"ECHOCARDIOGRAPHIC PREDICTORS OF EARLY IN- HOSPITAL HEART FAILURE DURING FIRST ST-ELEVATION MYOCARDIAL INFARCTION"

Submitted By

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in

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Under the guidance of

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2014

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation entitled **"ECHOCARDIOGRAPHIC PREDICTORS OF EARLY IN- HOSPITAL HEART FAILURE DURING FIRST ST-ELEVATION MYOCARDIAL INFARCTION"** is a bonafide and genuine research work carried out by me under the guidance of **Dr.BADIGER SHARANABASAWAPPA** **M.D.** Professor, Dept Of Medicine.

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LIST OF ABBREVIATIONS

AMI	-	Acute Myocardial Infarction.
HF	-	Heart Failure
CVS	–	Cardiovascular System
LV	-	Left Ventricular.
EF	-	Ejection Fraction.
DT	-	Deceleration Time
MPI	-	Myocardial Performance Index.
ECG	-	Electrocardiogram
LBBB	-	Left Bundle Branch Block.
STEMI	-	ST Elevation Myocardial Infarction.
ACS	-	Acute Coronary Syndrome.
MI	-	Myocardial Infarction.
RAAS	-	Renin Angiotensin Aldosterone System.
AT II	-	Angiotensin II.
ANP	-	Atrial Natriuretic Peptide
BNP	-	Brain Natriuretic Peptide
PCWP	-	Pulmonary Capillary Wedge Pressure.
HTN	-	Hypertension
LA	-	Left Atrium
ACC	-	American College of Cardiology.
AHA	-	American Heart Association.
2D	-	Two Dimensional
ASE	-	American Society of Cardiology

WMSI - Wall Motion Score Index

3D - Three Dimensional

CT - Computerised Tomography

MRI - Magnetic Resonance Imaging

ACE - Angiotensin Converting Enzyme

NSTEMI - Non-ST Elevation Myocardial Infarction

UA - Unstable Angina

ABSTRACT

INTRODUCTION

Acute Myocardial infarction (AMI) is a significant public health problem in developing countries. Heart failure (HF) following AMI is a leading cause of cardiovascular (CVS) mortality and morbidity. An increasing no of studies have reported the use of several indices like the Ejection fraction(EF), left atrial volume index (LAVI) and diastolic indices like E/A ratio , deceleration time(DT) in predicting Early in-hospital failure in patients with ST elevation myocardial infarction. Hence the study to analyze the role of left atrial volume compared to other conventional parameters of systolic and diastolic left ventricular function in patients with first ST elevation MI in predicting early heart failure.

METHODS

A total of 61 patients with ST elevation myocardial infarction were studied, a detailed history, general examination, routine investigations were performed and ECHO was performed within 72 hrs and EF,DT, E/A,LVEDVI,LVESVI and LAVI were estimated ,patients were then divided according to Killip class> II and < II and analyzed.

RESULTS

Of 61 patients 25 developed in-hospital heart failure(killip>II), the mean age of patient in our study was58.5yrs and mean age of patient who developed HF was64.12yrs.Diabetes was present in 28 patients ,of which 18 developed HF which was significant(p 0.01).46 patients had involvement of anterior wall of which 24 patients developed HF (p 0.001). EF was significantly associated with HF with a mean of 36% compared to that of 45% without HF(p 0.001).LAVI showed a

significant association with development of HF with a mean of 28.96 in those with HF compared to22.58 in those without HF..

CONCLUSION

In our study of patients with STEMI, there was increase in incidence of HF (Killip>II) in elderly patients (>60yrs) and in diabetic individuals. There was significant increase in incidence of HF in patients with infarction of anterior wall. Ejection fraction<35% was significantly associated with increased incidence of HF and a strong predictor of In-hospital HF. LAVI was also significant predictor of in-hospital HF in our study and a LAVI>28ml/m^2 was associated with a high incidence of In-hospital HF

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INTRODUCTION

Acute Myocardial infarction is a significant public health problem on increase in developing countries. ST elevation constitutes approximately 40% of all acute myocardial infarction. The risk of cardiac complications such as Heart failure, reinfarction , arrhythmias is substantial clinical evidence of heart failure in myocardial infarction is associated with increased mortality even if the manifestations of failure resolve within the first 24 hours. Assessment of myocardial function is essential to facilitate further management and enable further development of new approaches and algorithms to improve outcome and detection of patients with acute myocardial infarction at risk of development of in-hospital heart failure is necessary to limit myocardial injury and left ventricular dysfunction[1] .

Heart failure following AMI is a leading cause of cardiovascular mortality and morbidity and contributes to significantly to worldwide burden of CVS diseases[2].

The incidence of HF has dramatically increased due to increasing role of coronary artery disease.

Early detection of patients with AMI at risk of development of in hospital failure is necessary to limit myocardial injury and LV dysfunction.

Echocardiography was introduced in clinical practice in 1970 and it rapidly grew in popularity once its outstanding abilities to diagnose valve disease and assess left ventricular function was established, it is most widely used advanced imaging technique in cardiovascular system as it allows rapid non invasive assessment of cardiac structure and function in a wide variety of hospital settings.

Non invasive evaluation of LV function has been assessed by systolic as well as diastolic echocardiographic indices in correlation to short term clinical outcome .Left ventricular systolic functions have been extensively studied in relation to heart

failure, however it has now become apparent that left ventricular diastolic function contributes to signs and symptoms of clinical heart failure[3] .

An increasing number of studies have reported the use of several indices like the ejection fraction,left atrial volume and diastolic indices like E/A ratio , deceleration time in predicting Early in-hospital failure in patients with ST elevation myocardial infarction[3].

Some studies also shown the use Myocardial Performance Index and Left Atrial Volume comparison to conventional parameters like ejection fraction in predicting early in-hospital failure in patients with ST elevation myocardial infarction.

Hence the study to analyze the role of left atrial volume compared to other conventional parameters of systolic and diastolic left ventricular function in patients with first ST elevation MI in predicting early heart failure

OBJECTIVE OF THE STUDY

TO ANALYZE THE ROLE OF LEFT ATRIAL VOLUME COMPARED TO OTHER CONVENTIONAL PARAMETERS OF SYSTOLIC AND DIASTOLIC LEFT VENTRICULAR FUNCTION IN PATEINTS WITH FIRST TIME ST ELEVATION MYOCARDIAL INFARCTION, IN PREDICTING EARLY HEART FAILURE DURING IN-HOSPITAL EVOLUTION BY ECHOCARDIOGRAPHY

REVIEW OF LITERATURE

THE DEFINITION OF ACUTE MYOCARDIAL INFARCTION

Myocardial infarction can be defined from a number of different perspectives related to clinical, electrocardiographic , biochemical and pathologic characteristics. The term myocardial infarction reflects death of cardiac myocytes caused by prolonged ischemia[4].

A working definition for acute evolving myocardial infarction in the presence of clinically appropriate symptoms has been established as patients with ST-segment elevation, i.e. new ST-segment elevation at the J point with the cut-off points ≥0.2 mV in V1 through V3 and ≥0.1 mV in other leads, or patients without ST-segment elevation, i.e. ST-segment depression or T wave abnormalities. Clinically established myocardial infarction may be defined by any Q wave in leads V1through V3, or Q wave ≥0.03 s in leads I, II, aVL, aVF, V4, V5 or V6 [4].

Myocardial infarction can be recognized when blood levels of biomarkers are increased in the clinical setting of acute myocardial ischemia. An increased value of cardiac troponin or CPKMB is defined as one that exceeds the 99th percentile of a reference population.

The present guidelines pertain to patients presenting with ischemic symptoms and persistent ST-segment elevation on the ECG. The great majority of these patients will show a typical rise of biomarkers of myocardial necrosis and progress to Q-wave myocardial infarction[4].

Typically the ST-segment elevation in acute myocardial infarction, measured at the J point, should be found in two contiguous leads and be $\geq$0.25 mV in men below the age of 40 years. $\geq$0.2 mV in men over the age of 40 years, or $\geq$0.15 mV in

women in leads V2–V3 and/or ≥0.1 mV in other leads (in the absence of left ventricular hypertrophy or left bundle branch block)[5].

In patients with inferior myocardial infarction, it is advisable to record right precordial leads (V3R and V4R) seeking ST elevation, in order to identify the concomitant right ventricular infarction. Likewise, ST-segment depression in leads V1–V3 suggests myocardial ischemia, especially when the terminal T-wave is positive (ST-elevation equivalent), and may be confirmed by concomitant ST elevation ≥0.1 mV recorded in leads V7–V9[5].

In patients with a suspicion of myocardial ischemia and ST-segment elevation or new or presumed new LBBB, reperfusion therapy needs to be initiated as soon as possible. However, the ECG may be equivocal in the early hours and, even in proven infarction, may never show the classical features of ST-segment elevation and new Q waves. If the ECG is equivocal or does not show evidence to support the clinical suspicion of myocardial infarction, ECGs should be repeated and, when possible, the current ECG should be compared with previous tracings additional recordings of, for example, lead V7, V8 and V9 may be helpful in making the diagnosis in selected cases[5].

CLINICAL PRESENTATION

It is accepted that the term MI reflects a loss of cardiac myocytes (necrosis) caused by prolonged ischemia. Ischemia is the result of a supply and demand mismatch. Ischemia in a clinical setting can be identified from the patient's history and from the electrocardiogram[6].

Possible ischemic symptoms include chest, epigastric, arm, wrist or jaw discomfort with exertion or at rest and usually lasts at least 20 min, but may be shorter in duration, it may then radiate to the arm, jaw, back or shoulder.

The discomfort is usually not sharp or highly localized and may be associated with dyspnea, diaphoresis, nausea, vomiting or light-headedness. The atypical variant can develop in the epigastrium (often confused with indigestion), arm, shoulder, wrist, jaw or back, without occurring in the chest[6].

Symptoms can also include unexplained nausea and vomiting, persistent shortness of breath secondary to left ventricular failure and unexplained weakness, dizziness, lightheadedness or syncope, or a combination of these symptoms, they may be noted in association with chest discomfort or they may occur in the absence of chest symptoms[6].

THE PATHOGENESIS OF ACUTE MYOCARDIAL INFARCTION

An acute coronary syndrome is always a result of sudden reduction in coronary blood flow caused by atherosclerosis with thrombosis superimposed, with or without concomitant vasoconstriction. The clinical presentation and outcome depend on the location of the obstruction and the severity and duration of myocardial ischemia. Occlusive and persistent thrombosis prevails is the basic pathophysiology in myocardial infarction in ST elevation myocardial infarction. About 2/3 to 3/4 of fatal coronary thrombi are precipitated by sudden rupture of a vulnerable plaque (inflamed, lipid-rich plaque covered by a thin fibrous cap)[4]. Other poorly defined mechanisms such as plaque erosion account for the rest. As many as 3/4 of all infarct related thrombi appear to evolve over plaques causing only mild-to-moderate stenosis prior to infarction and after thrombolysis[4].However, severe stenosis are more likely to undergo plaque events leading to infarction than mild ones. Myocardial infarction caused by complete coronary artery occlusion begins to develop after 15–30 min of severe ischemia (no forward or collateral flow) and progresses from the subendocardium to the subepicardium in a time-dependent fashion[4].

THE NATURAL HISTORY OF ACUTE MYOCARDIAL INFARCTION

The true natural history of myocardial infarction is hard to establish for a number of reasons: the common occurrence of silent infarction, the frequency of acute coronary death outside hospital and the varying methods used in the diagnosis of the condition. Community studies have consistently shown that the overall fatality of acute heart attacks in the first month is between 30% and 50%, and of these deaths about one-half occur within the first 2hours[4.]

The mortality of STEMI is influenced by many factors, among them: age, Killip class, time delay to treatment mode of treatment, history of prior myocardial

infarction, diabetes mellitus, renal failure, and number of diseased coronary arteries, ejection fraction, and treatment. The in-hospital mortality of unselected STEMI patients in the national registries of the ESC countries varies between 6% and 14% [6].

The 30 day mortality rate was 8.6% for STEMI patients in India according to CREATE registry data which was similar in comparison to ESC countries[7]

COMPLICATIONS OF ST ELEVATION MYOCARDIAL INFARCTION

ST elevation myocardial infarction can result in a number of complications. Complications are stratified into the following categories: ischemic, mechanical, arrhythmic, embolic, and pericarditis[4, 8].

TABLE NO 1: COMPLICATIONS OF STEMI

Sl. No	COMPLICATIONS	
1.	Ischaemic	Reinfarction
2.	Hemodynamic	Cardiogenic shock Myocardial rupture Left ventricular aneurysm Pseudoanuerysm Rupture of ventricular septum
3.	Conduction abnormalities	Post infarction conduction abnormalities
4.	Arrhythmic	Sudden cardiac death
5.	Embolic	Stroke
6.	Pericardial	Post myocardial infarction pericarditis Dressler's syndrome.

ACUTE HEART FAILURE AS A COMPLICATION OF AMI

Around 10-20% of patients with Acute coronary syndrome have concomitant heart failure and upto 10% of ACS develop heart failure during hospitalization[9]. Patients with ACS with ST elevation have high levels of cardiac biomarkers corresponding to high levels of myocardial injury. Patients who are complicated by heart failure have increased short and long term mortalities. The rates are higher if Patients with ACS develop heart failure later than those who develop at presentation.

Cardiac pump failure due to myocardial injury is the leading cause of circulatory failure and in-hospital death from acute MI (myocardial infarction). Manifestations of circulatory failure may include a weak pulse, low blood pressure, cool extremities, a third heart sound, pulmonary congestion, oliguria, and cold sweat perspiration. However, several distinct mechanisms, hemodynamic patterns, and clinical syndromes characterize the spectrum of circulatory failure in acute myocardial infarction. The degree of left ventricular dysfunction correlates well with the extent of acute ischemia/infarction[2].

Hemodynamic compromise becomes evident when impairment involves 20% to 25% of the left ventricle, and cardiogenic shock or death occurs with involvement left ventricular muscle of 40% or more. Pulmonary congestion and S3 and S4 gallops are the most common physical findings[8].

Left heart weakness is linked with both long- and short-term poor prognosis. Clinical symptoms begin with dyspnea, sinus tachycardia, a third heart sound and murmurs which are first detected on the pulmonary basis but then spread and involve whole lungs. However, developed pulmonary congestion is not necessarily followed by the auscultator signs[10].

Cardiogenic shock is a form of severe left ventricular failure characterized by marked hypotension (systolic pressures less than 80mm Hg) and reductions in cardiac index (<1.8 L/min/m2) despite high left ventricular filling pressure (pulmonary capillary wedge pressure greater than 18 mm Hg). The cause is loss of a critical functional mass (>40%) of the left ventricle. Cardiogenic shock is associated with mortality rates of more than 70% to 80% despite aggressive medical therapy[8].

Risk factors include age, large (usually anterior) acute myocardial infarction, previous myocardial infarction, and diabetes[8].

The goal of risk stratification before and early after discharge for acute myocardial infarction is to assess ventricular and clinical function, latent ischemia, and arrhythmic risk, and to use this information for patient education and prognostic assessment and to guide therapeutic strategies[2.]

LV dysfunction is the single strongest predictor of mortality following STEMI. The mechanisms responsible for LV dysfunction in the acute phase include myocardial loss and remodelling due to infarction, ischemic dysfunction (stunning), atrial and ventricular arrhythmias and valvular dysfunction. There is frequently evidence of both systolic and diastolic dysfunction. The degree of heart failure following myocardial infarction may be categorized according to the Killip classification[5,8].

PATHOPHYSIOLOGY:

Heart failure is the multisystem disorder which is characterized by the abnormalities of cardiac, skeletal muscle and renal function with stimulation of the sympathetic nervous system and complex pattern of neurohormonal changes[10].

Myocardial Systolic Dysfunction:

The primary abnormality in HF is an impairment in the left ventricular Function leading to fall in cardiac output. This fall in cardiac output leads to activation of several neurohormonal compensatory mechanisms aimed at improving the mechanical environment of heart[9,10,11].

Activation of sympathetic system tries to maintain cardiac output with increase in heart rate, increases myocardial contractility and peripheral vasoconstriction, activation of Renin- Angiotensin-Aldosterone system results in vasoconstriction and increase in blood volume with salt and water retention. Concentration of vasopressin and natriuretic peptides increase. Furthermore there may be progressive cardiac dilatation or alterations in cardiac structure or both[9].

Renin – Angiotensin – Aldesterone system (RAAS)

Simulation of RAAS leads to increased concentration of Renin, Angiotensin II and Aldosterone. AT II is a potent vasoconstrictor of renal and systemic circulation where it stimulates release of Nor Adrenaline from sympathetic nerve terminals, which inhibits vagal tone and promotes release of Aldosterone. This leads to sodium and water retention, in addition, AT – II has an important effects on cardiac myocytes and may contribute to endothelial dysfunction[12].

Sympathetic Nervous System:

Sympathetic nervous system is activated in HF via low and high pressure baroreceptors as an early compensatory mechanism which provides inotropic support and maintains cardiac output. In long term, the ability of the myocardium to respond to chronic high concentration of catecholamines is activated by down regulation of receptors. This may be associated with baroreceptor dysfunction and further increase in sympathetic activity .

Natriuretic Peptides

There are three natriuretic peptides of similar structure and these exert a wide range of effects on heart, kidneys and cardio vascular system. Atrial natriuretic peptide is released from atria in response to stretch, leading to natriuresis and vasodilatation. In humans, Brain natriuretic peptide is also released from heart and its actions are similar to those of ANP.C- type natriuretic peptide is limited to vascular endothelium and CNS and has only limited effects on natriuresis and vasodilatation[13].

Vasopressin:

Vasopressin concentration is also increased in severe chronic HF. High concentration of the hormone are particularly common in patients receiving diuretic treatment and this may contribute to development of hyponatremia [12].

Endothelin:

Secreted by vascular endothelial cells, is a potent vasoconstrictor on renal vasculature. Its concentration is also correlated with indices of severity such as pulmonary capillary wedge pressure and need for hospitalisation and death [12].

Diastolic dysfunction:

Diastolic dysfunction refers to clinical syndrome of HF with preserved Left Ventricle Ejection Fraction (EF – 40%or more) in the absence of major valvular disease. In diastolic HF, LV cavity is stiff due to increased LV mass. It relaxes slowly in early diastole and offers greater resistance to filling in late diastole so that diastolic pressure is increased. The low cardiac output manifests as fatigue while the increase in end diastolic pressure is transmitted backwards through valve less pulmonary veins to pulmonary capillaries resulting in exertional dyspnea[5].

Mechanisms contributing to abnormal LV diastolic properties include stiff arteries, hypertension, ischemia, Diabetes and intrinsic myocardial changes with or without associated hypertrophy.

The prognosis of diastolic HF is generally better than that of systolic Heart failure. Diastolic Heart failure is common in clinical practice. The Diagnosis of diastolic HF may be considered in patients with HF who have normal LV EF (40%) or more) [14].

Myocardial Dysfunction due to Remodeling, Hibernation and Stunning:

After extensive myocardial infarction, cardiac contractility is frequently impaired and neurohormonal activation leads to regional eccentric and concentric hypertrophy of Non- infarcted segment with expansion of infarct zone. This is known as Ventricular Remodeling.

Particular risk factors for this development of progressive ventricular dilatation after an MI include large infarcts, anterior infarctions, occlusion of artery related to infarction and hypertension. Myocardial dysfunction may also occur in response to stunning which describes delayed recovery of myocardial function despite restoration of coronary blood flow in the absence of irreversible damage. This is in contrast to hibernating myocardium which describes persistent myocardial dysfunction due to reduced perfusion although cardiac myocytes remain viable and myocardial contraction may improve with revascularization[11].

Echocardiography was introduced into clinical practice in the 1970's and rapidly grew in popularity once its outstanding ability to diagnose valve disease and to assess LV function was established. Utilisation of echocardiography has continued to grow and ultrasound has advanced, particularly with the introduction of harmonics, tissue Doppler and digital imaging, allowing rapid assessment of cardiac structure and function. Echocardiography is the most widely used advanced imaging technique in cardiovascular medicine because it allows rapid, noninvasive assessment of cardiac function and structure in a wide variety of hospital and community settings at moderate cost[15,16].

As our knowledge of the underlying pathogenesis of HF has extended beyond the assessment of pump function, so too have echocardiographic methods. Using the theory of pressure changes and gradients within the heart, it is possible to use Doppler ultrasound to assess both left atrial and LV pressures and their changes relative to one another in order to determine the effectiveness of diastolic filling of the LV. Diastolic filling, as assessed by Doppler echocardiography, is a useful surrogate for diastolic function of the LV and is used to identify, diagnose and quantify diastolic filling abnormalities and is an important predictor of morbidity and mortality after acute myocardial infarction[15,17].

Diastolic function may contribute significantly to the forward output of the ventricle. Frank Starling first described the importance of myocardial stretch upon subsequent contraction of the muscle. Whether HF is primarily due to systolic or diastolic LV dysfunction, the clinical syndrome and symptoms may be similar and difficult to differentiate in individual patients. Whilst it is rare for systolic dysfunction to exist in solitude (diastolic filling abnormalities are almost always present when contractile function is reduced) it is possible for diastolic dysfunction to exist in

isolation. From an echocardiographic perspective, the differences are clearly apparent. In the setting of systolic impairment, the LV is likely to be dilated, spherical in shape and show impaired myocardial thickening, i.e. fibre shortening and depressed pump function. In patients with primarily diastolic abnormalities, the LV may be small, with hypertrophied walls and the LA is usually dilated. The ejection fraction may appear normal but the stroke volume that is generated from a small ventricular chamber is inadequate and thus forward output of the LV is significantly reduced[18].

Although the diagnosis of HF is based upon clinical findings, echocardiography is widely used and indeed advocated in patients with HF to document the degree of ventricular dysfunction and determine reversible or treatable causes of HF. Recently published guidelines for HF diagnosis from both the American College of Cardiology/American Heart Association[11] and include an objective measure of ventricular dysfunction[18,19].

In the case of HF patient, echocardiography is useful for determining the severity of the cardiac disease, such as degree of dilatation, hypertrophy and haemodynamic compromise. This will often include emphasis upon the LV, in particular on size and function[19].

VENTRICULAR SIZE

Left ventricular dilatation can be assessed by M-mode or two-dimensional echo methods. The easiest and most commonly used method measures the chamber diameter from the parasternal M mode or 2D view. Dilatation, if present, will almost certainly be detected by this method but may in fact be significantly underestimated in patients with HF due to the spherical shape of the LV or indeed overestimated if the M-mode beam is not orthogonal to the LV long axis. The use of single dimension to

accurately reflect volumes and global LV function requires symmetrical geometry of the LV to minimize error[20].

Left ventricular volume is one of the best prognostic parameters in patients after myocardial infarction and is prerequisite for calculation of ejection fraction[20].

In addition, many of the M-mode and simple 2D methods use algorithms to calculate LV volumes that make significant assumptions about the LV geometry. Left ventricular size is more accurately determined using a 2D biplane volumetric approach (typically the modified Simpson's or summation of discs method) from the apical four and two chamber views. It is important that both M-mode linear dimensions and LV end-systolic and end-diastolic volume[20].

The quantification of left ventricle volumes and EF is an important aspect of cardiac evaluation, its quantification is mainly related to global LV function[20,21].

THE LEFT ATRIUM

The left atrial response to LV dysfunction is complex and differs depending upon the stage of ventricular involvement. Initially, LA contraction compensates for the compromised early filling, but as LV relaxation slows and end-diastolic pressure increases, this is diminished. LA pressure rises in response to increased LV end-diastolic pressure and at the same time increased LV stiffness shortens the passive filling time (corresponding to short deceleration time of the mitral inflow) and patients develop restrictive filling and LA dysfunction[22]. LA diameter is often measured by M-mode and it may be inaccurate and significantly underestimate LA size[23].

Significant errors may occur due to beam angulation or where LA dilatation occurs along its long and axis. LA volume (or area) can be estimated using a modified

Simpson's method (summation of discs) from the apical two and four chamber views[24].

Left atrial volume is related to both systolic and diastolic LV function, is correlated with LV filling pressure and independently associated with HF. Thus, it is recommended that LA area or volume be measured from the 2-D apical views in patients in whom ventricular dysfunction in suspected and indexed to account for body size[22,24,25].

Left atrial function, or LA stroke volume, may also contribute significantly to forward output and is related to filling pressures and LA dysfunction may occur in the presence of preserved LV EF and may be a contributing factor to the development of diastolic HF[24].

LV SYSTOLIC FUNCTION

An assessment of global LV systolic function is central to diagnosis of heart failure the assessment of LV systolic function is an important goal of echocardiography in patients with HF and there are a number of validated quantitative echocardiographic approaches for comprehensive assessment of systolic function. The sensitivity of this technique depends on both operator and nature of measurement. There are a variety of approaches, from expert qualitative data (normal, mild, moderate or severe impairment) to complex quantitation[17,19].

The LV volume is one of the best prognostic parameter after acute myocardial infarction. The simplest quantification uses chamber diameter change over the cardiac cycle, or fractional shortening (FS), as a measure of global systolic function[19].

This is usually done by placing an M mode cursor through the LV cavity and provides a good estimate of systolic function. However, this M-mode method is biased towards the basal segments and is unreliable when regional wall motion

abnormalities are present. The simplest method of global assessment, and the most commonly used in clinical practice, is subjective assessment of systolic function. Typically, LV function is viewed in several views and a thoughtful judgment is made about overall systolic function - the so called “eye-ball” EF. Because this requires individual assessment of segmental function it is unquestionably subjective and dependent upon the interpreter’s experience[19].

Assessment of regional wall motion abnormalities becomes particularly important in these cases. Quantification of assessment of regional wall motion as recommended by the American Society of Echocardiography[19].

It requires allocating a numeric score based on the assessment of function (normal, hypokinetic, akinetic, dyskinetic, aneurysmal), for all segments of the ventricle and averaging the results. Many echocardiographers already do this in their mind but do not quantify or document the results. Despite being a simple incremental step from eyeballing ventricular function, this method is rarely used in clinical practice[19,26].

Each segment is assigned a score on the basis of its contractility as assessed visually: normal = 1, hypokinesis = 2, akinesis = 3, dyskinesis = 4, and aneurysm = 5. On the basis of this wall motion analysis scheme, a wall motion score index (WMSI) is calculated to semiquantitate the extent of regional wall motion abnormalities:

WMSI=sum of wall motion scores/no of segments visualized

A normally contracting LV has a WMSI of 1 (each of the 16 segments receives a wall motion score of 1; hence, the total score is 16 and WMSI is 16/16 = 1). The larger the infarct the higher the WMSI because wall motion abnormalities become more severe.LV volume is one of the best prognostic parameters in patients

with acute MI and is essential for calculating EF and LV mass, several methods are used like the prolate ellipse area length method truncated ellipsoid[19,20] .

The gold standard of echocardiographic assessment of systolic LV function is currently 2D biplane volume assessment. Typically, this requires manual tracing of the blood-endocardial interface in diastole and systole in both the apical four and two chamber views. There are several adaptations of this method, based on different formulae but the Simpson's summation of discs is the most accurate in a wide range of clinical scenarios and is thus the recommended method[20] .

This method is time-consuming, and requires significant operator expertise, but does provide a reasonable assessment of global systolic function and possibly more importantly, provides measurements such as LV volumes and EF that many clinicians are familiar with.

ASSESSMENT OF DIASTOLIC FUNCTION

Echocardiography does not allow direct assessment of diastolic function, but filling pressure may be estimated by measuring the pressure gradients, blood flow and annular motion during the diastolic phase of the cardiac cycle. Based upon the ratio of early to late mitral valve diastolic filling and deceleration time, five progressive filling categories have been described: normal, abnormal relaxation, pseudonormal, reversible restrictive filling and non-reversible restrictive filling[19,27].

Pulsed wave Doppler assessment of the mitral valve is now routinely used in clinical practice to non-invasively assess LV diastolic filling, although this is complicated in the presence of AF or a paced rhythm. The addition of pulmonary venous Doppler flow measurements helps to differentiate between true normal and pseudonormal filling and is useful for estimating LA pressure in patients with systolic

dysfunction and advanced diastolic filling abnormalities as well as in ischemic patients with normal EF and only mild filling abnormalities[19,27,28] .

Although these methods of assessing diastolic function are reliable and extensively researched, they are not always well applied and often echocardiographic reports may be confused in their nomenclature. One suggested approach describes the filling phases as grades from 0 to 4 (0 =normal, 1 = abnormal relaxation,
2 = pseudonormal, 3 = reversible restriction, 4 = non-reversible restriction).

This is attractive given that many other clinical scores are based on a similar system. Coupled with an objective measure of left atrial pressure elevation, such as the E/Ea ratio, this grading would provide a clinically useful assessment of diastolic function. The reporting of diastolic function needs to be consistent and based upon easily understood terminology.

However, in HF patients the simple assessment of the diastolic filling pattern and its reversibility, together with an estimate of LA pressure (pulmonary venous Doppler and/or mitral annular TDI) may be helpful for both clinical management and prognosis .

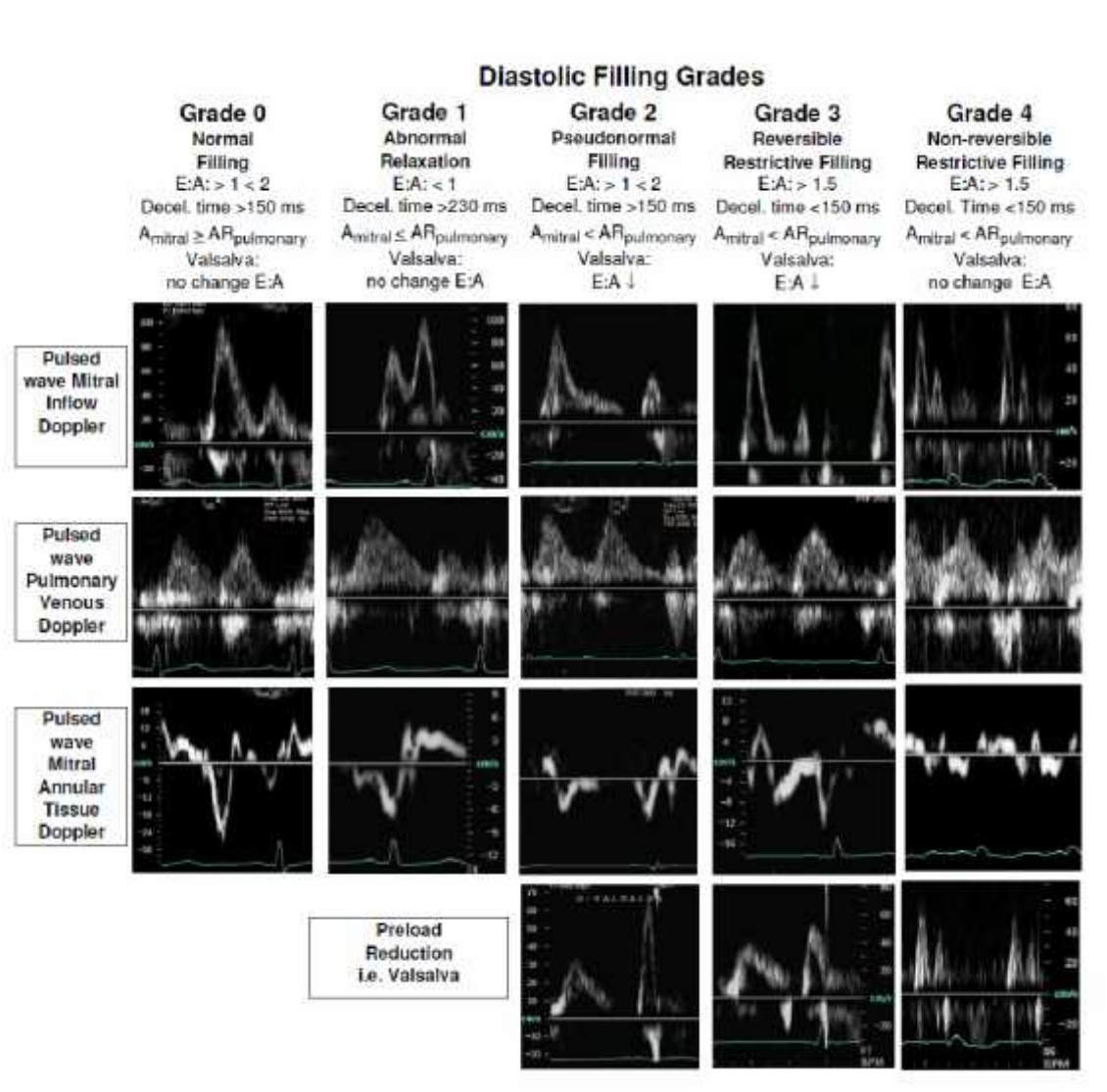


Figure 1: Daistolic filling grades based upon mitral,pulmonary and mitral annular pulsed wave doppler

SYSTOLIC VERSUS DIASTOLIC HEART FAILURE

Many studies suggest that around 40-50 percent of patients with clinical HF have no discernable systolic dysfunction by echocardiography or ventriculography. This group of patients with so-called “diastolic HF” is often described as having HF with preserved systolic function[27].

This is probably a misnomer and a more appropriate description might be “HF with preserved LV EF” as subtle systolic dysfunction may be present but the assessment method lacks the sensitivity for detection. Methods such as strain-rate imaging, stress echocardiography and tissue Doppler imaging may prove to be particularly useful in this area, where crude measurements of EF fail. The ACC/AHA guidelines for diagnosing diastolic HF, or rather HF with preserved systolic function, do not currently advocate a role for diagnostic echocardiography beyond assessment of systolic function[19].

LEFT ATRIAL VOLUME

The estimated ejection fraction of left ventricle (LV) determined by echocardiography has been used to predict cardiovascular outcomes. Recent evidence Highlights the importance of left atrial volume with regards to prediction of cardiovascular outcomes. The LA volume is a reflection of long- standing hemodynamic condition and has been compared to the “glycated hemoglobin of diabetes mellitus”[29].

The mechanical function of LA has been described in three phases within the cardiac cycle: 1) ‘reservoir’ 2) the ‘conduit’ and the 3)‘ contractile’ machinery. The LA functions as a ‘reservoir’ receiving blood from pulmonary veins during systole and isovolumic relaxation. It acts as a “conduit” during the early phase of ventricular diastole for blood passing from the pulmonary veins into the LV. This is followed by

atrial contraction during which the LV stroke volume is augmented by approximately twenty Percent[29].

The relative contribution of LA function to LV filling is dependent upon the diastolic properties of LV. In subjects with normal diastolic function, the relative contribution of the reservoir, conduit and contractile function of the LA to the filling of LV is approximately 40%, 35% and 25% respectively. As LV relaxation gradually worsens, the relative contribution of LA reservoir and contractile function increases while conduit function decreases. But with advanced diastolic dysfunction the LA serves predominantly as a conduit.

ASSESSMENT OF LA SIZE

LA is not a symmetrically shaped three-dimensional (3D) structure. Furthermore, LA enlargement may not occur in a uniform fashion. Therefore antero posterior measurement of LA by M-mode echocardiography is likely to be an insensitive assessment of any change in LA size. In contrast, LA volume by 2D or 3D echocardiography provides a more accurate and reproducible estimate of LA size as compared to magnetic resonance imaging (MRI) and cine-computerised tomography (CT). The LA size is measured at the ventricular end-systole when the LA chamber is at its greatest dimension. It is imperative to avoid foreshortening of the LA for computing LA volume. The confluence of the pulmonary veins and LA appendage should be excluded, when performing planimetry[29].

Echocardiographic assessment of LA volume is done by Simpson's method, area –length method and real time 3D echocardiography[29].

The simplest method for estimating LA volume is the cube formula, which assumes that the LA volume is that of a sphere with a diameter equal to the LA antero posterior dimensions[19,29].

LA volumes are calculated using either an ellipsoid model or Simpson's rule. The ellipsoid model assumes that the LA can be adequately represented as a prolate ellipse with a volume of $4\pi/3$ (L/2) (D1/2) (D2/2), where L is the long axis (ellipsoid) and D1 and D2 are orthogonal short-axis dimensions[15].

LA volume can be estimated using this biplane dimension-length formula by substituting the LA antero posterior diameter acquired from the parasternal long axis as D1, LA medial-lateral dimension from the parasternal short-axis as D2, and the LA long-axis from the apical 4-chamber for L[15].

Simplified methods using nonorthogonal linear measurements for estimation of LA volume have been proposed. To estimate the LA minor-axis dimension of the ellipsoid more reliably, the long-axis LA areas can be traced and a composite dimension derived. This dimension takes into account the entire LA border, rather than a single linear measurement. When long-axis area is substituted for minor-axis dimension, the biplane area length formula is used: 8 (A1) (A2)/3π (L), where A1 and A2 represent the maximal planimetered LA area acquired from the apical 4- and 2-chamber views, respectively, and L is length. The length remains the LA long-axis length determined as the distance of the perpendicular line measured from the middle of the plane of the mitral annulus to the superior aspect of the LA. In the area-length formula the length is measured in both the 4- and 2-chamber views and the shortest of these 2 length measurements is used in the formula. The area-length formula can be computed from asingle plane, typically the apical 4-chamber, by assuming A1 _ A2, such that volume _ 8 (A1)2/3π(L)

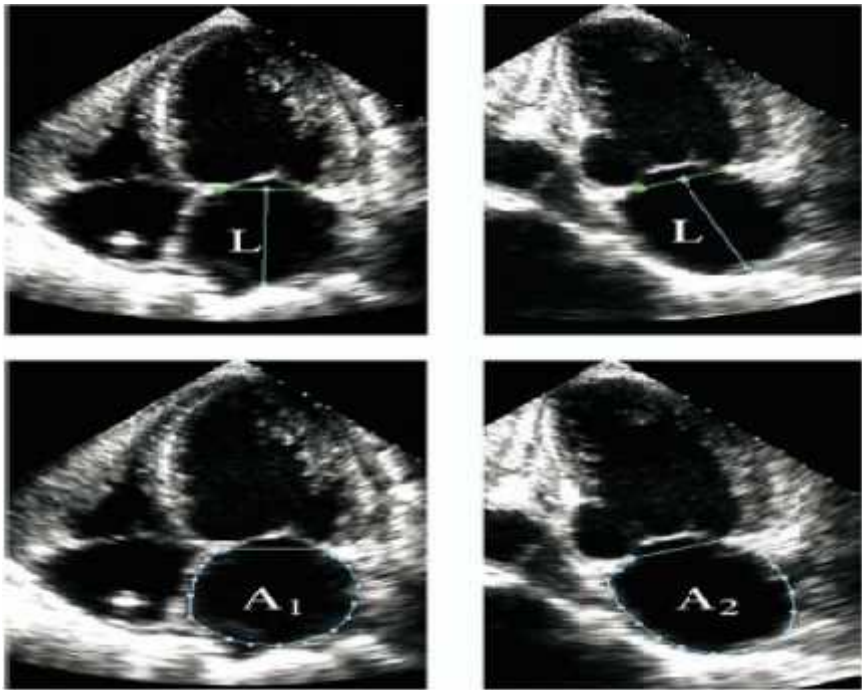


Figure2 : Measurement of left atrial (LA) volume from area-length (*L*) method using apical 4-chamber (*A4C*) and apical 2-chamber (*A2C*) views at ventricular end systole (maximum LA size). L is measured from back wall to line across hinge points of mitral valve. Shorter L from either A4C or A2C is used in equation

LA volume may also be measured using Simpson's rule, similar to its application for LV measurements, which states that the volume of a geometric figure can be calculated from the sum of the volumes of smaller figures of similar shape. Most commonly, Simpson's algorithm divides the LA into a series of stacked oval disks whose height is h and whose orthogonal minor and major axes are D1 and D2 (method of disks). The volume of the entire LA can be derived from the sum of the volume of the individual disks. Volume= $\pi/4$ (h) Σ (D1) (D2).

The formula is integrated with the aid of a computer and the calculated volume provided by the software.

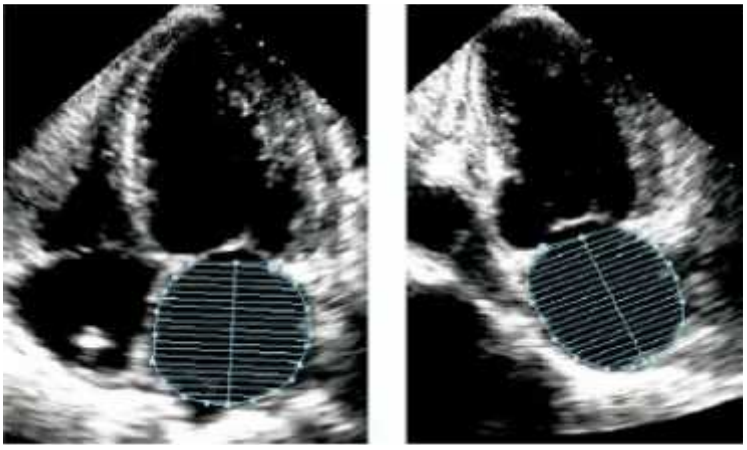


Figure 3: Measurement of left atrial (LA) volume from biplane method of disks (modified Simpson's rule) using apical 4-chamber (*A4C*) and apical 2-chamber (*A2C*) views at ventricular end systole (maximum LA size).

Normal indexed LA volume has been determined in several studies involving several hundred patients using the preferred biplane technique. Most trial results indicate a value of 22 ± 6 ml/m2 as the normal range and the same is recognized by ASE[15].

MATERIALS AND METHODS

1. SOURCE OF DATA:

- The material for the present study was collected from patients who were admitted to BLDEU'S Shri B.M. PATIL Medical College Hospital and Research centre, Bijapur and diagnosed with ST elevation acute Myocardial infarction according to inclusion criteria and period of study was from October 2011 To March 2013.

2. METHOD OF COLLECTION OF DATA:

- By detailed history and detailed clinical examination and examined for signs of heart failure, relevant investigations like ECG changes, lipid profile and CPK-MB enzyme levels and patients were subsequently subjected to Echocardiography within 72 hours of admission and various indices noted and analysis of various Echocardiographic indices was done in correlation to clinical signs .

3. SAMPLE SIZE:

Time period of study from October 2011 To March 2013,With prevelance rate of Acute Myocardial infarction – 7%[30]

At confidence interval of 95% allowing 5% margin of error, the sample size is 100 , $n = \frac{(1.96)^2 \times p \times q}{d^2}$

Among cases of myocardial infarction 40% cases were ST elevation MI 40% of 100 cases will be 40, so minimum 40 cases were to be included in the study

4. INCLUSION CRITERIA :

- Pateints age of Adult age>18 years
- Characteristic chest pain>20 min
- ST elevation> 1mm in atleast 2 contiguous leads
- Transient rise in CPKMB

5. EXCLUSION CRITERIA:

- Non ST elevation MI
- Early reinfarction
- In- hospital Death
- Previous Coronary Bypass
- Valvular heart disease
- Congenital heart disease
- Left bundle branch block
- Chronic heart failure

6. STATISTICAL METHOD:

- All results are expressed as Mean $\pm$ one SD.
- Two samples were compared by Mann Whitney rank sum test or unpaired ’t’test
- For Qualitative data Chi square test or Fischer”s exact test was applied

METHOD OF TEST

Echocardiography was preformed within 72 hours of chest pain in 61 patients with first ST elevation MI admitted to BLDEU'S Shri B.M.Patil Medical College Hospital and Research centre Several Clinical and Echocardiographic Variables were analysed

ECHOCARDIOGRAPHY VARIABLES:

A Comprehensive 2D Color Doppler Echo was performed in all patients with above inclusion criteria

The various indices used to predict early in hospital failure on Echo was determined by a single observer throughout the study

The various indices were calculated on Echo using the M mode to determine the various indices

The following parameters were done to establish the HF

1) LVEF: The ejection fraction was calculated using the simpson method

   EDV-ESV/EDV*100 On M mode

2) LA volume: The Left Atrial volume was calculated by the 2 Dimensional 4 chamber view by simpson's method
3) E/A RATIO and Deceleration time(DT):

   *Mitral diastolic inflow velocities at the tip of leaflets

   * LV systolic outflow curves obtained just below aortic valve

   Using these following were calculated

   a) E/A ratio diastolic velocities
   b) Deceleration time of early diastolic filling(DT)
4) Left ventricular end diastolic volume(LVEDV)
5) Left ventricular end systolic volume(LVESV)

Patients were also clinically defined as per KILLIP classification

KILLIP CLASSIFICATION

I. No clinical signs of heart failure.
II. Rales or crackles in lungs,S3, raised JVP
III. Frank pulmonary edema
IV. Cardiogenic shock or hypotension(peripheral vasoconstriction, BP<90mmhg, oliguria)

Investigations or interventions required in this study are routine standardized procedures.

INVESTIGATIONS

Blood examination: –Hb%, Total count ,Differential count, ESR. Serum creatinine, Blood urea, Blood sugars (fasting and post prandial), Creatine Phosphokinase (CPKMB), Fasting Lipid Profile

Urine examination:- Albumin, sugar, microscopy

Electrocardiography: 12 Lead ECG

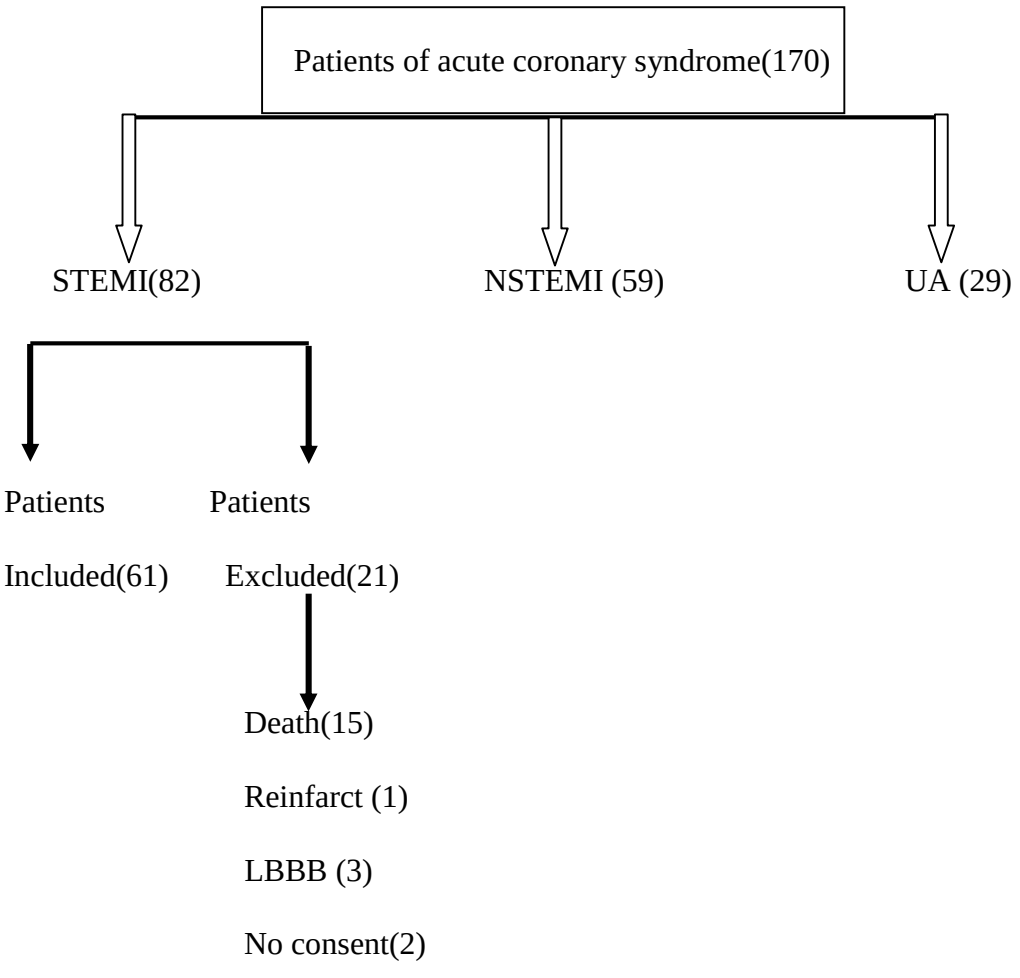
Radiological examination: Chest Xray PA view:

2D Echocardiography and color doppler

OBSERVATION AND RESULTS

A total of 61 patients were included in the study, who were admitted in Shri B.M.Patil Medical College, Hospital and research centre, bijapur ,during the study period.

A total of 170 Acute coronary syndrome patients were admitted,82 patients presented as ST elevation myocardial infarction(STEMI),59 patients were Non ST elevation myocardial infarction(NSTEMI) and 29 patients presented as unstable angina(UA). 82 patients presented as STEMI, out of which 61 patients were included in our study, 21 were excluded. Of the 21 excluded patients, 15 patients died in hospital,1 patient had reinfarction, 3 patients had a left bundle branch block and 2 patients did not undergo echocardiography.



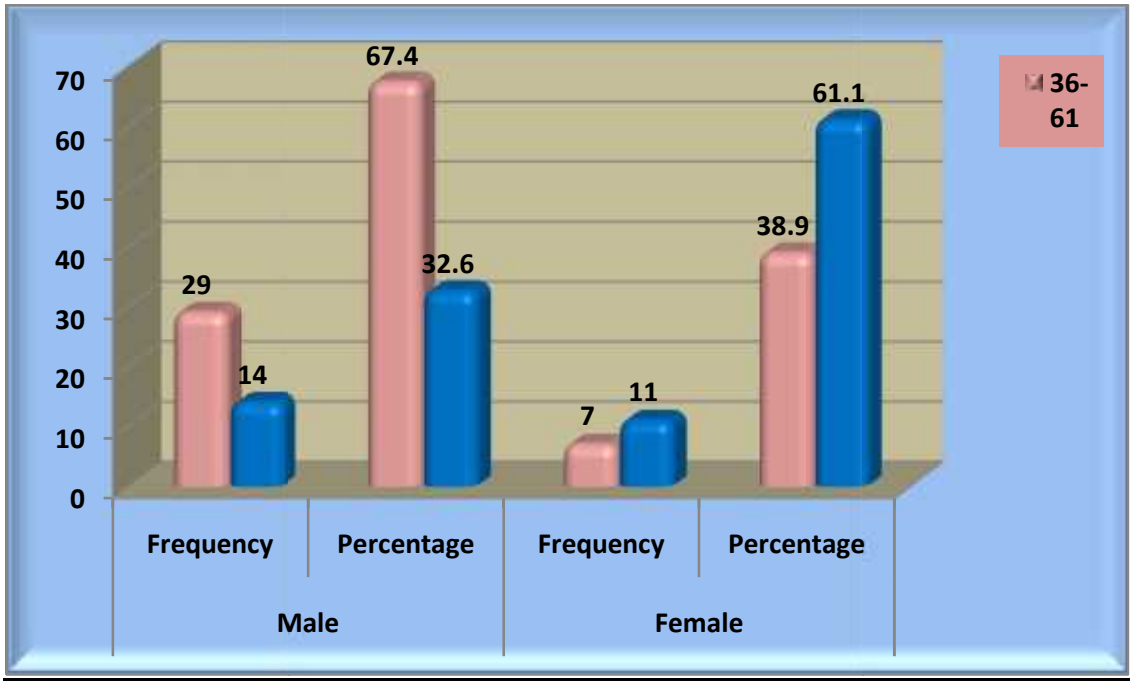
Of the 61 patients included 45 patients(73.7%) were male and 16 were female(26.2%) The average age of the patients included in study was 56.4years and no of patients with diabetes mellitus was 24(39.3%) and patients and those with hypertension were 13(21.3%) and 18(29.5%) were smokers the number of patients who were thrombolysed was 55(90.1%), and the number of patients with infarction of anterior wall were 46(75.4%) and those with infarction of inferior wall were 15(24.5%) and 39(63.9%) patients developed heart failure within first week of hospitalization

The number of patients who developed heart failure with Killip class>II were 25(40.9%), all of them being in class III and none in class IV.

Table no 2: AGE AND SEX DISTRIBUTION OF PATIENTS

	Male		Female	
Age(years)	Frequency	Percentage (%)	Frequency	Percentage (%)
36-61	29	67.4	7	38.9
61-81	14	32.6	11	61.1
	43	100.0	18	100.0

GRAPH NO 1: AGE AND SEX DISTRIBUTION OF PATIENTS



From the above table and graph, in our study of the total 61 patients 43 (70.4%) were male patients and 18(29.6%) were female patients. There were 36 patients in the age group of 36-60 years with 29 of them being male and 7 being females and 25 of patients were in age group of 60-80years and 14 of them being male and 11 being female.

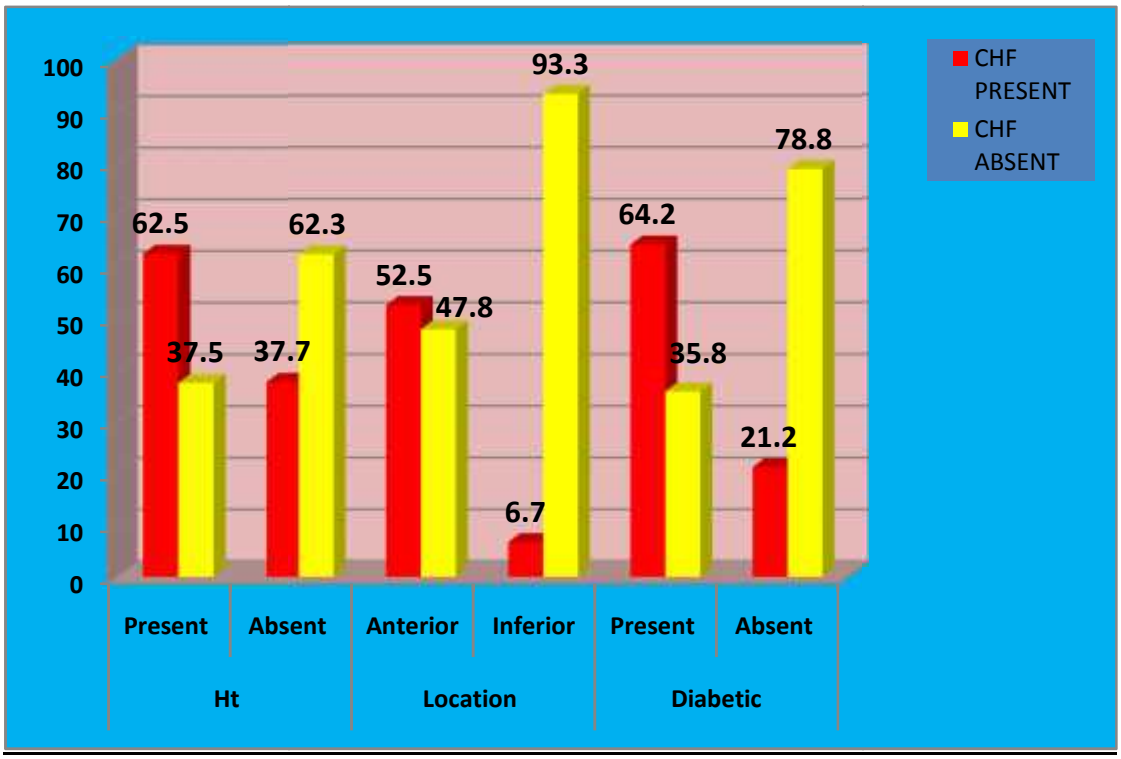
DATA ANALYSIS

Data Analysis in patients with and without heart failure (Killip>classII) Clinical variables according to presence or absence of heart failure.

Table no 3: CLINICAL VARIABLES ACCORDING TO ABSENCE OR PRESENCE OF HF

		Heart Failure PRESENT (25)		Heart Failure ABSENT (36)		P-VALUE
		PATIENTS	(%)	PATIENTS	(%)	
HTN	Present	05	62.5	03	37.5	0.254
	Absent	20	37.7	33	62.3	
Location	Anterior	24	52.5	22	47.8	0.001*
	Inferior	01	6.7	14	93.3	
Diabetes Mellitus	Present	18	64.2	10	35.8	<0.0001*
	Absent	07	21.2	26	78.8	
		Mean	Standard deviation	Mean	Standard deviation	
Age (years)		64.12	9.3	52.97	11.00	<0.0001
CKMB		72.12	15.08	52.802	12.92	<0.0001*

GRAPH NO 2: CLINICAL VARIABLES ACCORDING TO ABSENCE OR PRESENCE OF HF

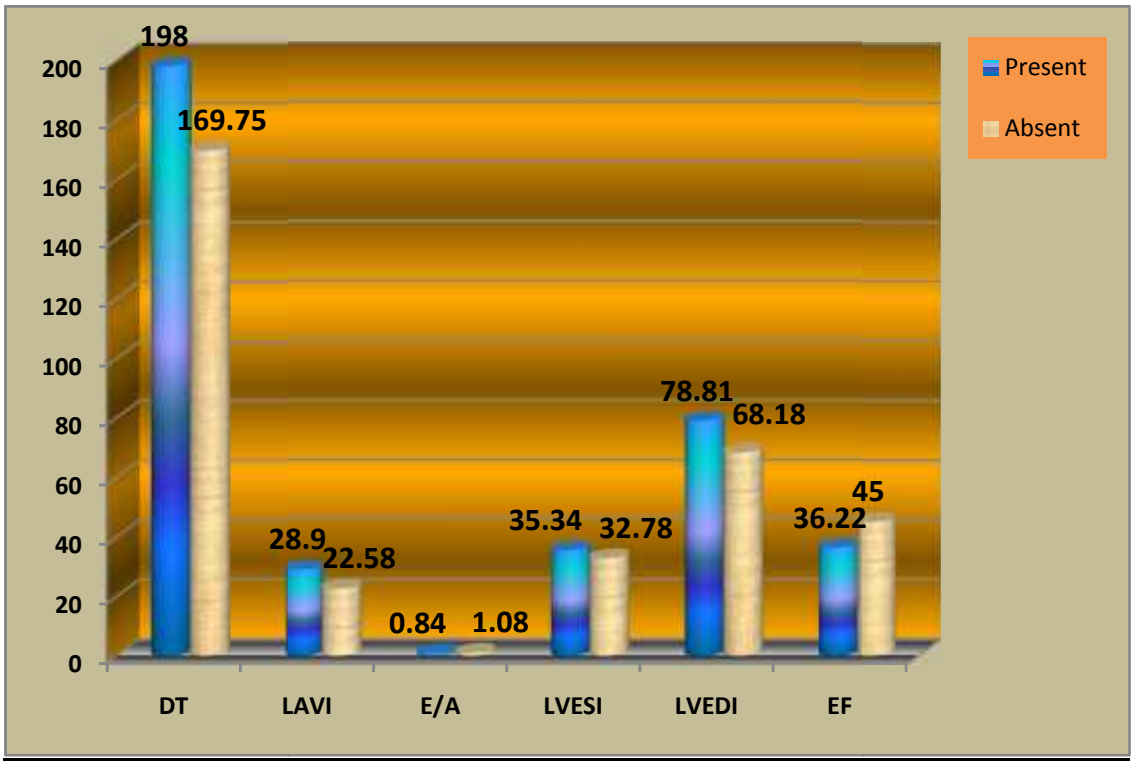


From above Table and graph, in our study ,there was no significant association between HTN and heart failure with a p value of 0.254,however there was significant association between anterior wall infarction and heart failure with a p value<0.001 and the p value was<0.0001 indicating the difference is highly signficant and strong association between Diabetes mellitus and heart failure and there was a significant association between age and heart failure with a p value<0.001 there was also a strong significance between levels of CPKMB with heart failure with a p value<0.001.

TABLE NO 4: ECHO CARDIOGRAPHIC VARIABLES ACCORDING TO PRESENCE OR ABSENCE OF HF

	Heart Failure PRESENT (25)		Heart Failure ABSENT (36)		P-VALUE
	Mean	Standard deviation	Mean	Standard deviation	
DT(ms)	198.0(25)	27.53	169.75(36)	27.81	0.01
LAVI ($ml/m^{2)}$	28.96(25)	3.30	22.58(36)	2.94	0.001*
E/A RATIO	0.844	0.15	1.0892	0.21	ns
LVESI(ml/m^2)	35.34	3.94	32.78	3.27	0.08
LVEDI(ml/m^2)	78.81	7.61	68.18	7.63	0.072
EF(%)	36.20	4.15	45.00	6.54	<0.001*

GRAPH NO 3: ECHOCARDIOGRAPHIC VARIABLES IN SUBJECTS ACCORDING TO PRESENCE OR ABSENCE OF HF

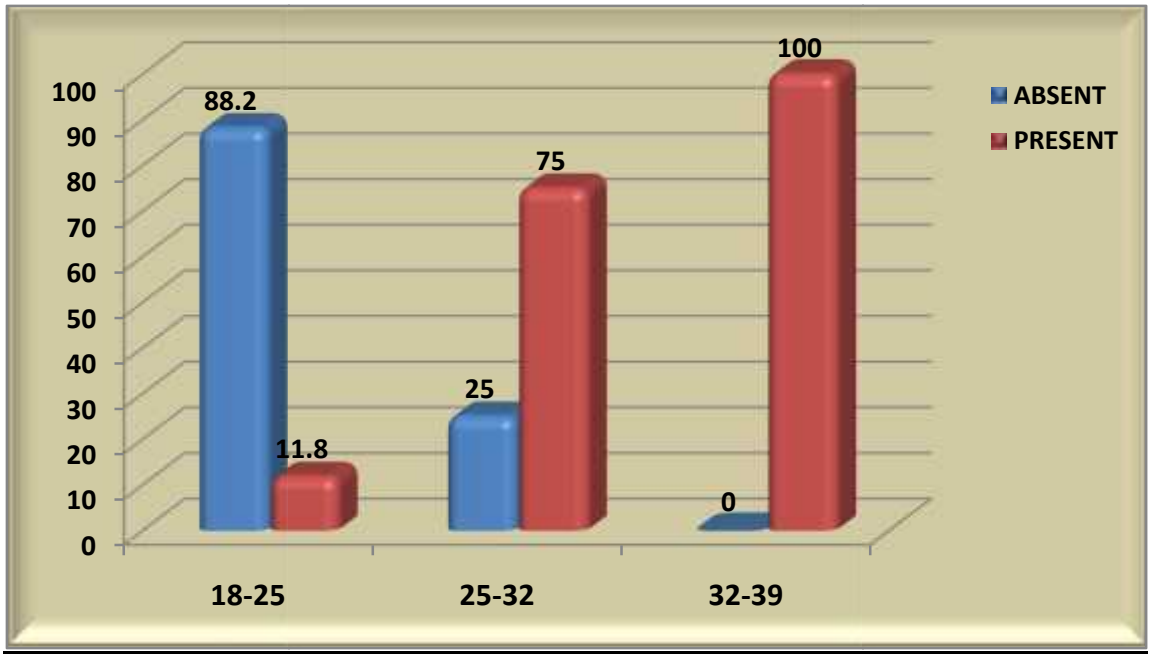


From above table, we infer that in our study the statistical difference was significant suggesting a strong association between deceleration time and heart failure with p value of 0.01 ,the left atrial volume index also had a strong association with heart failure with a p value 0.001 and there was strong association between Ejection fraction and heart failure with a p value<0.001,however there was no significant association between heart failure and diastolic indices like E/A ratio, left ventricular end diastolic volume index and left ventricular end systolic volume index.

TABLE NO 5: FREQUENCY AND PERCENTAGE DISTRIBUTION LAVI AMONG PATIENTS ACCORDING TO PRESENCE AND ABSENCE OF HEART FAILURE (HF)

LAVI (ml/m^2)	ABSENT	(%)	PRESENT	(%)
18-25	30	88.2	04	11.8
25-32	06	25.0	18	75.0
32-39	00	00	03	100

GRAPH NO 4: PERCENTAGE DISTRIBUTION OF LAVI ACCORDING TO PRESENCE OR ABSENCE OF HF



The above table and graph shows the distribution of LAVI among patients according to presence or absence of heart failure, Only 11.8% patients had heart failure with a LAVI in range of 18-25ml/m^2 and 75% patients had heart failure whose LAVI was in range of 25-32ml/m^2 and 100% patients had heart failure whose LAVI was in range of 32-39ml/m^2, signifying a strong association between increasing value of LAVI and development of heart failure

TABLE NO 6: BASELINE CHARACTERISTICS OF LAVI IN COMPARISON TO OTHER VARIABLES

	LAVI (ml/m^2)		
	<=24	>25	P-VALUE
Diabetes Mellitus			
present	24	09	0.04*
absent	10	18	
Hypertension			
present	30	23	0.726
absent	04	04	
LOCATION			
Anterior	21	25	0.005**
Inferior	13	02	
Diastolic grade			
0	29	04	
I	07	20	0.001
II	00	04	
III	00	01	

The cut -off median value of LAVI was $24ml/m^2$ in the analysis, and a value of LAVI $>24ml/m^2$ was strongly associated with Anterior wall location and grade I diastolic dysfunction as seen in the table there was association between presence of diabetes mellitus and LAVI value $>25ml/m^2$ with a p value of 0.04

TABLE NO 7: ASSCOIATION BETWEEN KILLIP AND LAVI

Heart Failure	LAVI (ml/m^2)		CHI-SQUARE	P-VALUE
	<=24	>24		
ABSENT	30	06	27.11	< 0.00001**
PRESENT	04	21		
TOTAL	34	26		

There is a significant association and a positive correlation between LAVI and Killip class with a p value <0.0001

TABLE NO 8: ODDS RATIOS FOR LEFT ATRIAL VOLUME INDEX (LAVI) AND EF PREDICTING HEART FAILURE

Variables	Odds ratio	p-value
LAVI $\geq 28ml/m^2$	37.91	0.009
EF $\leq 45\%$	2.7	0.0140

The cut-off value for significant variables was analyzed, a value of $28ml/m^2$ for LAVI and 45% for EF was considered, there were 14 subjects with LAVI>$28ml/m^2$, with 13 of them having Killip>II and one patient with Killip<II the odds ratio was significantly high and statistically significant($p<0.009$) and EF<45% was also analyzed, with 25 patients with Killip>II out of 46 patients which is statistically significant (p 0.014).

DISCUSSION

In our study, we analyzed the role of echocardiographic indices to predict in hospital heart failure in patients with first ST elevation myocardial infarction. 61 patients included in our study were analyzed to compare various echocardiographic indices to predict in hospital heart failure.

Out of 61 patients, 25 patients developed in hospital heart failure(Killip>II) associated risk factors were also analyzed in co relation with development of in hospital heart failure.

The various studies have shown a co relation between various echocardiographic indices like ejction fraction, Left atrial volume, E/A ratio and deceleration time in assessing the myocardial function which helps to predict going to failure following myocardial infarction.

Lilian P Souza, Orlando Campos et al[3] ,conducted a study on 95 patients admitted with ST elevation Myocardial infarction(MI) with Echo done in first 78 hrs. Early in-hospital failure occurred in 29%. They concluded that LVEF<45% was the single, strong and accurate predictor of early in hospital HF ,superior to LA volume .They included LA volume and Myocardial performance index and found that MPI alone could not predict HF in first ST elevation MI patients and Left Atrial volume was not associated with early HF in such patient. The MPI index was defined as sum of isovolumetric contraction time and isovolumetric relaxation divided by ejection time. They concluded that index was significantly higher in patients with MI.

S.H. Pouselen, S.E. Jensen et al[28] , in 1997 conducted a study in 65 patients with ST elevation MI. On analysis they identified age, ejection fraction <45% ,deceleration time<130ms ,E/A ratio>1.5 as variables significantly related to development of heart failure They concluded that assessment of left ventricular

diastolic function complements measurements systolic function in evaluation of cardiac function and mitral deceleration time<130ms best identifies patients at risk of development of HF following acute MI.

Jacob E Moller, Graham S.Hillis et al[31] 2003 conducted a study on 314 patients to analyze the role of left atrial volume as predictor of survival after acute myocardial infarction, several left ventricle systolic and diastolic function and measurement of LA volume was performed during admission and were divided into two groups >32ml/m^2 and <32ml/m^2 , they concluded that increased LA volume is powerful predictor of mortality after Acute myocardial infarction.

Roy Beinart, Valentine Boyko et al[32] in 2004 conducted a study on 395 individuals with AMI to evaluate the significance of left atrial volume within first 48 hours of admission as a long term predictor of outcome in patient with acute myocardial infarction and patients were divided into two groups >32ml/m^2 and<32ml/m^2 they concluded that LAVI>32ml/m^2 is an independent predictor of 5-year mortality.

Monika Maheshwari and S.K Kaushik[33] conducted a study in 2013 to assess LAVI in elderly patients with acute MI, they included 75 patients, compared LAVI with EF and found that LAVI was significantly raised in elderly patients who suffered from Acute MI.

ANALYSIS

Age:

The mean age of the subject in our study was 56.4 years,the mean age of the patients who developed heart failure(Killip>II) was 64.12±9.3 yrs and those without heart failure was 52.97±11 yrs which was statistically significant(p 0.001) indicating a higher incidence of heart failure with increasing age. In study by Lilian P Souza et al[3], the average age of patients who developed heart failure (Killip>II) was 65.8±14.2 yrs and in study by Monika Maheshwari et al[33], the average age of patients with heart failure was 65±3 yrs

Hypertension :

In our study, the number of patients with hypertension were 8,out of them 5 developed heart failure (Killip >II) and 3 patients did not. There was no significant association between HF and HTN, similar observations were made in study by Lilian P Souza et al[3] 49% of patients without HF had HTN and 62% had HTN among those with HF which was not significant with a p value 0.22.

Diabetes Mellitus :

The number of patients who had diabetes mellitus in our study was 28(45.6%), among them 18 developed in hospital heart failure(Killip>II) and 10 of them did not develop. It was statistically significant (p 0.001) suggesting a strong co relation between heart failure and diabetes mellitus. In study by Lilian P Souza et al[3] , they observed no significant association between diabetes mellitus and development of HF following Acute myocardial infarction,it was present in 18% of patients without HF and in 17% with HF. Peter H Stone et al[34], in their study observed a significant increase in heart failure in patients with Diabetes (in-hospital) post AMI, they concluded diabetics had more adverse outcomes compared to non diabetics. The

factors responsible for the increased incidence of adverse outcomes among diabetic patients may be related to an acceleration of the atherosclerotic process, diastolic left ventricular dysfunction associated with diabetic cardiomyopathy or other unidentified unfavorable processes[34] .

Site of infarction :

In our study, of 61 patients included 46 patients had involvement of anterior wall and 15 patients had inferior wall infarction, Of 46 patients with anterior wall infarction,24 patients developed HF(Killip >II) which was statistically significant(p 0.01), Lilian P Souza et al[3], in their study did not observe any such significance, 53% of patients with anterior wall infarction had no HF and 66% with anterior wall had HF, in their study with a study sample of 95 ,there were significant no of patients with inferior wall infarction 41 patients as compared to anterior wall 54 patients, where as in our study the total no of patients included were only 61 and and majority were anterior wall 75.4% in our study. Monika Maheshwari et al[33] ,in their study observed a significant association between increasing left atrial volume index and heart failure in patients with infarction of anterior wall, the study group included mainly patients with anterior wall infarction.

Among the echocardiographic variables, diastolic indices deceleration time, E/A ratio, Left ventricle end diastolic volume index,Left ventricle end systolic volume index and Diastolic patterns, Ejection fraction and Left atrial volume index were analyzed.

Deceleration time, in our study showed a significant association with a presence of heart failure (p value 0.002), mean DT in those with HF was 198±27.53 and those without HF was 169±27.81.In study by Lilian P Souza et al[3] there was no significant association between DT and heart failure(Killip >II) ,there was a tendency

of restrictive pattern (DT<140) to be associated with early HF (P-0.06) but it was not statistically significant, where as S.H. Poulsen et al[28] with a DT of <130ms,identified a risk of developing HF, where as in our study only two patients showed restrictive pattern with DT<130ms , the overall sample size included was less in our study compared to other studies.

E/A ratio was analyzed, a mean of 0.844 was observed in patients with HF Killip>II and a mean 1.08 in those with absent HF which was not statistically significant and did not show association with early in hospital HF, Similar result were observed in study by Lilian P Souza et al[3] with a mean of 1.02 in those with absent HF and 0.99 in those with HF , where as in study by S.H. Poulsen et al[28] , they observed a significant association between E/A >1.5(restrictive pattern) and HF, in their study they included patients with Killip>I as HF as compared to our study where we included Killip>II as HF.

LVESVI and LVEDVI were also analyzed no significant association was obsereved LVESVI was 35.4 with HF and 32.78 without HF which was not statistically significant (p 0.08) and LVEDVI was 78.81 with HF and 68.18 without HF (0.07), similar conclusion was made by Lilian P Souza et al[3] and S.H.Poulsen et al[28] in their studies.

Left atrial volume index was also analyzed, the mean LAVI in patients with HF Killip>II was 28.96ml/m^2 and those without HF was 22.55ml/m^2 which was statistically significant (p 0.001) suggesting a strong co relation of higher LAVI and in hospital HF. Lilian P Souza et al[3] did not observe any significant result between HF and LAVI, they attributed this to the short period follow up and less likely chance of LA remodeling to occur within 48 hours of initial presentation. Jacob E Moller et al[31] also analyzed the role of LAVI and other indices of diastolic and systolic function

after AMI in 314 patients, patients were also analyzed according to LAVI>32ml/m^2 and LAVI<32 ml/m^2, it was follow up study with large sample size and they concluded that LAVI is powerful predictor of mortality after AMI and provides significant information incremental to clinical data and conventional measures of LV systolic and diastolic function. Roy Beinart et al[32] in their study observed that left atrial volume index >32 ml/m^2 was found in 63 patients (19%) who had a higher incidence of heart failure on admission (24% vs. 12%, p -<0.01), a higher incidence of mitral regurgitation, increased LV dimensions, and reduced LV ejection fraction when compared with patients with LAVI <32 ml/m^2.They also analysed prediction of long term mortality of LAVI and their included large sample of 451 patients. Monika Maheshwari et al[33] found a strong correlation between LAVI and increasing LV dysfunction in elderly patients and those with anterior wall infarction, in the same study they observed a significant association between LAVI and Killip class with a p value<0.001, they mainly included control subjects who were elderly>70 years and study group were age and sex matched anterior wall infarction patients.

Ejection fraction was also analyzed and the mean in patients with HF present was 36.25% and in those with HF absent was 45%, which was statistically significant p <0.001,EF has been established as a powerful index of systolic function and also its role in predicting in hospital heart failure has been well established. Lilian P Souza et al[3] in their study concluded that EF<40% was powerful strongest and independent variable associated with development of HF Killip>II, it was superior to other indices of systolic and diastolic function. S.H.Poulsen et al[28] also observed that EF<45% was significant prognostic parameter in assessing LV function and predicting in hospital heart failure.

The variables were also compared in relation to cut-off values of LAVI as shown in table 5, the median value of LAVI was derived at 24ml/m^2 in our study. Diabetes mellitus was correlated with LAVI >24ml/m^2 and LAVI<24ml/m^2, it was found to be significant but not with hypertension. Anterior location of infarction was significantly associated with LAVI with p value (0.005), 25 patients of 46 had a LAVI >25ml/m^2.Grade I diastolic dysfunction was associated with significantly with a LAVI of>24ml/m^2 .In our study only 4 patients were found to have gradeII diastolic dysfunction and only patient had grade III diastolic dysfunction, owing to smaller sample size.

Jacob E Moller et al[31] in their study made a similar comparison with a cut-off value of LAVI of 32ml/m^2,they observed HTN, Diabetes mellitus were significantly associated with LAV>32ml/m^2,Anterior wall MI was not significantly associated unlike in our study, they observed association of grade III diastolic dysfunction with a value of LAVI >32ml/m^2 , they included patients with whole spectrum of ACS where as in our study only patients with STEMI were included. Roy Beinart et al [32] analyzed Left atrial volume index by dividing patients into two groups >32ml/m^2 and<32 ml/m^2 and found that in LAVI>32ml/m^2 was in 63 patients (19%). Compared with patients with LAVI <32 ml/m^2, these patients were older, had more prevelance of cerebrovascular attack or transient ischemic attack. They presented more frequently with heart failure (Killip score >II) on admission. There was no difference in the prevalence of hypertension, diabetes mellitus, and their study included whole spectrum of ACS patients.

The frequency and distribution of LAVI was studied in patients with and without HF. A LAVI of 32-39ml/m^2 predicted a 100% chance of developing in

hospital heart failure, a value between 25-32ml/m^2 predicted a 75% chance of developing in hospital heart failure as shown in table no.4

The odds ratio for LAVI to predict heart failure was used, the cut off value of 28ml/m^2 was derived, out of 14 patients with LAVI>28ml/m^2, 13 had HF(Killip>II) with a odds ratio of 37.91 which was highly significant(p 0.009).

THE ROLE OF ECHOCARDIOGRAPHY FOR DIAGNOSIS OF HEART FAILURE IN PRIMARY CARE

In a cross-sectional study performed in the United States, where 63% of HF patients in the community received an echocardiogram within three weeks of their symptomatic event, the patients who received an echo were less likely to be admitted to hospital, twice as likely to receive angiotensin converting enzyme inhibitors and had better five-year survival (adjusted for gender, age and NYHA class)[35].

In a recent review of five open access echo services in the United Kingdom it was found that 80% of the patients were referred with suspected HF, and approximately 20% of those patients had demonstrable systolic impairment[36].

Further, whilst it is easy to diagnose structural abnormalities or systolic impairment, it is more challenging to diagnose diastolic HF.

In addition to providing confirmation of diagnosis, physicians anticipate open access echocardiography will contribute to their treatment decisions and prognostication.

PROGNOSTIC VALUE OF ECHOCARDIOGRAPHY IN HEART FAILURE

Despite optimal medical therapy, mortality associated with congestive HF remains high. One-year mortality rates after the first hospitalisation for HF are approximately 30-40%. This prognosis is worse than many cancers[37].

Several clinical, functional and echocardiography parameters predict survival, including New York Heart Association (NYHA) classification, end-systolic volume[38], EF[39] creatinine clearance[40] and echo-Doppler indices of diastolic function[41]

Echocardiography may allow clinicians to determine which patients will fare worst, to identify those patients who may benefit the most from newer or more intensive treatments, or simply allow patients to better plan for their remaining.

SUMMARY

Sixty one patients with ST elevation myocardial infarction were studied in B.L.D.E.U's Shri B.M.Patil Medical College, Hospital and Research Center, Bijapur from a period of October 2011 to July 2013.

This study was conducted to analyze the left atrial volume compared to other conventional parameters of systolic and diastolic left ventricle function in patient with first ST elevation myocardial infarction, in predicting early heart failure during In-hospital evolution by echocardiography.

1. Of the 61 patients with ST elevation myocardial infarction studied ,25 patients developed in-hospital heart failure(Killip>II)
2. The mean age of the patients in our study was 58.5 yrs and mean age of patients who developed heart failure(Killip>II) was 64.12 yrs which was statistically significant
3. Risk factors like Hypertension was present in 5 patients who developed Heart Failure and was not significant where as Diabetes mellitus was present in 28 patients and 18 of them developed Heart Failure which was statistically significant.
4. Of 61 patients, 46 patients had involvement of anterior wall and of 46 patients 24 patients had Heart Failure which was highly significant.
5. Deceleration time was found to be significant with development of Heart failure (p 0.02), where as E/A ratio, Left ventricular end systolic volume ,left ventricular end diastolic volume were not found to be significant with Heart failure

6. Left atrial volume index was significantly associated with development of in-hospital heart failure, with a mean of 28.96ml/m^2 in patients with Killip>II compared to that of 22.58ml/m^2 in that patients with killlp <II
7. There was significant association between increasing Left atrial volume index and anterior location of infarction, 25 patients of 46 with anterior wall involvement had a left atrial volume index>25ml/m^2
8. There was no significant association between increasing value of Left atrial volume index and Hypertension and Diabetes mellitus
9. Ejection fraction showed a very significant association between with Heart failure (Killip >II) and was a strong predictor of in-hospital heart failure.

CONCLUSION

In our study of patients with STEMI, there was increase in incidence of Heart failure in elderly patients (>60yrs) and in diabetic individuals and **t**here was significant increase in incidence of Heart failure in patients with infarction of anterior wall**.** Ejection fraction<35% was significantly associated with increased incidence of Heart failure and a strong predictor of In-hospital Heart failure and left atrial volume index was also significant predictor of in-hospital Heart failure in our study and a Left atrial volume index>28ml/m^2 was associated with a high incidence of In-hospital Heart failure.

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ANNEXURE-I

ETHICAL CLEARANCE

BIJAPUR-586103
OUT Ward
No 27/2011
Date 21-10-11
Shri B. M. PATIL Med Coll

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 20-10-2011 *at* 10-30 am *to scrutinize the Synopsis/Research projects of postgraduate/undergraduate student/Faculty members of this college from Ethical Clearance point of view. After scrutiny the following original/corrected & revised version synopsis of the Thesis/Research project has been accorded Ethical Clearance.*

Title Echocardiographic predictors of early in - Hospital heart failure during first STelevation myocardial infarction,

Name of P.G./U.G. student/Faculty member Dr. Avinash.S. Alashetty
Dept of Medicine

Name of Guide/Co-investigator Dr. S.R. Badiger Prof of Medicine

DR.M.S.BIRADAR,
CHAIRMAN
INSTITUTIONAL ETHICAL COMMITTEE
BLDEU'S, SHRI.B.M.PATIL
MEDICAL COLLEGE, BIJAPUR.
Chairman
Ethical Committee
BLDEA'S Shri. B.M. Patil
Medical College
Bijapur-586103

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

ANNEXURE –II

INFORMED CONSENT FORM BLDEU'S SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, BIJAPUR- 586103

TITLE OF THE PROJECT ECHOCARDIOGRAPHIC PREDICTORS OF EARLY IN- HOSPITAL HEART FAILURE DURING FIRST ST ELEVATION ACUTE MYOCARDIAL INFARCTION

PRINCIPAL INVESTIGATOR - Dr. AVINASH S ALASHETTY

P.G.GUIDE NAME - Dr. BADIGER SHARANABASWAPPA
PROFESSOR OF MEDICINE

All aspects of this consent form are explained to the patient in the language understood by him/her.

I) **INFORMED PART**

1) PURPOSE OF RESEARCH:

I have been informed that this study echocardiographic predictors of early in-gospital failure during first ST elevation myocardial infarction. I have also been given a free choice of participation in this study.

2) PROCEDURE:

I am aware that in addition to routine care received I will be asked series of questions by the investigator. I have been asked to undergo the necessary investigations and treatment, which will help the investigator in this study

3) RISK AND DISCOMFORTS:

I understand that I may experience some pain and discomfort during the examination or during my treatment. This is mainly the result of my condition and the procedure of this study is not expected to exaggerate these feelings that are associated with the usual course of treatment.

4) BENEFITS:

I understand that my participation in this study will help to patients survival and better outcome.

5) CONFIDENTIALITY:

I understand that the medical information produced by this study will become a part of Hospital records and will be subject to the confidentiality and privacy regulation. Information of a sensitive personal nature will not be a part of the medical records, but will be stored in the investigator's research file and identified only by a code number. The code-key connecting name to numbers will be kept in a separate location.

If the data are used for publication in the medical literature or for teaching purpose, no name will be used and other identifiers such as photographs and audio or videotapes will be used only with my special written permission. I understand that I may see the photographs and videotapes and hear the audiotapes before giving this permission.

6) REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at anytime. Dr. AVINASH S ALASHETTY is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of the study, which might influence my continued participation.

If during the study, or later, I wish to discuss my participation in or concerns regarding this study with a person not directly involved, I am aware that the social worker of the hospital is available to talk with me. A copy of this consent form will be given to me to keep for careful reading.

7) REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and that I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice to my present or future care at this hospital. I also understand that Dr. AVINASH S ALASHETTY may terminate my participation in the study after she has explained the reasons for doing so and has helped arrange for my continued care by my own physician or physical therapist, if this is appropriate.

8) INJURY STATEMENT:

I understand that in the unlikely event of injury to me resulting directly from my participation in this study, if such injury were reported promptly, the appropriate treatment would be available to me, but no further compensation would be provided. I understand that by my agreement to participate in this study I am not waiving any of my legal rights.

I have explained to _____the purpose of the research, the procedures required and the possible risks and benefits to the best of my ability in patient's own language.

Dr. AVINASH S ALASHETTY Date

(Investigator)

II) **STUDY SUBJECT CONSENT STATEMENT:**

I confirm that Dr. AVINASH S ALASHETTY has explained to me the purpose of research, the study procedures that I will undergo, and the possible risks and discomforts as well as benefits that I may experience in my own language. I have read and I understand this consent form. Therefore, I agree to give consent to participate as a subject in this research project.

Participant / Guardian Date

Witness to signature Date

ANNEXURE–III

CASE PROFROMA

BLDE'S SHRI B.M.PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, BIJAPUR

"ECHOCARDIOGRAPHIC PREDICTORES OF EARLY IN HOSPITAL FAILURE DURING FIRST ST ELEVATION ACUTE MYOCARDIAL INFARCTION"

Name: CASE NO:

Age: IP NO:

Sex: DOA:

Religion: DOD:

Occupation:

Residence:

Presenting complaints with duration:

History of presenting complaints:

Past History:

History of hypertension

History of diabetes mellitus

Personal History:

Diet/appetite

Sleep

Bladder and bowel habits :

Smoking/Tobacco chewing/Snuff Inhalation

Duration

Number of cigarettes/beedis pack year smoked

Amount of tobacco chewed/snuff inhaled

Alcohol

Duration

Quantity/Frequency

Type

Family History:

History of suggestive of Ischemic Heart Disease/hypertension diabetis mellitus

Treatment History :

General Physical Examination

Pallor: present/absent

Icterus: present/absent

Clubbing: present/absent

Generalized Lymphadenopathy: present/absent

Built: Poor/Middle /Well

Nourishment: Poor / Middle / Well

Vitals

PR:

BP:

RR:

Temp:

SYSTEMIC EXAMINATION.

- Respiratory System
- Cardiovascular System
- Central Nervous System
- Per abdomen

INVESTIGATIONS

HAEMATOLOGY –

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

BIOCHEMISTRY–

Random blood sugar	(mg/dl)
Blood Urea	(mg/dl)
Serum creatinine	

URINE EXAMINATION -

Albumin	
Sugar	
Microscopy	

Lipid Profile:

Chest X Ray PA view:

12 lead ECG :

CPK-MB :

ECHOCARDIOGRAPHY:

1) LA volume
2) Ejection fraction
3) E/A ratio
4) Deceleration time of early diastolic filling(DT)
5) LVEF
6) LVEDV
7) LVESV

FINAL DIAGONOSIS :

ANNEXURE–IV

KEY TO MASTERCHART

DM - Diabetes Mellitus.

HTN - Hypertension

Killip - Killip Class For Heart Failure.

F - Female

M - Male

PE - Pulmonary Edema

N - Normal

R_x - Treatment

T - Thrombolysed

C - Conservative

ANT - Anterior

INF - Inferior

EF - Ejection Fraction

LAVI - Left Atrial Volume Index

LVESVI - Left Ventricle End Systolic Volume Index

LVEDVI - Left Ventricle End Diastolic Volume Index

DT - Deceleration Time

ml/m^2 - Milliliter Per Meter Square

m/s - Meter Per Second

MASTER CHART

Sl. No	Name	Age (yrs)	Sex	DM	HTN	Smoker	Killip	CPKMB	X-Ray	Rx	ECG Location	EF (%)	LAVI (ml/m^2)	LVESI (ml/m^2)	LVEDI (ml/m^2)	E/A	DT (m/s)	Disatolic
1	Tukaram Gadge	55	M	yes	yes	no	III	68	PE	T	Ant	30	38	31	68	0.6	230	I
2	Gurappa	66	M	no	yes	no	II	72	N	T	Ant	35	24	32	62	0.7	230	I
3	Chandrabai	48	F	no	yes	no	I	60	N	T	INF	50	19	33	65	1	190	0
4	Balu	42	M	yes	yes	no	I	58	N	T	INF	45	21	36	66	1	150	0
5	Irayya	56	M	no	no	yes	I	50	N	T	INF	50	21	30	48	1	190	0
6	Janabai	75	F	yes	yes	no	III	80	PE	T	Ant	45	25	36	68	1.5	150	II
7	Saibanu	75	M	yes	no	no	III	80	PE	T	Ant	40	26	37	68	1.5	130	III
8	Imamsab	36	M	no	no	no	I	50	N	T	Ant	45	21	37	69	1.1	170	0
9	Umesh	45	M	no	no	no	III	60	PE	T	Ant	30	26	38.2	72	0.7	240	I
10	Gurubai	68	F	yes	yes	no	III	72	PE	T	Ant	35	25.2	39	73	0.7	150	I
11	Huchappa	37	M	yes	no	yes	II	68	N	T	Ant	40	18.6	38	62	1.1	160	0

12	Bhumanna	64	M	no	no	no	III	80	PE	T	Ant	35	24.2	36	68	0.8	220	I
13	Parubai	74	F	no	no	no	III	80	PE	T	Ant	35	27.2	36	59	0.7	220	I
14	Roshanbee	65	F	no	no	no	II	72	N	C	INF	45	19.2	32	61	1.1	160	0
15	Ramchandra	72	M	no	no	no	III	68	N	R	Ant	40	28.1	31	64	0.8	220	I
16	Shantappa	43	M	no	no	no	I	48	N	R	Ant	45	18.6	28	59	1.1	150	0
17	Mallappa Palarai	48	M	no	no	no	II	62	N	R	Ant	45	20.1	31	60	1.1	158	0
18	Kasturibai	80	F	no	no	no	III	80	PE	C	Ant	35	24.2	32	61	1.1	180	0
19	Gundu	60	M	no	no	no	III	60	N	T	INF	35	24.1	28	52	1.5	140	II
20	Umesh	41	M	no	no	no	I	48	N	T	INF	50	19.1	29	59	1.1	166	0
21	Shantappa	55	M	yes	no	yes	II	68	N	T	Ant	35	25.2	33	66	0.7	210	I
22	Polaram	55	M	yes	no	yes	III	45	N	T	Ant	30	29	45	80	0.8	200	I
23	Siddaling	60	M	no	no	no	II	48	N	C	Ant	30	25	36	71	1.1	156	0
24	Saibanna	55	M	no	no	no	I	40	N	T	Ant	50	24.2	34	68	1.1	166	0
25	Shantappa	50	M	no	no	no	I	45	N	T	Ant	40	22	29	88	1.2	170	0

26	Tippanna	65	M	yes	no	yes	I	48	N	T	INF	45	21.2	31	68	1.1	150	0
27	Mohdsaab	55	M	no	no	no	I	54	N	T	Ant	40	21	34	70	1.1	160	0
28	Sanganna	42	M	no	no	no	I	60	N	T	Ant	45	23.2	38	72	1.1	160	0
29	Bujang	40	M	yes	no	no	I	44	N	T	Ant	55	21.2	31.6	71	1.1	160	0
30	Mallamma	66	F	yes	no	no	III	102	PE	T	Ant	40	29.6	36.2	79.2	0.9	200	I
31	Babu Pattar	60	M	yes	no	yes	III	80	PE	T	Ant	35	29.2	34	82.2	0.8	210	I
32	Sangamma	63	M	no	no	no	I	60	N	T	INF	45	21	26	62.2	1.1	160	0
33	Srikanth	62	M	yes	no	no	II	102	N	T	Ant	50	20	30	68.2	1.1	150	0
34	Kashibai	60	F	yes	yes	no	III	102	PE	T	Ant	40	28	26	74.2	0.8	200	I
35	Chandrawwa	65	F	no	no	no	II	60	N	T	Ant	30	31.2	41	80	0.7	220	I
36	Sangamesh	65	M	no	no	no	II	50	N	T	INF	40	29	30	78	0.6	230	I
37	Iranna	52	M	yes	no	no	II	40	N	T	INF	40	22	30	85.2	0.7	236	I
38	Chidambar	62	M	yes	no	yes	III	48	PE	T	Ant	35	32.2	40	76	1.5	150	II
39	Rajesh	52	M	yes	no	no	II	50	N	T	INF	50	23.6	34.2	64	1.1	180	0

40	Raju	40	M	no	no	no	I	40	N	T	INF	50	26	35	74	1.1	170	0
41	Sangamesh	65	M	no	no	no	I	48	N	T	INF	50	22.6	38	62	1.1	150	0
42	Vishwanath	53	M	no	no	no	I	40	N	T	Ant	50	22.2	28	70	1.1	160	0
43	Sayyed Hussain	65	M	yes	no	yes	III	68	PE	T	Ant	35	29.2	34.8	76	0.8	220	I
44	Rudragouda	75	M	yes	no	no	III	70	PE	T	Ant	30	32.2	39.2	86	1.1	140	II
45	Asagavli	40	M	yes	no	no	I	48	N	T	INF	55	24	33.5	70	1.1	160	0
46	Sangappa	77	M	no	no	no	II	40	N	T	Ant	45	24	31.5	68	0.8	220	I
47	Ramaling	60	M	no	yes	no	III	80	PE	C	Ant	30	31.2	38.2	77.8	0.7	210	I
48	Manappa	45	F	yes	no	no	III	60	PE	T	Ant	40	31	36.2	74	0.8	210	I
49	Renukabai	42	F	no	no	no	I	40	N	T	INF	55	23	33.4	70	0.8	220	I
50	Appasab	58	F	yes	no	no	III	60	PE	T	Ant	40	30.2	31.8	78	0.9	200	I
51	Revabai	70	F	no	no	no	II	48	N	T	Ant	35	26	36	80	1.1	164	0
52	Sangamesh	55	M	yes	no	yes	III	80	PE	T	Ant	40	31.2	36	70	0.8	210	I
53	Kamalawwa	70	F	no	no	no	II	40	N	T	Ant	40	28	32	68	1.2	160	0

54	Samabai	75	F	yes	no	no	III	100	PE	T	Ant	35	33	36	78	0.8	220	I
55	Krishaji	42	M	yes	no	no	I	60	N	T	Ant	50	24	32	70	1.2	150	0
56	Gangabai	48	F	no	no	no	I	40	N	T	Ant	50	21	33	68	1.1	150	0
57	Malathi Natwadi	44	F	no	no	no	I	60	N	T	Ant	45	20	31	64	1.2	140	0
58	Dvarkawwa	67	F	yes	no	no	III	60	PE	T	Ant	40	31.2	34	70	1.1	160	0
59	Md. Akil Sindgikar	72	M	yes	no	no	III	60	PE	T	Ant	35	30	36	71	0.6	210	I
60	Vasanth	53	M	no	no	no	I	40	N	T	Ant	50	21	33	68	1.2	150	0
61	Iranna	60	M	yes	no	no	III	60	PE	T	Ant	40	29	36	70	0.9	200	I