USEFULNESS OF PULMONARY REGURGITATION DOPPLER TRACINGS IN PREDICTING OUTCOME IN PATIENTS WITH ACUTE INFERIOR WALL MYOCARDIAL INFARCTION

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CERTIFICATE

This is to certify that this dissertation titled **"Usefulness Of Pulmonary Regurgitation Doppler Tracings In Predicting Outcome In Patients With Acute Inferior Wall Myocardial Infarction"** submitted by **DR.HEMANATH.T.R** to the faculty of Cardiology, The Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfilment of the requirement for the award of DM degree Branch II (Cardiology), is a bonafide research work carried out by him under our direct supervision and guidance. The period of post-graduate study and training was from August 2011 to July 2014.

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DECLARATION

I, **Dr. Hemanath T R,** solemnly declare that the dissertation titled **"Usefulness Of Pulmonary Regurgitation Doppler Tracings In Predicting Outcome In Patients With Acute Inferior Wall Myocardial Infarction"** has been prepared by me under the guidance and supervision of **Prof.M.S.Ravi M.D, D.M,** Professor and Head, Department of Cardiology, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai. This is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfilment of the rules and regulations for the award of DM degree (branch II) Cardiology.

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CONTENTS

ABBREVIATIONS

RVMI	: Right Ventricular Myocardial Infarction
IWMI	: Inferior Ventricular Myocardial Infarction
PR	: pulmonary Regurgitation
PHT	: Pressure Half Time
MPI	: Myocardial Performance Index
RVOT	: Right Ventricular Outflow Tract
RVEDP	: Right Ventricular End Diastolic Pressure
RVSP	: Right Ventricular Systolic Pressure
TR	: Tricuspid Regurgitation
TRPG	: Tricuspid Regurgitation Peak Gradient
TAPSE	: Tricuspid Annular Plane Systolic Excursion
TASV	: Tricuspid Annular Systolic Velocity
EDA	: End Diastolic Area
ESA	: End Systolic Area
FAC	: Fractional Area Change
EF	: Ejection Fraction
IVCT	: Isovolumic Contraction Time
IVRT	: Isovolumic Relaxation Time
ET	: Ejection Time

INTRODUCTION

Right ventricular (RV) acute myocardial infarction (AMI) occurs almost exclusively in setting of inferior wall left ventricular AMI [1-4]. It is known that impaired left ventricular (LV) function is a major determinant of prognosis in patients surviving acute myocardial infarction (MI). However, little and controversial information is available on the relationship between right ventricular (RV) dysfunction and mortality. In a recent report focusing on the relationship between RV ejection fraction and long-term prognosis in patients with MI, Pfisterer and associates [5] concluded that RV dysfunction contributes to the occurrence of cardiac death after MI independent of and in addition to LV impairment.

Non invasive hemodynamic diagnostic criteria, available at the bedside, may be useful in the acute phase of MI to allow recognition of high-risk patients with RV involvement. Zehender et al [6-7] reported that ST-segment elevation in lead V4R at the time of admission was a strong predictor of in-hospital complications. However, the diagnostic accuracy of non invasive diagnostic criteria varies in different studies [7-13].

RV echocardiographic study may represent a valuable alternative. Evaluation of RV systolic function as well as wall motion abnormalities or global RV function index is difficult because of inadequate apical windows and the unusual geometry of the right side of the heart. Continuous-wave Doppler

tracings of physiologic pulmonary regurgitation (PR) are highly promising tools because PR flow is directly related to the pressure gradient between the pulmonary artery and the right ventricle by the Bernoulli equation[14].

Pulmonary regurgitation (PR) flow-derived Doppler curve was useful in recognizing RV involvement during the first 24 hours of AMI. A PR Doppler pattern depends mainly on the diastolic RV pressure pattern, which is altered during RV ischemia and characterized by a disproportionate increase of RV end diastolic pressure. This physical relation led us to hypothesize that a modification of RV pressure could modify the regurgitant flow pattern.

To test this hypothesis, the present study was designed to systematically search for the presence of a pulmonary regurgitant jet in patients with inferior wall acute myocardial infarction and to compare the modifications of the flow pattern with clinical outcome.

AIMS OF THE STUDY

1. To evaluate the Doppler predictors of physiological pulmonary regurgitation in patients with right ventricular myocardial infarction in the setting of acute inferior wall acute myocardial infarction.

2. To assess the prognostic implications of Doppler characteristics of physiological pulmonary regurgitation with PR PHT $\leq$ 150 milliseconds and ratio between minimum and maximum Vmin / Vmax < 0.5 with respect to in-hospital events in patients with acute inferior wall myocardial infarction.

REVIEW OF LITERATURE

Anatomy of Pulmonary Valve

Semilunar valves are valve connecting between great arteries to that of corresponding ventricles to maintain blood flow in single direction. Annulus, cusps and commissures form three parts of these semilunar valves. Being simple in architecture when compared to atrio-ventricular valves, most of the opening and closure of the semilunar valves are passive in nature. Pulmonary valve lies closer to the chest wall than other cardiac valves. The pulmonary valve is also a valve which is situated away at some distance from other valves and the plane of the valve is towards left and towards posterior and the opening of the pulmonary valve is towards the left shoulder. The right ventricular myocardium has extensions into the pulmonary valve. The pulmonary valve does not have a proper fibrous annulus which is very important for tight closure of lunules. There is fibrous core inside the valve cusps are variously developed and covered by fold of endocardium. The cusps have small perforations near its free margin. These factors may attribute the presence of physiological valvular regurgitation in pulmonary valve which happens to be the most common valve having high incidence of physiological regurgitation.

The left pulmonary sinus has extension from septal band's antero-superior limb. Trabeculation lying parallel to parietal bands insert into right

pulmonary sinus. Pulmonary valve leaflets are thinner as they are operating on low pressure zone.

Anatomy of the Right Ventricle

Heart as a four chambered organ was first described by Leonardo da Vinci. He first described about moderator band in his drawings. Right ventricle is the anterior most chamber and it is situated behind the sternum. Right ventricle is a crescent shaped chamber while left ventricle is ellipsoidal in shape.

Right ventricular wall is thin of 3 to 5 mm thickness. Right ventricular wall is made up of circumferential fibres in the superficial layer and sub endocardial longitudinal muscle. Functionally both the right and left ventricles are bound together by the continuity between the muscle fibres which contribute to the ventricular interdependence.

Right ventricle has three regions namely inlet, trabecular and outlet segments [15]. Inlet part of right ventricle extends from the tricuspid valve annulus to the attachment of the papillary muscles. Trabecular part of right ventricle is below the papillary attachments up to the ventricular apex. Outlet part of right ventricle is also known as conus or infundibulum. It is smooth walled and contains pulmonary valve [14, 15].

Right ventricle is supplied in major part by the right coronary artery. A segment of the posterior part of right ventricle is supplied by postero-lateral branches of left circumflex artery in about 10%. Posterior descending artery which is a branch of right coronary artery supplies a major part of posterior segment of right ventricle. Even in a right dominant supply where right coronary artery supplies major part of the right ventricle, anterior wall and antero-septal region of right ventricle are supplied by branches of the left anterior descending coronary artery [16]. In about 24% of the human population, 30% of the right ventricular free wall is supplied by right ventricular branches of the left anterior descending coronary artery [17]. In 22% of population where the left anterior descending artery wraps around the apex, it may also supply the infero-posterior free wall of the right ventricle adjacent to the apex [18].

Morphologically right ventricle is different from left ventricle by the following features. First, atrio - ventricular valve which is attached to the right ventricle is a tricuspid valve while mitral valve attached to the left ventricle is bicuspid. Second, tricuspid valve has septal attachment while mitral valve has no septal attachment. Third, myocardium of the right ventricle is heavily trabeculated while that of the left ventricle is not trabeculated. Fourth, the right ventricle has a band of muscle attached from the base of the anterior papillary

muscle to the inter ventricular septum called moderator band while it is absent in the left ventricle [19].

Physiology of the Right Ventricle

Output of the right ventricle is the same as that of the left ventricle but the stroke work of the right ventricle is 75% less than that of the left ventricle. This is due to the highly compliant pulmonary vasculature when compared to the aorta. Hence according to Laplace's law which states that pressure is directly proportional to the product of the wall tension and wall thickness and inversely proportional to the radius of the cavity, right ventricle is thin walled.

When compared to the left ventricle, the endocardial layer of the right ventricle is thick especially in the inflow portion and the middle myocardial fibre layer in thin. Hence longitudinal fibre shortening plays a major role in ejection of blood from this chamber. 80% of the combined right ventricular volume is from the inflow portion of the right ventricle and hence more than 85% of the right ventricular stroke volume is from the sinus inflow portion of the right ventricle [20].

Another important difference between right and left ventricle is that the entire pattern of right ventricular contraction is different from that of the left ventricle. Unlike in left ventricle, the contraction of the right ventricle starts in

the inflow portion of the right ventricle and it moves like a peristaltic wave towards the infundibulum of the right ventricle [21].

Anatomy of the right ventricle is complex. The sinus portion (inlet) is separated from the outlet portion (infundibulum) by the crista supraventricularis. Right ventricular stroke volume is mainly due to the longitudinal fibre shortening than due to the circumferential fibre shortening [22]. There is a continuous interplay between the right and left ventricles due to the shared inter ventricular septum, common muscle bundles, right ventricular free wall attachment to the septum, shared blood flow and common pericardium.

Right ventricular function depends on the interplay between the intrinsic and extrinsic factors like ventricular interdependence, preload and after load. Right ventricular contraction is due to three major factors namely movement of right ventricular free wall towards the inter ventricular septum, tricuspid annulus descent to the apex producing long axis shortening and traction of the right ventricular free wall by the movement of the septum towards left ventricle during left ventricular systole [16]. This makes right ventricular contraction to occur as a peristaltic pattern and the right ventricular outflow tract contracts later than the inflow portion of the right ventricle by about 50 milli-seconds.

Functionally both the right and left ventricles are seen as two pumps working in series with right ventricle related to the highly compliant pulmonary

circulation and the left ventricle related to the highly resistant systemic circulation. Bernheim first described that alteration in the function of one ventricle will alter the function of the other ventricle. Bernheim effect is that left ventricular hypertrophy produces compression of the right ventricle which leads to right ventricular dysfunction. Reverse Bernheim effect is that development of left ventricular dysfunction due to the right ventricular pressure and volume overload. This is due to the shift of inter ventricular septum towards left ventricular cavity producing left ventricular dysfunction. The pericardium plays a major role in the diastolic interaction between the ventricles.

The contraction of the anterior wall of the left ventricle and inter ventricular septum plays a major role in the contraction of the right ventricle and hence in the right ventricular cardiac output. Inter ventricular septum and the left ventricle are mainly responsible for about 20 - 50% of the function of the right ventricle.

Echocardiographic assessment of right ventricular function [22]

Initially echocardiographic evaluation was more on the structure and function of the left ventricle. Evaluation of the right ventricle was prevented by the more complex anatomy of the right ventricle and poor echo window of the right ventricle as it is situated behind the sternum. As right ventricle gained

more importance in the management of patients with cardiac and pulmonary disorders and newer echocardiographic techniques were invented, echocardiographic evaluation of the right ventricle came into light.

Evaluation of the right ventricular dimension and function were first brought into guidelines by the recommendations of American society of echocardiography and European association of echocardiography which was published in 2005 [(23)]. However this recommendation gave only little importance to right ventricle when compared to the left ventricle. After this recommendation, there was a great advancement in the evaluation of the functions of the right ventricle.

Similar to left ventricle, right ventricle ejection fraction is considered to be the determinant of right ventricular function. However because of the complex anatomy of the right ventricle, right ventricular ejection fraction could not be measured accurately. In recent years many other parameters have been developed which are indicators of the right ventricular function.

The common parameters and echocardiographic views measured for RV functional assessment are shown in table.

Table : Echocardiographic assessment of RV

Echocardiographic views	Parameters
RV focussed Apical four chamber view	RV and RA size
Subcostal view	IVC dimension
Apical four chamber view PSAX at basal level Apical four chamber view Apical four chamber view Apical four chamber view Apical four chamber view Apical four chamber view	RV systolic function RIMP, TAPSE, 2D RV FAC, 2D RV EF, 3D RV EF, S' of tricuspid annulus, IVA

Abbreviations: EF: Ejection fraction; FAC: Fractional area change; IVA: Iso-volumic myocardial acceleration index; IVC: Inferior vena cava; TAPSE: Tricuspid annular plane systolic excursion

Definitions of parameters used for assessment of RV function

1. Fractional Area Change (FAC):

It is a measure of RV systolic function which has been shown to correlate well with RV ejection fraction on MRI. It is currently one of the recommended methods of quantitative estimation of RV function. The formula for estimation of FAC is as follows

$$\frac{\text{EDA-ESA}}{\text{ESA}} \times 100$$

Where EDA is RV end diastolic area and ESA is RV end systolic area.

2. 2D RV EF estimation:

The complex crescent shaped geometry of right ventricle precludes the accurate assessment of RV ejection fraction precisely using conventional methods. RVEF is measured using the area length method or disc summation method using the apical four chamber view predominantly. The major disadvantage with the use of this parameter is that the RV volumes are underestimated because of exclusion of RVOT. This parameter is not currently recommended because of heterogeneity of methods and geometric complexity of the RV. The formula for estimation of RV EF is as follows

$$\frac{\text{EDV-ESV}}{\text{EDV}} \times 100$$

EDV is the end diastolic volume and ESV is the end systolic volume.

Definitions of parameters used for hemodynamic assessment:

1. RVSP/SPAP:

This is estimated using TR velocity with a simplified Bernoulli equation and combining this value with an estimate of RA pressure. RA pressure is

calculated from IVC diameter and its respiratory variations. In the absence of gradient across the pulmonary valve or RVOT, SPAP equals RVSP. Doppler sweep speeds of 100mm/sec to be used for tracings. Signal can be augmented with agitated saline or contrast if the same is weak. Overestimation of spectrum can be avoided by ensuring that only well defined dense spectral profile is measured. This parameter is measured using the following formula

$$\textbf{RVSP} = \textbf{4V}^2 \textbf{ + RA pressure}$$

Where V is peak TR velocity in m/sec

The cut off value for peak TR velocity is 2.8-2.9 m/sec, whereas the peak gradient is usually less that 35-36 mm Hg. Estimation of RA pressure on the basis of IVC diameter and collapse is shown in the following table.

Table RA pressure versus IVC diameter

RA pressure	Normal (0-5 mm Hg)	Intermediate (5-10 mm Hg)		High (>10 mm Hg)
IVC diameter	<2.1 cm	<2.1 cm	>2.1 cm	>2.1 cm
Collapse with sniff	>50%	<50%	>50%	<50%

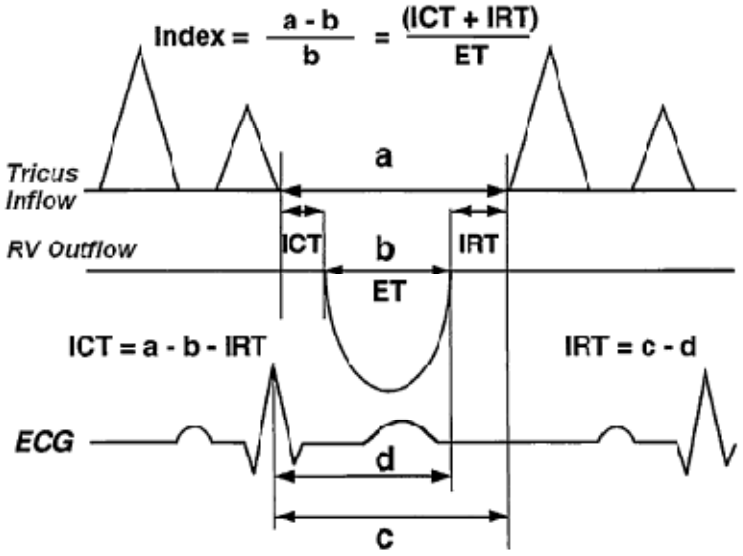
2. Non volumetric assessment: RV has superficial circumferential muscle fibers responsible for its inward bellow movement as well as inner longitudinal fibers that result in base apex contraction. Assessment of RV function includes global and regional assessment. Global assessment includes RV-MPI, RV dp/dt, RVEF, RVFAC and IVA. Regional assessment includes Doppler derived systolic annular velocity (S') and TAPSE.

(a). RV dp/dt: this gives the rate of pressure rise in the ventricle and is an index of ventricular contractility. This can be accurately estimated from TR continuous wave Doppler signal. It is load dependent and is calculated by measuring time required for TR jet to increase in velocity from 1 to 2 m/sec. A value of <400mmHg/sec is considered as abnormal.

(b): RV-MPI: This is also known as the RV Tei index. In 1995, Chuwa Tei et al published in the Journal of Cardiology about new non invasive index to measure the global ventricular function [(24)]. This index is known by the author's name Tei index. It is also known as myocardial performance index. This index was first used in 1995 to study the global function of the ventricle in dilated cardiomyopathy patients [(25)] and to study the systolic and diastolic function of the patients with cardiac amyloidosis [(26)].

It gives a global measure of both systolic and diastolic function of the RV. It is basically derived from the following formula:

RV Tei index = ratio of IVCT+IVRT/ET



This parameter can be measured by two methods:

(i)PW method: ET is measured with PW of RVOT and TV closure-opening time from measured from PW Doppler of tricuspid inflow or continuous wave Doppler of TR jet. These measurements are taken from different images.

(ii) Tissue Doppler method: all time intervals are measured from a single beat by pulsing the tricuspid annulus. It can be recorded from medial or lateral annulus of TV. The advantage of recording from the lateral mitral annulus is that errors due to changes in the heart rate can be avoided.

A value of >0.40 on PW Doppler and >0.55 on tissue wave Doppler is considered as abnormal. The advantages of measuring this parameter include reproducibility and feasibility and avoidance of geometric assumptions. The

disadvantages are that it is load dependent and is also unreliable when measured with different R-R intervals as in atrial fibrillation.

Right ventricular myocardial performance index is calculated as the ratio of isovolumic time and right ventricular ejection time. Isovolumic time is the sum of isovolumic contraction time and isovolumic relaxation time.

The mean normal value of myocardial performance index for right ventricle is 0.28 ± 0.04 [32]. According to ASE/EAE guidelines, Values less than 0.40 is considered normal for the right ventricle. Values more than 0.40 are indicative of right ventricular dysfunction.

Tei index is a simple, non invasive, reproducible index. It has been documented in many studies that it is independent of heart rate, ventricular dimension, arterial pressure, regurgitation of the atrio ventricular valve, preload and after load [25].

In a study published in Journal of American College of Cardiology in 1996, chewa Tei et al showed good correlation of Doppler derived myocardial performance index with the global cardiac function in patients with cardiac amyloidosis [29].

In a study published in Echocardiography (2008), Karnati et al has shown excellent correlation between right ventricular myocardial performance index and right ventricular ejection fraction calculated by nuclear ventriculography [52]. In this study, the sensitivity and specificity for right ventricular performance index value more than 0.50 were 45.4% and 100% respectively while using right ventricular ejection fraction measured by nuclear ventriculography as less than 45%. The study had a conclusion that right ventricular dysfunction is present when myocardial performance index value is more than 0.50.

In a study published in Echocardiography (August 2012), Vizzardi et al has shown that right ventricular Tei index had a more prognostic impact on moderate chronic heart failure when compared with other functional parameters of the right ventricle like tricuspid annular plane systolic excursion and right ventricular fractional area change [27].

In another study Maheswari et al compared right ventricular Tei index with right ventricular ejection fraction calculated by Simpson's method in patients with isolated left ventricular anterior wall myocardial infarction [28]. This study showed that Right ventricular myocardial performance index was more sensitive in detecting early right ventricular dysfunction than Simpson's method of right ventricular ejection fraction.

In another study published in Journal of American Society of Echocardiography 2004, Miller et at compared TAPSE and myocardial performance index with the right ventricular ejection fraction calculated using Simpson's method [29]. Using Simpson's method of right ventricular ejection fraction less than 50% myocardial performance index less than 0.40 had 100% sensitivity and 100% negative predictive value. However this study showed myocardial performance index was less specific and had a less positive predictive value.

(c): Isovolumic contraction myocardial acceleration index (IVA):

This is defined as peak isovolumic myocardial velocity divided by time to peak velocity. It is measured by Doppler tissue imaging at the lateral tricuspid annulus and is considered as the most consistent tissue Doppler index for evaluation of RV function. It has been demonstrated to correlate with severity of illness in conditions affecting RV function like mitral stenosis. It normally lies between 1.5-3 m/sec^2. The advantages include that it measures global RV function and is less load dependent. The disadvantages are that it is age dependent, heart rate dependent and angle dependent.

Regional assessment of RV function:

(i)TAPSE: Tricuspid annular plane systolic excursion:

This is a method to measure the distance of systolic excursion of RV annular segment along its longitudinal plane. It is measured in the apical four chamber view and represents the longitudinal function of the RV. The right ventricular free wall contracts predominantly in a longitudinal axis due to the longitudinal muscle fibres, systolic movement of the base of the right ventricular free wall towards the apex is one of the most prominent movements seen in echocardiography.

It is acquired by placing the 'M' mode cursor through the tricuspid annulus. TAPSE correlated strongly with radionuclide angiography in a study by Kaul et al[30] the normal value is <17 mm. the advantages include simplicity and reproducibility. The disadvantages are that it is angle dependent and load dependent. It has been recommended that TAPSE should be routinely used as a simple method of estimating RV function. TAPSE is m-mode displacement of basal portions of right ventricle during cardiac contraction which is measured from apical four chamber view, has sensitivity of 59% and specificity of around 94% for the detection of RV ejection fraction <50%. According to ASE/EAE guidelines, value less than 16 cm was considered abnormal.

In a study published in Post graduate Medicine Journal 2008, Lopez - Candales et al, studied about right ventricular function in patients with pulmonary hypertension [31]. TAPSE correlated well with right ventricular

dysfunction. TAPSE value below 20 mm was seen with severe pulmonary hypertension.

In another study published in Journal of American Society of Echocardiography 2004, Miller et at compared TAPSE and myocardial performance index with the right ventricular ejection fraction calculated using Simpson's method [29]. Using Simpson's method of right ventricular ejection fraction less than 50%, TAPSE had a good correlation with right ventricular function. With TAPSE value less than 1.5 cm, it had 89% specificity and 92% negative predictive value.

In a study published in International Journal of Cardiology 2007, Tamborini et al compared right ventricular function in various cardiac disorder patients with age matched normal control people. This study concluded that TAPSE had high specificity in detecting right ventricular dysfunction [32].

In another study done by Stephano Ghio which was published in the American Journal of Cardiology 2000, 140 patients with left ventricular ejection fraction less than 35% and chronic heart failure underwent echocardiographic evaluation and were followed for two years. Tricuspid annular plane systolic excursion added prognostic information and correlated well with patients having NYHA class III or IV [33].

(ii) Tissue Doppler imaging:

Tricuspid Annular Peak Systolic Velocity (S')

This measures the longitudinal velocity of excursion and termed as RV S' or systolic excursion velocity. The PW Doppler sample volume is placed in either the tricuspid annulus or middle of the basal segment of RV free wall. The peak S' wave form which is due to the right ventricular contraction occurs during mechanical systole and it follows pulmonary valve opening.

An S' value of <10cm/sec raises the suspicion of abnormal RV function. The advantages are that it is simple and reproducible. The disadvantage is that it is angle dependent. The waveforms should be properly understood to measure the right ventricular systolic and diastolic function using Doppler tissue imaging.

In a study published in the European Heart Journal in 2001, Meluzin et al studied tissue Doppler imaging in patients with heart failure. In this study, S' calculated correlated well with right ventricular ejection fraction. Tricuspid annular peak systolic velocity less than 11.5 cm/s was found to have 90% sensitivity and 85% specificity with right ventricular dysfunction having ejection fraction less than 45% [(34)].

In another study in 2006 which was published in Echocardiography journal, Saxena et al compared tricuspid annular peak systolic excursion (TAPSE), tricuspid annular peak systolic velocity (S') and right ventricular

fractional area change (FAC) to assess right ventricular function in patients with pulmonary hypertension [35]. This study showed good correlation between S' and TAPSE and S' and right ventricular fractional area change. This study concluded that tricuspid annular peak systolic velocity should be used in the assessment of the right ventricular function as it is easy to measure and it is less time consuming.

In a Swiss study done by David Tiiller, systolic funtion of the right ventricle was assessed using tricuspid annular peak systolic velocity. This study showed that measurement of the systolic velocity of the lateral annulus of the tricuspic valve correlated with the right ventricular systolic function [36].

Diastolic function of the Right Ventricle.

Earlier right ventricle was considered as a passive chamber. But now its not true. Any acute right ventricular ischemia or injury produces severe diastolic dysfunction of the right ventricle, which leads to raised filling pressure of the right ventricle [37].

The diastolic function of the right ventricle is assessed using transtricuspid flow doppler velocities (E, A, and E/A), tricuspid annulus tissue Doppler velocities (e', a', e'/a'), deceleration time and isovolumic relaxation

time. E/A value between0.8 and less than 2.1 and E/e' value more than 6 suggests pseudo normal filling.

Age has a correlation with the E/A ratio. For each decade, there is a decrease of 0.1 in the E/A ratio [38, 39]. During inspiration, there is and increase in E and E/A ratio. There is a greater increase in A velocity when compared to E velocity during tachycardia and hence E/A ratio decreases during tachycardia [40].

Right ventricular diastolic dysfunction is an indicator of mortality in patients with chronic cardiac failure and pulmonary hypertension [32]. The response to treatment is reflected by the filling pattern of diastole. It has been shown in many studies that diastolic dysfunction of the right ventricle precedes right ventricular systolic dysfunction and hence it is a marker of subclinical right ventricular dysfunction.

Echocardiographic assessment of Pulmonary Valve

The Incidence of pulmonary regurgitation in normal individuals varies from 40-78 % [41-43]. Among four cardiac valves the incidence of physiological regurgitation is highest in pulmonary valve. Two-dimensional echocardiogram usually visualise one or two leaflets of pulmonary valve simultaneously. The pulmonary valve can also be seen as a short axis view in some individuals

where all the three leaflets are visualised simultaneously. In trans-oesophageal echocardiography pulmonary valve is difficult because it is far away from the transducer. [44]

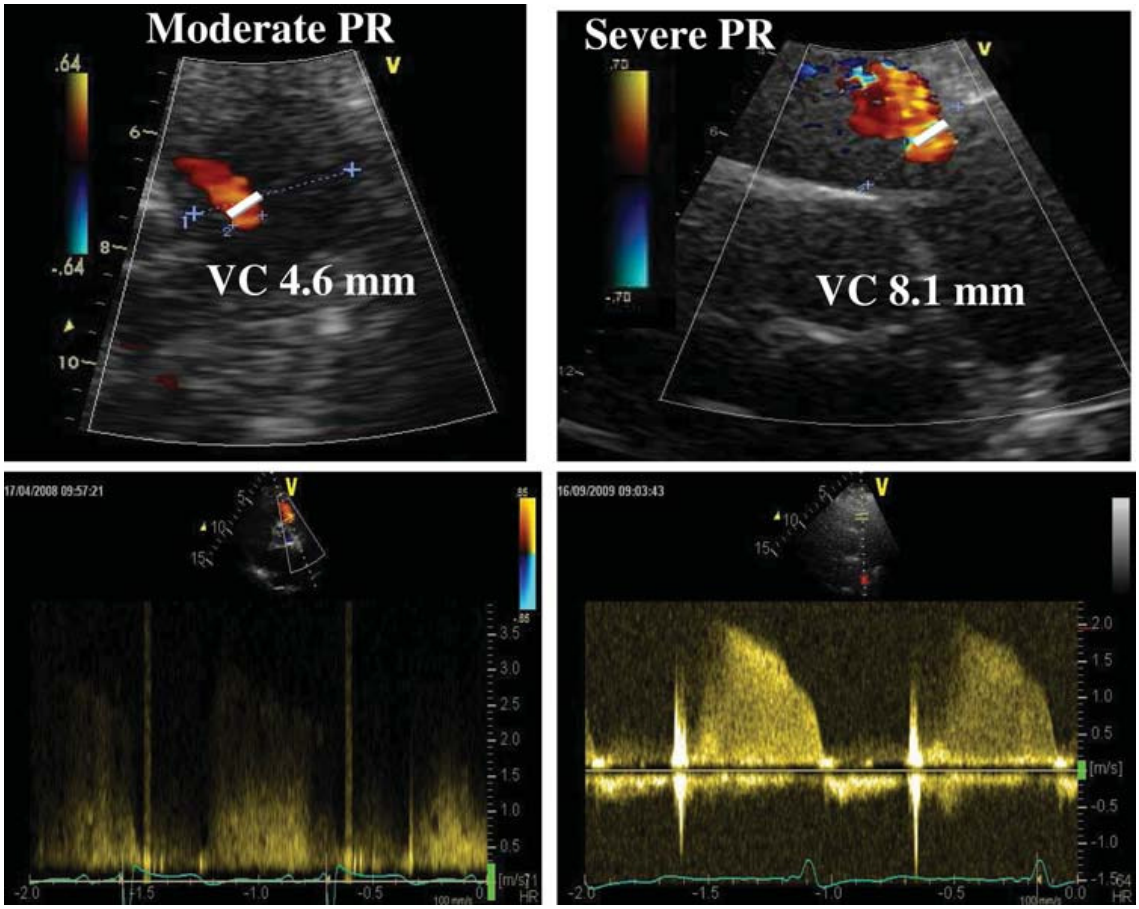
Colour Flow Doppler

Colour flow Doppler is the main method to detect pulmonary regurgitation by visualising a diastolic colour jet in right ventricular outflow tract towards RV cavity. In Pulsed Wave Doppler the forward flow and backward flow can be used for the assessment of regurgitant volume and regurgitant fraction. The density of the continuous wave Doppler gives a qualitative evaluation of PR. But both these were not validated. To assess the severity of pulmonary regurgitation jet width is taken into account which is to be compare with RVOT diameter. > 65% is the cut-off value for the Jet width / RVOT diameter ratio for diagnosing severe PR.

Vena Contracta

More accurate method for the assessment of severity of PR is vena contracta width and it is better predictor than of jet width. The 3D vena contracta width gives more accurate values in the quantitative assessment of PR.

Figure: Assessment of PR using Vena Contracta



The Volume of regurgitation can be accurately obtained by multiplying 3D vena contracta width with that of TVI of PR jet.

Pressure half-time:

In the presence of PR, the pressure half-time (time it takes for the pressure gradient between PA and right ventricular to decrease by 50%) is a useful indicator for assessing the RV end diastolic pressure and hemodynamic changes. PR can be graded by using the PHT as follows.

Mild PR > 100 msec

Moderate PR <100 msec.

Severe PR <100 msec

Grading of Severity of PR [44]

Parameters		**Mild**	**Moderate**	**Severe**
Qualitative	Pulmonary Valve Morphology	Normal	Normal / Abnormal	Abnormal
	Colour Flow PR jet width	Small usually <10 mm in length with a narrow origin	Intermediate	Large with a wide origin, May be brief in duration
	CW signal of PR jet	Faint/ Slow deceleration	Dense / Variable	Dense / Steep deceleration, Early termination of diastolic flow
Semi quantitative	VC width (mm)	Not defined	Not defined	Not defined
Quantitative	EROA (mm2)	Not defined	Not defined	Not defined
	Regurgitant Volume (ml)	< 15	15 - 115	> 115
+ RV Size				

Myocardial Infarction

Myocardial infarction is due to the sudden total occlusion of the coronary artery due to rupture of the atherosclerotic plaque with superimposed thrombus

formation. Myocardial infarction usually involves the anterior or the inferior wall of the left ventricle. Right ventricular infarction usually accompanies infero posterior infarction of the left ventricle. According to Kinch et al, right ventricular infarction or ischemia accompanies acute infero posterior myocardial infarction in up to 50% of patients and in 10% of anterior wall myocardial infarction [(46)].

Right ventricular infarction has gained more importance in recent years because of the associated complications like bradycardia, supraventricular arrhythmia, conduction block, hypotension and cardiogenic shock. Involvement of the right ventricle is an important predictor of complications and mortality [(47)].

RV infarction

Right ventricular is most commonly associated with inferior wall infarction of the left ventricle. ECG criteria for the diagnosis of ST segment elevation myocardial infarction is 1 mm ST elevation at the J point in two contiguous leads other than V2 and V3, where 2 mm is required in leads V2 and V3 for patients older than 40 years and 2.5 mm for patients younger than 40 years and less than 1.5 mm for women. Contiguous leads refer to group of leads such as anterior leads (V1–V6), inferior leads (II, III, aVF) or lateral/apical

leads (I, aVL). Supplemental leads such as V3R and V4R reflect the free wall of the right ventricle and V7–V9 the infero-basal wall.

Right Ventricular infarction has been described in many of autopsy studies done in the past. The initial description of the right ventricular failure in cases of right ventricular myocardial infarction had come from Cohn et al as early as 1974. Since that time right ventricular infarctions has been recognized more and remain to be a diagnostic and therapeutic challenge. The increase in immediate mortality and morbidity make the recognition of right ventricular myocardial infarction as an important clinical entity. The presence of right ventricular myocardial involvement in patients with inferior wall myocardial infarction makes them a special subgroup of patients who require early reperfusion.

Inferior wall myocardial infarction with right ventricular infarction is usually caused by acute occlusion of proximal part of right coronary artery proximal to the origin of right ventricular branch. But all patients who have occlusion of proximal right coronary artery proximal to the right ventricular branch do not develop significant right ventricular myocardial necrosis. The reasons attributed for this phenomenon may be one of the following (1) Right ventricle has thinner wall and hence much smaller muscle mass than that of left

ventricle and hence the right ventricular myocardial oxygen demand is less compared to that of left ventricle. (2) The coronary perfusion for the right ventricle occurs in both parts of cardiac cycle namely systole and diastole, whereas the coronary perfusion for left ventricle occurs mainly during diastolic phase of cardiac cycle. (3) There is more extensive collateral circulation for the left side of the heart to the right side. Extrapolating this, the presence of right ventricular hypertrophy may predispose to right ventricular infarction in these patients when they develop coronary artery disease.

The incidence of right ventricular infarction varies with various studies and also depends upon the criteria applied to the detection. In earlier times, the autopsy studies confirmed that the incidence of right ventricular infarction was 24-34% in patients with left ventricular infarction. Non invasive studies suggest that right ventricular infarction occurs in about 30% of patients with acute infero-posterior wall myocardial infarction. Hemodynamic pattern of right ventricular MI is somewhat less expected than anatomic evidence of right ventricular myocardial infarction.

Hemodynamic changes in RVMI

Ischemia or infarction of right ventricle causes compliance of right ventricle to decrease, decreased filling and reduced right ventricular stroke

volume. . Furthermore, when the right ventricular dysfunction is severe, it shifts the interventricular septum leftward, which narrows the left ventricular cavity Combination of these changes finally causes decrease in left ventricular filling and hence decreases cardiac output which causes systemic hypotension and shock. The hemodynamic abnormalities in right ventricular infarction depend upon various factors such as the extent of right ventricular ischemia, right ventricular dysfunction, restraining effects of pericardium, left ventricular function and ventricular interdependence. Right heart filling pressures such as central venous pressure, right atrial pressure and right ventricular end diastolic pressure are elevated, right ventricular systolic pressure and pulse pressure in pulmonary artery are decreased and hence cardiac output is markedly decreased.

The disproportionate increase in the pressure in right atrium compared to that of its left counterpart cause shunting of blood through patent foramen ovale whose direction flow is from right to left. If there is unexplained systemic hypoxemia and cyanosis present in cases of right ventricular myocardial infarction, the physician should have the suspicion of the above possibility of right to left shunting of blood at atrial level.

Another explanation for the persistent hypotension in cases of right ventricular myocardial infarction is due to the presence of abnormally high levels of atrial natriuretic peptide which are circulating in the blood stream causing hypotension.

Echocardiography in RV infarction

Right Ventricular infarction can be diagnosed by echocardiogram at the bedside. Two dimensional echocardiogram in patients with inferior wall myocardial infarction gives clue about right ventricular involvement. Right ventricular hypokinesia or akinesia or global dysfunction is an important finding in cases of RVMI. Also there may be presence of Right ventricular dilatation, tricuspid regurgitation, reduced TAPSE and the presence of dilated inferior vena cava.

MATERIALS AND METHODS

Study Design

The present study was a prospective study conducted in the Department of Cardiology, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai for a period of three months starting from January 2014. Informed written consent was obtained from all patients prior to the start of the study. Institutional ethics committee approval was obtained.

Study Population

112 Consecutive Patients admitted with acute inferior wall ST elevation myocardial infarction in the coronary care unit are included as study population for 3 months from January 2014. Among 112 patients, 18 patients were excluded as they did not fulfill the criteria to be included.

Inclusion Criteria

1. Presence of Physiological PR
2. Prolonged Chest Pain > 30 minutes
3. ECG evidence of $\geq$ 1 mm ST elevation in $\geq$ 2 inferior leads (II, III, aVF)
4. Positive CPK-MB or Troponin- T Test

5. Sinus Rhythm at the time of Echocardiography

Exclusion Criteria

1. Severe PR or No PR
2. Pulmonary Hypertension
3. Not willing for angiography
4. Allergic to contrast dye

METHODS

All the patients underwent a detailed history taking, physical examination, electrocardiogram and biochemical investigations were. Patients who were eligible for reperfusion were treated with streptokinase.

Echocardiographic Examination

Two dimensional and Doppler echocardiographic examination of the patients was done with Esaote MyLab echo machine for all patients.

The probe placed in left parasternal space and shot axis view is obtained. Colour Doppler was applied to find out physiological PR and continuous wave Doppler recordings done across PR jet yielding a positive flow spectrum during normal respiration. The following variables had been measured. Peak velocity

of PR jet (V max), minimum velocity in mid diastole just before the onset of A wave (V min), Pressure half time of Pulmonary regurgitation. The ratio between the maximum and minimum velocities (Vmax/Vmin) was calculated.

The other parameters studied are RV size and dilatation in apical 4 chamber view, RV wall thickness, LV diameter, LV ejection fraction, RV ejection fraction, RV fractional area change, RV tricuspid annulus planar systolic excursion (TAPSE), right ventricular myocardial performance index (MPI), and tricuspid annular peak systolic velocity (s').

The tricuspid valve is interrogated in A4C view and tricuspid regurgitation was recorded and quantified using colour Doppler. IVC diameter was recorded in both inspiration and expiration in subcostal view.

Figure showing PHT 26 msec

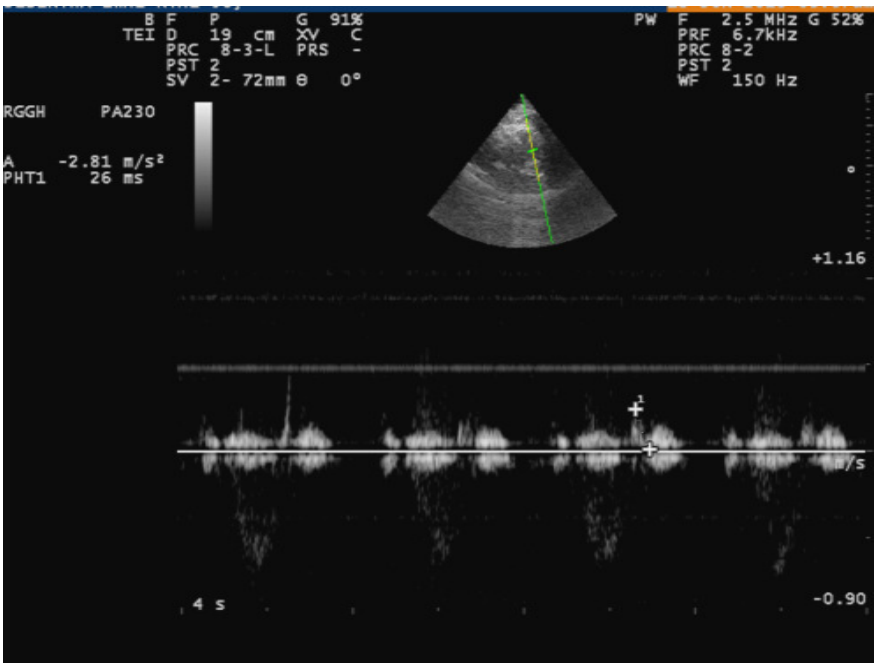
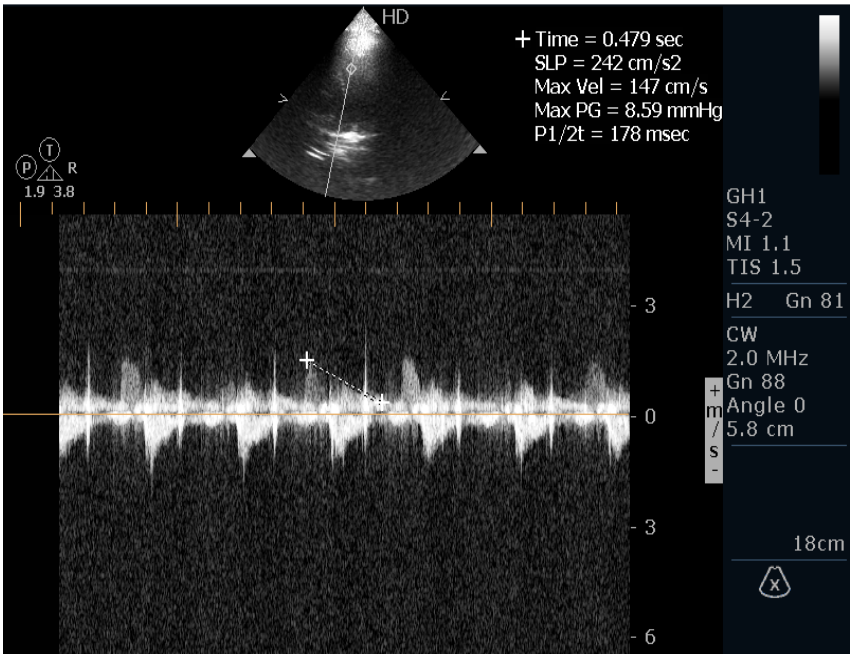


Figure Showing PHT of 178 sec



Electrocardiographic Data

Right precordial leads V4R and Posterior leads were recorded in all patients. RV involvement was suspected in electrocardiogram when there is ST elevation ≥ 1mm seen in V4R and similarly posterior wall MI is suspected when similar magnitude of ST elevation is seen in posterior leads.

Cardiac catheterisation

Coronary angiography is performed in all patients during the period of admission within 7 days to assess the extent of coronary artery lesion. Significant coronary artery disease in a vessel is defined as the presence of

significant (≥ 50%) stenosis on a main branch of the coronary angiogram. Patients are classified as having 1, 2 or 3 vessel disease according to the presence of lesions.

In-hospital events

The prognostic implication of RV involvement as derived by electrocardiographic and echocardiographic criteria, in short term was evaluated for the following events

1) Death
2) Severe arrhythmia (sustained VT, VF)
3) High degree AV block
4) Sinus node dysfunction
5) Need for temporary pacing implantation
6) Low output syndrome (SBP <90 mmHg, Reduced urine output, Need for volume loading, inotropic support)
7) Ischemic events
    a. Anginal pain
    b. Myocardial infarction
    c. Revascularisation (CABG/PCI)

The patient's clinical details and echocardiographic values were entered in a proforma and later tabulated using Microsoft Excel 2007 for statistical analysis.

STATISTICAL ANALYSIS

Patients were grouped according to the Doppler flow characteristics. Pulmonary regurgitation pressure half time (PHT) $\leq$ 150 msec was set as a cutoff value. The patients having PHT $\leq$ 150 msec were classified as Group 1 and those having PHT > 150 msec were classified as Group 2. Variables between these groups were compare using chi-square test or Fisher's exact test. Continuous variables are tabulated as Mean and Standard Deviation. Mann-Whitney U test had been used for the analysis of the Continuous variables as the test is very robust particularly in non- normal or skewed distributions compared to unpaired student t-test. Then Univariate analysis was done to predict in-hospital and 7 day overall events. The statistical analysis was performed by utilising Software Package for social Studies (SPSS) Version 17.0.

RESULTS AND ANALYSIS OF OBSERVED DATA

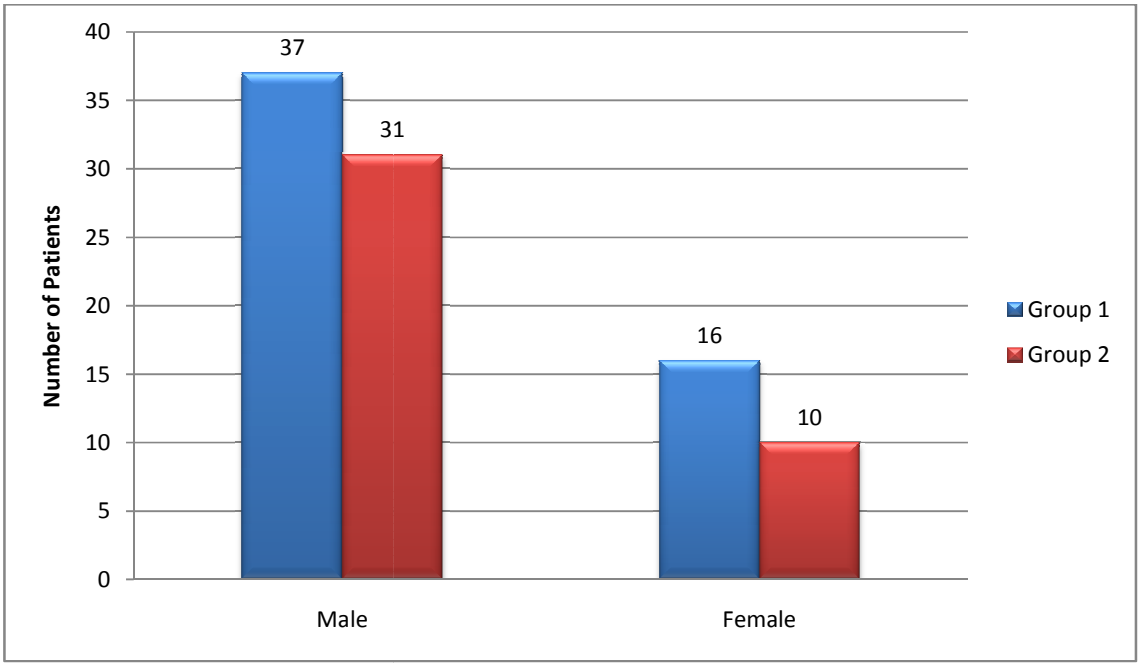
Total number of patients in our study is 94. Among these patients, 2 groups have been divided according to the presence of PR PHT ≤ 150 msec. The first group named "Group 1" has 53 patients who have PHT ≤ 150 ms and second group who have PHT > 150 was named as "Group 2".

Table No. 1 Sex wise distribution of patients

S. No	Group	Male		Female		Total	P Value
		No. of Patients	Percentage	No. of Patients	Percentage		
1	Group 1	37	69.8 %	16	31.2 %	53	**0.533**
2	Group 2	31	75.6 %	10	24.4%	41	
Total		68	72.3 %	26	28.7 %	**94**	

Among Group 1, 69.8 % were males and among Group 2 75.6% were males. There is no statistically significant difference between 2 groups regarding to sex distribution of the patients (P = 0.533). The details of the gender distribution of the patients is tabulated in Table 1 above and depicted in Chart No. 1.

Chart No 1 Gender wise distribution of patients

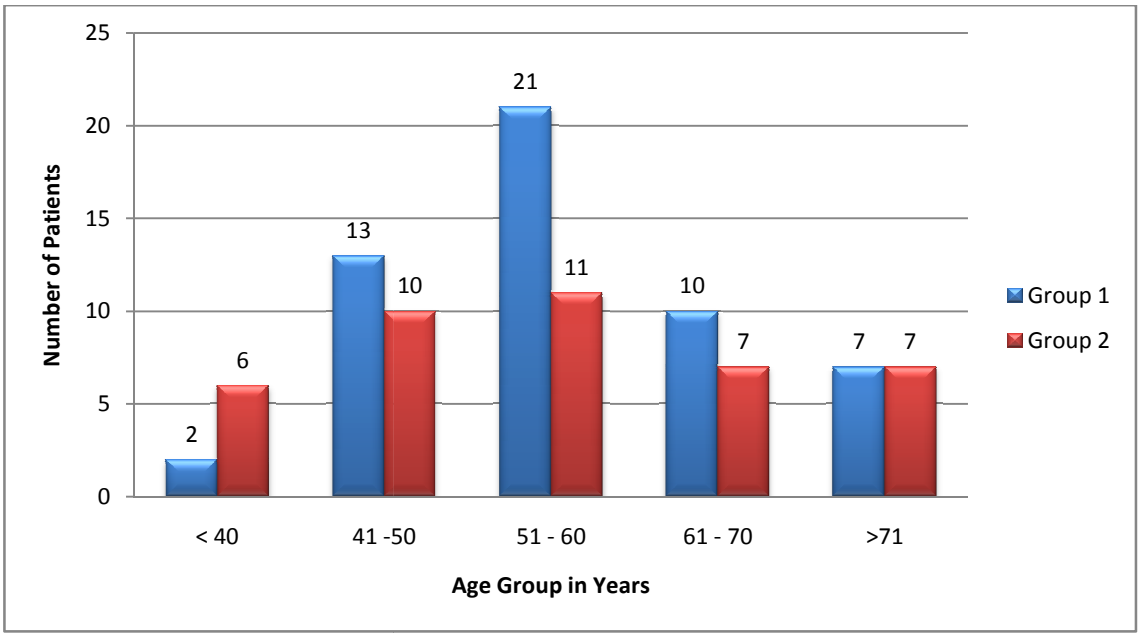


The mean age of patients in Group 1 is about 56.6 years and in Group 2 is 55.5 years. Among total 8 patients who are below the age of 40 years, 2 patients are in Group1. Patients above the age of 75 years have been considered as high risk for in-hospital and follow up events. The differences in age wise distribution of the patients between 2 groups is not significant statistically (p=0.625). The age wise distribution of the patients is shown in Table 2 below and depicted pictorially in Chart 2.

Table No.2 Age Wise Distribution of Patients

S. No	Group	Age Group of Patients in years					Mean ± SD	P Value
		< 40	41-50	51-60	61-70	>70		
1	Group 1	2	13	21	10	7	56.62 ± 10.37	**0.625 mann**
2	Group 2	6	10	11	7	7	55.56 ± 13.16	
Total		8	23	33	17	14		

Chart No 2 Age wise distribution of patients

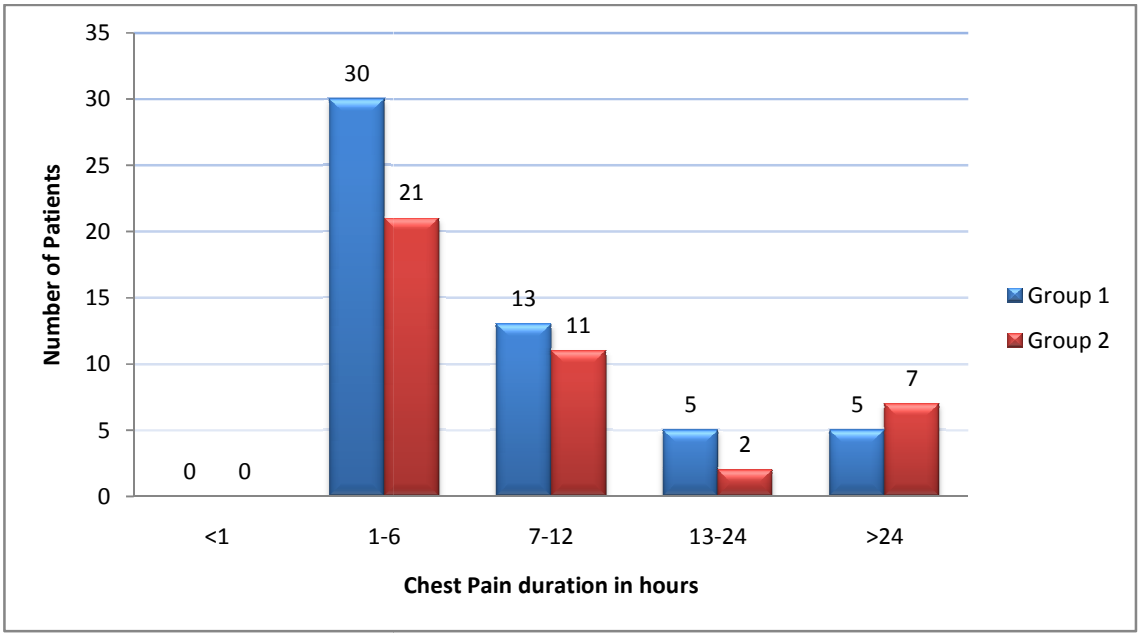


Patients admitted to coronary care unit with various duration of chest pain. No patients had come to CCU with chest pain < 1 hour duration. The minimum duration of chest pain which brought the patient to CCU was 2 hours.

Table No. 3 Chest Pain duration

S. No	Group	Chest pain duration in hours					Mean ± SD	P Value
		< 1	1 – 6	7 – 12	13 – 24	>24		
1	Group 1	0	30	13	5	5	8.73 ± 7.86	0.339
2	Group 2	0	21	11	2	7	12.02 ± 13.49	
Total		0	51	24	7	12		

Chart No. 3 Chest pain duration in hours - distribution



51 Patients were presented within initial 6 hours after onset of chest pain. 12 patients presented more than a day after onset of chest pain. In between groups, the average duration of chest pain in Group 1 is 8.7 hours compared to 12.0 hours in Group 2. Even though there appears to be having a difference

between averages, the difference is statistically not significant (p= 0.339). The results are shown in Table No.3 and depicted in Chart No.3.

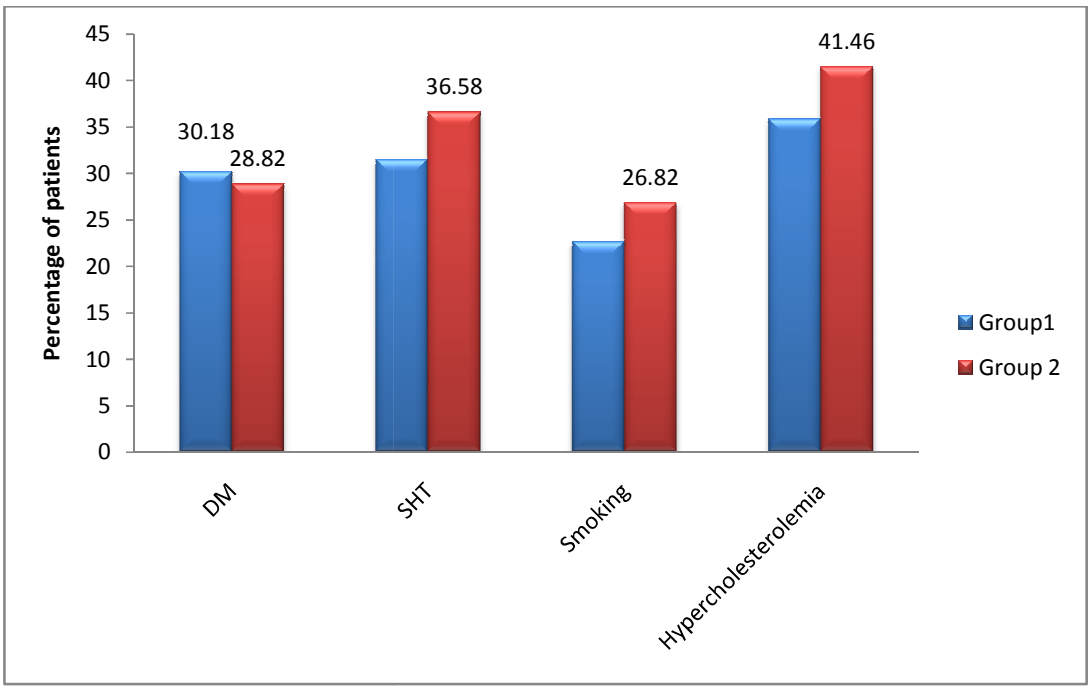
Comparing the risk factors between the two groups, 30.1 % of patients in Group 1 are diabetics and 28.8 % of patients in Group 2 are diabetics. The difference between these groups is statistically not significant. 17 patients in Group 1 who constitute about 31.8 % are having systemic hypertension and in Group 2, 15 patients are hypertensives who constitute about 36.5% of the Group 2 population. 22.6 % of patients in Group 1 are smokers and in Group 2, 26.8 % patients are smokers.

In Group 1, 35.8 % patients are having serum cholesterol level > 200 mg/dl and in Group 2, 41.46 % patients are having serum cholesterol > 200 mg/dl. The differences between individual risk factors between both groups were analysed and all found to be statistically not significant (p > 0.05). The risk factor distribution is depicted in Table 4 below and in Chart No.4.

Table No.4 Risk factors

S. No	Risk Factors	Group 1		Group 2		P Value chi
		No. of Patients	Percentage	No. of Patients	Percentage	
1	Diabetes	16	30.18 %	11	28.82 %	**0.932**
2	SHT	17	31.48 %	15	36.58 %	**0.765**
3	Smoking	12	22.64 %	11	26.82 %	**0.821**
4	Serum Cholesterol > 200 mg/dl	19	35.84 %	17	41.46 %	**0.732**

Chart No. 4 Risk factors comparision

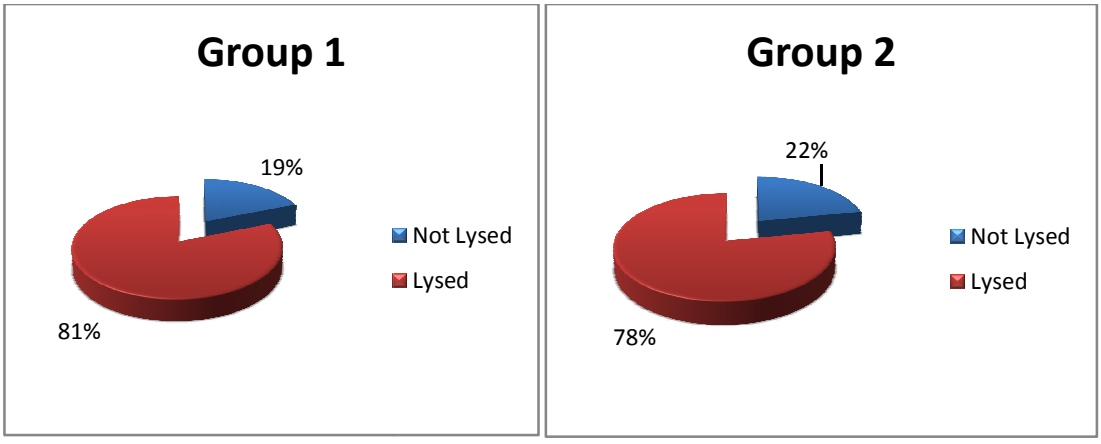


43 patients out of total 53 patients in Group 1 were thrombolysed who constitute about 81.1%. 32 patients out of total 41 patients in Group 2 were thrombolysed constituting about 78%. Total number of patients thrombolysed in our study were 75 constituting about 79.7% of the whole study population. The difference between groups was not statistically significant (p = 0.910). The thrombolysis details are shown in Table 5 and in Pie Chart 5.

Table No. 5 Thrombolysed status

S. No	Group	Thrombolysed		Not thrombolysed		Total	P Value
		No. of Patients	Percentage	No. of Patients	Percentage		
1	Group 1	43	81.1 %	10	18.9 %	53	**0.910 chi**
2	Group 2	32	78.0 %	9	22.0 %	41	
Total		75	79.7 %	19	20.3 %	**94**	

Chart No. 5 Thrombolysed Status

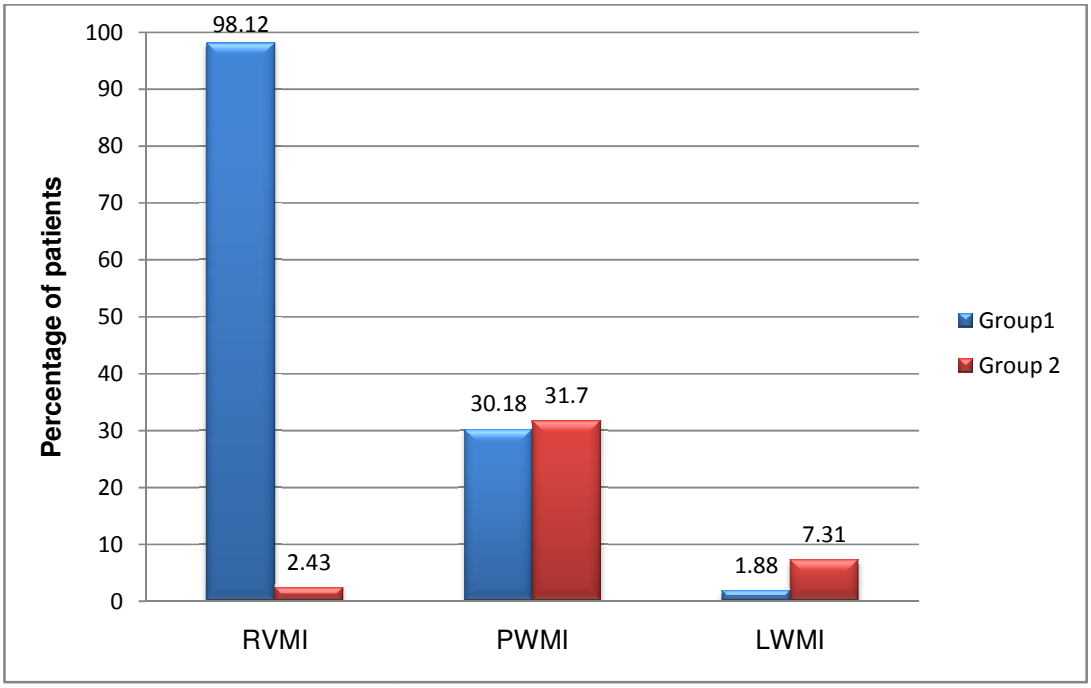


ST segment elevation in electrocardiogram ≥ 1 mm is seen in right sided V4R lead in 98.1 % patients in Group 1. One person in Group 1 does not show ST elevation in V4R. In contrary, only one person in Group 2 has shown significant ST elevation in V4R. This observation of difference between the groups is statistically significant ($p<0.0001$). Posterior wall MI as diagnosed by ST elevation ≥ 1 mm in posterior leads such as V9 is seen in 16 patients in Group 1 and in 13 patients in Group 2 which is statistically not significant. Similarly presence of significant ST elevation in V6 suggesting associated lateral wall involvement is seen in 3 patients in Group 2 and in only one person in Group 1 which is also not significant statistically. The details are shown in Table 6 below and in Chart 6.

Table No. 6 ECG Changes ST Elevation Leads

S. No	Group	V4R		V9		V6	
		No. of Patients	Percentage	No. of Patients	Percentage	No. of Patients	Percentage
1	Group 1	52	98.12 %	16	30.18 %	1	1.88 %
2	Group 2	1	2.43 %	13	31.70 %	3	7.31 %
Total		53	56.38 %	29	30.85 %	4	4.25 %
P Value		< 0.0001		0.946		0.436	

Chart No. 6 Electrocardiographic changes



Mean right ventricular wall thickness is 2.9 mm in Group 1 and 2.9 mm in Group 2 patients. Mean RV dimension was 36.6 mm in Group 1 patients and 25.7 mm in Group 2 patients. The mean left ventricular end diastolic dimension was 36.1 mm in Group 1 whereas 37.9 mm in Group 2 patients. Left ventricular ejection fraction does not significantly differ between both groups. The RV/LV ratio in Group 1 patient has mean of 1.01 whereas in Group 2 is it is 0.68. Mean right ventricular fractional area change in Group 1 is 26.3% and in Group 2 the mean RVFAC is 41.1%. Mean right ventricular ejection fraction in Group 1 patients is 32.4% and in Group 2 is 44.7%. The measurement of TAPSE has a mean of 12.7 mm in Group 1 patients and in Group 2 patients is 19.1 mm. The

tissue Doppler derived value of TASV (S') differ in both groups with mean of 7.9 cm/s in Group 1 and 14.3 cm/s in Group 2.

Table No. 7 Echo Parameters

S. No	Parameters	Units	Group 1	Group 2	P Value Mann
			Mean ± SD	Mean ± SD	
1	RV Wall Thickness	mm	2.90 ± 0.79	2.97 ± 0.87	**0.747**
2	RV Dimension	mm	36.62 ± 5.09	25.78 ± 3.37	**< 0.0001**
3	LVIDd	mm	36.11 ± 5.11	37.90 ± 4.46	**0.096**
4	LV EF	%	54.98 ± 6.16	51.07 ± 12.26	**0.159**
5	RV/LV Ratio	Ratio	1.01 ± 0.07	0.68 ± 0.11	**< 0.0001**
6	RV FAC	%	26.37 ± 5.24	41.18 ± 7.78	**< 0.0001**
7	RV EF	%	32.44 ± 4.40	44.74 ± 6.18	**< 0.0001**
8	TAPSE	mm	12.74 ± 3.26	19.11 ± 3.70	**< 0.0001**
9	TASV (S')	cm/sec	7.91 ± 2.46	14.32 ± 3.61	**< 0.0001**
10	RV MPI (TEI Index)	-	0.49 ± 0.08	0.30 ± 0.11	**< 0.0001**
11	IVC Diameter Inspiration	mm	8.09 ± 2.68	8.65 ± 3.04	**0.316**
12	IVC Diameter Expiration	mm	19.81 ± 4.21	18.09 ± 4.13	**0.031**

The right ventricular myocardial performance index (Tei index) is different in both groups with mean of 0.49 in Group 1 and mean of 0.3 in Group 2. Statistical analysis of all the above parameters shows that the following parameters are statistically significant in the following – RV dimension, RV/LV ratio, RV FAC, RV ejection fraction, TAPSE, MASV and RV Tei index. Others are not statistically significant.

Pulmonary regurgitation Doppler flow characteristics are compared between two groups. Peak PR velocity does not vary significantly between 2 groups. Similarly the end diastolic velocity does not vary between both groups significantly. Mid diastolic minimum velocity has a mean of 0.33 m/sec in Group 1 and 0.69 m/sec in Group2. The difference between the groups is statistically significant with p value < 0.0001. The ratio between Vmax / Vmin differ between the groups. The difference is statistically significant (p value < 0.0001). The mean PHT in Group 1 is 100.1 millisec and in Group 2 is 229 msec. The values are tabulated below in Table 8.

Table No. 8 Echo Parameters – Pulmonary Regurgitation

S. No	Parameters	Unit	Group 1	Group 2	P Value Mann
			Mean ± SD	Mean ± SD	
1	Peak PR Velocity	m/sec	1.41 ± 0.41	1.33 ± 0.28	**0.331**
2	End Diastolic PR Velocity	m/sec	0.72 ± 0.27	0.70 ± 0.27	**0.752**
3	Mid Diastolic Minimum	m/sec	0.45 ± 0.19	0.89 ± 0.31	**< 0.0001**
4	V min/ V max	Ratio	0.33 ± 0.15	0.69 ± 0.31	**<0.0001**
5	PHT of PR	msec	100.1 ± 29.5	229.2 ± 55.5	**< 0.0001**

Tricuspid regurgitation was not present in all cases. TR is present in only in 32.0 % of patients in Group 1 and only 17.0 % in Group 2 patients. The difference between these observations was not statistically significant. (p = 0.156). Regarding severity of TR, severe TR was present in 4 persons in Group 1 accounting for 7.54 % and No patient in group 2 had any severe TR. The difference in presence of severe TR does not achieve statistical significance.

Similarly regarding pulmonary regurgitation severity, moderate PR was present in 3 of the patients of Group 1 and in 1 of the patients of Group 2. There is no statistically significant difference between the two groups regarding the severity of pulmonary regurgitation. The results are displayed in Table No. 9.

Table No. 9 Echo Parameters – Regurgitation severity

S. No	Group		Group 1		Group 2		P Value
			No. of Patients	Percentage	No. of Patients	Percentage	
1	TR Present		17	32.07 %	7	17.07 %	**0.156**
2	TR Severity	Trivial	5	9.43 %	3	7.31 %	**0.994**
		Mild	7	13.20 %	3	7.31 %	**0.561**
		Moderate	1	1.96 %	1	2.43 %	**0.576**
		Severe	4	7.54 %	0	0 %	**0.200**
3	PR Severity	Trivial	20	37.73 %	18	43.91 %	**0.693**
		Mild	30	56.60 %	22	53.66 %	**0.940**
		Moderate	3	5.67 %	1	2.43 %	**0.797**
		Severe	0	-	0	-	-

40 out of the total 94 patients had in-hospital complications in total study population. Percentage of the patients in Group 1 who had in-hospital complications is 54.7 % and in Group 2 is 26.8 %. When the number of overall in-hospital complications in each group was analysed for significant difference, it turned out to be statistically significant ($p = 0.012$). Hypotension was present in 11 patients, reduced urine output was present in 5 patients, and requirement of volume loading was present in 11 patients. 5 patients were in cardiogenic shock and 6 patients were in congestive cardiac failure. Except for oliguria all

the above indices for low volume status have statistically significant difference between two study groups.

Total number of in-hospital deaths in our study is 7. The mortality rate in our study population in the in-hospital set up is 7.44 %. All deaths occurred in Group 1 and Group 2 does not have any in-hospital mortality. The statistical analysis showed that there is significant difference in mortality between two groups with p value 0.043. Heart blocks occurred significantly in Group 1 patients where 7 of them developed this complication while none of the Group 2 patients developed any heart block. This occurrence of heart block in Group 1 patients is statistically significant with a p value of 0.043.

Significant arrhythmias were seen in 6 patients out of whom 4 patients belong to Group 1 and 2 patients belong to Group 2. The difference was statistically not significant. ($p = 0.92$) 9 out of total 94 patients received temporary pacemaker during the course of illness. 8 patients belonged to Group1 and one patient belonged to Group 2. No statistical difference was made out in between these groups regarding TPI placement ($p = 0.08$)

Regarding clinical events, 11 patients developed anginal chest pain during the stay in the hospital accounting for 11.7 % of the study population. There is no statistically significant difference between two groups regarding recurrent angina ($p = 0.84$). The reinfarction rate in the study population was

4.25 %. Three persons form Group 1 and one person from Group 2 has recurrence of MI and the difference is not statistically significant (p = 0.79). Revascularisation was required in 7 patients in whom 4 belonged to Group1. No statistically significant difference was observed in between groups pertaining to revascularisation (p = 0.72). The details of the in-hospital outcome group wise are shown in Table No.9 below.

Table no. 9 In-Hospital Complications

S. No	In-hospital Complications	Group 1		Group 2		Total	P Value
		No. of Patients	Percentage	No. of Patients	Percentage		
1	Overall Complications	29	54.71 %	11	26.82 %	40	**0.012**
2	Hypotension	10	18.86 %	1	2.43 %	11	**0.032**
3	Oliguria	5	9.43 %	0	0 %	5	**0.119**
4	Requirement for volume loading	12	22.64 %	2	4.87 %	15	**0.035**
5	Requirement for inotropes	9	16.98 %	1	2.43 %	11	**0.053**
6	Cardiogenic Shock	5	9.43 %	0	0 %	5	**0.119**
7	CCF	3	5.66 %	3	7.31 %	6	**0.919**
8	Death	7	13.20 %	0	0 %	7	**0.043**
9	Heart Block	7	13.20 %	0	0 %	7	**0.043**
10	Arrhythmia	4	7.54 %	2	4.87 %	6	**0.920**
11	Requirement for TPI	8	15.09 %	1	2.43 %	9	**0.087**
12	Recurrent Angina	7	13.20 %	4	9.75 %	11	**0.847**
13	Recurrent MI	3	5.66 %	1	2.43 %	4	**0.799**
14	Revascularisation	4	7.54 %	3	7.31 %	7	**0.723**

Univariate analysis showed that following variables were associated significantly with total in-hospital events – Age > 65 years (p = 0.049), ST elevation in V4R (p = 0.011), RV dilatation in echocardiography (p = 0.018), Doppler criteria indicating RV involvement such as PR pressure half time ≤ 150 msec (0.018) and combined Vmin / Vmax ≤ 0.5 and PR PHT ≤ 150 msec (p = 0.042). The in-hospital events are also associated significantly with the presence of triple vessel disease in coronary angiogram (p < 0.0001). The odds ratio is highest for the presence of triple vessel disease for the occurrence of in-hospital events. (Relative risk 3.5, CI 1.2 – 9.9, p value <0.0001). Other factors which predict in-hospital events are Age > 65 years, ST elevation in V4R and Doppler flow characteristics of PR. The odds ratios for the variables with confidence intervals are tabulated below in Table No. 10.

Table No. 10 Univariate analysis of in-hospital events

S. No	Parameter	Odds ratio	95% Confidence Interval	P value
1	Age > 65 Yrs	1.6	0.9 – 7.1	0.049
2	Diabetes Mellitus	1.0	0.6 – 1.7	0.925
3	SHT	0.7	0.4 – 1.1	0.229
4	Serum cholesterol > 200 mg/dl	1.0	0.5 – 2.3	0.831
5	Smoking	1.0	0.6 – 1.9	0.792
6	Thrombolysis	0.8	0.4 – 1.6	0.645
7	ST elevation in V4R	1.5	1.1 – 2.1	0.011

8	ST elevation in V9	1.0	0.6 – 1.6	0.988
9	RV dilatation	1.9	1.0 – 3.3	0.018
10	TASV < 10 cm/sec	1.6	0.9 – 2.6	0.060
11	V min/ Vmax ≤ 0.5	1.3	0.7 – 2.2	0.275
12	PR PHT ≤ 150 msec	1.9	1.0 – 3.3	0.018
13	PR PHT ≤ 150 & Vmin/Vmax ≤ 0.5	1.4	1.0 – 2.0	0.042
14	3 vessel CAD	3.5	1.2 – 9.9	< 0.0001

DISCUSSION

The present study is done to evaluate the prognostic implications of Doppler derived parameters of pulmonary regurgitation in cases of acute inferior wall myocardial infarction.

The non invasive diagnostic criteria used in our study, which is based on validated hemodynamic and angiographic criteria, are highly sensitive of Right ventricular ischemia and therefore may be used as an more accurate method of differentiating patients with and without right ventricular involvement.

The Doppler evaluation of PR was done in patients with acute IWMI by Ariel Cohen et al [48] in 1995 which concluded that when the cut off for PR pressure half time was kept below 150 milliseconds and ratio between minimum and maximum velocities V min / V max $\leq$ 0.5, they indicated the presence of associated right ventricular infarction with the sensitivity of 100% and specificity of 89%. The study was done with hemodynamic confirmation of right ventricular MI in cases of IWMI. The same Doppler echocardiographic criteria applied in our study to analyse associated RVMI in IWMI patients and the prognostic impact of the right ventricular involvement.

Hector Bueno et al showed that in patients with RVMI, in-hospital case fatality rate was 47% (compared with 10% in patients without right ventricular involvement (P<0.001). The most common cause of death was non reversible

low cardiac output cardiogenic shock. In our study the most common cause of death was refractory hypotension and ventricular arrhythmias. [49]

In our study, the mortality rate in acute IWMI is about 7.4 % which is in contrast to the study done by Zehender et al where the mortality rate in inferior wall infarction patients was 19% [6]. In the same study, the case fatality rate for right ventricular involvement in acute IWMI was 31% compared to our study which is about 13.2%. The reduction of mortality after RVMI may be attributed to the early recognition of RVMI, better reperfusion strategies and improvement in quality of care in coronary care units. [6]

The presence of ST elevation in V4R is an important clinical variable available at bedside to assess the prognosis of the patient which predict in-hospital mortality with relative risk of 7.7 and major complications with relative risk of 4.7. In our study the presence of ST elevation in V4R has prognostic implications for the major in-hospital events with odds ratio of 1.5 which is statistically significant.

Results of our study are comparable to the study by Ariel Cohen et al, in terms of in-hospital mortality. Study done by Ariel Cohen found that the mortality in cases of IWMI was 6 % which is in our study is 7.4%. But in that study they cannot confirm the poor prognostic outcome of independent ST elevation in V4R which is demonstrated in our study. [50]

Among the risk factors, age > 65 years is the only risk factor which showed statistically significant association with in-hospital events. The other traditional risk factors of cardio vascular disease such as smoking, diabetes mellitus, systemic hypertension and hypercholesterolemia were not able to demonstrate prognostic implications in our study. This may be due to small number of study population and hence any small difference in prognosis may not be able to translate into statistical significance.

Recent studies have found out that the extent of right ventricular myocardial infarction and RV dysfunction as assessed by cine MRI after STEMI, are prognostic indicators, which correlates with our study showing echocardiographic indices of RV dilatation and RV dysfunction such as TAPSE, TASV are independent prognostic factors for early in-hospital events.[51]

CONCLUSIONS

1. In patients with inferior wall acute myocardial infarction, flow Doppler tracings of pulmonary regurgitation are useful in the prediction of in-hospital complications. PR derived parameters (PHT of PR ≤ 150 ms and V min /V max ≤ 0.5) were the excellent predictors of overall in-hospital complications.

2. PHT ≤ 150 ms and the minimal velocity of PR tracings in mid-diastole to the peak early diastolic velocity of PR ratio (V min /V max) ≤ 0.5 were excellent predictors of RV involvement in the setting of inferior wall myocardial infarction.

3. Low output syndrome is a frequent, specific, and potentially severe complication of RV infarction.

LIMITATIONS OF THE STUDY

1. The study population was small, hence it needs to be evaluated whether the results obtained in this study would generalize to other patient groups or not. Clinical trials with larger study populations are needed to assess this.
2. Long term follow up of patients was not done to assess the long term prognosis.
3. The characteristics of infarct related artery such as thrombus burden, lesion morphology, TIMI flow grade etc are not taken into consideration for the study purpose
4. The status of thrombolysis whether successful or failed is not considered in determining the prognosis, which might play a big role in determining in-hospital and long term prognosis.
5. The exclusion of sizeable number of patients who do not have physiological PR which may skew the results.

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PROFORMA

Name :		Age:		Sex:	
Address:					
Diagnosis:					

Clinical Profile

Chest pain duration : Thrombolysis done : Y / N

History : DM: Y / N SHT: Y / N Smoking: Y / N

CPK – MB : Units Trop- T :
ng/ml

Serum Cholesterol : mg/dL Blood Glucose : mg/dL

Blood Urea : mg/dL Serum Creatinine : mg/dL

ECG Parameters

Leads	II	III	aVF	V4R	V 9
ST Segment elevation (mm)					

ECHO Parameters

2D

RV wall thickness

RV dimensions : D1 mm D2 mm D3 mm

LV dimension : Sys mm Dias mm EF %

RV/LV ratio :

RV FAC : %

RV Ejection Fraction : %

M mode

TAPSE (TAM) : mm

TEI index

IVCT : ms

IVRT : ms

Ejection time : ms

Tei Index :

Colour Doppler **PR** **:** Present or Not

If PR present : Mild / Moderate / Severe

Continuous Doppler

PR Peak Velocity : m / sec

End diastolic Velocity: m / sec

Mid diastolic minimum: m / sec

Pressure half time : ms

TR Peak velocity : m / sec

TR PG : mmHg

Right Atrial Pressure Estimation

IVC Diameter Expiratory : mm

Inspiratory : mm

RV Stroke Volume

RVOT VTI : cm

RVOT Diameter : mm

RVOT Area : mm^2

RV Stroke Volume : ml

In hospital Prognosis - Clinical

SBP < 90 mmHg	Yes	No
Oliguria	Yes	No
Need for volume loading	Yes	No
Inotropic support	Yes	No
Shock	Yes	No
CHF	Yes	No
Death	Yes	No
Heart Block	Yes	No
Severe Arrhythmia	Yes	No
SSS / Need for TPI	Yes	No

In hospital Prognosis – Ischemic events

Recurrent Angina	Yes	No
Myocardial Infarction	Yes	No
Revascularisation	Yes	No
Need for hospitalisation	Yes	No

Coronary angiography

CAG done – ? Days after admission			
Number of vessels involved			
IRA - RCA / LCX / LAD / Others			
Percentage of stenosis of IRA			
RCA involvement	Proximal	Mid	Distal

Master Chart

Consent Form

சுய ஒப்புதல் படிவம்

<u>ஆய்வு செய்யப்படும் தலைப்பு</u>

மாரடைப்பு நோயின் பின் விளைவுகளை டாப்ளர் முறையில் முன் கணித்தல் குறித்து ஆராய்ச்சி

<u>ஆராய்ச்சி நிலையம்</u>: இருதய மருத்துவத் துறை,

இராஜீவ் காந்தி அரசு பொது மருத்துவமனை மற்றும் சென்னை மருத்துவக்கல்லூரி,

சென்னை – 600 003.

பங்கு பெறுபவரின் பெயர்: **உறவு முறை:**

பங்கு பெறுபவரின் எண்:

பங்கு பெறுபவர் இதனை (√) குறிக்கவும்

மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களைக் கேட்கவும், அதற்கான தகுந்த விளக்கங்களைப் பெறவும் வாய்ப்பளிக்கப்பட்டது. ☐

நான் இவ்வாய்வில் தன்னிச்சையாகத்தான் பங்கேற்கிறேன். எந்தக் காரணத்தினாலோ எந்தக் கட்டத்திலும் எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகிக் கொள்ளலாம் என்றும் அறிந்து கொண்டேன். ☐

இந்த ஆய்வு சம்மந்தமாகவும், மேலும் இது சார்ந்தஆய்வு மேற்கொள்ளும்போதும், இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளைப் பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்துகொள்கிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன். ☐

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும், பரிசோதனை முடிவுகளையும் மற்றும் சிகிச்சை தொடர்பான தகவல்களையும் மருத்துவர் மேற்கொள்ளும் ஆய்வில் பயன்படுத்திக் கொள்ளவும், அதைப் பிரசுரிக்கவும் என் முழு மனதுடன் சம்மதிக்கிறேன். ☐

இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக்கொள்கிறேன். எனக்குக் கொடுக்கப்பட்ட அறிவுரைகளின் படி நடந்துகொள்வதுடன், இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதியளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ அல்லது எதிர்பாராத வழக்கத்திற்கு மாறாக நோய்க்குறி தென்பட்டாலோ உடனே அதை மருத்துவ அணியிடம் தெரிவிப்பேன் என உறுதி அளிக்கிறேன். ☐

இந்த ஆய்வில் எனக்கு மருத்துவப் பரிசோதனை, இரத்தப் பரிசோதனை மற்றும்இதயஉத்புகுத்துசிகிச்சை பரிசோதனை செய்து கொள்ள நான் முழு மனதுடன் சம்மதிக்கிறேன். ☐

பங்கேற்பவரின் கையொப்பம் ..... இடம் ..... தேதி .....

கட்டைவிரல் ரேகை:

பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....

ஆய்வாளரின் கையொப்பம் ..... இடம் .....தேதி .....

ஆய்வாளரின் பெயர் .....

Information Sheet

ஆராய்ச்சி தகவல் தாள்

சென்னை அரசு பொது மருத்துவமனையில் மாரடைப்பு நோயின் பின் விளைவுகளை டாப்ளர் முறையில் முன் கணித்தல் குறித்து ஆராய்ச்சி செய்ய உள்ளோம்.

நீங்கள் இந்த ஆராய்ச்சியில் பங்கேற்க நாங்கள் விரும்புகிறோம். இந்த ஆராய்ச்சியில் பங்கேற்பதால் தங்களது நோயின் ஆய்வறிக்கையோ அல்லது சிகிச்சையோ பாதிக்கப்படாது என்பதையும் தெரிவித்துக் கொள்கிறோம்.

இந்த ஆராய்ச்சியின் முடிவுகளை அல்லது கருத்துகளை வெளியிடும் போதோ அல்லது ஆராய்ச்சியின் போதோ தங்களது பெயரையோ அல்லது அடையாளங்களையோ வெளியிடமாட்டோம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

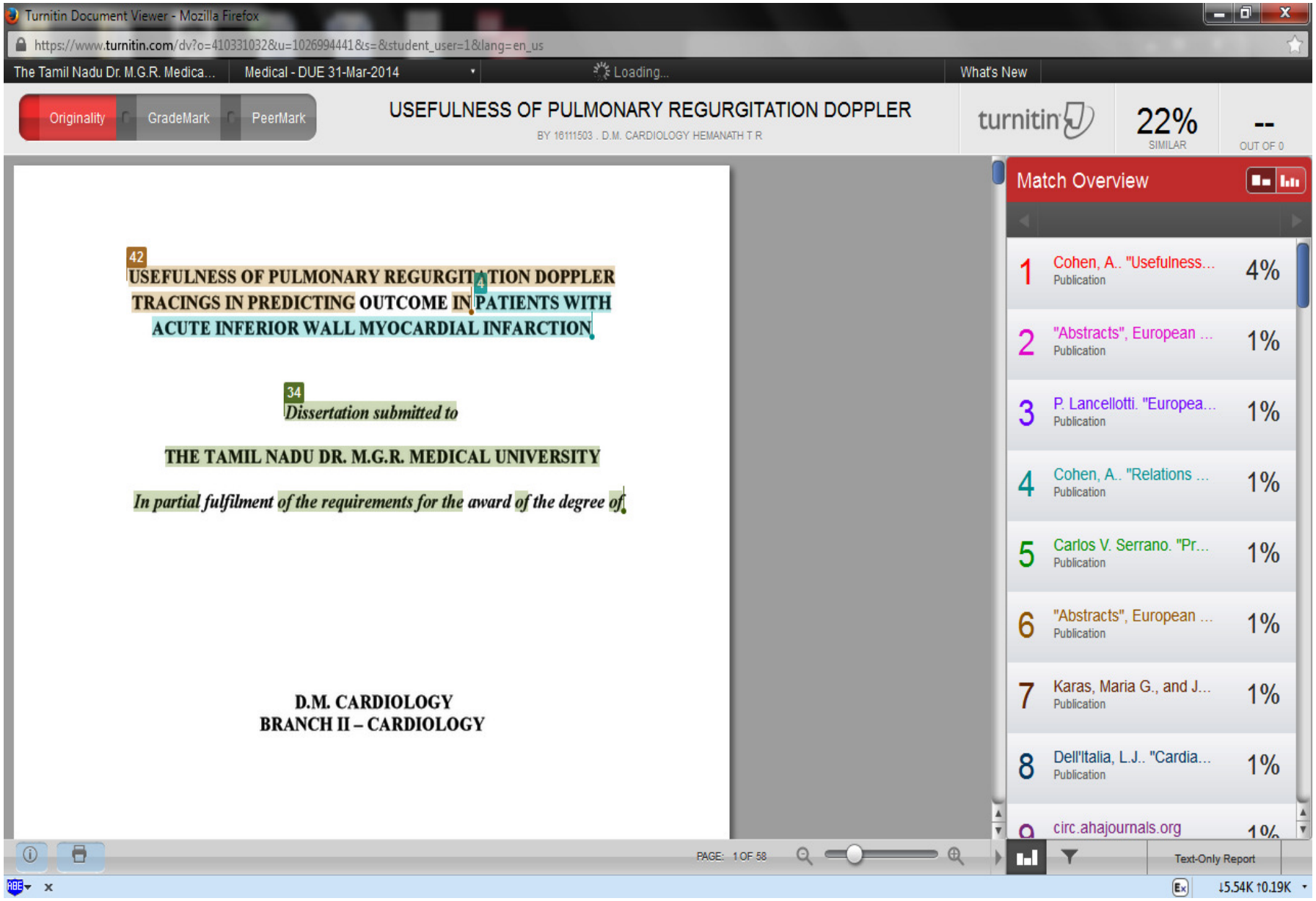
இந்த ஆராய்ச்சியில் பங்கேற்பது தங்களுடைய விருப்பத்தின் பேரில் தான் இருக்கிறது. மேலும் நீங்கள் எந்நேரமும் இந்த ஆராய்ச்சியில் இருந்து பின்வாங்கலாம் என்பதையும் தெரிவித்துக்கொள்கிறோம்.

இந்த சிறப்புப் பரிசோதனைகளின் முடிவுகளை ஆராய்ச்சியின் போதோ அல்லது ஆராய்ச்சியின் முடிவின் போதோ தங்களுக்கு அறிவிப்போம் என்பதையும் தெரிவித்துக்கோள்கிறோம்.

ஆராய்ச்சியாளர் கையொப்பம் பங்கேற்பாளர் கையொப்பம்

தேதி:

Ethical Committee Approval Letter



Anti Plagiarism Certificate

S. No	IP No	Age	Sex	Chest Pain duration (Hrs)	DM	SHT	Smoking	Serum Cholesterol	CPK MB	Trop T	SK	ST elevation Leads: II, III, aVF	V6	V4R	V9
1	200	53	F	6	0	0	0	240	1	1	1	1	0	1	0
2	282	59	M	26	0	0	1	154	1	1	0	1	0	1	0
3	492	58	M	48				264	1	1	0	1	0	0	0
4	534	52	M	7			Y	196	1	1	1	1	1	0	0
5	591	35	M	34	1	0	0	209	1	1	0	1	0	1	0
6	950	72	M	15	0	0	1	191	1	1	0	1	0	1	1
7	1099	53	M	25	0	0	0	163	1	1	0	1	0	1	0
8	1092	72	F	5	1	1	0	134	1	1	1	1	0	1	1
9	1197	65	F	10	0	0	0	325	1	1	1	1	0	1	0
10	1336	77	M	14				80	1	1	0	1	0	0	1
11	2512	70	F	18	1	1	0	122	1	1	0	1	0	1	1
12	2641	72	F	15	0	0	0	100	1	1	0	1	0	1	0
13	3646	51	M	2	0	0	1	159	1	1	1	1	0	1	0
14	3880	55	M	4	0	0	1	212	1	1	1	1	0	1	0
14	4354	55	M	7	0	0	0	96	1	1	1	1	0	1	0
15	4547	60	M	6	1	0	0	272	1	1	1	1	0	1	0
16	5458	42	M	35		Y		211	1	1	0	1	0	0	0
17	5657	68	F	6	0	0	0	181	1	1	1	1	0	1	0
18	5624	48	M	28	1	0	1	172	1	1	0	1	0	1	0
19	6542	65	F	5	0	0	0	196	1	1	1	1	0	1	0
20	6550	60	M	5				120	1	1	1	1	0	0	1
21	6969	58	M	9	Y		Y	200	1	1	1	1	0	0	1
22	7034	67	M	22	0	0	0	157	1	1	0	1	0	1	0
23	7241	74	M	5	1	1	0	119	1	1	1	1	0	1	0
24	7287	50	M	3	0	0	1	126	1	1	1	1	0	1	0
25	7403	59	M	8				225	1	1	1	1	0	0	0
26	7495	52	M	29	Y		Y	247	1	1	0	1	0	0	1
27	7486	51	M	8	0	1	0	151	1	1	1	1	0	1	0
28	8046	54	M	3		Y		121	1	1	1	1	0	0	0
29	8285	78	F	39	Y	Y		189	1	1	0	1	0	0	0
30	8335	45	M	5	Y			203	1	1	1	1	0	0	0
31	8576	65	M	7	0	0	1	267	1	1	1	1	0	1	0
32	8699	62	M	8		Y	Y	111	1	1	1	1	0	0	0
33	8792	50	M	6				151	1	1	1	1	0	0	0
34	8791	47	M	7				222	1	1	1	1	0	0	0
35	9653	60	F	4	1	1	0	191	1	1	1	1	0	1	1
36	9701	60	M	8	1	1	1	187	1	1	1	1	0	1	1
37	10048	63	M	50				235	1	1	0	1	0	0	0
38	10134	60	F	9	0	0	0	173	1	1	1	1	0	1	0
39	10460	55	F	5	1	0	0	221	1	1	1	1	0	1	0
40	11218	65	F	6				221	1	1	1	1	0	0	0
41	11629	36	M	1	0	0	0	200	1	1	1	1	0	1	0
42	12057	37	M	3	Y		Y	248	1	1	1	1	0	0	1
43	12482	38	M	3				195	1	1	1	1	0	0	0
44	12912	52	F	4	0	0	0	195	1	1	1	1	0	1	0
45	13200	48	M	6				156	1	1	1	1	0	0	1
46	13401	60	M	26	1	1	1	178	1	1	0	1	0	1	0
47	13587	45	M	24	0	0	0	260	1	1	0	1	0	1	0
48	14805	52	M	9				170	1	1	1	1	0	0	0
49	15181	73	M	6		Y	Y	108	1	1	1	1	0	0	0
50	15226	75	M	4	Y	Y		224	1	1	1	1	0	0	1

S. No	ECHO Parameters – 2D: RV wall thickness	RV dimension	LVIDd	LV EF	LV/RV ratio	RV FAC	RV EF	TAPSE	S'	TEI index	PR: Severity	Peak	End diastolic	Mid diastolic minimum	Vmin/Vmax	PHT	TR	TR Severity	TRPG
1	3	38	32	62	1.19	27	33	17	5.7	0.5	L	1.59	0.47	0.46	0.29	104	0		
2	3	24	25	56	0.96	35	36	10	12.4	0.6	L	0.92	1.07	0.54	0.59	124	0		
3	3	25	42	56	0.60	39	38	19	10.9	0.2	T	1.60	0.74	1.39	0.87	239	Y	T	26
4	3	26	38	57	0.68	48	51	25	18.0	0.4	T	0.75	0.38	0.49	0.66	273	N		
5	2	36	29	57	1.24	31	32	16	4.7	0.5	L	1.15	1.18	0.54	0.47	145	0		
6	3	40	41	54	0.98	33	31	17	11.3	0.3	L	1.95	1.09	0.93	0.48	83	1	L	30
7	4	34	36	59	0.94	25	33	11	10.1	0.5	L	0.61	0.47	0.10	0.16	133	0		
8	1	35	34	48	1.03	37	34	9	9.7	0.6	L	1.74	0.81	0.30	0.18	100	0		
9	3	29	30	54	0.97	24	40	13	8.0	0.6	L	1.80	0.71	0.34	0.19	99	1	L	28
10	2	25	38	43	0.66	38	51	17	12.8	0.2	T	1.24	0.63	0.90	0.73	163	N		
11	3	32	29	53	1.10	31	26	13	11.2	0.4	L	1.17	0.60	0.31	0.27	89	0		
12	3	34	30	30	1.13	32	36	13	7.2	0.5	T	1.74	1.05	0.44	0.25	91	1	T	26
13	3	35	38	57	0.92	24	27	10	6.8	0.5	T	0.91	0.74	0.41	0.45	126	0		
14	3	34	33	59	1.03	23	34	9	9.8	0.5	T	1.73	0.48	0.60	0.35	108	0		
14	3	45	45	54	1.00	25	30	13	3.5	0.5	L	0.95	0.91	0.46	0.49	123	0		
15	2	42	41	53	1.02	27	33	15	6.7	0.4	O	1.77	0.78	0.45	0.26	106	1	S	
16	4	24	32	57	0.75	44	46	18	17.4	0.4	L	1.50	0.84	0.86	0.57	256	N		
17	3	37	40	53	0.93	22	26	11	5.6	0.6	L	0.61	0.77	0.36	0.58	42	0		
18	3	30	32	58	0.94	26	31	13	6.5	0.5	O	1.40	0.82	0.14	0.10	115	1	S	35
19	4	39	42	52	0.93	20	38	13	7.9	0.5	T	1.27	0.74	0.58	0.46	149	1	L	32
20	3	25	34	49	0.74	48	48	24	14.7	0.3	T	1.54	1.04	0.87	0.57	238	N		
21	3	24	32	50	0.75	48	43	20	8.9	0.4	T	1.83	0.56	1.55	0.85	221	N		
22	2	35	30	50	1.17	26	32	18	7.7	0.4	L	1.67	0.70	0.46	0.28	117	0		
23	4	26	29	53	0.90	28	44	6	5.9	0.4	T	1.19	0.18	0.54	0.46	102	0		
24	4	29	30	57	0.97	23	20	12	6.4	0.5	L	1.79	0.65	0.59	0.33	96	1	L	18
25	2	27	41	59	0.66	44	44	17	7.1	0.3	L	1.96	1.13	0.98	0.50	321	N		
26	2	23	42	42	0.55	34	53	16	17.0	0.2	L	1.03	0.63	1.20	1.16	252	N		
27	5	30	32	53	0.94	22	38	16	3.5	0.4	T	1.27	0.38	0.48	0.38	59	0		
28	3	23	40	65	0.58	45	36	19	21.1	0.3	L	1.18	0.26	0.58	0.49	257	N		
29	3	27	43	61	0.63	45	43	27	15.2	0.1	T	1.08	0.95	0.42	0.38	165	N		
30	4	28	45	62	0.62	40	34	18	15.2	0.3	L	1.35	0.45	0.58	0.43	264	N		
31	3	36	32	59	1.13	21	32	17	4.1	0.6	L	0.68	1.07	0.46	0.67	121	0		
32	3	26	42	58	0.62	51	48	17	14.7	0.2	T	0.98	0.18	0.70	0.72	159	N		
33	2	25	40	57	0.63	24	45	18	10.4	0.1	L	1.46	1.17	0.70	0.48	243	Y	L	33
34	3	23	31	59	0.74	44	55	23	14.9	0.2	L	1.47	0.73	1.24	0.84	179	N		
35	2	35	38	49	0.92	36	27	10	9.0	0.4	T	1.22	0.67	0.42	0.35	94	0		
36	2	37	38	43	0.97	36	30	13	10.1	0.4	T	1.05	0.39	0.46	0.44	101	0		
37	4	27	31	59	0.87	45	46	18	14.7	0.3	L	1.44	1.14	0.57	0.39	197	N		
38	3	30	32	51	0.94	21	38	8	5.8	0.5	T	1.16	1.00	0.46	0.40	143	0		
39	4	28	30	57	0.93	31	38	16	10.5	0.4	L	1.06	0.67	0.23	0.22	137	1	S	45
40	2	19	40	62	0.48	35	50	21	14.9	0.3	L	1.30	0.70	0.91	0.70	291	N		
41	2	37	34	59	1.09	22	36	11	8.9	0.5	T	2.34	0.72	0.48	0.21	107	1	T	16
42	4	28	33	51	0.85	44	39	16	19.0	0.5	L	1.70	0.57	0.90	0.53	229	N		
43	3	30	37	54	0.81	41	40	16	20.4	0.2	T	1.38	0.18	0.58	0.42	176	N		
44	3	30	29	59	1.03	30	27	14	7.2	0.5	O	1.35	1.23	0.30	0.22	57	0		
45	1	31	45	42	0.69	38	44	14	8.2	0.2	T	1.33	1.09	1.02	0.77	288	Y	T	24
46	3	36	38	60	0.95	28	35	10	8.3	0.6	T	1.84	0.50	0.46	0.25	56	1	T	34
47	4	39	38	61	1.03	24	35	19	5.6	0.5	L	2.25	0.55	0.72	0.32	145	0		
48	3	27	42	53	0.64	32	50	18	16.2	0.4	T	1.12	0.84	0.90	0.80	265	N		
49	3	25	40	53	0.63	27	41	22	16.4	0.5	L	1.53	0.69	1.42	0.93	156	N		
50	4	21	31	41	0.68	37	44	18	14.8	0.4	L	1.63	0.87	0.55	0.34	233	N		

S. No	IVC: Ins	Exp	In-Hospital Prognosis – Clinical: SBP<90	Oliguria	Volume loading	Inotropes	Shock	CHF	Death	Heart Block	Arrhythmia	SSS/TPI	Ischemic: Recurrent angina	MI	Revascularisation	CAG Vessels
1	10	21	0	0	0	0	0	0	0	0	0	0	0	0	0	2
2	8	20	0	0	0	0	0	0	0	0	0	0	0	0	0	1
3	6	13	0	0	0	0	0	0	0	0	0	0	0	0	0	3
4	6	13	0	0	0	0	0	0	0	0	0	0	0	0	1	1
5	6	15	0	0	0	0	0	0	0	0	0	0	0	0	0	1
6	6	13	0	0	0	0	0	0	1	0	0	0	0	0	0	1
7	7	18	0	0	0	0	0	0	0	0	0	0	1	0	0	3
8	5	10	0	0	0	0	0	0	0	0	1	0	0	0	0	3
9	12	27	0	0	0	0	0	0	0	0	0	0	0	0	0	2
10	6	16	0	0	0	0	0	0	0	0	0	0	0	0	0	2
11	7	17	0	0	0	0	0	0	0	0	0	0	0	0	0	3
12	11	22	0	0	1	1	0	0	0	0	0	0	0	0	0	1
13	6	16	0	0	0	0	0	0	0	0	0	0	0	0	0	1
14	9	19	0	0	0	0	0	0	0	0	0	0	1	0	0	3
14	8	21	0	0	0	0	0	0	0	0	0	0	0	0	0	1
15	6	22	1	0	1	1	0	1	0	0	0	0	0	0	0	3
16	3	10	0	0	0	0	0	0	0	0	0	0	0	0	0	1
17	5	18	0	0	0	0	0	0	0	0	0	0	1	0	0	3
18	9	22	0	0	0	0	0	0	0	0	0	0	0	0	0	1
19	8	20	0	0	0	0	0	1	0	0	0	0	0	0	0	2
20	10	24	0	0	0	0	0	0	0	0	0	0	0	0	0	1
21	9	19	0	0	0	0	0	0	0	0	0	0	0	0	0	2
22	8	23	1	1	1	1	1	0	1	1	0	1	0	0	0	2
23	8	23	0	0	0	0	0	0	0	0	0	0	0	0	0	1
24	7	19	0	1	1	1	1	0	1	1	0	1	0	1	0	1
25	9	19	0	0	0	0	0	0	0	0	0	0	0	0	0	2
26	10	20	0	0	0	0	0	0	0	0	0	0	0	0	0	1
27	7	21	0	0	0	0	0	0	0	0	0	0	0	0	0	1
28	6	15	0	0	0	0	0	0	0	0	1	0	0	0	0	1
29	11	22	0	0	0	0	0	0	0	0	0	0	0	0	0	1
30	15	25	0	0	0	0	0	0	0	0	0	0	0	0	0	2
31	6	18	0	0	0	0	0	0	0	0	0	0	0	0	0	3
32	9	15	0	0	0	0	0	0	0	0	0	0	0	0	0	3
33	8	17	0	0	0	0	0	0	0	0	0	0	0	0	0	1
34	7	17	0	0	0	0	0	1	0	0	0	0	0	0	0	1
35	19	29	1	0	1	0	0	0	0	0	0	0	0	0	0	1
36	6	14	0	0	0	0	0	1	0	0	0	0	0	0	0	2
37	8	24	0	0	0	0	0	0	0	0	0	0	0	0	0	1
38	9	19	0	0	0	0	0	0	0	0	0	0	0	0	0	2
39	3	12	1	1	1	1	1	0	0	1	1	1	0	0	0	1
40	6	29	0	0	0	0	0	0	0	0	0	0	0	0	0	1
41	13	24	1	0	1	0	0	0	0	0	0	0	0	0	0	1
42	8	18	0	0	0	0	0	0	0	0	0	0	0	0	1	1
43	9	17	0	0	0	0	0	0	0	0	0	0	0	0	0	1
44	5	13	0	0	0	0	0	0	0	0	0	0	0	0	1	3
45	7	19	0	0	0	0	0	0	0	0	0	0	0	0	0	1
46	9	24	0	0	0	0	0	0	0	0	0	0	0	0	0	2
47	12	25	1	0	1	0	0	0	1	1	0	1	0	1	1	2
48	10	22	0	0	0	0	0	0	0	0	0	0	0	0	0	1
49	11	21	0	0	0	0	0	0	0	0	0	0	0	0	0	3
50	6	14	0	0	1	0	0	0	0	0	0	0	0	0	0	1

												ST elevation Leads				ECHO Parameters																					In- Hospital Prognosis													
																2D										PR									IVC		Clinical										Ischemic			
S. No	IP No	Age	Sex	Chest Pain duration (Hrs)	DM	SHT	Smoking	Serum Cholesterol	CPK MB	Trop T	SK	II, III, aVF	V6	V4R	V9	RV wall thickness	RV dimension	LVIDd	LV EF	LV/RV ratio	RV FAC	RV EF	TAPSE	S'	TEI index	Severity	Peak	End diastolic	Mid diastolic minimum	Vmin/ Vmax	PHT	TR	TR Severity	TRPG	Ins	Exp	SBP<90	Oliguria	Volume loading	Inotropes	Shock	CHF	Death	Heart Block	Arrhythmia	SSS/TPI	Recurrent angina	MI	Revascularisation	CAG Vessels
51	15260	43	M	3	1	0	0	215	1	1	1	1	0	1	1	3	39	32	46	1.22	24	33	16	10.9	0.5	T	2.15	0.55	0.00	0.00	116	0			8	18	0	0	0	0	0	0	0	0	0	0	0	0	0	2
52	15587	70	F	6		Y		141	1	1	1	1	0	0	0	3	20	38	54	0.53	32	48	18	8.1	0.3	T	0.81	0.61	1.34	1.65	303	Y	L	32	12	25	0	0	0	0	0	0	0	0	0	0	0	0	0	3
53	15616	60	M	6				352	1	1	1	1	0	0	1	2	22	33	2	0.67	47	32	20	10.0	0.4	L	0.77	0.66	1.09	1.41	181	N			12	23	0	0	0	0	0	0	0	0	0	0	0	0	0	1
54	15632	75	M	18	Y	Y		218	1	1	0	1	0	0	0	5	28	38	58	0.74	40	27	24	18.1	0.1	L	1.11	0.87	1.35	1.22	158	N			7	18	0	0	0	0	0	0	0	0	0	0	0	0	0	2
55	15943	42	M	2	0	0	0	250	1	1	1	1	0	1	0	2	40	41	60	0.98	28	30	15	5.0	0.4	L	0.78	0.91	0.41	0.53	95	0			8	19	0	0	0	0	0	0	0	0	0	0	0	0	0	2
56	16429	60	M	4	0	1	0	213	1	1	1	1	0	1	0	3	41	40	62	1.03	23	34	10	7.2	0.5	T	1.37	0.62	0.26	0.19	55	0			9	23	0	0	0	0	0	0	0	0	0	0	1	0	0	3
57	16892	55	M	2	0	0	0	277	1	1	1	1	0	1	0	3	46	45	61	1.02	29	39	8	11.0	0.5	L	1.61	0.97	0.81	0.50	88	0			6	21	0	0	0	0	0	0	0	0	0	0	0	0	0	1
58	17358	47	M	9	0	0	0	179	1	1	1	1	0	1	1	2	43	42	49	1.02	23	27	19	9.3	0.7	T	1.35	0.93	0.32	0.24	83	0			6	13	0	0	0	0	0	0	0	0	0	0	0	0	0	2
59	19023	48	M	36				103	1	1	0	1	0	0	0	3	26	35	50	0.74	30	44	16	12.2	0.5	L	1.43	0.82	0.36	0.25	277	N			9	17	0	0	0	0	0	1	0	0	1	0	0	0	0	1
60	20183	32	M	43		Y		172	1	1	0	1	1	0	0	2	24	39	60	0.62	56	51	19	16.2	0.3	T	1.38	0.37	0.74	0.54	193	N			10	15	0	0	0	0	0	0	0	0	0	0	1	0	0	1
61	20230	33	F	3				204	1	1	1	1	0	0	1	3	37	35	48	1.06	31	50	23	19.1	0.5	L	1.40	0.73	0.87	0.62	238	Y	L	28	8	16	0	0	0	0	0	0	0	0	0	0	0	0	0	2
62	20228	65	F	9	Y			169	1	1	1	1	0	0	1	2	28	45	46	0.62	39	47	11	14.5	0.1	L	1.39	0.32	0.70	0.50	248	N			11	23	0	0	0	0	0	1	0	0	0	0	1	0	0	1
63	20906	47	F	7		Y		136	1	1	1	1	0	0	1	4	29	41	46	0.71	36	42	10	20.7	0.4	L	1.12	1.13	1.09	0.97	201	N			6	14	0	0	0	0	0	0	0	0	0	0	0	0	0	3
64	21098	61	F	8				210	1	1	1	1	1	0	1	3	30	37	45	0.81	45	43	16	15.1	0.4	L	1.48	0.50	1.14	0.77	178	N			8	18	0	0	0	0	0	0	0	0	0	0	0	0	0	1
65	21488	47	M	1	0	0	0	181	1	1	1	1	0	1	1	4	42	40	50	1.05	22	37	15	7.6	0.4	L	1.02	0.47	0.45	0.44	96	1	L	27	9	19	0	0	0	0	0	0	0	0	0	0	0	0	0	1
66	21581	66	M	3	1	1	0	155	1	1	1	1	0	1	1	3	32	31	51	1.03	23	32	15	8.8	0.5	L	1.72	1.02	0.37	0.22	127	0			6	13	0	0	0	0	0	0	0	0	0	0	1	1	1	3
67	21989	58	F	7	0	1	0	312	1	1	1	1	0	1	1	3	38	35	52	1.09	21	34	8	6.5	0.5	L	1.85	0.65	0.10	0.05	26	0			8	16	0	0	0	0	0	0	1	0	1	0	1	0	0	2
68	22722	45	M	4	0	0	0	191	1	1	1	1	0	1	0	3	34	33	62	1.03	26	29	9	8.4	0.4	L	1.30	0.85	0.14	0.11	73	1	0	26	5	21	0	0	0	0	0	0	1	1	0	1	0	0	0	2
69	22828	62	F	8	0	1	0	240	1	1	1	1	0	1	1	4	40	39	53	1.03	23	27	8	10.5	0.3	L	1.48	0.12	0.74	0.50	118	1	S	32	7	18	0	0	0	0	0	0	0	0	0	0	0	0	0	1
70	23090	52	M	4		Y	Y	174	1	1	1	1	0	0	0	3	28	37	57	0.76	27	48	16	18.6	0.3	O	1.73	0.21	0.95	0.55	157	Y	O	28	20	21	0	0	0	0	0	0	0	0	0	0	1	0	0	1
71	23230	54	M	3	0	1	0	180	1	1	1	1	0	1	1	2	38	37	55	1.03	27	28	16	10.8	0.4	T	1.59	0.71	0.28	0.18	100	0			8	19	1	1	1	1	1	0	1	1	0	1	0	0	0	1
72	23606	52	M	10	0	0	0	218	1	1	1	1	0	1	0	2	41	40	63	1.03	19	42	17	9.8	0.4	L	1.55	0.41	0.59	0.38	112	0			9	23	0	0	0	0	0	0	0	0	0	0	0	0	0	2
73	23728	80	F	5		Y		154	1	1	1	1	0	0	0	3	21	30	53	0.70	40	54	20	15.4	0.4	L	1.29	1.13	1.44	1.12	219	N			4	10	0	0	0	0	0	0	0	0	0	0	0	0	0	1
74	24774	50	M	5			Y	277	1	1	1	1	0	0	0	4	26	40	53	0.65	43	43	21	9.5	0.3	T	1.62	0.62	0.59	0.37	342	N			9	17	0	0	0	0	0	0	0	0	0	0	0	0	0	1
75	24913	42	F	10	1	0	0	206	1	1	1	1	0	1	0	2	41	42	65	0.98	32	32	10	2.0	0.5	T	0.82	0.61	0.37	0.46	91	0			6	21	0	0	0	0	0	0	0	1	0	1	0	0	0	1
76	25150	72	F	9				151	1	1	1	1	0	0	0	2	30	37	52	0.81	38	44	20	10.2	0.4	T	0.94	0.69	1.09	1.16	163	N			7	14	0	0	0	0	0	0	0	0	0	0	0	0	0	1
77	25438	75	F	11	1	1	0	161	1	1	1	1	0	1	0	3	42	43	62	0.98	30	32	14	6.8	0.5	L	1.70	0.33	0.62	0.36	114	0			10	21	1	1	1	1	1	0	0	0	0	0	0	0	0	1
78	25444	52	M	3	0	0	1	180	1	1	1	1	0	1	1	3	40	42	52	0.95	29	34	11	9.9	0.5	L	1.63	0.55	0.39	0.24	143	0			13	29	1	0	1	1	0	0	0	0	0	0	0	0	0	2
79	26135	72	M	4	1	1	0	155	1	1	1	1	0	1	1	2	49	47	54	1.04	31	33	9	5.6	0.6	T	1.28	0.46	0.65	0.51	117	1	T	28	8	18	0	0	0	0	0	0	0	0	0	0	0	0	0	2
80	26213	65	M	5	0	1	0	176	1	1	1	1	0	1	1	3	39	35	59	1.11	23	33	14	5.9	0.5	T	1.46	0.38	0.93	0.64	87	0			7	18	0	0	0	0	0	0	0	0	0	0	0	0	0	2
81	26268	42	M	6	0	0	0	150	1	1	1	1	0	1	0	3	38	39	67	0.97	29	31	15	5.8	0.6	L	1.29	0.50	0.33	0.25	48	0			9	19	1	0	1	1	0	0	0	0	0	1	0	0	0	2
82	27103	46	F	5	Y	Y		174	1	1	1	1	0	0	0	2	28	32	51	0.88	47	48	26	14.7	0.2	T	1.33	0.96	1.16	0.87	322	N			8	16	0	0	0	0	0	0	0	0	0	0	0	0	0	2
83	27150	47	M	6	0	0	0	206	1	1	1	1	0	1	1	2	37	35	45	1.06	30	32	14	9.2	0.5	T	1.31	0.96	0.38	0.29	105	1	T	29	8	19	0	0	0	0	0	0	0	0	0	0	0	0	0	
84	27205	38	M	3	Y	Y	Y	221	1	1	1	1	0	0	0	3	24	42	9	0.57	41	40	19	12.3	0.4	L	1.12	0.58	0.70	0.63	156	N			7	17	0	0	0	0	0	0	0	0	0	0	0	0	0	1
85	27209	54	M	6	0	0	0	168	1	1	1	1	1	1	0	2	35	37	52	0.95	28	28	14	8.7	0.5	L	2.18	0.82	0.80	0.37	30	0			4	21	0	0	0	0	0	0	0	0	0	0	0	0	1	3
86	27586	48	M	3				287	1	1	1	1	0	0	0	5	25	47	59	0.53	49	39	16	12.0	0.3	T	1.06	0.96	0.80	0.76	162	N			14	20	0	0	0	0	0	0	0	0	0	0	0	0	0	1
87	27103	45	M	5	0	0	1	176	1	1	1	1	0	1	0	2	34	33	55	1.03	27	30	10	8.5	0.5	L	1.60	1.05	0.39	0.25	69	1	L	24	12	30	0	0	0	0	0	0	0	0	0	0	0	0	0	1
88	28609	69	M	2	Y	Y	Y	159	1	1	1	1	0	0	1	4	27	35	39	0.77	46	45	22	14.7	0.4	T	1.68	0.74	0.88	0.53	249	Y	T	32	6	15	0	0	0	0	0	0	0	0	0	1	0	1	1	2
89	28363	52	M	7				181	1	1	1	1	0	0	0	2	23	39	60	0.59	57	43	21	12.8	0.2	L	1.39	0.56	0.65	0.47	347	N			9	17	0	0	0	0	0	0	0	0	0	0	0	0	0	2
90	28402	35	M	4			Y	179	1	1	1	1	0	0	0	3	22	35	62	0.63	55	57	24	10.6	0.2	T	1.39	0.61	0.29	0.21	242	N			8	17	1	0	1	1	0	0	0	0	0	0	1	0	0	1
91	28436	63	M	3	0	0	1	198	1	1	1	1	0	1	0	4	38	37	56	1.03	25	32	11	7.9	0.6	T	1.42	0.96	0.41	0.29	90	0			10	21	0	0	0	0	0	0	0	0	0	0	0	0	0	1
92	28445	50	M	7	1	1	0	224	1	1	1	1	0	1	0	3	43	42	55	1.02	30	29	10	14.3	0.5	L	0.99	1.33	0.63	0.63	117	0			8	23	0	0	0	0	0	0	0	0	0	0	1	0	0	1
93	28876	75	F	3	0	1	0	238	1	1	1	1	0	1	0	4	39	40	58	0.98	6	31	16	9.5	0.4	L	1.57	0.97	0.45	0.29	126	1	L	23	10	22	0	0	0	0	0	0	0	0	0	0	0	0	0	1