMI subset of STEMI patients. Further a cut of value of 1.28 for troponin T had limited sensitivity and specificity in predicting LVD among the patients with STEMI. This study also confirm the strong association of LVD with overweight and obesity in patients with STEMI. Although, the sensitivity of Troponin T is limited, it is a simple, quick, inexpensive, noninvasive method which provides hint to the treating physician towards the likelihood of trend of LVEF. However, these findings require further validation due to potential limitations so this study.

Strengths

The strength of the study was that the present study extended the relationship between Troponin T and LVED for STEMI patients with IWTMI and AWTMI.

Limitations

The limitation of the study was that, the findings in this study were based on the data having relatively smaller sample size from a single center. Also owing to the smaller subset of patients, subgroup analysis like age and gender specific analysis

was not done and finally outcome was not considered as it was beyond the scope of this study.

Recommendations

Multicentric studies involving large sample size with assessment of EF based on coronary angiogram with gender and age specific EF values extending the relationship to short term and long-term outcomes may provide the exact relationship between Troponin T and LVEF in patients with STEMI.

CONCLUSION

From the results of this study, we can conclude that, there is moderate negative correlation between Troponin T and LVEF as assessed by 2D echocardiography with EF in both AWTMI and IWTMI subset of STEMI patients. Further a cut of value of 1.28 μ g/L for troponin T has limited sensitivity and specificity in predicting LVEF 40% among the patients with STEMI.

SUMMARY

Measurement of LVEF is important from both a prognostic and therapeutic perspective. The present study was aimed to assess relationship between Troponin T and LV dysfunction so as to identify its role as a non-imaging modality in identifying LV dysfunction.

The present one-year hospital based cross-sectional study was carried out under the Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum from January 2019 to December 2019. A total of 97 patients presenting with STEMI during the study were evaluated for troponin T and LVEF by 2D echocardiography. The important highlights of the study are summarized as below.

- The EF of the population studies ranged from 30% to 55%. The mean and median EF was $42.68 \pm 5.95\%$ and 45% (IQR 5%) respectively. Further EF of 40% as noted in 45.36% of the patients.
- Serum troponin T levels ranged from 0.05 to 17.11 $\mu\text{g/L}$. The mean and median troponin T levels were 2.41 ± 3.22 and 1.31 (IQR 2.16) $\mu\text{g/L}$. The median Troponin T (0.741 [IQR 1.15] vs 2.14 [IQR 6.22] years; $p=0.328$) levels in patients EF greater or less than 40% were comparable.
- Moderate significant negative correlation between troponin T and EF ($r=-0.474$; $R^2=0.2056$; $p<0.001$) was noted in the present study in all MI as shown in graph 12.

- There was weak negative correlation between troponin T and EF but the correlation was not significant in IWMI ($r = -0.474$; $R^2 = 0.0546$; $p = 0.125$).
- There was moderate negative significant correlation between troponin T and EF in AWMI ($r = -0.534$; $R^2 = 0.2272$; $p < 0.001$).
- Most of the patients (44.33%) of the patients had BMI from 25.00 to 29.99 Kg/m^2 and 9.28% had BMI of $\geq 30 \text{ Kg/m}^2$. Significant association was noted between BMI and EF ($p = 0.022$). But, body mass index in patients with normal and low EF were comparable (25.70 [IQR 4.15] vs 24.00 [IQR 6.30] Kg/m^2 ; $p = 0.693$).
- Troponin T levels of > 0.010 were noted in all the patients (greater than the upper reference normal).
- The median troponin T levels in patients with EF greater or lesser than 40% were comparable (0.741 [IQR 1.15] vs 2.14 [IQR 6.22] years; $p = 0.328$).
- The cut-off value of troponin T in the diagnosis of EF $\geq 40\%$ based ROC was 1.285 with AUC of 0.442 and not significant ($p = 0.328$) with sensitivity and specificity of 50% and 50.94% respectively.
- Majority of the patients were males (69.07%) and 30.93% were females with male to female ratio of 2.23:1. No association was found between sex and EF ($p = 0.186$).
- Most of the patients (38.14%) of the patients were aged from 61 to 70 years. No association was found between age and EF ($p = 0.486$). The median age in patients with normal and low EF were comparable (62.00 [IQR 12.00] vs 65.00 [IQR 15.50] years; $p = 0.226$).

- Majority of the patients (91.75%) of the patients presented with chest discomfort, 23.71% has breathlessness and 6.19% of the patients had syncope. Significant association was noted between breathlessness and EF ($p=0.002$).
- Most of the patients (51.55%) reported history of diabetes mellitus, 35.05% reported smoking, 31.96% reported hypertension and 22.68% reported history of alcohol consumption. No association was found between these risk factors with EF ($p>0.050$).
- ECG findings revealed AWMI or extensive anterior in 69.07% of the patients, IWMI in 28.87% and both in 2.06% of the patients. No association was found between ECG findings and EF ($p=0.079$).
- On 2D echocardiography, normal function was noted in 71.13% of the patients, mild PAH in 22.68% while moderate PAH was diagnosed in 6.19% of the patients. No association was found between pulmonary artery pressure and EF ($p=0.532$).
- Diagnosis of PE was noted in 6.19% of the patients. No significant association was found between PE and EF ($p=0.255$).
- Most of the patients (50.52%) were treated with streptokinase lysis, 39.18% with anticoagulation, 6.19% with reteplase and 4.12% with PAMI.

Overall, there is moderate negative correlation between Troponin T and LVEF as assessed by 2D echocardiography with EF in both AWMI and IWMI subset of STEMI patients. Further a cut of value of 1.28 for troponin T has limited sensitivity and specificity in predicting LVD among the patients with STEMI.

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ANNEXURE I

ETHICAL CLEARANCE CERTIFICATE

	K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH (Deemed - to - be - University)	
	Accredited 'A' Grade by NAAC (2 nd Cycle)	Placed in Category 'A' by MHRD (GoI)
JAWAHARLAL NEHRU MEDICAL COLLEGE, NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)		
Website: http://www.jnmc.edu E-Mail : dome@jnmc.edu	Phone: (+ 91-(0)831 Office : 2472550 Principal: 2471701 Fax No. +91 (0)831 -2470759	
Ref: MDC/DOME/ 29		Date: 24/11/2018
To,		
REG. NO. BG0118009 PG student in Medicine, J.N. Medical College, BELAGAVI		
Sub: Institutional Ethical Clearance for the study.		
<p>With reference to the above, we wish to inform you that your proposed research project titled "ROLE OF TROPONIN IN IDENTIFYING LEFT VENTRICULAR EJECTION FRACTION OF <math>\leq 40\%</math> IN ACUTE ST ELEVATED MYOCARDIAL INFARCTION: A ONE YEAR CROSS SECTIONAL STUDY IN KLE DR PRABHAKAR KORE HOSPITAL AND MRC BELAGAVI", is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.</p>		
 (Dr. Arathi Darshan) Member Secretary JNMC Institutional Ethics Committee on Human Subjects Research, J.N. Medical College, Belagavi.	 (Dr. Roopa M Bellad) Chairman, JNMC Institutional Ethics Committee on Human Subjects Research, J.N. Medical College, Belagavi.	

ANNEXURE II

CONSENT FORM

Title:“ROLE OF TROPONIN IN IDENTIFYING LEFT VENTRICULAR EJECTION FRACTION OF $\leq 40\%$ IN ACUTE ST ELEVATED MYOCARDIAL INFARCTION: A ONE YEAR CROSS SECTIONAL STUDY IN KLE DR PRABHAKAR KORE HOSPITAL AND MRC BELAGAVI”

Study investigator: REG. NO.BG0118009

P.G. M.D. GENERAL MEDICINE.

J.N. Medical College,

Belagavi-590010.

Introduction

You are being invited to participate in this study on Role of Troponin in identifying ejection fraction of the heart in Myocardial Infarction.

Explanation of procedure

An ECG will be taken at the time of presentation to the hospital.If patient fulfils inclusion criteria, 2cc blood sample will be drawn and sent to detect level of Troponin T. This might be repeated once more at a later time as well to study the trend. 2D Echo will be done to determine ejection fraction of the heart.

Possible benefits

The participant receives further evaluation of his condition which will help in the management.

Confidentiality

All information collected during the course of study will be kept confidential.

Withdrawal

Participation in this study is voluntary. If you don't wish to participate in this study; you will not lose benefits to which you are entitled. After starting the study, anytime during the study, if you feel to withdraw from the study, you are free to do so.

Cost of participation

The cost of the study will be borne by the researcher. There will be no additional cost to you for participation in the study.

Authorization to publish the results

The results of the study would be forwarded to the KLE University, Belgaum as part of requirement towards the completion of MD degree, review and publishing.

In case of the queries during study or in future you may contact following persons

CONSENT STATEMENT

I voluntarily agree to take part in this study by signing below. I may withdraw at any time. I am not giving up any of my legal rights by signing this form. My signature below indicates that I have read, or it has been read to me, this entire consent form, and have had all my questions answered.

Name of the Participant: _____ Signature / Thumb print

Name of the Witness _____ Signature/ Thumb print

Investigator Name: _____ Signature: _____

Date:

Place

ತಳುವಳಕಯಸಮ್ಮತಿರೂಪ

ಶೀರ್ಷಿಕೆ: “ಟ್ರೋಪೋನಿನ್‌ನೋಲಿ ಎಡವ್ಯಾಟ್ರಿಕ್ರೀಕರ್ಷಣಜಕ್ಷನ್ಯುರುತನ್ನುಗುರುತಿಸುವುದರಲ್ಲಿನಾನು <= 40%

ನಷ್ಟುಅಕ್ಕೂಟೆಎಸಿಎನ್ನಲಾದಮಯೋಕಾರ್ಡಿಯಲಿಒನ್ನಾರ್ಕ್ಸ್‌ನ್ - KLE

ಪ್ರಭಾಕರ್ಕೋರಆಸ್ಪತ್ರೆಯಲ್ಲಿಒಂದುವರ್ಷದಅಡ್ಡವಿಭಾಗೀಯಅಧ್ಯಯನ”

ಅಧ್ಯಯನಸಂಶೋಧಕ:

REG. NO.BG0118009.

ಸ್ನಾತಕೋತ್ತರಪದವಿ - ಆಂತರಿಕಜಞ್ಞವ್ಯದ್ಯರು

ಜಿವಾಹರಲಾಲ್‌ಜಿವ್ಯದ್ಯಕೇಯಕಾಲೇಜು

ಬಲಾಗವಿ - 590010

ಪರಿಚಯ

ಮಯೋಕಾರ್ಡಿಯಲಿಒನ್ನಾರ್ಕ್ಸ್‌ನ್‌ನಲ್ಲಿಹೃದಯದವಜಕ್ಷನ್ಯುಗವನ್ನುಗುರುತಿಸುವಲ್ಲಿಟ್ರೋಪೋನಿನ್ I

ಪಾತ್ರದಬಗ್ಗೆಈಅಧ್ಯಯನದಲ್ಲಿಭಾಗವಹಿಸಲುನಿಮ್ಮನ್ನುಆಹ್ವಾನಿಸಲಾಗುತ್ತಿದೆ

ಪ್ರಕ್ರಿಯೆಯವಿವರಣೆ

ಪ್ರಸ್ತುತಿಸಮಯದಲ್ಲಿಆಸ್ಪತ್ರೆಗಳಇಸಿಜಿತಗದುಕೂಳ್ಳಲಾಗುವುದು.ರೋಗಿಯಸೇರ್ಪಡಮಾನದಂಡ

ವನ್ನುಪೂರೈಸಿದರೆ, 2cc ರಕ್ತದಮಾದರಿಯನ್ನುಎಳೆಯಲಾಗುತ್ತದೆಮತ್ತುಟ್ರೋಪೋನಿನ್ I

ನಮಿಟ್ಟವನ್ನುಪತ್ತಹಚ್ಚಲುಕಳುಹಿಸಲಾಗುತ್ತದೆ.ಇದುವಂತರದಸಮಯದಲ್ಲಿಮತ್ತೊಮ್ಮೆಪ್ರವೃತ್ತಿಯನ್ನುಅಧ್ಯ

ಯನಮಾಡಲುಕೂಡಾಪುನರಾವರ್ತಿಸಬಹುದು.ಹೈದೆಯದವಜಕ್ಷನ್ಯುಗವನ್ನುನಿರ್ಧರಿಸಲು 2D

ಎಕೋಸ್ಕ್ಯಾನಾಡಲಾಗುತ್ತದೆ.

ಸಂಭವನೀಯಲಾಭಗಳು

ಪಾಲ್ಗುಳ್ಳವರುತನ್ನಸ್ಥಿತಿಯನ್ನುಮತ್ತಷ್ಟುಮೌಲ್ಯಮಾಪನವನ್ನುಪಡೆಯುತ್ತಾರೆಯೆಂದುನಿರ್ವಹಣೆಗ

ಸಹಾಯಮಾಡುತ್ತದೆ

ಗೌಪ್ಯತೆ

ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ಸಂಗ್ರಹಿಸಿದ ಎಲ್ಲಾ ಮಾಹಿತಿಯನ್ನು ಗೌಪ್ಯವಾಗಿ ರಸಲಾಗುತ್ತದೆ.

ವಿರೋಧ

ಈ ಅಧ್ಯಯನದ ಭಾಗವಹಿಸುವಿಕೆ ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿರುತ್ತದೆ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ನೀವು ಬಯಸದಿದ್ದರೆ; ನಿಮಗೆ ಅರ್ಹವಾದ ಲಾಭಗಳನ್ನು ನೀವು ಕಳೆದುಕೊಳ್ಳುವುದಿಲ್ಲ. ಅಧ್ಯಯನವನ್ನು ಪ್ರಾರಂಭಿಸಿದ ನಂತರ, ಯಾವುದೇ ಅಧ್ಯಯನದಲ್ಲಿ, ನೀವು ಅಧ್ಯಯನದಿಂದ ಹೊರಬರಲು ನೀವು ಬಯಸಿದರೆ, ನೀವು ಹಾಗೆ ಮಾಡಲು ಮುಕ್ತರಾಗುತ್ತೀರಿ.

ಪಾಲ್ಗೊಳ್ಳುವಿಕೆಯ ವಚನ

ಅಧ್ಯಯನದ ವಚನವು ಸಂಶೋಧಕರಿಂದ ಹುಟ್ಟಿಕೊಳ್ಳುತ್ತದೆ. ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ನಿಮಗ ಹೆಚ್ಚುವರಿ ವಚನವಿಲ್ಲ.

ಫಲಿತಾಂಶಗಳನ್ನು ಪ್ರಕಟಿಸಲು ಅನುಮೋದನೆ

ಅಧ್ಯಯನದ ಫಲಿತಾಂಶಗಳನ್ನು ಎಷ್ಟೆ ಪಡೆದು, ವಿಮರ್ಶೆ ಮತ್ತು ಪ್ರಕಾಶನ ಮುಗಿಸಲು ಅಗತ್ಯವಿರುವ ಭಾಗವಾಗಿ ಕೆಲವು ಇವಶ್ಯವಿದ್ದಾಯಿ, ಬಳಗಾವಿ ಗರ್ವಾ ಡ್ಯಾಡಲಾಗುವುದು.

ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ಅಥವಾ ಭವಿಷ್ಯದಲ್ಲಿ ನೀವು ಈ ಕೆಳಗಿನ ವ್ಯಕ್ತಿಗಳನ್ನು ಸಂಪರ್ಕಿಸಬಹುದು :

REG. NO. BG0118009

ಡಾ. _____

ಸ್ನಾತಕೋತ್ತರ ಪದವಿ -

ಉಪಪ್ರಾಂಶುಪಾಲರು

ಆಂತರಿಕ ಜ್ಞಾನ ವ್ಯವಸ್ಥಾಪಕರು

ಪ್ರೊಫೆಸರ್

ಆಂತರಿಕ ಜ್ಞಾನ ವ್ಯವಸ್ಥಾಪಕರು

ಜಿವಾಹರಲಾಲ್ ಹೈವೈದ್ಯಕೀಯ ಕಾಲೇಜು

ಬೆಂಗಳೂರು - 590010

ರಸಚ್ಛೇದನಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ಒಪ್ಪಿಗ

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ನಾನು ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿ ಒಪ್ಪುತ್ತೇನೆ. ನಾನು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಹಿಂಪಡೆಯಬಹುದು. ಈ ಫಾರ್ಮ್ ಅನ್ನು ಸಹಿ ಮಾಡುವ ಮೂಲಕ ನನ್ನ ಯಾವುದೇ ಕಾನೂನು ಹಕ್ಕುಗಳನ್ನು ನಾನು ಬಿಟ್ಟುಕೊಡುವುದಿಲ್ಲ.

ಕಳೆದ ನನ್ನ ಸಹಿ ನಾನು ಈ ಸಂಪೂರ್ಣ ಒಪ್ಪಿಗಯನ್ನು ಓದಿದ್ದೇನೆ ಅಥವಾ ನನಗೊದುತ್ತಿದ್ದೇನೆ ಎಂದು ಸೂಚಿಸಿದ

, ಮತ್ತು ನನ್ನ ದೇಶೀಯ ಭಾಷೆಯಲ್ಲಿ ನನಗೆ ಎರಡು ಸಲಾಗದ ಮತ್ತು ನನ್ನ ಎಲ್ಲ ಪ್ರಶ್ನೆಗಳಿಗೆ ಉತ್ತರಿಸಿದ.

ಈ ಸಮ್ಮತಿಯು ರೂಪದ ಪ್ರತಿಯನ್ನು ನನಗನೇ ಡಲಾಗುವುದು.

ಸಹಿ / ಪಾಲ್ಗೊಳ್ಳುವವರ ಅಥವಾ ಕಾನೂನುಬದ್ಧವಾಗಿ ಅಧಿಕೃತ ಪ್ರತಿನಿಧಿಗಳ ಎಡತಮ್ಮ ದ್ರಣ.

ಪಾಲ್ಗೊಳ್ಳುವವರ ಹೆಸರು:

ಸಹಿ / ಎಡತಮ್ಮರುತು:

ಕಾನೂನುಬದ್ಧವಾಗಿ ಅಧಿಕೃತ ಪ್ರತಿನಿಧಿ ಹೆಸರು:

.....

ಸಹಿ / ಎಡತಮ್ಮರುತು:

ವಿಚ್ಛೇದನ ಹೆಸರು:

ಸಹಿ / ಎಡತಮ್ಮರುತು:

ತನಿಖಾಧಿಕಾರಿ ಹೆಸರು ಮತ್ತು ಸಹಿ:

.....

ದಿನಾಂಕ ಮತ್ತು ಸ್ಥಳ:

सूचितसहमति

शीर्षक: “ट्रोपोनिनकीभूमिकामें<= 40%

बाएवेंट्रिकुलरएजेक्शनफ्रेक्शनकीपहचानमेंएसटीमेंनिर्मितमायोकार्डियलइंफार्क्शन :
एकसालकाक्रॉससेक्शनस्टडी, केएलईप्रभाकरकोरेअस्पतालबेलगावीमें”

अध्ययनजांचकर्ता:

REG. NO.BG0118009

स्नातकोत्तरछ - एमडीजनरलमेडिसिन

जे.एन.मेडिकलमहाविद्यालय,

के.एल.ईविश्वविद्यालय, बेलगावी-590,010

परिचय

म्योकार्डियलइंफार्क्शनमेंदिलकेनिकासअंशकीपहचानकरनेमेंट्रोपोनिन I
कीभूमिकापरइसअध्ययनमेंभागलेनेकेलिएआपकोआमंत्रितकियाजा रहा है

प्रक्रियाकाविस्ता

अस्पतालमेंप्रस्तुतिकेसमयएकईसीजीलियाजाएगा.यदिरोगीसमावेशमानदंडोंकोपूराक
रताहै, तो 2 सीसीरक्तनमूनातैयारकियाजाएगाऔरट्रोपोनिन I
केस्तरकापतालगानेकेलिएभेजाजाएगा।इसेबादमेंएकबारफिरसेदोहरायाजासकताहै - ट्रोपोनिन
I मेंप्रवृत्तिकाअध्ययनकरनेकेलिए.

2डीइकोदिलकेनिकासअंशनिर्धारितकरनेकेलिएकियाजाएगा

संभावितलाभ

प्रतिभागीकोअपनीहालतकाऔरमूल्यांकनप्राप्तहोताहैजोप्रबंधनमेंमददकरेगा

गोपनीयता

अध्ययनकेदौरानएकत्रकीगईसभीजानकारीगोपनीयरखीजाएगी।

वापसी

इसअध्ययनमेंभागीदारीस्वैच्छिकहै।यदिआपइसअध्ययनमेंभागलेनानहींचाहतेहैं; आपलाभकमनहींकरेंगेजिसकेलिएआपहकदारहैं।अध्ययनशुरूकरनेकेबाद, अध्ययनकेदौरानकभीभी, यदिआपअध्ययनसेहटनाचाहतेहैं, तोआपऐसाकरनेकेलिएस्वतंत्रहैं।

साझेदारीकीलागत

अध्ययनकीलागतशोधकर्ताद्वारापैदाकीजाएगी।अध्ययनमेंभागलेनेकेलिएआपकेलिए कोईअतिरिक्तलागतनहींहोगी।

परिणामोंकोप्रकाशितकरनेकेलिएप्राधिकरण

अध्ययनकेनतीजेएमडीडिग्री, समीक्षाऔरप्रकाशनकेपूराहोनेकीआवश्यकताकेहिस्सेकेरूपमेंकेएलईविश्वविद्यालय, बेलगामकोभेजेजाएंगे।

अध्ययनकेदौरानयाभविष्यमेंपूछताछकेमामलेमेंआपनिम्नलिखितव्यक्तियोंसेसंपर्ककरसकते हैं

REG. NO.BG0118009

स्नातकोत्तरछ -

एमडीजनरलमेडिसिन

डॉ _____

प्रोफेसर

चिकित्साविभा ,

जेएनमेडिकलकॉलेज

अनुसंधानअध्ययनमेंभागलेनेकीसहमति

मैंस्वेच्छासेनीचेदीगईरेखापरहस्ताक्षरकरकेइसअध्ययनमेंभागलेनेकेलिएसहमतहूँ।मैं
किसीभीसमयवापसलेसकताहूँ।मैंइसफॉर्मपरहस्ताक्षरकरकेअपनेकिसीभीकानूनीअधिकारकोन
हींछोड़रहाहूँ।नीचेदिएगएमेरेहस्ताक्षरसेसंकेतमिलताहैकिमैंनेइसपूरेसहमतिफॉर्मकोपढ़ाहैयायह
मुझेपढ़ागयाहै,

औरमुझेमेरीस्थानीयभाषामेंसमझायागयाहैऔरमेरेसभीसवालोंकेजवाबदिएगएहैं।मुझेइससहम
तिपत्रकीएकप्रतिदीजाएगी।

प्रतिभागीयाकानूनीरूपसेअधिकृतप्रतिनिधिकेहस्ताक्षर / बाएंथंबप्रिंट।

प्रतिभागीकानाम:

हस्ताक्षर / बाएंथंबइंप्रेशन:

कानूनीरूपसेअधिकृतप्रतिनिधिकानाम:

हस्ताक्षर / बाएंथंबइंप्रेशन:

गवाहकानाम:

हस्ताक्षर / बाएंथंबइंप्रेशन:

जांचकतोंकाकानामऔरहस्ताक्षर:

दिनांकऔरस्थान:

माहितीपूर्णसंमती

शीर्षक : “त्रोपोनिंप्रथमचींभूमिका,डावीकडीलवेंट्रिकुलरइजेक्शनअपूर्णांक<= ४०%
तीव्रएसटीमधीलमायोकार्डियल इन्फेक्शनआहे -
केएलईविकासकहॉस्पिटलआणिएमआरसीबल्लागवीमधीलएकवर्षांच्याविभागीयअभ्यासाचाअ
भ्यास’

अभ्यासअन्वेषक :

REG. NO.BG0118009

पदवीधरपदवी – एमडीजनरलमेडिसिन

जे.एन.मेडिकलमहाविद्यालय,

के.एल.ईविश्वविद्यालय, बेलगावी-590,010

परिचय

ट्रायपोनिन

1

चीभूमिकायाअभ्यासातसहभागीहोण्यासाठीआपल्यालामायोकार्डियलइन्फेक्शनमध्येहृदयाचे
पृथक्करणभागओळखण्यासाठीआमंत्रितकेलेजातआहे.

प्रक्रियांचीव्याख्या

हॉस्पिटलमध्येप्रस्तुतीच्यावेळीईसीजीघेण्यातयेईल.जररुग्णसमावेशनमानदंडपूर्णकरे

लतर

2

सीसीरक्तनमुनाकाढलाजाईलआणिट्रोपोनिन

1

च्यापातळीवरपोहोचण्यासाठीपाठविलाजाईल.प्रवृत्तीचाअभ्यासकरण्यासाठीनंतरपुन्हाएकदा

याचीपुनरावृत्तीहोऊशकते.हृदयाचेनिष्कासनअंशनिश्चितकरण साठी 2 डीइकोकेलाजाईल.

संभाव्यफायदे

सहभागीलात्याच्यास्थितीचेपुढीलमूल्यांकनमिळतेजेव्यवस्थापनासमदतकरेल.

गोपनीयता

अभ्यासादरम्यानगोळाकेलेलीसर्वमाहितीगोपनीयठेवलीजाईल.

सहल

याअभ्यासातसहभागस्वैच्छिकआ. आपणयाअभ्यासातसहभागीहोऊइच्छितनसल्या

स; आपणज्याहकदारआहातत्यांचेफायदेगमावणारनाहीत.

अभ्यासाच्यासुरुवातीसअभ्याससुरूकेल्यानंतर,

जरआपणअभ्यासातूनबाहेरपडूइच्छितअसालतरआपणतसेकरण्यासस्वतंत्रआहात.

सहभागाचीकिंमत

अभ्यासाचीकिंमतसंशोधकांद्वारेघेतलीजाईल.अभ्यासातसहभागघेण्यासाठीआपल्या
साठीअतिरिक्तखर्चहोणारनाही.

परिणामप्रकाशितकरण्यासाठीअधिकृतता

एमडीपदवी,

समीक्षाआणिप्रकाशनपूर्णकरण्यासाठीआवश्यकतेचाएकभागम्हणूनअभ्यासनिष्कर्षांचेपरिणा
मकेएलईविद्यापीठ, बेळगावयांनापाठवलेजातील.

अभ्यासादरम्यानच्याप्रश्नांच्याबाबतीतकिंवाभविष्यातआपणखालीलव्यक्तीशी

संपर्कसाधूशकता

REG. NO.BG0118009

पदवीधरपदवी –

एमडीजनरलमेडिसिन

संशोधनअभ्यासातसहभागीहोण्यासाठीसहमती

खालीदिलेल्याओळीवरसहीकरूनमीयाअभ्यासातभागघेण्यासस्वेच्छेनेसहमतआहे.मी कोणत्याहीवेळीमागेघेऊशकतो.मीयाफॉर्मवरस्वाक्षरीकरूनमाझ्याकोणत्याहीकायदेशीरअधिका रांनासोडतनाही.खालीमाझ्यास्वाक्षरीनेसूचितकेलेआहेकीमीहेसंपूर्णसंमतीफॉर्मवाचलेआहेकिंवा तेमलावाचलेगेलेआहेआणिमलामाझ्यास्थानिकभाषेतस्पष्टकेलेगेलेआहेआणिमाझ्यासर्वप्रश्नां चीउत्तरेदिलीआहेत. मलायासंमतीफॉर्मचीएकप्रतदिलीजाईल.

सहभागीकिंवाकायदेशीरअधिकृतप्रतिनिधीचेस्वाक्षरी / डावाथंबप्रिं .

सहभागीचेनाव:

स्वाक्षरी / डावाथंबप्रभाव:.....

कायदेशीरअधिकृतप्रतिनिधीचेनाव:

स्वाक्षरी / डावाथंबप्रभाव:.....

साक्षीदारांचीनावे:.....

स्वाक्षरी / डावाथंबप्रभाव:.....

तपासकर्तेचेनावआणिस्वाक्षरी:

तारीखआणिठिकाण:.....

ANNEXURE III

PROFORMA

PROFORMA USED FOR DATA COLLECTION

This Proforma, has been used for data collection for the study titled

ROLE OF TROPONIN IN IDENTIFYING LEFT VENTRICULAR EJECTION FRACTION OF $\leq 40\%$ IN ACUTE ST ELEVATED MYOCARDIAL INFARCTION: A ONE YEAR CROSS SECTIONAL STUDY IN KLE DR PRABHAKAR KORE HOSPITAL AND MRC BELAGAVI

Is as follows –

A) PATIENT IDENTIFICATION DATA -

1. Name: _____
2. IP No: _____
3. Age: _____
4. Sex: _____
5. D.O.A.: _____
6. Address: _____
7. Religion: _____
8. Marital Status: _____
9. Occupation: _____

B) PRESENTING COMPLAINTS -

C) PAST HISTORY -

1. Type 2 Diabetes –
2. Hypertension –
3. Other -

D) PERSONAL HISTORY -

1. Smoking –
2. Alcohol –
3. Other -

E) FAMILY HISTORY -

F) GENERAL PHYSICAL EXAMINATION -

1. Anthropometric measurements –

- Height -
- Weight -
- BMI -

2. Vitals –

- PR -
- BP -
- RR -

G) SYSTEMIC EXAMINATION -

1. CVS –
2. RS –
3. PER ABDOMEN –
4. CNS –

H) INVESTIGATIONS -

1. Troponin																
2. ECG	<p style="text-align: center;">ST Elevation in leads</p> <table style="width: 100%; border-collapse: collapse;"> <thead> <tr> <th style="width: 33%; text-align: center;">I</th> <th style="width: 33%; text-align: center;">II</th> <th style="width: 33%; text-align: center;">III</th> </tr> </thead> <tbody> <tr> <td style="text-align: center;">aVL</td> <td style="text-align: center;">aVF</td> <td style="text-align: center;">aVR</td> </tr> <tr> <td style="text-align: center;">V1</td> <td style="text-align: center;">V3</td> <td></td> </tr> <tr> <td style="text-align: center;">V2</td> <td style="text-align: center;">V4</td> <td></td> </tr> <tr> <td style="text-align: center;">V5</td> <td style="text-align: center;">V6</td> <td></td> </tr> </tbody> </table>	I	II	III	aVL	aVF	aVR	V1	V3		V2	V4		V5	V6	
I	II	III														
aVL	aVF	aVR														
V1	V3															
V2	V4															
V5	V6															

I) MANAGEMENT –

J) 2D ECHOCARDIOGRAPHY

Measurements:

LVSD -

LVDD -

RV Free Wall –

Aorta -

Pulmonary Artery –

Left Atrium (AP) –

EF% -

Valves :

Mitral Valve :

Aortic Valve :

Tricuspid Valve :

Pulmonary Valve :

Chambers :

Left Ventricle –

Right Ventricle –

Left Atrium -

Right Atrium –

Doppler Study :

Mitral Valve –

Aortic Valve –

Tricuspid Valve -

Pulmonary Valve –

RWMA :

Pericardial Effusion:

Clots/Vegetation :

ANNEXURE IV

KEY TO THE MASTER CHART

A	-	Anterior
ACG	-	Anticoagulation
ECG	-	Electrocardiogram
F	-	Female
I	-	Inferior
Kg/m ²	-	Kilograms per square meter
M	-	Male
N	-	No
PAH	-	Pulmonary artery hypertension
PAMI	-	Primary Angioplasty in Myocardial Infarction
STK	-	Streptokinase
Y	-	Yes
Rt	-	Retepase

Patient number	In patient Number	Age (Years)	Sex	Chest Discomfort	Breathlessness	Syncope	Diabetes mellitus	Hypertension	Smoking	Alcohol	Body mass index (Kg/m ²)	BMI	ECG	Ejection fraction (%)	Pulmonary artery pressure	Pericardial effusion	Troponin T	Management	I	II	III	aVL	avF	STE V1-V2	V3-V4	V5-V6
1	1017495	66	M	Y	N	N	Y	N	N	N	24	22.00 to 24.99	I	45%	Mild PAH	N	2.81	STK Lysis	0	1	3	0	3	0	0	0
2	969794	68	M	Y	N	N	Y	Y	Y	N	25.8	25.00 to 29.99	I	30%	Mild PAH	N	0.2	ACG	0	2	2	0	2	0	0	0
3	985429	63	M	Y	Y	N	Y	N	Y	Y	26.3	25.00 to 29.99	I	45%	Nr	N	1.31	STK Lysis	0	2	4	0	3	0	0	0
4	977197	65	F	Y	Y	N	N	Y	N	N	18.8	18.5 to 22.9	A	40%	Nr	N	0.1	STK Lysis	1	0	0	1	0	0	2	2
5	978114	65	M	Y	N	N	Y	N	N	N	27.7	25.00 to 29.99	A	45%	Nr	Y	0.47	STK Lysis	0	0	0	0	0	7	8	2
6	978206	60	M	Y	Y	N	Y	N	N	N	24.8	22.00 to 24.99	A	40%	Nr	N	5.73	STK Lysis	0	0	0	0	0	5	2	0
7	978915	55	F	Y	N	N	Y	Y	N	N	24	22.00 to 24.99	A	35%	Mild PAH	N	17.11	STK Lysis	1	0	0	0	0	4	4	2
8	978892	54	M	Y	N	N	N	Y	Y	Y	27	25.00 to 29.99	A	35%	Nr	N	9.23	STK Lysis	0	0	0	0	0	3	2	0
9	5463134	35	F	Y	Y	N	Y	N	N	N	20.8	18.5 to 22.9	A	35%	Nr	N	2.51	Rt	1	0	0	0	0	2	4	0
10	979429	65	F	N	N	Y	Y	Y	N	N	27.3	25.00 to 29.99	A	40%	Nr	N	1.43	ACG	0	0	0	0	0	3	3	0
11	979572	68	F	Y	N	N	N	N	N	N	23.7	22.00 to 24.99	A	45%	Nr	N	3.52	ACG	0	0	0	0	0	0	2	2
12	998450	73	F	Y	N	N	Y	Y	N	N	25.9	25.00 to 29.99	A	30%	Moderate PAH	N	10	ACG	1	0	0	1	0	3	2	0
13	979822	55	M	Y	N	N	Y	N	N	N	22.7	18.5 to 22.9	A	45%	Nr	N	1.1	STK Lysis	1	0	0	0	0	2	5	5
14	980920	58	M	Y	N	N	Y	N	N	N	26.7	25.00 to 29.99	A	50%	Moderate PAH	N	0.05	STK Lysis	1	0	0	1	0	3	2	0
15	986448	65	F	Y	N	N	N	N	N	N	20	18.5 to 22.9	A	30%	Nr	N	10.88	STK Lysis	1	0	0	1	0	3	2	0
16	987062	54	M	Y	N	N	N	N	Y	N	25.7	25.00 to 29.99	A	40%	Nr	N	8.83	STK Lysis	0	0	0	0	0	7	8	5
17	987297	73	M	Y	N	N	Y	Y	Y	N	29.4	25.00 to 29.99	I	50%	Mild PAH	N	0.97	ACG	0	1	2	0	2	0	0	0
18	983097	60	F	Y	Y	N	Y	Y	N	Y	31.7	>30	A	40%	Nr	N	1.71	PAMI	0	0	0	0	0	3	6	0
19	987670	40	M	Y	N	N	N	N	Y	Y	29.1	25.00 to 29.99	A	35%	Nr	N	3.06	STK Lysis	2	0	0	2	0	2	0	0
20	988062	40	M	Y	N	N	N	N	N	Y	32.3	>30	I	50%	Nr	N	1.12	STK Lysis	0	1	2	0	2	0	0	0
21	987917	40	M	Y	N	N	N	Y	Y	N	21.3	18.5 to 22.9	A	40%	Nr	Y	7.81	STK Lysis	0	0	0	0	0	4	8	3
22	989271	65	M	Y	Y	N	Y	Y	N	N	21.3	18.5 to 22.9	A	40%	Mild PAH	N	1.01	ACG	1	0	0	1	0	3	2	0
23	988662	63	M	Y	N	N	N	N	N	N	27.7	25.00 to 29.99	A	40%	Nr	N	5.41	STK Lysis	0	0	0	0	0	5	5	2

24	991047	50	M	Y	N	N	Y	N	N	N	22	18.5 to 22.9	A	45%	Nr	N	0.16	Rt	0	0	0	0	0	0	0	3	2
25	990011	65	F	Y	N	N	Y	N	N	N	21	18.5 to 22.9	A	35%	Nr	N	1.87	ACG	0	0	0	0	0	0	3	2	0
26	992656	80	M	N	N	Y	N	Y	Y	N	20.7	18.5 to 22.9	I	45%	Mild PAH	N	3.78	STK Lysis	0	2	2	0	2	0	0	0	0
27	992804	65	F	Y	N	N	Y	Y	N	N	28.4	25.00 to 29.99	A	45%	Moderate PAH	N	5.8	STK Lysis	1	0	0	1	0	2	4	3	3
28	992982	30	M	Y	Y	N	N	N	Y	Y	30.5	>30	A	40%	Nr	N	1.22	PAMI	4	0	0	4	0	10	9	3	3
29	996895	70	M	Y	N	N	N	N	N	N	27	25.00 to 29.99	I	50%	Nr	N	0.84	ACG	0	2	4	0	3	0	0	0	0
30	997125	73	F	Y	N	N	Y	N	N	N	25	25.00 to 29.99	I	45%	Nr	N	2.14	ACG	0	2	4	0	3	0	0	0	2
31	1005098	55	M	Y	N	N	Y	N	Y	N	25	25.00 to 29.99	A	40%	Nr	Y	0.98	STK Lysis	1	0	0	0	0	4	2	2	2
32	1005103	34	M	Y	N	N	N	N	Y	Y	25.3	25.00 to 29.99	I	45%	Nr	N	1.42	Rt	0	3	4	0	4	0	0	0	0
33	1004553	65	F	Y	N	N	N	N	N	N	27	25.00 to 29.99	A	50%	Nr	N	0.39	STK Lysis	0	0	0	0	0	4	8	3	3
34	1006157	70	M	N	Y	N	N	Y	Y	N	22.8	18.5 to 22.9	A	35%	Mild PAH	N	13.24	ACG	1	0	0	0	0	3	5	0	0
35	921739	48	M	Y	N	N	Y	N	N	N	26.3	25.00 to 29.99	I	45%	Nr	N	3.82	PAMI	0	3	3	3	0	4	4	2	2
36	925642	92	M	Y	N	N	Y	N	N	N	19	18.5 to 22.9	A	40%	Mild PAH	N	0.05	ACG	0	0	0	0	0	2	2	0	0
37	924202	58	M	Y	N	N	N	N	N	N	27.7	25.00 to 29.99	A	50%	Nr	N	0.25	STK Lysis	1	0	0	1	0	0	2	2	2
38	994898	78	M	Y	N	N	Y	N	Y	N	24.8	22.00 to 24.99	I	45%	Nr	N	0.69	ACG	0	3	4	0	4	0	0	0	0
39	923015	82	M	N	Y	N	N	Y	N	Y	24	22.00 to 24.99	A	30%	Moderate PAH	N	1.69	ACG	0	0	0	0	0	4	4	0	0
40	922154	40	M	Y	N	N	N	N	Y	Y	27	25.00 to 29.99	I	50%	Nr	N	0.05	Rt	0	1	2	0	2	0	0	0	0
41	922145	62	M	Y	N	N	Y	N	Y	N	20.8	18.5 to 22.9	I	40%	Nr	N	1.69	STK Lysis	0	4	5	0	4	0	0	0	0
42	920075	65	M	Y	N	N	Y	N	N	N	27.3	25.00 to 29.99	I	40%	Nr	N	2.88	STK Lysis	0	1	2	0	2	0	0	0	0
43	929410	93	F	N	Y	Y	N	Y	N	N	20.7	18.5 to 22.9	A	40%	Nr	Y	0.24	ACG	1	0	0	1	0	3	2	2	2
44	929197	55	F	Y	N	N	Y	Y	N	N	22.7	18.5 to 22.9	A	45%	Mild PAH	N	1.51	STK Lysis	0	0	0	0	0	4	8	5	5
45	930238	68	M	Y	Y	N	Y	N	Y	Y	26.7	25.00 to 29.99	A	45%	Mild PAH	N	0.17	ACG	2	0	0	1	0	6	8	3	3
46	984832	47	M	Y	N	N	N	Y	Y	N	20	18.5 to 22.9	A	55%	Nr	N	0.62	STK Lysis	0	0	0	0	0	4	2	0	0
47	985994	60	M	Y	N	N	N	N	N	N	25.7	25.00 to 29.99	I, A	45%	Nr	N	2.22	STK Lysis	2	2	0	0	1	8	9	3	3
48	937135	62	M	Y	N	N	Y	Y	N	Y	29.4	25.00 to 29.99	A	45%	Nr	N	1.26	ACG	0	0	0	0	0	7	8	4	4
49	936324	72	F	Y	N	N	Y	N	N	N	31.7	>30	I	40%	Nr	N	0.1	ACG	0	1	3	0	3	0	0	0	0
50	932642	49	M	Y	N	N	Y	N	N	N	29.1	25.00 to 29.99	A	50%	Nr	N	0.1	STK Lysis	1	0	0	1	0	3	2	0	0
51	935101	57	M	Y	N	N	N	N	Y	Y	32.3	>30	A	50%	Nr	N	0.2	STK Lysis	1	0	0	0	0	4	4	2	2
52	933327	82	M	Y	Y	N	Y	N	N	N	21.3	18.5 to 22.9	A	35%	Moderate PAH	N	0.11	ACG	1	0	0	1	0	0	2	2	2
53	933246	65	F	Y	Y	N	Y	N	N	N	21.3	18.5 to 22.9	I	35%	Nr	N	1.31	STK Lysis	0	2	4	0	3	0	0	0	2
54	935407	65	F	Y	N	N	N	N	N	N	27.7	25.00 to 29.99	I	45%	Nr	N	0.18	ACG	0	2	2	0	2	0	0	0	0

55	934013	62	M	Y	N	N	N	Y	N	N	22	18.5 to 22.9	A	50%	Mild PAH	N	0.84	STK Lysis	0	0	0	0	0	0	3	2	0
56	941387	91	M	Y	N	N	N	N	N	N	21	18.5 to 22.9	A	40%	Mild PAH	N	10	ACG	0	0	0	0	0	0	6	8	2
57	937472	35	M	Y	N	N	N	N	Y	Y	20.7	18.5 to 22.9	A	40%	Nr	N	1.57	Rt	0	0	0	0	0	5	5	2	
58	942201	52	M	Y	N	N	Y	N	Y	N	28.4	25.00 to 29.99	I	45%	Nr	N	0.29	STK Lysis	0	1	2	0	2	0	0	0	0
59	936454	54	M	Y	Y	N	Y	Y	N	N	30.5	>30	I	40%	Nr	N	7.61	STK Lysis	0	13	14	0	14	0	7	6	6
60	939721	60	F	N	N	Y	Y	Y	N	N	27	25.00 to 29.99	A	30%	Mild PAH	Y	1.55	STK Lysis	1	0	0	1	0	2	4	3	3
61	984631	60	F	Y	N	N	N	N	N	N	25	25.00 to 29.99	A	40%	Nr	N	2.4	ACG	4	0	0	4	0	10	9	2	2
62	944726	84	F	Y	N	N	N	N	N	N	25	25.00 to 29.99	I	45%	Nr	N	0.26	ACG	0	1	2	0	2	0	0	0	0
63	949244	60	M	Y	N	N	N	Y	N	N	25.3	25.00 to 29.99	A	50%	Mild PAH	N	0.75	STK Lysis	0	0	0	0	0	0	3	0	0
64	948272	65	F	Y	N	N	N	N	Y	N	28	25.00 to 29.99	A	50%	Mild PAH	N	0.28	STK Lysis	2	0	0	2	0	2	0	0	0
65	944459	36	M	Y	N	N	N	N	Y	Y	32.6	>30	A	55%	Nr	N	0.18	PAMI	0	0	0	0	0	0	3	2	2
66	952386	59	M	Y	N	N	Y	N	N	Y	24.2	22.00 to 24.99	A	50%	Nr	N	0.72	STK Lysis	0	0	0	0	0	2	2	0	0
67	952434	75	M	Y	N	N	Y	N	N	N	23.5	22.00 to 24.99	A	45%	Nr	N	1	ACG	1	0	0	1	0	3	2	2	2
68	951184	55	M	Y	N	N	Y	N	N	N	26	25.00 to 29.99	I	45%	Nr	N	2.56	STK Lysis	0	2	4	0	3	0	0	0	0
69	950781	72	M	Y	Y	N	N	Y	Y	N	21.5	18.5 to 22.9	A	35%	Moderate PAH	N	2.32	ACG	0	0	0	0	0	7	8	5	5
70	949462	70	M	N	Y	N	N	Y	N	N	23.6	22.00 to 24.99	A	30%	Nr	N	0.12	ACG	0	0	0	0	0	5	5	2	2
71	950817	60	M	Y	N	N	Y	N	Y	Y	27.2	25.00 to 29.99	A	45%	Mild PAH	N	2.17	STK Lysis	0	0	0	0	0	4	6	0	0
72	958060	65	M	Y	N	Y	Y	N	Y	N	24	22.00 to 24.99	A	40%	Nr	N	4.15	STK Lysis	5	0	0	0	0	10	13	10	10
73	956714	47	M	Y	Y	N	N	N	Y	Y	28.7	25.00 to 29.99	I	45%	Nr	N	0.29	Rt	0	3	4	0	4	0	0	0	0
74	960676	52	F	Y	N	N	N	N	N	N	32.9	>30	A	50%	Nr	N	0.3	ACG	0	0	0	0	0	0	2	2	2
75	958491	70	F	Y	N	N	N	N	N	N	20	18.5 to 22.9	I	45%	Nr	N	0.36	ACG	0	3	3	0	3	4	4	2	2
76	957936	65	M	Y	N	N	Y	N	N	N	22	18.5 to 22.9	A	50%	Nr	N	0.53	STK Lysis	0	0	0	0	0	3	3	0	0
77	965925	59	M	Y	N	N	N	Y	Y	N	22	18.5 to 22.9	A	50%	Mild PAH	N	0.55	ACG	1	0	0	0	0	2	4	0	0
78	967725	73	M	Y	N	N	Y	N	N	Y	24.5	22.00 to 24.99	A	45%	Nr	N	0.66	ACG	1	0	0	1	0	0	2	2	2
79	965346	56	M	Y	Y	N	Y	Y	Y	Y	23.4	22.00 to 24.99	A	45%	Nr	N	1.43	STK Lysis	0	0	0	0	0	7	8	5	5
80	965717	65	M	Y	N	N	N	N	Y	N	23.6	22.00 to 24.99	I	45%	Mild PAH	N	0.41	ACG	0	1	2	0	2	0	0	0	0
81	964480	67	F	Y	N	N	Y	N	N	N	27.3	25.00 to 29.99	I	40%	Nr	N	2.12	ACG	0	1	2	0	2	0	0	0	0
82	974003	62	M	N	Y	N	N	N	Y	N	26	25.00 to 29.99	A	45%	Mild PAH	Y	1.85	STK Lysis	1	0	0	1	0	3	2	0	0
83	969897	46	M	Y	N	N	Y	N	N	N	28.5	25.00 to 29.99	A	40%	Nr	N	7.28	STK Lysis	1	0	0	0	0	4	4	2	2
84	967674	48	M	Y	N	N	N	N	N	N	23.2	22.00 to 24.99	I	40%	Nr	N	7.52	STK Lysis	0	2	4	0	3	0	0	0	2
85	913177	63	F	Y	N	N	Y	Y	N	N	25	25.00 to 29.99	I	45%	Nr	N	0.98	ACG	0	1	2	0	2	0	0	0	0

86	968562	65	F	Y	Y	N	N	N	N	N	20.5	18.5 to 22.9	A	40%	Nr	N	2.17	ACG	1	0	0	1	0	3	2	0
87	968245	68	M	Y	N	N	N	N	Y	N	23.4	22.00 to 24.99	A	50%	Nr	N	1.44	ACG	1	0	0	1	0	2	4	3
88	980534	80	F	Y	N	N	Y	N	N	N	20.2	18.5 to 22.9	I	40%	Mild PAH	N	3.96	ACG	0	2	4	0	3	0	0	0
89	980297	60	F	Y	Y	N	N	Y	Y	Y	26.6	25.00 to 29.99	A	45%	Mild PAH	N	0.28	STK Lysis	4	0	0	4	0	10	9	3
90	978946	75	M	Y	N	N	Y	N	N	N	25.2	25.00 to 29.99	A	45%	Nr	N	2.08	ACG	0	0	0	0	0	4	8	3
91	978114	65	M	Y	Y	Y	Y	Y	N	Y	22.4	18.5 to 22.9	A	40%	Nr	N	0.47	ACG	2	0	0	2	0	3	0	0
92	994958	52	M	Y	N	N	N	N	Y	Y	21	18.5 to 22.9	A	40%	Mild PAH	N	1.73	STK Lysis	2	0	0	1	0	6	8	4
93	976842	60	M	Y	N	N	N	N	N	N	27.5	25.00 to 29.99	I, A	50%	Nr	N	0.28	STK Lysis	2	2	0	0	1	8	9	3
94	979601	61	F	Y	N	N	Y	Y	N	N	24	22.00 to 24.99	A	50%	Nr	N	0.41	STK Lysis	5	0	0	0	0	10	13	10
95	978892	54	M	Y	N	N	Y	N	N	N	29.5	25.00 to 29.99	A	35%	Nr	N	9.23	STK Lysis	0	0	0	0	0	3	2	0
96	977675	65	F	Y	Y	N	N	N	N	N	30	>30	A	30%	Nr	N	1.43	ACG	1	0	0	1	0	3	2	2
97	1016474	63	M	Y	N	N	N	Y	Y	N	23	22.00 to 24.99	A	45%	Nr	N	0.71	STK Lysis	0	0	0	0	0	5	5	2