"UTILITY OF REAL TIME THREE-DIMENSIONAL ECHOCARDIOGRAPHY IN POST MYOCARDIAL INFARCTION VENTRICULAR SEPTAL RUPTURE"

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"learn to heal"

CERTIFICATE

This is to certify that the dissertation entitled **"UTILITY OF REAL TIME THREE-DIMENSIONAL ECHOCARDIOGRAPHY IN POST MYOCARDIAL INFARCTION VENTRICULAR SEPTAL RUPTURE"** is the bonafide original work of **DR.P.GNANAVEL** in partial fulfillment of the requirements for D.M. Branch-II (CARDIOLOGY) examination of THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY to be held in August 2008.The period of post-graduate study and training was from August 2005 to July 2008.

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DECLARATION

I **Dr.P.GNANAVEL,** solemnly declare that this dissertation entitled, **"UTILITY OF REAL TIME THREE-DIMENSIONAL ECHOCARDIOGRAPHY IN POST MYOCARDIAL INFARCTION VENTRICULAR SEPTAL RUPTURE"** is a bonafide work done by me at the department of Cardiology, Madras Medical College and Government General Hospital during the period 2005 – 2008 under the guidance and supervision of the Professor and Head of the department of Cardiology of Madras Medical College and Government General Hospital, Professor **R.Alagesan M.D.D.M.** This dissertation is submitted to The Tamil Nadu Dr.M.G.R Medical University, towards partial fulfillment of requirement for the award of **D.M. Degree (Branch-II) in Cardiology.**

Place : Chennai

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Dr.P.GNANAVEL

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CONTENTS

INTRODUCTION

Ventricular septal rupture after an acute myocardial infarction is a life threatening complication which carries a high mortality. Ventricular septal rupture results from full thickness infarction of the interventricular septum followed by sufficient necrosis to result in the septal rupture. It is one of the three mechanical complications that can occur following myocardial infarction. The others are free wall rupture, which is usually rapidly fatal, and papillary muscle rupture, which results in sudden onset of mitral regurgitation. The respective frequencies of these complications are in approximate proportion to the respective volumes of muscle that are available to be involved, so that free wall rupture is most common, ventricular septal rupture next, and papillary muscle rupture least.

The differential diagnosis of postinfarction cardiogenic shock should exclude VSR, free ventricular wall rupture and rupture of the papillary muscles. In a recent report of the SHOCK (*SH*ould we emergently revascularize *O*ccluded *C*oronaries for shoc*K*) trial registry of 1160 patients with cardiogenic shock 74.5% of patients had predominant left ventricular failure, 8.3% had acute mitral regurgitation, 4.6% had ventricular septal rupture, 3.4% had isolated right ventricular shock, 1.7% had tamponade or cardiac rupture, and 8% had shock that was a result of other causes. Cardiogenic shock is most often associated with anterior myocardial infarction. In the SHOCK trial registry 55% of infarctions were anterior, 46% were inferior, 21% were posterior, and 50% were in multiple locations.

Reperfusion therapy has reduced the incidence of septal rupture. The event occurs 2-8 days after an infarction and often precipitates cardiogenic shock. Clinical studies report an average time of 2.6 days from infarction to septal rupture. However, recent data suggest that the initial treatment of MI with thrombolytics may affect both the time between infarction and septal rupture and outcome. The early use of thrombolytic agents may lead to reopening of the occluded vessels, thus reducing the incidence of VSR.

The age range of patients who sustain a postinfarction VSR is wide, from 44-81 years. Men are affected more commonly than women, although septal rupture is more common in women than would be predicted based on the prevalence of CAD alone. In postinfarction VSR the sites of rupture are the anterior septum (60%) and the posterior septum (40%). The most consistent finding is a murmur. Diagnosis is confirmed with the aid of echocardiography and the presence of a left-to-right shunt. Of patients treated without surgery, 90% die. The surgical mortality rate for anterior defects is 10-15% and for posterior defects is 30-35%.

To avoid the high morbidity and mortality associated with this disorder, patients should undergo emergent surgery. Concomitant coronary artery bypass may be required. Rapid diagnosis, aggressive medical management, and surgical intervention are required to optimize recovery and survival. Long-term survival can be achieved in patients who undergo prompt surgery.

The location and the size of the septal defect as well as haemodynamic conditions are considered important information and may influence patient prognosis. The exact location, shape and size of the VSR may be challenging to define accurately using 2DTTE because it views cardiac structures and any associated defects in two dimensions only. 2D echo requires mental conceptualization of a series of multiple orthogonal or tomographic images into an imaginary multidimensional reconstruction for better understanding of complex intracardiac structures and their spatial relation with surroundings.Three dimensional reconstructions of multiplane TEE images has been found useful in evaluating the location and size of the VSR but the technique is semi invasive and not completely without risk it would be beneficial if the information could be obtained with live 3DTTE. Real time 3D echocardiography is the only on-line 3D method based on real time volumetric scanning, as compared with other 3D imaging techniques such as MRI and CT, which are based on post- acquisition reconstruction not on volumetric scanning.

Comprehensive non invasive assessment of the location, shape, size of the septal rupture, which could be clearly visualized en face from both left and right ventricular aspects is

possible with Live 3DTTE. So, utilization of Real time 3D ECHO in diagnosis of VSR helps to complete visualization and comprehensive assessment of cardiac anatomy, location, shape, size, extent of VSR. 3D Echo serves as an electric knife, allowing left ventricular en face views to delineate the size and position of VSR. Once the defect can be determined, multiple imaging planes and rotation of the ventricular septum can be undertaken. 3D colour flow imaging can improve the depiction of the number, size, and position of the VSR inrelation to the septum.

AIM OF THE STUDY

This study aims at utilizing Real time three-dimensional transthoracic echocardiography (RT3DE) technique for comprehensive assessment of

- location, size, shape of post myocardial infarction ventricular septal rupture.
- Pathomorphology of post myocardial infarction ventricular septal rupture.
- cardiac anatomy and cardiac pathophysiology after acute myocardial infarction.
- Clinical characteristics of patients with Acute myocardial infarction complicated by ventricular septal rupture and its correlation with the findings of 3D echocardiography.

This study also aims to compare the findings between 2D TTE and Real time three-dimensional transthoracic echocardiography (RT3DE).

REVIEW OF LITERATURE

History

Latham first described ventricular septal rupture at autopsy in 1847, but he did not make the association between acquired VSR and coronary artery disease (CAD). Brunn made the first antemortem diagnosis of acquired VSR in 1923, and, in 1934, Sager established the clinical association between MI and VSR.

Incidence

In the era before reperfusion therapy, septal rupture complicated 1 to 3 percent of acute myocardial infarctions.[1-5] Among the 41,021 patients in the Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries (GUSTO-I) trial, ventricular septal rupture was suspected in 140 patients (0.34 percent) and confirmed by a retrospective review in 84 (0.2 percent).[6] Thus, reperfusion therapy has decreased the incidence of septal rupture.[6]

Risk Factors

Septal rupture occurs more frequently with anterior than other types of acute myocardial infarction.[2,6-9] The age range of patients who sustain a postinfarction VSR is wide, from 44-81 years. Risk factors for septal rupture in the era before thrombolytic therapy included hypertension, advanced age (60 to 69 years),10 female sex, [10-12] and the absence of a history of angina or myocardial infarction.[1,2,13-15] Angina or infarction may lead to myocardial preconditioning as well as to the development of coronary collaterals, both of which reduce the likelihood of septal rupture. In patients

undergoing thrombolysis, advanced age, female sex, and the absence of smoking are often associated with an increased risk of septal rupture, whereas the absence of antecedent angina has not been associated with an increased risk.

In the GUSTO-I trial, there was a nonlinear relation between the systolic and diastolic blood pressures at enrollment and septal rupture, since hypertension (a blood pressure of more than 130/75 mm Hg) and extensive myocardial infarction and right ventricular infarction (which are causes of hypotension) are also risk factors for septal rupture.

Pathogenesis

The septum adjacent to the rupture is often thin and necrotic. Without reperfusion, coagulation necrosis develops within the first three to five days after infarction, with numerous neutrophils entering the necrotic zone. The neutrophils undergo apoptosis and release lytic enzymes, hastening the disintegration of necrotic myocardium. The pathogenic process of the rupture changes over time. During the first 24 hours, coagulation necrosis is just beginning and there are relatively few neutrophils within the infarcted tissue. Early ruptures occur in infarcts with large intramural hematomas that dissect into tissue and rupture. If patients survive for several weeks, the septum becomes fibrotic.

Becker and van Mantgem classified the morphology of free-wall rupture into 3 types, which are also relevant to ventricular septal rupture[16]: type I ruptures have an abrupt tear in the wall without thinning; in type II, the infarcted myocardium erodes before rupture occurs and is covered by a thrombus; and type III has marked thinning of the myocardium, secondary formation of an aneurysm, and perforation in the central portion of the aneurysm.

The size of septal rupture ranges from millimeters to several centimeters. The two pathological types of ventricular rupture are simple and complex. In a simple rupture there is a through and through opening connecting the two ventricles, without gross haemorrhage or laceration and with the right and left ventricular openings at about the same horizontal level of the ventricular septum. The perforation is at the same level on both sides of the septum. Septal ruptures in patients with anterior myocardial infarction are generally apical and simple.

A complex rupture is an interventricular communication with a convoluted course, with a tract that might extend into regions remote from the primary acute myocardial infarction site, and with haemorrhage and disruption of myocardial tissue. Extensive hemorrhage with irregular, serpiginous tracts within necrotic tissue characterizes complex septal rupture.[7,13] In patients with inferior myocardial infarction, septal ruptures involve the basal inferoposterior septum and are often complex. Complex morphology is more common in ruptures complicating inferior myocardial infarct while simple morphology is more common after anterior myocardial infarction.[31]

Pathophysiology:

The blood supply to the septum originates from branches of the left anterior descending coronary artery, the posterior descending branch of the right coronary artery, or the circumflex artery when it is dominant. An infarction associated with a VSR is usually transmural and extensive. Approximately 60% of septal ruptures occur with infarction of the anterior wall; 40% occur with infarction of the posterior or inferior wall. Posterior VSR may be accompanied by mitral valve insufficiency secondary to papillary muscle infarction or dysfunction. At autopsy, patients with VSR usually show complete coronary artery occlusion with little or no collateral flow. The lack of collateral flow may be secondary to associated arterial disease, anatomic anomalies, or myocardial edema. Sometimes, multiple septal perforations occur. These may occur simultaneously or within several days of each other. Ventricular aneurysms are commonly associated with postinfarction VSR and contribute significantly to the hemodynamic compromise in these patients.

Hemodynamics

Septal rupture results in a left-to-right shunt, with right ventricular volume overload, increased pulmonary blood flow, and secondary volume overload of the left atrium and ventricle. As left ventricular systolic function deteriorates and forward flow declines, compensatory vasoconstriction leads to increasing systemic vascular resistance, which, in turn, increases the magnitude of the left-to-right shunt. The degree of shunting is determined by the size of the septal rupture, the level of pulmonary vascular resistance and systemic vascular resistance and the ratio of the two, and left ventricular and right ventricular function. As the left ventricle fails and the systolic pressure declines, left-to-right shunting decreases and the fraction of the shunt diminishes.

Natural history

The natural history of postinfarction VSR is greatly influenced by hypertension, anticoagulation therapy, advanced age, and, possibly, thrombolytic therapy. The natural course in patients with postinfarction VSR is well documented and short. Most patients die within the first week; almost 90% die within the first year. Reports indicate that fewer than 7% of patients are alive after 1 year. This grim prognosis results from an acute volume overload exacted on both ventricles in a heart already compromised by a large MI and occasionally by extensive CAD in sites other than that already infarcted. In addition, superimposed ischemic mitral valve regurgitation, a ventricular aneurysm, or a combination of these conditions may be present, which further compromises heart function. The depressed left ventricular function commonly leads to impaired peripheral organ perfusion and death in most patients.

A few sporadic reports indicate that some patients with medically treated postinfarction VSR live for several years. Although many medical advances have been made in the nonsurgical treatment of these patients, including intra-aortic balloon counterpulsation (IABCP), these methods have not replaced the need for surgery.

Angiographic Findings

Some studies have found that septal rupture is associated with multivessel coronary artery

disease.[2,7] However, others found a high prevalence (54 percent) of single-vessel disease among patients with ventricular septal rupture.[8,19] Ventricular septal rupture is likely to be associated with total occlusion of the infarct-related artery.[3,6,8] A post mortem study by Mann and Roberts compared hearts from victims of acute myocardial infarction with and without ventricular septal rupture. They found that more epicardial coronary arteries were narrowed in those without rupture than those with, indicating that patients with diffuse disease are less likely to develop septal rupture. In a

clinical study the coronary angiographic findings in 91 patients with ventricular septal rupture were compared with angiography in 123 infarct survivors without septal rupture. Rupture was associated with a higher incidence of single vessel disease and less evidence of collateral circulation.[32] This was confirmed in a more recent but smaller Swiss study.[33]

It has also been demonstrated that multi-vessel disease is more common when rupture complicates an inferior infarct rather than an anterior infarct.[34]

In the GUSTO-I study, total occlusion of the infarct-related artery was documented in 57 percent of patients with ventricular septal rupture, as compared with 18 percent of those without ventricular septal rupture[6]. Collaterals are less often evident in patients with ventricular septal rupture, supporting the hypothesis that collateral circulation reduces the risk of rupture of the cardiac free wall as well as septal rupture.

Time Course

Without reperfusion, septal rupture generally occurs within the first week after infarction [3,5,7,19,20] There is a bimodal distribution of septal rupture, with a high incidence on the first day and on days 3 through 5 and rarely more than two weeks after infarction. The advent of widespread use of thrombolysis has had a dramatic effect upon the nature of ventricular septal rupture. In the early days of thrombolysis it was thought that the incidence may be increased, but it has subsequently been demonstrated repeatedly

that the incidence is significantly reduced. The median time from the onset of symptoms of acute myocardial infarction to rupture is generally 24 hours or less in patients who are receiving thrombolysis.[21] The median time from the onset of infarction to septal rupture was 1 day (range, 0 to 47; 94 percent of cases were diagnosed within 1 week) in the GUSTO-I trial and 16 hours in the Should We Emergently Revascularize Occluded Coronaries in Cardiogenic Shock (SHOCK) trial.[6,22] Although thrombolytic therapy reduces the size of the infarct, it may in some cases promote hemorrhagic dissection in the myocardium, accelerating the onset of septal rupture. In the GUSTO-I (global utilization of streptokinase and t-PA for occluded coronary arteries) trial there was an incidence of 0.2% of ventricular septal rupture in over 41 000 patients, a 5–10 fold reduction compared with the pre-thrombolytic era**.** [35]

However, the nature of presentation has changed. Whereas the average time interval between infarction and rupture used to be 5–6 days, it is now closer to one day. [36] As we have already seen, the surgical mortality has increased at the same time. It is likely that the nature of the patients coming to surgery has changed. The thrombolytic treatment may increase the proportion of ruptures that are complex rather than simple, and therefore more difficult to repair. Furthermore, patients in the first 24–48 hours after infarction are probably less well able to sustain the insult of surgery than they would be a week or so later.

Clinical Manifestations

Symptoms of septal rupture include chest pain, shortness of breath, and those associated with low cardiac output and shock.[2,14] Acute septal rupture produces a harsh, loud holosystolic murmur along the left sternal border, radiating toward the base, apex, and right parasternal area, and a palpable parasternal thrill in half of patients.[19,23] With

cardiogenic shock and a low-output state complicating septal rupture, there is rarely a thrill, and the murmur is difficult to identify because turbulent flow across the defect is reduced. Right and left ventricular S3 gallops are common. The pulmonic component of the second heart sound is accentuated by PHT. Tricuspid regurgitation may also be present. Biventricular failure generally ensues within hours or days.As compared with acute mitral regurgitation, septal rupture has a loud murmur, a thrill, and right ventricular failure but is less often characterized by severe pulmonary edema. In patients with a low cardiac output, distinguishing between these two entities can be difficult. In addition, severe mitral regurgitation may occur in 20 percent of patients with septal rupture.[24-26]

Diagnosis

Pump failure in patients with myocardial infarction may be related to the major mechanical complications, such as ventricular septal rupture, papillary-muscle rupture, or free-wall rupture. Alternatively, it results from the infarction or ischemia of a large area, ischemic mitral regurgitation, right ventricular dysfunction, or hypovolemia. Doppler echocardiography is generally diagnostic.[26,27,30] Doppler techniques can be used to define the site and size of septal rupture, left and right ventricular function, estimated right ventricular systolic pressure, and the left-to-right shunt.[28] The sensitivity and specificity of color Doppler echocardiography have been reported to be as high as 100 percent. Three dimensional echocardiography has a role in both the quantitative and qualitative assessment of ventricular septal rupture. Three dimensional echocardiography overcomes the

limitations of image plane positioning inherent in conventional 2DE and offers a more precise approach to measurement of ventricular septal rupture, area.

No surgical treatment was available until 1957, when Cooley et al performed the first successful surgical repair of VSR in a patient 9 weeks after the diagnosis. Unfortunately, the patient died 6 weeks later. Principal treatment of post infarction VSR during the early 1960s consisted of aggressive medical management, although it was well known that survival was rare after medical treatment alone. Surgical therapy was generally reserved for patients who survived at least 6 weeks, primarily to allow for scarring of the edges of the defect. A secure and long-lasting closure was thought to occur if the edges of the VSR were strong enough to hold the sutures. By the late 1960s, early surgical repair was proposed for patients whose conditions were deteriorating despite medical therapy. The timely introduction of better prosthetic material significantly contributed to the successful surgical repair of acute VSR.

More recently, improved surgical techniques (eg, infarctectomy), myocardial protection, and better perioperative mechanical and pharmacological support have helped to lower mortality rates. In addition, the development of surgical techniques to repair perforations in different areas of the ventricular septum have led to improved results in the management of patients with post infarction VSR. So, preoperative detection of VSR by 3D ECHO will be helpful for the surgeon in better planning .

REAL TIME THREE DIMENSIONAL ECHOCARDIOGRAM IN VSR:

Whereas, conventional two dimensional (2D) Echocardiography is crucial to our understanding of the complex anatomy and three dimensional (3D) spatial relationships of cardiac structures, it requires the mental integration of a limited number of 2D imaging planes. This mental 3D reconstruction is inherently variable according to observer experience and

expertise, and can only be described to other clinicians (such as surgeons) rather than displayed reproducibly. The display of cardiac anatomy in three dimensions from any perspective would have clear advantages over conventional 2D imaging and provide an insight into the functional and anatomic properties of cardiac structures.

Recent advances in ultrasound and computer technology have been combined such that dynamic 3D echocardiography imaging is now a practical reality[37,38]. Three dimensional echocardiography (3DE) has been shown to be more accurate than 2DE in the quantification of cardiac volumes. These studies used either manually contoured, static "wire frame" reconstructions or dynamic "volumetric" automated reconstruction technology that is now commercially available.

The benefits of 3 dimensional echocardiography are particularly well suited to the study of the ventricular septal rupture. The assessment of patients with ventricular septal rupture is one of the most promising clinical applications of this technology.

The art of technology in RT3D echo, consists of

1. Transducer design **2.**Beam forming in three spatial dimensions.

3.Display of 3D information**.** **4.** Quantification in three dimensions.

1. Transducer design -Breakthrough in 3D technology is possible by piezoelectric crystals development.Conventional transducer- consist of 64 to 128 elements . These elements are arranged along a single row. 3D transducer-has more than 3000 elements arranged in rows and columns. 30-60 crystals are arranged in rows. These elements are electrically independent. 3D transducer has 150-Boards. In these dense array real-time 3D transducer, each square represents an element and entire crystal of the transducer head is sampled or covered with elements. The micro beam former is required for this arrangement to provide a communication of all of the (3000) elements to the ultrasound system. So, beam steering is very powerful.

2. Beam forming in three spatial dimensions- Beam forming constitutes the steering and focusing of transmitted and received scan lines. Each element must have independent electrical control by the ultrasound system. Cable size is reduced and power consumption is reduced, by micro beam formation.

3.Display of 3D information- where as in 2D display which has rows and blocks of pixels(picture elements) in 3D data set it consists of bricks and pixels called volume elements or voxels. 3D Data set (collection of voxels) can be rotated with respect to the computer screen.

Cropping-is a process used to cut into volume and make some voxels invisible.

(E.g.- one can cut away LA to visualize the Mitral valve.) Volume rendering- 3D data sets of voxels are turned into 2D images.

4.Quantification in three dimensions- 3D quantification of LV typically employs a surface rendered mesh. It allows accurate computation of volume, regional wall motion, and regional synchrony. Entire extend of the LV is taken into account, so no foreshortening errors or assumption of LV volume.

Artifacts in RT3D-Echo includes Ringing , reverberations, shadowing, attenuation artifacts. The constraints of 3D images are bounded by 1-Frame rate. 2-Three dimensional volume size. 3- Image resolution.

2D Echo images are obtained with 3D echo probe and then switched to live 3D Echo.

3D spatial modes consists of

1- Live 3D mode –instantaneous

2- Live 3D mode zoom-instantaneous

3- Full volume –gated.

4- 3D colour Doppler- gated.

Live 3D mode/zoom –instantaneous -The system scans in real time 3 dimensions. If the transducer comes off the chest –the image disappears. The volume pyramid may be reduced to zoom in three dimensions- Live 3D zoom-instantaneous RT3D is the only on-line 3D method based on real time volumetric scanning, as compared with other 3D imaging techniques like MRI, CT.

Full volume –gated. Gating allows a technique to generate wider volumes while maintaining frame rate. Gating is done by stitching 4 (or more) gates together in full- volume mode. This can generate > 90 degree scanning volumes at frame rates >30 Hz.

DATASET ACQUISITION, PROCESSING AND RECONSTRUCTION

A 3D dataset is composed of anatomical information from multiple 2D cross sectional images. For reconstruction of the ventricular septum and VSR in adult patients, transthoracic echocardiography (TTE) is the routine approach for 2D image acquisition as it offers a relatively stable site for the imaging probe and superior resolution of the ventricular septum and VSR. Images from transthoracic echocardiography (TTE) are interfaced with a 3D computer system which incorporates the steering logic for acquisition of a rotational dataset and software for 3D reconstruction and display.

Optimal temporal and spatial registration is achieved by ECG and respiratory gating. Offline processing involves the conversion from polar to cubic Cartesian co-ordinates and interpolation of missing information between 2D slices. From the resultant dataset, novel 2D cut planes in any orientation can be selected (any plane echo) and multiple parallel cross sectional 2D slices can be generated in any desired plane (Para plane echo). A volume rendered 3D image of the ventricular septal rupture can be reconstructed from any perspective. Threshold limits are used to separate cardiac structures from blood pool and background.

IMAGE DISPLAY AND ANALYSIS

The digitized data were reformatted and interpolated into a cubic data set by filling in the gaps between pixels to create individual volume elements or voxels. This cubic data set could then be rotated in any direction, allowing unlimited cut planes irrespective of the original ultrasonographic window. Once a cut plane was chosen, a volume-rendered three-dimensional image was produced by a combination of distance, gradient and texture shading.

For our study a parasternal long axis and short-axis, apical 4 chamber view of the interventricular septum (IVS) at the level of the VSR was used to view the defect (as if looking from the left ventricle side and from RV side).

In managing patients with VSR , it is essential to obtain reliable information about site, shape and area although conventional 2-dimensional echocardiography is still useful for evaluating VSR in clinical use. Identification and measurement of the VSR at times may be inaccurate with 2D Echo. With the recent introduction of a novel, high-speed, volumetric scanner system, real time, 3-dimensional echocardiography (RT3DE) could be used to display the IVS and VSR and its relation to neighboring structures in real-time. It has great potential in assessing morphologic characteristics of the VSR and in determining the exact location, size, shape and extent.

Imaging postinfarction VSR.

Live 3-D is used to interrogate the ventricular septum for defects using parasternal and apical windows. Once identified, the VSR can be evaluated for circumference and spatial orientation to other adjacent intracardiac structures in detail. After detecting the defect, use the 3-D color technique to confirm the shunt's size and location. To obtain clear shunt flow, arrhythmias to be avoided during data acquisition.

MATERIAL AND METHODS.

This study was performed in the Department of Cardiology, Government General Hospital, Chennai, during the year 2005 – 2008, after approval from the Institutional Ethical Committee, Madras Medical College, Chennai-3.(Ref- K.Dis.No 25406/P&D3/Ethics/Dean/GGH/07)

STUDY INDICATION

Study indication was for the comprehensive assessment of cardiac anatomy and pathomorphology of postmyocardial ventricular septal rupture and to analyze the clinical characteristics and its correlation with 3D echocardiography..

STUDY GROUP SELECTION

Study groups are those who were admitted in coronary care unit (CCU) with acute myocardial infarction complicated by VSR. All cases with VSR were complication of STEMI.

VSR	Male	Female	Total
AMI- without VSR	2221	1281	3502
AMI with VSR	19(43.2%)	25(56.8%)	**44**
Total	2240	1306	**3546**
VSR (%)	Male	Female	Total
No	99.15179	98.08576	
Yes	0.848214	1.914242	
Total	100	100	

Patients suspected of VSR were carefully evaluated by history taking, physical examination and laboratory tests, including electrocardiography chest radiographs, and echocardiography.

Color Doppler 2D echo was done in all cases.

CLINICAL EVALUATION

The study group consisted of 44 VSR patients consisting of 19 males and 25 Females, (Range 45 to 80. mean age= 61.95). Patients with acute ST elevation myocardial infarction were admitted in coronary care unit (CCU) and those within the time window of less than 12 hours were thrombolysed with streptokinase 1.5 million units intravenously after excluding contraindications. Standard medical treatment was given according to the guidelines. Presenting symptoms were analyzed in all cases. Risk factors like systemic hypertension, diabetes mellitus, smoking, and dyslipidaemia, family history of CAD, prior CAD, prior aspirin use, and prior surgical coronary revascularization were analyzed. Detailed clinical examination was done in all cases. ECG, X-ray chest, 2D echocardiogram were done in all cases on admission. ECG was taken in all cases periodically to look for new changes, arrhythmias, persistence of ST segment elevation. Cardiac enzymes were done in all cases. Patients suspected of VSR were done 2D echocardiogram immediately and presence of VSR was confirmed and at the same time all cases were analyzed by 3D echocardiogram.

Day of onset of VSR was noted in all cases. Hemodynamic status was monitored in all patients. Periodic blood pressure, heart rate, killip class, respiratory rate, and renal parameters noted at the time of admission and with the onset of VSR.

ECHOCARDIOGRAPHY STUDY -2D TRANSTHORACIC ECHOCARDIOGRAPHY

All patients admitted in coronary care unit with underwent 2D Echocardiographic evaluation at the time of admission and those suspected of VSR were again underwent 2D Echo and confirmed the presence of VSR. Wall motion abnormality in each segments, wall motion score index, LV ejection fraction by modified simpson method, diastolic dysfunction, RV function, LVH, pericardial effusion, and presence of Mitral regurgitation were noted in all cases. Location of VSR, size if possible, extend were analyzed by 2D and colour Doppler.

REAL TIME THREE DIMENSIONAL ECHOCARDIOGRAPHY IN VSR.

In each patient, RT3DE is performed immediately after 2D study using a Philips iE 33 ultrasound machine with an X3-1 probe. This probe is unique as it contains 3000 elements arranged in a rectangular format. Each transducer element is less than the size of the human hair. The foot print of the RT3DE probe is almost the same size as that of the 2D echocardiography probe[46].

RT3DE examination is performed from the same windows that are used for 2D echocardiography, namely parasternal long axis, parasternal short axis, apical, sub costal & suprasternal views. Hence the plane of examination and the views are exactly similar to that of 2D echocardiography[43].

Initially parasternal long axis short axis views of ventricular septum and VSR was obtained with routine 2D echo. Then it was switched over from 2 dimensional Echo to 3 dimensional Echo.

Two-dimensional Echocardiography examinations were performed with patients in the left lateral decubitus position. Electrocardiograms were simultaneously recorded in all subjects. A standard parasternal left ventricular long and short-axis view,A4C view was

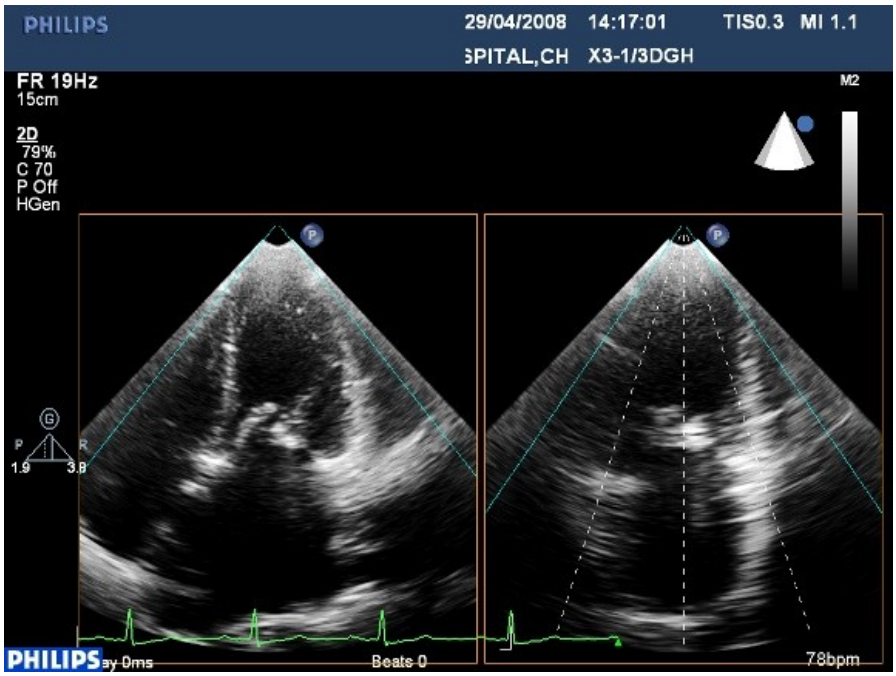
obtained, adjusting the transducer for optimal view of the VSR. New ultrasound equipment (Philips iE.33) was used to permit RT3DE of a ventricular septal rupture in the parasternal, apical, and sub costal views, We carefully adjusted the ventricular septal rupture to the center of the screen, opened the "live 3D" functional button, acquired pictures in real-time, then randomly rotated the picture, carefully observing the ventricular septal rupture and its relation to the neighboring structures. During the examination we paid special attention to adjusting the parameters to optimally display the 3-dimensional ventricular septal rupture at any given time.

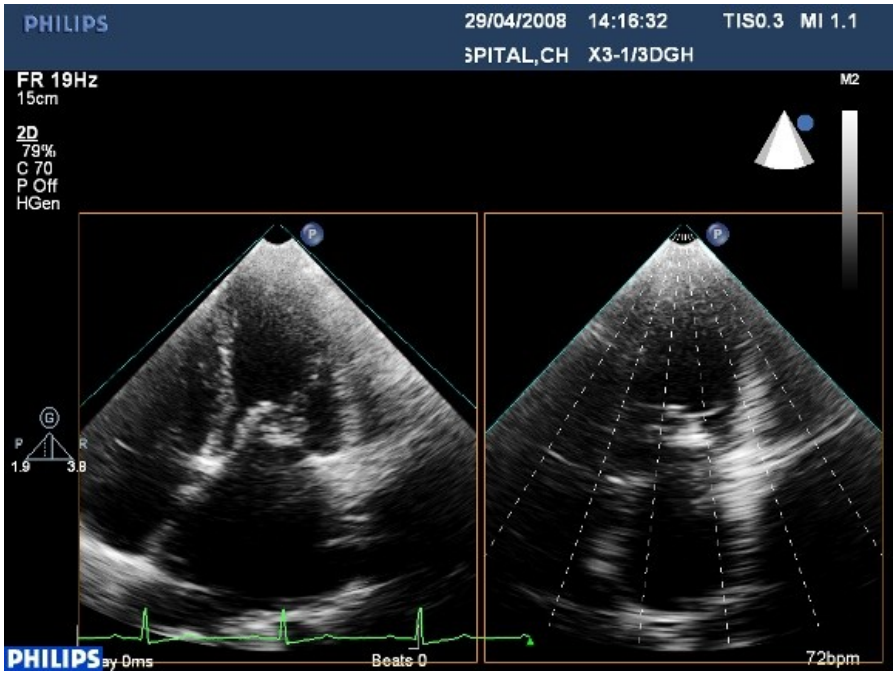
Then the "full volume" program was used (picture 1). With a standard apical 4-chamber view, we adjusted the ventricular septal rupture to the center of the screen, then opened the full-volume button, asked the subject to hold his/her breath, then acquired the "pyramid," 3-dimensional database (80° X 90°) from sequential 4 cardiac cycles. The "pyramid," 3-dimensional database with wider angle (90° X 110°) from sequential 7 cardiac cycles was also acquired. All images were stored on disks for off-line analysis. 3D TTE images were analyzed using integrated software system by cropping in 3 different colour-coded cutting planes perpendicular to each other. Besides these cutting planes, an additional oblique plane capable of cropping the image in any desired angulation may also be used, which helps in assessing the intracardiac structures placed at an angle other than the perpendicualr one.

3-D datasets consisting of the full LV volume allow accurate retrospective selection of measurement planes for calculation of stroke volumes and EF.In 3D Volume is not calculated by mathematical formula. It was calculated by volume data set.Like pixel,the data set of volume is – voxel. The No.of voxels can be calculated by the computer. LV volume analyzed by volume rendering automatic border detection method [37-42].

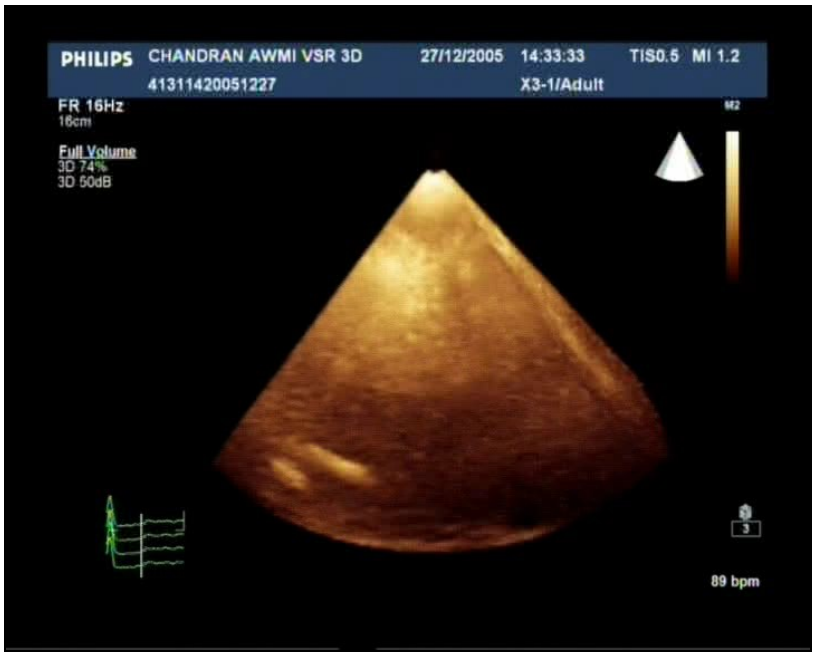
Data are presented as mean ± SD for descriptive statistics. We chose the average values for 3-time measurements. The linear regression analysis and paired Student's *t* test were used. To assess the effect of intra- and interobserver variabilities on the 3-dimensional measurements of the ventricular septal rupture, 10 randomly selected patients in the ventricular septal rupture, group were analyzed twice by one observer and at different times by two independent observers. A p value <0.05 was considered statistically significant.

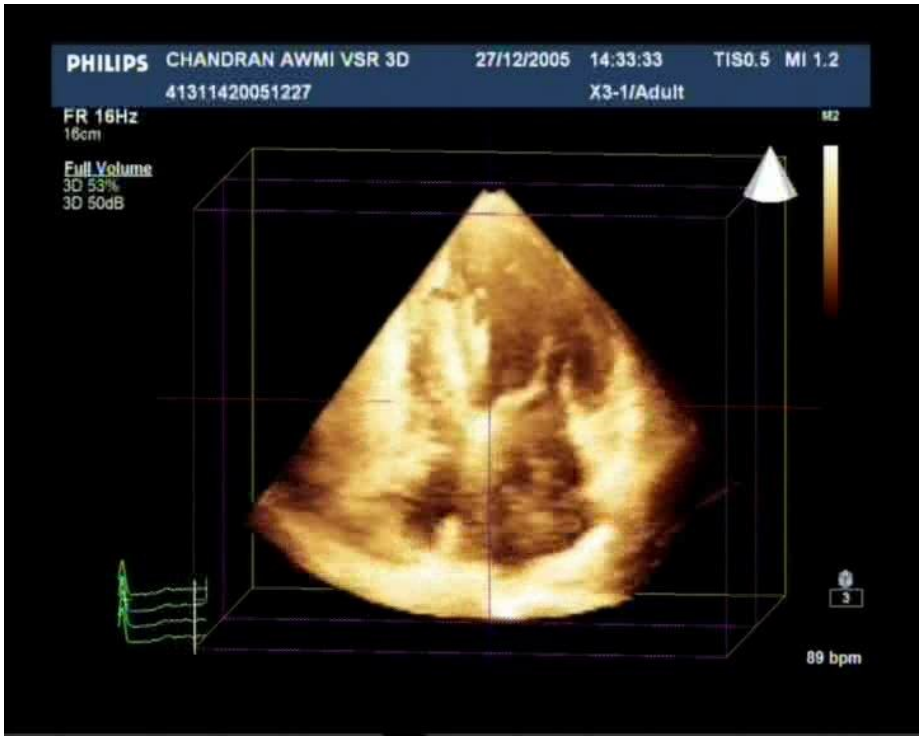
The 3-dimensional structures of the ventricular septal rupture, in 44 subjects were clearly and vividly demonstrated. We were able to observe the morphologic appearance of the ventricular septal rupture, and its relation to neighboring structures from a different azimuth and angle using a randomly rotated mode in the live 3-dimensional program.





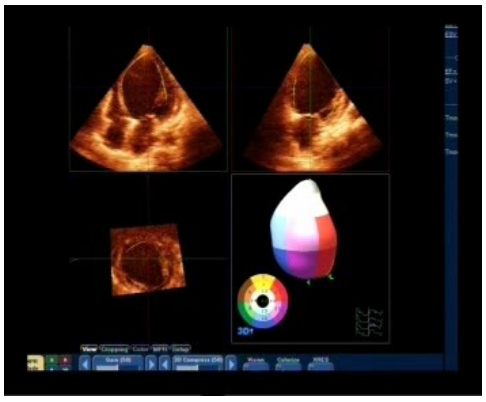
Picture 1(a): Full volume 3D image acquisition in 4 beats and 7 beats



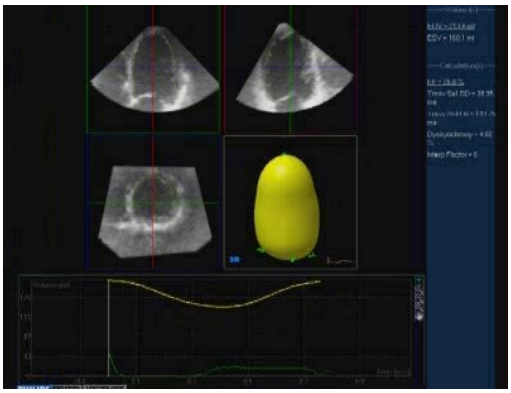


Picture 1(b): Full volume 3D acquisition image in 4 beats (80X90°)

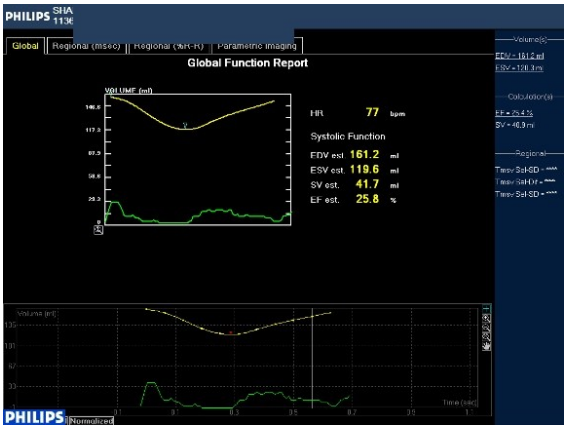
A



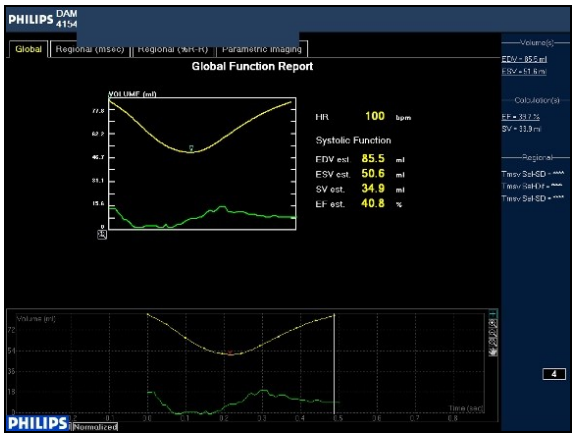
B



C



D



Picture 2 (A-D) 3D Echo assisted EF calculation. 3D Echo - (Q LAB) – EF Calculation. 3D Advanced True volume analysis.

Compared with the 2-dimensional technique, both static and dynamic 3-dimensional echocardiography provides more information about cardiac anatomic structures without any arbitrary geometric assumptions [44,45]. However, this complex acquisition and off-line data processing proved cumbersome and impractical for routine clinical use. With the new development of RT3DE, it became possible to rapidly evaluate the ventricular septal defect and area. RT3DE with a high-speed volumetric ultrasound system developed at Duke University is based on the use of a 16:1 parallel processing schema from a 2- to 4-MHz matrix,

phased-array scanner. The 16.times improvement in the data acquisition rate allowed one to interrogate an entire 3-dimensional pyramidal volume in real-time. Images in the region of interest may be viewed in real-time or in a 7 cardiac-cycle-captured volume without respiratory and electrocardiographic control.

The RT3DE examination can also be done in the zoom mode to visualize the VSR or any other region of interest. In the zoom mode a 30^0 X 60^0 sector of the heart is visualized. This live 3DE can be performed from the standard echocardiography window using the standard 2DE windows as starting point.

The entire heart and surrounding structures can be interrogated and we can obtain a pyramidal shape data set. The narrowest portion of pyramid being nearer to the

transducer and the widest portion is in other end of transducer. The pyramid is about 80^0 X 90^0 in size. The 80^0 view is obtained as four data set of 20^0 each from four cardiac cycles and the data set are merged to get full pyramid. During full volume acquisition mode the patient should hold the breath during expiration and the datas are collected from four cardiac cycle.

The full volume data can be analyzed later by cutting it from different direction and also by slicing. The entire ventricular septum and VSR orifice can be analyzed from the left ventricular side and RV side by post processing. This again helps us to obtain an enface view or surgeon's view of the septum and VSR. The VSR orifice can be measured from RV and LV side. Shape of the VSR was recorded clearly as, oval or elliptical from LV and RV aspects. Nature of the VSR, like simple or complex also recorded. Area of VSR was recorded in all cases during diastole. Associated complications like papillary muscle tear, free wall rupture and extension of VSR were analyzed.

The full volume acquisition can also be performed in the color Doppler mode. The size of pyramid is 60^0 X 90^0 and it is obtained from seven cardiac cycles with the patient holding the breath in expiration. The full volume data set can be post processed and we can clearly visualize the VSR. In case of mitral regurgitation, the area of the mitral regurgitation can be directly measured from the narrowest portion of the color Doppler jet.

In selected cases coronary angiogram was done and angiographic findings were recorded. 4 patients undergone surgery.

RESULTS

The study group consisted of forty four (n=44) post myocardial infarction, VSR patients consisting of 19 (43.2%) males and 25 (56.8%) females, (age range 45 to 80. mean age was 61.95). 3 cases were below 50 years (6.8%), 16 (36.4) cases were aged from 50-60 years, and 25 cases(56.8%)were above 60 years.

Out of 3546 patients with ST elevation myocardial infarction during the study period (male=2240; female= 1306) 0.84% of males and 1.91% of females had ventricular septal rupture. so, VSR complicated 1% of STEMI cases admitted in Coronary care unit from year 2005- 2008. Angina was the presenting manifestation in 56.8 % cases (31.8% in males and 25% in females) 31.8% of females compared with 11% in males, did not present with angina. Atypical presentations were more commonly noted in female groups.

Breathlessness was the presenting manifestation in 86% cases and it was more commonly observed in females in the absence of angina. Syncope was observed in 9 cases(20.5%) and cerebrovascular complication was noted in 2 cases(4.5%). Time window of patients presented in CCU varied from 6 Hrs to 120 Hrs. Those presented with in 12 Hrs of symptom onset were thrombolysed. Out of 44 cases 14 were thrombolysed (male 7, female 7). 34.1 % were presented within 12 hours.

Out of 44 cases 12 males and 18 females were not thrombolysed due to delayed presentation and one case was not thrombolysed due to persistent cardiogenic shock though the time window was less than 12 hours. SHT was present in 34 (77%) cases (male 16 and female 18). Diabetes mellitus type 2 was present in 19(43%) cases. Prior history of CAD was observed in 6 cases (13%). 7(16%) patients had family history of CAD. 18 % of males with VSR were smokers and remaining males and all females were non-smokers. Dyslipidemia was present in 29%.

Evidence of AWMI noted in 31 cases (70.5% of all types MI. Male 29.5% and female 40.5%), IWMI observed in 13 patients (29.5% of all types MI. Male 13.5% and female 15.5%), PWMI observed in 6 patients (13.6% of all types MI). 15.9% cases had lateral wall MI either in association with AMWI or IWMI. Evidence of RVMI noted in 14 cases (31.8 % of all types MI . Male 13.6 % and female 18.18 %),

From the symptom onset, day of VSR onset was noted and in 25% cases it was on day 1 and the same noted more commonly in females (15.9% in females; 9.09% in females). Another peaking of incidence observed on days 3 and 4 . On day 3, 8 cases (18.2%; male=4.5%; female=13.6%) developed VSR. On 4th day 14 cases (31.8%; male =15.9%; female=15.9%) and on day 5, 18 %(male = 9%, female=9%) developed VSR with equal incidence between males and females. No cases developed VSR beyond day 5 in this study.

Most of the patients were in cardiogenic shock. With the onset of VSR 50% patients presented with cardiogenic shock , 15.9% cases presented with Killip III, 31.8% cases presented with Killip II, and 1 patient presented in Killip I, whereas within 12 to 24 hours after the onset of VSR, 86.4% were in cardiogenic shock and remaining patients were in Killip III.

Prior to cardiac arrest sinus tachycardia was noted in 56.8% cases. Sinus tachycardia and ventricular tachycardia was observed in 13.6 % cases. AV block of high degree noted in 2 cases (4.5%). RBBB and LAHB were present in 15 cases and 12 cases were females. 3 cases developed LBBB. In 3 cases (6.8) sinus tachycardia and ventricular fibrillation was observed and these cases were resuscitated during cardiac arrest. Transient atrial fibrillation was noted in 1 patient (2.3%). 1 patient was received

with ventricular fibrillation and resuscitated. Blood urea and serum creatinine was elevated in 54.5% cases with onset of VSR.

Coronary angiogram was done in 9 cases (20.5%) and due to various reasons CAG was unable to do in remaining cases. Left main disease was present in 1 case which was also associated with LAD lesion. LAD lesion noted in 6 cases (13.6%), LCX lesion noted in 2 (4.5%) cases, and RCA lesion noted in 3 (6.8%) cases. None of the cases underwent coronary angiogram showed development of collaterals. In 90% cases the lesion was total occlusion of the target vessel.

By 2D Echo LV function was analyzed in all cases. LVEF was done by modified Simpson method. 27.3% were in LVEF of or below 35%. Severe LV dysfunction was noted in 9.1% cases. All the cases were below 48% of LVEF. 2D echo overestimated the LVEF. Whereas 3D echocardiographic LVEF measurement by wire loop method through automatic endocardial boundary detection method, LVEF were lower than that was measured by 2D echo. Mean LVEF calculated by 2D echo was 39.23%, whereas the same value calculated by 3D Echocardiogram was 36%.

T Test

The t test procedure is used to compares mean scores of LVEF for the 2D and 3D Echo methods. The procedure assumes that the variances of the two groups are equal and it was tested with Levene's test statistics. The results of the analysis are given in Table-

Null Hypothesis: H_0 : There is no significant difference between the mean scores of the two dimensions regarding with LVEF.

Echo	Mean	N	Std. Deviation	Std. Error Mean	t	df	P value	Remark

2D	39.23	44	5.295	.798	4.345	43	.000	Highly Significant
3D	36.00	44	5.681	.857				

The table displays the descriptive statistics of the sample size, mean, standard deviation and standard error. The table also shows that the t statistics, calculated as the ratio of the difference between sample means divided by the standard error of the difference. The column P value shows the probability value from the t distribution. Since the P value is less than 0.01 it is significant at 1%. Hence we reject the hypothesis. Hence there is significant difference in the mean scores of two groups with respect to the LVEF

Regarding diastolic dysfunction, 21 cases (47.7%) were in grade I, 5 cases (11.4%) in grade II , and 18 cases in grade III diastolic dysfunction. Wall motion score index varied from 1.2 to 2.1. All cases with acute AWMI showed regional wall motion abnormalities in LAD territory and in IWMI it was in RCA or LCX territory. Left ventricular hypertrophy was noted in 16 cases (36.4%).

Pulmonary hypertension was absent in 14 cases (31.8%). 18 cases (40.9%) had mild PHT, 7 cases (15.9%), had moderate PHT, 5 (11.4%) were in severe PHT. In all 31 cases (70.5) of AWMI with VSR the location was in apical ventricular septum. In all 13 cases (29.5) of IWMI with VSR the location was in basal ventricular septum. VSR pressure gradient in all cases ranged from 22 mmHg to 76 mmHg. Gradient of 40 and less than 40 mmHg noted in 31.8% cases. By 2D echocardiography size of the VSR could not be measured in 26 cases (59.1%) and only the location was identified by use of colour Doppler. in other cases size ranged from 3 mm to 16 mm. whereas size of the VSR as measured by longest diameter by 3D echocardiography varied compared with 2D echocardiogram. Size by 3D echo ranged from 3mm to 18 mm. area of the VSR was measured all cases, and it ranged from 0.8 sq.mm to 3.8 sq.mm. area of 2 or less than 2 sq.mm was noted in 63.6%

patients. Shape of the VSR defect from LV aspect was elliptical in 16 cases (AWMI=9; IWMI=7) and oval in 7 cases. From RV aspect morphology was same as from LV side in most of the cases and in 8 cases it was irregular and the shape altered during systole. Elliptical shaped VSR patients had more early mortality compared with oval shaped VSR defects.

3D echo evidence of simple direct through and through septal rupture was observed in 23 patients (52.3%) and complex VSR noted in 21cases (47.7%). 3 cases (6.8%) demonstrated extension of septal rupture into adjacent free wall. Free wall rupture

associated with VSR in 3 cases. Mitral regurgitation was observed in 75% cases varied from trivial to severe (mild MR-50%, moderate MR – 15.9, severe MR- 2.3%).

No cases were associated with papillary muscle rupture.2D and 3D evidence of pericardial effusion was observed in 12 cases (27.3%), and it was mild pericardial effusion in all of these patients. no evidence of tamponade noted.

VSR repair was done in 4 cases from our study. 3 cases survived and 1 patient developed electromechanical dissociation during the surgical procedure and could not be revived. 7 day mortality in patients with post myocardial infarction VSR was 36.4% (16 cases out of 44) and 30 days mortality was 43.2 % (19 cases). 7 day mortality in females was 20.5% and in males it was 15.5 %. 30 days mortality was also more in females (25%) compared with males(18%). 20.5% cases (n=9)survived at 30 days . Of the 9 cases ,3 survived after surgery . On further follow-up of the remaining 6 cases, 5 died at 3 months with onset of cardiac failure. One patient (male) is on follow-up who had AWMI and thrombolysed. So, 11% (n=4) survived after VSR.

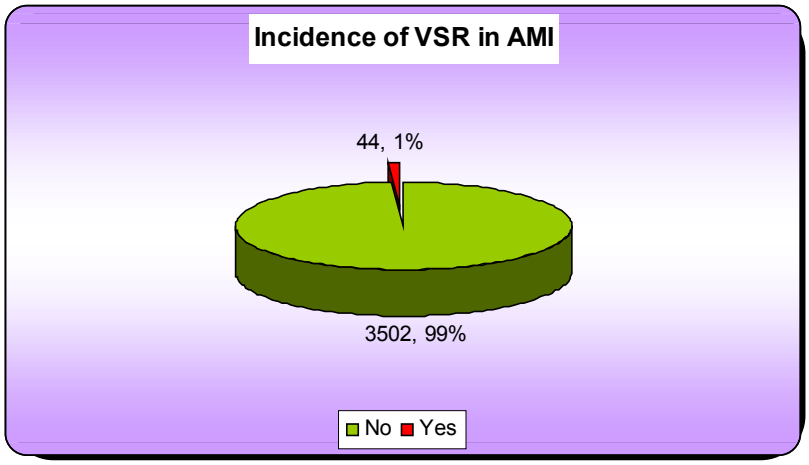
Within 7 days mortality, all cases had complex VSR. Patients survived 7 days were mostly of simple VSR in nature. 7 days mortality was more in patients who were not undergone thrombolysis (27% of all mortality cases) whereas in those thrombolysed, 7 days mortality was only 9%. Similarly 30 days mortality was also more in cases not thrombolysed (25% Vs 10% in thrombolysed).

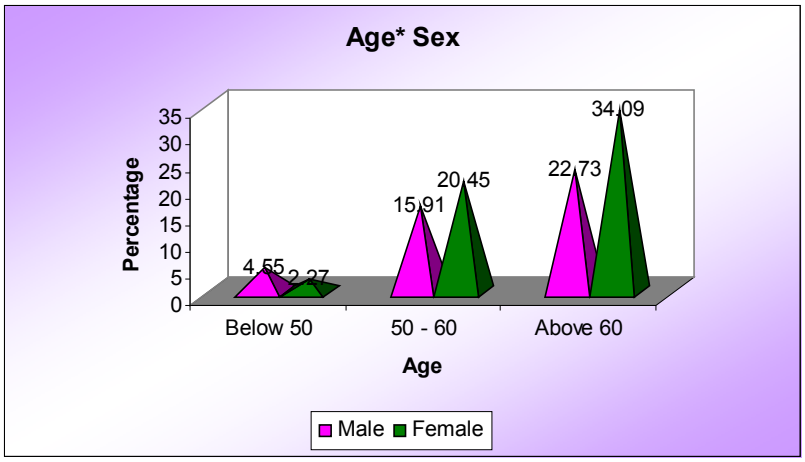
LVEF-2D%		Frequency	%	Valid %	Cumulative Percent
Valid	28	1	2.3	2.3	2.3
	30	3	6.8	6.8	9.1
	32	1	2.3	2.3	11.4
	34	2	4.5	4.5	15.9
	35	5	11.4	11.4	27.3
	36	4	9.1	9.1	36.4
	38	4	9.1	9.1	45.5
	40	9	20.5	20.5	65.9
	42	3	6.8	6.8	72.7
	44	1	2.3	2.3	75.0
	45	3	6.8	6.8	81.8
	46	6	13.6	13.6	95.5
	48	2	4.5	4.5	100.0
	Tot	44	100.0	100.0	

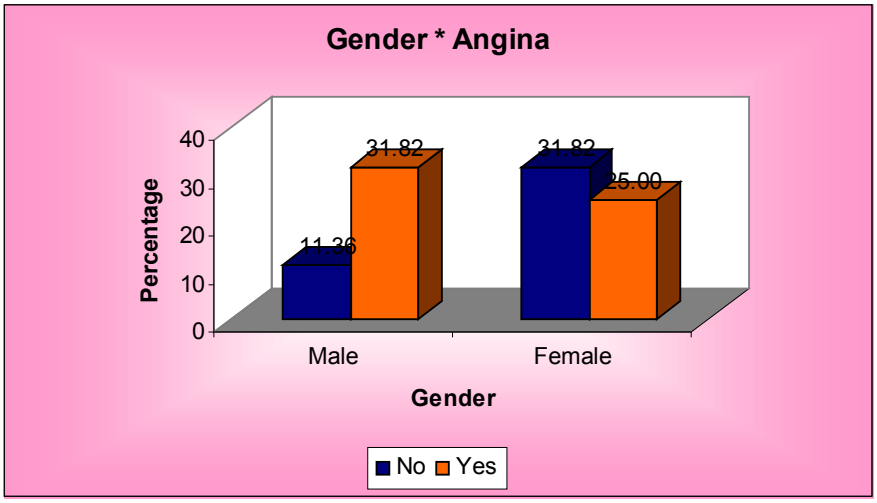
WMSI-2D Echo	Frequency	Percent	Cumulative Percent
1.20	2	4.5	4.5
1.25	4	9.1	13.6
1.30	1	2.3	15.9
1.37	2	4.5	20.5
1.50	6	13.6	34.1
1.60	1	2.3	36.4
1.62	5	11.4	47.7
2.00	3	6.8	54.5

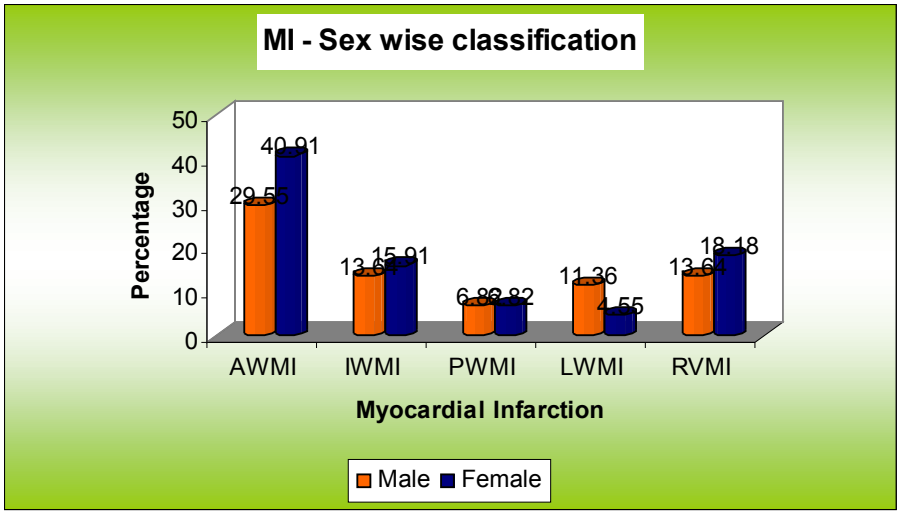
	2.10	20	45.5	100.0
	Total	44	100.0	

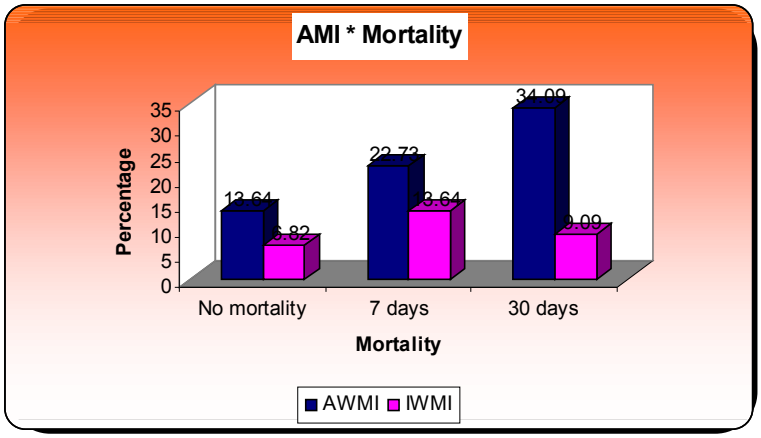
3D Size(mm)		Frequency	%	Cumulative Percent
Valid	3	1	2.3	2.3
	4	5	11.4	13.6
	5	10	22.7	36.4
	6	10	22.7	59.1
	7	7	15.9	75.0
	8	1	2.3	77.3
	9	2	4.5	81.8
	10	1	2.3	84.1
	11	1	2.3	86.4
	12	2	4.5	90.9
	13	1	2.3	93.2
	15	2	4.5	97.7
	18	1	2.3	100.0
	Total	44	100	

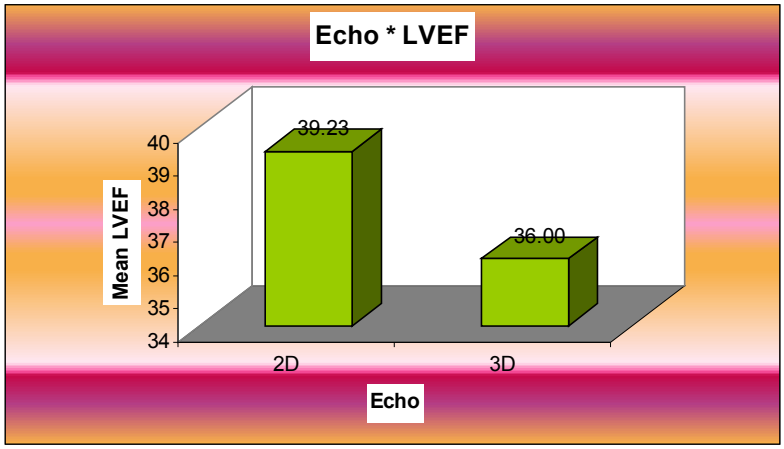

Incidence of VSR in AMI
44, 1%
3502, 99%
No
Yes

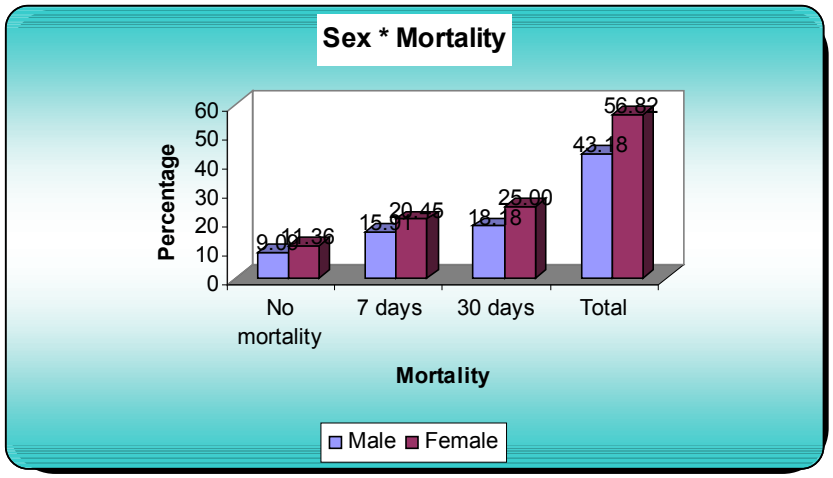

Age* Sex
Percentage
35
30
25
20
15
10
5
0
4.55
2.27
15.91
20.45
22.73
34.09
Below 50
50 - 60
Above 60
Age
Male
Female

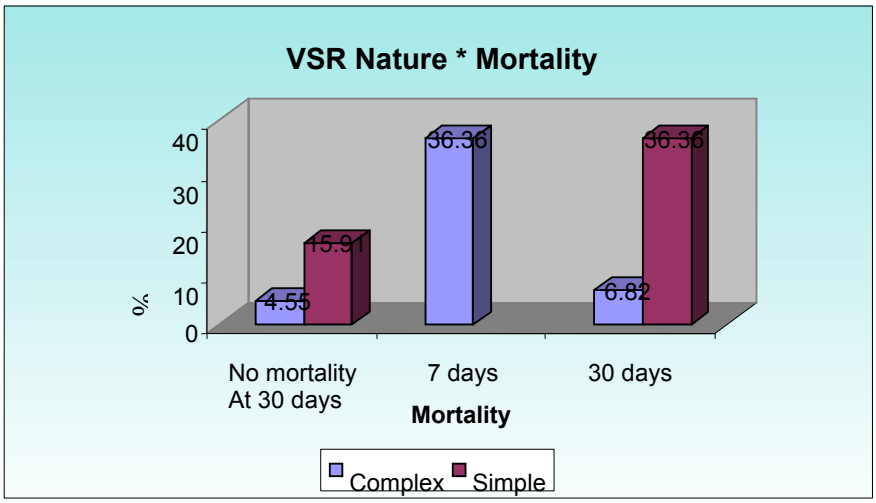

Gender * Angina
Percentage
40
30
20
10
0
11.36
31.82
31.82
25.00
Male
Female
Gender
No
Yes

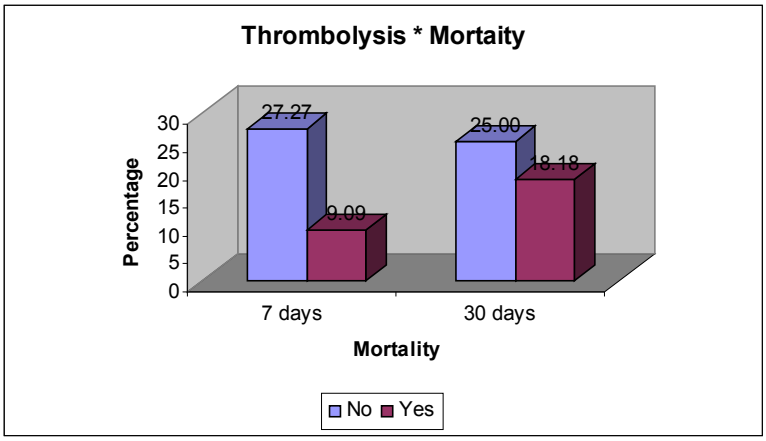

MI - Sex wise classification
Percentage
50
40
30
20
10
0
29.55
40.91
13.64
15.91
6.82
6.82
11.36
4.55
13.64
18.18
AWMI
IWMI
PWMI
LWMI
RVMI
Myocardial Infarction
Male
Female

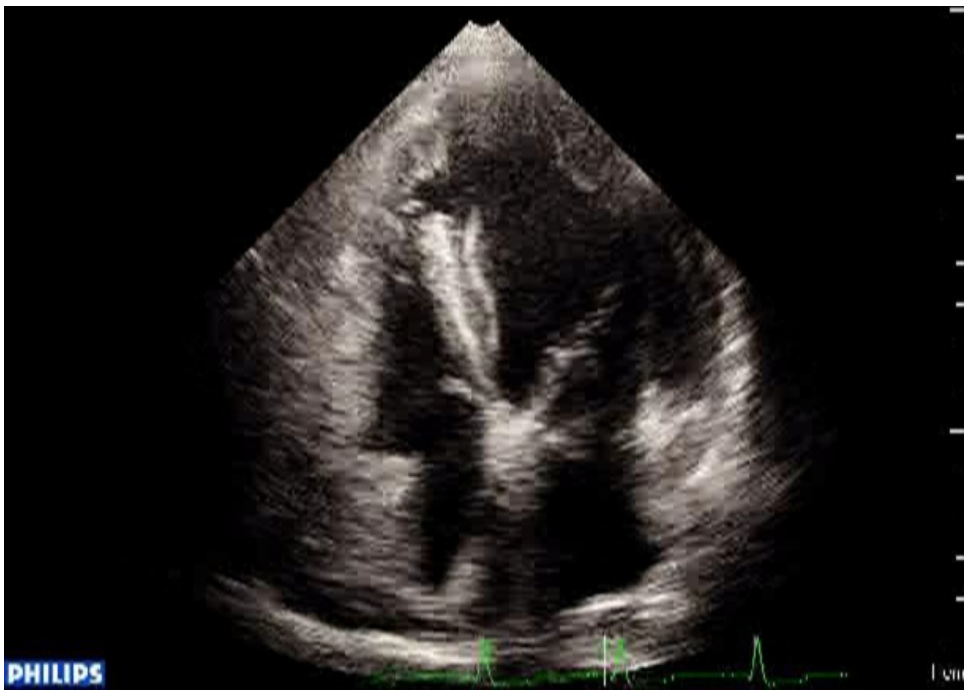

AMI * Mortality
Percentage
35
30
25
20
15
10
5
0
34.09
22.73
12.64
5.82
8.64
9.09
No mortality
7 days
30 days
Mortality
AWMI
IWMI


Echo * LVEF
Mean LVEF
40
39
38
37
36
35
34
39.23
36.00
2D
3D
Echo

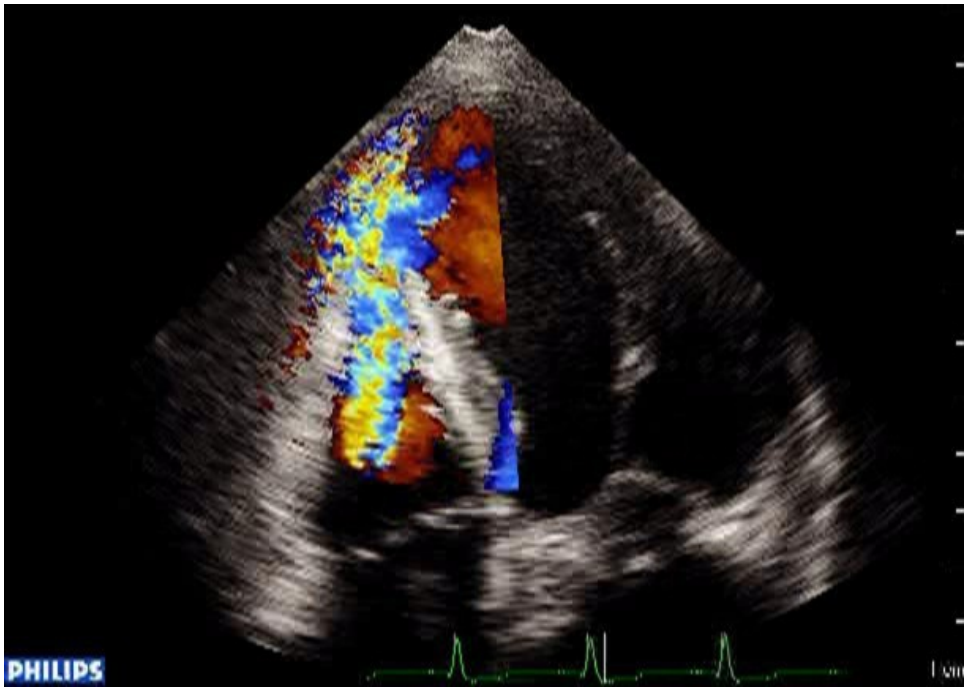

Sex * Mortality
Percentage
60
50
40
30
20
10
0
56.82
43.18
25.00
20.45
18.18
15.91
11.36
9.09
No mortality
7 days
30 days
Total
Mortality
Male
Female


VSR Nature * Mortality
40
30
20
10
0
%
4.55
15.91
36.36
6.82
36.36
No mortality
At 30 days
7 days
30 days
Mortality
Complex
Simple


Thrombolysis * Mortaity
Percentage
30
25
20
15
10
5
0
27.27
9.09
25.00
18.18
7 days
30 days
Mortality
No
Yes

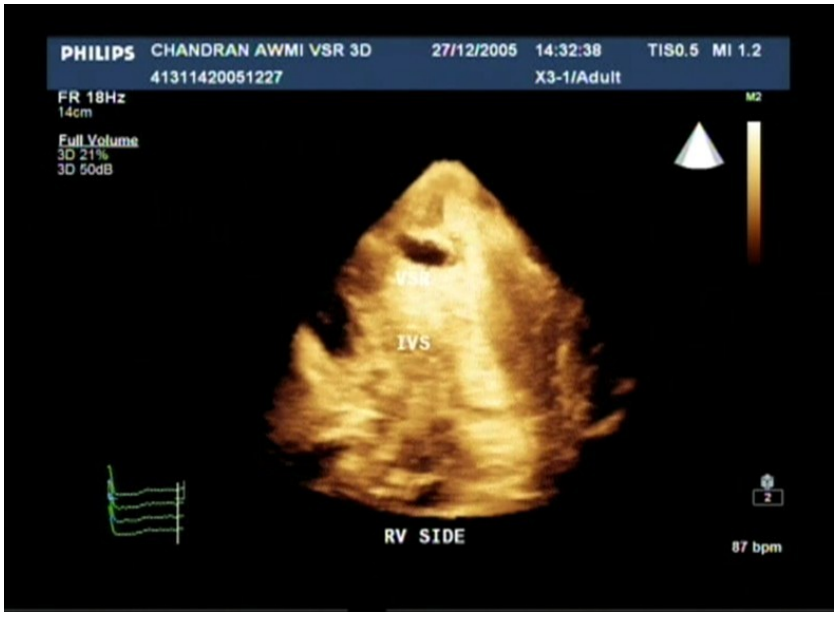
A 
PHILIPS

B



Picture 3(A&B): 2 Dimensional Echocardiographic imaging of apical ventricular septal rupture without and with colour Doppler in a 56 yrs old male after AWMI.

A



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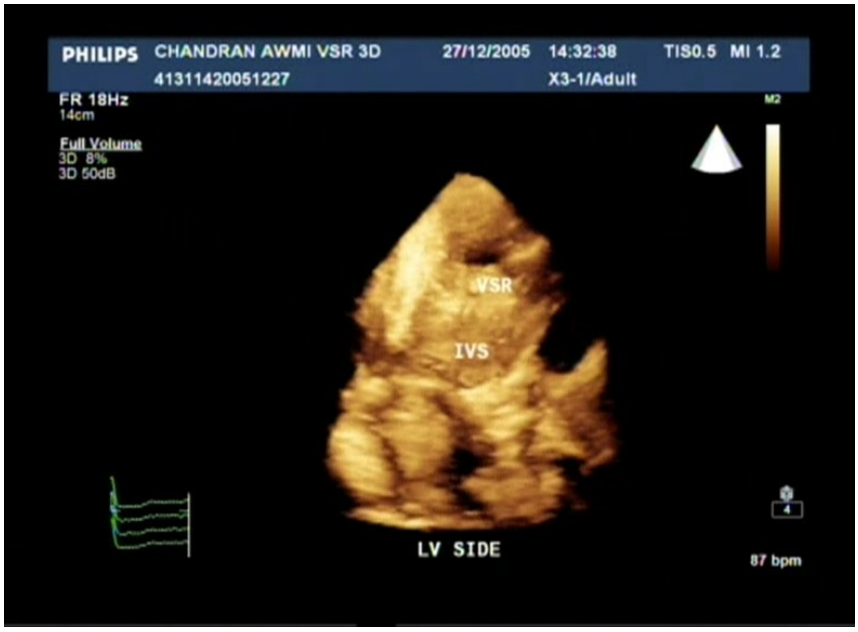
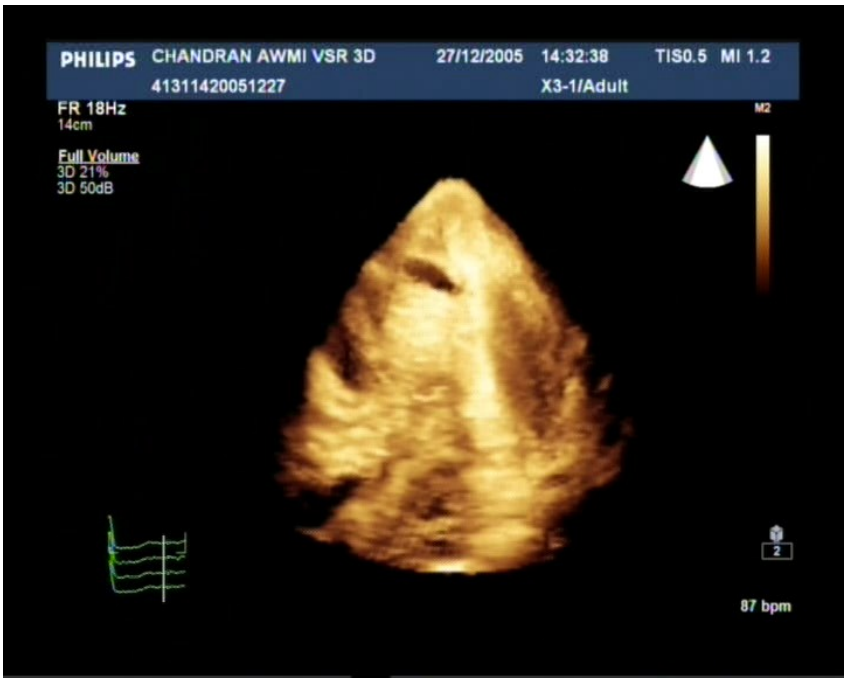
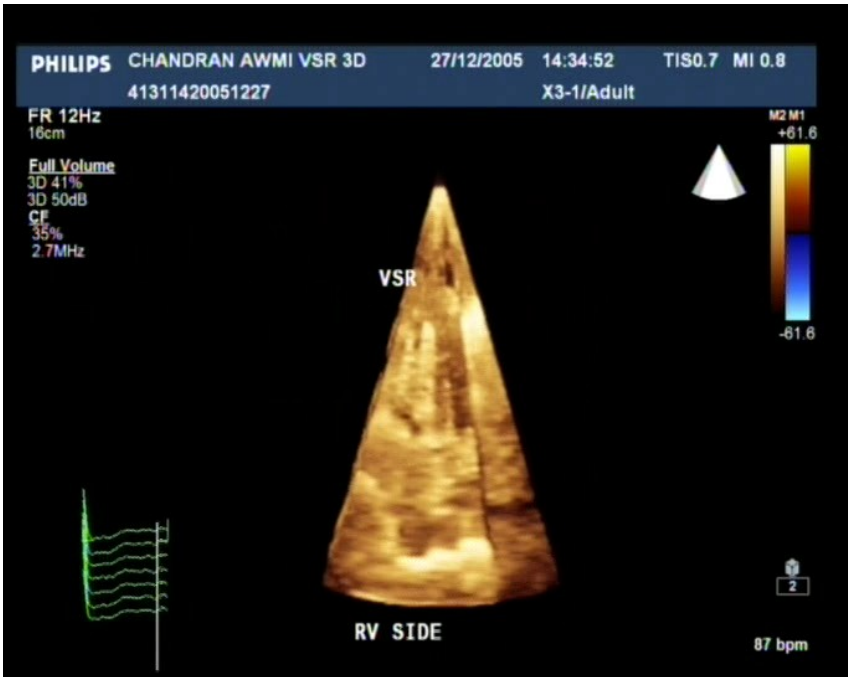


Picture 4(A&B): 3 Dimensional Echocardiographic imaging of apical ventricular septal rupture from right and left ventricular aspects in a 56 years old male after AWMI compared with his 2D Echo image (Picture 3).

C

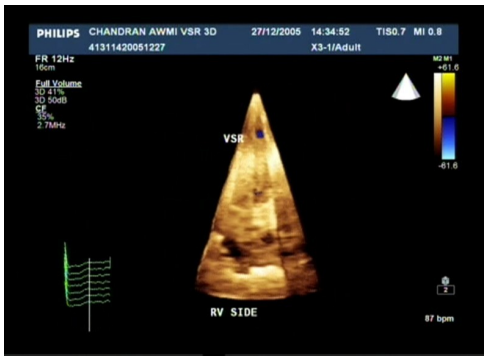


D

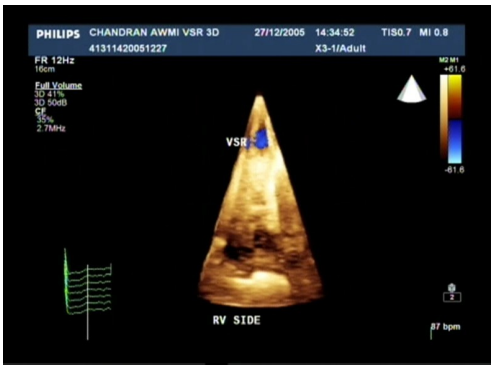


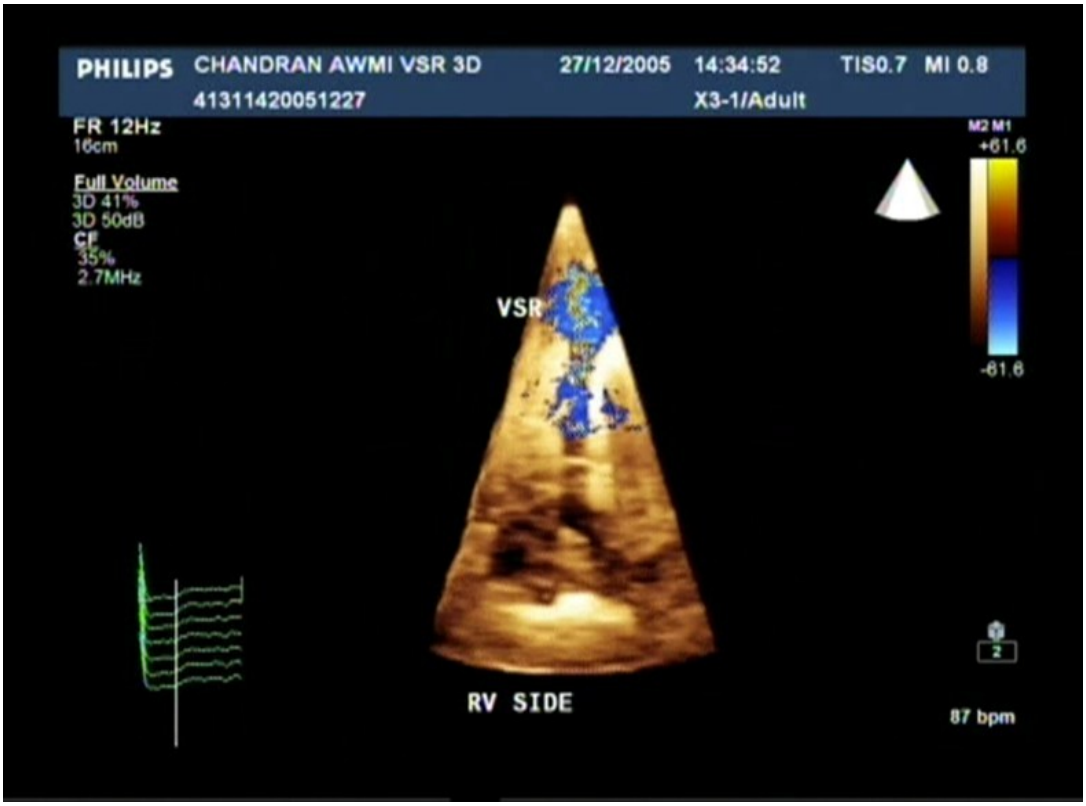
Picture 4(C&D): 3 Dimensional Echocardiographic imaging of apical ventricular septal rupture from right ventricular aspects in a 56 years old male after AWMI, compared with his 2D Echo image (Picture 3).

E



F



G 

Picture 4(E,F,G): 3 Dimensional colour Doppler Echocardiographic imaging of apical ventricular septal rupture from right ventricular aspects in a 56 years old male after AWMI, compared with his 2D Echo image (Picture 3).

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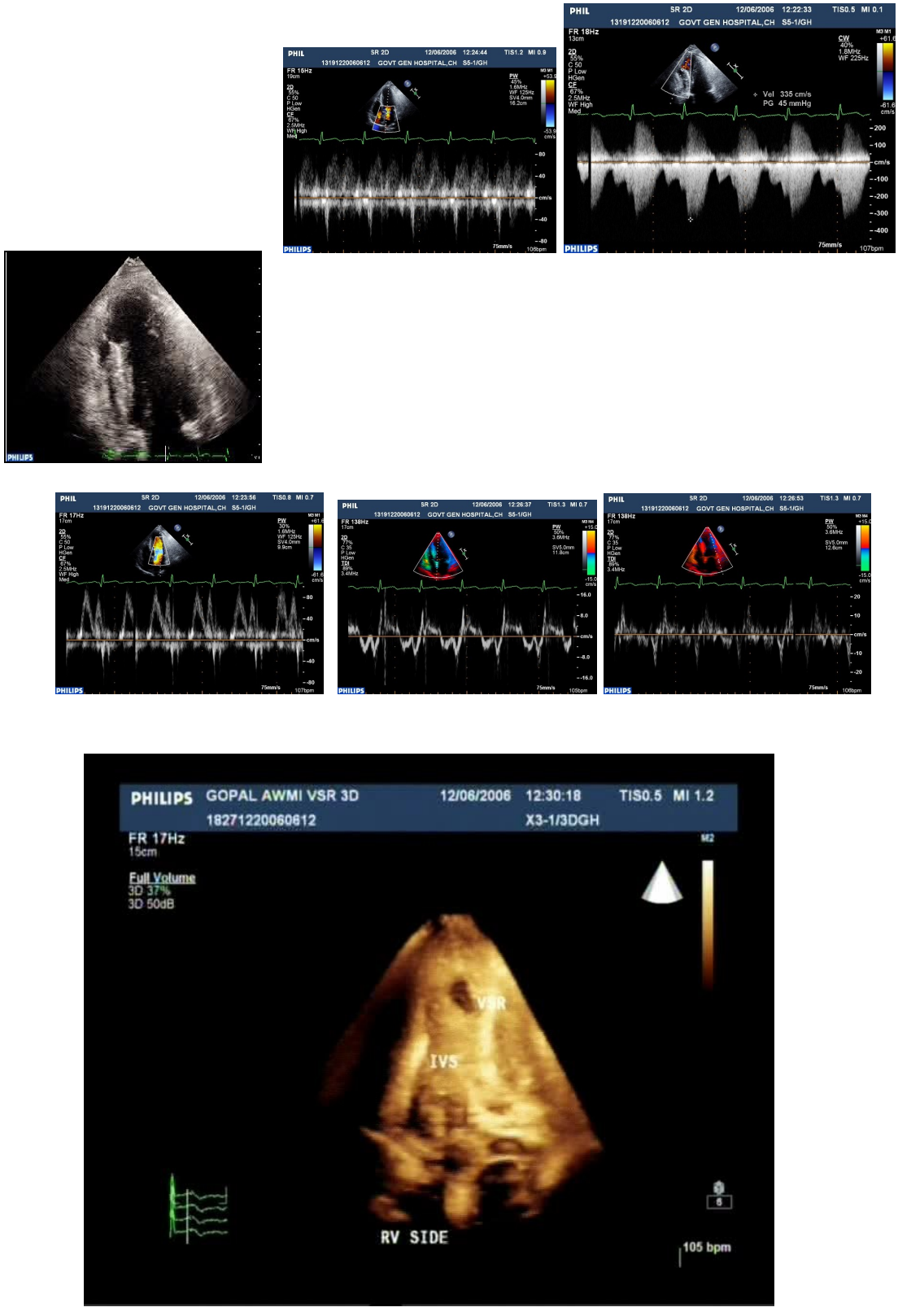
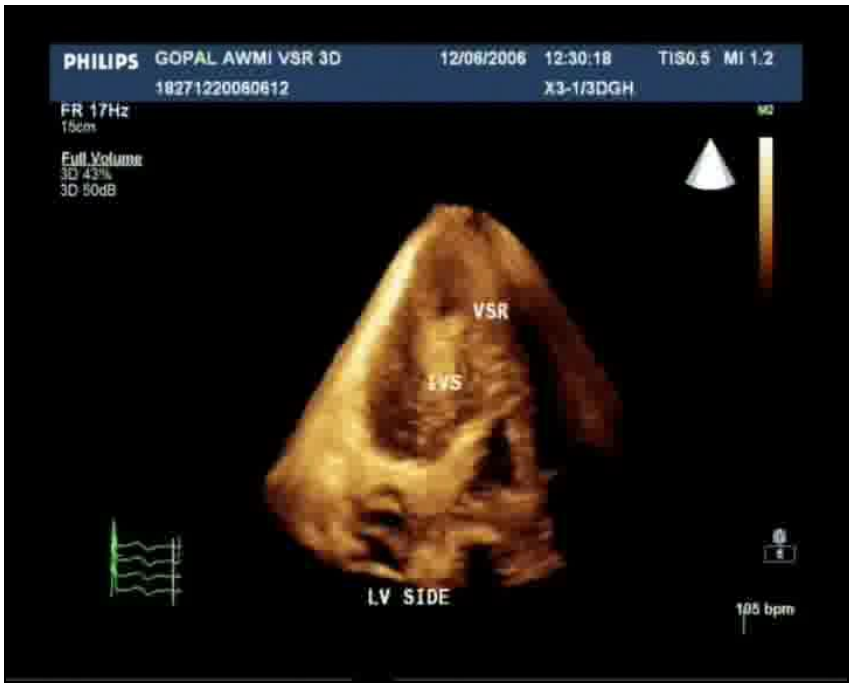


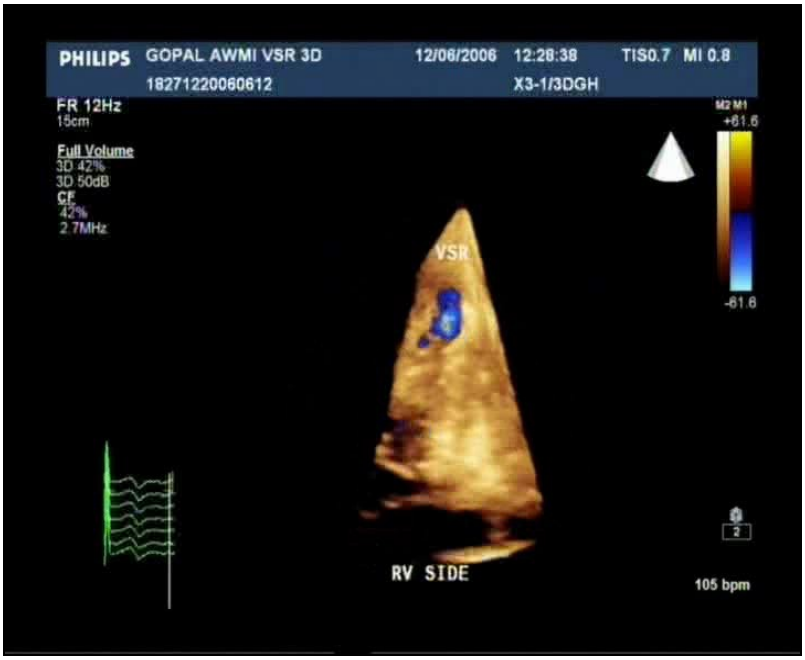
Picture 5: 3 Dimensional Echocardiographic imaging of apical ventricular septal rupture from right ventricular aspects in a 70 years old male after AWMI, in comparison with 2D Echo image

and Mitral inflow, Tissue Doppler images of the same patient.

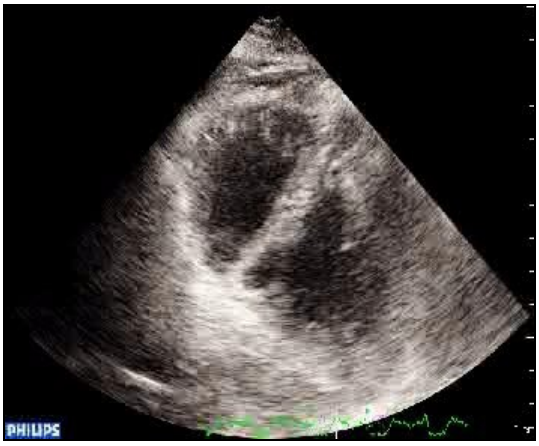
A

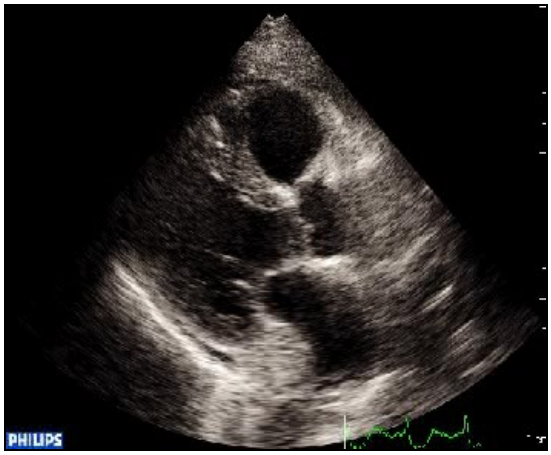


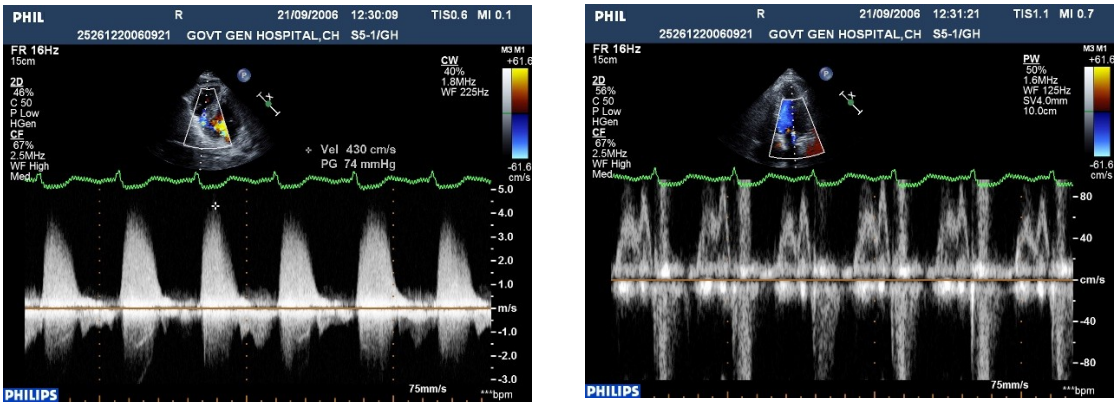
B



Picture 6: 3 Dimensional Echocardiographic imaging of apical ventricular septal rupture from right and left ventricular aspects in a 70 years old male after AWMI, in comparison with 2D Echo image of the same patient in picture 5.

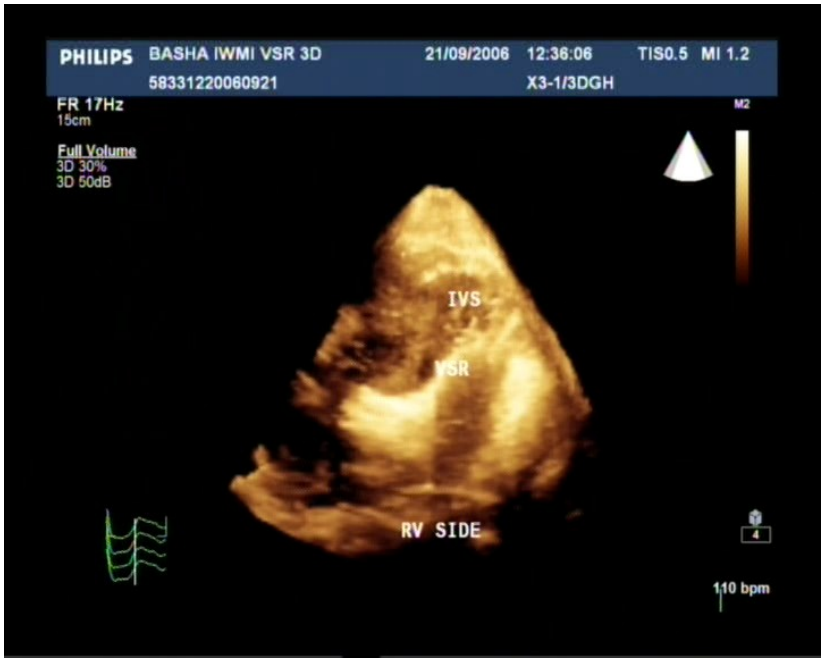


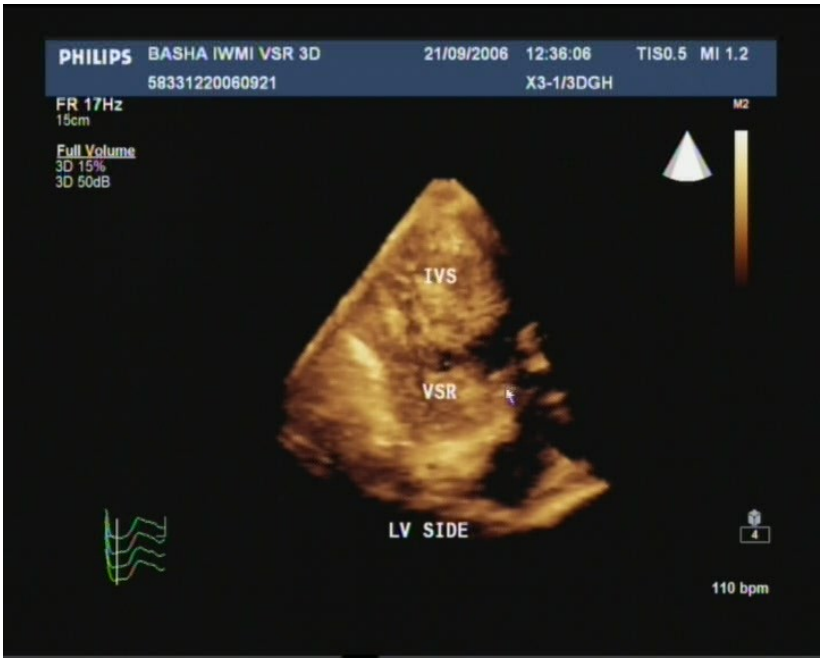




Picture 7: 2 Dimensional Echocardiographic images and mitral inflow Doppler images of basal ventricular septal rupture in a 64 years old male after IWMI.

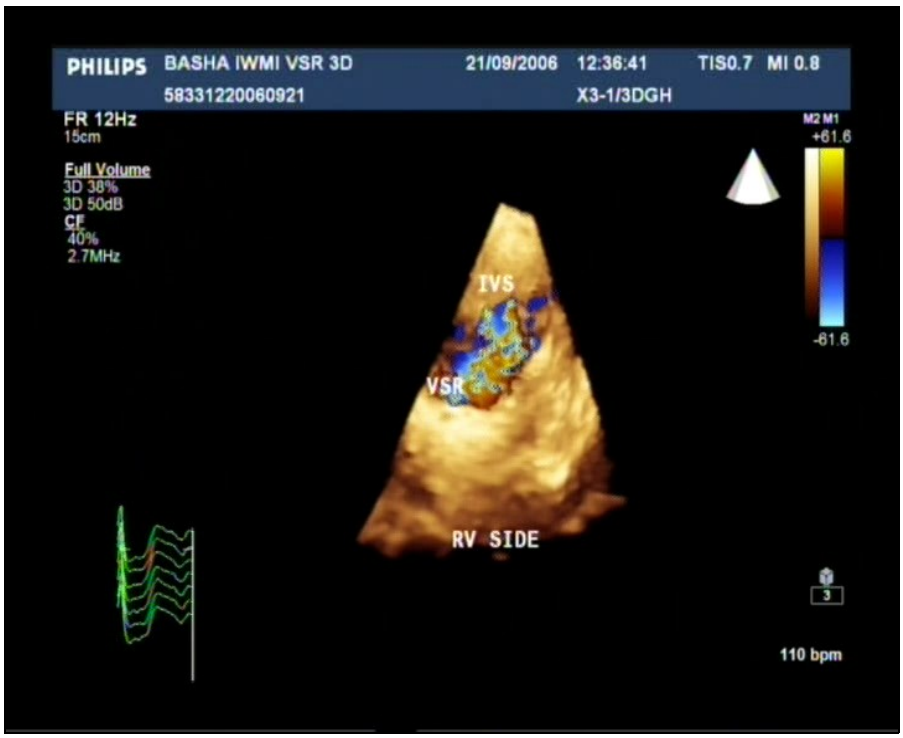
A B

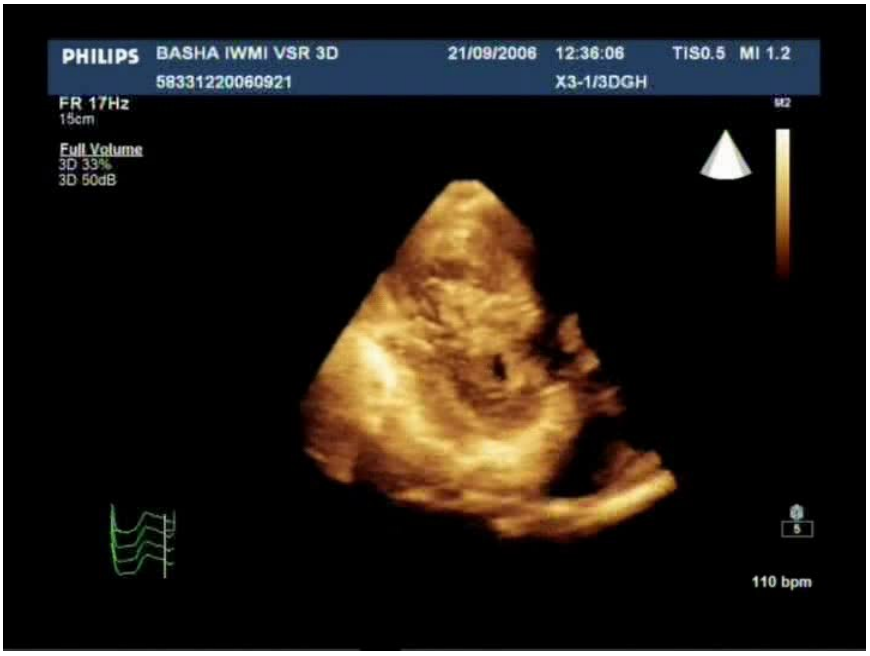




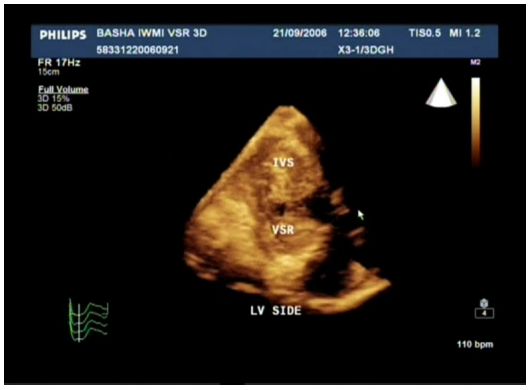
Picture 8: Three Dimensional Echo imaging of basal ventricular septal rupture from right and left ventricular aspects in a 64 years old male after IWMI, in comparison with 2D Echo image of the same patient (picture 7).

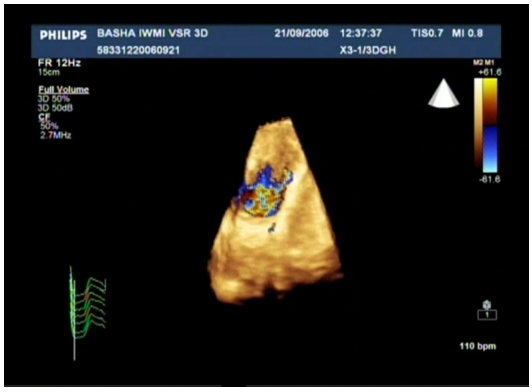
A B

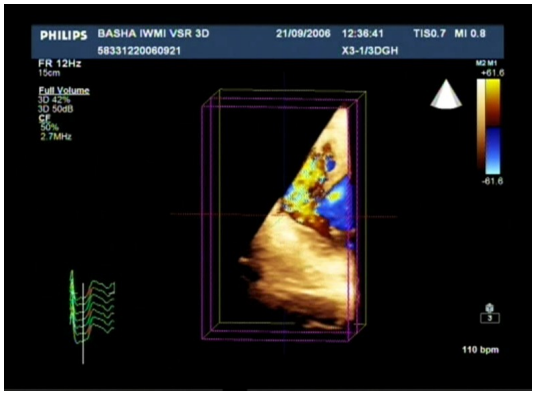


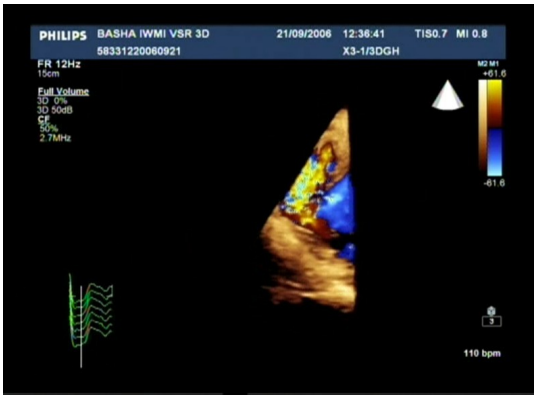


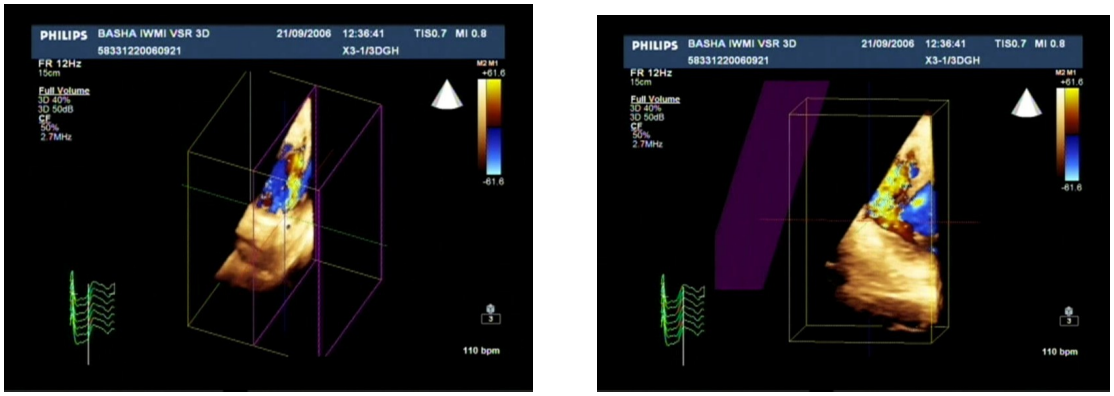
Picture 9 (A&B): Three Dimensional Echocardiographic imaging of basal ventricular septal rupture with and without colour Doppler in a 64 years old male after IWMI, in comparison with 2D Echo image of the same patient (picture 7).



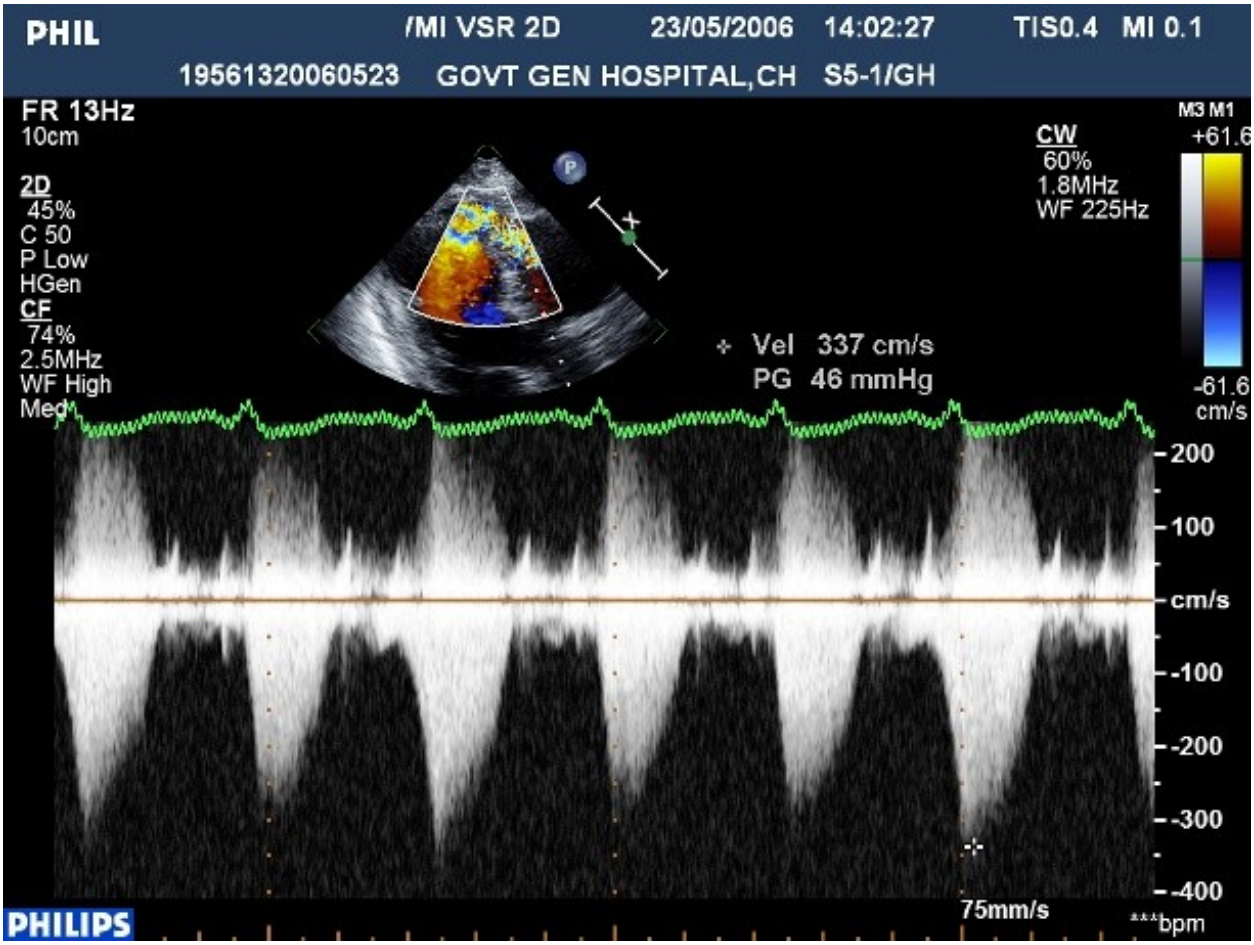




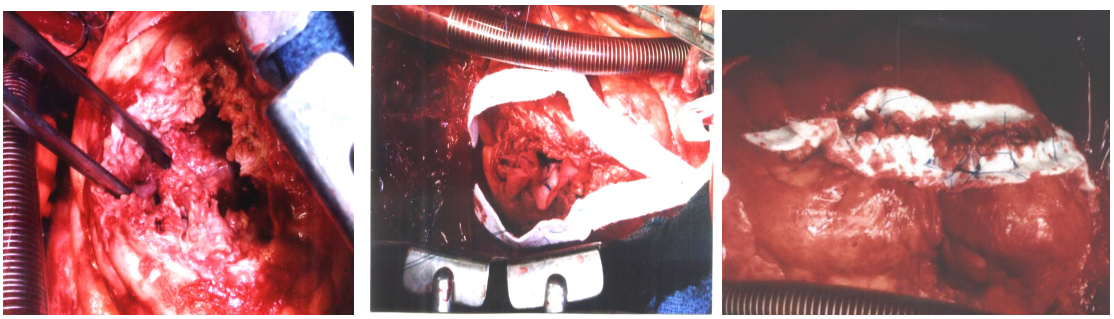




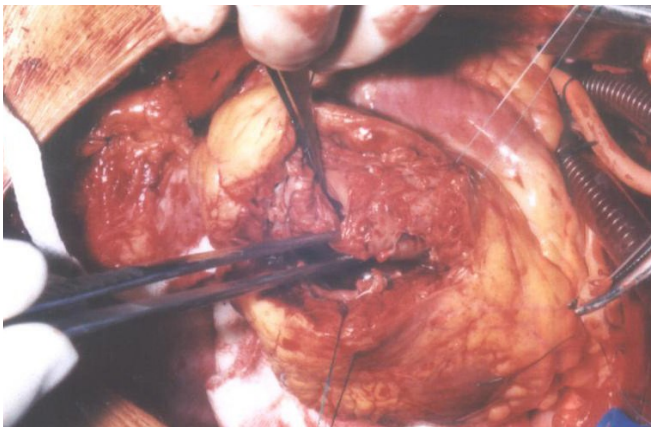
Picture 10. Methods of image acquisition in Live 3D Echo. Live 3D Echo images from a 64 years old male with IWMI and basal VSR.

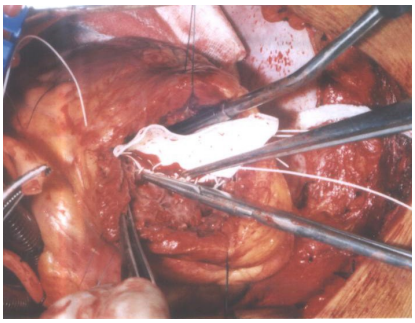


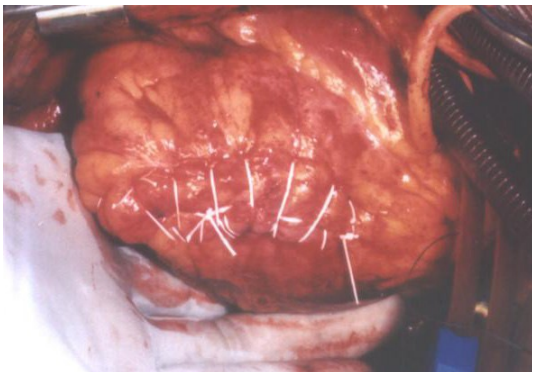
Picture 11: 2 D Echo imaging of apical ventricular septal rupture in a 62 years old female after AWMI .



Picture 12: Repair of a Ventricular septal rupture.







Picture 13: Repair of a complex ventricular septal rupture.

DISCUSSION

Before the introduction of thrombolysis ventricular septal rupture complicated 1–2% of all myocardial infarctions. The incidence has declined to about 0.2% (GUSTO-I trial) in the thrombolysis era. In our study group 1% of STEMI had VSR. Of the 270 000 myocardial infarctions suffered in the UK in 2002, approximately 550 will be complicated by a ventricular septal rupture.

In our study most of the cases that developed VSR on day 1 were already thrombolysed, The advent of widespread use of thrombolysis has had a dramatic effect upon the nature of ventricular septal rupture. In the early days of thrombolysis it was thought that the incidence may be increased, but it has subsequently been demonstrated repeatedly that the incidence is significantly reduced. In the GUSTO-I trial there was an incidence of 0.2% of ventricular septal rupture in over 41 000 patients, a 5–10 fold reduction compared with the pre-thrombolytic era. However, the nature of presentation has changed. Whereas the average time interval between infarction and rupture used to be 5–6 days, it is now closer to one day. Thrombolytic therapy may accelerate the time from myocardial infarction to VSD formation.

The relation of cardiac rupture to timing of thrombolytic administration is controversial however, none of the patients in our study were thrombolysed with time window beyond 12 hours. Some studies have shown an increased risk with late therapy (ie, >12 hours after symptom onset), but more recent evidence does not support this finding.[48] GUSTO-1 trial did not find a significant difference in the timing of thrombolytic administration between those who developed VSD and those who did not (3.1 versus 2.8 hours). However, enrollment criteria for GUSTO-I included a 6-hour limit from symptom onset; therefore, we cannot exclude the possibility of increased risk with thrombolytic administration after that period.

Risk Factors, Angiographic Patterns, and Outcomes in Patients With VSR Complicating Acute Myocardial Infarction was analyzed for the GUSTO-I Trial Investigators[49] and they compared enrollment characteristics, angiographic patterns, and outcomes (30-day and 1-year mortality) of patients enrolled in the Global Utilization of Streptokinase and TPA for Occluded Coronary Arteries

(GUSTO-I) trial with and without a confirmed diagnosis of VSR. In all, 84 of the 41 021 patients (0.2%) developed VSD, a smaller percentage than reported in the prethrombolytic era. The median time from symptom onset to VSD diagnosis was 1 day. Enrollment factors most associated with this complication were advanced age, anterior infarction, female sex, and no previous smoking. The infarct artery was more often the left anterior descending and more likely to be totally occluded in patients who developed VSD. Mortality at 30 days was higher in patients with VSDs than in those without this complication (73.8% versus 6.8%, $P<0.001$). Patients with VSDs selected for surgical repair (n=34) had better outcomes than patients treated medically (n=35; 30-day mortality, 47% versus 94%). These findings were well correlated with our study except the incidence of VSR in our study which was higher than the incidence in GUSTO-I . This may be due to the reason that all patients GUSTO-1 trial were treated with thrombolytics within 6 hours of symptom onset.

Reperfusion therapy, particularly if begun early, may prevent the extensive myocardial necrosis typically associated with mechanical complications.

In GUSTO-1, advanced age, anterior infarct location, female sex, and no current smoking were found to be the most important predictors of VSD and similar results were noted in our study. In GUSTO-1, unlike previous studies, hypertension and no previous MI or angina were found to be less helpful when all other variables were considered. But in our study still the above variables correlated with incidence VSR. In GUSTO-1, there was a bidirectional association of systolic and diastolic blood pressures at enrollment with the incidence of VSD. The positive correlations (increase in the incidence of VSD as systolic blood pressure increased to >130 mm Hg and the diastolic blood pressure to >75 mm Hg) reflect the association between hypertension and VSD. Extensive MI and right ventricular involvement, both known risk factors for VSD, may cause hypotension and cardiogenic shock on admission. The negative correlations between enrollment systolic (≤ 130 mm Hg) and diastolic (≤ 75 mm Hg) blood pressures with the incidence of VSD probably reflect the incidence of hemodynamic compromise associated with extensive MI or right ventricular infarction. In our study most of the hypertensive patients had either normal or high blood pressure prior to the onset of VSR and with the onset of VSR , developed hypotension. With the onset of VSR the systolic blood pressure ranged from 70 to 106 mmHg, an the diastolic pressure ranged from 50 to 74 mmHg.

In our study 4 cases (M=3, F=1) underwent surgery and in that 3 patients were not thrombolysed and 1 patient was thrombolysed and in whom surgery was done at the end of first week. It is likely that the nature of the patients coming to surgery has changed. The thrombolytic treatment may increase the proportion of ruptures that are complex rather than simple, and therefore more difficult to repair. Furthermore, patients in the first 24–48 hours after infarction are probably less well able to sustain the insult of surgery than they would be a week or so later. In the SHOCK Trial Registry of Cardiogenic shock in acute infarction, Rupture occurred a median 16 h after infarction. Patients with VSR tended to be older ($p = 0.053$), were more often female ($p = 0.002$) and less

often had previous infarction ($p < 0.001$), diabetes mellitus ($p = 0.015$) or smoking history ($p = 0.033$). These findings were well correlated with our study population.

TWO DIMENSIONAL ECHOCARDIOGRAPHY

2D echo failed to locate the site of VSR in majority of our cases. TEE may improve the detection rate, but this procedure is not without any risk.A study from Germany[50] reports that rapid and accurate diagnosis of ventricular septal rupture (VSR) remains difficult, TTE, TEE, color Doppler, and contrast echocardiography were evaluated in 17 patients with VSR in whom the diagnosis was confirmed by catheterization, surgery, or necropsy. In this study Routine transthoracic echocardiography visualized VSR only in 4 out of 17 patients and, with additional

views, in 12 out of 17 patients. Color Doppler echocardiography identified the rupture in 15 out of 16, and contrast echocardiography in 11 out of 11 patients. VSR was identified using TEE in six out of nine patients, and using color Doppler and contrast echocardiography in all patients. With the availability of 3D echo, it was possible to locate the VSR without any associated invasive procedures.

THREE DIMENSIONAL ECHOCARDIOGRAPHY

One of the main reasons for requesting an echocardiogram in routine clinical practice is the assessment of global and regional LV function. To date, this assessment is predominantly performed using visual interpretation or "eye balling" of dynamic ultrasound images of the beating heart, which requires adequate training and experience to accurately estimate LVEF, and wall motion abnormalities. However, the limitations of this subjective interpretation have been long recognized, and consequently the use of quantitative techniques has been recommended. Thus multiple methods to measure LV size and function have been developed, validated and refined for both M-mode and 2D B-mode images, and subsequently for reconstructed 3D images and more recently for volumetric real time 3D datasets.

The relative inaccuracy of the one and 2D echocardiographic approaches has been attributed to the need for geometric modeling of the ventricle. The "missing dimensions" have also been consistently referred to has the main source of the relatively wide inter measurement variability of the Echocardiographic estimates of the ventricular size and function.

In addition, the frequently encountered limitations in endocardial visualization, particularly in the apical-lateral segments of the LV are commonly compensated for by tilting the transducer. This maneuver generally improves endocardial visualization, but at the same time generates oblique or "foreshortened" views of the ventricle, resulting in even less accurate and reproducible measurements.

In this regard, the biggest advantage of 3D Echo is the lack of dependence of geometric modeling and image plane positioning, which theoretically should result in more accurate chamber quantification.

3D quantification of the LV typically employs a surface rendered mesh. This allows accurate computation of volume, RWMA, and regional synchrony. Because, the entire extend of the LV is taken in to the account, no foreshortening errors or assumption of LV volume are generated. Technically, a 3D deformable model is used to find the LV endocardial surface in 3 dimensions. This is the more accurate way to quantify LV volume. In our study LV volume and ejection fraction were calculated by this method and values were less than the values calculated by 2D method.

Indeed almost all studies that have directly compared the accuracy of 3D measurements of LV volumes and EF have demonstrated the superiority of the 3D approach over the 2D methodology, which was shown to consistently under estimate LV volumes. This superiority of 3D echoes was demonstrated in both accuracy and reproducibility when compared against independent reference techniques, such as radionuclide ventriculography or MRI [47].

These improvements have been shown to occur irrespective of the 3D acquisition strategy employed. Whereas in earlier 3D studies, quantification of LV size and function relied on tedious, manual, or at best semi-automated tracing of endocardial boundaries in multiple planes, today it is based on near fully automated frame- by- frame deduction of the 3D endocardial surface from real time 3D data sets. RA, LA, RV volumes also possible to measure by 3D techniques.

ANATOMICAL VISUALIZATION OF VSR BY 3D ECHOCARDIOGRAM.

In most cases of post myocardial infarction VSR 2D echo images unable to visualize the exact

size or site of VSR, and only after color Doppler it was possible to locate the VSR site, whereas 3D echo demonstrates the location and size clearly even before color Doppler. Shape of the VSR from LV side and RV side was possible by 3D echo image and nature of the VSR defined as simple or complex was possible. So that correlation with mortality was predictable in complex VSR and therefore early surgical planning may prevent mortality. In our study elliptical shaped VSR patients had more early mortality compared with oval shaped VSR defects, probably early remodeling may play a role and we believe myocardial fiber dehiscence may the added reason for the elliptical shape and it could explain the early morality. Presence of elliptical VSR in our study was almost equal in both AWMI and in IWMI patients.

Location, shape and extension into free wall will help the cardiac surgeon to plan the surgical procedure. As 3D echo provides the surgical view in enface, surgeon may not be surprised after opening the chamber. Right or left venticulatomy may be planned prior to cardiac surgery. Comprehensive noninvasive assessment of the location, shape, and size of the septal defect by Live 3D echo was clearly visualized en face from both RV and LV aspects in earlier reports. [51]

Classifying the types of lesion was accurate by 3D echo. VSR with aneurismal dilatation of the septum as in type III lesion will help the surgeon to plan about exact procedure. Associated surgical procedures like free wall repair mitral valve reconstruction in cases of PMD, and MR, could be planned by use of 3D echo images.

VENTRICULAR SEPTAL RUPTURE -MANAGEMENT

There existed a vogue some time ago for managing patients with ventricular septal rupture non-surgically in the first instant. After a period of perhaps six weeks, often with IABP, the patient then underwent surgery. The

advantages for the surgeon of this strategy were several. The procedure itself was a good deal more straightforward than the same operation in the acute phase, because the remaining septum was no longer mushy necrotic muscle, but had begun to fibrose and was thus more receptive to sutures. Furthermore the least well patients often failed to survive the weeks of non-surgical management, so that only the least ill survived to undergo surgery. This process of "unnatural selection" was first

expressed in the literature nearly 40 years ago by Honey and his colleagues from the London Chest Hospital.[52]

Some time later, from the same institution, Norell and colleagues reported a series of 55 consecutive patients who had presented with ventricular septal rupture and which they divided into two temporal groups.[53] Of the first group of 26 patients up to 1982, who were managed with delayed surgery, two had only minimal haemodynamic abnormality and did not go forward to operation. The remaining 24 patients included six who died without surgery and a further three who died after surgery. Thus the operative mortality was only 17%, and the overall mortality for the whole group was 38%. In the second era the philosophy was for early surgery. Even then there was an average delay of 10 days between clinical recognition and surgical repair. Of the 29 patients in this group, two were too well to require intervention, but a further five patients were considered to be too unwell for surgery and all of these died. Of the remaining 22 patients who underwent repair seven died, so that the operative mortality was 32%, and the overall mortality was 44%.

In view of the grim prognosis of medically treated patients, simply the diagnosis of postinfarction VSR is an indication for operation. The previous controversy surrounding the timing of surgical intervention is no longer an issue, and most surgeons agree that early surgery should be performed in order to incur the lowest risk of mortality

and morbidity. The success of the surgery depends on the prompt medical stabilization of the patient and the prevention of cardiogenic shock.

In most patients, postinfarction VSR rapidly leads to a worsening of the hemodynamic state, with cardiogenic shock, marked and intractable symptoms of congestive heart failure, and fluid retention. Immediate surgery is usually indicated. The high surgical risk of early repair is accepted because of the even higher risk of death without surgery under such circumstances. Generally, most patients who experience a postinfarction VSR are in need of emergent surgery. However, because of either delayed diagnosis or referral, an occasional patient may be in a state of multiorgan failure and may not be a candidate for surgery. The chances of such a patient surviving an operation are minimal; in these circumstances, supportive medical therapy may be adequate.

Surgical therapy: The first operations for repair of postinfarction VSR used an approach through the right ventricle, with an incision of the right ventricular outflow tract as was used to repair some congenital ventriculoseptal defects (VSDs). This approach proved inadequate because of limited exposure for lesions at the apex of the heart, injury to normal right ventricular muscle, interruption of coronary collateral vessels, and failure to excise the infarcted tissue.

Subsequently, a transinfarction approach was described. This technique, first pioneered by Heimbecker, incorporates infarctectomy, aneurysmectomy, and repair of the ventricular septal perforation. The technique of closure of these defects has resulted in several procedures. The choice of procedure is determined by the location of the defect.

Most defects are anteroapical, and closure uses a technique of buttressing the defect with viable muscle from the adjacent anterior left ventricular wall. Smaller defects located high in the ventricular septum are closed with a Dacron patch. The less common high posterior septal or inferior defect is approached through the inferior portion of the heart, usually in the distribution of the posterior descending coronary branch of the right coronary artery. The incision is made in the area of maximal infarction, which is usually on the right ventricular side of the septum. A well-proven principle of repair of these defects is the use of a synthetic patch closure to prevent tension. When a left ventricular aneurysm is associated with postinfarction VSR, it is excised as the initial step in the surgery. After repair of the VSR, the aneurysm is generally repaired.

In our study 4 cases underwent surgical repair at the end of first week,and 75% cases survived and 25% died. Patients with VSDs in GUSTO-I selected for surgical repair had better outcomes than those treated medically.

The operative mortality rate is directly related to the interval between infarction and surgical repair. If repair is performed 3 weeks or more after an infarction, the rate is approximately 20%. If surgery is performed prior to this time, the mortality rate approaches 50%. The most obvious reason for this is the fact that the greater the myocardial damage and hemodynamic compromise, the more urgent the need for early intervention. With the use of an early operative approach, most studies show an overall mortality rate of less than 25%. Mortality rates tend to be lower for patients with

anteriorly located VSRs and lowest for patients with apical VSRs. For anterior defects, mortality rates vary from 10-15%; For posterior defects, mortality rates vary from 30-35%.

None of the cases operated in our study did not develop residual VSR. Residual VSDs have been noted early or late postoperatively in 10-25% of patients. These residual defects are easily diagnosed with the aid of color-flow Doppler investigations. Residual VSDs may be attributable to the reopening of a closed defect, the presence of an overlooked VSD, or the development of a new septal perforation during the early postoperative period. Reoperation is required for closure of such residual defects when the Qp-to-Qs ratio is greater than 2. When these VSDs are small and asymptomatic, a conservative approach may be recommended because spontaneous closure may occur.

POST MYOCARDIAL INFARCTION VSR AND MORTALITY

6 cases in our study with IWMI and RVMI had severe RV dysfunction and early mortality (on day 2) was 100%.The most important risk factors for death in the early phase are poor homodynamic and associated right ventricular dysfunction. The amount and distribution of myocardial necrosis and scarring are responsible for both. Right ventricular dysfunction results from ischemic damage or frank infarction of the right ventricle and is present when stenosis occurs in the right coronary artery system. The severity and distribution of CAD are also risk factors. Similarly, advanced age, diabetes, and preinfarction hypertension are risk factors for death in the early phase.

Successful transcatheter closure of a post-MI ventricular septal rupture acutely following unsuccessful surgical repair has been reported. Catheter closure was accomplished by the use of a 26-mm Amplatzer atrial septal occluder[54].

The profile and outcomes of patients with VSR in the setting of cardiogenic shock (CS) complicating acute myocardial infarction (MI) was analyzed in SHOCK trial registry[55]. Despite surgical therapy, mortality in such patients was high. 939 patients enrolled. They also underwent right-heart catheterization, intra-aortic balloon pumping and bypass surgery significantly more often. Although patients with VSR had less severe coronary disease, their in-hospital mortality was higher (87% vs. 61%, $p < 0.001$). Surgical repair was performed in 31 patients with rupture (21 had concomitant bypass surgery); 6 (19%) survived. Of the 24 patients managed medically, only 1 survived. It was concluded there is a high in-hospital mortality rate when cardiogenic shock develops as a result of VSR. Ventricular septal rupture may occur early after infarction, and women and the elderly may be more susceptible. In our study, early mortality was more in patients presented with cardiogenic shock.

LV ANEURYSM AND VSR

3 cases in our study had aneurismal dilatation of the apical septum and complicated by VSR. A previous report from Kobe General Hospital of 72-year-old woman with inferior myocardial infarction presented with both a pseudo aneurysm and a ventricular septal rupture detected by 2D and Doppler echocardiography[57]. The pseudo aneurysm originated from the junctional area between the inferior portion of the ventricular septum and posterior left ventricular wall. The short-axis view of two-dimensional echocardiography revealed an abrupt discontinuity of the junctional area and an echo-free space behind the left ventricular cavity. The communication orifice was 5 mm wide. Color Doppler echocardiography showed a left-to-right shunt flow from the pseudo aneurysm to the right ventricle was visualized. There are reports where making the diagnosis of combined LV pseudo aneurysm and VSR by 3-D TEE proved superior to 2-D TEE in assessing the size of the LV rupture site.[59]

COMPLEX VSR AND PROGNOSIS

Risk factors, echocardiographic patterns, and outcomes in patients with acute VSR during myocardial infarction was studied from Mexico[56] in 2005 which examined the prognostic significance of Echo patterns in postinfarct VSR by postulating that complex rupture and RV involvement carry a worse prognosis. 17 patients(3=IWMI;14=AWMI) who had confirmed postinfarct VSR underwent 2D TTE and TEE followed by CAG. Type of septal rupture was classified as simple or complex based on autopsy proved echocardiographic criteria. ST-segment elevation persisted >72 hours in all 3 patients who had inferior wall MI and in 12 who had anterior wall MI. Segmental wall motion abnormalities helped in detecting the left ventricular entry site, and use of unconventional views superimposed with color flow Doppler provided the RV exit site. RV function was better appreciated with transesophageal echocardiography. Two patients who had IWMI and 7 who had AWMI had complex ruptures. All 3 patients who had inferior wall MI and 7 who had anterior wall MI had ECG and echocardiographic evidence of RV involvement. Mortality rate was higher in patients who had complex rupture (78% vs 38%, p <0.001) and in those who had RV extension (71% vs 29%, p <0.001). Persistent ST elevation was a common finding in patients who have postinfarct VSR. Complex VSR and RV involvement are significant determinants of clinical outcome. These findings were also well correlated with our study, where persistent ST elevation after 24 hours was noted in 75% cases. complex type of VSR as detected by 3D echo had early mortality in our study.

COMPARISION WITH OTHER INDIAN STUDIES ON VSR.

Clinical and Hemodynamic Profile, Surgical Results and Long-Term Outcome of VSR were studied from Sree Chitra Tirunal Institute of Medical Sciences and Technology, Thiruvananthapuram[58]. They analyzed 26 cases (21 males) of post MI VSR, who had undergone cardiac catheterization and angiography. The mean age was 55.14±7.2 years. Of the total, 3 had IWMI, 20 had AWMI and 3 had both IWMI and AWMI. Only 5 of them received thrombolytic therapy. All patients developed VSR on the second or third day after MI. Three patients presented with acute pulmonary edema, 5 with congestive heart failure and 6 with NYHA class III dyspnea; the rest were in class II. The VSD size varied between 4.5 and 14 mm (mean 8.6±2.4 mm),(In our study VSR size from 3 to 18 mm; and area by 3D method varied from 0.8 to 3.8 sq.cm) and the VSD gradients varied from 21 to 100 mmHg (mean 54.6±19.44 mmHg).(In our study VSR pressure gradients varied from 22 to 76 mmHg). Mean left ventricular (LV) ejection fraction was 56.6%±14.09%.(In our study LF by 3D echo method varied from 25 to 48%). Cardiac catheterization showed a left-to-right shunt which ranged from 1.4 to 4.4 (mean 2.34±0.75). The

mean pulmonary artery (PA) pressure was 33.42±11.80 mmHg. LVEDP ranged from 10 to 40 mmHg (mean 18.96±8.01 mmHg). Of the 3 patients who had IWMI, 2 had a totally blocked RCA and 1 had a totally blocked dominant LCX . Other vessels were normal in these patients. In patients who had AWMI, 16 had a totally blocked LAD artery with normal/insignificant lesions in the other vessels, 5 patients had 70%–80% lesions in the LAD while one had a reanalyzed LAD artery. One patient had 60% lesions in both the LAD and RCA. One patient with good ventricular function and a small VSD awaited surgery, while 14 patients underwent surgery Of the patients who underwent surgery, 2 died on the table, 1 had a small residual VSD and 2 had moderate LV dysfunction on follow-up. All the other patients were in NYHA class II on a mean follow-up of 8.5±5.2 years post surgery. Among the 7 patients kept on medical follow-up, 4 were in class I with good effort tolerance and negative TMTs, 5 were lost to follow-up, including 2 who were advised surgery. The overall prognosis in this study seems better than reported earlier. Smaller defect size, minimal left to right shunt and preserved RV function may be the factors responsible for long-term survival. But in our study out of 44 cases only 4 cases survived and 90.9 % died.

A report from Sanjay Gandhi Post Graduate Institute of Medical Sciences, Lucknow (GS Gill, Naveen Garg) on 9 (M=7,F=2)cases of post MI ,VSR from January 2004 to May 2005, with mean age was 56±10 years (range: 41- 66 years),VSD appeared on day 3 onward. None had any history of angina or MI. Eight (90%) patients had anterior wall MI (2 patients were thrombolyzed) and had apical muscular VSD (size varying from 8 - 15 mm). One patient had inferior wall MI and had basal septum VSD. Mean left ventricular ejection fraction (LVEF) was 38±7%. Coronary angiography performed in 6 patients revealed single vessel disease in 4 and triple vessel disease in other 2 patients. 2 patients died of cardiogenic shock soon after the admission. Patch closure of VSD was done in 5 patients. Two patients died after surgery. The clinical presentation correlated with our study, but our study population underwent 3D echocardiographic demonstration of VSR to predict the outcome better.

LIMITATIONS OF THREE DIMENSIONAL ECHOCARDIOGRAPHY

The standard of the 3D reconstructed display depends critically on the quality of the original 2D cross sectional images. Until recently, in adults, this necessitated TEE.

However, the development of harmonic imaging has made it feasible to reconstruct from a transthoracic rotational dataset. During data acquisition three-dimensional echocardiographic reconstructions are also very sensitive to both patient and operator movement, either of which can distort the image and result in dropout which may be misinterpreted. There are several possible limitations of three-dimensional volume-rendered echocardiographic reconstructions. This technique is extremely sensitive to proper gain settings from the two-dimensional data acquisition as well as to the level of threshold chosen (which defines the interface between tissue and blood) during the three-dimensional reconstruction.

Atrial fibrillation or a variable respiratory pattern prolongs the acquisition time and impairs the dataset resulting in artifact. Operator dependent changes in threshold settings, which define the tissue–blood interface on the 3D rendered display, can affect the apparent ventricular septal rupture .Therefore; measurements on reconstructed images should be made with caution.

THREE DIMENSIONAL ECHOCARDIOGRAPHY - FUTURE DIRECTIONS

Ongoing developments in 3D echocardiography include technological innovations and expanding clinical applications. Automated surface extraction and quantification, single-heartbeat full-volume acquisition, transesophageal RT3D imaging, the ability to navigate within the 3D volume, and

stereoscopic visualization of 3D images are some of the technological advances that can be expected over the next several years. These will further enhance the quality and clinical applications of 3D echocardiography. In addition, standardized and focused 3D protocols will be developed and refined to optimize clinical application of this technique.

Tagging and/or tracking the LV surface in real time may provide new approaches to quantifying myocardial mechanics, such as regional shape and strain. This approach has great potential and will complement and likely compare favorably with the quantitative ability of cardiac MRI. The superior temporal resolution of echocardiography should offer unique advantages for this purpose.

In the future, combining the greater temporal resolution of 3D echocardiography with the excellent spatial resolution of MRI (or computed tomography) may yield an imaging data set with unsurpassed anatomic and physiological information, an approach called “fusion imaging.”

CONCLUSIONS

1) Ventricular septal rupture complicates 1% of STEMI in our study population.Incidence of VSR was common among female sex, elderly age group,hypertensive and among non smokers.

2) Angina was absent in most cases of VSR in females .

3) All VSR cases were associated with first myocardial infarction.

4) VSR was common in AWMI than in IWMI.

5) Mortality was more in AWMI complicated by Ventricular septal rupture than in IWMI complicated by VSR.

6) VSR was more commonly noted in delayed present cases and in patients not thrombolysed.

7) Cardiogenic shock was noted in most patients with VSR . With the onset of VSR 50 % of patients were presented with cardiogenic shock, where as within 12 to 24 hours of onset of VSR 86% were in cardiogenic shock.

8) Majority of cases presented with sinus Tachycardia and Hypotension.

9) 2D Echo could not visualize the site of defect without colour Doppler in 30 % cases.

10) 2D Echo could not Identify the Exact shape of VSR and complexity of lesion and also the lesion extent.

11) 3D Echo imaging Identified the exact location of the VSR even prior to color Doppler and shape of the lesion as elliptical, oval or irregular was identified from RV and LV aspects.

12) With live 3D Echo VSR was demonstrated in en face from Left ventricular and Right ventricular side.

13) LV volume and EF was more accurate by 3D Echo, surface rendered method where as LVEF by 2D Echo under estimate the LV volume and LVEF.

14) Single vessel lesion with total obstruction was the major CAG findings noted and no collaterals noted during coronary angiogram.

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GLOSSARY AND ACRONYMS

VSR- ventricular septal rupture

AWMI- Anterior wall myocardial infarction.

IWMI- Inferior wall myocardial infarction.

RWMI- Right ventricular myocardial infarction.

LWMI- Lateral wall myocardial infarction.

CAD- coronary artery disease.

TEE- Trans esophageal echocardiogram.

TTE- Trans thoracic echocardiogram.

RT3D ECHO- Real Time 3 Dimentional Echocardiogram.

MRI- Magnetic Resonance Imaging.

STEMI- ST segment Elevation Myocardial Infarction.

LVEF- Left Ventricular Ejection Fraction.

CS- Cardiogenic Shock.

RV- Right ventricle; LV-Left ventricle.

PN	Name	ID/CD	Age	Sex	Angina	SOB	SYN	CVA	Others	TW	SK	SHT	DM	CAD	FH	Smok	Dysli	CABG
1	arumugam	4948	52	1	1	1	0	0	0	6	1	1	0	0	0	1	1	0
2	basha	839895	64	1	1	1	0	0	0	36	0	1	0	0	1	1	0	0
3	Chellamuthu	46401	72	1	1	1	1	0	0	30	0	1	1	1	0	0	1	0
4	jayaraman	164812	67	1	1	1	0	0	0	26	0	1	0	0	0	1	0	0
5	rajagopal	861856	45	1	0	1	1	0	0	18	0	1	0	0	0	0	0	0
6	srinivasan	813730	61	1	1	1	0	0	0	120	0	1	1	0	0	1	0	0
7	babuji	32654	67	1	0	1	0	0	0	32	0	1	0	0	1	0	1	0
8	chandran	413114	56	1	1	0	0	0	0	12	1	0	0	0	0	1	0	0
9	gopal	814508	70	1	0	1	0	0	0	27	0	1	0	0	0	0	0	0
10	manoharan	420514	67	1	1	1	0	0	0	10	1	1	0	0	0	1	1	0
11	selvam	505512	68	1	0	0	0	0	0	26	0	1	0	0	0	0	0	0
12	subramaniam	41388	55	1	1	1	0	0	0	8	0	1	1	1	1	1	0	0
13	dhawamani	809742	62	2	0	1	1	0	0	19	0	1	1	0	0	0	0	0
14	gangabai	792628	76	2	0	1	0	0	0	26	0	1	1	0	0	0	1	0
15	ghosbee	3102	57	2	1	1	0	0	0	6	1	1	1	1	0	0	0	0
16	kullammal	403012	67	2	0	1	0	0	0	26	0	1	0	0	0	0	0	0
17	muniammal	67182	70	2	1	1	1	0	0	19	0	1	1	0	1	0	0	0
18	novrojee	374812	56	2	0	1	0	0	0	14	0	0	0	0	0	0	0	0
19	shantha	462712	59	2	0	1	0	0	0	18	0	1	0	0	0	0	1	0
20	thallavi	303413	66	2	1	1	0	0	0	17	0	1	1	0	0	0	0	0
21	vairam	432012	54	2	0	1	0	0	0	30	0	0	0	0	0	0	0	0
22	chaellaiya	787622	60	1	1	0	0	0	0	12	1	0	0	0	0	0	0	0
23	laksmai	788669	60	2	0	1	0	0	0	27	0	1	0	1	0	0	1	0
24	dhanalaksmi	790880	80	2	0	1	1	0	0	18	0	1	0	0	0	0	0	0
25	pappathi	766447	61	2	0	1	1	1	0	6	1	1	1	0	1	0	0	0
26	verammal	797371	47	2	1	1	0	0	0	8	1	1	1	0	0	0	0	0
27	ameena bee	847452	65	2	0	1	0	0	0	10	1	1	0	0	0	0	1	0
28	mahalaksmi	803052	60	2	1	1	0	0	0	6	1	1	1	0	0	0	0	0
29	jaithumbee	805130	65	2	1	1	0	0	0	6	1	1	1	0	0	0	0	0
30	navaneethammal	839894	67	2	0	1	0	0	0	26	0	1	0	0	0	0	1	0
31	jayammal	847496	70	2	1	1	1	0	0	19	0	1	1	0	0	0	0	0
32	alamelu	860913	56	2	0	1	0	0	0	14	0	0	0	0	1	0	0	0

33	mohan	24076	59	1	1	1	0	0	0	18	0	1	0	1	0	0	1	0
34	sundaram	75222	55	1	1	1	0	0	0	17	0	1	0	0	0	1	0	0
35	jalil	78446	65	1	1	1	0	0	0	23	0	0	0	0	0	0	0	0
36	shanmabai	74630	60	2	1	0	0	0	0	22	0	0	1	0	0	0	0	0
37	govidaraj	83870	50	1	1	1	0	0	0	9	1	1	0	0	0	0	1	0
38	ellammal	54918	70	2	0	1	1	0	0	18	0	0	0	0	1	0	0	0
39	johnbasha	58388	64	1	0	1	1	1	0	6	1	1	1	0	0	0	0	0
40	shantha bai	74630	65	2	1	1	0	0	0	13	0	1	1	0	0	0	0	0
41	pitcahi	3533	49	1	1	1	0	0	0	8	1	0	0	0	0	0	0	0
42	annammal	1719	65	2	0	1	0	0	0	26	0	1	1	1	0	0	1	0
43	sivabhakkiyam	88683	60	2	1	0	0	0	0	34	0	0	1	0	0	0	1	0
44	kaliammal	16731	62	2	1	0	0	0	0	8	1	1	1	0	0	0	0	0

PN	Name	MI	AW	IW	PW	LW	ECG ST	RVMI	Kilip	Ki 24	Arr	HR	SBP	DBP	VSR_day	Thrill	Renal	vsr B/A	2D PG	2D Size	2DEF
1	arumugam	1	0	1	0	0	0	1	2	3	6	98	96	60	1	0	N	B	57	0	46
2	basha	1	0	1	0	0	1	1	4	4	2	104	80	58	3	1	E	B	74	0	48
3	Chellamuthu	1	1	0	0	0	0	0	2	4	1	120	94	60	4	0	N	A	40	0	36
4	jayaraman	1	1	0	0	0	1	0	2	3	2	110	100	70	3	0	N	A	56	3	42
5	rajagopal	1	1	0	0	1	1	0	3	4	2	116	98	72	1	0	N	A	47	4	40
6	srinivasan	1	1	0	0	0	1	0	3	4	3	98	100	74	5	0	E	A	56	0	38
7	babuji	1	1	0	0	0	1	0	2	3	4	105	104	74	4	1	N	A	36	0	40
8	chandran	1	1	0	0	1	0	0	2	3	2	110	94	62	4	1	N	A	54	12	45
9	gopal	1	1	0	0	0	1	0	4	4	1	105	90	70	5	0	E	A	45	0	35
10	manoharan	1	0	1	0	0	1	1	2	4	1	110	80	60	1	0	N	B	40	0	38
11	selvam	1	0	1	1	1	1	1	4	4	1	128	84	62	2	0	E	B	22	0	35
12	subramaniam	1	0	1	1	0	1	1	4	4	1	130	70	54	1	1	E	B	49	10	28
13	dhawamani	1	1	0	0	0	1	0	4	4	1	120	70	56	1	0	E	A	46	6	40
14	gangabai	1	0	1	0	0	1	1	4	4	1	130	74	52	3	0	E	B	48	16	46
15	ghosbee	1	1	0	0	0	1	0	3	4	1	108	96	60	1	1	N	A	66	0	44
16	kullammal	1	1	0	0	0	1	0	4	4	1	114	80	56	3	0	E	A	64	5	34
17	muniammal	1	1	0	0	0	0	0	2	4	5	120	94	58	5	1	N	A	76	0	36
18	novrojee	1	0	1	1	0	0	1	3	4	0	98	100	68	4	1	N	B	54	0	34
19	shantha	1	1	0	0	1	1	0	3	4	1	126	94	62	4	0	E	A	73	4	40
20	thallavi	1	1	0	0	0	1	0	3	4	0	98	100	74	5	1	E	A	42	0	42
21	vairam	1	0	1	0	0	0	1	2	3	1	125	104	68	4	1	N	B	46	0	40
22	chaellaiya	1	1	0	0	1	1	0	4	4	1	110	80	52	4	1	E	A	46	0	32
23	laksmai	1	1	0	0	0	1	0	4	4	1	117	90	70	5	1	E	A	42	4	35
24	dhanalaksmi	1	1	0	0	0	1	0	2	4	0	80	70	50	1	0	E	A	40	0	30
25	pappathi	1	0	1	1	0	1	1	4	4	1	120	80	62	4	1	E	B	34	0	35
26	verammal	1	1	0	0	0	1	1	4	4	4	130	70	54	1	0	E	A	46	5	35
27	ameena bee	1	1	0	0	0	1	0	4	4	2	110	70	50	4	0	E	A	32	4	30
28	mahalaksmi	1	1	0	0	0	0	0	4	4	1	130	70	50	1	0	E	A	46	7	36
29	jaithumbee	1	1	0	0	0	1	0	4	4	1	108	96	60	1	0	N	A	56	0	40
30	navaneethammal	1	1	0	0	1	1	0	4	4	1	114	80	56	3	0	E	A	64	0	46

31	jayammal	1	1	0	0	0	0	0	2	4	1	120	94	58	5	0	N	A	34	7	38
32	alamelu	1	0	1	1	0	1	1	3	4	0	98	100	68	4	1	N	B	48	0	46
33	mohan	1	1	0	0	1	1	0	4	4	2	126	94	62	4	0	N	A	34	5	40
34	sundaram	1	1	0	0	0	1	0	2	4	0	98	100	74	5	1	E	A	54	0	45
35	jalil	1	1	0	0	0	1	0	2	4	1	115	84	58	4	0	N	A	62	0	40
36	shanmabai	1	0	1	0	0	1	1	4	4	3	90	86	50	3	0	E	B	34	0	42
37	govidaraj	1	1	0	0	0	1	0	4	4	1	117	90	70	5	0	N	A	46	8	45
38	ellammal	1	1	0	0	0	0	0	2	4	1	102	80	62	4	0	E	A	34	7	38
39	johnbasha	1	0	1	1	0	1	1	4	4	1	120	80	60	4	0	E	B	28	10	46
40	shantha bai	1	0	1	0	0	0	1	4	4	1	130	70	54	3	0	E	B	34	0	36
41	pitcahi	1	1	0	0	0	0	0	1	3	0	90	106	70	2	0	N	A	64	0	46
42	annammal	1	1	0	0	0	1	0	4	4	1	130	70	50	1	0	E	A	46	0	30
43	sivabhakkiyam	1	1	0	0	0	0	0	2	4	1	110	86	54	2	0	N	A	34	0	48
44	kaliammal	1	1	0	0	0	0	0	4	4	4	110	86	60	3	0	N	A	46	7	40

PN	NAME	DD	FWR	PHT	MR	PE	LVH	RWMA	RV,LW	WMSI	LVEF3D	DD_3	RMWA_3	RV	PE 3D	LVH 3D	B/A
1	arumugam	I	0	2	11	0	0	IW	RV	1.25	48	I	IW	RV	0	0	B
2	basha	I	0	0	11	0	0	IW	RV	1.25	36	I	IW	RV	0	0	B
3	Chellamuthu	I	0	0	11	0	0	AW		1.37	40	I	AW		0	0	A
4	jayaraman	I	0	1	11	0	1	AW		1.5	40	I	AW		0	1	A
5	rajagopal	III	0	2	21	1	1	AW		2	36	III	AW		1	1	A
6	srinivasan	II	0	0	21	1	1	AW		2.1	40	II	AW		1	1	A
7	babuji	I	0	0	21	0	0	AW		2.1	42	I	AW		0	0	A
8	chandran	I	0	0	0	1	0	AW		1.5	36	I	AW		1	0	A
9	gopal	III	0	0	0	0	0	AW		2.1	30	III	AW		0	0	A
10	manoharan	I	0	1	11	0	0	IW		2.1	30	I	IW		0	0	B
11	selvam	III	0	3	31	0	1	IW	RV	1.62	30	III	IW	RV/LW	0	1	B
12	subramaniam	I	0	0	11	0	1	IW	RV	1.62	34	I	IW	RV	0	1	B
13	dhawamani	II	0	1	0	0	0	AW		2.1	30	II	AW		0	0	A
14	gangabai	III	1	1	11	1	0	IW		2.1	45	III	IW	RV	1	0	B
15	ghosbee	I	0	1	11	1	1	AW	LW	1.25	38	I	AW		1	1	A
16	kullammal	I	0	0	11	0	1	AW		1.25	30	I	AW		0	1	A
17	muniammal	III	0	0	11	0	0	AW		1.37	40	III	AW		0	0	A
18	novrojee	I	0	2	11	0	0	IW		1.5	35	I	IW		0	0	B
19	shantha	III	1	0	11	1	1	AW		2	36	III	AW		1	1	A
20	thallavi	I	0	2	0	1	0	AW		2.1	40	I	AW		1	0	A
21	vairam	II	0	2	0	1	1	IW	RV	2.1	40	II	IW		1	1	B
22	chaellaiya	III	0	1	1	0	0	AW		1.5	25	III	AW		0	0	A
23	laksmai	I	0	2	0	0	0	AW		2.1	30	I	AW		0	0	A
24	dhanalaksmi	III	0	1	11	0	0	AW		2.1	28	III	AW		0	0	A
25	pappathi	III	0	2	1	0	1	IW	RV	1.62	27	III	IW	RV	0	1	B
26	verammal	II	0	0	11	0	1	AW	LW	1.62	30	II	AW		0	1	A
27	ameena bee	III	0	1	0	0	1	AW		2.1	30	III	AW		0	1	A
28	mahalaksmi	I	0	0	21	0	0	AW		2.1	28	I	AW		0	0	A
29	jaithumbee	I	0	1	11	0	1	AW		1.2	40	I	AW		0	1	A
30	Navaneethal	III	0	1	11	1	0	AW		1.2	40	III	AW		1	0	A
31	jayammal	III	0	3	21	0	1	AW		1.3	36	III	AW		0	1	A
32	alamelu	I	0	1	11	0	0	IW		1.5	34	I	IW		0	0	B

33	mohan	III	0	1	11	0	0	AW		2	40	III	AW		0	0	A
34	sundaram	I	0	1	0	1	1	AW		2.1	45	I	AW		1	1	A
35	jalil	III	0	0	0	0	0	AW		2.1	40	III	IW		0	0	A
36	shanmabai	III	0	3	11	0	0	IW		1.5	35	III	IW		0	0	B
37	govidaraj	I	0	1	11	0	1	AW		2.1	45	I	AW		0	1	A
38	ellammal	III	0	1	21	1	0	AW		2.1	38	III	AW		1	0	A
39	johnbasha	III	0	3	1	0	0	IW	RV	1.6	36	III	IW	RV	0	0	B
40	shantha bai	II	0	1	11	0	0	IW	RV	1.62	36	II	IW	RV	0	0	B
41	pitcahi	III	0	3	0	0	0	AW		2.1	45	III	AW		0	0	A
42	annammal	I	0	0	0	0	0	AW		2.1	30	I	AW		0	0	A
43	sivabhakkiyam	I	0	1	21	0	0	AW		2.1	30	I	AW		0	0	A
44	kaliammal	I	1	1	11	1	0	AW		2.1	40	I	AW		1	0	A

PN	NAME	PG3D	Shap3D	Size3D	Area	Ext	Single	Simple	MR	PMT	FWR	PHT
1	arumugam	57	O	4	0.8	0	S	S	11	0	0	2
2	basha	74	O	5	1	0	S	C	11	0	0	0
3	Chellamuthu	40	E	3	0.8	0	S	S	11	0	0	0
4	jayaraman	56	E	7	2	0	S	S	11	0	0	1
5	rajagopal	47	O	5	1	0	S	C	21	0	0	2
6	srinivasan	56	E	6	1.3	0	S	C	21	0	0	0
7	babuji	36	O	4	0.8	0	S	S	21	0	0	0
8	chandran	54	E	15	3.2	0	S	C	0	0	0	0
9	gopal	45	E	5	1	0	S	S	0	0	0	0
10	manoharan	40	E	5	1	0	S	S	11	0	0	1
11	selvam	22	E	6	1.3	0	S	C	31	0	0	3
12	subramaniam	49	E	15	3.8	0	S	C	11	0	0	0
13	dhawamani	46	O	9	2.2	0	S	S	0	0	0	1
14	gangabai	48	O	18	3.2	1	S	C	11	0	1	1
15	ghosbee	66	O	5	1	0	S	S	11	0	0	1
16	kullammal	64	O	6	1.3	0	S	C	11	0	0	0
17	muniammal	76	O	6	1.3	0	S	S	11	0	0	0
18	novrojee	54	E	6	1.3	0	S	S	11	0	0	2
19	shantha	73	O	5	1	1	S	C	11	0	1	0
20	thallavi	42	O	4	0.8	0	S	S	0	0	0	2
21	vairam	46	O	6	1.3	0	S	S	0	0	0	2
22	chaellaiya	46	O	10	3	0	S	C	1	0	0	1
23	laksmai	42	O	8	2.7	0	S	S	0	0	0	2
24	dhanalaksmi	40	O	7	2.1	0	S	C	11	0	0	1
25	pappathi	34	E	6	1.3	0	S	C	1	0	0	2
26	verammal	46	E	9	2.8	0	S	S	11	0	0	0
27	ameena bee	32	O	7	2.2	0	S	S	0	0	0	1
28	mahalaksmi	46	O	12	3	0	S	C	21	0	0	0
29	jaithumbee	56	O	7	2	0	S	S	11	0	0	1
30	navaneethammal	64	O	6	1.3	0	S	C	11	0	0	1

31	jayammal	34	O	12	3	0	S	C	21	0	0	3
32	alamelu	48	E	6	1.3	0	S	S	11	0	0	1
33	mohan	34	O	5	1	0	S	C	11	0	0	1
34	sundaram	54	O	4	0.8	0	S	S	0	0	0	1
35	jalil	62	O	5	1	0	S	S	0	0	0	0
36	shanmabai	34	O	7	2.2	0	S	C	11	0	0	3
37	govidaraj	46	O	7	2.2	0	S	S	11	0	0	1
38	ellammal	34	E	5	1	0	S	S	21	0	0	1
39	johnbasha	28	E	6	1.3	0	S	C	1	0	0	3
40	shantha bai	34	O	5	1	0	S	S	11	0	0	1
41	pitcahi	64	O	4	0.8	0	S	S	0	0	0	3
42	annammal	46	O	7	2.2	0	S	C	0	0	0	0
43	sivabhakkiyam	34	E	11	3	0	S	C	21	0	0	1
44	kaliammal	46	E	13	3.4	1	S	C	11	0	1	1

PN	NAME	LCX	RCA	COLLAT	Mort7/30	VSR repair	CABG
1	arumugam	0	0	0	2	0	0
2	basha	0	0	0	1	0	0
3	Chellamuthu	0	0	0	2	0	0
4	jayaraman	1	0	0	0	1	0
5	rajagopal	0	0	0	1	0	0
6	srinivasan	0	0	0	1	0	0
7	babuji	1	0	0	0	1	0
8	chandran	0	0	0	2	0	0
9	gopal	0	0	0	2	0	0
10	manoharan	0	0	0	2	0	0
11	selvam	0	0	0	1	0	0
12	subramaniam	0	0	0	2	0	0
13	dhawamani	0	0	0	2	0	0
14	gangabai	0	1	0	1	0	0
15	ghosbee	0	0	0	2	0	0
16	kullammal	0	0	0	1	0	0

17	muniammal	0	0	0	2	0	0
18	novrojee	0	0	0	0	0	0
19	shantha	0	0	0	1	0	0
20	thallavi	0	0	0	2	0	0
21	vairam	0	0	0	0	0	0
22	chaellaiya	0	0	0	1	0	0
23	laksmai	0	0	0	2	0	0
24	dhanalaksmi	0	0	0	1	0	0
25	pappathi	0	0	0	1	0	0
26	verammal	0	0	0	2	0	0
27	ameena bee	0	0	0	2	0	0
28	mahalaksmi	0	0	0	1	0	0
29	jaithumbee	0	0	0	2	0	0
30	navaneethammal	0	0	0	1	0	0
31	jayammal	0	0	0	2	0	0
32	alamelu	0	1	0	0	0	0
33	mohan	0	0	0	1	0	0
34	sundaram	0	0	0	2	0	0
35	jalil	0	0	0	0	0	0
36	shanmabai	0	0	0	1	0	0
37	govidaraj	0	0	0	0	1	0
38	ellammal	0	0	0	2	0	0
39	johnbasha	0	0	0	1	0	0
40	shantha bai	0	1	0	2	1	0
41	pitcahi	0	0	0	2	0	0
42	annammal	0	0	0	1	0	0
43	sivabhakkiyam	0	0	0	0	0	0
44	kaliammal	0	0	0	0	0	0