EFFECT OF REPERFUSION ON

RIGHT VENTRICULAR FUNCTION IN ACUTE ANTERIOR WALL

ST ELEVATION MYOCARDIAL INFARCTION

Dissertation submitted to
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CERTIFICATE

This is to certify that the dissertation titled "**EFFECT OF REPERFUSION ON RIGHT VENTRICULAR FUNCTION IN ACUTE ANTERIOR WALL ST ELEVATION MYOCARDIAL FUNCTION"** is the bonafide original work of Dr**. K. SIDHARTHAN**, 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 2014.The period of post-graduate study and training was from August 2011 to July 2014.

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DECLARATION

I, **Dr. K. SIDHARTHAN**, solemnly declare that this dissertation entitled, "**EFFECT OF REPERFUSION ON RIGHT VENTRICULAR FUNCTION IN ACUTE ANTERIOR WALL ST ELEVATION MYOCARDIAL FUNCTION**" is a bonafide work done by me at the department of Cardiology, Madras Medical College and Government General Hospital during the period 2011 – 2014 under the guidance and supervision of the Professor and Head of the department of Cardiology of Madras Medical College and Government General Hospital, Professor M. S. Ravi 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: **SIGNATURE OF THE CANDIDATE**

Date:

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CONTENTS

Introduction

INTRODUCTION

Coronary artery disease is one of the leading cause of morbidity and mortality in the world. In 19th century coronary artery disease is more prevalent in the developed countries. Now it is becoming epidemic in developing countries also[1]. There is a five times increased prevalence of coronary artery disease in 1986 when compared to 1976. When compared to other countries in the world, SAARC countries have more prevalence of cardiovascular diseases [2,3,4].

According to the Global burden of Disease Study this region will have more cardiovascular disease than the rest of the world by 2020 [4]. The main reasons for the increased incidence of coronary artery disease in this part of the world are diabetes mellitus, hypertension, smoking, stress, obesity, sedentary life style susceptible genetics and unhealthy diet.

The spectrum of coronary artery disease is from acute coronary syndrome to stable angina. Acute coronary syndrome is the most important cause for morbidity and mortality among coronary artery disease. Acute myocardial infarction is usually due to anterior or inferior wall infarction.

Clinical and hemodynamic features of acute myocardial infarction depends upon the territory of coronary artery involved. Anterior wall ST elevation myocardial infarction has more adverse prognosis because of cardiogenic shock, ventricular tachycardia. Inferoposterior wall myocardial infarction often accompanied by right ventricular infarction.

In recent years in coronary artery disease right ventricular function has receive more importance. Cohn et at was one of the first who described about the hemodynamic and clinical features of acute right ventricular infarction [(5)]. Right ventricular infarction more frequently causes low cardiac output and shock which is an important cause of mortality [(6)].

Right ventricular function usually remains normal in anterior wall myocardial infarction. In recent years many studies have shown right ventricular dysfunction in isolated anterior wall myocardial infarction. In patients with left ventricular dysfunction after myocardial infarction, an important predictor of cardiovascular mortality is right ventricular function [(7)]. Both interventricular septum and right ventricular free wall contribute to the function of the right ventricle and hence right ventricular dysfunction is expected in septal involvement in anterior wall myocardial infarction [(8)].Heightened sympathetic drive caused by acute myocardial

infarction is one of the mechanism which affects right ventricular function [9].

Branches of the left anterior descending coronary artery supply anterior wall of right ventricle [10] and autopsy studies have shown that right ventricular infarction occurs in acute left anterior descending coronary artery occlusion. But this relation has not been studied so far [11, 12].

Echocardiography has helped to study the right ventricular function. Even though right ventricular volume and ejection fraction are not accurately measured by echocardiography, right ventricular function can be determined by two dimensional echo, M mode, pulse doppler and tissue doppler echocardiography [13, 14].

Review of literature

REVIEW OF LITERATURE

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 [15].

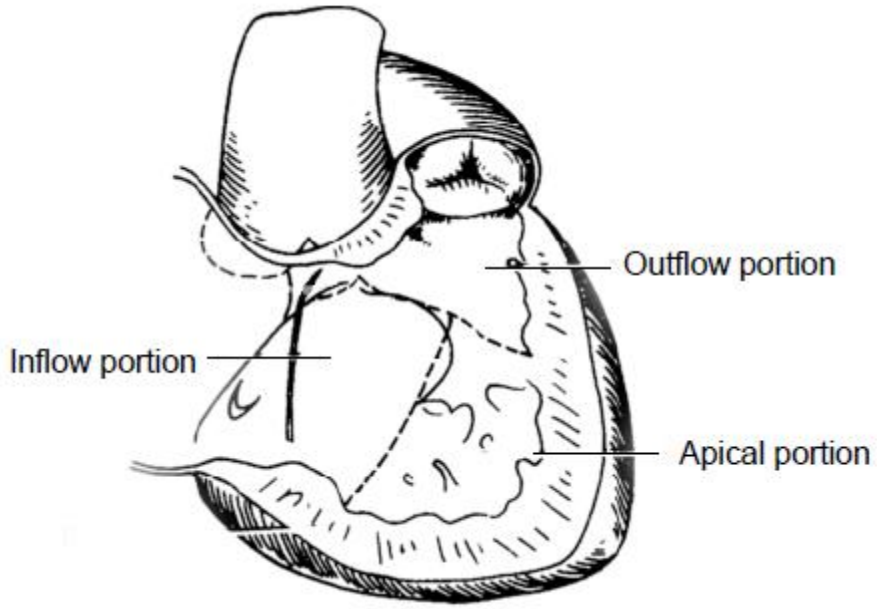
Right ventricle is the anterior most chamber and it is situated behind the sternum. Right ventricle is a crescent shaped chamber while left ventricle is a ellipsoidal chamber.

Right ventricular wall is thin of 3 to 5 mm thickness. Right ventricular wall is made up of circumferential fibers 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 fibers which contribute to the ventricular interdependence.

Right ventricle has three regions namely inlet, trabecualr and outlet segments [16]. 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 [16, 17].

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 [(10)]. 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 [(18)]. 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)].

Fig - 01. Anatomy of the right ventricle



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 trabecualted while that of the left ventricle is not trabecualted. Fourth, the right ventricle has a band of muscle attached from the base of the anterior papillary muscle to the interventricular 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 also 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 shortenening [22]. There is a continuous interplay between the right and left ventricles due to the shared interventricular 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 [10]. 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 a 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 was the first to describe 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 the 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 the inter ventricular septum plays a major role in the contraction of the right ventricle and hence in the right ventricular cardiac output. Interventricular septum and the left ventricle are mainly responsible for about 20 - 50% of the function of the right ventricle.

Definition of Myocardial Infarction

In 2012, Joint ESC/ACCF/AHA/WHF Task Force has given the definition for the myocardial infarction. Accordingly, the diagnosis of myocardial infarction needs any one of the following criteria

Detection of a rise and or fall of cardiac biomarker with at least one value above the 99th percentile and with at least one of the following

Ischemic symptoms.

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

New pathological Q waves in ECG.

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

Angiographic or autopsic identification of intracoronary thrombus.

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 [(23)].

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. Invlovement of the right ventricle is an important predictor of complications and mortality [(24)].

Clinical Features of Right Ventricular dysfunction

Right ventricular dysfunction leads to a reduction in right ventricular compliance, decreased filling and a fall in the stroke volume of the right ventricle. This produces a decreased preload to the left ventricle and finally decreased cardiac output. Furthermore, when the right ventricular dysfunction is severe, it shifts the interventricular septum leftward, which narrows the left ventricular cavity. It increases the end diastolic pressure of the left ventricle and decreases compliance and cardiac output [(25)]. In addition right ventricular dysfunction causes right ventricular dilatation which causes the intra pericardial pressure to rise and thus reduces the ventricular compliance[(26)]. These features of right ventricular dysfunction are more commonly seen in ischemia or

infarction of the right ventricle which usually accompanies the acute infero posterior wall myocardial infarction.

The clinical features of right ventricular dysfunction are hypotension, elevated jugular venous pressure and clear lung fields. Hypotension and cardiogenic shock are important cause of in hospital mortality.

Echocardiographic Evaluation of the Right Ventricle

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 [(27)]. 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.

Myocardial Performance Index

In 1995, Chuwa Tei et al published in the Journal of Cardiology about new non invasive index to measure the global ventricular function [(28)]. This index is known by the author's name Tei index. 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 [(29)] and to study the systolic and diastolic function of the patients with cardiac amyloidosis [(30)].

Myocardial performance index is used to measure the global ventricular function and for the evaluation of both right and left

ventricular function [31]. It combines both the diastolic and systolic performance of the heart. Tei index is derived using pulse wave Doppler echocardiography. Initially it was calculated from sequential pulse wave Doppler recording of both ventricular inflow and outflow. But now it is also calculated from tissue Doppler recording from lateral mitral annulus [32]. The advantage of recording from the lateral mitral annulus is that errors due to changes in the heart rate can be avoided. Similarly, Tei index calculated using tissue Doppler imaging of the tricuspid annulus also correlated with that calculated with pulse wave Doppler of the right ventricular inflow and outflow tracts [33].

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.

Right ventricular myocardial performance index is calculated with the formula

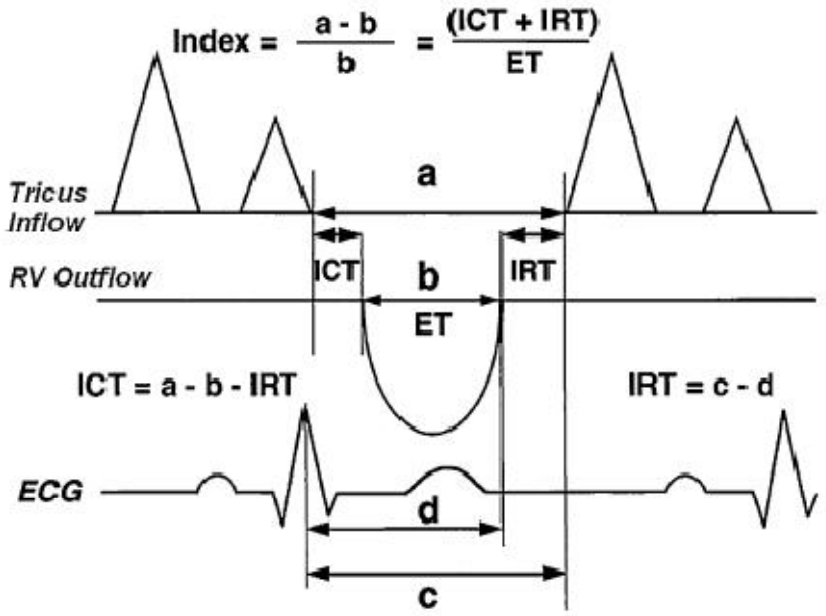
$$\text{RV MPI} = [\text{IVCT (ms)} + \text{IVRT (ms)}] / \text{ET (ms)}$$

IVCT = Isovolumic contraction time

IVRT = Isovolumic relaxation time

ET = Ejection time

Fig - 02. Pulse wave Doppler of the trans tricuspid flow and pulmonary flow to measure Tei index



The mean normal value of myocardial performance index for right ventricle is 0.28 $\pm$ 0.04 [(31)]. 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 [(29)].

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 [30].

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 [34]. 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 [35].

In another study Maheswari et al compared right ventricular Tei index with right ventricular ejection fracion calculated by Simpson's

method in patients with isolated left ventricular anterior wall myocardial infarction [36]. 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 [37]. 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.

Tricuspid Annulus Planar Systolic Excursion (TAPSE)

Unlike in left ventricle, right ventricular contraction is complex and it begins in the sinus or inflow portion of the right ventricle and the last part of the right ventricle to contract is the right ventricular outflow tract and the infundibulum [14]. Also the right ventricular free wall contracts predominantly in a longitudinal axis due to the longitudinal muscle fibers, systolic movement of the base of the right ventricular free

wall towards the apex is one of the most prominent movement seen in echocardiograpy.

TAPSE is calculated in the apical four chamber view as the vertical displacement of the lateral tricuspid annulus during ventricular systole. According to ASE/EAE guidelines, value less than 16 cm was considered abnormal.

Fig - 03. Apical four chamber view showing the M - mode cursor aligned over lateral tricuspid annulus

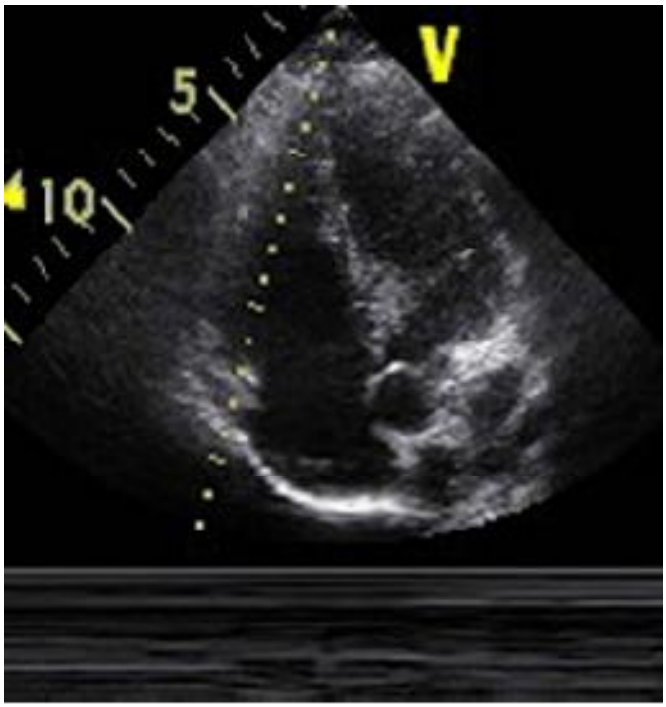
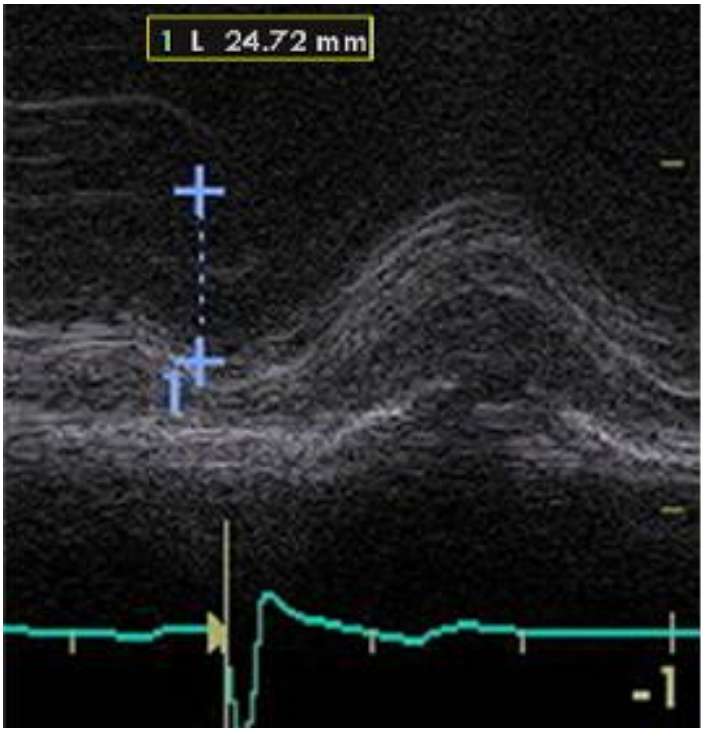


Fig - 04. M - mode measurement of the tricuspid annulus planar systolic excursion



It has been validated from many studies that TAPSE has good correlation with the right ventricular systolic function. In a study done by Kaul et al, TAPSE correlated well with ejection fraction measured with radionuclide angiography [(14)]. It also had very low inter observer variability.

In a study published in Post graduate Medicine Journal 2008, Lopez - Candales et al, studied about right ventricular function in patients with pulmonary hypertension [38]. 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 [37]. 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 [39].

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

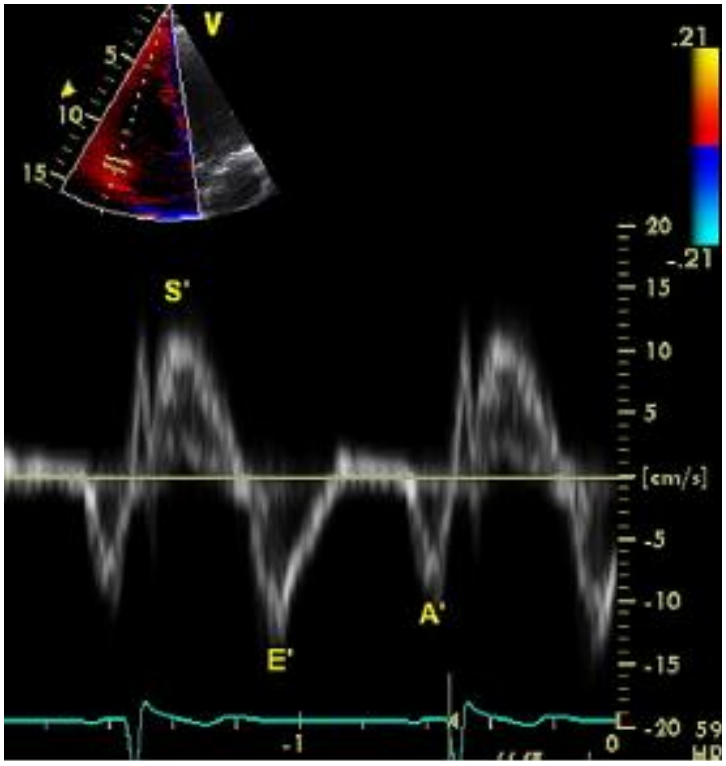
underwent echocardiographic evaluation and were followed for two years. Tricuspid annular plane systolic excursion added prognostic information and correlated well with patients having NYHA class III or IV [(40)].

Tricuspid Annular Peak Systolic Velocity (S')

It is measured using tissue Doppler imaging of the lateral tricuspid annulus. The waveforms should be properly understood to measure the right ventricular systolic and diastolic function using Doppler tissue imaging. Right ventricular dysfunction often reduces the peak velocities. The peak S' wave form which is due to the right ventricular contraction occurs during mechanical systole and it follows pulmonary valve opening.

Tricuspid annular peak systolic velocity was evaluated in Umea general population Heart study among healthy people. Normal value of S' was found to be 15 cm/s at the tricuspid annulus and basal right ventricular free wall and velocities recorded at the mid and apical region of the right ventricle were lower[(41)].

Fig - 05. Tissue Doppler imaging of the lateral tricuspid annulus to measure tricuspid annular peak systolic velocity (s')



Tricuspid annular peak systolic velocity (S') calculated by tissue Doppler imaging is used for the assessment of the function of the basal right ventricular free wall. According to ASE guidelines for the assesment of the right ventricular function, it should be used for the assesing right ventricular function. S' value less than 10 cm/s denotes right ventricular dysfunction.

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% [(42)].

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 [(43)]. 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.

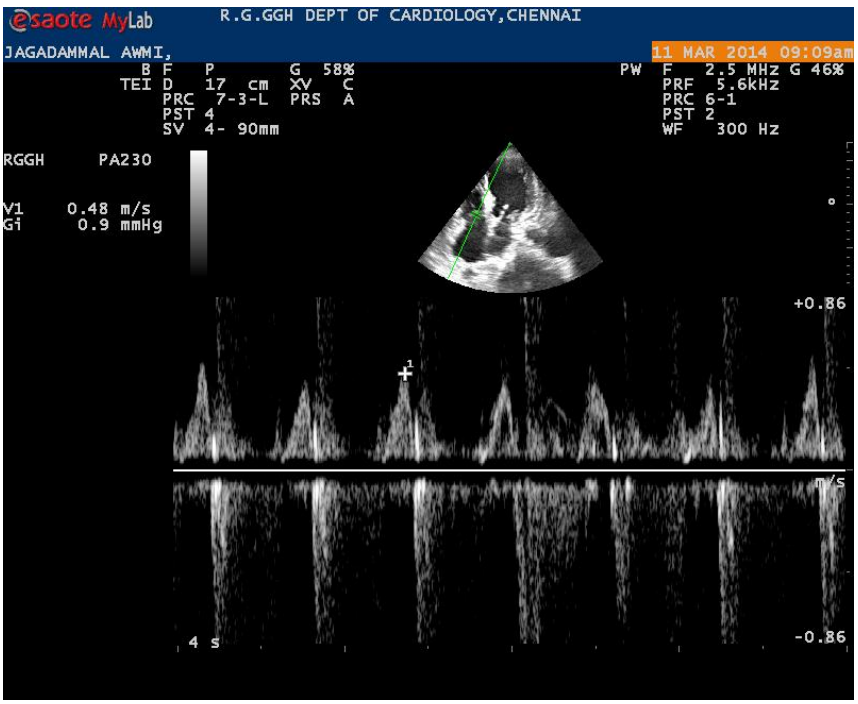
In a Swiss study done by David Tiiller, systolic funtion of the right ventricle was assessed using tricuspid annular peak systolic velocity. This study showed that measurement of the systolic velocity of the lateral annulus of the tricuspic valve correlated with the right ventricular systolic function [(44)].

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 [45].

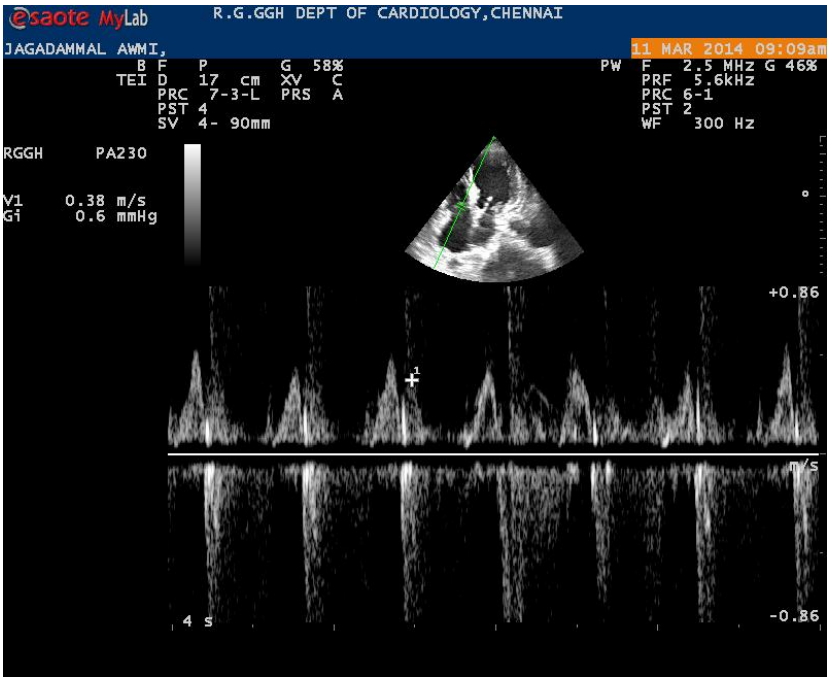
The diastolic function of the right ventricle is assessed using transtricuspid flow doppler velocities (E, A, and E/A), tricuspid annulus tissue Doppler velocities (e', a', e'/a'), deceleration time and isovolumic relaxation time. E/A value between0.8 and less than 2.1 and E/e' value more than 6 suggests pseudo normal filling.

Fig - 06. Pulse wave Doppler method of measuring Early diastolic trans tricuspid flow velocity (E)



Age has a correlation with the E/A ratio. For each decade, there is a decrease of 0.1 in the E/A ratio [46, 47]. 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 [48].

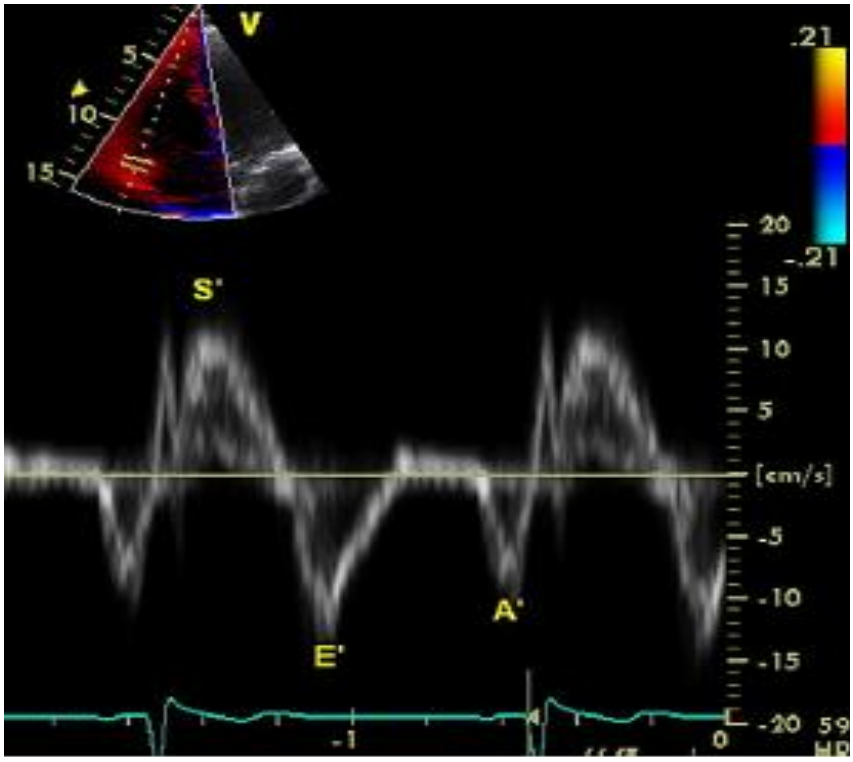
Fig - 07. Pulse wave Doppler method of measuring Late diastolic trans tricuspid flow velocity (A)



Right ventricular diastolic dysfunction is an indicator of mortality in patients with chronic cardiac failure and pulmonary hypertension [31]. 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.

Fig - 08. Tissue Doppler imaging of the lateral tricuspid annulus to measure early (e') and late (a') tricuspid annular diastolic velocity



Right ventricular is most commonly associated with inferior wall infarction of the left ventricle. The association of the right ventricular involvement in antero septal wall myocardial infarction has not been studied extensively. It was first reported in American Heart Journal in 1938 by Feil et al that anteroseptal myocardial infarction was associated with right ventricular infarction in 22% of cases at autopsy [(49)].

In a study published by Paula Azevedo et al, right ventricular dysfunction was present in 13.4% of patients with anterior wall myocardial infarction [50]. This study suggested that left ventricular diastolic dysfunction may be an important predictor of right ventricular dysfunction after six months.

Naeem Tahirkheli et al showed in a study that right ventricle was involved in 10% of patients with antero septal myocardial infarction [51]. He suggested that the mechanism for the involvement of the right ventricle is due to the blood supply of a part of the free wall of the right ventricle from the right ventricular branches of the left anterior descending coronary artery.

In another study done by Cabin et al, 13% of patients with anterior wall myocardial infarction had right ventricular infarction. He also showed right ventricular dysfunction in anterior wall myocardial infarction patients by radionuclide angiography [52].

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.

Diagnosis of failed thrombolysis

Failed thrombolysis is diagnosed by the persistence of chest pain, ECG evidence of less than 50% resolution of the ST segment in the lead with maximum ST segment elevation before thrombolysis.

Diagnosis of failed lysis based on the ecg criteria is maximum ST segment elevation before and after thrombolysis at 80ms from J point. Preferred criteria for failed thrombolysis is failure of the ST segment to decrease > 50% from the pre thormbolytic stage, preferably at 60 min than at 90 minutes [(53)].

Aims and objectives

AIMS AND OBJECTIVES

1. To study the effect of reperfusion therapy on right ventricular function in patients with acute anterior wall ST elevation myocardial infarction.

2. To study the utility of echocardiographic parameters like tricuspid annular plane systolic excursion, tricuspid annular peak systolic velocity, right ventricular myocardial performance index in the evaluation of right ventricular function in patients with acute anterior wall ST elevation myocardial infarction.

Materials and methods

MATERIALS AND METHODS

Setting :

The Study was carried out in the Department of Cardiology, Madras Medical College, Chennai.

Design of the study : Prospective analytical study

Period of the Study : Three months

Sample size : 40 patients

Ethical committee approval :

The present project was approved by the Institutional ethics committee.

Inclusion criteria :

Patients admitted with acute anterior wall ST elevation myocardial infarction at Coronary care unit, Department of Cardiology, Madras Medical College were included.

Exclusion criteria :

01. Inferior wall ST elevation myocardial infarction

02. Previous history of myocardial infarction

03. Previous history of coronary artery bypass grafting

04. Previous history of percutaneous coronary intervention

05. Chronic obstructive lung disease

06. Chronic kidney disease

07. Arrhythmias including atrial fibrillation, supraventricular tachycardia or ventricular ectopics

08. Bundle branch block

09. Atrio ventricular block

10. Severe valvular heart disease

11. Active malignancy

12. End stage liver disease

Consent:

The study group thus identified by the above criteria (inclusion and exclusion criteria) was first instructed about the nature of the study. Willing participants were taken up after getting a written informed consent from them.

Details of the study subjects:

Patients admitted with acute anterior wall ST elevation myocardial infarction in the coronary care unit are included as study subjects. Detailed history, physical examination, electrocardiogram and biochemical investigations were done. Patients who were eligible for reperfusion were treated with streptokinase.

Echocardiographic examination of the patients was done with Esaote my lab echo machine. The parameters studied are tricuspid annulus planar systolic excursion (TAPSE), right ventricular myocardial performance index (MPI), trans tricuspid early diastolic flow (E), lateral tricuspid annulus early (e') and late (a') diastolic velocity, and tricuspid annular peak systolic velocity (s').

Tricuspid Annulus Planar Systolic Excursion

Tricuspid annular motion is measured using M - mode echocardiography. Using apical four chamber view, M - mode cursor is aligned through anterior tricuspid annulus. M - mode cursor should be parallel to the tricuspid annulus. The longitudinal displacement of the annulus from the base to the apex is measured.

Myocardial Performance Index

To obtain Tei index, pulse wave Doppler recording of the tricuspid valve inflow and pulmonary valve outflow is recorded. The duration from the end of A wave of the tricuspid valve inflow to the starting of the E wave of the tricuspid inflow is measured. This is taken as total contraction time. The ejection time is measured from the pulmonary valve outflow tracing. The isovolumic time is calculated by subtracting ejection time from total contraction time. Isovulumic time divided by ejection time gives the myocardial performance index.

Tricuspid Annular Peak Systolic Velocity (s'), tricuspid annulus early diastolic velocity are measured using tissue Doppler imaging of the tricuspid annulus. Trans tricuspid early diastolic flow velocity (E) is measured using pulse wave Doppler of the trans tricuspid flow.

Statistical Analysis:

The collected data was entered in Microsoft excel spread sheet and analysed using Statistical Package for Social Sciences software (SPSS version 17.0). Categorical data are presented as absolute values and percentages, whereas continuous data are summarized as mean value ± standard deviation. Independent sample 't' test and Chi - square tests were used for comparison of categorical variables as appropriate. Significance was considered if the 'p' value was below 0.05.

Results

RESULTS

Forty patients admitted for acute anterior wall ST elevation myocardial infarction were included in the study. Among them 30 patients were lysed and 10 patients were not lysed.

Analysis with respect to sex

Of the total forty patients 11 patients were females and 29 patients were males. Among 11 female patients, 7 patients were treated with streptokinase and 4 people were not treated with streptokinase as they came after the time window period of 12 hours. Of 7 patients 5 patients were considered to have successful lysis and 2 patients were considered to have not successful lysis.

The study population included 72.5% of the subject as male patients and 27.5% as female patients. 79.3% of male patients were lysed with streptokinase and 20.7% of male patients were not lysed. 63.6% of female patients were lysed with streptokinase and 36.4% were not lysed.

Of the 30 patients who were lysed, 76.7% patients were male patients and 23.3% were female patients. Of 10 patients who were not lysed, 60% of them were male and 40% of them were female patients.

Fig - 09. Gender Distribution

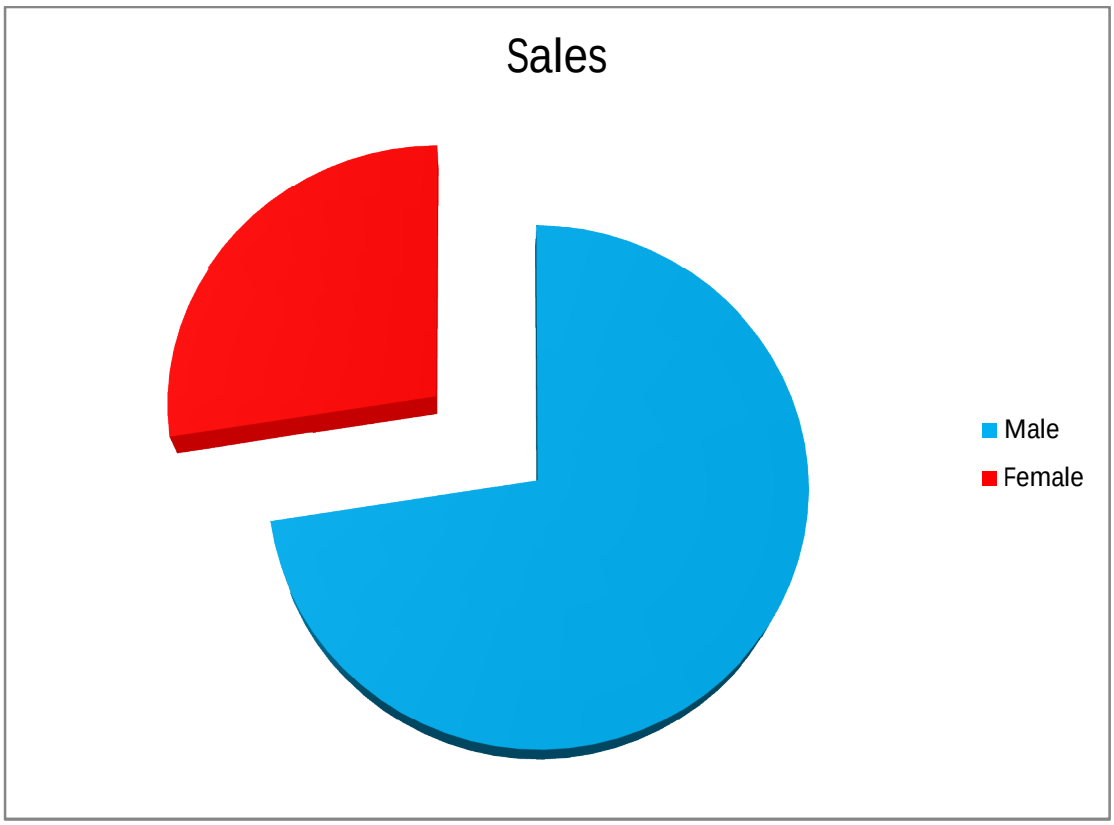
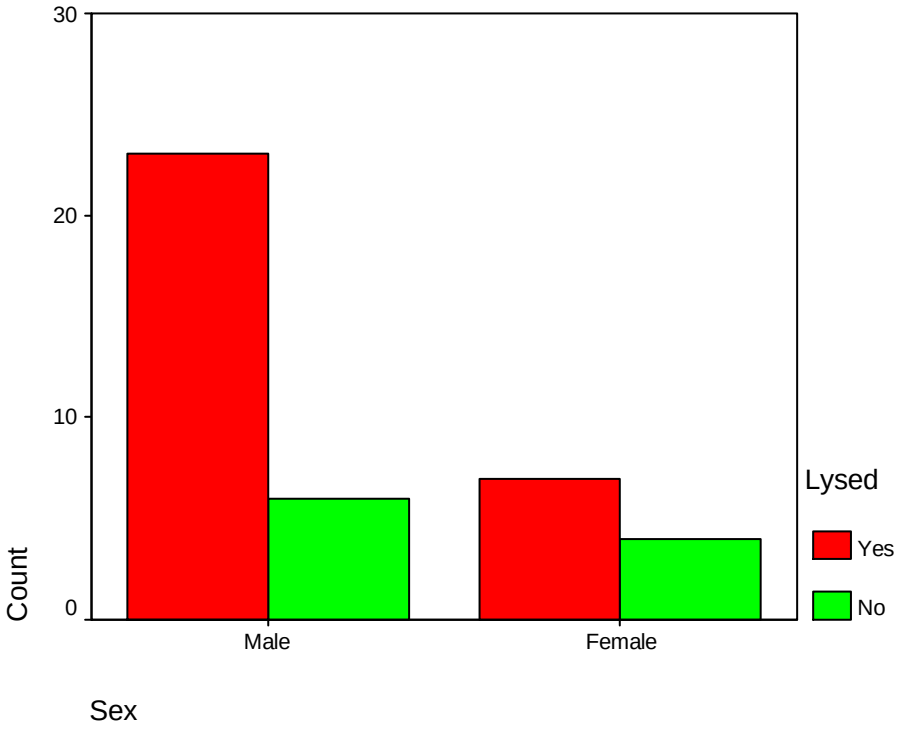


Table – 01 Gender Distribution

			Lysed		Total	P value
			Yes	No		
Sex	**Male**	Count	23	6	29	0.307
		% within Sex	79.3%	20.7%	100.0 %	
		% within Lysed	76.7%	60.0%	72.5%	
	Female	Count	7	4	11	
		% within Sex	63.6%	36.4%	100.0 %	
		% within Lysed	23.3%	40.0%	27.5%	
Total		Count	30	10	40	
		% within Sex	75.0%	25.0%	100.0 %	
		% within Lysed	100.0 %	100.0 %	100.0 %	

Among 29 male patients 23 were treated with streptokinase and 6 patients were not lysed. Of 23 patients, 15 patients had successful lysis and 8 patients had failed lysis.

Fig - 10. Gender Distribution of lysed and not lysed patients



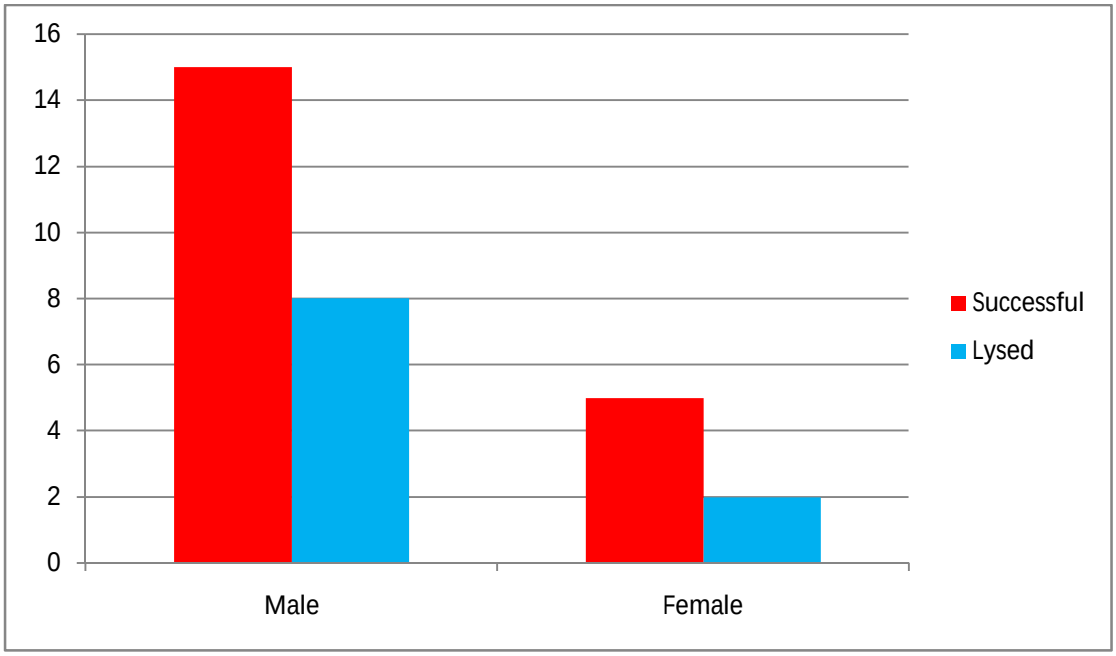
Of 29 male patients, 51.7% of patients had successful lysis and 27.6% had failed lysis and20.7% were not lysed.

Of 11 female patients, 45.5% of patients had successful lysis, 18.2% had failed lysis and 36.4% were not lysed.

Table - 02. Gender Distribution of successful and failed lysis patients

			Successful		**Total**	**P value**
			Yes	**No**		
Sex	**Male**	Count	15	8	23	
		% within Sex	65.2%	34.8%	100.0%	
		% within Successful	75.0%	80.0%	76.7%	
	Female	Count	5	2	7	
		% within Sex	71.4%	28.6%	100.0%	
		% within Successful	25.0%	20.0%	23.3%	
Total		Count	20	10	30	0.76
		% within Sex	66.7%	33.3%	100.0%	
		% within Successful	100.0%	100.0%	100.0%	

Fig - 11. Gender Distribution of successful and failed lysis patients



Analysis with respect to age

The age group of the patients who were treated with streptokinase ranged from 29 years to 80 years.

The age group of the patients who had successful lysis ranged from 29 years to 72 years.

The age group of the patients with failed lysis ranged from 40 to 80 years.

The age group of the patients who were not lysed ranged from 42 to 65 years.

Table - 03. Age distribution

	Lysed	**N**	**Mean**	**Std. Deviation**	**P value**
Age in years	Yes	30	53.77	11.901	0.650
	No	10	55.60	7.306	

The mean age of the patients in the lysed group was 53.77 ± 11.9 years and the mean age of the patients who were not lysed was 55.6 ± 7.3 years. Both had no statistical significance.

Fig - 12. Age Distribution

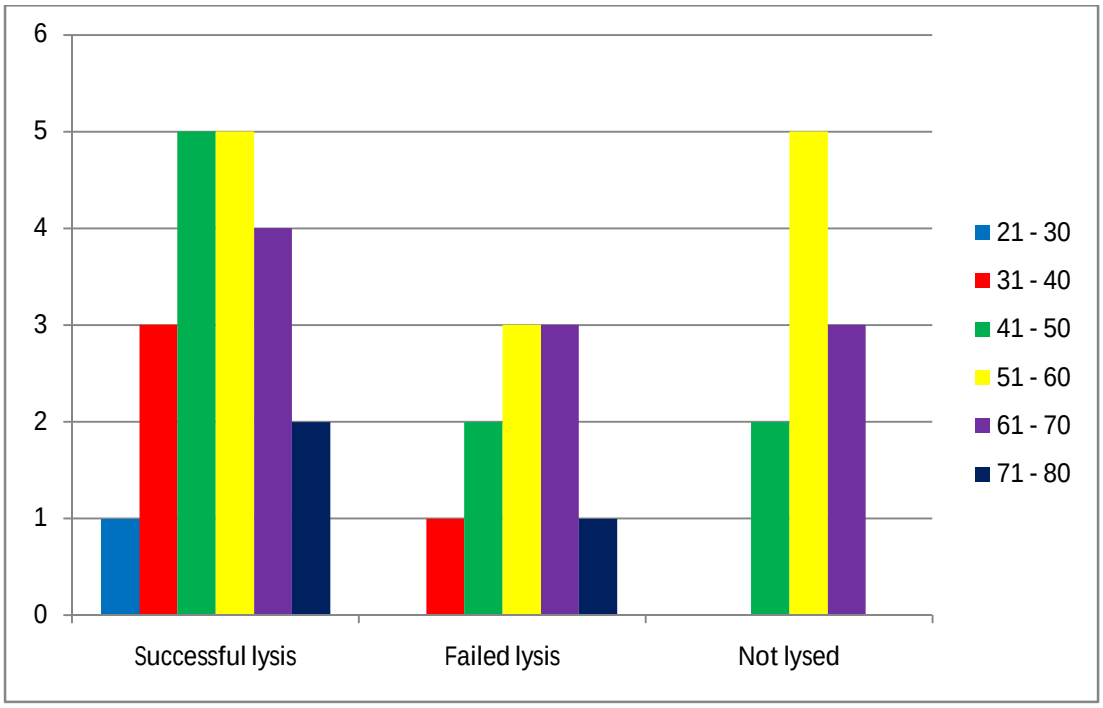
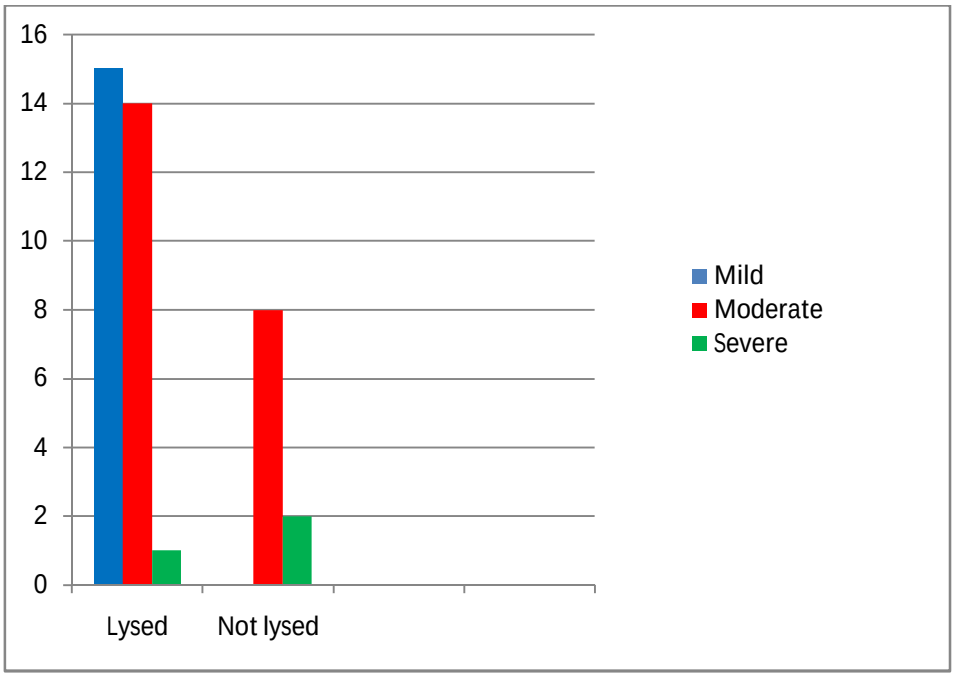


Table - 04. Age Distribution

AGE	LYSED SUCCESS		LYSED FAILED		NOT LYSED	
	NO.	%	NO.	%	NO.	%
20 - 30	1	5	0	0	0	0
31 - 40	3	15	1	10	0	0
41 - 50	5	25	2	20	2	20
51 - 60	5	25	3	30	5	50
61 - 70	4	20	3	30	3	30
71 - 80	2	10	1	10	0	0
TOTAL	20	100%	10	100%	10	100%

Of the patients who had successful lysis, most (50%) of the patients were in the age group of 41 to 60 years. Of the patients who had failed lysis most (60%) of the patients were in the age group of 51 to 70 years. Of the patients who were not lysed most (80%) of the patients were in the age group of 51 to 70 years.

Fig - 13. Left ventricular systolic function of patients



Analysis with respect to left ventricular function

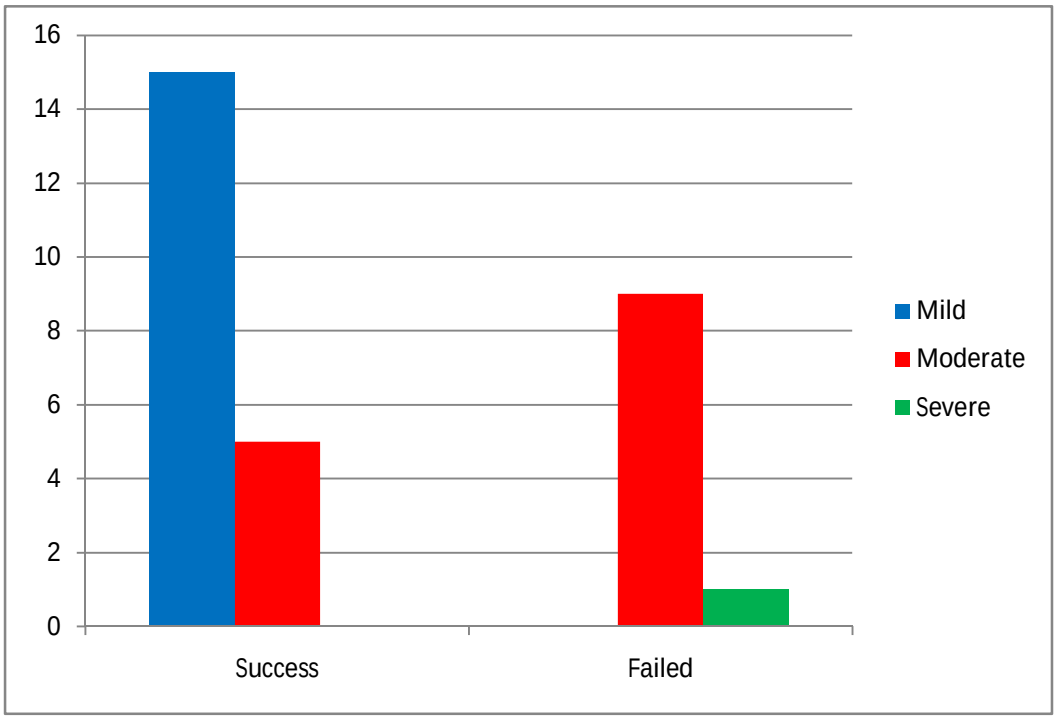
Of 30 patients who were lysed, 15 patients had mild LV systolic dysfunction, 14 patients had moderate LV systolic dysfunction and 1 patient had severe LV systolic dysfunction. Of patients who were not lysed, 8 patients had moderate LV systolic dysfunction and 2 patients had severe LV systolic dysfunction.

Of patients who had successful lysis, 15 patients had mild LV systolic dysfunction and 5 patients had moderate LV systolic dysfunction. Of patients who had failed lysis , 9 patients had moderate LV systolic dysfunction and 1 patient had severe LV systolic dysfunction.

Table - 05. Analysis of patients with respect to Left ventricular systolic function

LYSED	**MILD** **46 - 55**	**MODERATE** **31 - 45**	**SEVERE** **≤ 30**
YES	15	14	1
NO	-	8	2

Fig - 14. Left Ventricular function of lysed patients



Of 30 patients who were lysed, 50% patients had mild LV systolic dysfunction, 46.7% patients had moderate LV systolic dysfucntion and 3.3% patients had severe LV systolic dysfunction.

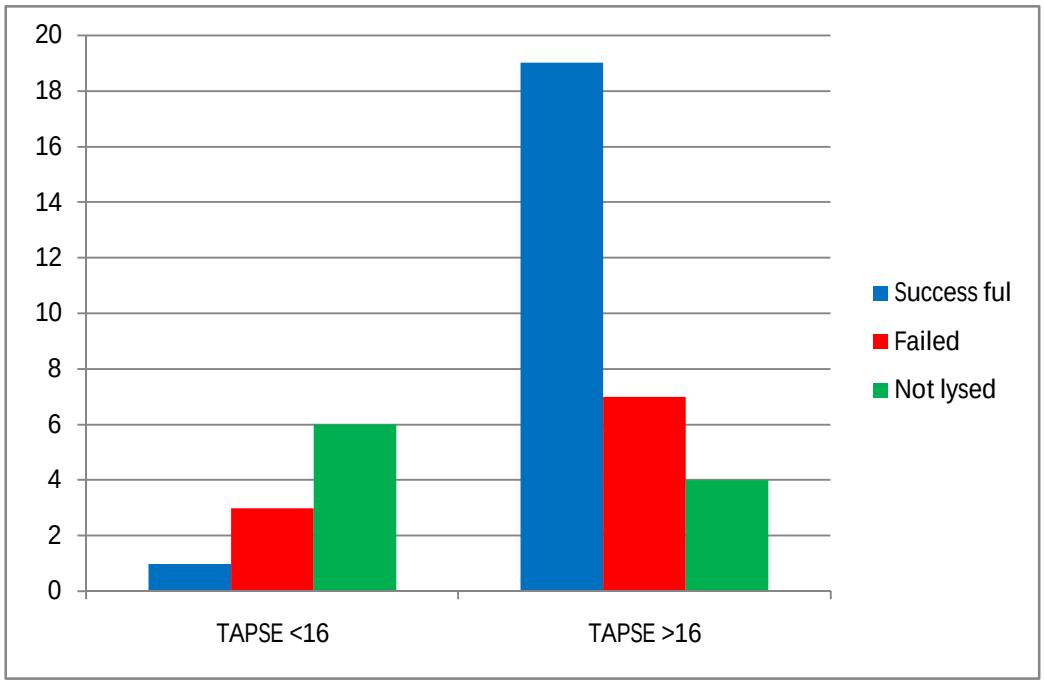
Of 10 patients who were not lysed, 80% patients had moderate LV systolic dysfunction and 20% patients had severe LV systolic dysfunction.

Table - 06. Left Ventricular function of lysed patients

LYSED	**MILD** **46 - 55**	**MODERATE** **31 - 45**	**SEVERE** **≤ 30**
SUCCESSFUL LYSIS	15	5	0
FAILED LYSIS	1	9	0

Of patients who had successful lysis 75% patients has mild LV systolic dysfunction and 25% patients had moderate LV systolic dysfunction. Of patients who had failed lysis, 90% patients had moderate LV systolic dysfunction and 10% patients had severe LV systolic dysfunction.

Fig - 15. Tricuspid Annulus Planar Systolic Excursion



Analysis of Right ventricular function

When right ventricular function of the patients with acute anterior wall ST elevation myocardial function was analysed using Tricuspid annulus planar systolic excursion, right ventricular dysfunction was significantly present in the patients who were not lysed.

Table - 07. Tricuspid Annulus Planar Systolic Excursion

	N	TAPSE	STD DEVIATION	P VALUE
LYSED	30	17.90	2.383	0.002
NOT LYSED	10	14.8	2.781	

Table - 08. Tricuspid Annulus Planar Systolic Excursion

LYSIS	TAPSE < 16 MM	TAPSE >16 MM
SUCCESSFUL LYSIS	1	19
FAILED LYSIS	3	7
NOT LYSED	6	4

From the above data, using TAPSE value of less than 16 mm as right ventricular dysfunction, 60% of the patients who were not lysed had right ventricular dysfunction. Only 5% of the patients who had successful lysis had tricuspid annulus planar systolic excursion value less than 16 mm. In patients who had failed lysis, 30% of the patients had right ventricular dysfunction with reference to TAPSE.

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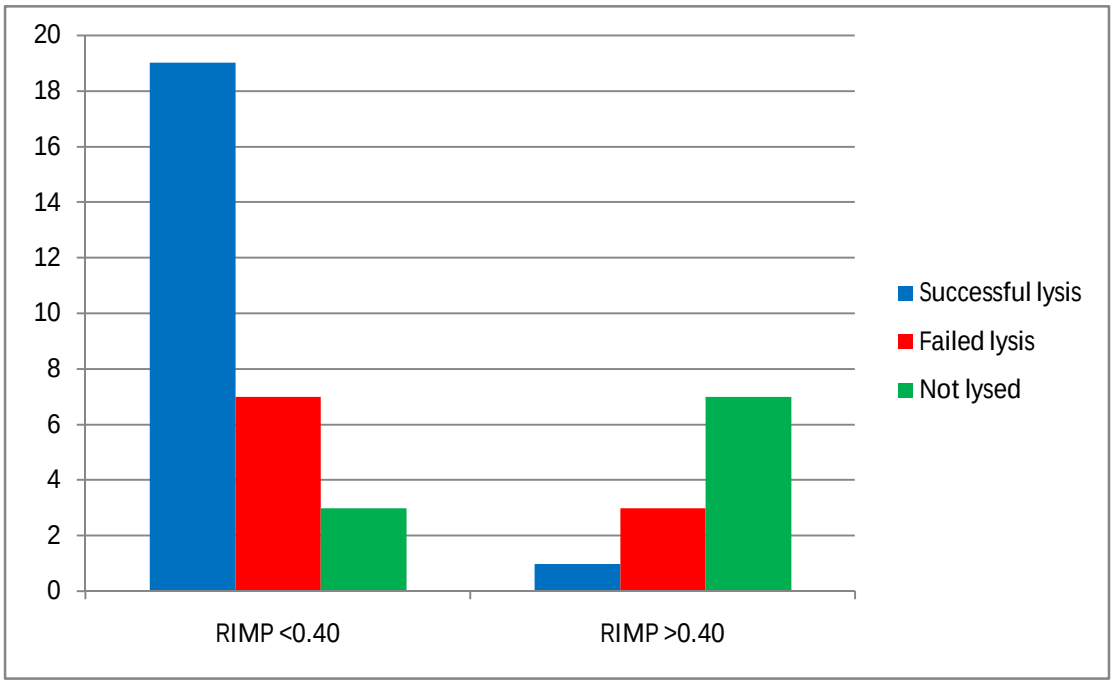
Fig - 16. Right ventricular mycardial performance index



Right ventricular function was assessed using right ventricular myocardial performance index value less than 0.40 as normal and values above 0.40 as abnormal.

Table - 09. Right ventricular mycardial performance index

	N	RIMP	STD DEVIATION	P VALUE
LYSED	30	0.358	0.086	0.003
NOT LYSED	10	0.459	0.093	

Table - 10. Right ventricular mycardial performance index

LYSIS	RIMP <0.40	RIMP >0.40
SUCCESSFUL LYSIS	19	1
FAILED LYSIS	7	3
NOT LYSED	3	7

The above table shows that the patients who were not lysed had more right ventricular dysfunction. 70% of patients who were not lysed had right ventricular dysfunction. 30% of patients who had failed lysis had right ventricular dysfunction, while only 5% of patients who had successful lysis had right ventricular dysfunction.

Tricuspid annular peak systolic velocity (S') represents the systolic function of the right ventricle. It is measured at the anterior tricuspid annulus using Tissue Doppler imaging. Value of less than a10 cm/s is considered abnormal and representative of right ventricular dysfunction. In our study tricuspid annular peak systolic velocity was normal among lysed patients and abnormal among not lysed patients.

Table - 11. Tricuspid Annulus Peak Systolic Velocity

LYSED	N	S' m/s	STD DEVIATION	P VALUE
YES	30	0.154	0.217	0.49
NO	10	0.096	0.029	

The value of s' is 0.15 ±0.21 m/s in patients who were lysed and 0.09 ± 0.03 m/s in patients who were not lysed. However the p value is not statistically significant.

Fig - 17. Tricuspid Annulus Peak Systolic Velocity

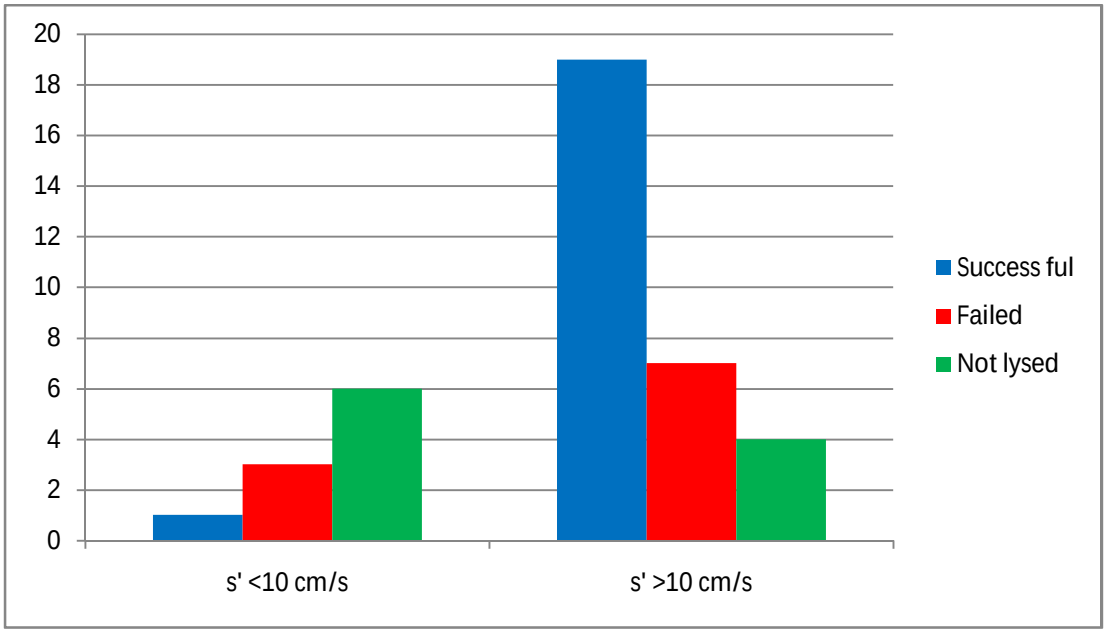


Table - 11. Tricuspid Annulus Peak Systolic Velocity

LYSIS	S' < 10 cm/s	S' >10 cm/s
SUCCESSFUL LYSIS	1	19
FAILED LYSIS	3	7
NOT LYSED	6	4

Tricuspid annular peak systolic velocity was abnormal mainly in patients who were not lysed. In 60% of the patients who were not lysed, it was less than 10 cm/s. In patients who had failed lysis, s' was less than 10 in 30% of the patients.

When compared with left ventricular dysfunction, right ventricular dysfunction was present 46.7% of patients with moderate left ventricular systolic dysfunction and 42.9% of patients with severe left ventricular dysfunction. Of the patients who had right ventricular dysfunction in patients with moderate left ventricular dysfunction, 26.7% of the patients were not lysed and13.3% of the patients were with failed lysis.

Table - 12. RV function with respect to LV function

STUDY GROUP	LV DYSFUNCTION	RV FUNCTION NORMAL	RV FUNCTION ABNORMAL
SUCCESSFUL LYSIS	MILD	14	0
	MODERATE	5	1
	SEVERE	0	0
FAILED LYSIS	MILD	1	0
	MODERATE	6	2
	SEVERE	0	1
NOT LYSED	MILD	0	0
	MODERATE	4	4
	SEVERE	0	2

In this study when comparing the diastolic function of the patients lysed with the patients not lysed, patients who were lysed have tricuspid valve E/A ratio of impaired relaxation as per ASE echo guidelines and patients who were not lysed have tricuspid valve E/A ratio of pseudo normal filling.

Table - 13. Diastolic Function

LYSED	N	E/A	SD	P VALUE
YES	30	0.83	0.26	0.002
NO	10	1.28	0.57	

Table - 14. Diastolic Function

LYSED	N	E/e'	SD	P VALUE
YES	30	5.38	0.26	0.005
NO	10	6.99	0.57	

Tricuspid valve E/A ratio for lysed patients is 0.83 ± 0.26 and for not lysed patients is 1.28 ± 0.57. This has statistically significant corelation. E/e' ratio for lysed patients is 5.38 ± 1.27 and for not lysed patients is 6.99 ± 2.04. This is also statistically significant.

-

Discussion

DISCUSSION

This study was done to find the presence of right ventricular dysfunction in patients presenting with acute anterior wall ST elevation myocardial infarction. Parameters like TAPSE, TASV, right ventricular myocardial performance index, tricuspid valve E/A ratio and E/e' ratio were done in patients with acute anterior wall ST elevation myocardial infarction to study right ventricular function.

This study showed that tricuspid annulus planar systolic excursion correlated more with the right ventricular function. It was simple to use and assess the right ventricular function. In a study done by Monika Maheswari et al, TAPSE was compared with right ventricular ejection fraction calculated with Simpson's method in patients with isolated anterior wall myocardial infarction [(54)]. This study showed that TAPSE calculated at the septal side of the tricuspid valve in patients with anterior wall myocardial infarction was significantly lower than control subjects.

In our study, the occurrence of right ventricular dysfunction was more among patients who were not lysed. Patients who had failed lysis had lesser incidence of right ventricular dysfunction when compared with patients who were not lysed.

Our study also showed that most of the patients who had right ventricular dysfunction, had moderate left ventricular systolic dysfunction. Of 10 patients who had right ventricular dysfunction, 70% had moderate left ventricular systolic dysfunction and the remaining 30% patients had severe left ventricular dysfunction.

Ozlem karakurt et al shown in his study that right ventricular Tei index was increased in patients with anterior wall myocardial infarction who were treated with thrombolysis and percutaneous coronary intervention when compared with non anterior wall myocardial infarction[55].

Paula Azevedo et al studied the prevalence of right ventricular dysfunction in patients with anterior wall myocardial infarction [50]. They considered that right ventricular dysfunction was due to the mechanism of ventricular interdependence which was mediated through the interventricular septum.

Contraction of the right ventricle is dependent on three mechanisms. Contraction of the free wall of right ventricle towards the interventricular septum, contraction of the longitudinal muscle fibres of the right ventricle and the movement of the interventricular septum into the right ventricle during left ventricular contraction are the three

mechanisms considered to be responsible for the right ventricular hemodynamics [10]. TAPSE represents the longitudinal systolic function of the tricuspid annulus and hence correlate with the systolic function of the right ventricle.

Around 20 to 40% of right ventricular stroke volume is contributed mainly by the left ventricular contraction along with the movement of the interventricular septum into the right ventricle [56]. This dependence of the right ventricular function on the interventricular septum is ventricular interdependence. Hence both the right and left ventricles which are not only connected anatomically by sharing common muscle bundles, they are also dependent on each other functionally. In sinus rhythm, the right ventricular systolic function is contributed by the pressure generated by the left ventricular contraction and the interventricular septum which is supported during systole by the increased left to right trans septal pressure gradient [57].

In this study there is a good correlation between tricuspid annulus planar systolic excursion and the left ventricular ejection fraction. TAPSE was significantly decreased more in patients with moderate and severe left ventricular systolic dysfunction. This indicates that left ventricular systolic dysfunction was associated with right ventricular systolic

dysfunction because of ventricular interdependence. Hence it implies that good right ventricular function was associated with an increase in tricuspid annulus planar systolic excursion.

It has been previously stated that both the right and left ventricle share common myocardial fibers. Hence when this common myocardial fiber over the left ventricle is damaged as in acute anterior wall myocardial infarction, the part of the common myocardial fiber which encircles over the right ventricle will also be abnormal and hence it may contribute to the right ventricular dysfunction.

It has been shown in the GISSI - 3 Echo sub study that right ventricular dysfunction which occurred with acute ischemia recovered once the left ventricular ejection fraction improved [58].

Right ventricular dysfunction causes right ventricular dilatation which raises the diastolic pressure and shift the inter ventricular septum towards the left ventricle and affects the left ventricular diastolic function and when the reverse occurs, it affects the right ventricular diastolic function. This leads to the raise of intra pericardial pressure because of the noncompliant pericardium [58, 60].

Another mechanism by which right ventricular dysfunction occurs in the acute anterior wall myocardial infarction is explained by the blood supply to the right ventricle. James et al has reported that in about 24% of the human hearts, 30% of the right ventricular free wall was supplied by the right sided branches of the left anterior descending coronary artery [61]. Hence in proximal or mid left anterior descending coronary artery occlusion which occurs in patients with antero septal myocardial infarction, right ventricle may involve producing right ventricular dysfunction.

Also the left anterior descending coronary artery wraps around the apex in 22% of human heart and supply the inferior wall of the right ventricle and end in the posterior inter ventricular groove [61]. Hence occlusion of this artery produces inferior wall myocardial infarction of both the right and left ventricles at the apex. This may also produce right ventricular dysfuncion.

Another potential mechanism is that right coronary artery may have a high grade stenosis and hence right ventricle may be supplied by the collateral branches from the left anterior descending coronary artery. In this situation also occlusion of the left anterior descending coronary artery produces right ventricular dysfunction.

Conclusion

CONCLUSION

This study has showed that right ventricular dysfunction may also occur in patients with acute anterior wall ST elevation myocardial infarction patients.

Right ventricular dysfunction is explained by many factors, namely the role of left ventricular dysfunction causing right ventricular dysfunction, ventricular interdependence which was mediated mainly through the inter ventricular septum, common myocardial fibers shared by both the right ventricle and the left ventricle and the blood supply of the anterior wall of right ventricle by the left anterior descending coronary artery.

Even though both tricuspid annulus planar systolic expansion and Tei index are more sensitive to recognize right ventricular dysfunction, tricuspid annulus planar systolic expansion is a simple, easy and rapid method to measure the right ventricular function.

It is important to recognize right ventricular dysfunction early because right ventricular dysfunction may cause cardiogenic shock and hence treating right ventricular dysfunction may reduce morbidity and mortality.

Limitations of the study

LIMITATIONS OF THE STUDY

01. Coronary angiography was not done

02. Patients were treated with less fibrin specific thrombolytic agent streptokinase and not with highly fibrin specific thrombolytic agents as in our Government Institution they were not sanctioned.

03. Patients who were treated with primary percutaneous coronary intervention were not included in the study.

04. Long term follow up of patients was not done.

Appendix

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ACRONYMS

TAPSE - Tricuspid annulus planar systolic excursion

RIMP - Right ventricular index of myocardial performance

TASV - Tricuspid peak annular systolic velocity

RV - Right ventricle

LV - Left ventricle

EF - Ejection fraction

EFFECT OF REPERFUSION ON RIGHT VENTRICULAR FUNCTION IN ACUTE ANTERIOR WALL ST ELEVATION MYOCARDIAL INFARCTION

PROFORMA

NAME: AGE: SEX:

ADDRESS: CD NO:

RISK FACTORS

1.	Gender	
2.	Smoker	
3.	Diabetes Mellitus	
4.	Hypertension	
5.	Hyperlipidemia	

CLINICAL EXAMINATION

PR: BP:

1.	Killip Class		
2.	Median Delay		
3.	Thrombolytic Therapy		
4.	Thrombolysis	Successful	
		Failed	

ECG

ECHO

SL. NO.			
01.	LV EF	LV EDV ml	
		LV ESV ml	
		LV EF (MODIFIED SIMPSON)	
		LVIDD cm	
		LVIDS cm	
		EF%	
02.	MV	MV E VELOCITY cm/s	
		MV A VELOCITY cm/s	
		E/A RATIO	
		DECELERATION TIME ms	
03.	MVA TDI	e' cm/s	
		a' cm/s	
		S cm/s	
		E/e'	
04.	RIMP	TCT ms	
		IVCT +IVRT = TCT - ET ms	
		ET ms	
	RIMP	IVCT+IVRT/ET	
05.	TAPSE	cm	
06.	TA TDI	e' cm/s	
		a' cm/s	
		s' cm/s	
07.	TV	E VELOCITY cm/s	
		A VELOCITY cm/s	
		E/A	
		DT ms	
		E/e'	

MASTER CHART

Sl.No.	age	sex	smoking	diabetes	hypertensio	killip class	median dela	lysed	successful	LV EF	MV E/A	MV E/e'	TAPSE	RIMP	S'	TV E/A	TV E/e'
1	38	m	yes	no	no	2	12	yes	yes	31	0.87	4.4	13	0.41	0.09	0.67	4.5
2	40	m	yes	no	no	1	12	yes	no	39	2.2	7	19	0.25	0.11	0.84	2.2
3	80	f	no	no	no	2	11	yes	no	25	1.1	11.2	12	0.72	0.08	1.2	9.4
4	52	m	yes	yes	no	2	16	no	-	32	0.7	12.6	14	0.6	0.09	1.4	11.8
5	61	m	yes	no	no	2	8	yes	yes	52	1.2	5.3	19	0.35	0.12	1.1	4.9
6	46	m	no	yes	yes	1	7	yes	yes	43	0.69	14.1	17	0.39	0.11	1.6	8.3
7	48	f	no	yes	yes	1	5	yes	yes	48	1.3	5.8	21	0.31	0.12	0.72	5.2
8	57	f	no	yes	no	3	18	no	-	33	0.79	13.3	10	0.53	0.06	1.3	8.2
9	61	m	yes	yes	no	2	21	no	-	38	0.9	9.6	18	0.38	0.16	0.66	5.7
10	29	m	yes	no	no	1	5	yes	yes	53	1.1	7.7	18	0.31	0.16	0.79	5.1
11	44	m	no	no	no	1	6	yes	yes	50	0.8	6.1	21	0.28	0.12	0.73	5.9
12	71	m	no	yes	yes	1	8	yes	yes	41	1.2	9	17	0.39	0.11	0.78	5.2
13	61	f	no	yes	no	2	9	yes	yes	47	1.1	8	19	0.33	0.14	0.64	4.9
14	66	m	yes	no	yes	2	10	yes	no	36	0.71	12.6	12	0.45	0.09	1.2	6.2
15	49	m	no	no	no	1	4	yes	yes	45	0.84	7.6	18	0.33	0.11	0.79	5.5
16	39	m	yes	no	no	2	7	yes	yes	52	1.1	8.2	19	0.31	0.12	0.83	4.6
17	57	f	no	yes	no	2	9	yes	yes	49	1.3	9.2	21	0.39	0.11	0.76	5.9
18	63	f	no	yes	yes	2	15	no	-	30	1.2	9.2	15	0.46	0.09	1.2	6.2
19	43	m	yes	no	no	2	10	yes	yes	52	1.1	7.9	18	0.32	0.12	0.76	5.3
20	58	m	no	yes	yes	3	5	yes	no	36	1.3	8.9	17	0.39	0.12	0.71	4.9
21	67	m	no	no	no	1	3	yes	yes	51	1.2	6.4	19	0.29	0.13	0.65	4.2
22	59	m	yes	no	no	2	7	yes	no	31	0.92	11.6	15	0.49	0.08	1.7	7.2
23	42	m	yes	no	no	2	13	no	-	44	1.4	9.3	17	0.41	0.11	0.75	4.8
24	49	m	no	yes	yes	1	9	yes	no	42	1.5	11.4	18	0.34	0.12	0.78	5.9
25	51	f	no	yes	no	2	7	yes	yes	50	1.3	7.8	19	0.38	0.12	0.71	4.5
26	64	f	no	yes	yes	2	11	yes	no	41	1.4	12.3	17	0.39	0.1	0.78	5.3
27	57	m	yes	yes	no	3	16	no	-	32	1.1	9.6	19	0.36	0.11	0.69	5.1
28	37	m	yes	no	no	1	4	yes	yes	48	1	7.1	20	0.35	0.12	0.71	5.8
29	54	m	no	yes	yes	2	17	no	-	40	1.2	9.2	14	0.48	0.09	1.9	6.8
30	65	f	no	yes	no	2	20	no	-	36	1.4	11.2	12	0.56	0.08	2	7.9
31	56	m	yes	yes	no	2	5	yes	yes	47	1.1	7.6	18	0.31	0.12	0.68	4.9
32	43	m	yes	no	no	2	11	yes	no	43	1.4	9.4	17	0.34	0.11	0.69	5.8
33	51	f	no	yes	yes	1	6	yes	yes	54	1.1	7.6	19	0.29	0.12	0.76	4.2
34	61	m	no	yes	no	2	7	yes	no	42	1.3	8.4	20	0.32	0.11	0.65	4.9
35	46	m	yes	no	no	2	22	no	-	28	1.6	12.3	13	0.51	0.06	2.1	7.5
36	72	m	no	no	yes	1	4	yes	yes	49	1.1	7.2	17	0.34	0.13	0.64	5.1
37	59	f	no	yes	no	2	16	no	-	37	1.6	9.7	16	0.33	0.11	0.76	5.9
38	56	m	no	no	yes	2	8	yes	yes	44	1.2	6.2	21	0.31	0.13	0.72	4.8
39	53	m	yes	no	no	2	5	yes	no	46	1.3	8.1	19	0.38	0.11	0.69	5.2

INSTITUTIONAL ETHICS COMMITTEE

MADRAS MEDICAL COLLEGE, CHENNAI – 600 003.

EC Reg. No. ECR /270/Inst/TN/2013

Telephone No. 044 25305301

Fax 044 25363970

CERTIFICATE OF APPROVAL

To

Dr . K.Sidharthan ,

Post graduate in DM Cardiology,

Department of Cardiology,

Madras Medical College, Chennai 600 003.

Dear Dr. K. Sidharthan ,

The Institutional Ethics Committee of Madras Medical College , reviewed and discussed your application for approval of the proposal Effect of Reperfusion on Right Ventricular Function in Acute Anterior Wall ST Elevation Myocardial Infarction No. 27122013.

The following members of the Ethical Committee were present in the meeting held on 11.12.2013 conducted at Madras Medical College, Chennai – 3.

1.	Dr .G.Sivakumar , MS FICS FAIS	Chairperson
2.	Prof.B.Kalaiselvi , MD Vice Principal, MMC, Ch3	Member Secretary
3.	Prof.Ramadevi , Director i/c, Institute of Biochemistry, Chennai	Member
4.	Prof . P.Karkuzhali , MD; Prof.Inst .of Pathology, MMC, Ch 3	Member
5.	Thiru .S.Govidasamy , BA., BL.,	Lawyer
6.	Tmt . Armold Saulina , MA MSW	Social Scientist

We approve the proposal to be conducted in its present form.

Sd / Chairman & other Members.

The Institutional Ethics Committee expects to be informed about the progress of the study , and SAE occurring in the course of the study , any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

27/12/13

Member Secretary , Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

INFORMATION SHEET

- We are conducting a study of the **"Effect of reperfusion on right ventricular function in acute anterior wall ST elevation myocardial infarction**'' at the Department of Cardiology, Rajiv Gandhi Govt. General Hospital, Chennai. The purpose of this study is to investigate the right ventricular function in the reperfused acute anterior wall ST elevation myocardial function

- The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

- Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time. Your decision will not result in any loss of benefits to which you are otherwise entitled.

- The results of the study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of the investigator **Signature of the participant**

Date:

ஆராய்ச்சி தகவல் தாள்

சென்னை அரசு பொது மருத்துவமனையில் இதய தீவிர மருத்துவப் பிரிவில் மாரடைப்பு நோயுடன் சேரும் நோயாளிகளின் வலது கீழரையின் செயல்பாட்டை ஆராய 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:

PATIENT CONSENT FORM

Study Details : **Effect of reperfusion on right ventricular function in acute anterior wall ST elevation myocardial infarction**

Study Centre : **Department of Cardiology,**
Madras Medical College and
Rajiv Gandhi Government General Hospital,
Chennai - 600 003.

Patient may check (□) these boxes:

I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask question and all my questions and doubts have been answered to my complete satisfaction. ☐

I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected. ☐

I understand that the investigator of the clinical study, others working on his behalf, the ethical committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study. ☐

I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well being or any unexpected or unusual symptoms. ☐

I hereby give permission to undergo complete clinical examination and diagnostic tests including hematological, biochemical, radiological, EMG, EEG, NCS, Lumbar puncture and muscle biopsy, appropriate to the clinical diagnosis. ☐

I hereby consent to participate in this study. ☐

Signature / Thumb impression: Place : Date :

Patient Name and Address:

Signature of Investigator: Place : Date :

Study Investigator's Name :

சுய ஒப்புதல் படிவம்

<u>ஆய்வுசெய்யப்படும்தலைப்பு</u>: சென்னை அரசு பொது மருத்துவமனையில் இதய தீவிர மருத்துவப் பிரிவில் மாரடைப்பு நோயுடன் சேரும் நோயாளிகளின் வலது கீழரையின் செயல்பாடு.

<u>ஆராய்ச்சி நிலையம்</u>: இருதய மருத்துவத் துறை,
இராஜீவ் காந்தி அரசு பொது மருத்துவமன
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. ☐

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