"LEFT ATRIAL VOLUME INDEX AS A PREDICTOR OF IN HOSPITAL EVENTS IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION" – BY 2D AND DOPPLER ECHOCARDIOGRAPHY

Dissertation submitted to

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CHENNAI, INDIA

CERTIFICATE

This is to certify that this Dissertation titled **"Left atrial volume index as a predictor of in hospital events in Patients with Acute Myocardial Infarction" – by 2D and Doppler Echocardiography** is a bonafide work done by **Dr. K. TAMILARASAN** Post Graduate Student (2008-2011) in the Department of Cardiology, Govt. Stanley Medical College, Chennai under the direct guidance and supervision and in partial fulfillment of the regulations laid down by the Tamilnadu Dr. M.G.R. Medical University, Chennai for DM Branch II, Cardiology Degree examination.

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I **Dr. K. TAMILARASAN** solemnly declare that this dissertation titled "**Left atrial volume index as a predictor of in hospital events in Patients with Acute Myocardial Infarction" – by 2D and Doppler Echocardiography** is a bonafide work done by me in the Department of Cardiology, Govt. Stanley Medical College and Hospital under the guidance and supervision of my Professor **Dr. G. KARTHIKEYAN,** M.D. D.M., Professor & HOD, Department of Cardiology, Govt. Stanley Medical College, Chennai – 600 001.

This Dissertation is submitted to the Tamilnadu Dr. M.G.R. Medical University, Chennai towards the partial fulfillment of the University regulations for the award of DM Branch II, cardiology degree examination.

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CONTENTS

Introduction

INTRODUCTION

Multiple Doppler echo-cardio graphic variables may be used to assess left ventricular (LV) diastolic function.[1-3] However, these variables reflect the beat-to-beat interaction of LV filling pressures and ventricular compliance, making them sensitive to rapid alternations in ventricular preload and afterload.[4] Because of opposing effects of preload and compliance on transmitral velocities, the mitral inflow pattern may appear normal (pseudonormal) despite abnormal filling pressures.[1-2] Despite these limitations, Doppler indices of diastolic function have been shown to predict morbidity and mortality in patients with acute myocardial infarction (AMI).[5-8] In particular, a restrictive diastolic filling pattern, characterized by an abbreviated mitral E-wave deceleration time, predicts a poor outcome.[6-8]

During ventricular diastole, the left atrium (LA) is directly exposed to LV pressures through the open mitral valve. LA size is therefore largely determined by the same factors that influence diastolic LV filling.[9-10] It is, however, a more stable indicator, reflecting the duration and severity of diastolic dysfunction.[11] Left atrial (LA) enlargement has been proposed as a barometer of diastolic burden and a predictor of common cardiovascular outcomes such as atrial fibrillation, stroke, congestive heart failure, and cardiovascular death. It has been shown that advancing age alone does not independently contribute to LA enlargement, and the impact of gender on LA volume can largely be accounted for by the differences in body

surface area between men and women. Therefore, enlargement of the left atrium reflects remodelling associated with pathophysiologic processes. For this reason, it is hypothesized that LA volume would predict long-term outcome after AMI and might be superior in this respect to conventional Doppler indices of diastolic function. There is strong evidence that left atrial (LA) enlargement, as determined by echocardiography, is a robust predictor of cardiovascular outcomes. Recently, it has been shown that LA volume provides a more accurate measure of LA size than conventional M-mode LA dimension [12]. To optimize the use of LA volume for risk stratification, an understanding of the physiologic determinants of LA size and the methods for accurate quantitation is pivotal. To address this, we performed a study of patients who had comprehensive assessment of LV systolic and diastolic function, including assessment of LA volume, early after AMI.

Coronary heart disease remains the number one cause of death in the country, for both men and women. The magnitude of these age-related conditions is expected to increase because of the burgeoning older population. Significant progress has been made in the evaluation and treatment of certain clinical risk factors for primary and secondary prevention of cardiovascular diseases. The value of echocardiographic assessment of these patients with coronary artery disease is of great value. Patients with STEMI and NSTEMIs had progressive LA enlargement with reductions in conduit and active emptying volumes, reflecting persistent left

ventricular diastolic dysfunction consequent to coronary artery disease and associated diabetes [13]. The measurement of LA volumes after STEMI and NSTEMI may be useful to monitor chronic diastolic dysfunction resulting from ischemic burden and the severity of coronary artery disease [13].

Review of literature

REVIEW OF LITERATURE

Doppler Echocardiographic Assessment of Diastolic Function

After an AMI, myocardial ischemia, cell necrosis, microvascular dysfunction, and regional wall motion abnormalities will influence the rate of active relaxation. In addition, interstitial edema, fibrocellular infiltration, and scar formation will directly affect LV chamber stiffness. Thus, abnormalities in LV filling are common in this setting.

Spectral Pulsed-Wave Doppler Echocardiography

The pulsed-wave Doppler technique allows assessment of flow velocities (<2 m/s) at a distinct spatial position, making the technique suitable for assessment of changes in inflow velocities across the mitral valve during diastole. With mitral valve opening, the early inflow velocity will be determined largely by ventricular suction and the pressure gradient between the LA and LV.[1, 2] This is followed by a steady decrease in inflow velocity, with a normal duration of 140 to 240 ms (early mitral deceleration time [DT]) (Figure 2). After a period of diastasis, atrial contraction will cause a new increase in inflow velocity less than that of the early inflow; thus, the ratio of early to atrial inflow velocities (E/A ratio) will usually be 1 to 1.5. If active relaxation is impaired, the early mitral inflow velocity will decrease, increasing the atrial contribution to filling, resulting in a reversal of the E/A ratio and a prolonged DT. This "impaired relaxation" pattern, indicative of grade 1 diastolic

dysfunction, is usually associated with normal LV filling pressure (Figure 3). With worsening of diastolic dysfunction, LA pressure increases, and the gradient between the LA and LV at mitral valve opening increases; hence, the velocity of early inflow will increase even though relaxation is impaired. Because of rapid equilibration, early ventricular filling is terminated abruptly, causing a shortening of the time period during which early filling occurs; hence, DT returns to normal.

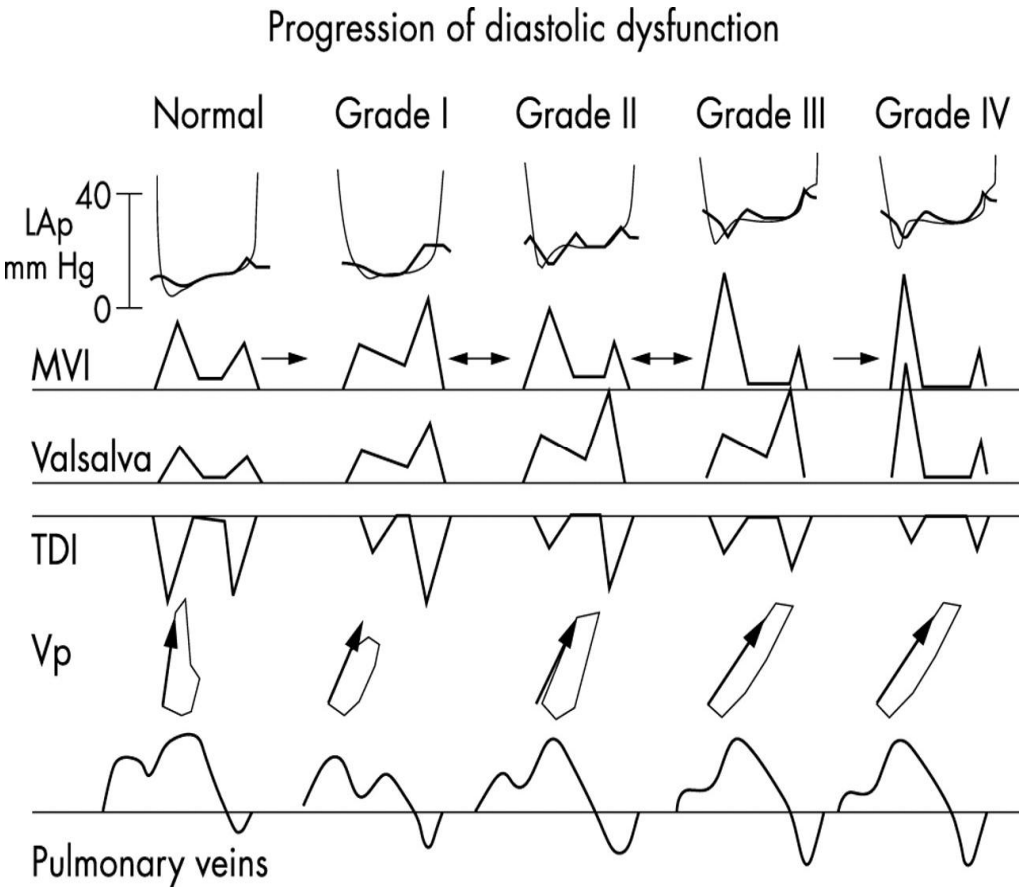


Figure 1

Therefore, the combination of delayed relaxation and elevated LA pressure may create an apparently normal transmitral inflow pattern that has been termed *pseudonormal* (grade 2 diastolic dysfunction) (Figure 1). With further deterioration, early filling will terminate abruptly because of the increase in LV stiffness. The DT will be abnormally short and the E/A ratio will be high, a pattern termed *restrictive* (grade 3 diastolic dysfunction) (Figure 3). The restrictive filling pattern can be subdivided further as *reversible*, if preload reduction, accomplished either by treatment or by the Valsalva maneuver, causes reversal of the filling pattern to the nonrestrictive pattern, or *irreversible*, if preload reduction causes no reversal of the filling pattern.[1, 2]

In patients with previous AMI, short DT (<140 ms) is associated with elevated LV filling pressures, even in the presence of atrial fibrillation and irrespective of the severity of mitral regurgitation. In contrast, DT >140 ms, especially in patients with preserved LV systolic function, correlates poorly with filling pressures. Although transmitral filling patterns are fundamental to the assessment of LV diastolic function, they have several limitations. They may change rapidly with variations in preload. Pseudonormalization of the inflow pattern despite moderate elevation of filling pressures is a further major shortcoming. To overcome this, less load-dependent indices of LV filling can be used, usually in combination with transmitral parameters. These may include assessment of the pulmonary venous flow pattern. This, however, is difficult to obtain in all patients and is greatly affected by heart rhythm. Thus, other

techniques have been developed. The most extensively validated of these are the determination of blood flow propagation within the LV with the use of color M-mode and tissue Doppler assessment of mitral annulus motion during diastole.

Color M-Mode Doppler Echocardiography

The color M-mode Doppler technique, performed in the apical 4-chamber view, reflects the distribution of blood velocities along a vertical line from the mitral plane to the apex of the LV. Color M-mode therefore provides spatiotemporal information on the propagation of blood into the LV (Figure 2). The slope of this early surge of blood into the LV has been termed *flow propagation velocity* (V_p), which is slowed when relaxation is impaired and, in contrast to the mitral E wave, remains reduced when LA pressure increases.V_p is also affected by LV geometry, intraventricular pressure gradients, and synchrony of wall relaxation.[3] Several studies have demonstrated a negative correlation between V_p and invasive measures of LV relaxation during myocardial ischemia and during both blockade and stimulation of ß-adrenergic receptors. Under physiological conditions, V_p has been demonstrated to be relatively preload independent.

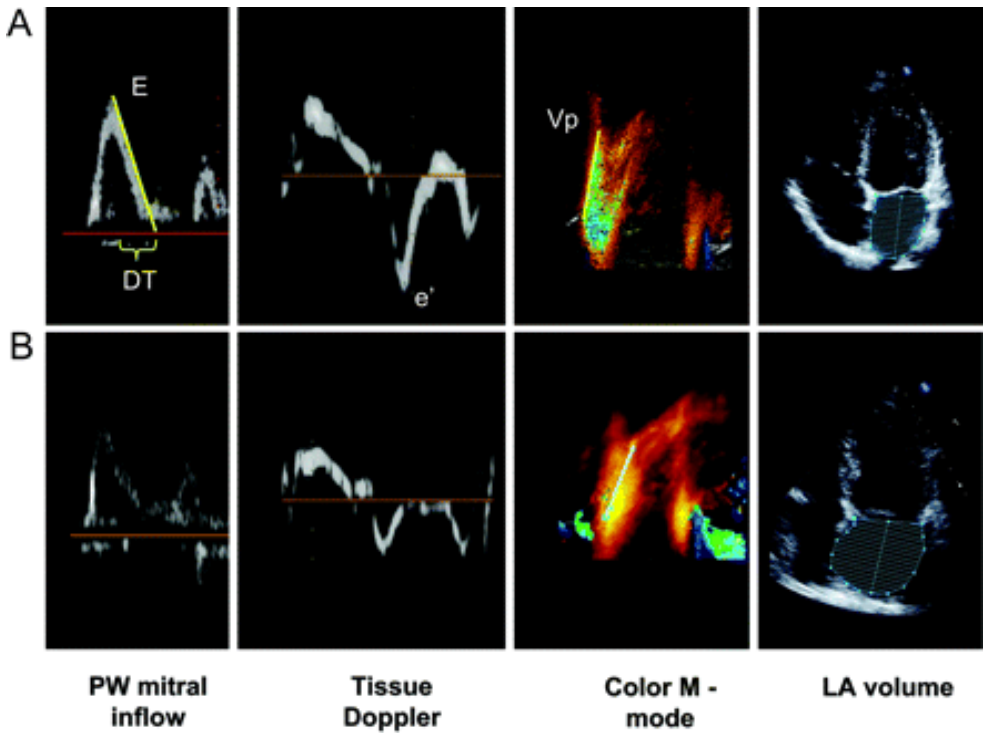


Figure 2

Based on this, V_p has been used in combination with peak mitral E-wave velocity to assess filling pressures and has proven useful in detecting a pseudonormalized LV filling pattern. The ratio of mitral E to V_p allows estimation of filling pressure during sinus rhythm or atrial fibrillation; E/V_p ratio >1.5 is suggestive of increased (>15 mm Hg) pulmonary capillary wedge pressure. Although useful in many situations, the assessment of LV filling with flow propagation has limitations. In ventricles with severe hypertrophy, V_p may appear normal because of enhanced intraventricular gradients despite delayed relaxation. In addition, several different methods for acquisition and analysis of color M-mode recordings have been used. In the majority of more recent studies, the method proposed by Garcia et al[3] has been adopted. According to

this method, the M-mode cursor is positioned in the center of LV inflow, avoiding boundary regions. V_p is measured as the first aliasing velocity (45 cm/s) from the mitral annulus in early diastole to 4 cm distally into the LV cavity. In patients with a low mitral E-wave velocity, baseline shift is adjusted to alias at ≈75% of the E-wave velocity. Even when this method is used, the interobserver variability may be as high as 10% to 20%, with the greatest variability for high (normal) values of V_p.

Spectral Pulsed-Wave Tissue Doppler Echocardiography

The motion of myocardium during the cardiac cycle can be displayed as a spectral pulsed-wave Doppler image, in which the signal will reflect the movement of myocardium parallel with the Doppler cursor. Because the apex of the LV is relatively fixed throughout the cardiac cycle and the motion of the LV base is nearly parallel with the long axis, assessment of the movement of the basal LV segments reflects the longitudinal vector of contraction and relaxation. Early diastolic mitral annulus velocity (e') is a useful indicator of LV relaxation (Figure 2). Invasive studies have demonstrated that e' correlates inversely with invasive indices of relaxation. In the presence of low (<0.1 m/s) velocities, e' is less affected by changes in preload and may be used to identify pseudonormal LV filling. Using the ratio of peak mitral E-wave velocity to early mitral annulus velocity (E/e'), numerous studies have demonstrated a good approximation of LV filling pressures. This relationship has been validated in the presence of atrial fibrillation, sinus tachycardia, preserved or

depressed LV systolic function, secondary mitral regurgitation, and LV hypertrophy. Ommen et al demonstrated that E/e' >15 accurately detects elevated filling pressures, and E/e' <8 accurately detects normal LV filling pressures. However, because the Doppler method tracks the velocity of movement, tissue Doppler cannot separate active contraction from passive tethering. Annular velocities vary depending on the location sampled, with the velocity of the lateral annulus usually higher than that of the septal annulus. This has led to controversy about which site should be used. Local myocardial damage may affect the mitral annular velocity, which may be a theoretical disadvantage of this measurement in AMI.

Tissue Doppler or Color M-Mode for Assessment of LV Filling

Although different in methodology, both tissue Doppler and color M-mode are relatively preload insensitive, allow estimation of filling pressures with reasonable accuracy, and facilitate identification of the pseudonormal LV filling pattern. In patients with small LV cavities due to hypertrophy, tissue Doppler is preferred because of pseudonormalization of V_p. Although V_p has a good reproducibility for distinguishing normal from abnormal, the reproducibility of e' is superior. In assessment of filling pressures and detection of pseudonormal LV filling, most studies but not all that have compared the techniques have favored E/e'. Thus, the better reproducibility and lesser dependence on LV geometry make tissue Doppler echocardiography e' measurement the preferred technique.

LA Phasic Function and Size

The LA mechanical function can be described broadly by three phases within the cardiac cycle[14] (figure 3). First, during ventricular systole and isovolumic relaxation, the LA functions as a "reservoir" that receives blood from pulmonary venous return and stores energy in the form of pressure. Second, during the early phase of ventricular diastole, the LA operates as a "conduit" for transfer of blood into the left ventricle (LV) after mitral valve opening via a pressure gradient, and through which blood flows passively from the pulmonary veins into the left ventricle during LV diastasis. Third, the "contractile" function of the LA normally serves to augment the LV stroke volume by approximately 20%[15]. The relative contribution of this "booster pump" function becomes more dominant in the setting of LV dysfunction[16,17].

The size of the LA varies during the cardiac cycle[18-22]. Generally, only maximum LA size is routinely measured in clinical practice. However, various LA volumes[19-22] can be used to describe LA phasic function:

1. Maximum LA volume occurs just before mitral valve opening.

2. Minimum LA volume occurs at mitral valve closure.

3. Total LA emptying volume is an estimate of reservoir volume, which is calculated as the difference between maximum and minimum LA volumes.

4. LA passive emptying volume is calculated as the difference between maximal LA volume and the LA volume preceding atrial contraction (at the onset of the P-wave on electrocardiography).

5. LA active emptying (contractile) volume is calculated as the difference between pre-atrial contraction LA volume and minimum LA volume.

6. LA (passive) conduit volume is calculated as the difference between LV stroke volume and the total LA emptying volume.

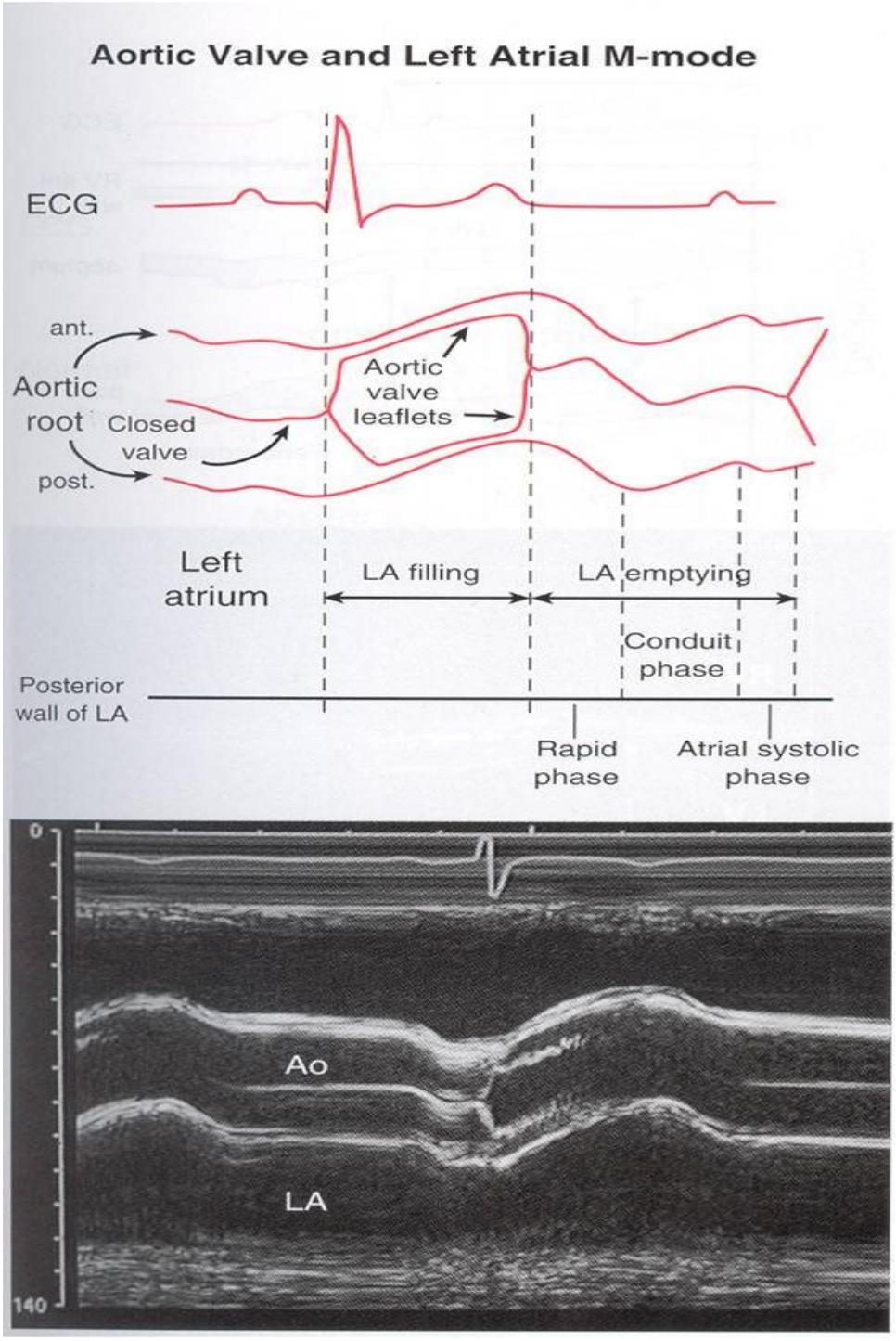


Figure 3

The LA mechanical function described broadly by three phases within the cardiac cycle and measurement of anteroposterior LA linear dimension by M-mode echocardiography

The relative contribution of LA phasic function to LV filling is dependent upon the LV diastolic properties[23] and therefore varies with age[19]. In subjects with normal diastolic function, the relative contribution of the reservoir, conduit, and contractile function of the LA to the filling of the LV is approximately 40%, 35%, and 25%, respectively[23]. With abnormal LV relaxation, the relative contribution of LA reservoir and contractile function increases and conduit function decreases. However, as LV filling pressure progressively increases with advancing diastolic dysfunction, the LA serves predominantly as a conduit[23].

Assessment of LA Size and Function

Two-dimensional and Doppler methods have been used increasingly for the assessment of LA size and function, respectively.

LA size assessment. Measurement of anteroposterior LA linear dimension by M-mode echocardiography[24-25] (Figure 3) is simple and convenient but not reliably accurate, given that the LA is not a symmetrically shaped three-dimensional (3D) structure[26]. Furthermore, because LA enlargement may not occur in a uniform fashion[27], one-dimensional assessment is likely to be an insensitive assessment of any change in LA size. In contrast to LA dimension, LA volume by two-dimensional (2D) or 3D echocardiography provides a more accurate and reproducible estimate of LA size, when compared with reference standards such as magnetic resonance imaging (MRI) and cine computerized tomography (CT)[28-31], and has a stronger association with cardiovascular outcomes[13,32,33]. Accordingly, the American Society of Echocardiography has recommended quantification of LA size by biplane 2D

echocardiography using either the method of discs (by Simpson's rule) or the area-length method[14] (Figure 4). Although we have routinely used the area-length method in our laboratory, we have found that the biplane Simpson's method is comparable in accuracy and reproducibility. Critical elements and common pitfalls for accurate and reproducible measurement of biplane LA volume assessment are detailed and outlined in Table 1. The Biplane area length method requires measuring LA area from two orthogonal apical views (A1 and A2) and LA length (L), from which LA volume is calculated as (0.85 x A1 x A2)/L (Figure 4). When LA length is measured from two apical views, the shorter value is used to calculate LA volume.

Echocardiographic methods systematically underestimate LA volume when compared with CT[34] or MRI quantitation[30], which in turn underestimates true LA size[22]. More recently, magnetic electroanatomic mapping has also been used for assessment of LA volume[35]. However, because of its portability and safety, echocardiographic assessment of LA volume is preferable to other imaging methods in clinical practice.

LA volume reference limits. Reference values for 2D Echocardiographic maximum LA volumes have been estimated using data collected on persons free of cardiovascular disease, although few samples have been population based[32,36]. Published reference values for maximum and minimum LA volumes are 22 ± 6 ml/m^2 [(37)] and 9 ± 4 ml/m^2 [(38)], respectively. In a study of LA function, mean total LA emptying volume was 13.5 ± 4.3 ml/m^2 (representing 37 ± 13% of LV stroke volume), fractional emptying of the LA was 65 ± 9%, and conduit volume was 23 ± 8 ml/m^2 [(39)].

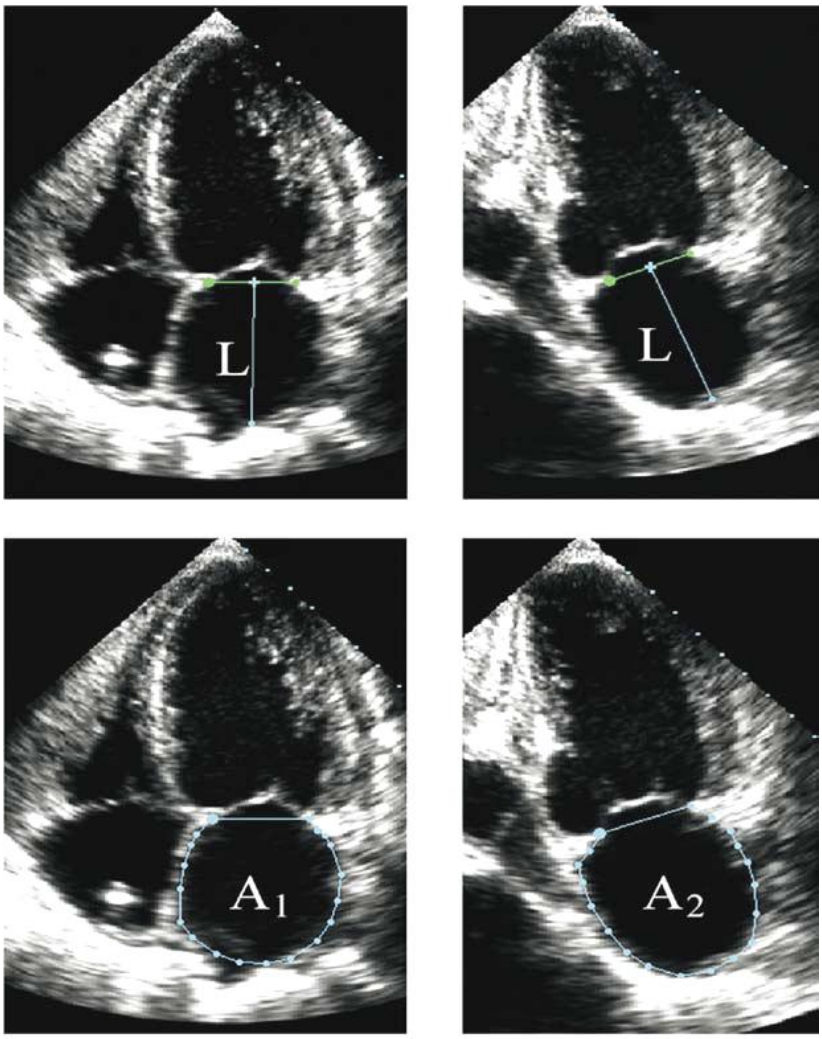

L
L
A1
A2
A4C
A2C
Left Atrial
Volume =
8/3π[(A1)(A2)/(L)] *
* (L) is the shortest
of either the A4C
or A2C length

Figure 4

Table 1 Critical elements and common pitfalls for accurate and reproducible measurement of biplane area- length method LA volume assessment

Step	Common Limitations/Errors	Suggestions
A. Optimize LA image quality	Atria are located in the far field of the apical views. Reduction of lateral resolution may result in apparently thicker LA walls.	Not improved by modifying the gain settings: Increase in gain will further reduce LA lumen size.Decrease in gain may lead to image "drop out" and difficulties in planimetry of LA area. Use high resolution sample box to increase pixel density and facilitate accurate tracing of the endocardial border. Capture at least five beats for each cine loop to maximize likelihood of obtaining adequate image quality.
B. Obtain maximal LA size	LA is foreshortened	Modify transducer angulation or location (place the transducer one intercostal space lower) until LA image is optimized and not foreshortened. If discrepancy in the two lengths measured from the orthogonal planes is >5 mm, acquisition should be repeated until the discrepancy is reduced.
C. Timing of maximum LA size	Correct frame for measurement is not selected	Choose frame just before mitral valve opening

Step	Common Limitations/Errors	Suggestions
D. LA area planimetry	LA border is inconsistently defined	Consistently adhere to convention: Inferior LA border—plane of mitral annulus (not the tip of leaflets). Exclude atrial appendage and confluences of pulmonary veins
E. Long-axis LA length	LA long axis is inconsistently delineated	Consistently adhere to convention: Inferior margin—midpoint of mitral annulus plane. Superior (posterior) margin—midpoint of posterior LA wall
F. Interpretation	Qualitative categorization of LA size	LA volume indexed to BSA is optimally interpreted as a continuous variable (using a reference point of 22 ± 5 ml/m^2 as "normal")

Assessment of LA functions by echocardiography. Pulsed-wave Doppler evaluation of transmitral and pulmonary venous blood flow velocity can be used for assessment of LA function, in addition to its widespread use for the evaluation of LV diastolic function and filling pressure[40-42]. The normal pulmonary venous flow pattern reflects flow from the pulmonary veins to the LA during early ventricular systole (PV_{s1}; seen distinctly in about 30% of transthoracic echocardiography studies[43]), late ventricular systole and isovolumic relaxation (PV_{s2}), early ventricular diastole (PV_d), and reversal of flow from the left atrium to pulmonary veins during atrial systole (PV_{ar}).

Apart from flow in late ventricular systole (reflected by PV_{s2}), which represents propagation of the right ventricular pressure pulse through the pulmonary circulation[44], blood flow in the pulmonary veins is determined by events that regulate phasic LA pressure[45]. The magnitude and velocity-time integral of the PV_s waves reflect LA reservoir function and are determined by LV systolic function and LA relaxation (PV_{s1}), LA compliance (PV_{s1} and PV_{s2}), and right ventricular stroke volume (PV_{s2})[44]. Peak velocity and velocity-time integral of PV_d is an index of LA conduit function[46] and is dependent on factors that influence LA afterload: LV relaxation and early filling[23] and mechanical obstruction from the mitral valve apparatus[47]. During LA contraction, blood is ejected from the LA into the LV and the pulmonary veins. Thus, assessment of transmitral (peak A-wave velocity, A-wave velocity-time integral, and atrial filling fraction)[17,48] and pulmonary venous blood flow (PV_{ar})[49] provides additive information for the evaluation of LA booster pump function.

More recently, global and regional atrial contractile function has been evaluated with pulse wave and color tissue Doppler imaging [19], but the incremental clinical utility of this assessment remains to be determined. Further, new echocardiographic techniques, such as with automated border detection using acoustic quantification, are being developed to facilitate evaluation of LA size and function[19].

Determinants of LA Size and Remodeling

Demographic and anthropometric influences: Body size is a major determinant of LA size. To adjust for this influence, LA size should be indexed to a measure of body size, most commonly to body surface area[32,36]. It remains to be clarified if this approach attenuates obesity-related variations in LA volume, which may be prognostically significant[50]. Gender differences in LA size are nearly completely accounted for by variation in body size[19,32,51,52]. In persons free of cardiovascular disease, indexed LA volume is independent of age from childhood onward[53]. Indeed, age-related LA enlargement is a reflection of the pathophysiologic perturbations that often accompany advancing age rather than a consequence of chronologic aging[20]. The relation of LA size to race or ethnicity has not been sufficiently studied.

Atrial structural remodelling: Many conditions are associated with LA remodelling and dilatation. The atria will enlarge in response to two broad conditions: pressure and volume overload. The relationship between increased LA size and increased filling pressures has been validated against invasive measures in subjects with[54,55] and without[41,56] mitral valve disease. Left atrial enlargement due to pressure overload is usually secondary to increased LA afterload, in the setting of mitral valve disease or LV dysfunction. Case reports have suggested that LA dilatation can also occur in response to pressure overload resulting from fibrosis and/or calcification of the LA. This condition, known as "stiff LA syndrome"[57,58], causes a reduction of LA compliance, a

marked increase in LA and pulmonary pressures, and right heart failure. Chronic volume overload associated with conditions such as valvular regurgitation, arteriovenous fistulas, and high output states including chronic anemia and athletic heart[59,60] can also contribute to generalized chamber enlargement. Both volume and pressure overload can increase atrial size. However, pressure overload is uniformly accompanied by abnormal myocyte relaxation, while volume overload is characteristically associated with normal myocardial relaxation physiology.

LA volume as an expression of LV filling pressures. In subjects without primary atrial pathology or congenital heart or mitral valve disease, increased LA volume usually reflects elevated ventricular filling pressures. During ventricular diastole, the LA is exposed to the pressures of the LV. With increased stiffness or noncompliance of the LV, LA pressure rises to maintain adequate LV filling[61], and the increased atrial wall tension leads to chamber dilatation and stretch of the atrial myocardium. Thus, LA volume increases with severity of diastolic dysfunction[33,62]. The structural changes of the LA may express the chronicity of exposure to abnormal filling pressures[33,56] and provide predictive information beyond that of diastolic function grade[63], which is determined from evaluating multiple load-dependent parameters and therefore reflective of the instantaneous LV diastolic function and filling pressures. In this way, analogous to the relationship between hemoglobin A_{1C} and random glucose levels, LA volume reflects an average effect of LV filling

pressures over time, rather than an instantaneous measurement at the time of study[64]. Thus, Doppler and tissue Doppler assessment of instantaneous filling pressure is better suited for monitoring hemodynamic status in the short term, whereas LA volume is useful for monitoring long-term hemodynamic control.

Left atrial size as an expression of diastolic function and filling pressures has not been fully evaluated in specific conditions. Most studies of LA size and outcomes have excluded patients with atrial fibrillation (AF). The relationship between AF and LA volume is complex[65]. It has been difficult to establish the causal relationship between AF and LA structural remodeling. In patients with AF and cardiac disease, structural LA alterations may be related to the underlying cardiac pathophysiology rather than solely the arrhythmia itself[66, 67]. Experimental animal studies have documented that sustained atrial tachyarrhythmias induce electrical, contractile and structural remodeling[68]. In some cases, it appears that LA structural remodeling may be related to high ventricular rate and increased ventricular filling pressures rather than to the atrial tachyarrhythmia itself[69,70]. However, in other individuals, the size of the LA varies widely for given LV relaxation and filling properties, suggesting a hysteresis between LA sizes and filling pressures. Few studies have assessed the impact of sustained AF on atrial structure in patients with lone AF[71].

LA Volume as a Marker of Diastolic Dysfunction

The LA acts as a conduit between the pulmonary vascular bed and the LV, receiving blood from the pulmonary veins and conveying it to the LV through passive and active filling. In addition, the atrium acts as an efficient volume sensor, releasing natriuretic peptides and other neurohormones to the circulation as a consequence of increased atrial wall stress. After opening of the mitral valve, the LA and LV diastolic pressures will rapidly equalize, and emptying of the LA will be determined largely by LV filling dynamics.[72,73] Thus, when the LA empties against a noncompliant LV and/or there is an increase in LV end-diastolic pressure, LA pressure will rise. This is poorly tolerated by the thin wall of the LA, and subsequent dilation will occur.[73] Chronic LA pressure overload will cause reduced myocardial energy production, alterations in contractile proteins, and myocyte atrophy, which eventually will cause LA wall fibrosis. Thus, with chronic distension there is little elastic recoil in the LA, and a chronically enlarged atrium will be relatively unaffected by transient changes in LA pressure.[73,74] Because of this relative insensitivity to transient changes in filling pressures, LA size can be considered a biomarker of sustained elevations in LV filling pressures.

With the use of echocardiography, LA size has traditionally been estimated with M-mode measurements obtained in the parasternal long-axis view, reflecting the anteroposterior dimension of the LA. However, the LA does not dilate symmetrically because of physical restraint.[75] Thus, with

expansion of the LA, the anteroposterior dimension by M-mode will underestimate the true volume.[75] With the use of planimetry performed in the apical window, the LA volume may be assessed by either single or biplane methods, with high reproducibility and good correlation with volumetric assessment with the use of magnetic resonance and 3D-cine computed ventriculography.[76-78] Compared with magnetic resonance, echocardiographic measurement of LA volume results in a slight underestimation.[79] This is less important when echocardiographic reference ranges are used. These are indexed to the body surface area of the patients, and the normal upper limit (mean +2 SD) of echocardiographically determined LA volume index has been determined to be 32 mL/m^2.[80]

Relation between LA Size and Prognosis after AMI

Two recent studies have investigated the relation between LA dilatation and all-cause mortality after AMI.[85,92] In a retrospective design including 314 patients, an increase >32 mL/m^2 in LA volume index was associated with a high all-cause mortality rate. In multivariate analysis, LA volume, Killip class, and a restrictive transmitral filling pattern were independent predictors of death, whereas LVEF or wall motion score index did not provide any additional prognostic information. A striking finding was that among patients with LVEF <40%, the LA appeared normal in size in one third of patients (27 of 82). One death occurred in this group as opposed to 22 deaths among 55 patients with LVEF <40% and LA enlargement. In addition, the prognostic importance of

LA volume was unrelated to the presence and severity of mitral regurgitation and atrial fibrillation. This finding has subsequently been confirmed by Beinart et al[85] in a prospective study of 395 patients with AMI in which multivariate analysis also identified restrictive filling, Killip class, and LA volume as independent predictors of adverse outcome.

Why Do Patients With Abnormal LV Filling/Enlarged LA Have a Poor Prognosis?

Consistently, irrespective of the method of assessment, it is evident that if there are direct or indirect signs of increased LV filling pressures, the risk of death is increased. Although the prevalence and severity of filling abnormalities are associated with the severity of systolic dysfunction, a considerable proportion of patients present with Doppler signs of elevated filling pressures despite only mildly reduced LVEF. The reason why these patients poorly tolerate what appears to be a relatively small myocardial injury is incompletely understood. These patients are older and more likely have a history of hypertension and diabetes compared with patients with no signs of elevated filling pressures. They also have evidence of more generalized overt atherosclerotic disease. The progression of cardiovascular disease can be regarded as a continuum of events in which the presence of risk factors such as hypertension, diabetes, and dyslipidemia predisposes to the development of atherosclerosis, LV hypertrophy, and eventually overt coronary artery disease and cardiac failure.[102] LA volume has been shown to correlate positively with

age and clinical cardiovascular risk score and negatively with LVEF.[80] We speculate that patients with increased LV filling pressures immediately after AMI have an increased burden of risk and poorly tolerate an acute loss of even relatively small amounts of myocardium. This is supported by the fact that a considerable number of patients, even when evaluated during the first 24 hours of AMI, present with LA enlargement. Based on the physiology of the LA, it would not be anticipated that acute elevation of filling pressures within hours can cause LA dilatation. This suggests that even before AMI, some patients had abnormal LV filling and possibly abnormalities in chamber stiffness and active relaxation with subsequent poor adaptation to the hemodynamic changes during acute myocardial ischemia.

LV pressure overload will cause myocyte stretch, increased wall stress, poorer subendocardial perfusion, and reduced energy production. These in turn are associated with neurohormonal activation and ventricular remodeling. Although the remodeling process will initially restore stroke volume and systemic hemodynamics, continuing dilation will have a detrimental effect on long-term LV function and survival. Previous studies of unselected patients with AMI,[91,103,104] patients with preserved systolic function,[105] and patients with ST-segment elevation AMI treated with fibrinolysis[84] or successful primary angioplasty[106] have demonstrated that a restrictive filling pattern in the early postinfarction phase predicts LV remodeling, defined as a dilatation (>20%) of the LV end-diastolic volume. This provides an important link to long-term prognosis.

LA Size for the Prediction of Cardiovascular Outcomes

There is considerable data confirming the relationship between increased LA size, principally maximal but also minimal [41,109], and the development of adverse cardiovascular outcomes in subjects without a history of AF or significant valvular disease.[110-123]

AF: Atrial fibrillation is the most common of the serious cardiac arrhythmias and is associated with increased morbidity and mortality in the community. Prospective data from the large population-based studies have established a relationship between M-mode anteroposterior LA diameter and the risk of developing AF.[124,125] In the Framingham study, a 5-mm incremental increase in anteroposterior LA diameter was associated with a 39% increased risk for subsequent development of AF.[124] In the Cardiovascular Health Study, subjects in sinus rhythm with an anteroposterior LA diameter >5.0 cm had approximately four times the risk of developing AF during the subsequent period of surveillance.[125] More recently, LA volume has been shown to predict AF in patients with cardiomyopathy[116,117] and first-diagnosed nonvalvular AF in a random sample of elderly Olmsted County residents who had undergone investigation with a clinically indicated echocardiogram.[110,111] The relationship between LA volume and LA dimension was nonlinear,[116] and it has been confirmed that LA volume represented a superior measure over LA diameter for predicting outcomes including AF[110,116,123] and provided prognostic information that was incremental to clinical risk factors.[110]

Stroke: Stroke is the leading cause of severe long-term disability and the third largest contributor to mortality in the U.S.[126] Despite the strong association between AF and ischemic stroke, 85% of strokes occur in patients who are in apparent sinus rhythm.[126] In the general population, LA size has been determined to be a predictor of stroke and death.[127] Increased LA volume has also been shown to predict the onset of first stroke in clinic-based elderly persons who were in sinus rhythm and did not have a history of ischemic neurologic events, AF, or valvular heart disease.[118] Even after censoring for the development of documented AF, an indexed LA volume >32 ml/m^2 was associated with an increased stroke risk (hazard ratio [HR] 1.67, 95% confidence interval [CI] 1.08 to 2.58) over 4.3 ± 2.7 years, independent of age and other clinical risk factors for cerebrovascular disease.

Heart failure: As previously discussed, LA volume is a barometer of LV filling pressure and reflects the burden of diastolic dysfunction in subjects without AF or significant valvular disease[33]. Elevation of filling pressure is uniformly found in the presence of symptomatic congestive heart failure (CHF). Because the majority of individuals in the community with LV dysfunction (systolic or "isolated" diastolic) are in a preclinical phase of the disease,[128] methods to quantify the risk of progression to symptomatic heart failure would be clinically useful. Evidence for a prognostic role for LA volume to predict incident CHF is emerging.[121,122] In a large prospective, population-based study, subjects with incident CHF during follow-up had slightly higher

baseline LA linear diameters (39 mm vs. 37 mm for women [$p < 0.01$], 41 mm vs. 39 mm for men [$p < 0.01$]).[129] In a study of older adults referred for echocardiography, LA volume >32 ml/m^2 was associated with increased incidence of CHF, independent of age, myocardial infarction, diabetes mellitus, hypertension, LV hypertrophy, and mitral inflow velocities (HR 1.97, 95% CI 1.4 to 2.7).[121] Furthermore, in subjects with an LV ejection fraction >50% at baseline and within four weeks of incident CHF, there was an increase of 8 ml/m^2 in LA volume from baseline to CHF diagnosis, reflecting the added burden of diastolic dysfunction during the period of transition from preclinical to clinical status.

Mortality: The relationship between LA size and death has been demonstrated in high-risk groups, such as patients with dilated cardiomyopathy,[112] LV dysfunction,[130] atrial arrhythmias,[131] acute myocardial infarction,[114,119] and patients undergoing valve replacement for aortic stenosis[132] and mitral regurgitation[133]. The LA diameter has also been shown to independently predict death in the general population[129]. However, in other population-based studies, the relationship between LA size and death has been attenuated when LV mass[127], LV hypertrophy[134], or diastolic function[62] has been considered. Thus, owing to the intimate relationship between LA volume, LV mass/hypertrophy, and diastolic dysfunction, the incremental value of each parameter for the prediction of death is diminished when considering the others.

Although a dilated LA is associated with a number of adverse outcomes, there is increasing evidence suggesting that LA size is potentially modifiable with medical therapy,[135-144] but whether LA size reduction translates to improved outcomes remains to be established.

Correlation of LA Size with the of severity of coronary artery disease

Patients with STEMI and NSTEMIs had progressive LA enlargement with reductions in conduit and active emptying volumes, reflecting persistent left ventricular diastolic dysfunction consequent to coronary artery disease and associated diabetes. The measurement of LA volumes after STEMI and NSTEMI may be useful to monitor chronic diastolic dysfunction resulting from ischemic burden[13] and the severity of coronary artery disease[13].

LA maximum volume was significantly larger at baseline in patients with NSTEMIs. At 12 months, maximum LA volume increased (27.6 ± 7.4 vs 30.2 ± 8.9 mL/m2, $P = .002$), with LA remodeling present in 64% of the patients with NSTEMIs. LA passive emptying volume increased, with concurrent reductions in conduit and active emptying volumes. Although diabetes, **major coronary artery disease**, and a larger myocardial score were predictive of LA remodelling, E′ velocity was the only independent predictor[13].

How to Treat Abnormal LV Filling

A major unresolved question is how to manage optimally patients with abnormal LV filling especially if LVEF is normal or only mildly reduced. To date, no interventional trial has been undertaken with hard end points in which

patient selection has been based on abnormalities in LV filling. However, assessment of the inflow pattern and E/e' ratio may provide important information on the hemodynamic status and guide the use of vasodilators and diuretics. In addition, previous randomized data have demonstrated that attenuation of the renin-angiotensin-aldosterone system with captopril in patients with mildly to moderately depressed LVEF after AMI is associated with a major improvement in central hemodynamics (LV end-diastolic and pulmonary artery pressure), whereas the improvement in LVEF is modest.[108] Likewise, small studies have demonstrated improvements in LV filling on intervention with ß-blockers after AMI, which was associated with improved exercise capacity.[108] However, although this reduction in LV filling pressure would be anticipated to improve functional status, it is not known whether this is associated with a better outcome.

Conclusions

Left atrial enlargement carries important clinical and prognostic implications. Left atrial volume is superior to LA diameter as a measure of LA size, and should be incorporated into routine clinical evaluation. Future studies are warranted to further our understanding of the natural history of LA remodelling, the extent of reversibility of LA enlargement with medical therapy, and the impact of such changes on outcomes. The utility of LA volume and function for monitoring cardiovascular risk and for guiding therapy is an evolving science and may prove to have a very important public health impact.

Aim of the study

AIM OF THE STUDY

1. To evaluate the role of left atrial volume index as a predictor of In- hospital events in patients with acute myocardial infarction by two Dimensional echocardiography and Doppler.

2. To assess the role of LA volume index as a prognostic tool, incremental to the standard Echocardiographic predictors of outcome, including LV systolic function (EF) and Doppler assessment of diastolic function.

Materials and methods

MATERIALS AND METHODS

1) Patient Selection:

This study was carried out in the period of February 2010 to February 2011 in the department of cardiology, Govt. Stanley Medical College Hospital, Chennai.

110 consecutive Patients presenting to ICCU with First episode of Acute myocardial Infarction (STEMI) were enrolled in this study after Excluding Patients based on Exclusion Criteria. The Diagnosis of Myocardial Infarction was based on following 3 criteria.

1. Typical chest pain
2. ECG changes suggestive of STEMI
3. Elevated cardiac enzymes

Exclusion criteria

The following groups of Patients were excluded from the study

- Patients with previous history of myocardial infarction
- Unstable angina / NSTEMI
- Previous history of Left ventricular dysfunction
- Previous history of Percutaneous coronary intervention

- Previous history of coronary artery bypass graft
- Previous history of valve diseases or arrhythmias such as atrial fibrillation.
- Cardiomyopathies
- Pericardial diseases

2) Ethical issues:

Since this study involve investigations, blood tests, certain life saving interventions, medications which could alter the outcome, all patients and their relatives were briefed about the study design at the time of enrollment. Contact details were established for further communication as and when necessary.

3) Study design

This is prospective observational study of all Newly diagnosed First episode of acute myocardial infarction Patients.

4) Data collection

Following Information was obtained at the time of Admission

1. Detailed history recording
2. Thorough physical examination
3. Blood samples for relevant blood investigations
4. Serial ECG's and complete Echocardiogram was done for all patients.

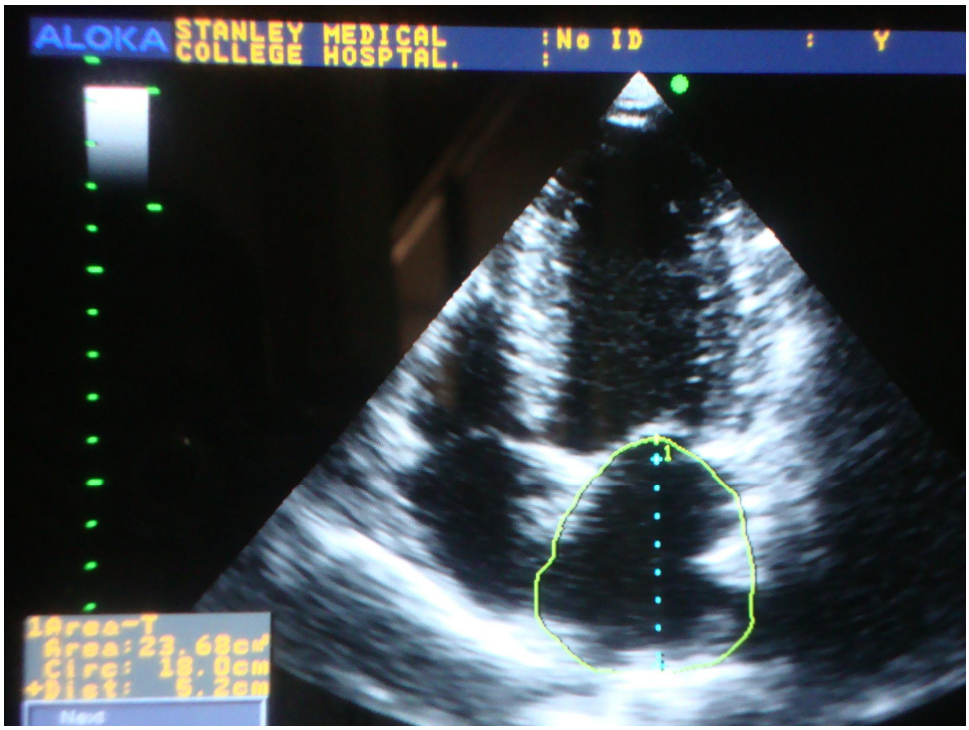
5) Echocardiography:

Echocardiography was performed on a median of 1 day (range 0 to 4 days) after admission using Aloka SSD 4000 Phased Array System equipped with Tissue Doppler and Harmonic Imaging technology.

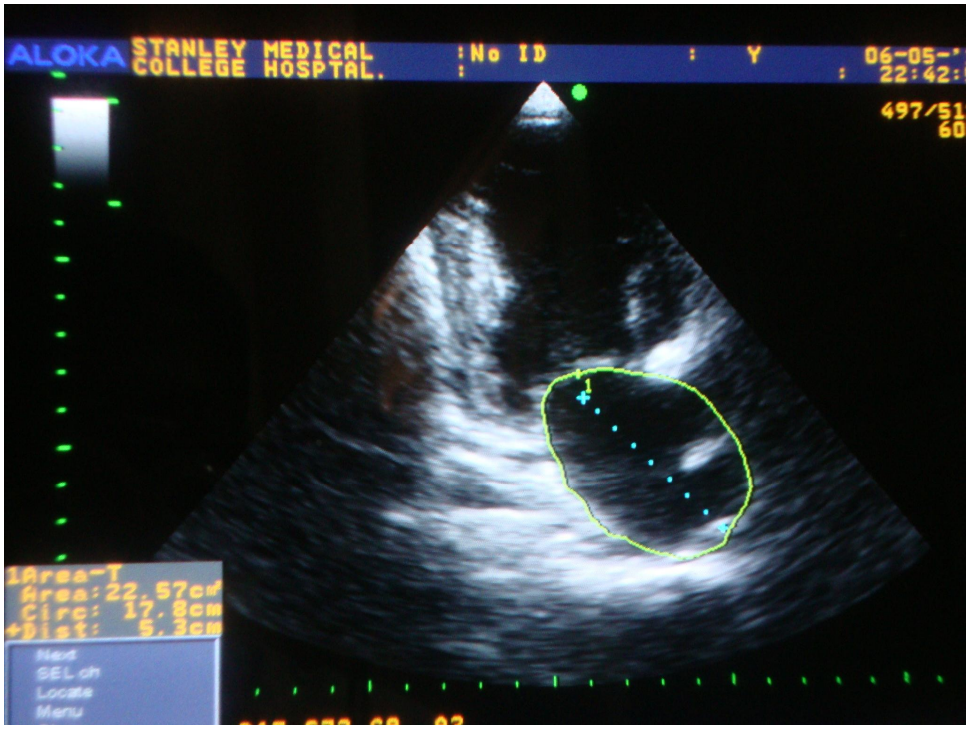
The Biplane area- length method was used which requires measuring LA area from two orthogonal apical views (A1 and A2) and LA length (L), from which LA volume is calculated as (0.85 x A1 x A2)/L (Figure 4). When LA length is measured from two apical views, the shorter value is used to calculate LA volume.

The normal value of indexed LA volume has been reported to be 20±6 mL/m^2.[148] Patients were therefore divided according to the mean value plus 2 SDs, corresponding to 32 mL/m^2.

LV systolic function was assessed semiquantitatively with a visually estimated ejection fraction and wall-motion score index. Excellent agreement between subjective interpretation of ejection fraction and volumetric assessment (95% limits of agreement -6% to 7%), with low interobserver variability (95% limits of agreement -5% to 10%) [145] has been established in previous studies.[145] Each of 16 LV segments was assigned a score (1 to 5) based on myocardial thickening.[146] A wall-motion score index was calculated by dividing the sum of scores by the number of segments visualized. Mitral regurgitation was graded with color flow imaging.



APICAL 4 CHAMBER VIEW



APICAL 2 CHAMBER VIEW

Mitral inflow was assessed with pulsed-wave Doppler echocardiography from the apical 4-chamber view. The Doppler beam was aligned parallel to the direction of flow, and a 1- to 2-mm sample volume was placed between the tips of mitral leaflets during diastole.[147] From the mitral inflow profile, the E- and A-wave velocity and E/A velocity ratio were measured. Doppler tissue imaging of the mitral annulus was also obtained. From the apical 4-chamber view, a 1- to 2-mm sample volume was placed in the septal mitral annulus.

Diastolic filling was categorized as normal (grade 0), impaired relaxation (grade 1), or restrictive (grade 3) by a combination of transmitral flow patterns. LA volume was assessed by the biplane area-length method from apical 4- and 2-chamber views.[11] Measurements were obtained in end systole from the frame preceding mitral valve opening, and the volume was indexed for body surface area.

6) In hospital complications

- **Death,**
- **Re-MI,**
- **Arrhythmias,**
- **LV Dysfunction**
- **Mechanical Complication: VSR / MR**

Complete patient characteristics, treatment details, Thrombolysis, Inotropic Therapy were included in the Proforma for further analysis.

Patients with acute myocardial infarction who had undergone thrombolysis were alone included in this study.

7) Analysis of the Results

The complete data collected during the study period was compiled, analyzed and interpreted keeping the objectives in mind.

8) Statistical Analysis

The data are expressed as Mean ± SD for quantitative data. For qualitative data are expressed as frequency and percentage. The probability value less than 0.05 was considered significant by using SPSS software (V.16.0). Pearson chi square test was used to compare LA volume index with all the parameters including In-hospital events.

Results and analysis

RESULTS AND ANALYSIS

Results

110 Consecutive patients admitted in our ICCU with First episode of Acute Myocardial Infarction were included in our study. Among 110 Patients 80 (72.72%) were males with the mean age of 53 ± 11 Yrs and 30 (27.27%) were Female with the mean age of 61 ± 10 Yrs. AWMI was more common (66.36%) than IWMI (33.63%)

I. Basic characteristics

1. Sex (Table 1 & Figure. 1)

Table 1

Male	80	72.72%
Female	30	27.27%
Total	110	100%

2. Age (Table 2)

Table 2

Sex	Age (Mean ± SD) Yrs
Male	53 ± 11 Yrs
Female	61 ± 10 Yrs

3. Diagnosis (Table 3 & Figure. 2)

Table 3

AWMI	73	66.36%
IWMI	37	33.63%
Total	110	100%

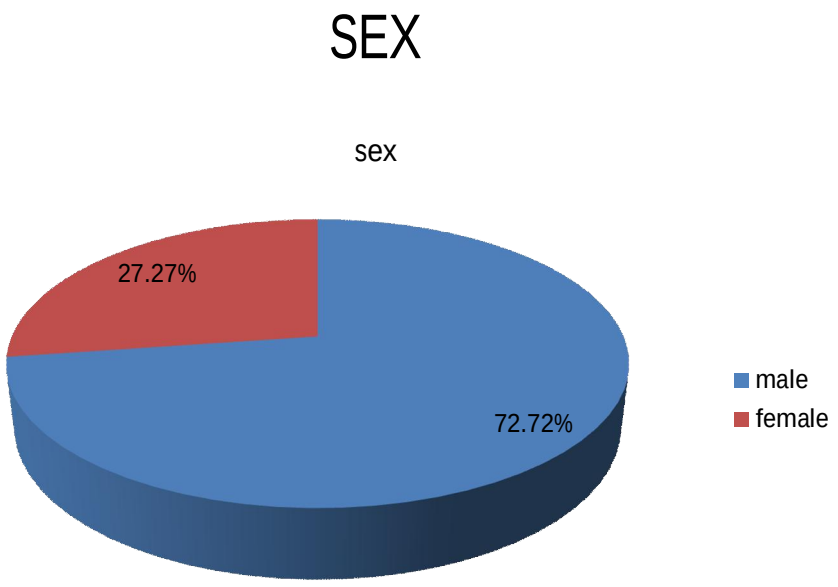


Figure. 1

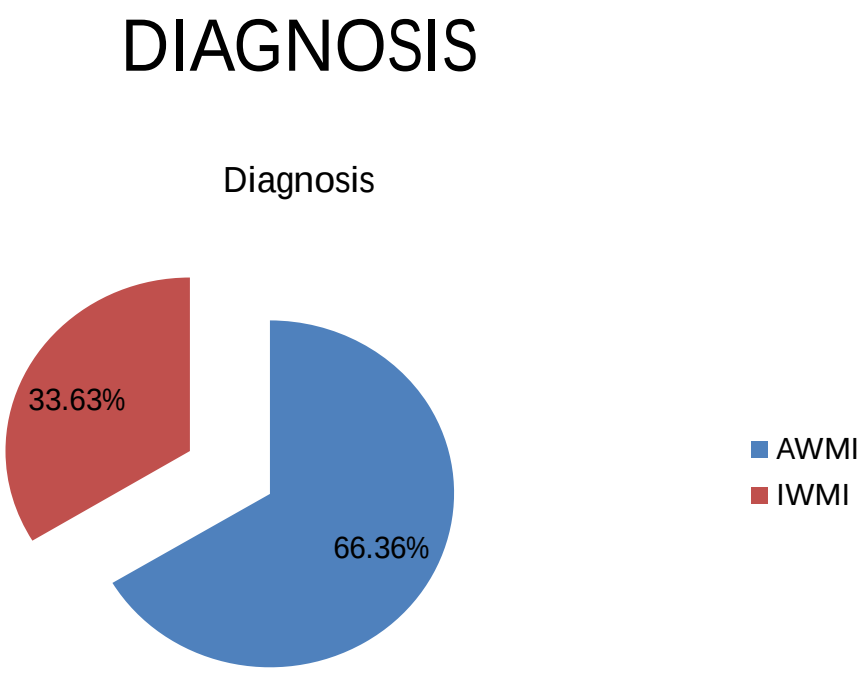


Figure. 2

4. Risk Factors (Table 4 & Figure. 3)

1. Smokers – 39.09%
2. Hypertension - 44.54%
3. Diabetes – 43.63%
4. Dyslipidemia – 53.63%
5. Obesity - 14.54%

Table 4

Smoking	Yes	43	39.09%
	No	67	60.90%
Hypertension	Yes	49	44.54%
	No	61	55.45%
Diabetes	Yes	48	43.63%
	No	62	56.36%
Dyslipidemia	Yes	59	53.63%
	No	51	46.36%
Obesity	Yes	16	14.54%
	No	94	85.45%

Risk Factors

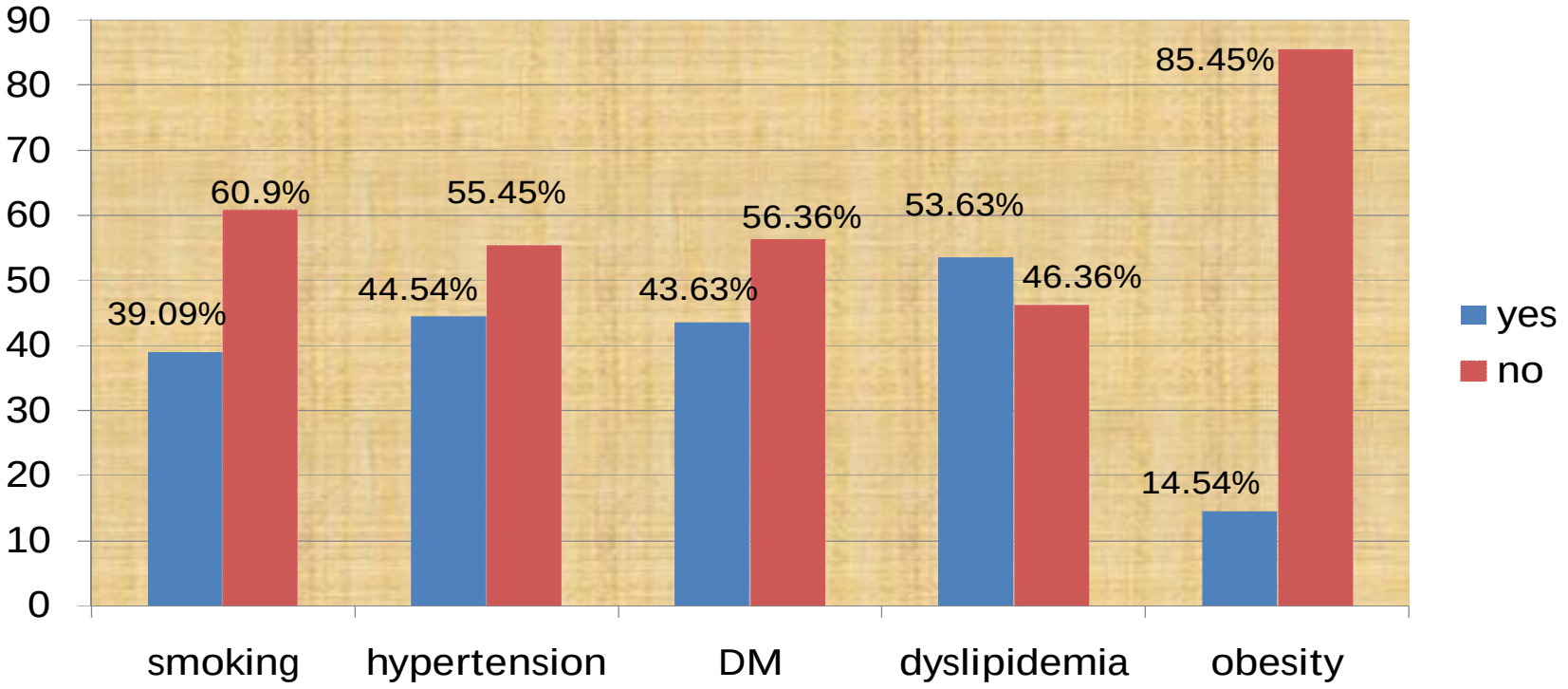


Figure. 3

5. Killip Class (Table 5 & Figure. 4)

Killip Class I – 42.72%

Killip Class II - 36.36%

Killip Class III – 20.90%

Killip Class IV – 0%

Table 5

Kilip Class	I	II	III	IV	Total
No. of Patients	47	40	23	0	110
Percent	42.72%	36.36%	20.90%	0%	100%

6. Inotropic Support (Table 6 & Figure. 5)

31 Patients (28.18%) required inotrophic support

Table 6

Yes	31	28.18%
No	79	71.81%
Total	110	100%

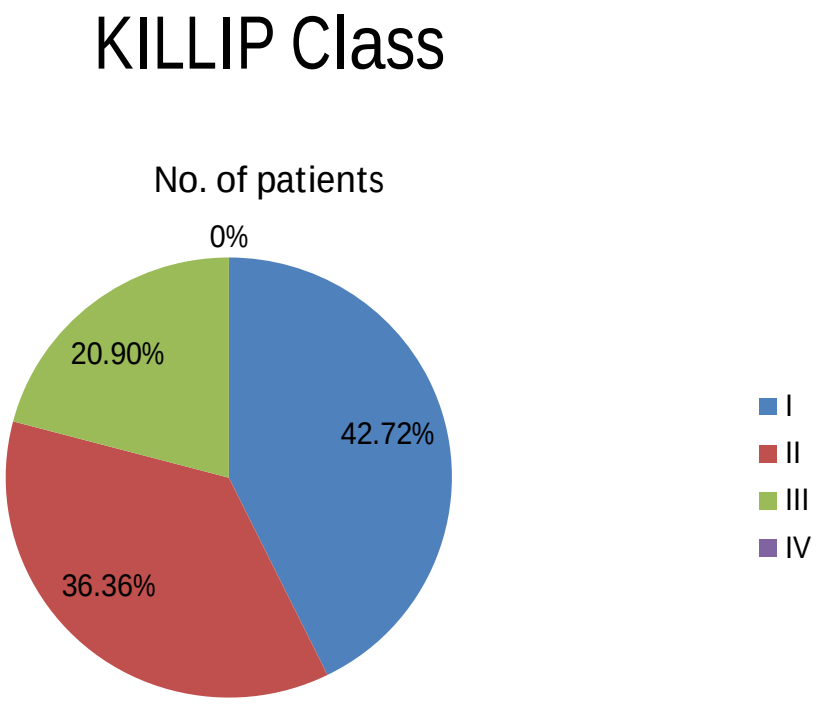


Figure. 4

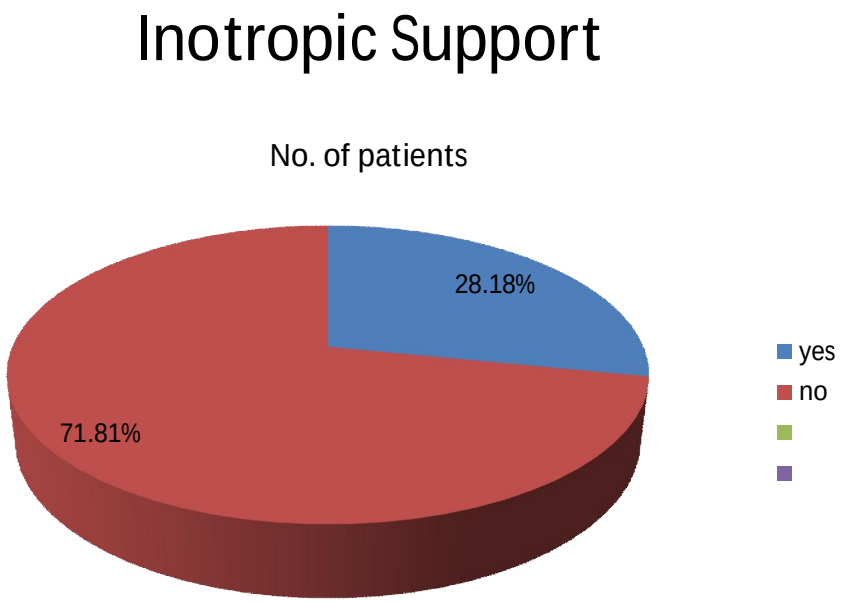


Figure. 5

II. ECHO Characteristics

7. Diastolic Dysfunction (Table 7 & Figure. 6)

Normal – 19.09%

Grade I – 40.90%

Grade II – 30.90%

Grade III - 9.09%

Grade IV – Nil

Table 7

Normal	Grade I	Grade II	Grade III	Total
21	45	34	10	110
19.09%	40.90%	30.90%	9.09%	100%

8. LA Volume Index (Table 8 Figure. 7)

72 Patients (65.45%) had LA Volume Index < 32 ml / m2 and 38 Patients (34.54%) had LA Volume Index > 32 ml / m2

Table 8

< 32 ml / m2	72	65.45%
> 32 ml / m2	38	34.54%
Total	110	100%

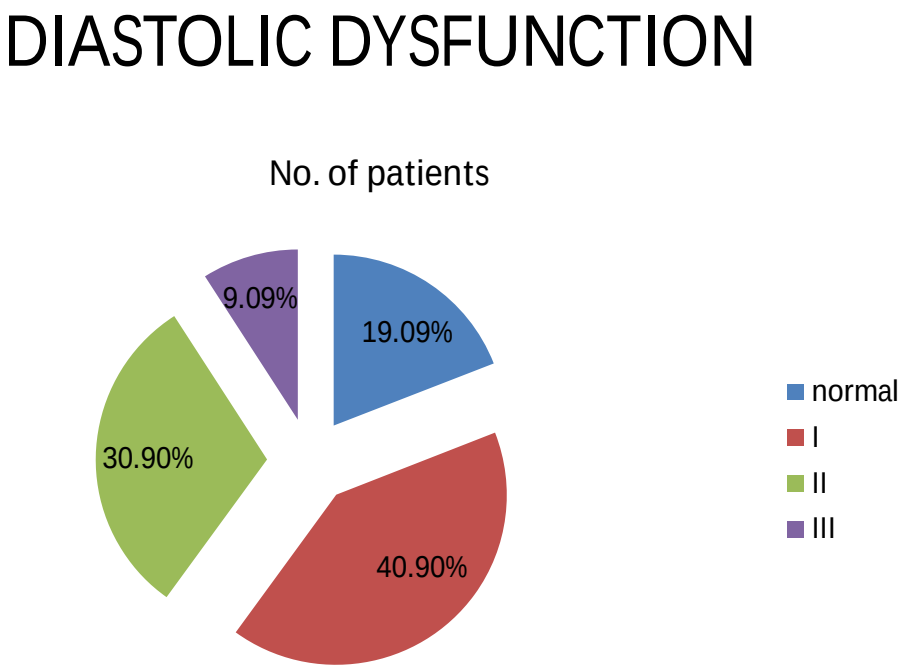


Figure. 6

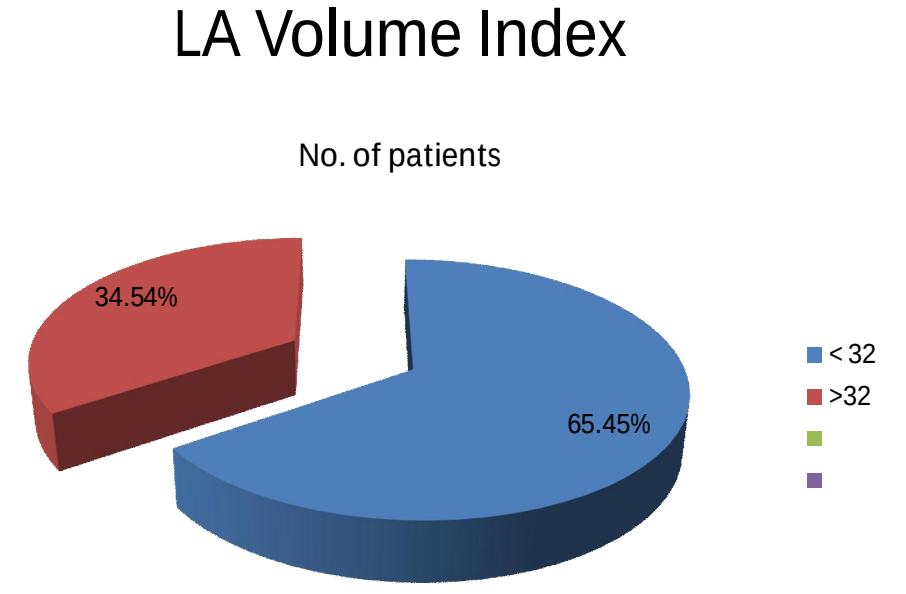


Figure. 7

III. IN HOSPITAL EVENTS (Table 9 & Figure. 8)

Death – 8.18%

Re – MI – 10%

Arrhythmias – 35.45%

LV dysfunction – 75.45%

Mechanical Complications – 7.3%

Table 9

Death	Yes	9	8.18%
	No	101	91.81%
Re MI	Yes	11	10%
	No	99	90%
Arrhythmia	Yes	39	35.45%
	No	71	64.54%
LV dysfunction	Yes	83	75.45%
	No	27	24.54%
Mechanical Complications	MR	6	5.5%
	VSR	2	1.8%
	No	102	92.7%

In Hospital Events

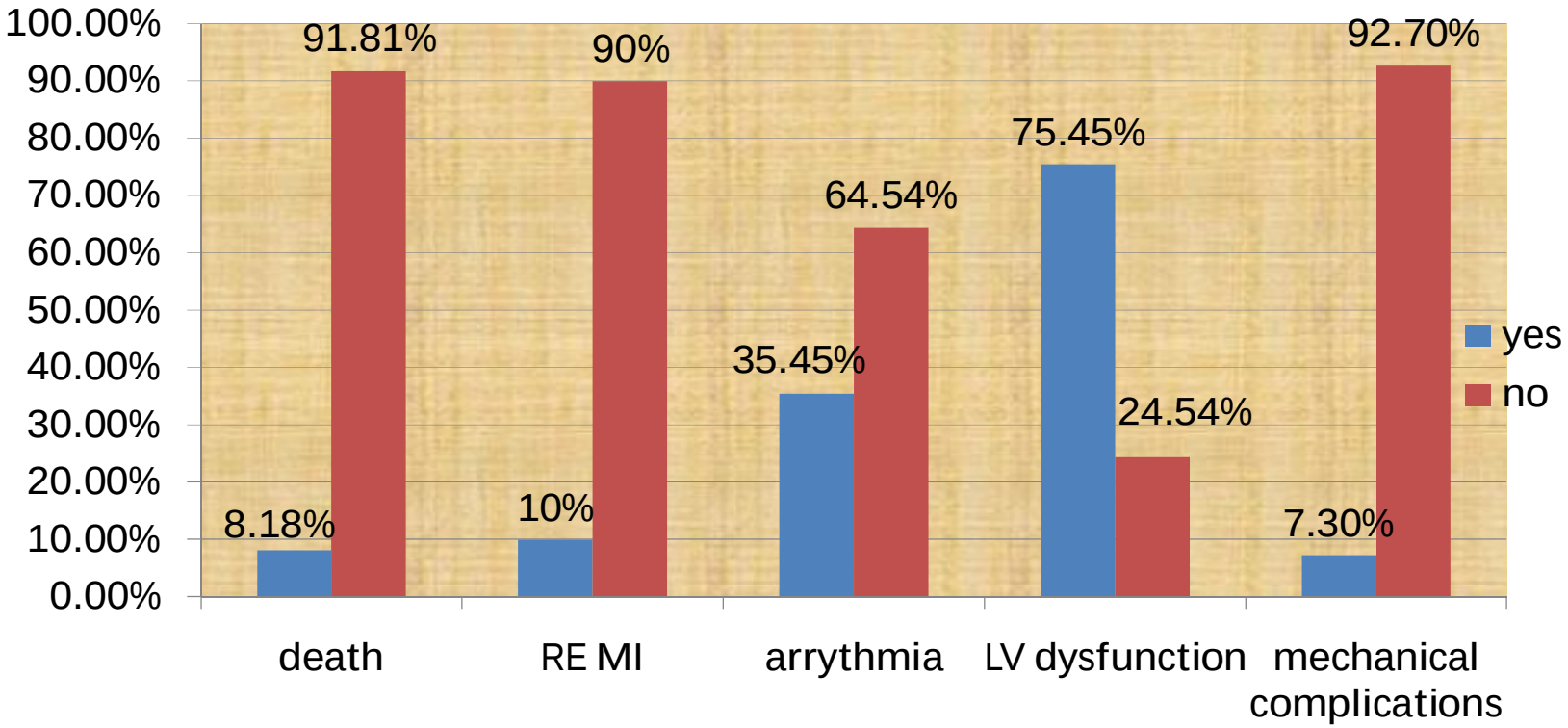


Figure. 8

DATA ANALYSIS

1. Gender and LA Volume Index (Table no 10 and Figure 9)

In our study, the overall patient population is predominantly male but no statistical significance was noted.

Table 10: Gender and LAVI

LAVI	Male	Female	Total	P Value
z	57(79.16%)	15(20.83%)	72(100%)	0.0626 (Corrected)
>32 ml/m^2	23(60.52%)	15(39.47%)	38(100%)	
Total	80(72.72%)	30(27.27%)	110(100%)	

2. Age and LA Volume Index (Table no 11 and Figure. 10)

In our study, age group of 40 – 60 years are more compared to age group above 60 years, hence no statistical significance was noted.

Table 11: Age and LAVI

LAVI	<40yrs	40-60yrs	>60yrs	Total	p Value
<32ml/m^2	5(6.94%)	41(56.94%)	26(36.11%)	72(100%)	0.6281
>32ml/m^2	3(7.89%)	18(47.36%)	17(44.73%)	38(100%)	
Total	8(7.27%)	59(53.63%)	43(39.09%)	110(100%)	

GENDER AND LAVI

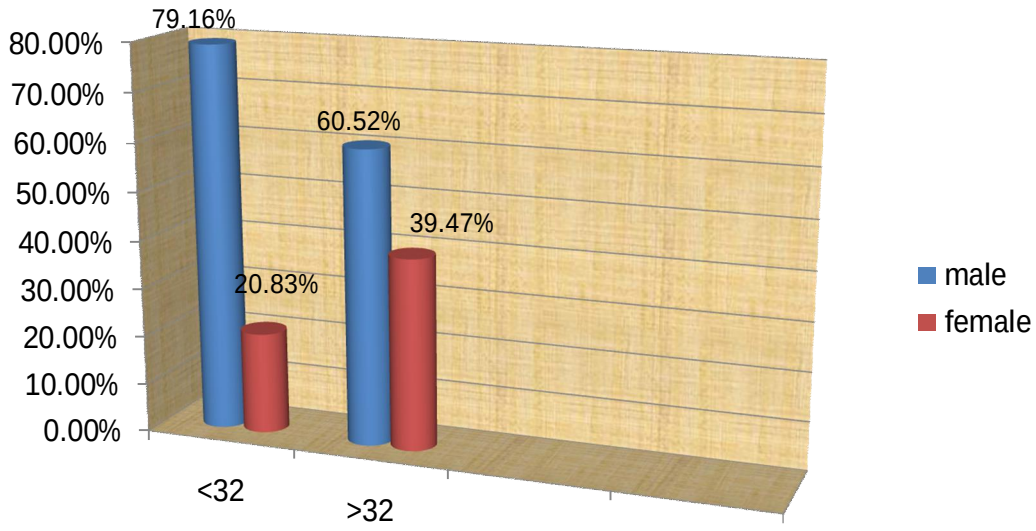


Figure. 9

AGE AND LAVI

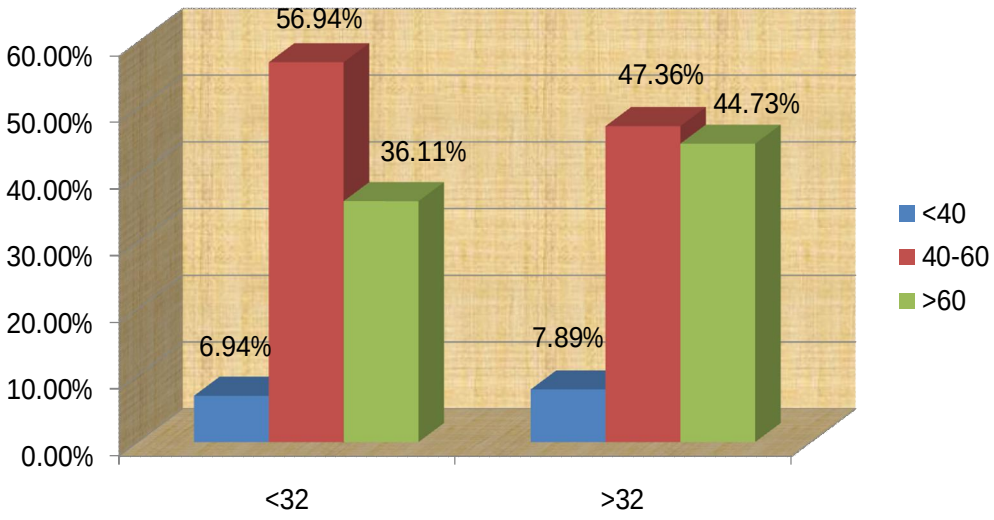


Figure. 10

3. Diagnosis and LA Volume Index (Table no 12 and Figure. 11)

Anterior wall myocardial infarction were more common than inferior wall myocardial infarction in LA Volume Index >32 ml/m^2 but not enough for a statistical significance

Table 12: Diagnosis and LAVI

LAVI	AWMI	IWMI	Total	p Value
<32ml/m^2	48(66.66%)	24(33.33%)	72(100%)	0.9058 (Corrected)
>32ml/m^2	25(65.78%)	13(34.2%)	38(100%)	
Total	73(66.36%)	37(33.63%)	110(100%)	

4. Smoking and LA Volume Index (Table no 13 and Figure. 12)

Smokers were more common (65.78%) in larger LA volume index group than smaller LA volume index group (25%) with statistically significant p value noted.

Table 13: Smoking and LAVI

LAVI	Smoker	Non smoker	Total	P Value
<32 ml/m^2	18(25%)	54(75%)	72(100%)	0.0001 (Corrected)
>32 ml/m^2	25(65.78%)	13(34.21%)	38(100%)	
Total	43(39.09%)	67(60.90%)	110(100%)	

DIAGNOSIS AND LAVI

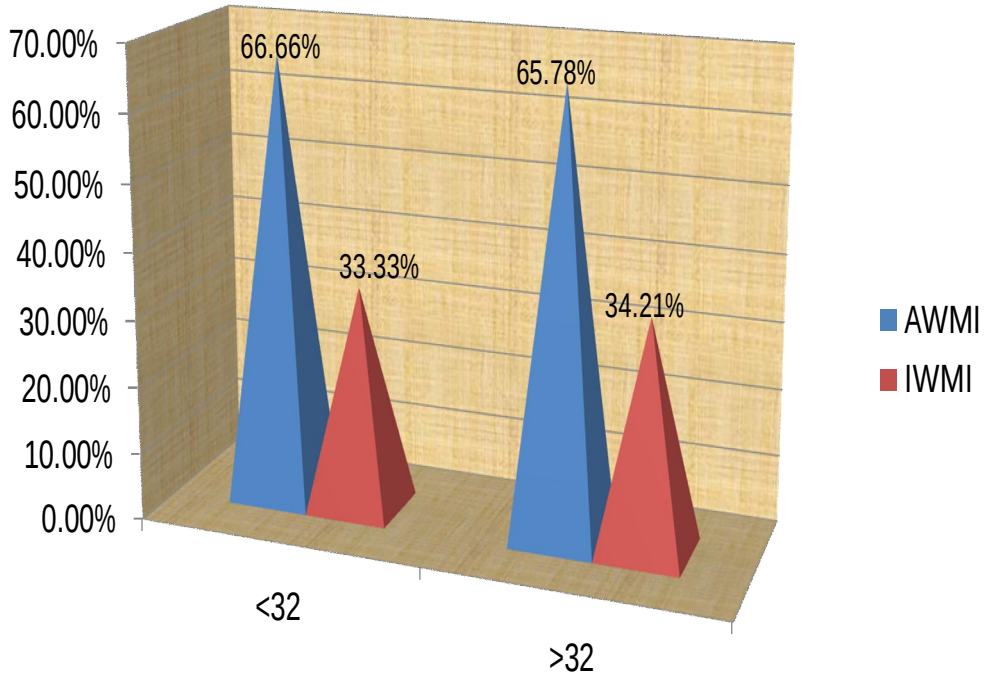


Figure.11

SMOKING AND LAVI

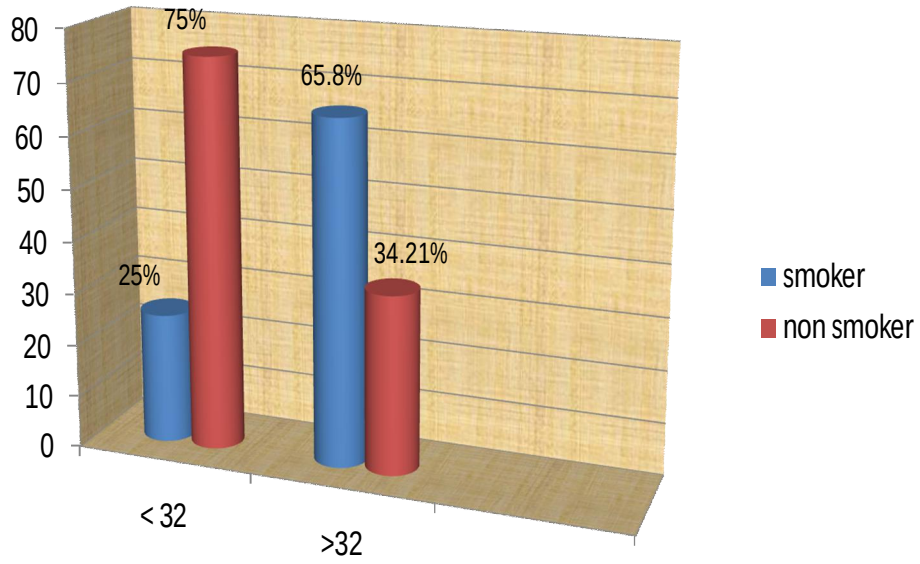


Figure. 12

5. Hypertension and LA Volume Index (Table no 14 and Figure. 13)

Hypertension was more common (73.68%) in larger LA Volume Index group than in small LA Volume Index group (29.16%) resulting in statistically significant p value (0.0000).

Table 14: SHT and LAVI

LAVI	Hypertensive	Not known Hypertensive	Total	P Value
<32 ml/m^2	21(29.16%)	51(70.83%)	72(100%)	0.0000 (Corrected)
>32 ml/m^2	28(73.68%)	10(26.31%)	38(100%)	
Total	49(44.54%)	61(55.45%)	110(100%)	

6. Diabetes and LA Volume Index (Table no 15 and Figure. 14)

More diabetic patients in larger LA Volume Index group(73.68%) than smaller lA volume index group (27.77%) resulting in a statistically significant p value (0.0000).

Table 15: Diabetes and LAVI

LAVI	Diabetics	Non Diabetics	Total	p Value
<32 ml/m^2	20(27.77%)	52(72.22%)	72(100%)	0.0000 (Corrected)
>32 ml/m^2	28(73.68%)	10(26.31%)	38(100%)	
Total	48(43.63%)	62(56.36%)	110(100%)	

HYPERTENSION AND LAVI

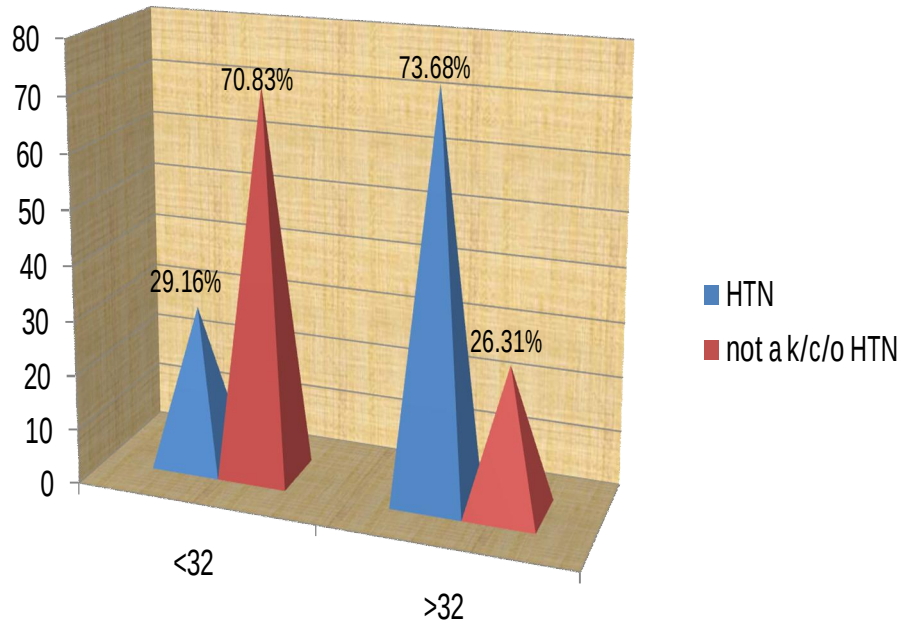


Figure. 13

DIABETES AND LAVI

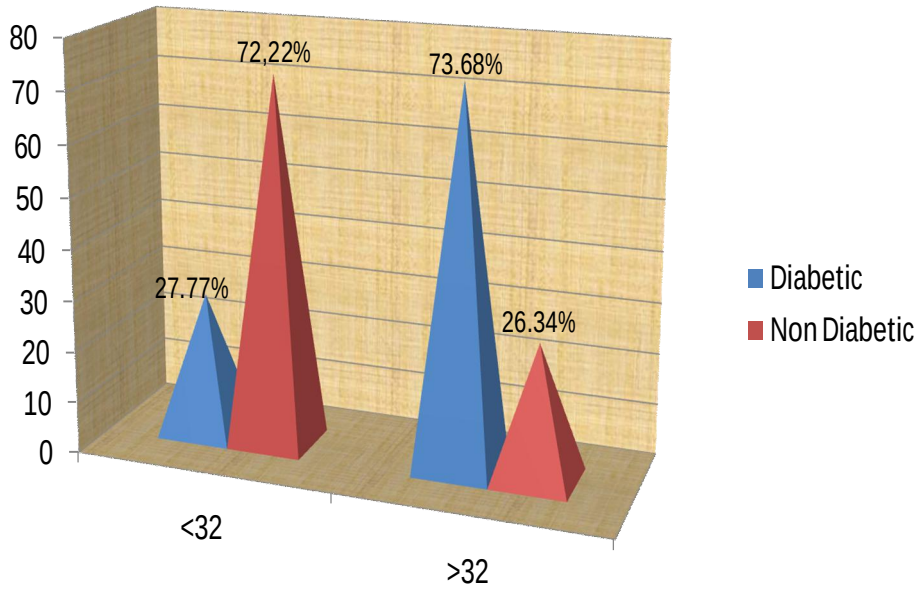


Figure. 14

7. Dyslipidemia and LA Volume Index (Table no 16 and Figure. 15)

Patients with larger LA Volume Index group has more dyslipidemic patients(86.84%)than smaller LA volume index group(36.11%) , resulting in statistically significant p value (0.000).

Table 16: Dyslipidemia and LAVI

LAVI	Present	Absent	Total	p Value
<32 ml/m^2	26(36.11%)	46(63.88%)	72(100%)	0.0000 (Corrected)
>32 ml/m^2	33(86.84%)	5(13.15%)	38(100%)	
Total	59(53.63%)	51(46.36%)	110(100%)	

8. Obesity and LA Volume Index (Table no 17 and Figure. 16)

Obese patients are less in our study group (14.54%) and they are also less common among larger LA Volume Index group (13.15%). Hence no statistical significance was noted.

Table 17: Obesity and LAVI

LAVI	Present	Absent	Total	P Value
<32 ml/m^2	11(15.27%)	61(84.72%)	72(100%)	1 (Corrected)
>32 ml/m^2	5(13.15%)	33(86.84%)	38(100%)	
Total	16(14.54%)	94(85.45%)	110(100%)	

DYSLIPIDEMIA AND LAVI

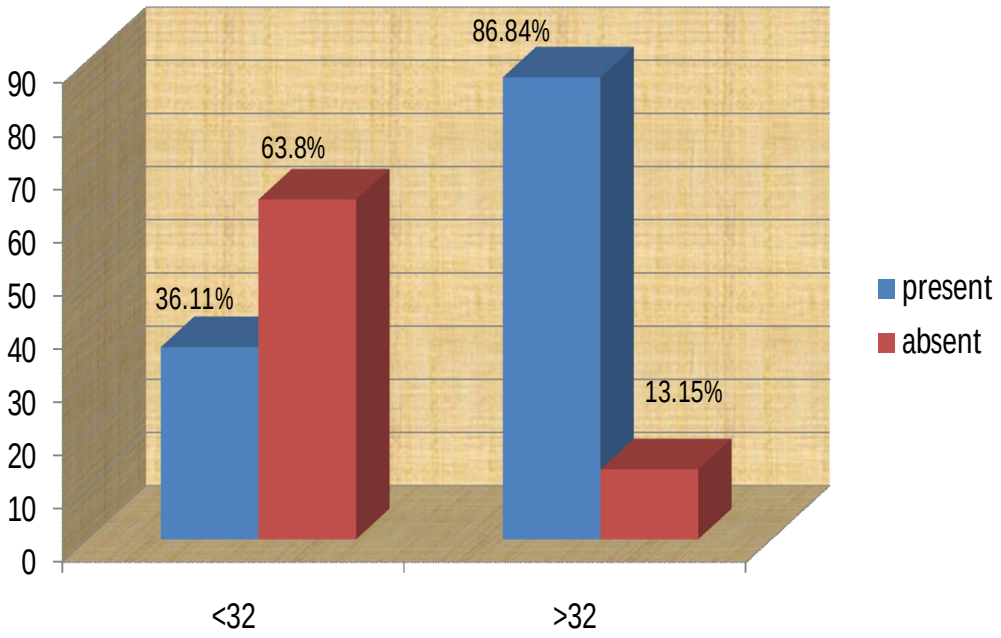


Figure. 15

OBESITY AND LAVI

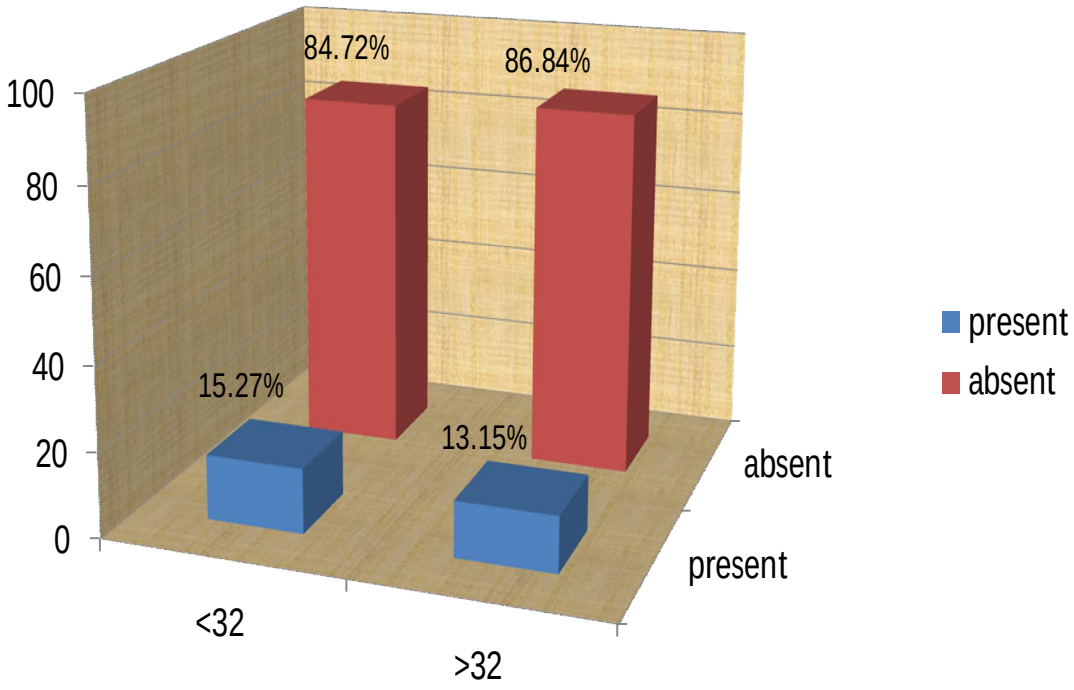


Figure.16

9. Killip Class and LA Volume Index (Table no 18 and Figure. 17)

Patients with larger LA volume Index group had more no of patients in Killip class II (52.63%) and Killip Class III (36.84%) in comparison to smaller LA Volume Index group which had more no of patients in killip class I (59.72%). This resulted in statistically significant p value (0.0000).

Table 18: Killip Classification and LAVI

LAVI	Class 1	Class 2	Class 3	Class 4	Total	p Value
<32 ml/m^2	43(59.72%)	20(27.77%)	9(12.5%)	0	72(100%)	0.0000
>32 ml/m^2	4(10.52%)	20(52.63%)	14(36.84%)	0	38(100%)	
Total	47(42.72%)	40(36.36%)	23(20.90%)	0	110(100%)	

10. Inotropic support and LA Volume Index (Table no 19 and Figure. 18)

More patients required inotropic support (42.10%) in larger LA Volume Index group compared to less number of patients (20.83%) requiring inotropic support in smaller LA Volume Index group, resulting in statistically significant p value (0.0327).

Table 19: Inotropic Support and LAVI

LAVI	YES	NO	Total	P Value
<32 ml/m^2	15(20.83%)	57(79.16%)	72(100%)	0.0327
>32 ml/m^2	16(42.10%)	22(57.89%)	38(100%)	
Total	31(28.18%)	79(71.81%)	110(100%)	

KILLIP CLASS AND LAVI

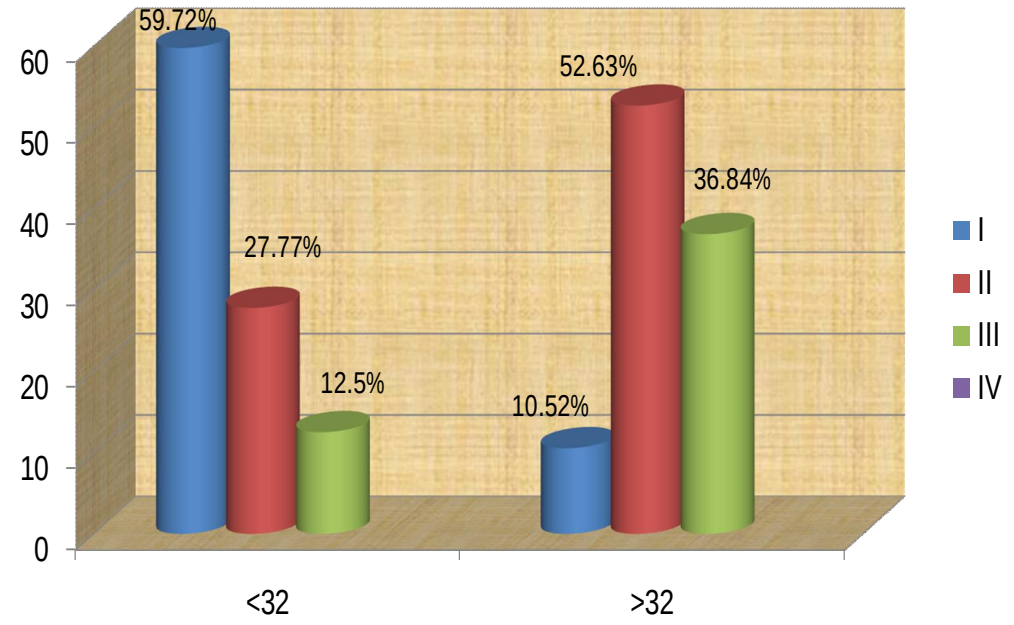


Figure. 17

INOTROPIC SUPPORT AND LAVI

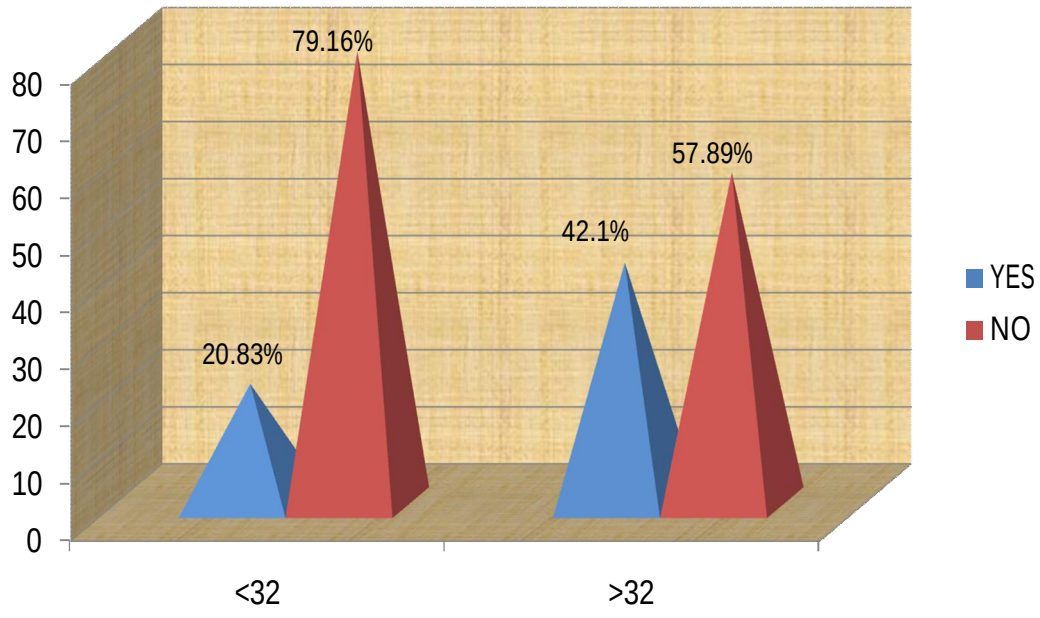


Figure. 18

11. Re MI and LA Volume Index (Table no 20 and Figure. 19)

Re MI occurred in 26.31% of patients in larger LA Volume Index group compared to 1.38% in smaller LA Volume Index group resulting in statistically significant p value (0.0001).

Table 20: Re MI and LAVI

LAVI	YES	NO	Total	p Value
<32 ml/m^2	1(1.38%)	71(98.61%)	72(100%)	0.0001
>32 ml/m^2	10(26.31%)	28(73.68%)	38(100%)	
Total	11(10%)	99(90%)	110(100%)	

12. Arrhythmia and LA Volume Index (Table no 21 and Figure. 20)

Recurrent arrhythmia occurred in 68.42% of patients in larger LA Volume Index group compared to 18.05% in smaller LA Volume Index group .This resulted in a statistically significant p value (0.0000).

Table 21: Arrhythmia and LAVI

LAVI	YES	NO	Total	P Value
<32 ml/m^2	13(18.05%)	59(81.94%)	72(100%)	0.0000
>32 ml/m^2	26(68.42%)	12(31.57%)	38(100%)	
Total	39(35.45%)	71(64.54%)	110(100%)	

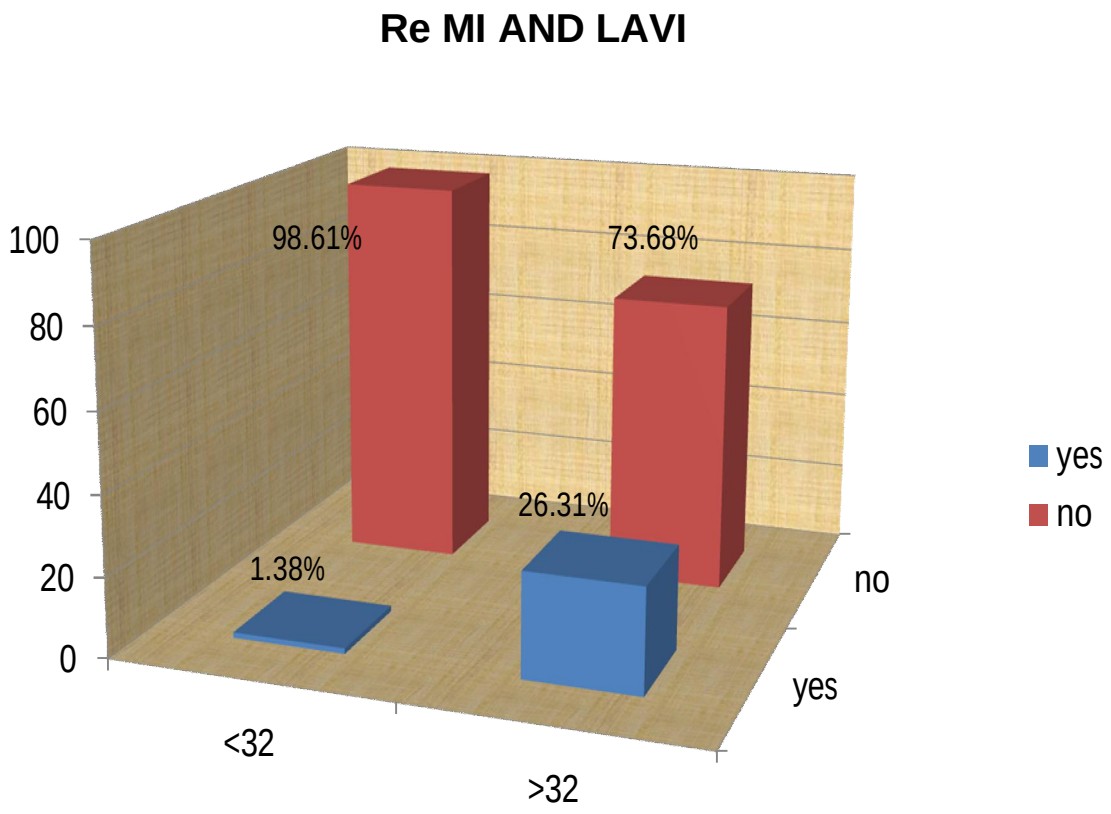


Figure. 19

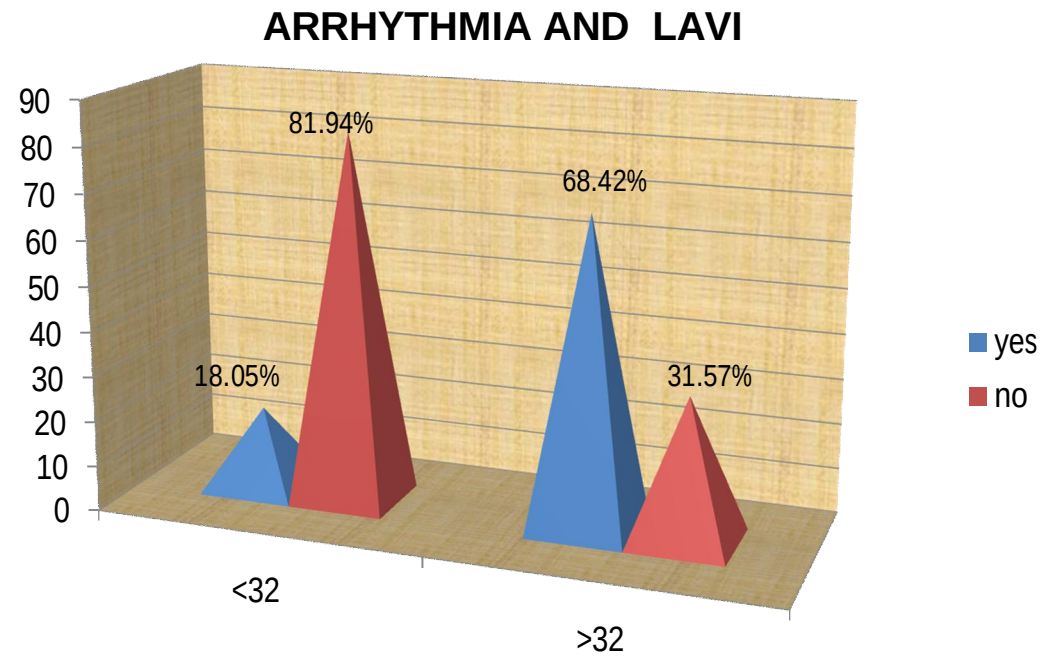


Figure. 20

13. LV Systolic Dysfunction and LA Volume Index (Table no 22 and Figure. 21)

In patients with large LA volume index group, 89.47% of patients had LV systolic dysfunction compared to 68.05% with smaller LA volume index group. This resulted in statistically significant p value (0.0245).

Table 22: LV Systolic Dysfunction and LAVI

LAVI	YES	NO	Total	p Value
<32 ml/m^2	49(68.05%)	23(31.94%)	72(100%)	0.0245
>32 ml/m^2	34(89.47%)	4(10.52%)	38(100%)	
Total	83(75.45%)	27(24.54%)	110(100%)	

14. Mechanical complication and LA volume Index(Table no 23 and Figure. 22)

Mechanical complications were more common among larger LA volume index group but the number is very less. Hence no statistically significant p value.

Table 23: Mechanical Complication and LAVI

LAVI	MR	VSR	NO	Total	P Value
<32 ml/m^2	2(2.77%)	0(0%)%	70(97.22%)	72(100%)	Not significant
>32 ml/m^2	4(10.52%)	2(5.26%)	32(84.21%)	38(100%)	
Total	6(5.5%)	2(1.8%)	102(92.7%)	110(100%)	

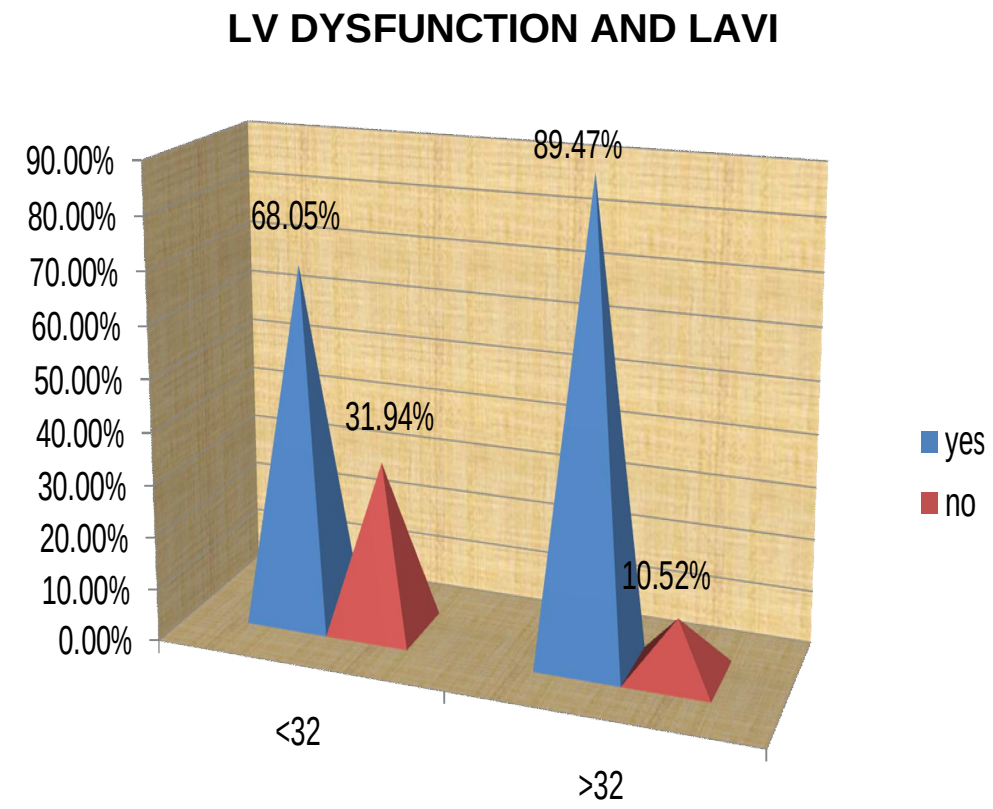


Figure. 21

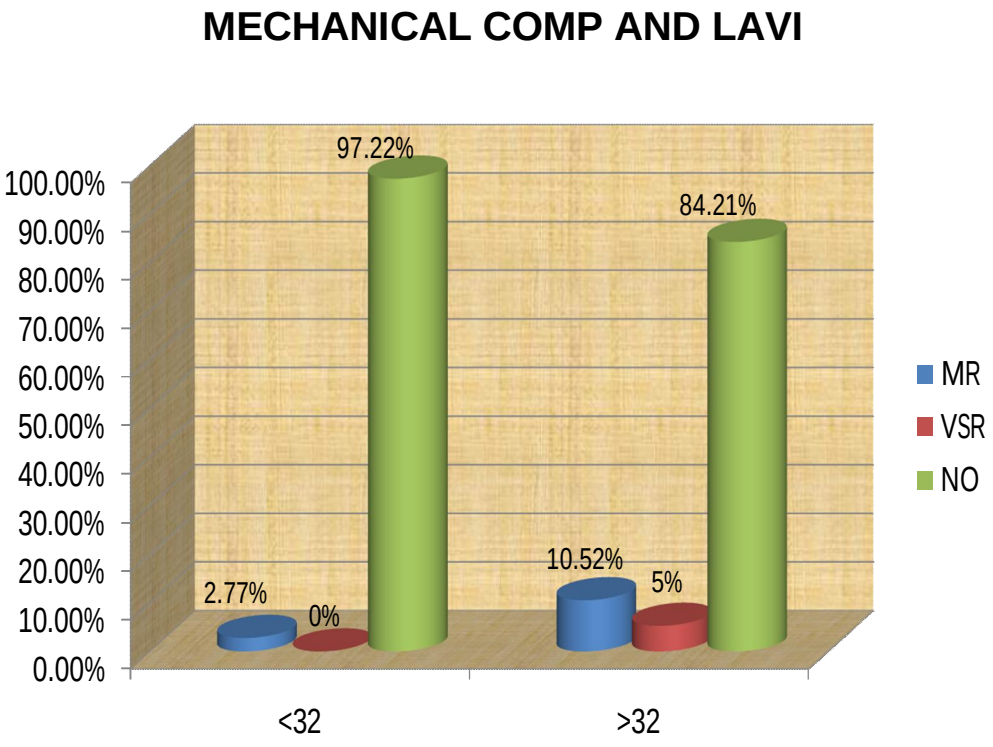


Figure. 22

15. Death and LA Volume Index (Table No 24 and Figure. 23)

Death was more common in larger LA volume index group (21.05%) compared to small LA Volume Index (1.38%) This resulted in statistically significant p value (0.0013).

Table 24: Death and LAVI

LAVI	YES	NO	Total	P Value
<32 ml/m^2	1(1.38%)	71(98.61%)	72(100%)	0.0013
>32 ml/m^2	8(21.05%)	30(78.94%)	38(100%)	
Total	9(8.18%)	101(91.81%)	110(100%)	

16. Diastolic dysfunction and LA Volume Index (Table no 25 and Figure. 24)

Grade II (50%) and Grade III (21.05%) diastolic dysfunction were more common in larger LA volume index group but normal (25%) and Grade I (51.38%) diastolic dysfunction were more common in smaller LA volume index group. This resulted in statistically significant p value (0.0000).

Table 25: Diastolic Dysfunction and LAVI

LAVI	Normal	Grade I	Grade II	Grade III	Total	P Value
<32 ml/m^2	18(25%)	37(51.38%)	15(20.83%)	2(2.77%)	72(100%)	0.0000
>32 ml/m^2	3(7.89%)	8(21.05%)	19(50%)	8(21.05%)	38(100%)	
Total	21(19.09%)	45(40.90%)	34(30.90%)	10(9.09%)	110(100%)	

DEATH AND LAVI

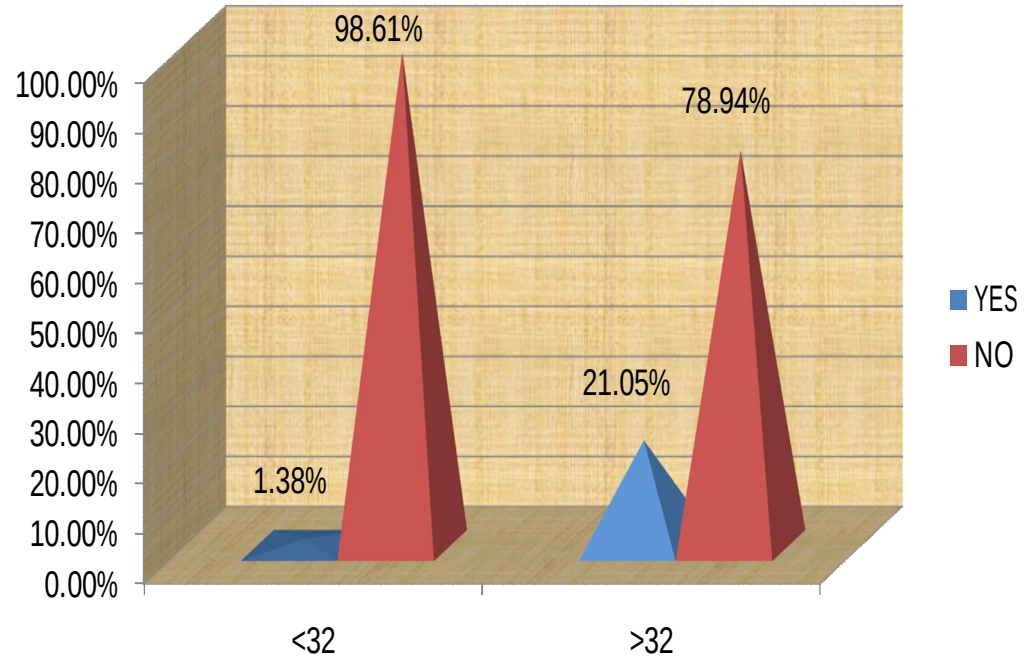


Figure. 23

DIASTOLIC DYSFUNTION AND LAVI

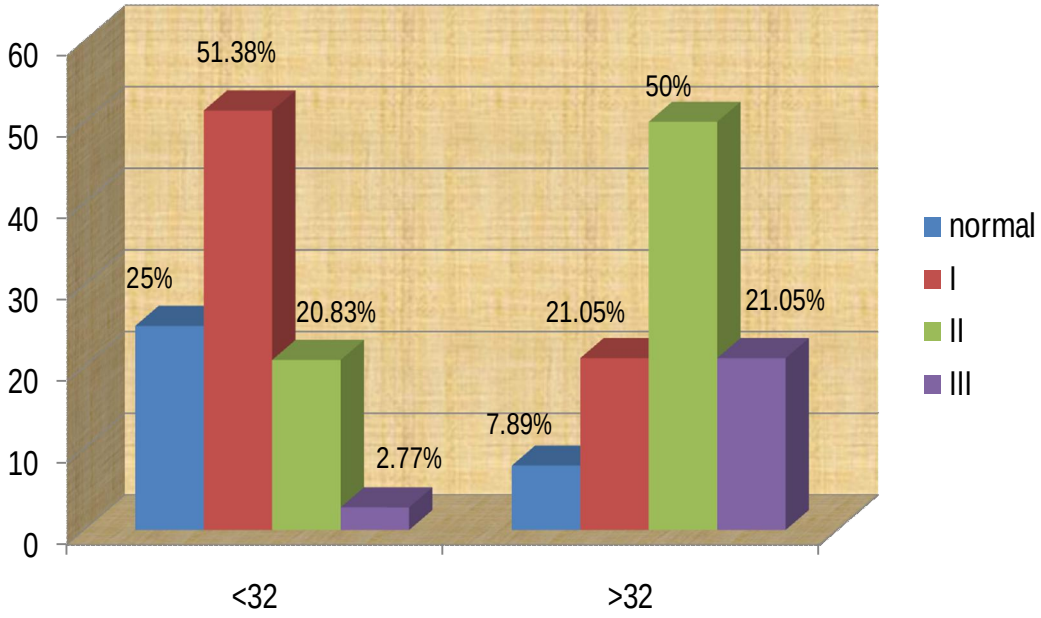


Figure. 24

17. E/e' and LA volume Index (Table no 26 and Figure. 25)

In patients with larger LA volume index group, 26.31% of patients were having E/e' of more than 15 compare to only 2.77% of patients was having E/e' of more than 15 in smaller LA volume index group. This resulted in statistically significant p value (0.0004).

Table 26: E/e' and LA Volume Index

LAVI	<15	>= 15	Total	p Value
<32 ml/m^2	70(97.22%)	2(2.77%)	72(100%)	0.0004
>32 ml/m^2	28(73.68%)	10(26.31%)	38(100%)	
Total	98(89.09%)	12(10.90%)	110(100%)	

In patients with larger LA Volume Index group, mean E/e' was 12.39 compared to mean E/e' of 8.52 in smaller LA Volume Index group.

LAVI	E/e'
<32 ml/m^2	8.52 ±2.91
>32 ml/m^2	12.39 ± 3.62

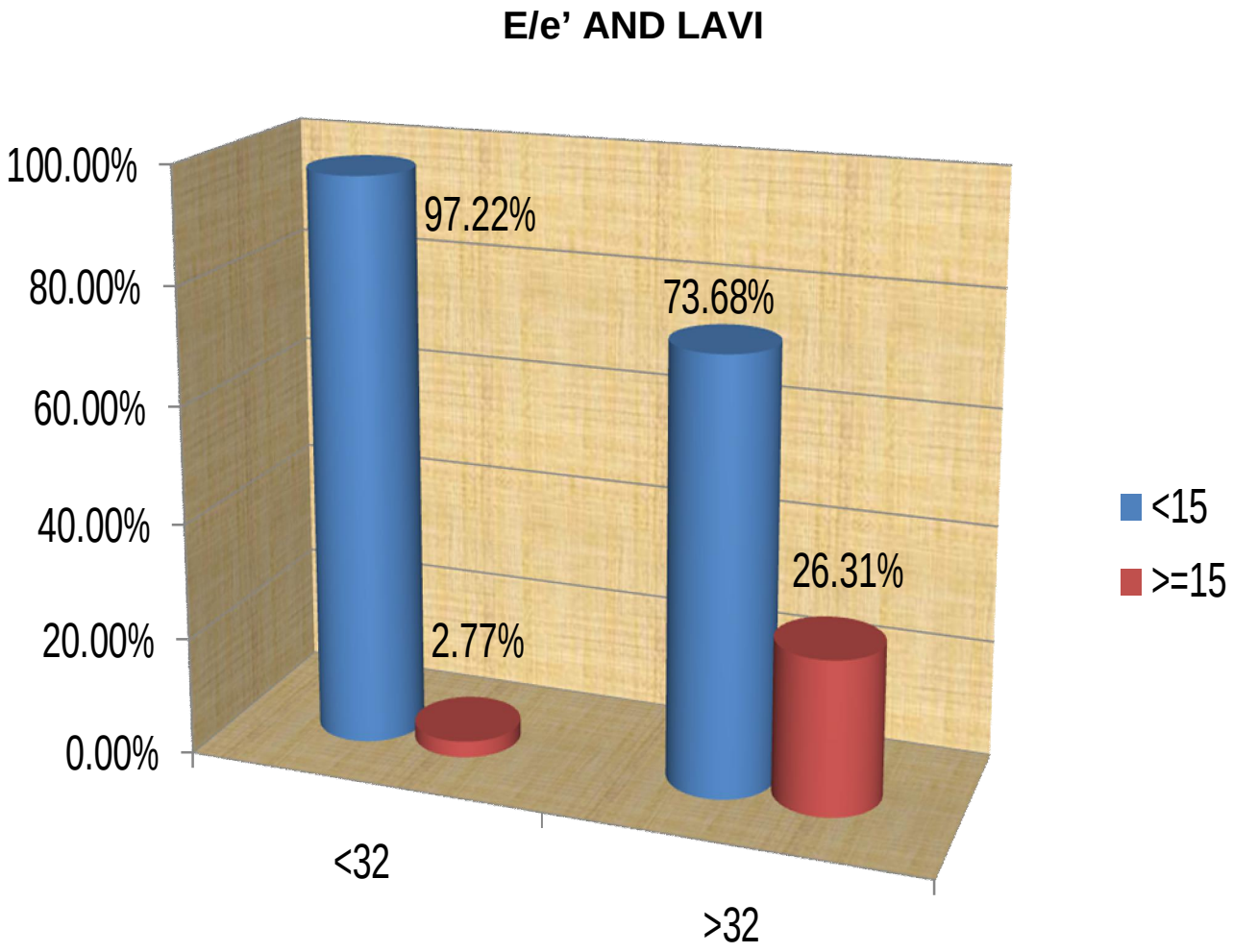

E/e' AND LAVI
100.00%
80.00%
60.00%
40.00%
20.00%
0.00%
97.22%
2.77%
73.68%
26.31%
<32
>32
<15
>=15

Figure. 25

18. LV EF and LA Volume Index (Table no 27 and figure. 26)

More number of patients with LV EF less than 40% (57.89%) were in larger lA volume index group compare to Less Number (27.77%) of patients with LV EF less than 40% were in smaller LA volume index group.This resulted in significant p value (0.0020).

Table 27:LV EF and LAVI

LAVI	>= 40%	<40%	Total	P Value
<32 ml/m^2	52(72.22%)	20(27.77%)	72(100%)	0.0020
>32 ml/m^2	16(42.10%)	22(57.89%)	38(100%)	
Total	68(61.81%)	42(38.18%)	110(100%)	

In patients with larger LA Volume Index group, the mean LV EF was 37.66% compared to mean LV EF of 43.56% in small LA Volume Index group.

LAVI	EF%
<32 ml/m^2	43.56 ± 8.36%
>32 ml/m^2	37.66 ± 7.47%

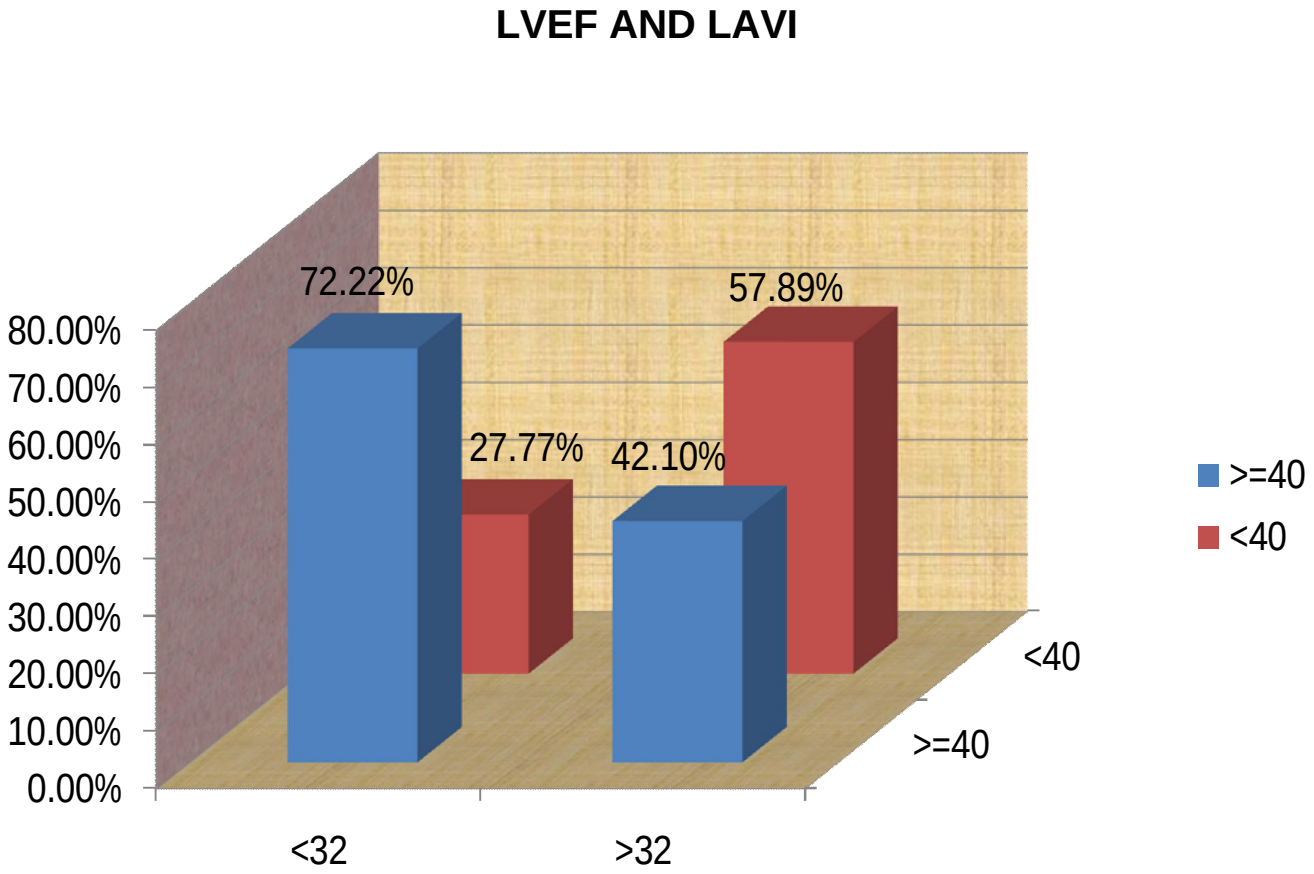

LVEF AND LAVI
80.00%
70.00%
60.00%
50.00%
40.00%
30.00%
20.00%
10.00%
0.00%
72.22%
27.77%
42.10%
57.89%
<32
>32
<40
>=40
>=40
<40

Figure. 26

19. WMSI and LA Volume Index(Table no 28 and Figure. 27)

In patients with larger LA Volume Index group, 92.10% of patients had WMSI of > 1.3 compared to 52.77% of patients in smaller LA Volume Index group. This resulted in statistically significant p value (0.0001).

Table 28: WMSI and LAVI

LAVI	<1.3	>= 1.3	Total	P Value
<32 ml/m^2	34(47.22%)	38(52.77%)	72(100%)	0.0001
>32 ml/m^2	3(7.89%)	35(92.10%)	38(100%)	
Total	37(33.63%)	73(66.36%)	110(100%)	

In patients with larger LA Volume index group, mean WMSI was 1.49 compared to mean WMSI of 1.34 in smaller LA volume index group.

LAVI	WMSI
<32 ml/m^2	1.3410 ± 0.1547
>32 ml/m^2	1.4942 ± 0.1788

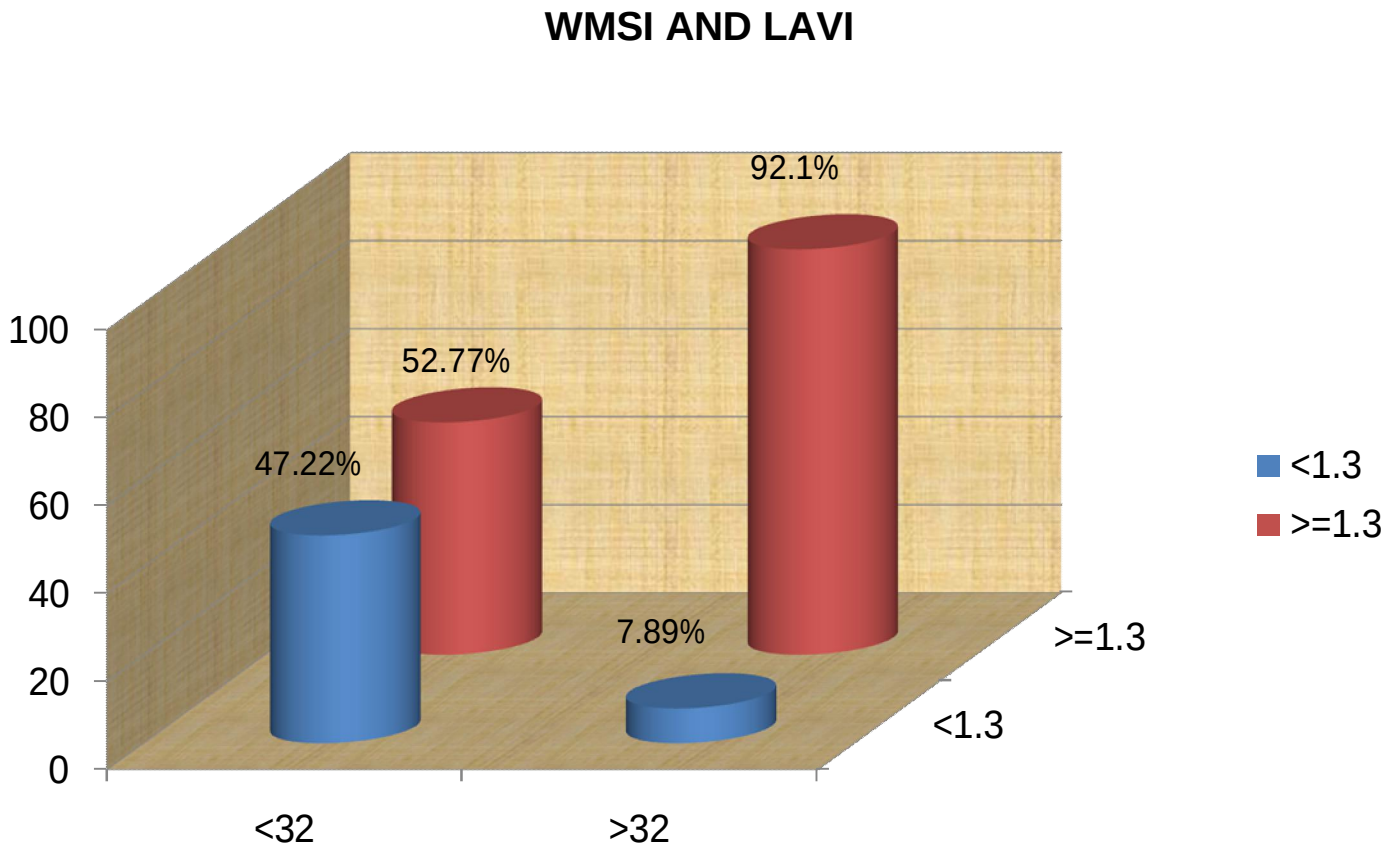


Figure. 27

Discussion

DISCUSSION

This study has been conducted to demonstrate that LA volume index is a predictor of In-hospital events after Acute Myocardial Infarction (STEMI). Furthermore, LA volume index provides prognostic information incremental to clinical data and standard echocardiographic predictors of outcome, including LV systolic function, Doppler assessment of diastolic function.

LA volume index, on comparison with various clinical characteristics

1. Gender and LA volume index, (Table No. 10 & Figure. 9)

- In our study population of 110 Patients, 72.72% were males and 27.27% were females. Males were more in number, both in LA volume index > 32 ml/m^2 and < 32 ml/m^2 categories.

- Gender differences in LA Volume index does not occur as per reviewed literature[19, 32, 51, 52]. Similarly in this study also there were no significant gender bias in LA Volume index analysis (p value 0.0626).

2. Age Group and LA volume index, (Table No. 11 and Figure. 10)

- There exists a direct correlation between advancing age and increased LA Volume Index i.e. Old age (< 60 years) is associated with physiologically increased LA volume index . So inclusion of older age patients in a study population alters the variables.

- Age group In this study were divided in to 3 categories i.e. < 40 yrs, 40 to 60 yrs & > 60 yrs.

- In this study, patients > 60 yrs were less number ($p < 0.625$), thus enabling a balanced study.

3. Location of MI and LA volume index, (Table No. 12 and Figure. No. 11)

- No independent correlation exists between regional location of MI and magnitude of LA volume index. Similarly in this study also, though anterior wall MI was more common, in the total study population, as well as in the 2 categories of LA volume index, there was no statistically significant difference ($p < 0.905$).

4. Smoking and LA volume index (Table No. 13 & Figure. No. 12)

- In the article by Teresa S M Sang MD et al [153] Left atrial volume was found to correlate positively with age, history of systemic hypertension, diabetes mellitus, hyperlipidemia, and smoking.

- Smokers were more common among patients with LA volume index > 32 ml/m^2 (65.78%), than LA volume index < 32 ml/m^2 (25%) resulting in a significant p value (0.0001).

- In this study smokers had a higher incidence of increased LA volume index (> 32ml/m^2) indicating significant cardiovascular risk.

5. Hypertension and LA volume index (Table No. 14 & Figure. 13)

- Hypertension was more common among patients with LA volume index > 32 ml/m^2 (73.68%) but only 29.16% in the group of patients with LA volume index < 32 ml/m^2. As a result the p value was significant (0.000).

- In a study of older adults referred for echocardiography, LA volume ≥32 ml/m^2 was associated with increased incidence of CHF, independent of age, myocardial infarction, diabetes mellitus, hypertension, LV hypertrophy, and mitral inflow velocities [121].

- In the article by Teresa S M Sang MD et al [153] LA volume was found to correlate positively with cardiovascular risk score which includes hypertension.

6. Diabètes and LA volume index (Table No. 15 and Figure. No. 14)

- In our study, 73.68% of patients were diabetic among patients with LA volume > 32 ml/m^2 and only 27.77% were diabetic in the group with LA < 32 ml/m^2 giving rise to a significant p value (0.000)[121].

- This is similar to the study by Teresa S M Sang MD et al [153] where LA volume was found to correlate positively with Diabetes mellitus and other parameters of cardiovascular risk score.

7. Dyslipidemia and LA volume index (Table No. 16 & Figure. No. 15)

- The number of patients with dyslipidemia among LA volume index > 32 ml/m^2 was 86.84% and 36.11% among LA volume index < 32 ml/m^2. The p value was significant (0.000).

- LA volume was found to correlate positively with cardiovascular risk score based on age, gender, history of systemic hypertension, diabetes mellitus, hyperlipidemia, and smoking [153]. In our study the total study population with dyslipidemia was 53.63% with a significant P value

8. Obesity and LA volume index (Table No. 17 and Figure No. 16)

- 14.54% of Patients in this study population were obese. Of these only 13.15% had a larger LA volume index (> 32 ml/m^2), and 15.27% of obese patients had smaller LA volume index (< 32 ml / m^2) hence the p value was not significant (1.0).

- Body size is a major determinant of LA size. To adjust for this influence, LA size should be indexed to a measure of body size, most commonly to body surface area[32, 36]. It remains to be clarified if this approach attenuates obesity-related variations in LA volume, which may be prognostically significant[50].

LA volume index, on comparison with In-hospital management and events

9. Killip Class and LA volume index (Table No. 18 & Figure No. 17)

- In this study those with killip class 2 and 3 had a larger LA volume index (> 32 ml m^2) with a statistically significant p value (0.000).

- Moller JE, Hillis GS et al [92] in their study also has proven that Killip class was higher in patients with larger LA volume and killip class is a predictor of increasing LA volume with disease progression.

- Beinart et al[85] in a prospective study of 395 patients with AMI multivariate analysis identified 3 parameters i.e. restrictive filling, killip class, and LA volume as independent predictors of adverse outcome.

10. Inotropic support and LA volume index (Table No. 19 & Figure. No. 18)

- The number of patients who required inotropic support was higher (42.10%) in the group of LA volume > 32 ml/m^2 resulting in a significant p value (0.0327).

- Moller JE, Hillis GS et al [92] in their study have demonstrated that patients with LV dysfunction in the larger LA volume index group needed Inotropic support.

11. Re-MI and LA volume index (Table No. 20 & Figure No. 19)

- Re-MI occurred more commonly in the LA volume index > 32 ml/m^2 which was 26.31% while it was only 1.38% in the LA volume index < 32 ml/m^2 group resulting in a significant p value (0.0001).

- Moller JE, Hillis GS et al [92] have suggested in their study that most patients with multivessel and severe disease resulted in increased In-hospital Re – MI.

12. Arrhythmia and LA volume index (Table No. 21 & Figure No. 20)

- Arrhythmia were more common, (68.42%) in those with LA index volume > 32 ml/m^2 group compared to 18.05% in LA volume index <32 ml/m^2, resulting in a significant p value (0.000).

- The relationship between LA size and death has been demonstrated in high-risk groups, such as patients with atrial arrhythmias[131]. LA volume has been shown to predict AF in patients with cardiomyopathy (Tani T, Tanabe K, Ono M, et al). Further the relationship between LA volume and LA dimension was non linear[116,117], and it has been confirmed that LA volume represented a superior measure over LA diameter for predicting outcomes including AF[110,116,123] and provided prognostic information that was incremental to clinical risk factors[110].

- LA structural remodelling in some cases, may be related to higher ventricular rate and increased ventricular filling pressures rather than to the atrial tachyarrhythmia itself.[69, 70]

13. LV dysfunction and LA volume index (Table No. 22 & Figure No. 21)

- The number of patients who had LV dysfunction among LA volume index > 32 ml/m^2 is 89.47% which is much higher than among LA volume index < 32 ml/m^2 which was only 68.05% resulting in a significant p value (0.0245).

- LA volume >32 ml/m^2 was associated with increased incidence of CHF, independent of age, myocardial infarction, diabetes mellitus, hypertension, LV hypertrophy, and mitral inflow velocities.[121] LV dysfunction is more common with larger LA volumes.[92]

14. Mechanical complication and LA volume index (Table No. 23 & Figure No. 22)

- Mechanical complication which included moderate MR and VSR, was more common among LA volume index > 32 ml/$m^{2.}$ The p value was 0.124 which is not significant since the number of subjects under these categories were less (7.3%).

- LA volume index was greater in patients with moderate or severe mitral regurgitation, (Moller JE, Hillis GS et al.[92])

15. Death and LA volume index (Table No. 24 Figure No. 23)

- More deaths (21.05%) occurred in those with LA volume index > 32 ml/m^2, resulting in significant p value (0.0013).

- Relationship between LA size and death has been demonstrated in high-risk groups, such as patients with acute myocardial infarction [114,119]. More importantly LA diameter has also been shown to independently predict death in the general population [129].

- Two recent studies have investigated the relation between LA dilatation and all-cause mortality after AMI.[85,92] In a retrospective design including 314 patients, an increased LA volume index (> 32 ml/m^2) was associated with a higher all-cause mortality rate. LA volume index was a predictor of mortality after AMI, even after adjustment for conventional indices of systolic and diastolic function.[92]

ECHO Characteristics and LA volume index

16. Diastolic function and LA volume index (Table No. 25 & Figure No. 24)

- Proportion of subjects in Grade 2 and 3 diastolic dysfunction were 50% and 21.05% respectively in LA volume index > 32 ml/m^2 while it was only 20.83% and 2.77% in LA volume index < 32 ml/m^2. The p value was significant (0.0000).

- Greenberg B, Chatterjee K et al have suggested that with increased stiffness or non-compliance of the LV, LA pressure rises to maintain adequate LV filling[61], and the increased atrial wall tension leads to chamber dilatation and stretch of the atrial myocardium.

- Tsang TS, Barnes ME et al have established that, LA volume increases with severity of diastolic dysfunction. Furthermore LA volume is a barometer of LV filling pressure and reflects the burden of diastolic dysfunction in subjects without AF or significant valvular disease[33]

17. E/e' ratio and LA volume index (Table No. 26 & Figure No. 25)

- In this study, higher E/e' ratio (> 15) was found in patients with larger LA volume index (> 32 ml/m^2) than those with smaller LA volume index (< 32 ml/m^2) with significant p value (0.0004).

- LA volume index was found to correlate positively with tissue Doppler E/e′ based on the study by Teresa S M Sang MD et al.[153] Among patients in whom Doppler tissue imaging was performed, a modest correlation was found between the E/e' ratio and LA volume index.[92]

18. LVEF, and WMSI and LA volume index (Table No. 27, 28 & Figure 26, 27)

- In patients with LA volume index > 32 ml/m^2, the LVEF was lower (<40%) and WMSI (> 1.3%) was higher when compared with LA volume index < 32 ml/m^2, with a significant P value (0.0020 and 0.0001 respectively).

- LA volume was found to correlate positively with the grade of diastolic function, and negatively with LV ejection fraction in the study by Teresa S M Sang MD et al [153].In this study also patients with EF<40%, had more cardiac events in the group with larger LA volume index (>32 ml/m^2) than with smaller LA volume index (<32 ml/m^2) as in above study. . Moller JE, Hillis GS et al [92] proved that there is a positive correlation with the wall motion score index (WMSI) and LA volume index >32 ml/m^2 .

Study Limitations

- This study was a single center study
- Sample size was small.
- Entry criterion for this study was measurement of LA volume index. This may have introduced a selection bias. Although it appears unlikely that this could have affected the observed results, it may reduce their applicability to a more general population.
- Patients with previous history of myocardial infarction were excluded but subclinical coronary artery disease which was undiagnosed could have changed the diastolic function of the heart.
- Our study cannot assess the predictive value of late LA remodelling and subsequent morbidity, mortality as no long term follow up is available.
- LA is a complicated three-dimensional structure and the geometric algorithm used only estimates its volume. Hence Real time 3 D Imaging (RT – 3DE) may be a more accurate method of assessing LA volume and function.

- Doppler assessment may have resulted in the misclassification of diastolic function in a few cases, highlighting the limitations of such measurements. In particular, some patients with normal Doppler parameters had LA enlargement and vice versa. Assessment of mitral annulus motion appears to be particularly useful for assessment of diastolic function. Unlike other Doppler parameters of diastolic function, it appears to be relatively independent of preload and recently. It has been shown to be the most accurate non-invasive predictor of elevated LV filling pressure. However, the role of Doppler tissue imaging in the prediction of outcome after AMI remains to be defined.

Conclusion

CONCLUSION

1. The present study demonstrates that LA enlargement implies a poor prognosis in patients with AMI. It has proved as a predictor of In-hospital events in patients with acute myocardial infarction.

2. LA Volume index provides prognostic information incremental to clinical data and standard Echocardiographic predictors of outcome. Including LV systolic function and Doppler assessment of Diastolic Function.

3. Measurement of LA volume is simple and important tool which can be easily done and reproducible and may be incorporated in routine assessment of diastolic function.

4. Measurement of LA volume index could emerge as a simple and important tool for Risk stratification and as a guide for future surveillance and therapy in patients with Acute myocardial infarction if confirmed by perspective studies.

5. The utility of LA volume and function for monitoring cardiovascular risk and for guiding therapy is an evolving science and may prove to have a very important public health impact.

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13. Left Atrial Enlargement and Phasic Function in Patients Following Non–ST Elevation Myocardial Infarction Anita C. Boyd, BMedScHons, Arnold C.T. Ng, MBBS, Da T. Tran, BApplSc (MRS), Ee May Chia, MBBS, John K. French, BMedSc, MBChB, PhD, Dominic Y. Leung, MBBS, PhD, Liza Thomas, MBBS, PhD JASE Volume 23, Issue 12, Pages 1251-1258 (December 2010)

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APPENDIX I

ABBREVIATIONS

2D	-	Two Dimensional
3D	-	Three Dimensional
AF	-	Atrial Fibrillation
AMI	-	Acute Myocardial Infarction
AWMI	-	Anterior Wall Myocardial Infarction
BMI	-	Body Mass Index
BP	-	Blood Pressure
BSA	-	Body Surface Area
CABG	-	Coronary Artery By-Pass Graft
CAD	-	Coronary Artery Disease
CAG	-	Coronary Angiogram
CHF	-	Congestive Heart Failure
CI	-	Confidence Interval
CPK	-	Creatinine Phospho Kinase
CT	-	Computed Tomography
CVS	-	Cardio Vascular System
DT	-	Deceleration Time
DVD	-	Double Vessel Disease
HR	-	Hazard Ratio
HT	-	Height
IABP	-	Intra Aortic Balloon Pump

IWMI	-	Inferior Wall Myocardial Infarction
LA	-	Left Atrium
LAVI	-	LA Volume Index
LV	-	Left Ventricle
LVEDD	-	Left Ventricular End Diastolic Dimension
LVEF	-	Left Ventricular Ejection Fraction
LVESD	-	Left Ventricular End Systolic Dimension
MR	-	Mitral Regurgitation
MRI	-	Magnetic Resonance Imaging
PCI	-	Percutaneous Coronary Intervention
PR	-	Pulse Rate
RS	-	Respiratory System
SVD	-	Single Vessel Disease
TDI	-	Tissue Doppler Imaging
TVD	-	Triple Vessel Disease
V_P	-	Flow Velocity Propagation
VPC	-	Ventricular Pre-Mature Complex
VSR	-	Ventricular Septal Rupture
WMSI	-	Wall Motion Score Index
WT	-	Weight

APPENDIX II

PROFORMA

Name: **Age:**

Sex: **Occupation:**

Address:

Presenting Complaints:

Diagnosis: AWMI

IWMI

Risk Factors: Male Gender:

Smoker:

Hypertension:

Diabetes:

Obesity:

Hyperlipidemia:

Family History of CAD:

In Hospital Therapy: Thrombolysis:

Inotropic Therapy:

On Examination:

HT:	WT:	BMI:	BSA:
PR:	BP:		
CVS:	RS:		

Killip Class On Admission:

Investigations:

Complete Blood Count:

Urea: Creatinine:

Electrolytes: Lipids:

CPK/MB:

ECG:

Chest X-Ray PA View:

ECHO:

LVEF, %:

LVESD, mm:

LVEDD, mm:

WMSI:

Diastolic Function:

Ratio E/e':

LA Dimension, mm:

LA Volume, mL:

LA Volume index, mL/m^2:

MR:

In Hospital Events:

Death:

Re-MI:

Arrhythmias:

LV Dysfunction:

Mechanical Complication: VSR / MR

APPENDIX - III
MASTER CHART

S. No.	NAME	AGE	SEX	Diagnosis	smoker	SHT	DM	obesity	dyslipidemia	family history	thrombolysis	inotropic	Height	Weight	BSA	BMI
1	thanaraj	60	M	AWMI	+	+	+	-	-	+	+	-	168	65	1.7	23.04
2	venkatesan	45	M	AWMI	+	-	-	-	-	-	+	-	160	60	1.6	23.43
z3	Shankar	28	M	AWMI	+	-	-	-	+	-	+	-	166	74	1.8	26.9
4	Jayapal	55	M	IWMI	+	-	-	-	+	-	+	-	177	92	2	29
5	Sundari	36	F	IWMI	-	-	-	-	-	+	+	-	173	64	1.6	21.4
6	srinivasan	42	M	AWMI	-	-	+	-	+	-	+	-	160	60	1.6	23.4
7	murugesan	48	M	AWMI	+	-	-	-	-	-	+	-	150	50	1.4	22.2
8	rajangam	70	M	AWMI	-	+	-	+	-	-	+	+	170	85	1.95	29.8
9	radhakrishanan	63	M	AWMI	+	+	+	-	+	-	+	+	163	65	1.7	24.5
10	appas hussain	57	M	IWMI	+	-	+	-	+	-	+	+	162	52	1.5	19.8
11	krishanan	50	M	AWMI	-	-	-	-	-	-	+	+	156	56	1.5	23
12	krishanan	52	M	AWMI	-	-	-	-	-	-	+	-	169	69	1.75	24.2
13	loganathan	53	M	AWMI	-	+	-	-	+	-	+	-	165	65	1.65	23.8
14	Chandra	60	F	AWMI	+	+	+	+	+	-	+	+	151	79	1.75	34.6
15	abdul agis	64	M	AWMI	+	-	+	-	-	-	+	-	176	72	1.85	23.3
16	balaraman	58	M	IWMI	+	+	-	-	-	-	+	+	170	72	1.8	24.9
17	Santosh	50	M	AWMI	-	-	+	-	+	-	+	-	155	68	1.7	25
18	Rikiram	55	M	AWMI	-	-	-	-	-	-	+	-	163	65	1.7	24.52
19	armugam	55	M	AWMI	-	-	+	-	-	-	+	+	173	79	1.9	26.42
20	md. Basha	48	M	IWMI	-	-	-	+	-	-	+	+	170	90	1.95	31.14
21	Azhagu	46	M	IWMI	+	-	-	-	-	+	+	+	176	65	1.75	21.03
22	pappamal	60	F	IWMI	-	-	+	-	+	-	+	-	172	75	1.85	25.95
23	kanchana	68	F	IWMI	-	+	+	-	+	-	+	+	155	65	1.65	27.08
24	Robert	68	M	AWMI	+	+	+	-	+	-	+	+	155	63	1.65	26.75
25	Stephan	51	M	IWMI	-	+	+	-	-	-	+	-	173	57	1.7	19.06
26	Murugesan	62	M	IWMI	+	+	-	+	-	-	+	-	152	70	1.65	30.3

S. No.	NAME	AGE	SEX	Diagnosis	smoker	SHT	DM	obesity	dyslipidemia	family history	thrombolysis	inotropic	Height	Weight	BSA	BMI
27	Palani	47	M	AWMI	+	+	-	-	+	-	+	-	175	85	2	28.33
28	kashinathan	66	M	AWMI	+	-	+	+	-	-	+	-	151	54	1.5	23.68
29	dhandapni	70	M	AWMI	+	+	+	-	+	-	+	+	150	60	1.5	26.54
30	neelavati	73	F	AWMI	-	+	-	-	-	-	+	-	176	72	1.85	23.3
31	Mastahn	57	M	AWMI	+	+	-	-	-	-	+	-	159	65	1.65	25.79
32	Erumal	70	M	AWMI	-	-	-	-	+	-	+	-	165	65	1.65	23.89
33	vermuthu	45	M	IWMI	-	-	-	-	+	+	+	-	155	65	1.54	27.08
34	kothanapani	82	M	AWMI	+	+	+	-	+	-	+	+	155	65	1.54	27.08
35	james	39	M	AWMI	+	-	+	-	+	-	+	+	150	60	1.5	26.54
36	chandra	60	F	AWMI	-	-	-	-	+	-	+	-	171	65	1.71	22.03
37	ismail	38	M	AWMI	-	-	-	-	+	-	+	+	177	65	1.75	22.03
38	shahulameed	74	M	IWMI	-	-	+	-	+	-	+	-	159	72	1.7	28.57
39	raji	50	M	IWMI	-	-	-	-	-	-	+	+	161	72	1.75	24.91
40	arokyraj	52	M	AWMI	+	+	+	-	+	-	+	-	162	75	1.8	28.62
41	syed abulla	48	M	IWMI	-	-	-	-	+	-	+	-	166	74	1.8	26.9
42	shanmugam	50	M	IWMI	+	+	+	-	+	-	+	-	162	52	1.5	19.84
43	selvaraj	60	M	IWMI	-	-	-	+	-	-	+	-	170	63	1.7	21.79
44	kamala	70	F	AWMI	-	+	+	+	+	-	+	+	165	75	1.8	27.57
45	xavier	67	M	IWMI	-	-	+	-	-	+	+	-	177	92	2	29.39
46	radhammal	55	F	AWMI	-	+	-	-	+	-	+	+	170	65	1.7	21.03
47	ramu	40	M	AWMI	-	-	-	+	+	-	+	-	170	85	1.95	32.81
48	rengraj	32	M	AWMI	-	-	-	-	+	-	+	-	170	76	1.91	29.41
49	muthu	46	M	AWMI	+	-	-	-	+	-	+	+	150	49	1.54	21.8
50	anthony	53	M	IWMI	-	-	-	-	-	-	+	+	173	64	1.6	21.4
51	vallimmal	60	F	AWMI	-	+	-	+	+	-	+	+	165	65	1.65	23.89
52	kuthuputin	50	M	AWMI	+	+	+	-	+	-	+	+	165	65	1.65	23.89
53	shanmugavel	61	M	AWMI	-	-	-	+	-	-	+	-	163	85	1.85	31.69
54	dhandapni	46	M	AWMI	+	+	-	-	-	-	+	+	150	56	1.5	24.88
55	lakshmi	60	F	AWMI	-	+	+	-	+	-	+	+	151	54	1.5	23.68

S. No.	NAME	AGE	SEX	Diagnosis	smoker	SHT	DM	obesity	dyslipidemia	family history	thrombolysis	inotropic	Height	Weight	BSA	BMI
56	jamaluddin	61	M	AWMI	-	-	+	-	+	-	+	-	163	60	1.6	22.64
57	kahanbri	60	F	AWMI	-	+	-	+	-	-	+	-	163	84	1.85	31.69
58	velu	41	M	AWMI	-	-	-	-	+	+	+	+	175	85	2	28.33
59	ponnusamy	72	M	IWMI	+	+	-	-	-	-	+	-	168	65	1.7	23.04
60	keshvan	64	M	AWMI	-	+	+	-	-	-	+	-	161	72	1.75	24.91
61	anif	54	M	IWMI	-	-	-	-	-	-	+	+	170	90	1.95	31.14
62	kaushalya	55	F	AWMI	-	+	+	-	+	-	+	+	163	65	1.7	24.52
63	narayannan	65	M	IWMI	-	+	+	-	+	-	+	-	163	75	1.75	28.3
64	ravichandran	50	M	AWMI	+	-	-	-	+	-	+	-	166	74	1.8	26.9
65	dhanalakshmi	65	F	AWMI	-	+	+	+	+	-	+	+	163	85	1.85	31.69
66	ravi	52	M	IWMI	+	+	+	+	+	-	+	-	173	57	1.7	19.06
67	chellatai	75	F	AWMI	-	+	+	-	+	-	+	+	165	75	1.8	27.57
68	shakuntala	70	F	AWMI	-	-	-	-	+	-	+	-	150	60	1.5	26.66
69	shankar	46	M	IWMI	+	-	-	-	-	-	+	-	162	67	1.7	25.57
70	prabhavati	51	F	IWMI	-	-	-	-	-	-	+	-	170	49	1.45	16.95
71	xavier francis	42	M	AWMI	+	-	-	-	-	-	+	-	163	84	1.85	31.69
72	abdulla	48	M	AWMI	-	+	-	-	-	-	+	-	163	75	1.75	28.3
73	kolandi mary	75	F	IWMI	-	-	-	-	-	-	+	-	155	65	1.65	27.08
74	anbu	43	M	AWMI	+	-	+	-	+	-	+	-	168	65	1.7	23.04
75	mukundan	59	M	AWMI	-	-	-	-	-	-	+	-	150	50	1.4	22.22
76	shanta	58	F	AWMI	-	+	-	-	+	-	+	-	165	70	1.75	25.73
77	meena	35	F	AWMI	-	-	-	-	-	-	+	-	165	90	1.95	33.08
78	rafiq	59	M	AWMI	-	-	+	+	+	-	+	-	170	85	1.95	29.41
79	ponram	68	M	IWMI	+	+	+	-	+	-	+	-	173	57	1.7	19.06
80	emanuvel	32	M	AWMI	+	-	-	-	-	-	+	-	156	63	1.75	30.04
81	jesudoss	71	M	AWMI	+	+	+	-	+	-	+	-	170	89	2	30.79
82	sesammal	60	F	AWMI	-	+	+	-	+	-	+	+	165	73	1.75	26.83
83	george	51	M	AWMI	-	-	+	-	+	-	+	-	172	64	1.65	21.69
84	fathima	70	F	AWMI	-	+	+	+	+	-	+	-	165	90	1.95	33.08

S. No.	NAME	AGE	SEX	Diagnosis	smoker	SHT	DM	obesity	dyslipidemia	family history	thrombolysis	inotropic	Height	Weight	BSA	BMI
85	sundarbabu	60	M	AWMI	-	-	-	-	-	-	+	-	160	60	1.6	23.43
86	vallimmal	59	F	IWMI	-	+	+	+	+	-	+	-	156	50	1.45	19.76
87	damayendi	55	F	AWMI	+	+	-	-	+	-	+	-	170	72	1.8	24.91
88	dhanalakshmi	65	F	AWMI	-	+	+	-	-	-	+	+	165	70	1.77	25.7
89	murugan	50	M	AWMI	+	-	+	-	+	-	+	+	167	70	1.79	25.1
90	venkatesan	55	M	IWMI	-	-	-	-	-	-	+	-	160	70	1.7	27.34
91	chandra	65	F	AWMI	-	+	+	-	+	-	+	+	155	55	1.53	22.9
92	shakuntala	70	F	IWMI	-	+	-	-	-	-	+	-	150	50	1.4	22.22
93	vishalkshi	70	F	AWMI	-	+	-	-	+	-	+	+	168	85	1.95	29.41
94	arokydass	40	M	IWMI	+	-	-	-	-	-	+	-	163	65	1.7	24.52
95	rajendran	54	M	AWMI	+	-	+	-	+	-	+	+	155	68	1.7	25
96	ellappan	40	M	IWMI	+	-	-	-	-	-	+	-	170	70	1.75	24.22
97	sarvanan	43	M	AWMI	-	-	-	-	-	-	+	-	170	85	1.95	29.82
98	srinivasan	42	M	IWMI	+	+	+	-	+	-	+	-	163	75	1.75	28.3
99	sundari	56	F	IWMI	-	+	+	-	+	-	+	-	168	49	1.45	16.95
100	nahour miran	60	M	AWMI	-	+	+	-	+	-	+	-	175	85	2	28.33
101	mani	39	M	AWMI	+	-	-	-	-	-	+	-	151	54	1.5	23.68
102	jarrish	65	M	AWMI	-	+	+	-	+	-	+	+	161	72	1.75	24.91
103	subramaniyan	57	M	AWMI	-	+	-	-	+	-	+	-	170	85	1.95	29.82
104	santhanm	48	M	IWMI	-	-	-	-	-	+	+	-	173	64	1.6	21.4
105	kairunisaa	47	F	IWMI	-	-	-	-	+	-	+	-	169	57	1.7	19.06
106	annamalia	62	M	IWMI	-	-	+	-	-	-	+	-	163	65	1.7	24.52
107	arumugam	47	M	IWMI	+	-	-	-	+	-	+	-	166	74	1.8	26.9
108	salim	55	M	AWMI	+	-	+	-	+	-	+	-	172	64	1.65	21.69
109	kumayangani	48	M	AWMI	+	-	-	-	-	-	+	-	176	72	1.85	23.3
110	sulochana	57	F	AWMI	+	+	+	-	+	-	+	+	151	54	1.5	23.68

S. No.	Killip	CPK	CXR	EF	LVIDd	LVIDs	WMSI	DD	e/e'	LAV	LAVI	MR	Re MI	arrthytmia	LV Dysf	VSR	DEATH
1	1	raised	-	55	43	23	1.35	1	9.23	24	14.11	-	-	-	-	-	-
2	2	raised	congestion	35	42	29	1.64	1	6.75	30.19	18.86	trivial	-	-	+	-	-
3	2	raised	congestion	30	47	37	1.41	2	12.78	58.3	32.38	trivial	-	+	+	-	-
4	1	raised	-	50	43	28	1.11	0	4.25	48.6	24.3	-	-	-	+	-	-
5	1	raised	-	45	43	26	1.35	1	9.47	30.2	18.87	trivial	-	-	+	-	-
6	1	raised	-	35	44	27	1.35	1	9.87	45.6	24	-	-	-	+	-	-
7	1	raised	-	46	42	30	1.29	1	7.97	34	17.43	-	-	-	+	-	-
8	3	raised	congestion	30	43	32	1.7	1	5.57	33.2	17.02	trivial	-	-	+	-	-
9	2	raised	congestion	30	43	32	1.7	3	13.17	54.55	32.08	trivial	-	+	+	-	+
10	3	raised	congestion	45	40	25	1.58	2	11.2	48.09	32.06	mod	-	-	+	-	-
11	2	raised	congestion	40	38	29	1.52	2	14.34	20	13.46	-	-	-	+	-	-
12	2	raised	congestion	35	43	26	1.52	1	9.8	31.4	16.1	-	-	-	+	-	-
13	2	raised	congestion	40	42	36	1.29	1	9.45	30.2	18.3	-	-	+	+	-	-
14	2	raised	congestion	35	44	29	1.41	2	11.63	63.2	32.41	trivial	-	+	+	-	-
15	1	raised	-	45	37	24	1.3	1	5.17	61.01	32.97	-	-	-	+	-	-
16	2	raised	congestion	45	48	28	1.35	2	15.2	57.69	32.05	mild	+	-	+	+	-
17	1	raised	-	30	45	30	1.64	1	6.04	24.1	14.17	-	-	-	+	-	-
18	1	raised	-	45	46	35	1.29	2	13.5	24.2	14.23	-	-	-	+	-	-
19	2	raised	congestion	30	41	28	1.47	2	10.65	32.1	16.89	trivial	-	-	+	-	-
20	3	raised	congestion	35	48	36	1.47	2	13.06	31.1	15.94	-	-	+	+	-	-
21	2	raised	congestion	30	49	36	1.64	2	11.34	56.04	32.02	trivial	+	-	+	-	-
22	3	raised	congestion	45	43	30	1.35	2	16.2	59.23	32.01	trivial	-	-	+	-	-
23	3	raised	congestion	30	43	31	1.58	1	8.78	54.3	32.9	mild	-	+	+	-	+
24	2	raised	congestion	30	41	36	1.64	2	11.9	52.9	32.06	mild	-	+	+	-	-
25	1	raised	-	50	40	23	1.11	0	7.7	28.7	16.88	-	-	-	-	-	-
26	3	raised	congestion	45	44	31	1.3	2	10.02	52.9	32.06	trivial	+	-	-	-	-
27	1	raised	-	55	49	29	1.1	0	8.8	38.79	19.39	-	-	-	-	-	-

S. No.	Killip	CPK	CXR	EF	LVIDd	LVIDs	WMSI	DD	e/e'	LAV	LAVI	MR	Re MI	arrthytmia	LV Dysf	VSR	DEATH
28	1	raised	-	40	39	27	1.35	1	6.49	22.3	12.05	mild	-	-	+	-	-
29	3	raised	congestion	30	50	40	1.7	3	17.1	48.09	32.06	mod	-	+	+	-	+
30	1	raised	-	45	37	24	1.23	1	5.17	28.7	15.51	-	-	+	+	-	-
31	1	raised	-	65	34	16	1.1	0	6.4	36.4	18.66	-	-	-	-	-	-
32	3	raised	congestion	35	48	39	1.47	3	15.6	29.23	17.71	mild	-	+	+	-	-
33	1	raised	-	55	49	29	1.23	0	8.3	28.89	17.5	-	-	-	-	-	-
34	3	raised	congestion	30	43	31	1.6	1	8.76	53.6	32.48	mild	-	+	+	+	+
35	2	raised	congestion	30	50	40	1.7	3	16.2	48.01	32.6	mod	-	+	+	-	-
36	1	raised	-	50	46	32	1.29	0	8.9	28	16	-	-	-	-	-	-
37	1	raised	-	41	41	26	1.35	2	13.2	32	18.34	-	-	-	+	-	-
38	1	raised	-	50	46	32	1.29	0	8.9	28.2	16.58	-	-	-	-	-	-
39	1	raised	-	45	47	31	1.29	1	5	30.01	17.14	-	-	-	+	-	-
40	2	raised	congestion	30	46	32	1.6	3	14.5	15.75	32.08	-	-	+	+	-	-
41	1	raised	-	50	52	30	1.29	0	4.6	25.6	14.2	-	-	-	-	-	-
42	3	raised	congestion	45	40	25	1.58	1	11.45	48.02	32.01	-	+	-	+	-	-
43	1	raised	-	50	45	28	1.23	0	6.5	28.7	16.88	-	-	-	-	-	-
44	3	raised	congestion	35	45	29	1.64	2	16.02	57.62	32.01	trivial	-	+	+	-	-
45	1	raised	-	50	43	28	1.11	0	4.25	38.9	19.45	-	-	-	-	-	-
46	2	raised	congestion	30	49	36	1.7	2	13.78	56.1	32.05	trivial	-	+	+	+	-
47	1	raised	-	65	34	16	1.23	0	6.4	36.4	18.66	-	-	-	-	-	-
48	2	raised	congestion	40	48	38	1.29	1	4.76	28.9	14.82	-	-	-	+	-	-
49	3	raised	congestion	28	50	45	1.87	3	13.75	52.267	36.8	mild	-	+	+	-	+
50	2	raised	congestion	45	43	26	1.35	1	9.47	30.1	18.81	trivial	-	-	-	-	-
51	3	raised	congestion	40	42	36	1.36	1	6.56	23.1	14	-	-	+	+	-	-
52	2	raised	congestion	40	41	30	1.35	2	11.4	52.91	32.06	trivial	+	+	+	+	-
53	1	raised	-	50	39	28	1.3	2	12.56	29.4	15.89	-	-	-	-	-	-
54	3	raised	congestion	35	44	32	1.47	2	12.89	24.6	16.4	-	-	-	+	-	-
55	2	raised	congestion	35	48	35	1.47	2	14.02	48.07	32.04	-	-	+	+	-	-

S. No.	Killip	CPK	CXR	EF	LVIDd	LVIDs	WMSI	DD	e/e'	LAV	LAVI	MR	Re MI	arrthytmia	LV Dysf	VSR	DEATH
56	2	raised	congestion	35	44	31	1.47	1	7.54	24.5	15.31	-	-	-	+	-	-
57	1	raised	-	40	39	27	1.35	1	6.44	26.4	14.27	-	-	-	+	-	-
58	2	raised	congestion	40	48	38	1.47	1	4.76	38.7	19.35	trivial	-	+	+	-	-
59	1	raised	-	41	41	26	1.35	2	13.2	29.89	17.58	trivial	-	-	+	-	-
60	1	raised	-	50	39	28	1.2	2	12.56	25	14.28	-	-	-	-	-	-
61	2	raised	congestion	35	38	36	1.41	2	13.06	26	13.33	trivial	-	+	+	-	-
62	2	raised	congestion	30	33	24	1.6	2	10.8	54.67	32.15	trivial	-	+	+	-	-
63	1	raised	-	45	47	30	1.23	1	9.8	29.8	17.02	mild	-	+	+	-	-
64	1	raised	-	30	47	37	1.49	2	12.78	22.3	12.3	-	-	+	+	-	-
65	2	raised	congestion	50	39	28	1.3	2	6.44	32	17.29	-	-	-	-	-	-
66	2	raised	congestion	50	40	23	1.11	0	10.09	54.45	32.02	-	-	+	-	-	-
67	3	raised	congestion	35	45	29	1.64	2	15.09	57.75	32.08	-	-	+	+	-	+
68	2	raised	congestion	40	42	25	1.35	1	8.82	24.6	16.4	-	-	+	+	-	-
69	1	raised	-	50	52	30	1.29	0	4.66	54.9	32.29	mild	-	-	-	-	-
70	2	raised	congestion	45	44	28	1.29	2	9.62	27.89	19.23	mod	+	-	+	-	-
71	3	raised	congestion	35	44	32	1.64	2	12.89	29	15.67	-	-	-	+	-	-
72	1	raised	-	45	37	24	1.23	1	5.17	27	15.42	-	-	-	+	-	-
73	3	raised	congestion	40	42	36	1.35	1	9.45	26.9	16.3	mild	-	+	+	-	-
74	1	raised	-	55	43	24	1.35	0	7.6	25.1	14.76	-	-	-	-	-	-
75	1	raised	-	45	35	25	1.11	1	8	23.1	16.5	trivial	-	-	+	-	-
76	3	raised	congestion	35	45	39	1.49	3	15.6	24.5	14	trivial	-	+	+	-	-
77	2	raised	congestion	35	43	26	1.52	1	9.8	31.4	16.1	-	-	-	+	-	-
78	2	raised	congestion	40	48	38	1.47	1	4.76	28.4	14.56	trivial	-	-	+	-	-
79	1	raised	-	50	40	23	1.11	0	10.7	54.5	32.05	-	-	+	-	-	-
80	3	raised	congestion	45	46	28	1.41	1	15.9	56.01	32	trivial	-	+	+	-	-
81	2	raised	congestion	35	48	36	1.47	2	11.2	64.25	32.25	-	+	+	+	-	-
82	3	raised	congestion	25	64	59	1.64	3	15.2	56.13	32.07	trivial	+	+	+	-	-
83	1	raised	-	40	38	26	1.27	1	6.71	28.5	17.27	-	-	-	+	-	-

S. No.	Killip	CPK	CXR	EF	LVIDd	LVIDs	WMSI	DD	e/e'	LAV	LAVI	MR	Re MI	arrthytmia	LV Dysf	VSR	DEATH
84	2	raised	congestion	45	44	28	1.11	1	8	30.14	15.45	-	-	-	+	-	-
85	2	raised	congestion	35	42	29	1.64	1	6.75	23.56	14.72	-	-	-	+	-	-
86	3	raised	congestion	45	41	25	1.3	2	10.2	46.52	32.08	mod	+	-	+	-	-
87	2	raised	congestion	45	48	28	1.35	1	10.21	57.89	32.16	-	+	-	+	-	-
88	3	raised	congestion	35	42	30	1.64	1	9.8	56	32	trivial	-	+	+	-	+
89	2	raised	congestion	35	55	44	1.62	3	18	72.85	41.62	trivial	-	+	+	-	+
90	1	raised	-	63	44	24	1.11	0	7.07	24.6	14.47	-	-	-	-	-	-
91	2	raised	congestion	38	53	45	1.67	3	22.5	56.52	36.94	mild	+	-	+	-	+
92	1	raised	-	45	35	25	1.11	1	8	20.01	14.35	-	-	-	+	-	-
93	3	raised	congestion	30	43	32	1.7	1	5.57	29.4	15.07	-	-	-	+	-	-
94	1	raised	-	50	46	32	1.29	0	8.45	24.1	14.17	trivial	-	-	-	-	-
95	2	raised	congestion	30	45	32	1.64	1	6.04	28.41	16.71	-	-	+	+	-	-
96	1	raised	-	38	41	30	1.23	1	6.5	26.4	15.08	mild	-	-	+	-	-
97	1	raised	-	46	42	30	1.29	1	7.97	28.4	14.56	-	-	-	+	-	-
98	2	raised	congestion	45	47	30	1.3	2	9.8	56.16	32.09	trivial	-	+	+	-	-
99	2	raised	congestion	45	44	28	1.3	2	13.2	46.55	32.1	mild	-	+	+	-	-
100	1	raised	-	55	49	29	1.23	0	8.8	35.67	17.83	-	-	-	-	-	-
101	2	raised	congestion	35	48	35	1.47	1	7.04	32.1	21.4	mod	-	-	+	-	-
102	1	raised	-	50	39	28	1.3	2	12.56	25	14.28	-	-	-	-	-	-
103	1	raised	-	46	42	30	1.29	1	7.97	28.4	17.58	-	-	-	+	-	-
104	2	raised	congestion	45	44	26	1.35	1	9.47	31.1	18.81	-	-	-	+	-	-
105	1	raised	-	55	43	20	1.25	0	7.6	25.1	14.76	trivial	-	-	-	-	-
106	1	raised	-	50	46	28	1.29	0	6.8	29.89	17.58	-	-	-	-	-	-
107	1	raised	-	50	52	30	1.11	0	4.66	25.6	14.2	trivial	-	-	-	-	-
108	1	raised	-	40	38	26	1.37	1	6.71	28.5	17.27	-	-	-	+	-	-
109	1	raised	-	45	37	24	1.39	1	5.17	61.01	32.97	-	-	-	+	-	-
110	2	raised	congestion	35	48	35	1.47	2	14.02	48.07	32.04	trivial	-	+	+	-	-