TO STUDY THE CORRELATION BETWEEN ECG AND 2D ECHOCARDIOGRAPHY IN LOCATING ACUTE MYOCARDIAL INFARCTION AND PREDICTING COMPLICATIONS.

By

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In partial fulfillment of the requirements for the degree of

MD

IN

GENERAL MEDICINE

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LIST OF ABBREVIATIONS USED

2D-Echo	:	Two Dimensional Echocardiography
AMI	:	Acute Myocardial Infraction
Asymp	:	Asymptomatic
CAD	:	Coronary artery disease
CHD	:	Coronary heart disease
Chol	:	Total cholesterol
CKMB	:	Creatine kinase MB
CVA	:	Cerebrovascular accidents
CVD	:	Cardiovascular disease
DM	:	Diabetes mellitus
ECG	:	Electrocardiography
EF	:	Ejection fraction
Epig. Pain	:	Epigastric pain
Haemte	:	Haematemesis
HDL	:	High density lipoproteins
HTN	:	Hypertension
IHD	:	Ischemic heart disease
Impr	:	Improved
LBBB	:	Left Bundle Branch Block
LDL	:	Low density lipoproteins
LH	:	Left sided hemiplegia
LVF	:	Left ventricular failure
M	:	Mixed
NSTEMI	:	Non ST-elevation myocardial infarction
Occ VPC's	:	Occasional Ventricular premature contraction

RBBB	:	Right Bundle Branch Block
RH	:	Right sided hemiplegia
RVMI	:	Right ventricle myocardial infarction
SGOT	:	Serum glutamic oxaloacetic acid transferase
STEMI	:	ST-elevation myocardial infarction
TC	:	Tobacco chewing
Tg	:	Triglycerides
UAP	:	Unstable angina pectoris
UN	:	Uneventful
V	:	Vegetarian
Vomit	:	Vomiting
VT	:	Ventricular tachycardia
ACS	:	Acute coronary syndrome
AF	:	Atrial fibrillation
AIVR	:	Accelerated idio ventricular rhythm
ALMI	:	Anteriolateral wall myocardial infarction
AVN	:	Atrio-ventricular node
AWMI	:	Anterior wall myocardial infarction
BBB	:	Bundle branch block
CHB	:	Complete heart block
IWMI	:	Inferior wall myocardial infarction
LAHB	:	Left anterior hemiblock
NSTEMI	:	non-ST-elevation myocardial infarction
PAT	:	Paroxysmal atrial tachycardia
RA	:	Reperfusion arrhythmia.
RCA	:	Right coronary artery
RV	:	Right ventricle

SAN	:	Sino-atrial node
SB	:	Sinus bradycardia
SK	:	Streptokinase
ST	:	Sinus tachycardia
VPB	:	Ventricular premature beat

ABSTRACT

Background and purpose:

Cardiovascular diseases are at present the leading causes of death in the developed and also developing countries. IHD is a great killer accounting for15% of all mortality in India.

The purpose of this study is to correlate between ECG & 2D Echocardiography in locating acute myocardial infarction and Predicting complications.

Methods:

86 cases of acute myocardial infarction admitted in ICCU of BLDE (DEEMED TO BE UNIVERSITY) Shri. B. M. Patil Medical College HOSPITAL and Research Centre, VIJAYAPURA, India between December 2016 to August 2018 were studied. All patients were evaluated for risk factors. 12-leadECG and 2D ECHOCARDIOGRAPHY with cardiac enzyme estimation was done. Complication such as Arrhythmias, cardiogenic shock, LVEF status and mortality were noted, result were studied.

Results:

On ECG, infarct site were as follows in the order of decreasing frequency.

Inferior wall with right ventricle > extensive anterior >anterior septal >antero-lateral. The lesions seen on ECG correlated broadly with those seen on echocardiography with more elaborated detail was noticed. 62% were males and 38% were females of which incidence being more common between 4th to 7th decade of life. Incidence of AMI was more common in patients with smoking/tobacco chewing and hypertension. Incidence of AWMI was equal to IWMI. Out of all arrhythmias, VT was seen in most common preponderance to AWMI. CHB and SB were commonly seen with IWMI,. BBB was more common in AWMI than IWMI

Conclusion:

The location of MI seen on ECG correlated broadly with those seen on echocardiography. Echo was able to elaborate regional wall motion abnormalities in detail than ECG. Tachyarrhythmias are more common with AWMI and bradyarrhythmias in IWMI.

Key Words : ECG, echocardiography, AMI

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INTRODUCTION

Cardiovascular diseases are at present the leading cause of death in developed as well as developing countries[1]. Ischemic heart disease is a great killer accounting for 25.1% of total death in urban areas are attributable to disease of circulatory system among which IHD is major entity[2]. Age adjusted death rates for coronary heart disease declined by two-third in the last decade, reflecting the identification and reduction of risk factors as well as improved treatment and intervention. A growing prevalence of obesity type 2 diabetes and metabolic syndrome which are important risk factors, now threatens to reverse the progress that has been made in the age adjusted reduction in the mortality rate of coronary heart disease.

For many years cardiovascular disease was considered to be more common in men than in women, but today the numbers has risen in women than men. Exercise ECG has lower diagnostic accuracy in the prediction of epicardial obstruction in women than in men.

ECG & 2D echocardiography helps in the diagnosis and prognostification of myocardial infarction. These investigation are non invasive and can be done in less advanced centers. Hence this study is undertaken to correlate the site of infarction by Electrocardiography & 2D Echocardiography

Myocardial infarction is often depicted as a modern disease, it was recognized before the modern era by Morgagni in 1761 and more clearly by Heberden . It is one of the most common diagnoses in hospitalized patients in industrialized countries& developing countries. Although after admission for myocardial infarction has declined

by approx. 30%. Over past two decades approx 1 of every 25 patients who survive the initial hospitalization dies in the first year after Acute myocardial infarction.[3]

In most patients it results from thrombotic occlusion of the related vessels resulting in infarct myocardial infarction and necrosis set in within about 20 to 40 minutes this occurs as a wave front starting from the sub endocardial region and progressing to the sub epicardial region the entire process usually takes 6 hours to complete therefore any intervention for limiting in fact size should be initiated in this time window of 6 hours .

It has been observed that various risk factors such as age male sex smoking obesity tobacco family history, hypertension, hyperlipidemia ,Diabetes mellitus, type A personality ,sedentary Lifestyle play a role in occurrence of myocardial infarction.[4]

Hence, this study is undertaken to correlate the site of infarction and EF by ECG and 2D Echo and also to assess the severity and prognosis of myocardial infarction

OBJECTIVES OF STUDY

1. To study the correlation between ECG and 2D ECHOCARDIOGRAPHY in locating acute Myocardial Infarction and predicting complications.

REVIEW OF LITERATURE

Historical view

Although myocardial infarction is Always depicted as a modern disease it was clearly recognised before the modern era by morgagni in 1761 and more clearly by Hamilton An early personal description of myocardial infarction was given by John Hunter surgeon to Saint George Hospital London who himself experienced what was probably a Myocardial Infarction in 1773 the description of his subsequent autopsy describes the scarred areas in his heart.

In 1910 Sir William osler delivered a lecture to the Royal College of physicians which is known that he had found the condition to be more common among guest is private or upper class patients than the poor classes he saw at Saint bartholomew Hospital[5]

Electrocardiography

Nobel Prize in 1924 was given to William into one of Netherlands for his discovery of the mechanism of the lectrocardiogramWilliam enthovan a physiologists found the capillary electrometre 2 sluggish and invented the string Galvanometer which is still in use today . the electrocardiogram would be considered a necessary confirmatory test for the clinical diagnosis of myocardial ischemia or infarction[6]

Wilson predicted the value of ECG as a Diagnostic effect in 1930 first recognise the characteristic curves of cardiac Infarction in ECG. In 1940 singer and team describe the relation between angina pectoris, coronary thrombosis and myocardial infarction and importance of the intern coronary Collateral circulation.

In 1945 successfully visualised coronary arteries in men. In 1948 Prinzmetal confirmed that anastomosis are conspicious in the superficial layer of the myocardium

then in the inner layer. In 1948 anticoagulant therapy was introduced. In 1954 Carmen introduced estimation of serum glutamate oxaloacetate transaminase for the diagnosis of myocardial infarction.

Winbergs operation of direct implantation of an internal mammary artery into the estimate area of myocardium was the only one which gave objective evidence of promoting a Collateral blood supply. In 1962 the development of selective coronary angiography by Mason, made a direct operation in the coronary arteries a practical consideration.

In present day ECG generally first investigation available for making diagnosis of acute Myocardial Infarction in patient presenting with acute severe chest pain. It is reliable reproducible can be applied serially when properly interpreted the cornerstone of laboratory diagnosis of myocardial infarction.

Echo cardiography One of the most fruitful collaboration in the history of cardiology was between once Edler Swedish cardiologist and helmet hertz a Swedish physicist in 1952 they adopted in human after use of Sonar device for detecting submarines in World War 2 and recorded across from the walls of the heart of one of the co inventors hertz heart and launched a field of electrocardiography these investigators provided continuous recordings of the movements of the heart valve and of the normal and disease. The mitral valve non invasive imaging represents enormous advances both in diagnosis of heart disease and in care of cardiac patients.[7]

Echocardiography uses ultrasound to image the heart and great vessels. A transducer containing piezoelectric element which when electrically activated emit ultrasound energy.Transducer will be described by its frequency range from 128 to 512 channels in modern scanners recently with a rectangular array of piezoelectric crystals

developed a three-dimensional Beam to be propagated in real time there are two basic mechanism by which an ultrasound transducer creates the fan shaped sector of ultrasound required for 2D imaging. A mechanical scanner utilizes one to three isoelectric crystals are rotated high velocity to create an Arc. The piezoelectric crystals corporate into the scan head serve the dualpurpose of transmitting and receiving ultrasound energy.[8]

In echocardiography three Types of studies are performed M mode two-dimensional and Doppler. In M mode echocardiography a single transducer emitting frequency 1000 to 3000 along a single line provides “icepick” view of heart ok with excellent temporal resolution. Two dimensional echocardiography produces an image into two distance dimension by scattering the sound beam through an Arc of up to 90 degree 30 times per second. It Provides excellent spatial resolution permitting analysis of movement in real time from multiple transducer position on the chest and upper abdomen it is all possible to obtain imaging and Doppler echocardiography via the oesophagus.[9]

Anatomy of Heart is composed of four Chambers right and left Atrium right and left ventricles four valve mitral tricuspid pulmonary and aortic.

Coronary circulation :

The left and the right coronary arteries supply the heart. The left main coronary divides into the left anterior descending artery (LAD)and the left circumflex artery (CX.) The LAD runs in the anterior interventricular groove supplies the anterior part of the septum and wall and apex of the left ventricle. The left circumflex artery gives marginal branches that supply, posterior inferior segments of the left ventricle. The right coronary artery runs in the right atrioventricular groove giving

branches that supply atrium ventricle infero-posterior aspect. The venous system drains ntocoronary sinus, in inferior atrioventricular groove and to the right Atrium.

Lymphatics drain into thoracic duct. Nerve supply of the heart sympathetic and parasympathetic.

The conduction system of the sinoatrial node, atrioventricular node, the his bundle right and left branches and purkinje network

PHYSIOLOGY

The basic unit of myocardial contraction is the sarcomere. During contraction, shortening of the sarcomere results from the actin and myosin molecules interdigitating, without altering the length of either molecule. Contraction is initiated when calcium is made available during the plateau phase of the action potential by calcium ions entering the cells and being mobilized from the sarcoplasmic reticulum. As its concentration rises, calcium binds to troponin, precipitating contraction. The force of cardiac muscle contraction is regulated by the influx of calcium ions through 'slow calcium channels'. The extent to which the sarcomere can shorten determines stroke value of the ventricle[10]

CORONARY ANATOMY PLUS PATHOLOGICAL ANATOMY

Coronary arteriographic studies have clarified the usual pathological anatomy associated with STEMI (ST elevation myocardial infarction). Generally in patients with STEMI who come to necropsy, more than 1/3 rd of coronary artery is severely narrowed.

One-third to two thirds of patients with STEMI have critical obstruction (to less than 25% of luminal area) of all three coronary arteries, whereas the remainder are equally divided between those having one-vessel disease and those having two-vessels disease. Most STEMI with transmural infarcts occur distal to an acutely totally occluded coronary artery with thrombus superimposed on ruptured plaque. However, the converse is not the case, in that total occlusion of coronary artery is not always associated with myocardial infarction. Collateral blood flow and other factors such as the level of myocardial metabolism, the presence and location of stenosis in other coronary arteries, the rate ofdevelopment of obstruction and the quantity of myocardium supplied by the obstructed vessels – all influence the viability of myocardial cells distal to the occlusion. In many series of patients studied at necropsy or by coronary arteriography, a small number (5%) of patients with STEMI are found to have normal coronary vessels. In these patients, anembolous that has lysed, a transiently occlusive platelet aggregate, or a prolonged episodeof severe coronary spasm may have been responsible for the reduction in coronaryflow.[11]

EFFECTS OF THE OCCLUSION OF THE CORONARY VESSELS:

I) Left coronary artery divides into:

A) Left anterior descending artery (LAD)

B) Left circumflex.

A) Left anterior descending artery (LAD)

a) Occlusion of the main trunk of anterior descending branch of LAD ⇒extensive anterior wall infarction.

b) Occlusion of LAD right branch leads to anteroseptal infraction.

c) Occlusion of LAD left branch to antero-lateral infarction.

d) Peripheral occlusion of main trunk of LAD leads to apical infarction.

B) Left circumflex artery:

Occlusion of the circumflex branch leads to postero-lateral infarct in most cases and extensive posterior wall infarction in 10% of cases.

II) Right coronary artery occlusion:

a) Occlusion of main trunk leads to posterior wall infarction including inferior wall.

b) Occlusion of the terminal branches leads to postero-lateral infarcton.[12]

PHYSIOLOGY AND PATHOPHYSIOLOGY OF CORONARY CIRCULATION

Normally the coronary arteries are end arteries, though the functional anastomoses are present and become active under unphysiological state. These anastomoses are between:

a) Branches of one coronary artery with that of the other.
b) Thebesian vessels and cavity of the heart.
c) Arterioluminal and arteriosinusoidal vessels with the cavity of the heart and
d) Extracardiac anastomosis.

The normal values of coronary inflow are:

During rest, for each 100gm of the left ventricle, the left coronary inflow varies from 65 to 85 ml/min. The arteriovenous O2 difference is very high and is about 10-15 ml per 100 ml. So the extraction of O2 by the cardiac muscle is very high.

During heavy exercise, minute volume increases about ten times, and coronary inflow also rises ten folds, i.e., about 2 liters. The O2 usage of heart is very high. The blood in the anterior cardiac veins or in the coronary sinus shows 20% saturation with O2.

During excerise, heart consumes about 250ml of O2 per minute (Same as the resting O2 consumption of the whole body). The arteriovenous O2 difference of heart is about 10-15% (Skeletal muscle 5-6%). This is equivalent to a minute flow of about 2 liters of blood through the coronary vessels.

Variations of coronary inflow during different phases of cardiac cycle:

Ventricular action affects coronary circulation in two ways:

a) By altering the aortic pressure
b) By exerting a variable degree of compression on the coronary vessels. The following phases are seen.

Coronary Inflow:

1) Left coronary artery:

During isometric contraction phase: Coronary inflow sharply falls and reaches minimum and even falls below the zero level due to backflow. Because, the aortic pressure is at minimum and the compression on the coronary vessels is maximum.

During maximum ejection phase: coronary inflow rises rapidly due to sudden rise of aortic pressure and reaches its maximum when the aortic pressure is maximum.

During reduced ejection phase: the coronary inflow again falls, because, the aortic pressure is falling and the compression is continued.

During isometric relaxation phase: the coronary inflow sharply rises, because, the aortic pressure is fairly high and the compression is minimum. Maximum coronary filling takes place during this phase due to fall of coronary vascular resistance.

During rapid ventricular filling phase: the coronary inflow continues to rise but slowly, because, the relaxation of the cardiac musculature continues, and the vessels open up further.

In the later part of diastole, the coronary inflow slowly diminishes, because, theaortic pressure is falling and the coronary vessels are stretched due to filling of heart and the consequent elongation of cardiac muscles.

2) Right coronary artery:

During isometric contraction phase, the coronary inflow sharply falls & rapidly rises again during the maximum ejection phase & falls during the reduced ejection phase. In the isometric relaxation phase the coronary inflow rises but not so steeply like the left coronary inflow.

Factors influencing coronary circulation:- The amount of blood passing through the coronary vessels is directly proportional to the work done by the heart. Blood flow can be adjusted on two principles:

1. By adjusting the lumen of the coronary vessels.
2. By adjusting the mean aortic pressure.

The following factors modify coronary circulation, through one or both the above principles:

1) Mean aortic pressure: It is the chief motive force for driving blood into the coronary vessels. Any alteration of aortic pressure wall, therefore, cause parallel changes in coronary circulation.
2) Role of cardiacoutput: Obviously, the coronary inflow is directly proportionalto the cardiac output. Increased output raises coronary inflow in two ways:
   a) By raising the aortic pressure.
   b) By reflex inhibition of the vagal vasoconstrictor tone.

3) Metabolic factors: With the increased metabolism of heart, the O2 requirement is increased and the circulation is greatly increased. There is a causal relationship between the myocardial metabolic activity, oxygen consumption & coronary blood flow. In the normal heart blood oxygen content of coronary sinus is low under a variety of physiological conditions which supports the view of metabolic regulation of coronary blood flow (CBF) by reactive hyperemia.

4) CO2 & O2: It has been observed through various experiments that if O2requirement of the heart is increased then the coronary circulation is increased. Furthermore the O2 supply to the heart muscle is decreased then the coronary flow increased. But, if the O2 is supplied more than it is required then the coronary circulation is decreased.

5) Heart rate: when the heart rate is increased, minute cardiac output, aortic blood pressure may increase but the stroke volume decreases.

Other factors also have an effect on the coronary circulation like:

i) Effects of Ions ii) Polypeptides iii) Adenine nucleotides

iv) Cardiac sympathetic & parasympathetic nerves

v) Hormones (thyroid, adrenaline & noradrenaline, pitressin and acetylcholine)

vi) Temperature vii) Muscular exercise and excitement viii) Anaemia

ix) Intraventricular pressure x) Transfusion xi) Extravascular pressure

xii) Viscero cardiac reflex.

Pathological physiology of angina pectoris & acute myocardial infarction

Ischemia is the cause of pain of angina pectoris &of acute myocardial infarction. Angina pectoris is thus a symptom of cardiac ischemia but not a disease. The anginal pain is often associated with inadequate blood supply to myocardial requirement. In ischemic heart disease the patient often complains of this pain following a heavy meal or physical exertion or an excitement. The pain is due to accumulation of the metabolites during coronary ischemic. The metabolites stimulate the nerve endings.

Predisposing cause of myocardial infarction is the coronary obstruction due to atherosclerosis. The obstruction may be due to gradual shortening of the vessel itself by the deposition of lipid in vessel or by the locally formed thrombus. If the ischemia is prolonged then it may lead to irreversible change or necrosis.[12]

Etiology & risk factors for acute MI

Almost all myocardial infarctions occur due to atherosclerosis of coronary arteries. The concept that occlusion of coronary artery leading to acute myocardial infarction appears to be the final common pathway resulting from a complex & dynamic interaction among coronary atherosclerosis, vasospasm, plaque rupture, platelet activation & aggregation &fibrin deposition-all ultimately leading to the thrombosis in the coronary artery.

Myocardial infarction was previously considered as disease of adults, but the incidence of myocardial infarction in young adults i.e., 40years or below is increasing because of the life style especially in India & South East Asia.

The major risk factors are tobacco, family history, hypertension, hyperlipidemia, diabetes mellitus, obesity, type A personality & sedentary life style.

The minor risk factors are age, male, sex, hypercoagulability, hyperhomocystinemia, post menopausal state & women on oral contraceptives.

Smoking:

In a study by Kantiz[13] in USA, 81% were males & 19% female, the major riskfactors were tobacco use 81%, followed by family history 40%, hypertension 26%hyperlipidemia 20%. In his study smoking emerged as the main coronary risk factor &atherosclerosis continues to be the major etiology.

Cigarette smoking increases risk two to three fold & interacts with other risk factors to multiply risk. There is no evidence that filters or other modification of the cigarette reduce risk[14]

Acute myocardial infarction in young adults, although earlier considered to be a disease of upper class people because of their dietary & sedentary habits, low class individuals were found even more prone because of widespread chronic smoking habit prevalent in them. Majority of them ignored chest pain as muscular, resulting in high "door to needle time for thrombolytic therapy". [15]

Family history of IHD, Hyperlipidemia, Hypertension:

James et al.[16] (1999) in his study states that, a strong family history of CAD infirst-degree relatives, the risk was considered 90% alone.

Hyperlipidemia in 52%, defined as above the 95th or 80th centile for patient's age & sex. Systemic HTN, if the blood pressure is more than 160/95 mm of Hg on three

occasions. Diabetes mellitus was defined as two fasting plasma glucose value [(> 6.6mmol/L) i.e., ≥ 126 mg/dl] or insulin dependence.

In another study by Gregory et al.[17] a family history of significant atherosclerosis as a risk factor was present in 69% of the younger patients. Death from MI was relatively more common in younger patients 27% than older patients 17%.

Hypertension was considered if ≥ 160/90mm of Hg at the time of discharge which was more common in older patients.

Of the younger infarction patients, 58% were obese exceeding the upper limits of ideal body weight by 20% or more. Hypertension, diabetes mellitus, & hyper lipidemia have been associated with obesity. Hyperlipidemia was seen in 61% of younger patients as a risk factor.

Elevated LDL cholesterol is considered a major cause of CHD. Numerous prospective epidemiological studies have identified a continuous, graded, direct relationship between serum total cholesterol level & CHD incidence.[18] The relation between triglycerides & CHD has been less clear. But hypertriglyceridemia has been found to be an independent risk factor in women. [19]

Peter. P. Toth et al.[20] showed that high HDL levels are associated with longevity& are protective against the development of atherosclerotic disease. In Framingham study, risk for CAD increases sharply as HDL levels fall progressively below 40 mg/dl.

In Quebec cardiovascular study, for every 10% reduction of HDL, risk for CAD increased 13%. So low HDL is an independent risk factor for CAD.

Obesity:

Hubert H.B et al.[21]in his study showed that association between obesity, increased incidence & earlier atherosclerotic coronary heart disease & reduced survival have been observed for many years. Obesity may lead to atherosclerosis by a variety of mechanisms, including predisposition to hypertension, diabetes mellitus, hyperlipidemia with elevated LDL & decreased HDL levels & possibly the association with decreased physical activity. If an individual who is only moderately obese & without other risk factors, obesity does not appear to impart an independent risk.

Study by Pisundeyer[22]in 1993, found that obesity strongly & positively correlates with the risk of CHD in univariate analysis. Obesity promotes insulin resistance, hyperinsulinemia, hypertension, hypertriglyceridemia, low HDL cholesterol &LVH.

Obesity was defined as relative body weight over 0.28, corresponding to an overweight of 20-25% i.e. Weight (Kg) /Height (cm2)* 100.[23]

Diabetes Mellitus:

In Reavan's[24] study in 1998, resistance to insulin stimulated glucose uptake &compensatory hyper insulinemia are the common metabolic bases for a cluster of coronary risk factors, particularly hypertension, diabetes, hypertriglyceridemia, low HDL, predominance of small, dense LDL, & an increased plasminogen activator inhibitor concentration.

In a study by Deirdre R. Blake[25] (2004) examined the prevalence of CHD risk factors & incident CHD events according to baseline impaired glucose tolerance (IGT) &impaired fasting glucose (IFG) using current American Diabetes Association

(ADA)criteria and modified criteria that used a lower cut point of IFG of 5.55mmol/L(100mg/dl) which was recently adopted. Those with isolated IGT or IFG + IGT had more atherogenic lipid profiles, with higher triglyceride & lower HDL cholesterol levels than those with NGT or isolated IFG. They showed that IGT, compared with NGT or isolated IFG, is associated with elevated baseline CHD risk factors. Thus individuals with IGT should be more aggressively targeted with strategies to lower CHD risk than those with IFG.

Acute myocardial infarction is the leading cause of death in diabetic patients &approximately 25% of MI survivors have diabetes. DM is an independent risk factor for CHD increasing risk by 2-3 times for men & 3-5 times for women. [26]

Personality types:

Individuals with certain personality characteristics appear predisposed to develop atherosclerosis, as in type A personality with time urgency, aggressive, ambitious, competitive, impatient & frequently frustrated, coronary risk is increased. This is seen in both men & women. The association is more prone among white collar than blue collar workers. The mechanism remains obscure, but possible atherogenic features include high circulating catecholamine levels that may predispose to hypertension, abnormalities in platelet function, mobilization of fatty acids & hyperlipidemia. [27,28]

In a study by Badui E et al [29], 142 patients were studied, 124 were males& 18 females showed that 106 i.e. 75% had emotional stress due to type A personality &101 (71%) smoking & 24% had no risk factors.

In another study by Shah. S.U., White. A. et al. (2004), there is a quite convincing evidence regarding relationship between disease of the psyche & conditions affecting the cardiovascular system.

A type A personality had always been considered as a significant risk of developing IHD compared with type 'B' (calm, laid back & non – aggressive) individuals. There is evidence that the risk of depression in women with IHD maybe twice as high as that of men. The prevalence of depression is also increased in patient with stable IHD & in patients who under went CABG (coronary artery bypass grafting).

After MI, depression may increase the risk of future coronary events, both over short &longer periods of time.

Chronic anxiety and anxiety disorders such as panic & phobias appear to exert a negative influence on the heart. High levels of phobic anxiety were associated with an almost fourfold increase in the relative risk for fatal IHD in the Northwick park study.

Depressive symptom seems to be influential in predicting IHD. There is a clear association with increased risk of sudden cardiac death & phobic anxiety. [30]

In Rozanskis[31] study showed that an episode of anger is capable of triggering acute MI. In the presence of atherosclerosis, mental stress induces silent MI. Alcohol:

Shaper et al[32] (2000) noted that higher levels of alcohol intake $\geq$ 3 drinks per day are associated with increased mortality in men with previous myocardial infarction.

Other risk factors:

Sex:

Male sex is another best-documented & most consistent risk factor for coronary risk factors. The females have a decreased incidence of myocardial infarction because ofa protective function exerted by estrogens. Paradoxically large doses of estrogenic hormones appear to increase cardiovascular mortality in women who had myocardial infarct & among men under treatment for prostate cancer. Hence the actual differences for sex is not understood. [33]

In the early days, it has been noted that for CAD, aging in women has different consequences from than in men. The Framingham heart disease study established seven times more mortality in the third decade of life in men as compared to women. The reason for delayed onset of CHD in women compared to men is still unclear. Though ovarian function may be partly responsible for the above observations, there is definite inter linking with other risk factors i.e. smoking, HTN, DM, obesity oral contraceptives, dyslipidemia. [34]

Oral contraceptives:

In a study by Lewis et al [35] in 1997 shows that use of OC pills increases the risk of myocardial infarction in young females. This is related to estrogen content of the drug. HDL tends to increase with increasing estrogen dose & decreases with increasing progesterone dose.

Homocysteinemia:

There is an independent association between total plasma homocysteine level &atherosclerotic risk & there is also an increased risk of myocardial infarction. [36]

Metabolic Syndrome:

A growing body of evidence suggests that the metabolic syndrome & hostility are independent risk factors for development of CHD. In one study by Todaro et al, the combined effect of metabolic syndrome &hostility incidence on MI was made. It appeared that hostility may provide additional prognostic information to the assessment of CHD risk in patients with metabolic syndrome &should be evaluated routinely as risk factor assessment. [37]

Acute myocardial infarction with ST – T changes in snakebite are very rare but can occur with cobra or viper bite.

Tanajura et al[38] 1990 shows that most frequent risk factor observed were cigarette smoking in 88%, hypertension in 22%, hypercholesterolemia in 16% & diabetes4% & 9% had no risk factor.

In another study by Weinberger[39] the most common risk factor among youngerage group was smoking 66% followed by hyperlipidemia, HTN & DM & 23% had no risk factor.

Non- Atherosclerotic cause of coronary artery disease:

Although atherosclerotic disease is the most common cause of luminal narrowing & coronary heart disease, there are multiple non-atherosclerotic causes of severe luminal narrowing. 4 to 7% of all the patients with MI & nearly four times this percentage for patients under age 35 do not have atherosclerotic coronary artery disease. [40]

Causes:

1. Congenital Anomalies:

   Anomolous origin from the aorta, Single coronary artery, Atresia of coronary ostium, Ostial ridges, Myocardial bridges, High-takeoff coronary ostium, Fistulla, Anomolous origin from the pulmonary trunk.

2. Embolus:

   Natural, Tumour, Calcium, Vegetation, Thrombus, Iatrogenic, Cardiac surgery & Catheterization, Coronary angioplasty, Prosthetic valves

3. Dissection:

   Coronary artery, Aortic

4. Trauma:

   Non penetrating, Penetrating, Surgery

5. Arteritis:

   Takayasu's disease, Polyarteritis nodosa, SLE, Kawasaki's Syndrome, Syphilis

6. Metabolic disorders:

   Mucopolysaccharidoses, Homocystinuria, Fabry's disease, Amyloid

7. Intimal proliferation:

   Irradiation therapy, Ostial cannulation, Fibromuscular hyperplasia, External, compression:, Aortic aneurysm, Tumour metastasis

8. Thrombosis without underlying artherosclerotic plaque:

   Polycythemia, Thrombocytosis, Hypercoagulability

9. Substance abuse:

   Cocaine, Amphetamines

10. Myocardial Oxygen demand-supply disproportion:

    Aortic stenosis, Systemic hypertension

11. Intramural coronary disease.

12. Hypertrophic

    Cardiomyopathy:, Amyloid, Cardiac transplantation, Neuromuscular,

    Diabetes mellitus

13. Normal coronary arteries. [41]

PATHOLOGY

Almost all myocardial infarctions result from coronary atherosclerosis generally with superimposed coronary thrombosis. [42]

Important advances have occurred in our understanding of the pathophysiology of acute myocardial infarction leading to a reorganization of clinical presentations into what is now referred to as the acute coronary syndrome the spectrum of which includes unstable angina, NSTEMI & STEMI.

Role of acute plaque change:

Slowly occurring high-grade stenosis of epicardial coronaries may progress to complete occlusion but does not usually precipitate acute myocardial infarction probably because of the development of a rich collateral network over time. However during the natural evolution of atherosclerotic plaques especially those that are lipid laden, an abrupt & catastrophic transition may occur characterized by plaque rupture & exposure of substances that promote platelet activation &thrombin generation.[43,44,45,46]

The resultant thrombus interrupts blood flow & leads to an imbalance between O2 supply and demand and if this imbalance is severe & persistent, may lead to myocardial necrosis.

Atherosclerosis is a chronic disease potentially involving the whole arterial system that causes a spectrum of clinical manifestations like acute MI to stable angina or stroke. The continuous accumulation of lipids & fibrous & inflammatory elements in the arterial wall of the coronary tree leads to progressive lumen narrowing with subsequent ischemia. Acute coronary syndromes (unstable angina & acute MI) have a

more complex& dynamic pathogenesis of which coronary plaque rupture & thrombosis represent only the final common pathway.[47]

In one study by Hayashi T et al.[48] it was showed that patients with eroded plaque lesions have necrosis smaller infarctions than those with ruptured plaque lesions, suggesting that an eroded plaque less potently thrombogenic than ruptured plaque.

Myocardial infarction generally occurs when there is an abrupt decrease in coronary blood flow following a thrombotic occlusion of a coronary artery previously narrowed by atherosclerosis.

The fundamental pathological alteration under lying left ventricular dysfunction in acute myocardial infarction is loss of functional myocardium. Depression of cardiac function in myocardial infarction is directly related to the extent of left ventricular damage. [49]

Left ventricular function:

1. Systolic function: Whenever interruption of antegrade flow in an epicardial coronary artery occurs, the zone of the myocardium supplied by that vessel immediately loses its ability to shorten & perform contractile work.

Four abnormal contraction patterns develop in sequence

i) Dys-synchony i.e., dissociation in the time course of contraction of adjacent segments.
ii) Hypokinesia i.e., reduction in the extent of shortening.
iii) Akinesis i.e., cessation of shortening.
iv) Dyskinesis i.e., paradoxical expansion or systolic bulging. [50,51]

Diastolic function:

Left ventricular diastolic properties are altered in infracted & ischemia myocardium leading initially to an increase but later to a reduction in left ventricular compliance. Over a period of several weeks end diastolic volume increases & diastolic pressure begins to fall towards normal. As with impairment of systolic function the magnitude of the diastolic abnormality appears to be related to the size of the infarct.

In a study conducted from the divisions of cardiology & metabolism Washington University school of Medicine, by Dr. Ronald P Karlsberg, Philip E Cryer, Robert Roberts, St. Louis M.O. Catecholamines have been shown to enhance myocardial oxygen consumption, increase the propensity to serious ventricular arrhythmias, enhance intravascular coagulation & increase the extent of myocardial damage during evolving acute myocardial infarction. The presence of elevated plasma catecholamines & the reported beneficial effects of beta adrenergic blockade in patients with acute myocardial infarction support the concept that stimulated sympathoadrenal activity may be deleterious in patients with acute myocardial infarction. The observation in their study showed that patient with higher plasma catecholamine levels within the first few hours of acute MI subsequently appeared to have greater myocardial damage & higher mortality rate, supports the concept that catecholamines may in themselves exert a deleterious effect.

However, the possibility that the level of catecholamines simply reflects the extent of myocardial damage rather than being an independent risk factor itself cannot be excluded. Nevertheless sympathetic activation begins early in the course of acute myocardial infarction at a time when myocardial necrosis is only beginning to evolve

& this provides a potential means where by beta-adrenergic blockade may provide rational therapy to curtail or minimize the amount of myocardial damage.[52]

CLINICAL FEATURES

Allison et al.[53] (1995), shows that in one half of patients with acute myocardial infarction, a precipitating factor or prodromal symptoms can be identified. Although adequate control studies have not been carried out, evidence suggests that unusually heavy exercise (particularly emotionally or stressed patients) may play a role in precipitating acute myocardial infarction.

Such infarction could be a result of marked increase in myocardial oxygen consumption in the presence of severe coronary arterial narrowing. Patients with known coronary disease who have been hospitalized for treatment of an acute coronary syndrome related event & who subsequently report a high level of stress in their life have an increased risk of re-hospitalization for cardiovascular reasons & also for hard events such as death & myocardial infarction. [54]

The prodrome is usually characterized by chest discomfort resembling classic angina pectoris, but it occurs at rest or with less activity than usual & can therefore be classified as unstable angina. Among patients who are hospitalized for unstable angina fewer than 10% develop acute myocardial infarction.

Of the patients with acute myocardial infarction presenting with prodromal symptoms of unstable angina, approximately one third have had symptoms from 1-4 wks before hospitalization, in the remaining two thirds symptoms predated admission by one week or less with one third of these patients (20% of all with prodromes) having had symptoms for 24 hours or less. [55]

Muller et al. [56] revealed a pronounced circadian periodicity for the time of onset of acute myocardial infarction with peak incidence of events between 6 am & 12 noon.

Circadian rhythms affect many physiological & biochemical parameters, the early morning hours are associated with rise in plasma catecholamine, cortisol & increase in platelet aggregability.

Interestingly the characteristic circadian peak was absent in patients receiving beta-blocker or aspirin therapy before their presentation with acute myocardial infarction.[57,58]

SYMPTOMS

Chest pain:

Pain is the most common presenting symptom in patients with myocardial infarction. Pain of acute myocardial infarction is severe & in some instances intolerable. The pain is prolonged usually lasting for more than 30 minutes & frequently for a number of hours. The pain is usually restro sternal in location spreading frequently to both sides of the anterior chest with predilection for the left side. Often the pain radiates down the ulnar aspect of the left arm producing a tingling sensation in the left wrist, hand & fingers. Pain of acute MI are thought to arise from nerve endings in ischemia or injured, but not necrotic myocardium. [59]

Nausea & vomiting occur in more than 90% of patients with transmural myocardial infarction with severe chest pain. These symptoms occur more commonly in patients with anterior myocardial infarction than in those with inferior myocardial infarction.[60]

Other symptoms: Other symptoms include feeling of profound weakness, dizziness, pain abdomen, palpitations, cold perspiration, sudden onset of breathlessness & sense of impending doom. On occasion, symptoms arising from an episode of cerebral embolism or other systemic arterial embolism are the first signs of STEMI.[61]

Silent infarction occurs more commonly in patients without antecedent angina pectoris & in patients with diabetes & hypertension.

Atypical presentation of STEMI include:

1) Heart failure i.e., dyspnoea without pain
2) Classic angina pectoris without a particularly severe or prolonged episode.
3) Atypical location of the pain.

4) CNS manifestations, resembling those of stroke, secondary to a sharp reduction in cardiac output in a patient with cerebral arteriosclerosis.
5) Apprehension & nervousness.
6) Sudden mania or psychosis.
7) Syncope.
8) Overwhelming weakness;
9) Acute indigestion and,
10) Peripheral embolisation

DIAGNOSIS

Detection of a rise and/or fall of cardiac biomarker values [preferably cardiac troponin (cTn)] or troponin I with at least one value above the 99th percentile upper reference limit (URL) and with at least one of the following:

Symptoms of ischaemia.

New or presumed new significant ST-segment–T wave (ST–T) changes or new left bundle branch block (LBBB).

Development of pathological Q waves in the ECG.

Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.

Identification of an intracoronary thrombus by angiography or autopsy[62]

Electrocardiogram in Myocardial Infarction:

Diagnosis of Myocardial infarction:

One of the most valuable contributions of the ECG is in the diagnosis of myocardial infarction. Usually it is the first laboratory test performed. The technique is reliable, reproducible, can be applied serially and when properly interpreted is the corner stone of the laboratory diagnosis of myocardial infarction. [63]

The initial ECG is diagnostic of acute infarction in slightly more than half of the patients. Serial tracings increase the sensitivity to near 95%. A single ECG may never be diagnostic.

However a pattern of ST segment displacement, especially with associated Q waves and T waves changes, and a clinical history of ischemia heart disease which is highly suggestive of acute MI. [64]

Classical Pattern and Evolution of Infarction:

The sequence of ECG evolution of myocardial infarction in man is in many respects, similar to that recorded in the experimental animal. If the ECG is inscribed at the onset of myocardial infarction, the characteristic early change, namely an abnormal T wave is often recorded. The T-wave may be prolonged, increased in magnitude and either upright or inverted. This is followed by ST segment elevation in leads facing the area of injury with reciprocal depression in the remote opposite leads. The upright T wave may exhibit terminal inversion at a time when ST segment is still elevated. A Q wave may be present in the first ECG or may not appear for hours or sometimes days. The amplitude of the QRS complex may be diminished and may be replaced by a QS pattern. As the ST segment returns to the baseline, symmetrically inverted T

wave evolve. The time of appearance and the magnitude of the changes vary amongst patients.

"Evolution of the characteristic ST segment and T wave changes coupled with appearance of Q wave is highly specific for acute myocardial infarction".

An abnormal QRS complex, ST segment and T wave may normalize transiently in the course of evolution of acute myocardial infarction and this may be due to reversible ischemia or injury or conduction defects. [65]

Correlation with evolutionary changes on ECG:

Autopsy data have shown that the ECG lacks sufficient sensitivity and specificity to permit reliable distinction of transmural from subendocardial infarcts because patients with transmural infarcts may not develop Q waves and Q waves may be seen in patients with autopsy evidence of a subendocardial (nontransmural) acute myocardial infarction.[66]

These remarks not with standing, a crude categorization of patients into Q wave and non – Q wave patterns based on the ECG is useful because Q wave acute myocardial infarctions are usually associated with greater ventricular damage, a greater tendency to infarct expansion, remodeling and a higher mortality rate.[67]

Myocardial infarction results in myocardial necrosis, injury and ischemia, each of which is reflected by different and distinct electrocardiographic manifestation.

1) Myocardial necrosis is represented by an QS complex and/or Q wave.
2) Myocardial injury is represented by elevated and coved and convex upward ST segment.

3) Myocardial ischemia is reflected by inverted, symmetrical and pointed T wave, which is increased in magnitude.[68]

Myocardial infarction on ECG is shown as a presence of abnormal Q wave.

Abnormal Q wave is noted:

a) When Q wave is 0.04 sec or more in width.

b) Depth of the Q wave is more than 25% of the height of succeeding "R" wave. [69]

Infarction implies necrosis and an electrically inert myocardium. The diagnostic feature of infarction is the "Q" wave. The theory of proximity – The "Window" theory suggests that the electrically inert myocardium allows an electrode to record the intracavitary negativity. There is an ample evidence to suggest however that a Q wave can be recorded in the absence of a transmural infraction. Heterogenicity of electrophysiological changes associated with the dynamic events of ischemia and subsequent healing with intermingling of fibrous and viable tissue has been suggested as an explanation.

According to the vectorial concepts, the electrically inert myocardium fails to contribute to the normal electrical forces and the result is a vector that point away from the area of infarction, reflected by Q wave. Theoretically, the infarction vector represents the force that alters the normal vector. It is equal to but opposite in direction form the vector generated by the infarcted myocardium prior to infarction. If the set vector is directed normally but is reduced in magnitude, a Q wave will not be recorded, but the amplitude of the QRS complex is reduced, indicating loss of myocardium. However the specificity of such a change for infarction is low. [65]

ECG and the site of infarction:

In 12 lead ECG, the site of myocardial infarction can be localized depending upon the leads showing typical changes of infarction.

- Massive anterior infarction is shown by the typical changes of infarction in chest leads from V1 – V6.
- Anteroseptal infarction is shown by changes in V1 to V3 while apical infarction is shown in V5 – V6.
- Inferior myocardial infarction is evident by Q wave in standard leads II, III and avF, which persist after deep inspiration.
- Anterolateral infarction is present when typical changes are present in standard leads I and avL as well as in chest leads V5 and V6.

There is no conventional electrode, which is oriented directly to the posterior wall of the heart (except for esophageal leads) to record the typical changes of infarction in 12 leads ECG. So the diagnosis of posterior wall MI is made by mirror image changes of this classic presentation in the right precordial (Chest) leads V1 to V3 especially V2 as:

i. Mirror image of Q wave is reflected by a tall slightly widened R – wave.
ii. The mirror image of coved and elevated ST segment is reflected by the depressed and concave upwards ST segment.
iii. The mirror image of inverted T wave is reflected by an upright, widened and tall T wave.

The ECG recognition of right ventricular infarction is usually based on manifestations of elevated ST segment in extreme right oriented leads i.e., V1 and V4R. Right

ventricular infarction almost always accompanies inferior wall infarction. Right ventricular infarction should be suspected in clinical setting of inferior wall infarction.

With ST segment elevation of 1mm or more in V1, V4R or any one of the extra right precordial leads V4R to V6R. [69]

The diagnosis of RVMI clinically relies upon clinical and haemodynamic evidence of predominant right ventricular dysfunction during acute MI.

QS complexes or QR complexes associated with ST segment elevation in leadsV4R or V4R to V3R are specific (100%)and sensitive (75%) indicators of acute diapharagmatic infarction.

Erhardt et al[70]found a significant correlation between ST segment elevation in CR4R or V4R and major anatomic involvement of right ventricle during inferior myocardial infarction in patients.

Croft et al.[71] stressed that ST segment elevation in any one of a combination of leads V4R to V6R is more sensitive than ST elevation in any single right precordial lead in identifying RVI.

ECG and the site of Obstruction:

ECG has a definite useful role in predicting the site of obstruction. In a study, it was shown that the sensitivity, specificity and predictive value for – (1) ECG indicative of anterior wall infarction and obstruction of left anterior descending artery was,95 and 96 percent respectively. (2) ECG indicative of inferior wall infraction and obstruction of right coronary artery was, 97 and 80% respectively. (3) ECG indicative of posterior or lateral wall infarction and obstruction of left circumflex coronary artery was, 98and 75% respectively. (4) ECG indicative of inferior wall infarction and obstruction of

right or left circumflex coronary artery was ,98 and 94% respectively, and (5) ECG indicative of posterior or lateral wall infarction and obstruction of right or left circumflex coronary was , 98 and 94% respectively.[72]

Role of Echocardiography in MI:

Cardiovascular imaging has significantly enhanced the practice of cardiology over the past few decades. Two- dimensional echocardiography is able to visualize the heart directly in real time using ultrasound providing instantaneous assessment of the myocardium, values, pericardium and the great vessels.

Basic Principles: Two-dimensional echocardiography uses the principle of ultrasound reflection. For a cardiac structure to produce images of heart, the imaging is performed from multiple acoustic windows with different transducer rotations, so the entire heart and great vessels can be displayed in real time and in various 2 dimensional planes. Maximum information from a study is obtained from a visual analysis of 2D images.

Echocardiography is an excellent technique for detecting the early changes in function, which occur with acute myocardial infarction. [73]

One of the principle ways of detecting ischemic muscle is that its motion is altered almost immediately after ischemia [74]

Another specific finding for ischemia muscle is alteration in systolic thickening. Normal myocardial muscle increases in thickness with systolic contraction. With acute ischemia or infraction one may actually note systolic thinning whereby the thickness of LV wall becomes greater in diastole than in systole.

Thus the affected wall segment exhibits not only dyskinetic motion, but also systolic thinning, which is probably more specific for ischemia.

Two-dimensional echocardiography is now the principle ultrasonic technique for detecting regional wall motion abnormalities in patients with CAD.

The long axis, two chambers and four chambers views are essentially for longitudinal acute myocardial infarction-nations from base to apex. The short axis view is perpendicular to the longitudinal axis. The three longitudinal views divide the short axis into six segments. Using both axis and longitudinal views, one has the opportunity to visualize segments from more than one perspective.

Using correlative studies with coronary angiography and exercise echocardiography in patients with acute myocardial infarction, it has been possible to develop a scheme where by certain areas of the 2D echocardiogram can provide a reasonable prediction as to the arteries perfusing certain areas of the heart.[75]

In patients with chest pain compatible with acute myocardial infarction but with non-diagnostic ECG, the finding on echocardiography of a distinct region of disordered contraction can be helpful diagnostically. [76]

Early use of echocardiography can aid in the early detection of potentially viable but stunned myocardium (contractile reserve), residual provocable ischemia, patients at risk for the development of congestive heart failure following acute myocardial infarction, and mechanical complications of acute MI.[77]

Old infarction leads to fibrosis and thinning. Fibrous tissue is more echogenic as compared to the normal myocardium. So the echogenic features of an old infarction are, thinning of myocardium and an increased echogenicity of the involved area.[78]

Comparison of ECG and 2D-Echo in acute MI:

According to Mitamura et al.[79] 2D-echocardiography provides a reliable method for detecting infarct lesions and anatomic location of myocardial infarction reflecting a specific coronary artery disease as compared to the ECG.

The results of coronary angiography have shown that 2D-echocardiography allows us to identify the infarct-related coronary artery in large number of patients (90%) compared to ECG (80%).

According to Scharti et al.[80] the reliability of ECG and 2D-echocardiography in the diagnosis of acute myocardial infarction was found to be similar; sensitivity 89% 2D-echo 96% for ECG; Specificity 89% VS 73%; positive predictive value 95% VS 89%; negative predictive value 79% VS 90%. This means that in most cases the ECG is a sufficient and reliable method in detecting or excluding acute myocardial infraction. Because of the equipment needed the limited practicability and because of specific problems (distinction between new and old infarction), the 2D-echocardiography should be regarded as a supporting method to the ECG, but not as an essential one in the diagnosis of acute myocardial infarction.

According to Engelen et al.[80] in anterior acute MI, the ECG is useful to predict the LAD occlusion site in relation to its major side branches as compared to echocardiography.

According to Izumi et al. [81]electrocardiography has limitations in diagnosing inferoposterior myocardial infarction especially during the acute phase, but 2D-echocardiography is an additional useful diagnostic procedure.

Mahajan Devinder Singh [82] concluded that localization of the site of myocardial infarction on ECG correlated broadly with that seen on Echo.

Echo was able to elaborate regional wall motion abnormalities in detail i.e., Echo could detect abnormalities in those areas, which could not be shown by ECG.

COMPLICATIONS

In patients with acute STEMI whose ventricular contractile performance is not normal, it is important to assist the degree of hemodynamic compromise in order to initiate therapy with drugs such as vasodilators and diuretics. Major advances in the management of STEMI have resulted from hemodynamic monitoring that has become widespread in coronary care units.

1) Left Ventricular Failure:

Left ventricular dysfunction remains the single most important predictor of mortality following STEMI. In patients with STEMI, heart failure is characterized by systolic dysfunction alone or by both systolic and diastolic dysfunction. Left ventricular diastolic dysfunction leads to pulmonary venous hypertension and pulmonary congestion, whereas systolic dysfunction is principally responsible for a depression of cardiac output and of the ejection fraction. Clinical manifestations of left ventricular failure become more common as the extent of the injury to the LV increases. Mortality increases with the severity of the hemodynamic deficit.

2) Cardiogenic Shock:

Cardiogenic shock is the most severe clinical expression of left ventricular failure and is associated with extensive damage to the left ventricular myocardium in more then

80% of STEMI patients in whom it occurs; the remainder have a mechanical defect such as ventricular septal or papillary muscle rupture or predominant right ventricular infarction.

3) Arrhythmias:

The incidence of arrhythmias is higher in those patients seen earlier after the onset of symptoms. Many serious arrhythmias develop before hospitalization even before the patient is monitored. When patients are seen very early during the course of STEMI, they almost invariably exhibit evidence of increased activity of the autonomic nervous system.

Thus sinus bradycardia, sometimes associated with AV block, and hypotension reflect augmented vagal activity.

i. Ventricular premature beats
ii. Ventricular tachycardia
iii. Ventricular fibrillation
iv. Sinus bradycardia
v. Accelerated idioventricular rythm

4) Atrioventricular and Intraventricular Block:

Ischemic injury can produce conduction block at any level of the AV or intraventricular conduction system. Such blocks may occur in the AV node and the bundle of His, producing various grades of AV block in either main bundle branch producing right or left bundle branch block; and in the anterior or left posterior divisions of the left bundle producing left anterior or left posterior (fascicular) divisional blocks.

a) First degree AV Block

b) Second degree AV Block

i)Mobitz Type I or Wenckebach

ii)Mobitz Type I1

c) Complete (third) degree AV Block

5) Supraventricular Tachyarrhythmias:

a) Sinus tachycardia

b) Atrial Premature contractions

c)Paroxysmal supraventricular tachycardia

d) Atrial flutter and fibrillation

6) Other complications:

a) Recurrent ischemia and infarction

b) Pericardial effusion

c) Pericarditis

d) Dressler Syndrome

e) Venous thrombosis and pulmonary embolism

f) Left ventricular Aneurysms

g) Mitral regurgitation

METHODOLOGY

SOURCE OF DATA

All patients with history of ischemic type of chest pain attending and admitted in ICCU OF SHRI. B.M. PATIL Medical College Hospital during the study period will be evaluated for the conditions.

Period of study will be from December 2016 to August 2018.

METHODOLOGY

ELIGIBILITY CRITERIA

1. All patients with history of ischemic type of chest pain

2. Evolutionary changes of serially obtained ECG

3. Rise in serum cardiac biomarkers.

4. 2D echocardiography showing wall motion abnormality.

EXCLUSION CRITERIA

1. Patient above the age of 70 years

2. Patient presenting with

    a. Previous history of Myocardial Infarction

    b. Subendocardial Myocardial Infarction, Posterior infarction

    c. Cardiomyopathy and previous cardiac surgeries.

    d. Valvular heart disease.

    e. congenital heart disease & Pericardial disease.

Statistical analysis

All characteristics were summarized descriptively. For continuous variables, the summary statistics of mean± standard deviation (SD) were used. For categorical data, the number and percentage were used in the data summaries and diagrammatic presentation. If the p-value was < 0.05, then the results were considered to be statistically significant otherwise it was considered as not statistically significant. Data were analyzed using SPSS software v.23.0. and Microsoft office 2007.

RESULTS

TABLE 1: DISTRIBUTION OF CASES ACCORDING TO AGE AND SEX

AGE(YRS)	Male		Female		Total		p value
	N	%	N	%	N	%	
30-40	11	20.37	0	0.00	11	12.79	0.047*
41-50	12	22.22	6	18.75	18	20.93	
51-60	21	38.89	14	43.75	35	40.70	
61-70	7	12.96	9	28.13	16	18.60	
>70	3	5.56	3	9.38	6	6.98	
Total	54	100.00	32	100.00	86	100.00	

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

Table 1 shows incidence of AMI in different age groups, number of males and females in each group and their respective percentage in that group. As indicated in the table, the maximum incidence of AMI was in 51-60 years of age. Percentage of females is steadily rising from 41 years of age onwards and is MORE to that of males after 41 years of age group.

There are only 20.37% cases below the age group of 40 years that too only males. Overall the number of female cases is highly significant as compared to males

FIGURE 1: DISTRIBUTION OF CASES ACCORDING TO AGE AND SEX

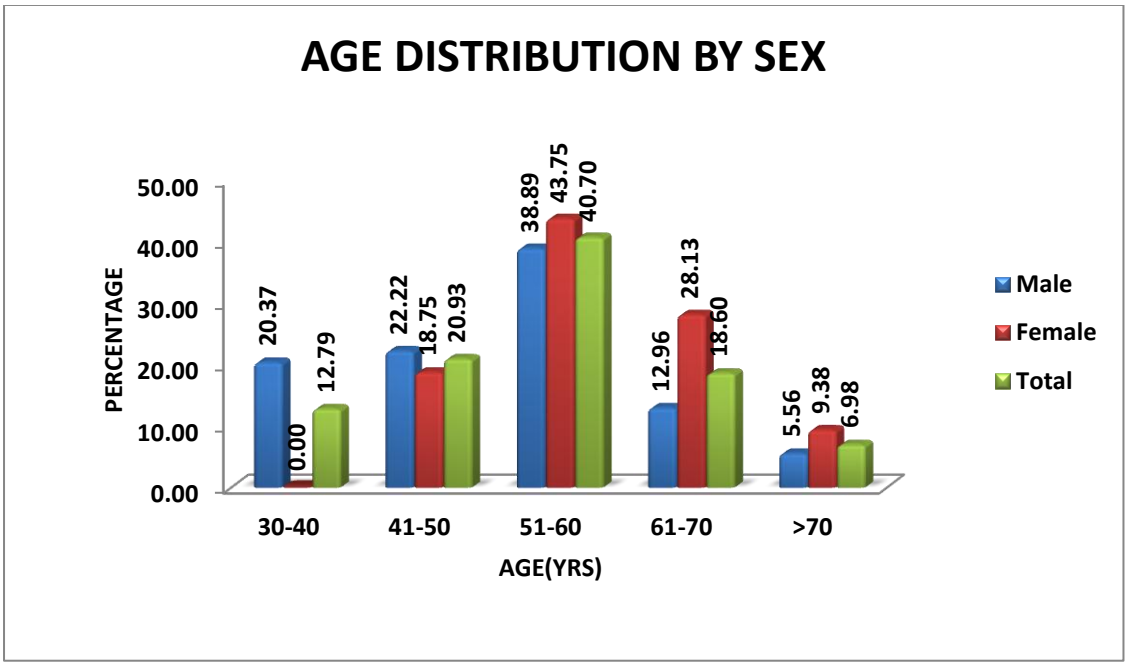


TABLE2: MEAN AGE ACCORDING TO SEX

	Male		Female		p value
	Mean	SD	Mean	SD	
AGE(YRS)	52.9	12.1	59.9	9.5	0.007*

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

Mean age for Male was 52.9(SD 12.1) and Female was higher 59.9(SD 9.5)

FIGURE2: MEAN AGE ACCORDING TO SEX

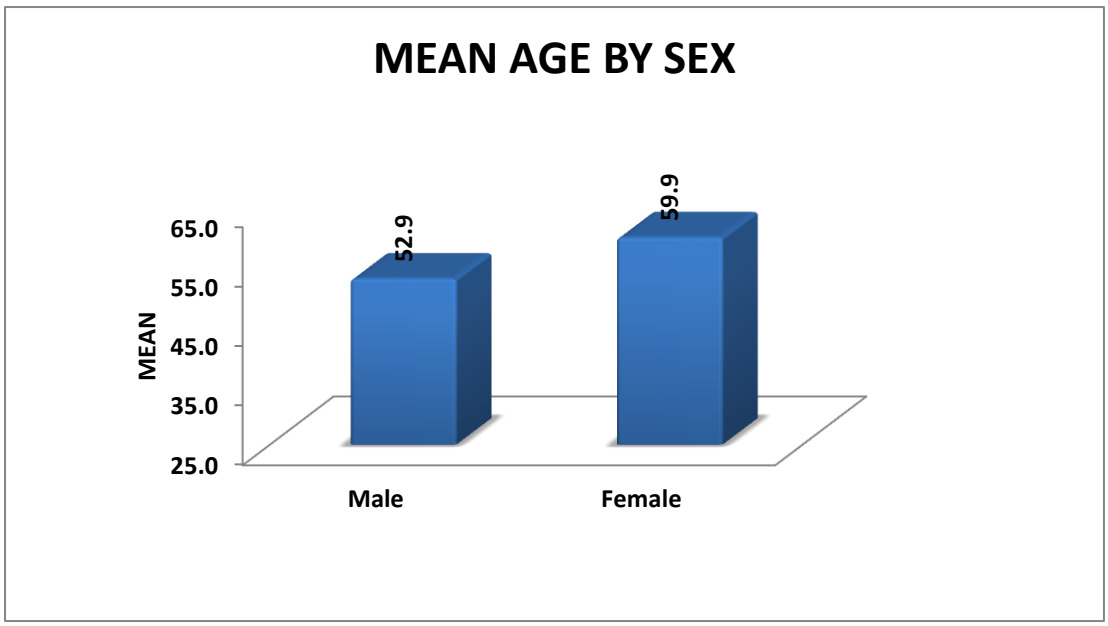


TABLE 3: DISTRIBUTION OF CASES ACCORDING TO SYMPTOMS

SYMPTOMS	N	%
CHEST PAIN	83	96.5
BREATHLESSNESS	30	34.9
SWEATING	32	37.2
VOMITING	13	15.1
PAIN ABDOMEN	10	11.6

Table 3 shows the pattern and incidence of various symptoms of AMI. Chestpain was the most common presenting symptom which was present in 96.5% of patients and another 10% had all together epigastrium pain and pain in left arm alone.

Sweating was 2nd most common symptom (37%) followed by dyspnoea (34.9%). Vomiting was present in 15% and palpitation in 11%. Another 8% of patients presented with giddiness / syncope

FIGURE 3: DISTRIBUTION OF CASES ACCORDING TO SYMPTOMS

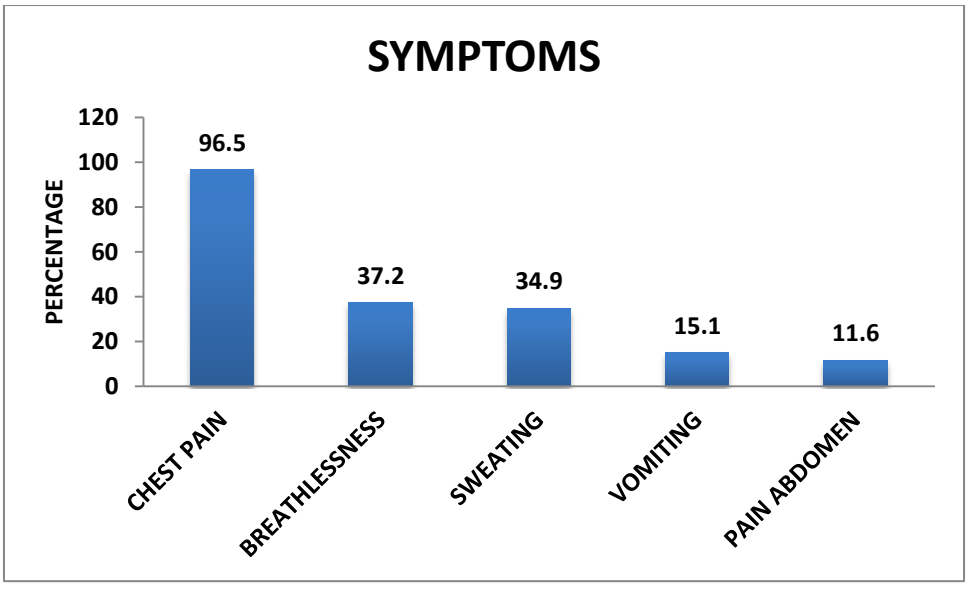


TABLE4 : DISTRIBUTION OF CASES ACCORDING TO RISK FACTORS

RISK FACTORS	N	%
HYPERTENSON	22	25.6
DIABETIS MELLITUS	15	17.4
SMOKING/TOBACCO CHEWING	38	44.2
ALCOHOL	20	23.3
OBESITY	5	5.8
FAMILY HISTORY	2	2.3

Table 4 shows major risk factors for AMI was smoking or tobacco chewing (44.2%) followed by hypertension (25.9%), alcohol intake (23.3%), diabetes mellitus (17%), obesity(5.8%) positive family history for CHD(2.3%).

FIGURE 4: DISTRIBUTION OF CASES ACCORDING TO RISK FACTORS

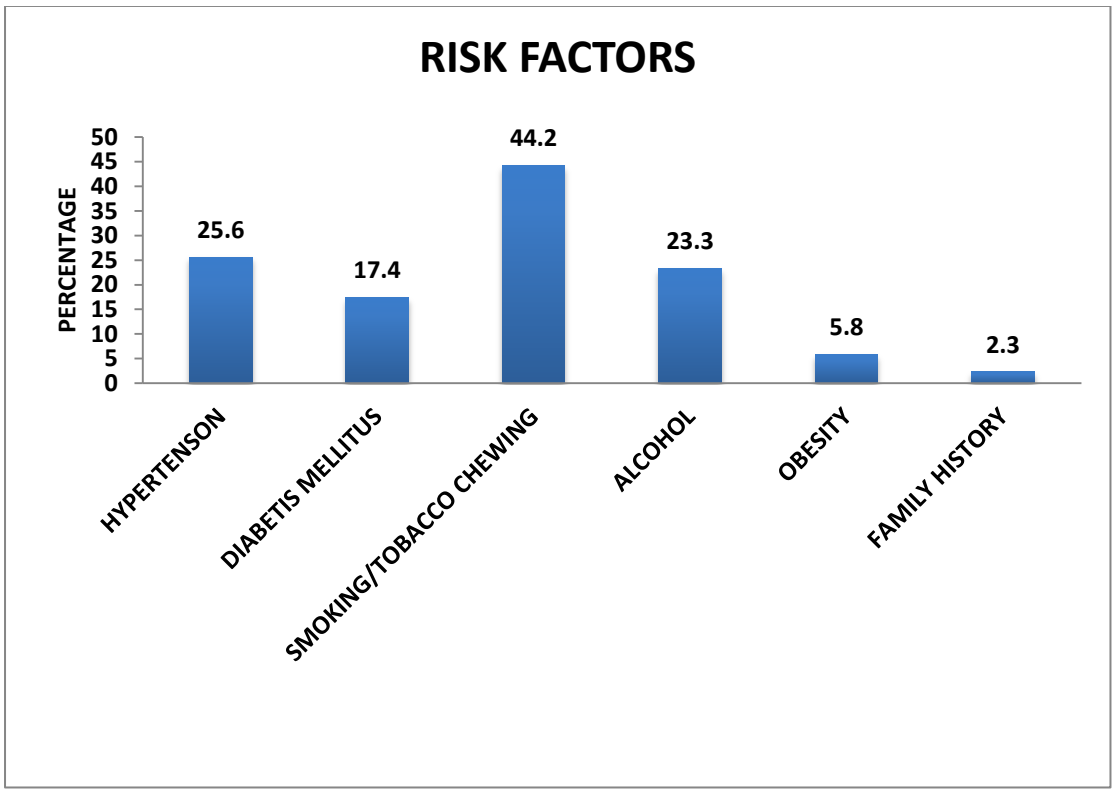


TABLE 5: DISTRIBUTION OF CASES ACCORDING TO VITAL PARAMETERS

PARAMETERS	Mean	SD
PULSE RATE/MIN	87.6	17.1
SBP	133.8	29.7
DBP	83.8	16.9
RESPIRATORY RATE (cpm)	19.7	3.4

TABLE 6: DISTRIBUTION OF CASES ACCORDING TO LIPID PROFILE

PARAMETERS	Mean	SD
TOTAL CHOLESTROL(mg/dl)	171.1	38.4
TG(mg/dl)	157.3	93.7
LDL(mg/dl)	102.8	33.9
HDL(mg/dl)	38.4	13.3

TABLE 7: DESCRIPTIVE STATISTICS OF CPK-MB, TROP-T AND TROP-I

CPK-MB(U/L)	N	%
≤27	10	11.6
>27	72	83.7
TROP-T (ng/ml)		
NEGATIVE	7	8.1
POSITIVE	44	51.2
TROP-I (ng/ml)		
≤0.12	1	1.2
>0.12	34	39.5
MEAN±SD	13.03±24.08	

Table shows details regarding raised in cardiac enzymes in AMI. CPK-MB raised in 72(83%) of subjects. Troponin-I or Troponin T(either one was done) was raised in 78(90%) of subjects.

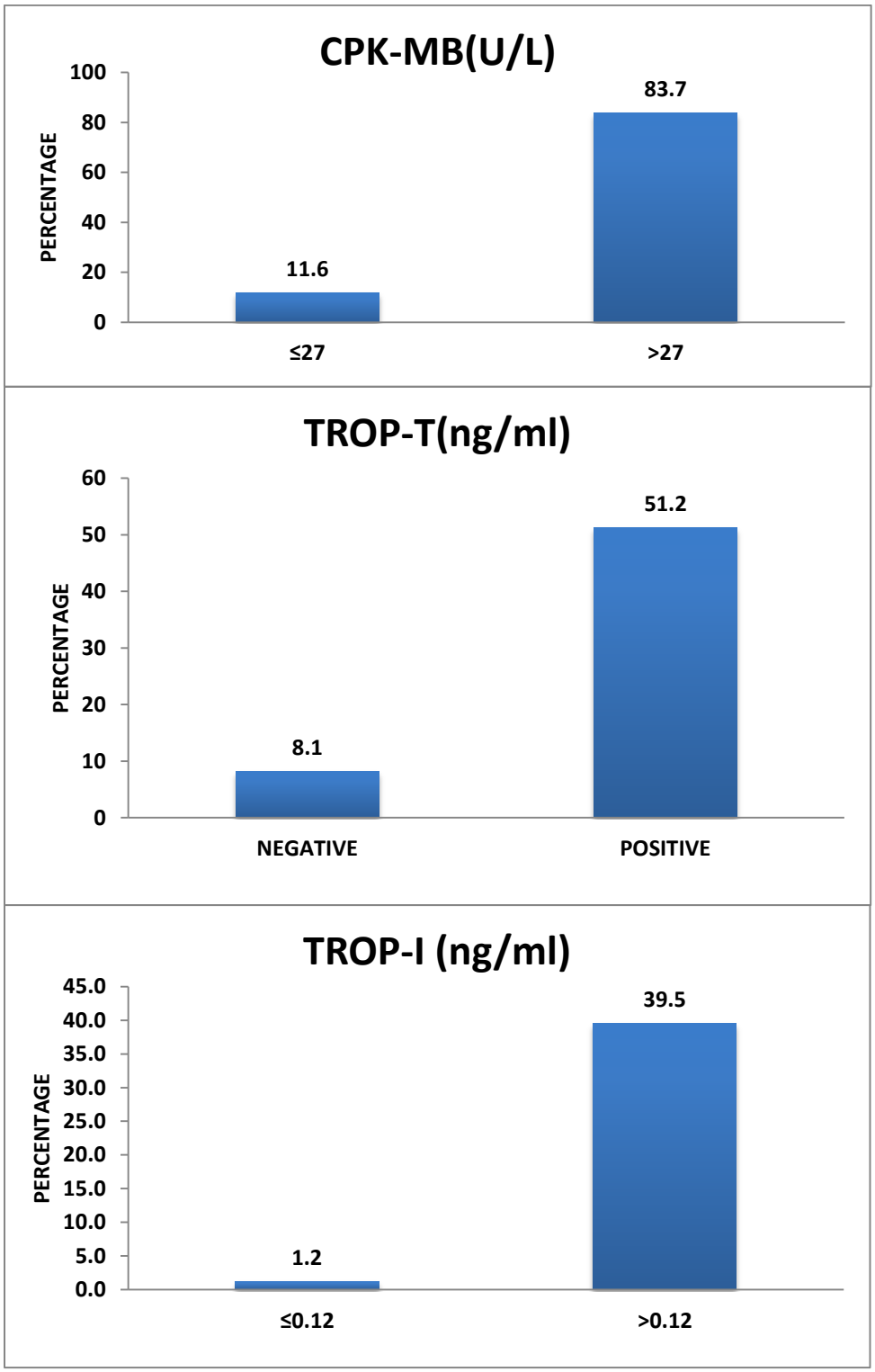


FIGURE: DESCRIPTIVE STATISTICS OF CPK-MB, TROP-T AND TROP-I

TABLE 8: DISTRIBUTION OF CASES ACCORDING TO LVEF %

LVEF %	N	%
NORMAL	25	29.1
MILD DYSFUNCTION	37	43
MODERATE DYSFUNCTION	22	25.6
SEVERE DYSFUNCTION	2	2.3
Total	86	100

FIGURE 5: DISTRIBUTION OF CASES ACCORDING TO LVEF %

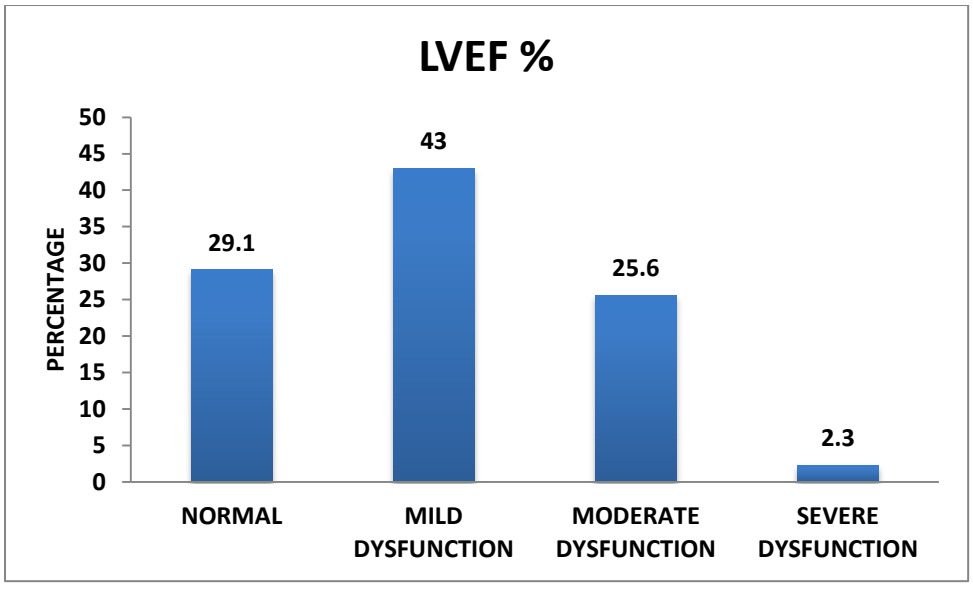


TABLE 9: DISTRIBUTION OF LVF% ACCORDING TO SITE OF INFRACTION BY ECG

	NORMAL	MILD DYSFUNCTION	MODERATE DYSFUNCTION	SEVERE DYSFUNCTION
INFERIOR WALL & INFERIOR WALL +RVMI	19	17	7	0
EXTENSIVE ANTERIOR	3	10	7	0
ANTERIOR SEPTAL WALL	2	6	7	1
ANTERIOR LATERAL WALL	2	0	1	0
LATERAL WALL	0	1	0	0

FIGURE 6: DISTRIBUTION OF LVF% ACCORDING TO SITE OF INFRACTION BY ECG

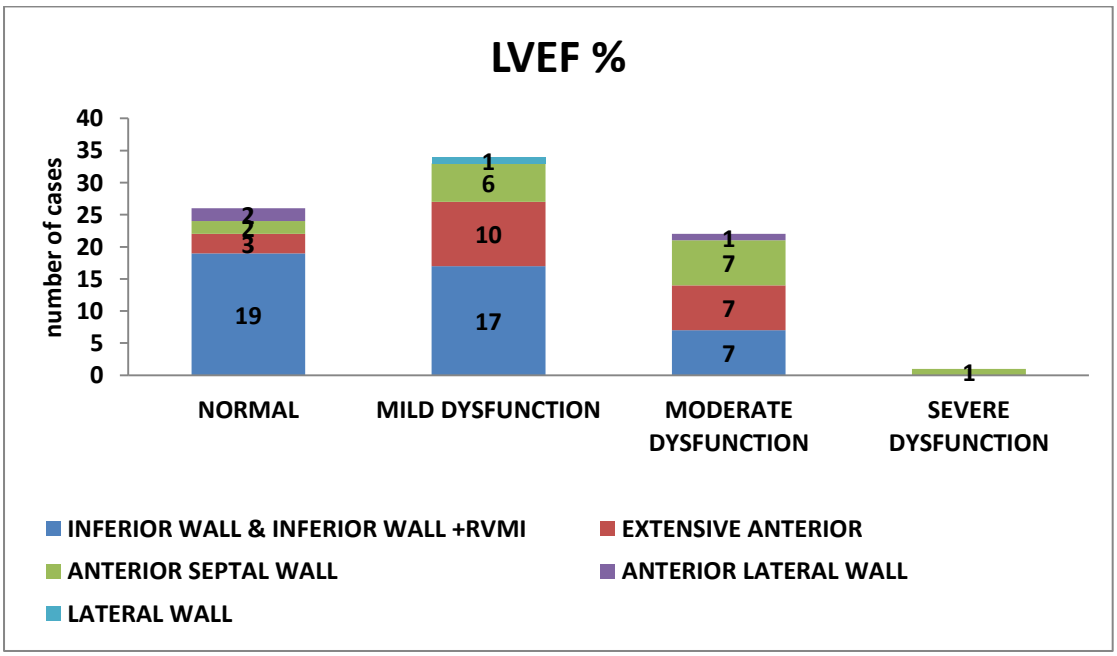


TABLE 10: DISTRIBUTION OF CASES ACCORDING TO SITE OF INFRACTION BY ECG

SITE OF INFRACTION BY ECG	N	%
INFERIOR WALL & INFERIOR WALL +RVMI	43	59.3
EXTENSIVE ANTERIOR	20	23.3
ANTERIOR SEPTAL WALL	15	17.4
ANTERIOR LATERAL WALL	8	9.3
LATERAL WALL	1	1.2

Table 9 shows the pattern and incidence of various AMI, according to the site overall all together anterior wall (i..e anterior + anterolateral + anteroseptal)almost equal which was in 51% of patients followed by inferior wall (i.e. inferior +infero-lateral + inferior and right ventricular + inferior and posterior wall) occurred in49% of patients.

FIGURE 7: DISTRIBUTION OF CASES ACCORDING TO SITE OF INFRACTION BY ECG

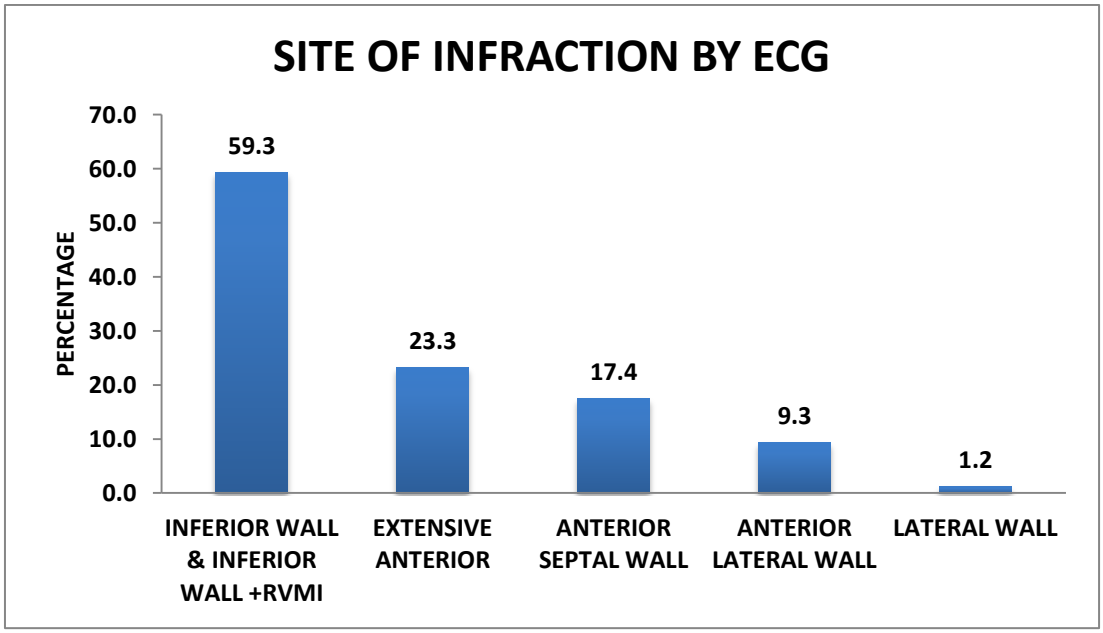


TABLE 11: DISTRIBUTION OF CASES ACCORDING TO SITE OF MI BY 2D ECHO

SITE OF MI BY 2D ECHO	N	%
ANTERIOR LATERAL WALL	9	10.5
ANTERIOR SEPTAL WALL	36	41.9
ANTERIOR WALL	31	36.0
APEX WALL	26	30.2
GLOBAL WALL	1	1.2
INFERIOR LATERAL WALL	27	31.4
INFERIOR SEPTAL WALL	15	17.4
INFERIOR WALL	45	52.3
LATERAL WALL	9	10.5
POSTERIOR WALL	6	7.0
SEPTUM WALL	1	1.2

Table 10 shows overall distribution of cases according to site of infarction based on 2D echocardiography.

FIGURE 8: DISTRIBUTION OF CASES ACCORDING TO SITE OF MI BY 2D ECHO

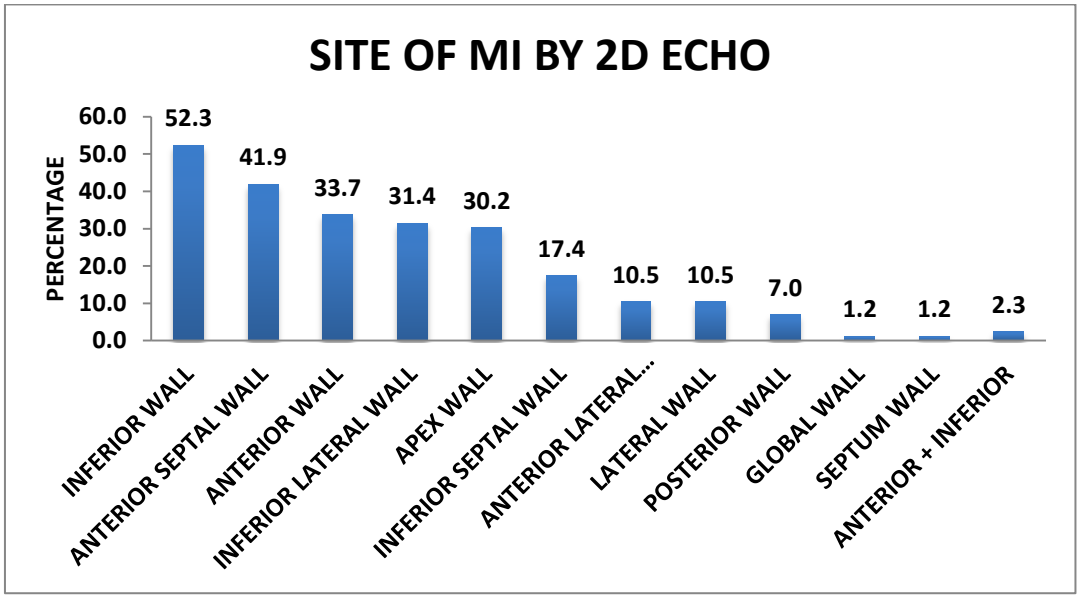


TABLE 12: SITE OF MI BY 2D ECHO AMONG 43 CASES OF INFERIOR WALL & INFERIOR WALL +RVMI

SITE OF INFRACTION BY 2D ECHO	N	%
INFERIOR WALL	35	40.7
INFERIOR LATERAL WALL	26	30.2
APEX WALL	1	1.2
INFERIOR SEPTAL WALL	15	17.4
ANTERIOR LATERAL WALL	2	2.3
LATERAL WALL	6	7.0
POSTERIOR WALL	5	5.8

Table 11 shows among 43 cases with IWMI & IWMI +RVMI the distribution according to 2D echocardiography. Predominantly inferior wall(40.7%) , followed by infero-lateral wall(30.3%), infero-septal(17.4%), lateral wall(7%), posterior wall(5.8%).

FIGURE 9: SITE OF MI BY 2D ECHO AMONG 43 CASES OF INFERIOR WALL & INFERIOR WALL +RVMI

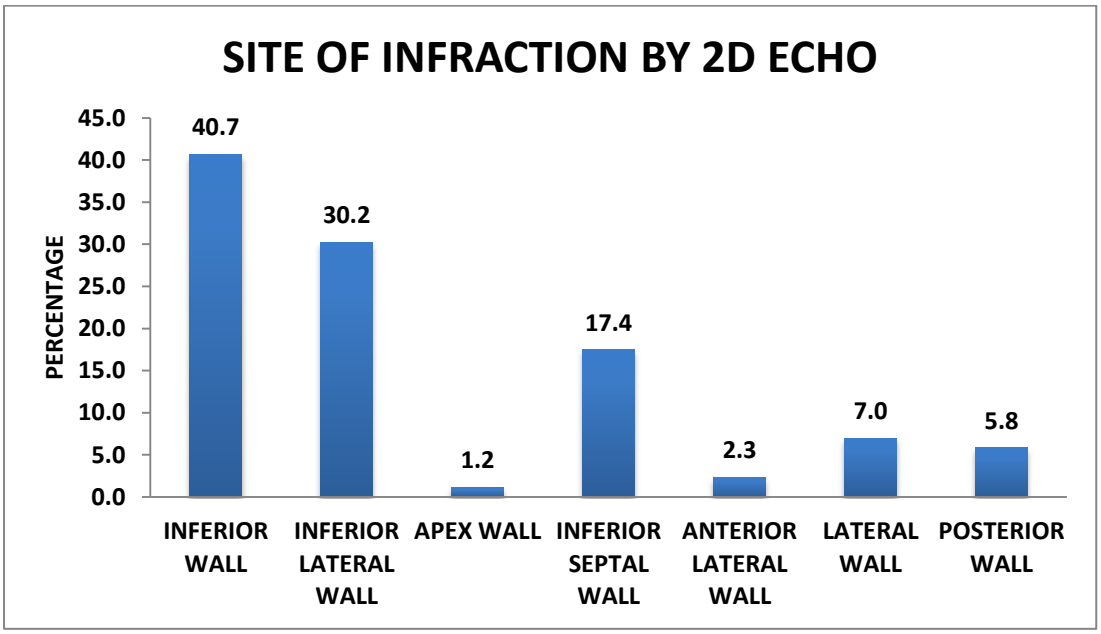


TABLE 13: SITE OF MI BY 2D ECHO AMONG 20 CASES OF EXTENSIVE ANTERIOR

SITE OF INFRACTION BY 2D ECHO	N	%
ANTERIOR SEPTAL WALL	13	15.1
ANTERIOR WALL	11	12.8
APEX WALL	11	12.8
INFERIOR WALL	2	2.3
ANTERIOR LATERAL WALL	2	2.3
INFERIOR LATERAL WALL	1	1.2
INFERIOR SEPTAL WALL	1	1.2
LATERAL WALL	1	1.2
GLOBAL WALL	1	1.2
SEPTUM WALL	1	1.2

Table 12 shows distribution of cases with extensive anterior wall involvement in ECG, with anterior septal (15%), anterior wall(12%), apex(12.8%), inferior wall(2.3%), inferior septal, inferior lateral, global, septum wall (1.2% each).

FIGURE 10: SITE OF MI BY 2D ECHO AMONG 20 CASES OF EXTENSIVE ANTERIOR

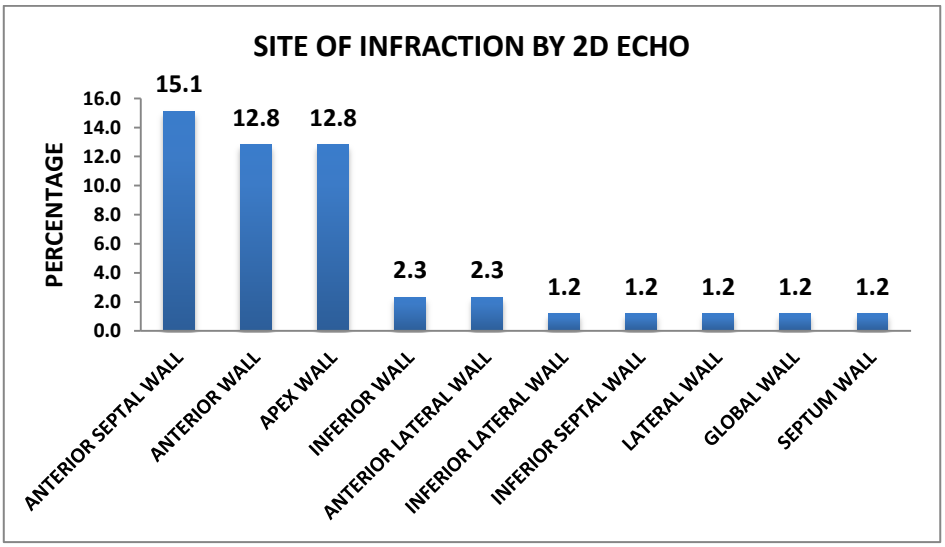


TABLE 14: SITE OF MI BY 2D ECHO AMONG 15 CASES OF ANTERIOR SEPTAL WALL

SITE OF INFRACTION BY 2D ECHO	N	%
ANTERIOR SEPTAL WALL	15	17.4
ANTERIOR WALL	12	14.0
APICAL WALL	10	11.6
ANTERIOR LATERAL WALL	3	3.5
LATERAL WALL	1	1.2

Table 13 shows 2D echo elaboration of 15 cases showing anterior septal wall, anterior septal wall(17.4%), anterior wall (14%) apical wall(11.6%) antero-lateral wall(3.5%)

FIGURE 11: SITE OF MI BY 2D ECHO AMONG 15 CASES OF ANTERIOR SEPTAL WALL

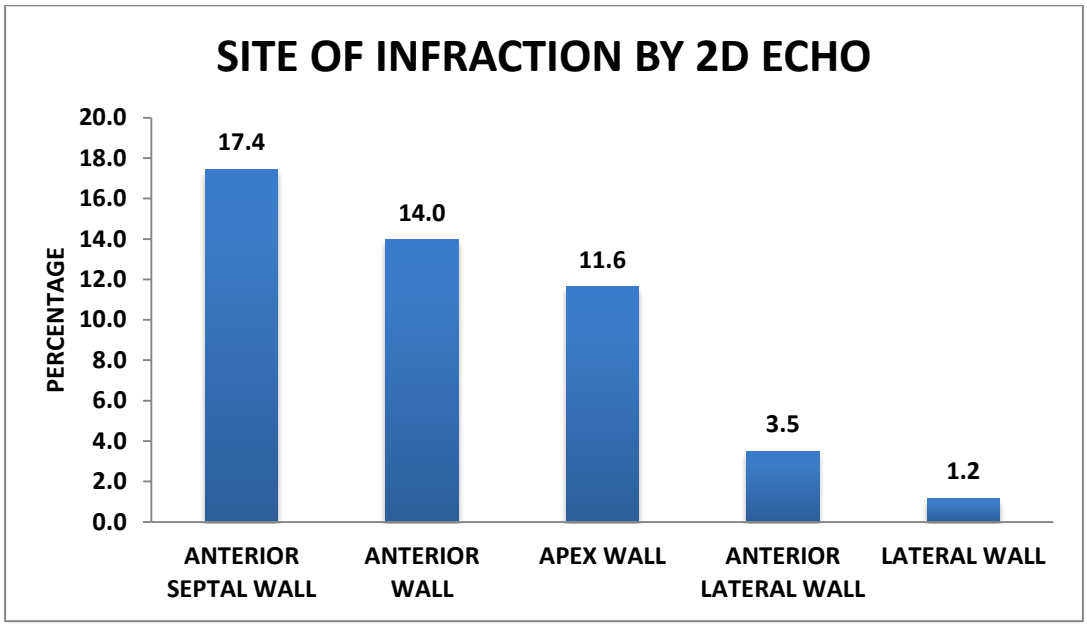


TABLE 15: HYPOTENSION ACCORDING TO SITE OF INFRACTION BY ECG

SITE OF INFRACTION BY ECG	HYPOTENSION
INFERIOR WALL & INFERIOR WALL +RVMI	19
EXTENSIVE ANTERIOR	5
ANTERIOR SEPTAL WALL	6
ANTERIOR LATERAL WALL	1
LATERAL WALL	0

Table 14 shows hypotension as complication of AMI is seen majorly in IWMI+RVMI followed by AWMI

FIGURE 12: HYPOTENSION ACCORDING TO SITE OF INFRACTION BY ECG

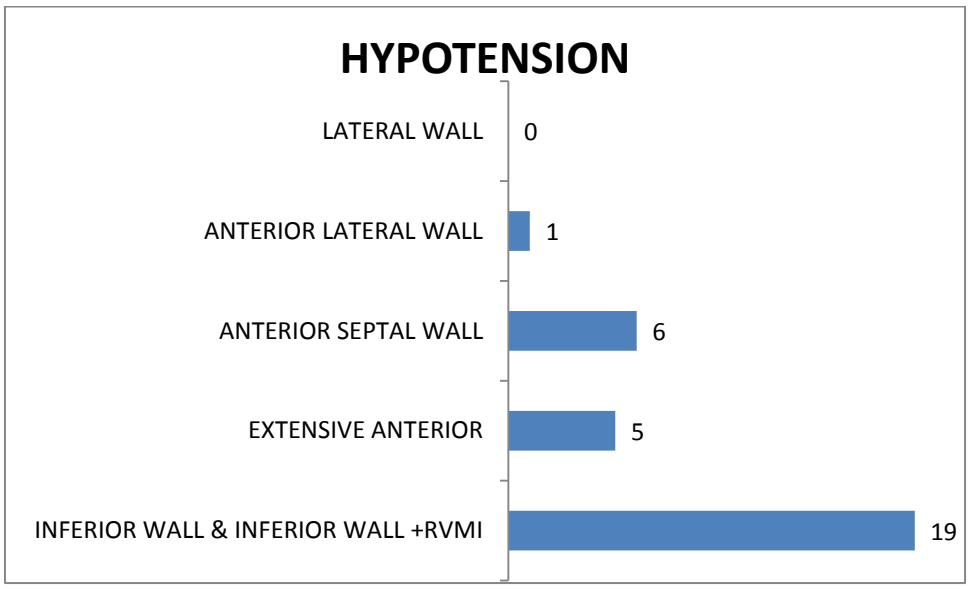


TABLE 16: DISTRIBUTION OF CASES ACCORDING TO ARRHYTHYMIAS/CONDUCTION BLOCK

ARTHYMIAS/CONDUCTION BLOCK	N	%
NO	58	67.4
YES	28	32.6
TOTAL	86	100

Total of 28 cases encountered arrhythmias or conduction block.

TABLE 17: ARTHYMIAS/CONDUCTION BLOCK ACCORDING TO SITE OF INFRACTION BY ECG

SITE OF INFRACTION BY ECG	VT	CHB	SB	ST	RBBB	LBBB	AV I°	AV II°	VPB
INFERIOR WALL & INFERIOR WALL +RVMI	3	5	5	0	0	1	2	1	0
EXTENSIVE ANTERIOR	3	0	0	1	2	0	0	0	1
ANTERIOR SEPTAL WALL	2	0	0	2	1	1	1	0	0
ANTERIOR LATERAL WALL	0	0	0	2	1	1	0	0	1
LATERAL WALL	0	0	0	0	0	0	0	0	0

Table 16 shows varies arrhythmia and conduction block with distribution among site of infarction involved most common was ventricular tachycardia 8 cases, CHB & SB 5 cases each. Sinus tachycardia 5 , RBBB 5, LBBB 3, Av 1st degree block 3, 2nd degree 1 VPB 2 cases.

FIGURE 13: ARTHYMIAS/CONDUCTION BLOCK ACCORDING TO SITE OF INFRACTION BY ECG

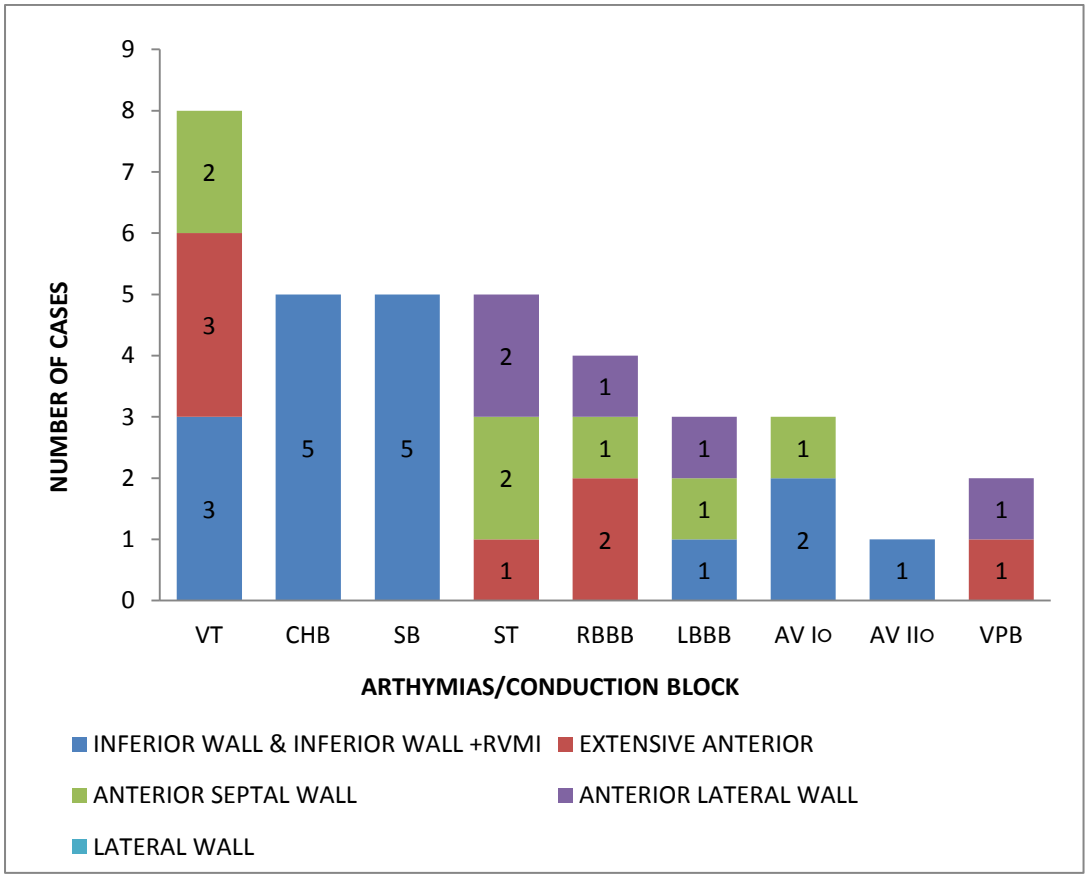
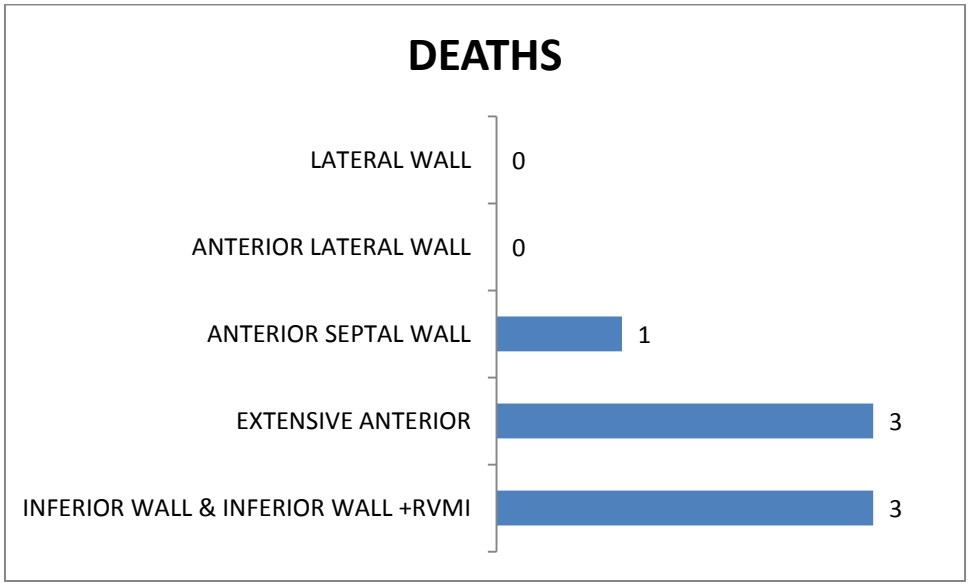


TABLE 18: DEATHS ACCORDING TO SITE OF INFRACTION BY ECG

SITE OF INFRACTION BY ECG	DEATHS
INFERIOR WALL & INFERIOR WALL +RVMI	3
EXTENSIVE ANTERIOR	3
ANTERIOR SEPTAL WALL	1
ANTERIOR LATERAL WALL	0
LATERAL WALL	0

Total of 7 cases expired during hospital stay, a total 4 cases had AWMI and 3 had IWMI.

FIGURE 14: DEATHS ACCORDING TO SITE OF INFRACTION BY ECG



DISCUSSION

Age & Sex:

IN his study 86 patients were studied in the present study. The age ranged from 30-80yrs. 54 patients were males (62%) & 32 patients were females (37%). The maximum number of cases were noted in 51 – 60 years (35 cases). Less number of cases were noted in 30 – 40 years & above 70(6 cases). The male to female ratio was 3:2mean age in male was 52.9 and in female 59.9. Study conducted by Khanna et al, the mean age was 40 yrs ranging from30 – 68 yrs. The male to female ratio was 11:2.

In another study conducted by Shah et al, the mean age was 54. 4 yrs, male to female ratio was 11.3:1. The maximum number of cases noted in this study were between 51 – 60 yrs.

In Cole & Katz series 83% of cases came within the age group of 40 – 69 yrs. They reported 25% incidence of infarction in females. They reported 25% incidence of infarction in females. The reported finding of male to female ratios was varied from 3.6:1. Our results correlate to these studies.

Symptoms:

Malliani study shown that chest pain was the most common symptom and the associated symptom was excessive sweating. In the present study, chest pain is the commonest symptom (96%) & sweating is the most common associated symptom (35%).So there is no much difference between these studies.

Risk Factors:

Among risk factors, the present study shows that smoking is the commonest risk factor (44%), followed by hypertension 25.6%, diabetes mellitus (17.4%),alcholic (23.3%) &obesity (5.8%).

In Tanajura et al[78]study, smoking was the commonest risk factor (88%) followed by hypertension (22%), hyperlipidemia 16% diabetes (4%) & no risk factors (9%). Smoking has been observed as a significant risk factor among patients of coronary artery disease in(70%) by Oscar roth (1967), in (75%) by Chandurkar (1975)

The study done by Sameer Thanavaro et al[83], found diabetes in 18 % of patients & hypertension 39%So there is no much significant difference noted when all the above studies were compared

ECG AND 2D ECHOCARDIOGGRAPHY CORRELATION.

ECG helps to localize the site of infarction. The changes of infarction are seen in lead II, III and avf in inferior wall infarction, in lead I, avl and V5-6 in anterolateral infarction, in lead V1-3 anteroseptal infarction, lead V5-6 apical infarction and lead V1-6 in extensive anterior infarction.

In patients with acute chest pain syndrome without diagnostic ECG findings of acute myocardial infarction, echocardiography may provide a rapid, sensitive and specific tool to aid in establishment of correct diagnosis. In patients with non-transmural myocardial infarction, severe hypokinesia appears to be the best discriminator of myocardial infarction.

In our data, as shown in results, 20 patients out of 86 patients had extensive anterior wall myocardial infarction on ECG. Echocardiography in these patients further

elaborated that 11 patients had extensive anterior wall infarction, 13 patients had antero-septal and apical wall myocardial infarction, 11 had anterior & apical,

2 had anterior & inferior infarction & 2 antero-lateral, 1 lateral wall, 1 global infarction, thus elaborating the extensive anterior infarction seen on echocardiography in great details.

43 patients, out of 86 patients had inferior wall myocardial infarction andinferior wall with right ventricle infarction on ECG. When echo was done in these patients, 35 patients had inferior wall myocardial infarction, 5 patients had inferior walland right ventricle infarction, 26 INFERO LATERAL& 15 infero septal 2 patients had inferior wall and anterior-lateral myocardial.

15 patients out of 86 patients had antero-septal infarction on ECG. One chocardiographic examination in these patients, 15 patients had antero-septal myocardial infarction, 10 patients had anteroseptal apical infarction, 3 patients had antero-lateral, thereby lending credence to the fact that echocardiography delineates ischemic changes more extensively.

According to Izumi et al [84](1995) electrocardiography has limitations in Diagnosing infero-posterior myocardial infarction especially during the acute phase, but 2D echocardiography is an additional useful diagnostic procedure.

Mahajan Devinder Singh[85] concluded that localization of the site of myocardial infarction on ECG correlated broadly with that seen on ECHO, ECHO was able to elaborate regional wall motion abnormalities in detail i.e., ECHO could detect abnormalities in those areas, which could not be shown by ECG.

Arrhythmias :

Sinus bradycardia (SB) :

Bradyarrhythmias[86]and hypotension are common in proximal occlusion of right coronary artery commonly leading to inferior myocardial infarction, because of reflexes arising from the ischemic right ventricle.

In the present study 5 had SB, out of which all 3 were purely in inferior and 2 in inferior wall with right ventricular extension and inferior + posterior wall MI. In all these patients, SB was transient and majority of the patients had normal sinus rhythm (NSR) by the end of 1st day. All the patients had NSR at discharge.

Similar observations were made by Swart G et al.[87]

Sinus tachycardia (ST) :

In my study, ST was observed in 5 patients and it was most commonly associated with anterior and antero-lateral wall than inferior and inferolateral wall MI. Same observation of anterior wall being commonly involved was made by Crimm A et al.[88]

AV blocks (Atreioventricular blocks) : Complete (III0) AV block in present study was 5,all of which together in inferior MI (inferior, inferolateral and inferior with right ventricular extension

Other studies done by Rathore SS et al[89] and Garcia Garcia C et al [90] also showed increased incidence of CHB in inferior wall MI, the incidence being 7.3% and 12% respectively. Though incidence is less as compared to present study but still more than any other site of AMI.

In the present study, the combined incidence of I^0 and II^0. AV block was 4. inferior wall (inferior and inferoposterior) having 75% and anterior wall (anterior and antero-septal) having 25% incidence.

In the study done by Majumder AA et al[91]the combined incidence of I0 and II0A-V block was 15%, with inferior wall showing 30.3% incidence and anterior wall showing 4.45% incidence.

Though the incidence of both studies differ, both show an affinity of AV blocks for inferior wall.

Bundle branch blocks (BBB) :

In the present study, the incidence of BBB was 7 with more affinity for anterior wall MI than inferior wall.

In present study mortality was high in RBBB associated with anterior

Ventricular arrhythmias :

In the present study, overall ventricular arrhythmias were seen in 10 patients of which VT were 8, VPB 2

The frequency of VT was seen more in anterolateral MI than anteroseptal MI which compares well with study done by Horvat D et al[92]in 2008.

SUMMARY

1) In the present study males outnumbered females (3:2).
2) Majority of cases was seen in 51 – 60 years.
3) Smoking emerged as the main risk factor in acute myocardial infarction patients.
4) both anterior wall MI & inferior wall MI were equal in incidence
5) The lesions seen on ECG correlated broadly with those seen on echocardiography. Echo was able to elaborate regional wall motion abnormalities in detail than ECG.
6) LVEF in anterior wall MI was less compared to inferior wall MI Anterior & Global MI had lower LVEF.
7) ICCU mortality was 8%.
8) Sinus tachycardia (ST) was observed in 5 patients with higher incidence in anterior and antero-lateral
9) Sinus bradycardia (SB) was observed in 5 patients predominantly seen in inferior wall MI. However, no death was observed in patients with SB and inferior wall MI.
10) Overall incidence of complete (III0) AV block in present study was 5 cases predominantly seen in inferior wall MI .mortality was higher in anterior wall.
11) Bundle Branch Blocks have high affinity for anterior wall (Anterior + anteroseptal + anterolateralits 10%. Among these mortality was higher in patients associated with RBBB.
12) Overall incidence of ventricular arrhythmas was 10 of which VT's were 8

CONCLUSION

- The location of MI seen on ECG correlated broadly with those seen on echocardiography. Echo was able to elaborate regional wall motion abnormalities in detail than ECG.
- Cardiac arrhythmias routinely manifest during or following ACS. Early recognition and management of post myocardial infarction arrhythmias can significantly modify the morbidity and mortality in myocardial infarction.
- AMI is one of the major causes for hospital admission in elderly with male preponderance between 4th to 7th decade and incidence being equal in both sexes beyond 7th decade.
- AMI is commonly seen in anterior wall with high incidence of tachyarrhythmias and higher mortality where as inferior wall MI was less common and was commonly associated with bradyarrhythmias with lesser mortality.
- Sinus Bradycardia, if transient, has protective role in AMI where as persistence of sinus tachycardia has high mortality in AMI

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ANNEXURES

ETHICAL CLEARANCE CERTIFICATE

B.L.D.E. UNIVERSITY'S

SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR-586 103

INSTITUTIONAL ETHICAL COMMITTEE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 04/10/2016 *at* 3-00pm *to scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected & revised version synopsis of the Thesis has been accorded Ethical Clearance.*

Title "To Study the correlation between ecg and 2d Echocardiography in locating acute myocardial infaction and Predicting complications"

Name of P.G. student Devendra Nair N

General Medicine

Name of Guide/Co-investigator Dr G.S. Mahishale

Associate professor

DR.TEJASWINI. VALLABHA
CHAIRMAN
INSTITUTIONAL ETHICAL COMMITTEE
BLDEU'S, SHRI.B.M.PATIL
MEDICAL COLLEGE, BIJAPUR.

Following documents were placed before E.C. for Scrutinization
1) Copy of Synopsis/Research project.
2) Copy of informed consent form
3) Any other relevant documents.

CHANGE OF GUIDE CERTIFICATE

BLDE
(DEEMED TO BE UNIVERSITY)
Declared as Deemed-to-be-University u/s 3 of UGC Act, 1956
The Constituent College
SHRI. B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPURA

BLDE(DU)/REG/PG/2017-18/010 April 04, 2018

To,
The Professor and HOD
Department of Medicine,
BLDE (DU)'s Shri B. M. Patil Medical College,
Hospital and Research Centre,
Vijayapura

Sir,

Sub: Regarding change of PG Guide.
Ref: Your letter no. 33 dated 30th January, 2018.

With reference to the subject and letter cited above, on approval of the Hon'ble Vice-Chancellor, the change of PG Guide is permitted in respect of PG Student of your department:

Sl. No.	Name of the Student	Previous Guide	New Guide
1.	Dr. Devendra R.	Dr. G. S. Mahishale	Dr. S. G. Balaganur

This is for your information.

REGISTRAR
REGISTRAR
BLDE (Deemed to be University)
Vijayapura-586103. Karnataka

Copy to:
- The Dean, Faculty of Medicine and Principal
- The Controller of Examinations
- Dr. S. G. Balaganur, Guide
- Dr. Devendra R., PG Student

04/04/18

Smt. Bangaramma Sajjan Campus, B. M. Patil Road (Sholapur Road), Vijayapura - 586103, Karnataka, India.
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ANNEXURE VIII

INFORMED CONSENT FORM

TITLE OF RESEARCH : TO STUDY CO RELATION BETWEEN ECG & 2D ECHOCARDIOGRAPHY IN LOCATING ACUTE MYOCARDIAL INFARCTION & PREDICTING COMPLICATION

GUIDE : DR S G BALAGANUR

P.G.STUDENT : DR DEVENDRA NAIK N

PURPOSE OF RESEARCH:

I have been informed that the purpose of this study Is TO STUDY CO RELATION BETWEEN ECG & 2D ECHOCARDIOGRAPHY IN LOCATING ACUTE MYOCARDIAL INFARCTION & PREDICTING COMPLICATION

PROCEDURE:

I understand that I will undergo detailed history and clinical examination and investigations.

RISKS AND DISCOMFORTS:

I understand that there is no risk involved in this study and I may experience mild pain during the above mentioned procedures.

BENEFITS:

I understand that my participation in this study will help to study the correlation of locaton of the myocardial infarction according to ECG & 2D echocardiography.

CONFIDENTIALITY:

I understand that the medical information produced by the study will become a part of hospital record and will be subjected to confidentiality and privacy regulation of hospital. If the data is used for publication the identity will not be revealed.

REQUEST FOR MORE INFORMATION:

I understand that I may ask for more information about the study at any time.

REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and I may refuse to participate or withdraw from study at any time.

INJURY STATEMENT:

I understand in the unlikely event of injury to me during the study I will get medical treatment but no further medical compensation.

_____ _____

(Signature of Guardian) (Signature of patient)

STUDY SUBJECT CONSENT FORM:

I confirm that Dr. Devendra Naik N has explained to me the purpose of this research, the study procedure that I will undergo and the possible discomforts and benefits that I may experience, in my own language.

I have been explained all above in detail in my own language and I understand the same. I agree to give my consent to participate as a subject in this research project.

SIGNATURE OF PARTICIPANT DATE

SIGNATURE OF WITNESS DATE

PROFORMA

Name of the patient :

Age in years :

Sex :

Address:

Religion:

Occupation:

IP no/OP no:

Presenting Complaints :

Past history:

Personal history:

1. Tobacco chewing
2. Smoking
3. Alcoholism
4. Diet- Veg/Mixed

Family history:

GENERAL PHYSICAL EXAMINATION:

Built :

Nourishment :

Ht(Cm) :

Wt(Kg) :

BMI:

Pallor

Icterus

Clubbing

Cyanosis

Edema

6. Vital parameters a. Pulse :

b. BP :

c. Respiratory rate :

d. Temperature

SYSTEMIC EXAMINATION:

CARDIOVASCULAR SYSTEM

RESPIRATORY SYSTEM

ABDOMEN EXAMINATION

CENTRAL NERVOUS SYSTEM

CONCLUSION:

SIGNATURE

DATE:

BIOCHEMISTRY

❖ Trop T / I	
❖ CPK MB	
❖ Fasting lipid profile	
❖ FBS/PPBS/RBS/HBA1C	
❖ Serum creatinine	
❖ Blood urea	

PATHOLOGY 1)Urine Routine	
Urine albumin	
Urine sugar Urine bile salts Urine bile pigments	
Urine microscopy RBC's Pus cells Cast's Epithelial cells	
2)Complete blood count:	
Hb	gm/dl
Total count	Cells/cumm
Differential count Neutrophils	%

Lymphocytes	%
Eosinophils	%
Basophils	%
Monocytes	%
ESR	At end of 1st hour.

2D-ECHOCARDIOGRAPHY:

LVIVSd : cm

LVIDd:	cm			Aorta :	cm
LVPWd :	cm	LVISd :	cm	LA (AP):	cm
RVIDd :	cm	EF% :		PA :	cm

VALVES :

Mitral Valve :

Aortic Valve :

Tricuspid Valve :

Pulmonary Valve :

CHAMBERS :

Left Ventricle :

Right Ventricle :

Left Atrium

Right atrium :

SEPTAE :

GREAT ARTERIES

Aorta :

Pulmonary Artery :

DOPPLER STUDY

Mitral Valve :

Aortic Valve :

Tricuspid Valve :

Pulmonary Valve :

REGIONAL WALL MOTION ABNORMALITIES :

PERICARDIAL EFFUSION :

CLOT/VEGETATION :

CONCLUSION

ECG: STANDARDISATION:

RATE:

RHYTHM:

P WAVE:

PR INTERVAL:

QRS COMPLEX:

ST SEGMENT:

T WAVE:

S-1 R:

MEAN QRS AXIS:

CONCLUSION:

SIGNATURE

DATE:

KEY TO MASTER CHART

1DHB	1ST DEGREE HEART BLOCK
2DHB	2ND DEGREE HEART BLOCK
A	ANTERIOR WALL MYOCARDIAL INFARCTION
AL	ANTERO LATERAL
ALWMI	ANTERIOR LATERAL WALL MYOCARDIAL INFARCTION
AS	ANTERIOR SEPTAL
ASWMI	ANTERIOR SEPTAL WALL MYOCARDIAL INFARCTION
AW	ANTERIOR WALL
AWMI	ANTERIOR WALL MYOCARDIAL INFARCTION
AX	APICAL
BC	BASAL CREPITATION
CHB	COMPLETE HEART BLOCK
F	FEMALE
G	GLOBAL
I	INFERIOR WALL
IL	INFERO LATERAL
ILWMI	INFERIOR LATERAL WALL MYOCARDIAL INFARCTION
IS	INFERIOR SEPTAL
IWMI	INFERIOR WALL MYOCARDIAL INFARCTION
L	LATERAL
LBBB	LEFT BUNDLE BRANCH BLOCK
LV	LEFT VENTRICLE
M	MALE

N	NO
NAD	NO ABNORMALITY DETECTED
NEG	NEGATIVE
POS	POSITIVE
PW	POSTERIOR WALL
RBBB	RIGHT BUNDLE BRANCH BLOCK
RV	RIGHT VENTRICLE
SB	SINUS BRADYCARDIA
ST	SINUS TACHYCARDIA
VPC	VENTRICULAR PREMATURE CONTRACTION
VT	VENTRICLE TACHYCARDIA
Y	YES