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A Prospective Observational Study on Echocardiographic Assessment of Left Ventricular Diastolic Dysfunction in Patients with Anterior Wall Myocardial Infarction Post Revascularization

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Abstract: ***Introduction: Diastolic Function are described as the rate and duration of the left ventricular pressure drop after systole and compliance, which is determined by volume changes over pressure changes during diastolic filling.LV diastolic function can be diagnosed noninvasively using two-dimensional and Doppler echocardiography. Three aberrant LV filling patterns are found based on Doppler study of mitral and pulmonary venous flow: poor relaxation, "pseudo normalization," and restrictive patterns. Myocardial ischemia causes abnormal myocardial relaxation that can be restored to normal by restoring normal myocardial blood flow. With the aid of mitral and pulmonary venous flow analysis, the diastolic anomaly can be detected within a matter of seconds and is accompanied by a distinctively defective relaxation filling pattern. In which Diastolic dysfunction is known to exist with or without LV systolic failure, diastolic dysfunction has been identified in the early as well as the post-MI phase. Both an aberrant relaxation pattern and a restrictive LV filling pattern are evident in the acute phase. Diastolic dysfunction in acute anterior wall MI is a common complication that can significantly impact patient outcomes. Diastolic dysfunction occurs prior to Systolic dysfunction according to systemic Cascade. Aim and Objectives: A prospective observational study, to assess Left Ventricular Diastolic dysfunction using 2D echocardiography in patients with revascularized Acute Anterior wall MI. Materials and Methods: A total of 120 patients who presented with acute anterior wall MI has been assessed for Left Ventricular diastolic dysfunction after Revascularization by using 2D echocardiography. Results: Out of 120 patients,44 patients were in Group 1 having Grade 1 and Grade 2 Diastolic dysfunction and 76 patients were in Group 2 having Grade 3 and Grade 4 diastolic dysfunction. This showed higher number individuals 63.3% have severe Diastolic Dysfunction (elevated filling pressure (E/A ratio), reduced tissue Doppler E/e' ratio, IVRT and reduced S/D ratio), following a revascularized MI. Conclusion: LV diastolic function evaluation is crucial in acute MI assessment. Doppler echocardiography provides key parameters, Mitral inflow E/A ratio, Tissue Doppler E/e', Isovolumetric relaxation time, Deceleration time. These parameters help stratify patients' risk of MI. Furthermore, LV diastolic dysfunction is common in acute MI patients, even after successful percutaneous coronary intervention (PCI). Diastolic dysfunction significantly increases MI risk, with higher grades of dysfunction associated with greater risk. Accurate assessment of LV diastolic function is essential for identifying patients at higher risk of adverse outcomes***

Keywords: AMI - Acute myocardial infarction AWMI - Anterior wall myocardial infarction DD – Diastolic dysfunction IVRT - Isovolumetric relaxation time S/D ratio - Systolic to diastolic ratio PASP- Pulmonary artery systolic pressure

1. Introduction

Diagnosis of AWMI

An ECG should be recorded as soon as possible and will typically show ST segment elevation >1mm in two contiguous leads or > 2mmin chest leads V1-V3. Cardiac troponin levels should be measured at presentation and at 12 and 24 hours after symptom onset. It should be noted however, that the release of troponin from the myocardial contractile apparatus into blood occurs slowly, so troponin levels in blood can take at least 6 hours to reach the discrimination threshold thus patients who present very early after symptom onset may initially test negative for troponins. CKMB levels should be measured at24 -36hrs. to provide a baseline for the detection of subsequent reinfarction. cardiac troponin levels may remain elevated for 10-14 days after the index infarction and so troponin testing cannot reliably used to identify reinfarction within this time frame.

LV Diastolic Function

Doppler echocardiography measures a number of factors, including mitral inflow, annular motion .The assessment of transmittal velocities by placing pulse wave Doppler cursor at mitral valve leaflets tips in apical four chamber view, with ideal sample volume of approximately 1 to 3 mm. Tissue Doppler can be used to acquire velocities along the medial and lateral aspects of mitral annulus to assess left ventricular end diastolic pressures (LVEDP) when used with E /e', and to determine the effects of LV relaxation properties on mitral inflow E wave velocities. sample volume is placed within 1cm of septal and lateral mitral annulus with baseline adjusted and sweep speeds set around 50 to 100mm/sec at end expiration.

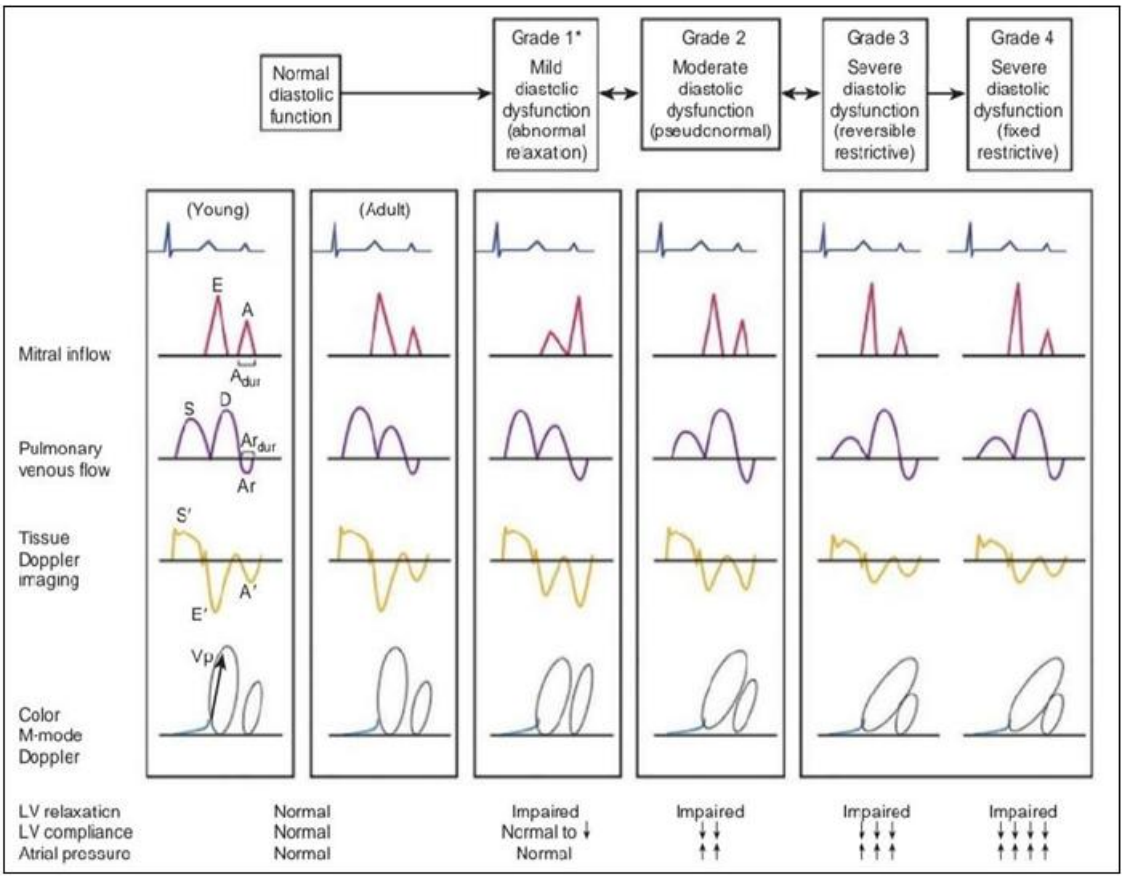

Figure 1: Diastolic function grading pattern

Table 1: Grades of diastolic dysfunction

Parameters	Normal	Grade I	Grade II	Grade III
LV RELAXATION	Normal	Impaired	Impaired	Impaired
LADIAMETER	Normal	Low or normal	Elevated	Elevated
MITRALE/A RATIO	>0.8	<0.8	>0.8to<2	>2
DECELERATION TIME(MS)	150-240	>240	<150	<150
E/e' RATIO	<10	<10	10-14	>14
ISOVOLUMETRIC CONTRACTION TIME (IVRT)	70-90	>90	<70	<70
PEAKTRVELOCITY(m/s)	<2.8	<2.8	>2.8	>2.8
ARDURATION (msec)	<35	<35	>35	>35
PULMONARYVENOUS DURATION(S/D)ratio	>1	>1	<1	<1
LAVOLUME INDEX	Normal	Normal orincreased	Increased	Increased
PRPOGATION VELOCITY(CM/SEC)	>55	>45	<45	<45

Thrombolysis or Primary Angioplasty:

In myocardial infarction with ST segment elevation (STEMI) occlusive and persistent thrombosis prevails. Most cases are precipitated by sudden rupture of vulnerable plaque followed by superimposed thrombus formation. reperfusion therapy whether catheter based or pharmacologic is critically needed to restore antegrade flow through the infarct – related artery and thus arrest the propagating wave of necrosis. To preserve myocardium and reduce morbidity and mortality reperfusion must be rapid, complete and sustained. Traditionally, pharmacological and catheter –based reperfusion therapies have been considered distinct and mutually exclusive strategies. Each strategy has its own advantages and shortcomings. Fibrinolytic therapy is widely available and can be given rapidly emergency departments. Even with the most efficacious fibrinolytic agents however normal (thrombolysis in myocardial infarction TIMI grade 3) flow at 90minutes is achieved in only 50-60% of patients. Intracranial hemorrhage occurs in approximately 1% of patients treated with fibrinolytic therapy and is fatal in >50% of patients. almost 90% of patients with patent infarct - related artery at 90 minutes after fibrinolytic therapy have a residual stenosis >50% at the culprit lesion, leaving the vessel more prone to re-occlusion. Re-infarct occurs in 5-6% of patients within first week of the treatment. In contrast primary angioplasty results in TIMI grade 3 follow in more than 70% of patients, and patency (TIMI 2 or 3 flow) in < 90% of patients without the hemorrhagic risk of fibrinolytic therapy. It also significantly reduces the risk of reinfarction because primary angioplasty not only restore brisk antegrade flow but also treats (eliminates) the underlying stenosis.

Primary Angioplasty Versus Fibrinolysis:

Primary angioplasty was better than fibrinolysis at reducing overall short-term mortality, non-fatal reinfarction, stroke, and combined end point of death. A better outcome was seen with primary angioplasty. Primary angioplasty is considered a superior strategy both for efficacy and safety. For primary

angioplasty clinical trials have defined the benefit of adjunctive stenting and use of platelet glycoprotein (Gp IIb /II a blockade). For fibrinolysis Gp IIb/IIIa inhibitor combined with reduced –dose fibrinolytic therapy was expected to improve mortality but did not improve the outcome Therefore primary angioplasty should be made available to more patients with STEMI. It is the superior to fibrinolytic as initial reperfusion strategy for patients with STEMI.

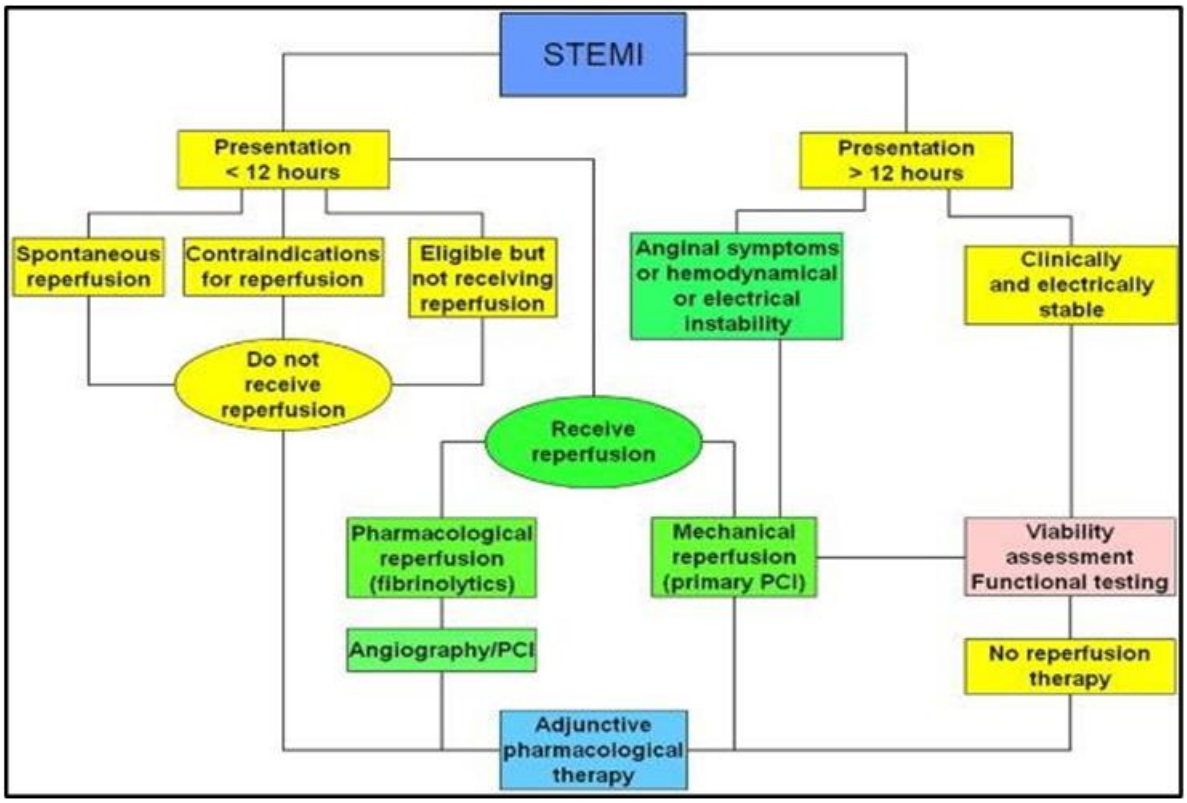

Figure 2: Revascularization Therapy

LV DIASTOLE

LV diastole begins with the closure of aortic valve, which results in drop in LV pressure. The time interval between Av closure and mitral valve opening is the isovolumetric relaxation time. During that time, LV pressure is decreasing while its volume is unchanged (in patients without mitral and aortic regurgitation). Mitral valve opening follows the drop in LV pressure below left atrial pressure. LV filling pressure during the early diastolic filling 18 period occurs as LV relaxation leads to lower LV early diastolic pressures and positive trans mitral pressure gradient. With ongoing LV filling, LA pressure drops and LV pressure rises, leading to decreased trans mitral pressure gradient and reduced LV filling. The rate of decline in early diastolic filling is related to LV stiffness such as that higher LV stiffness leads to faster deceleration of LV filling in late diastole, LA contracts and leads to another positive trans mitral pressure gradient and another peak of LV filling in late diastole.

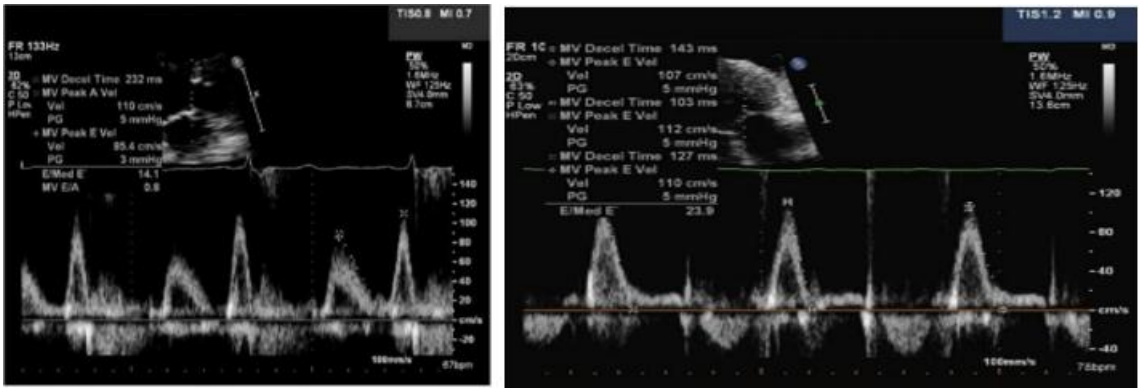

Figure 3: Mitral Inflow Pattern

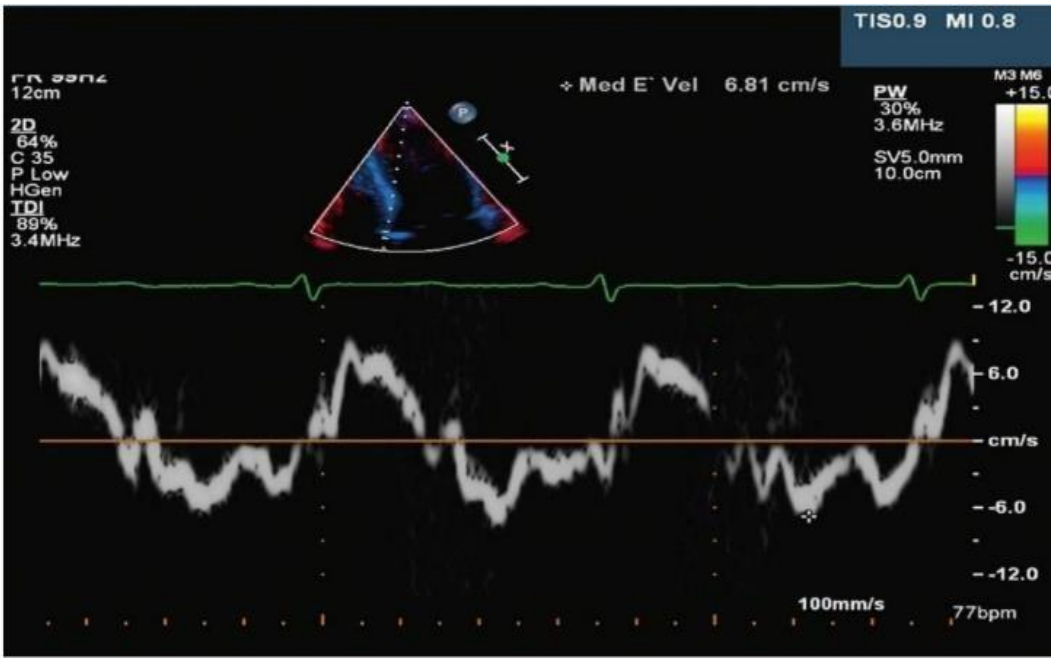

Figure 4: Tissue Doppler velocity of septal wall

LV Relaxation:

Load, inactivation, and asynchrony have an impact on LV relaxation. A decrease in LV desynchrony will result in an improvement in LV relaxation. Desynchrony can be exacerbated and caused by an increase in load. LV STIFFNESS: LV stiffness determine the diastolic volume pressure relationship. It is possible to derive LV operating chamber stiffness (dP /dV) using conductance catheters that simultaneously measure LV volume and pressure. Several factors affect chamber stiffness including LV geometry, myocardial stiffness and factors extrinsic the LV such as pericardial, RV and LV interactions, incomplete relaxation can contribute to elevated LV diastolic pressure for any given volume. Myocardial stiffness is determined by the sarcomere proteins, microtubules and extracellular matrix composition

Doppler Mitral Flow Velocity Patterns:

Doppler measurements of the mitral flow velocity provide unique information about the velocity of blood flow across the mitral valve into ventricle. This velocity is complex function of pressure gradient across mitral valve, described in the law of conservation of energy equation hence flow velocity represents the intermediate link between hemodynamic conditions indicated by instantaneous left atrial and left ventricular pressures and the filling characteristics of the ventricle. Mitral flow velocity variables are recorded from apical four chamber view with pulse wave (PWD) Doppler by placing a 1-3 mm sample volume between the mitral leaflet tips at their narrowest point which is visualized with 2 D echo at end expiration during normal breathing. The Doppler gain and the filter settings should be as low as possible with sweep speed at 50 – 100 mm/sec. Variables that should be measured include peak mitral flow velocity in early diastole (E wave) and during atrial contraction a wave mitral deceleration time (DT), the E wave velocity just before atrial contraction. The duration of mitral A wave velocity (A dur) (sample volume at the mitral annulus level) and isovolumetric relaxation time (IVRT). In young healthy individuals there is a rapid acceleration of blood flow from the left atrium (LA) to the left ventricles (LV) early peak filling velocity of 0.6 to 0.8 m/s occurring 90 to 110 m/s after the onset of mitral valve opening. This E wave occurs simultaneously with the maximum pressure gradient between the LA and LV that in turn depends on the pressure difference along the flow stream, LV relaxation, and the relative compliance of two chambers; normal E wave pattern shows rapid acceleration and deceleration; normal deceleration slope is 4.3 to 6.7 m/s. Mitral DT defined as the time interval from the peak of e wave to its extrapolation to baseline, typically ranges from 150 to 240ms. DT is prolonged in patients with LV relaxation abnormalities because it takes longer for LA and LV pressure to equilibrate. A low normal DT can be seen in normal young subjects in whom there is a vigorous LV relaxation and elastic recoil and short DT if there is a decrease in LV compliance or marked increase in LA pressure as in advanced diastolic dysfunction (DT < 150ms), Early diastolic filling is then often followed by a variable period of minimal flow (Diastasis). The duration of diastasis is dependent on heart rate, it is longer with slow heart rates and entirely absent with faster heart rates. A wave which is the result of an atrial kick pushing the remaining blood from LA to LV follows diastasis and is influenced by LV compliance and LA contractility. The normal A wave velocity typically ranges from 0.19-0.35m/s and is significantly smaller than the E wave resulting in E/A ratio greater than 1. Sinus tachycardia premature atrial contraction and first-degree atrioventricular block may result in fusion of E and A waves. The peak A wave in fused E and A velocity with an E at A wave velocity greater than 20cm/s is larger than it would have been at a slower heart rate, when mitral flow velocity has time to decrease before atrial contraction. E/A wave ratio may reduce, compared with values obtained at slower heart rate, so that more reliance on the Doppler variables is needed when interpreting the fused LV filling pattern. With aging the LV relaxation takes longer primarily because there is a gradual increase in systolic blood pressure and LV mass, resulting in reduced LV filling in early diastole and increased filling at atrial contraction.

DT and IVRT become longer with age and atrial contraction contributes up to 35 to 40% of LV diastolic stroke volume. With progressively worsening diastolic function, trans mitral flow evolves in recognized pattern.

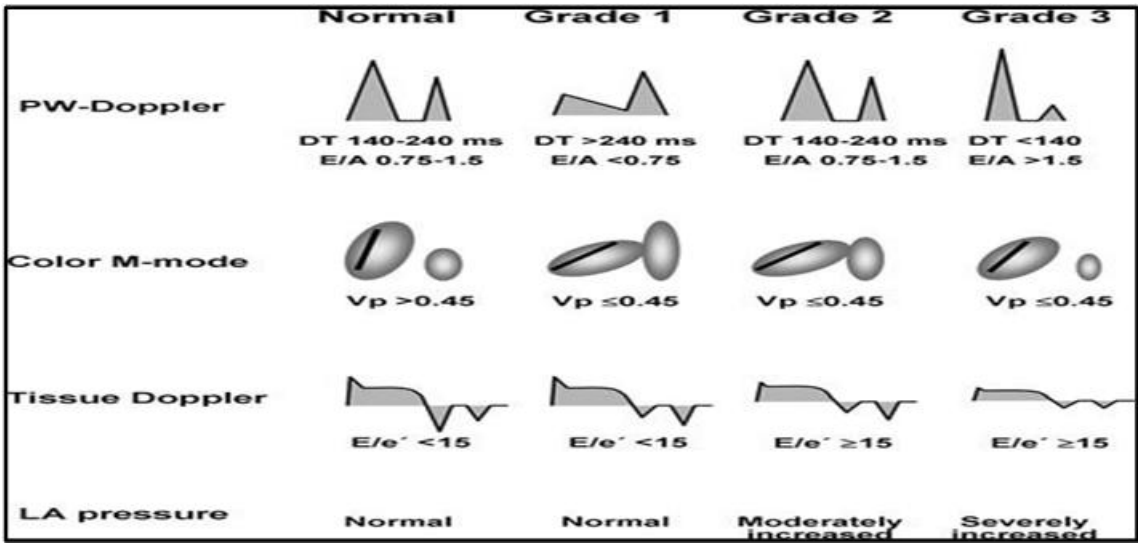

Figure 5: Diagrammatic representation of diastolic grades

Pulmonary Venous Flow:

Accurate pulmonary vein (PV) flow velocity can be obtained from apical 4ch view with Pulse wave Doppler the right upper PV is the most frequently visualized and accessible from transthoracic echocardiographic examination.to properly obtain the pulmonary vein flow with Doppler the sample volume should be placed approximately 1 to 2 cm into pulmonary vein with box size adjusted to 3 to 4mm, Doppler filter set to 200Hz and sweep speed adjusted to 50 to 100mm/sec The flow from PV to RA occurs in three phases: antegrade systolic, antegrade diastolic and retrograde following atrial contraction. The pulmonary venous waveforms are triphasic or Quadri phasic. In 70% of patients, it is difficult to discriminate between the two systolic components of LV flow .in patients with low filling pressure, systolic forward flow becomes biphasic and PV flow pattern is Quadri phasic. 22 The last phase is peak reverse flow velocity at atrial contraction (PVa or PV AR). It occurs in late diastole and influenced by late diastolic pressures in LV, atrial preload and LA contractility. The first phase is early systolic (PVs1) → it occurs in early systole and represents the increase in pulmonary venous flow secondary to atrial relaxation. The second phase, late systolic (PVs2) →occurs in mid to late systole .it is caused by increase in pulmonary venous pressure propagated through the pulmonary arterial tree from right side of the heart. The apical systolic annular motion of the mitral annulus is also believed to contribute to this finding. This phase is early diastole (PVd) which occurs during ventricular relaxation phase and is influenced by LV filling.

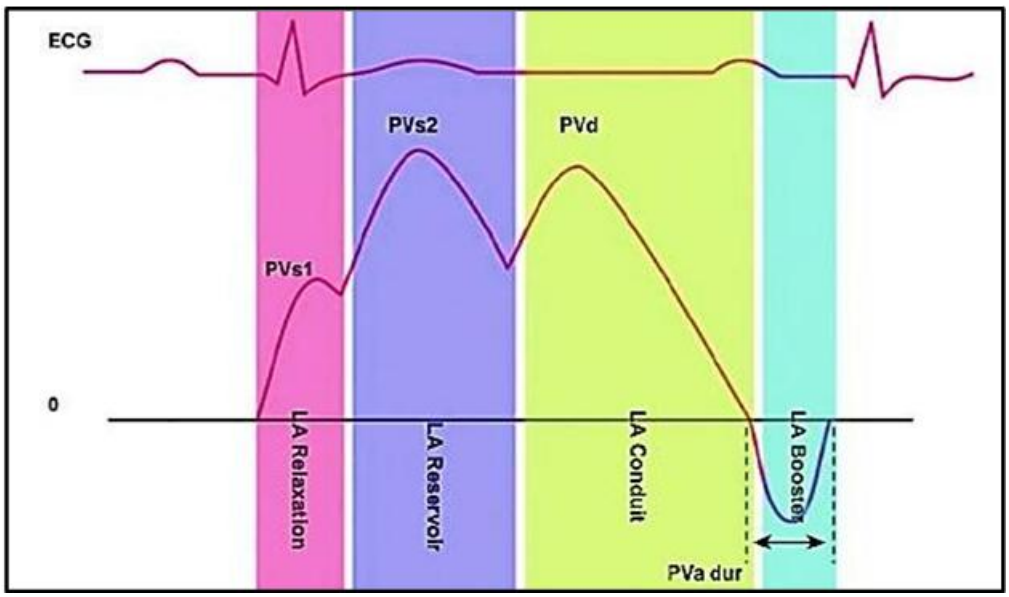

Figure 6: Pulmonary venous flow pattern

Diastolic Dysfunction in STEMI

Acute coronary occlusion is accompanied by significant diastolic dysfunction and elevated filling pressures. Clinically this is manifested as frequent development of acute pulmonary edema in STEMI patients as a result of elevated LA pressures. Severe diastolic dysfunction and elevated filling pressures during STEMI results in immediate improvements were noted in LVEDP, end –

diastolic compliance, stiffness and wall stress were noted post revascularization. The changes in LV diastolic performance and pressures could be tracked using conventional Doppler and tissue –Doppler parameters. Severe diastolic dysfunction is represented by restrictive mitral inflow pattern with an increased E wave velocity. This is accompanied by significantly reduced E' velocities, hence the E/E' ratio is increased and reflects the elevated filling pressures. In stable patients the diastolic function evolves over weeks to months. Early abnormalities in relaxation are followed by changes in compliance/ stiffness over a period of time. mirroring these changes are alterations in both LA size and function in response to left ventricular diastolic changes. In the setting of anterior wall myocardial infarction, onset of significant diastolic dysfunction and elevation of filling pressures occurs rapidly. The effect of these acute changes on LA size and function quantified but LA size likely does not enlarge in acute setting. A second considerations that affects the TDI indices relates to the regional nature of the ischemic process. Tissue velocity during acute infarction depends on whether or not it is in an infarcted segment, where the E' may be expected to be reduced or in an adjacent segment to the infarct where E' may in fact be elevated due to compensatory hyperkinesia. Grade III and Grade IV are associated with poor outcomes in the setting of anterior wall Myocardial Infarction.

2. Methods

A prospective observational study, to assess Left Ventricular Diastolic dysfunction using 2D echocardiography in patients with revascularized Acute Anterior wall MI. A total of 120 patients who presented with acute anterior wall MI has been assessed for Left Ventricular diastolic dysfunction after Revascularization, by using 2D echocardiography.

We have included patients aged between 30-60 years of age, who were diagnosed with Acute anterior wall MI with no past comorbid conditions.

We have excluded patients with previous history of ischemic heart disease, known comorbidities such as Diabetes, Systemic hypertension, chronic kidney diseases and Dyslipidaemia on medical management, pregnant female, patients with cardiogenic shock and hemodynamic instability, arrhythmias.

In 100 consecutive patients with Acute Anterior wall MI Post revascularization using conventional Echocardiography 2D, Pulse wave Doppler, Continuous wave Doppler will be performed using Vivid S5 GE machine in Department of Cardiology. Continuous wave Doppler records maximum transmittal velocity and tissue Doppler records the systolic septal and lateral annular wall motion.

3. Results

Table 2: Patient demography

Demographic data		Subjects	Percentage
Gender	Male	69	57.50%
	Female	51	42.50%
Age	30-40 y	40	33.30%
	41-50 y	28	23.30%
	51-60y	52	43.40%

Table 3: Revascularization procedure for Study Population

Procedure	Number of Subjects	Percentage
Fibrinolysis	33	27.5%
Primary Angioplasty	60	50%
Coronary Artery Bypass Graft	27	22.5%

Table 4: Parameters for Diastolic dysfunction in study population

Parameter	Group 1	Group 2
E/A	1.2±0.23	1.6±0.44
DT	169±11	119±35
E/E'	7.3±0.8	19±4.2
SEPTAL E'	7.6±0.2	4.3±0.1
IVRT	66±10.9	47±0.9
S/D RATIO	0.5±0.02	0.4±0.00

Table 5: Grades of Diastolic Dysfunction in Study Population

	Group 1		Group 2	
Diastolic Dysfunction	Grade 1	Grade 2	Grade 3	Grade 4
Number of Subjects	24	20	59	17
Percentage	36.6% (44)		63.3% (76)	

4. Discussion

In the present study 120 subjects were included, among them 69(57.5%) were males and 51(42.5%) females. 40 subjects (33%) were between age group 30-40years, 28 subjects (23.3%) were above 41-50 years of age and 52subjects (43.4%) were aged between 51-60 years of age, depicted in Table 2.

Jan pyszko et al in 2019 in their study with 999 subjects concluded that Echocardiographic parameters of diastolic function physiologically deteriorate with age, as in our study we have seen more individuals of age group 51-60 years have severe diastolic dysfunction.[1][2] Diabetes causes diastolic dysfunction and is highly prevalent in patients with Diabetes and seems to be positively correlated with HbA1c level, obesity, dyslipidemia and the duration of diabetes [3][4][5]. In our study since diabetes affects diastolic dysfunction, we have excluded Diabetic patients from our study.

Left ventricular diastolic dysfunction (LVDD) is common in hypertensive individuals and is associated with increased morbidity and mortality. Hypertension is the leading etiology for diastolic dysfunction, which is ubiquitous in elderly individuals and contributes to the development of heart failure.[6][7] In our study we have excluded hypertensive patients, since hypertension is a confounding factor which will affect the true statistics of our study.120 patients who presented with Acute Anterior wall MI underwent Revascularization in our hospital. Among them, 60(50%) underwent Primary Angioplasty, 33(27.5%)

underwent Fibrinolysis and 27(22.5%) underwent CABG, which is depicted in Table 3.

We have divided the 120 patients into two Groups depending on their Diastolic Function. Group 1 included Grade 1 and 2 Diastolic Dysfunction and Group 2 patients had Grade 3 and 4 Diastolic dysfunctions. The mean parameters of Diastolic dysfunction are depicted in Table 4.

In our study, 76(63.3%) subjects have severe Diastolic Dysfunction (elevated filling pressure (E/A ratio), reduced tissue Doppler E/e' ratio, IVRT and reduced S/D ratio), which are significantly higher in number and suggest that Diastolic dysfunction can be a crucial determinant in predicting the outcome of MI.

Nesto et al in his study showed that Diastolic dysfunction occurs early in the ischemic cascade.[8][9] Diastolic dysfunction is first to develop followed by systolic dysfunction but the recovery happens in the opposite direction, with systolic dysfunction recovering earlier than diastolic dysfunction. Hence, assessing early diastolic dysfunction is important for predicting future outcomes of MI. Despite the recovery of LV systolic function after the index AMI, LV diastolic function had not improved by the 6-month follow-up in a significant proportion of patients. Impaired diastolic functional recovery was an independent predictor of MACE after AMI. [10][11][12][13] In our study, we faced this major limitation as we could not follow up the patients to assess the status of diastolic function.

5. Conclusion

LV diastolic function evaluation is crucial in acute MI assessment. Doppler echocardiography provides key parameters, Mitral inflow E/A ratio, Tissue Doppler E/e', Isovolumetric relaxation time, Deceleration time. These parameters help stratify patients' risk of MI. Furthermore, LV diastolic dysfunction is common in acute MI patients, even after successful percutaneous coronary intervention (PCI). Diastolic dysfunction significantly increases MI risk, with higher grades of dysfunction associated with greater risk. Accurate assessment of LV diastolic function is essential for identifying patients at higher risk of adverse outcomes

6. Limitations

There are several limitations in our present study

1) We could not compare the severity of diastolic dysfunction in patients presenting with Acute Anterior wall MI before and after revascularization.
2) Lack of follow-up study to assess the severity of diastolic dysfunction over time, since serial monitoring of diastolic function over a period of at least 6 months would be useful in predicting future clinical adverse event in patient with Acute MI.
3) We did not assess whether there was any advantage of early intervention in development of diastolic dysfunction and its future adverse events in terms of severity.
4) It is a single-centered conducted study with limited subjects

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