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Relation between Myocardial Blush Grade and Left Ventricular Systolic and Diastolic Function after Primary Percutaneous Coronary Intervention in Patients with Type 2 Diabetes Mellitus

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ABSTRACT

Background One of the main risk factors for coronary artery disease (CAD) is diabetes mellitus. The myocardial wall function can be Doppler objectively analyzed with tissue imaging (TDI) echocardiography, which can also provide insight into the genesis of heart illness. The aim of this study was to investigate the predictive potential of myocardial blush grade and the tissue Doppler echocardiographymeasured E/(e'×s') ratio for left ventricular remodeling in patients with type 2 diabetes who had primary percutaneous intervention for a ST segment elevation myocardial infarction. Methods: A total of one hundred patients with ST-elevation myocardial infarction (STEMI) who were admitted to the cardiology department of Zagazig University Hospitals participated in this prospective study; fifty of them had diabetes, while the other fifty did not. Trans mitral Doppler early and late diastolic velocities (E and A waves). The septal, lateral, average early diastolic (e'), peak systolic (s') using tissue Doppler mitral annulus velocities were measured. Using visual assessment procedures, two experienced interventional cardiologists were trained to determine the Myocardial blush grade (MBG) and TIMI flow score while being blinded to the data. **Results:** Individuals with a greater $E/(e' \times s')$ ratio can predict systolic dysfunction with a cut-off value of >1.63 and diastolic dysfunction with a cut-off >1.68. Myocardial blush grade and TIMI flow were significantly better in non-diabetic group (P value <0.05).80% of non-diabetic achieved TIMI flow III score and 84% of them showed better myocardial blush (grad 3). Conclusion: In patients with type 2 uncontrolled diabetes, the TDI $E/(e' \times s')$ is a quick and easy way to measure LV diastolic and systolic dysfunction. In these individuals, it might be used to forecast heart failure. Keywords: Type 2 diabetes mellitus; Acute myocardial infarction; LV dysfunction; Tissue Doppler imaging.

INTRODUCTION

Patients with diabetes are at increased risk of developing cardiovascular disease (CVD) with its manifestations of coronary artery disease (CAD), heart failure, atrial fibrillation and stroke .[`] Examining the risk factors prevalent in Egyptian society that have contributed to the creation of this expanding issue is crucial, as diabetes mellitus (DM) is regarded as a serious public health concern in Egypt. Considering the rising prevalence of diabetic mellitus (DM), efficient management techniques are required. Poor eating habits, sedentary lifestyles, and obesity are the main causes of diabetes mellitus. Preventive cardiology represents a unique and significant concept in the management of diabetes, with the aim of evaluating cardiovascular risk and preventing potentially fatal cardiovascular events that are commonly observed in people with diabetes.[^Y]

When admitted for treatment for acute myocardial infarction (AMI), over thirty percent of patients did not have diabetic mellitus (DM), even though DM is associated with a higher risk of heart disease[3]. Diabetes mellitus patients have a two-to four-fold increased risk of dying from cardiovascular disease (CVD) as compared to healthy people without the condition.[ξ]

The myocardial wall performance can be objectively analyzed using tissue Doppler imaging (TDI) echocardiography, which can also shed light on the pathogenesis of heart disease. This noninvasive imaging technique measures myocardial velocities at different points during the cardiac cycle. Since it is independent of the reflected wave's amplitude, myocardial wall motion information can be obtained from regions where 2-D echocardiography's grayscale data may not be sufficient.[°]

It's unclear how diastolic function and systolic performance relate to one another. It is conceptually very difficult to distinguish between contraction and relaxation; therefore, it is preferable to think of them as one continuous cycle. Mitral annular velocities are a reflection of the ventricle's long axis motion, which is a crucial component of LV systolic and diastolic function. In conditions such as congestive heart failure, abnormalities in the diastolic and systolic phases affect the left ventricular filling pressure at different levels.[¹]

Numerous publications claim that following an acute myocardial infarction, the e' velocity and the ratio (E/e') of the early transmitral flow velocity to the early diastolic mitral annulus velocity are significantly predictive of worse outcomes. Using the s' wave is one method of quantifying the entity of a regional mobility limitation. This velocity is related to the longitudinally orientated cardiac fibers in the sub-endocardium, which are the most susceptible to ischemia.[^Y]

Previous work demonstrated a novel TDI index, $E/(e'\times s')$, using the idea of a complex marker that combines numerous tissue Doppler properties. an indication that connects the diastolic function marker, $E/(e'\times s')$, and a measurement of LV systolic performance.

Aim of the work

The aim of this study is to assess the relationship between myocardial blush grade and left ventricular systolic and diastolic function as measured by

in patients who present with ST elevation acute myocardial infarction in type 2 diabetes after primary percutaneous intervention.

METHODS

conventional and tissue Doppler echocardiography

One hundred patients with ST-elevation myocardial infarction (STEMI) were included in this prospective study; fifty of the patients had diabetes, while the other fifty did not. Between April 2023 and May 2024, the patients were admitted to the Sixth of October Hospital for Health Insurance, Zagazig University Hospitals, and the Cardiology Department. Each patient provided written informed permission, and Zagazig University (IRB #10606) authorized the study. The study was carried out in compliance with the Declaration of Helsinki, the global medical association's code of ethics for human subjects' research .

Inclusion criteria: Patients with sustained STsegment elevation (or equivalents) and acute chest pain (or symptoms and signs comparable to chest discomfort) on ECG were among those who arrived within a 12-hour window [8]. Exclusion criteria: Acute inflammatory diseases, cancer, congenital heart disease, hematological disorders, recent chemotherapy history, fever, pregnant women, poor image quality, hepatic failure, acute inflammatory diseases, hepatic failure, and patients with a history of coronary artery bypass graft (CABG) or PCI are some of the conditions that could be causing this variation Every patient underwent я comprehensive clinical examination, which included vital sign monitoring, a general and heart examination, in addition to demographic data collection on height, weight, age, sex, and body mass index (BMI). Testing for liver and renal function, lipid profiles, complete blood counts (CBCs). cardiac biomarkers, and glycated hemoglobin were among the laboratory tests performed (HbA1c).

ECG: If left ventricular hypertrophy or left bundle branch block are absent, there is a new ST elevation at the J-point in at least two contiguous leads. For leads V2–V3, this elevation has a value of at least 1.5 mm, and/or 1 mm for the other leads; for men 40 years of age or older, it is ≥ 2 mm, and for those under 40, it is ≥ 2.5 mm [LBBB].[\wedge]

Transthoracic echocardiography was performed on each patient both on the first day of hospitalization and 12 weeks following the operation. A skilled echocardiographer preserved all the images on a hard drive. This is how the global LV systolic function was evaluated: 2-D for the modified Simpson technique that was applied to compute LVEF in the two and four chamber apical views. ((LVEDV-LVESV)/LVEDV). LV The biplane method was used to calculate the left atrial (LA) volume, and the LA volume index (LAVI) was subsequently indexed to the BSA.[⁹]

Wall motion abnormality and score index (WMSI) was determined and characterized as follow; being akinetic = 3, dyskinetic = 4, hypokinetic = 2, and normal = 1. The whole of each segment's wall motion scores is divided by 17 to determine the index (WMSI) .[\uparrow ,]

The metrics employed to evaluate the left ventricular diastolic function were the projected E/A ratio, the isovolumetric relaxation time (IVRT). the E wave deceleration time (DT), and the transmitral Doppler early and late diastolic velocities (E and A waves). Measured mitral annulus velocities included septal, lateral, peak systolic (s'), average early diastolic (e'), and tissue Doppler. E/mean e' was utilized to obtain E/ey, and (e' septal + e' lateral)/2 was applied to compute mean e'. The end-expiration numbers that are shown reflect three heartbeats on average. Together with the pulsed Doppler E/A (septal and lateral mitral annulus as well as the average), the tissue Doppler E/e' and $E/(e' \times s')$ ratios were calculated Four criteria are specifically mentioned in the 2016 guidelines from the European Association of Cardiovascular Imaging/American Society of Echocardiography (EACVI/ASE) to diagnose diastolic dysfunction: an annular e' velocity (septal e' <7 cm/s, lateral e' <10 cm/s), a maximum left atrial (LA) volume index of >34 mL/m2, a peak tricuspid regurgitation velocity (TRV) of >2.8 m/s, an average E/e' ratio of >14, and a maximum left atrial (LA) volume index of >34 mL/m2.[\\]

Another method to assess systolic and diastolic functions simultaneously is the myocardial performance index, which includes both systolic and diastolic time intervals to show global systolic and diastolic ventricular function $[1^{3}]$ Systolic dysfunction results in a shortened ejection time and an extended isovolumic contraction time (IVCT). The aberrant myocardial relaxation caused by both systolic and diastolic dysfunction lengthens the isovolumic relaxation time (IVRT). According to reports, the MPI or Tei Index is a trustworthy technique for assessing left ventricular function $[1^{3}]$

Angiographic evaluation: Standard femoral or radial approaches were used to perform coronary angiography (CA). Revascularization Strategy: Primary PCI procedures were used on all patients. Each patient received a loading dose of 180 mg of ticagrelor and 300 mg of aspirin prior to primary angioplasty. With the Xience Prime coronary stent system, all patients had coronary stenting, and there was no discernible residual stenosis in the artery connected to the infarct.

The MBG and TIMI flow score were evaluated as follows: When there is no antegrade flow after a cardiac blockage, it is referred to as "TIMI 0 flow" or no perfusion. TIMI 1 flow (penetration without perfusion) is a weak antegrade coronary flow that, after overcoming an obstruction, partially fills the distal coronary bed. When the distal area fills entirely, antegrade flow slows down or is delayed, resulting in incomplete reperfusion, or TIMI 2 flow. In TIMI 3, the distal coronary bed is fully filled, indicating normal flow.[12]

The operator's assessment of the degree of contrast opacification of the myocardium supplied by the infarct-related artery (IRA) in relation to its provision of epicardial density is known as the myocardial blush grade (MBG).[¹°] MBG 0: The impacted myocardium does not exhibit contrast opacification. MBG1: A slight opacification or enduring staining is observed. MBG2: A decreased myocardial blush in the region of the infarct relative to the unaffected areas. MBG3: The dye's passage via the microvasculature is normal.[¹]

Following training and data blinding, two seasoned interventional cardiologists visually evaluated the TIMI flow score and the myocardial blush grade (MBG). The outpatient clinic conducted a clinical examination and echocardiography as part of the follow-up following three months of PCI.

One hundred individuals with ST-elevation myocardial infarctions (STEMIs) made up the study

population. Two groups of patients were formed:

group A (n = 50) had diabetes, and group B (n = 50) did not.

Statistical analysis SPSS v26 was utilized by IBM Inc., Chicago, IL, USA, for statistical analysis. According to Von Bibra et al., quantitative data were presented as mean and standard deviation, and the unpaired Student's t-test was used to compare the two groups. Chi-square was used to analyze the frequency and percentage (%) of the qualitative variables. Univariate and multivariate for predictor of improved LV function, low TIMI score, and improper myocardial blush was performed and for odds ratios. ROC analysis for the best cutoff values of different parameters for sensitivity and specificity. Hazard ratio and Odds ratio were evaluated as the predictor for LV dysfunction. If the two-tailed P value of a result was less than 0.05, it was deemed statistically significant.

Results

Clinical and demographic data showed no appreciable difference between the two groups (table 1). On the first day of hospitalization, the diabetic group's EF, s, and e were significantly lower (P value <0.05). The diabetes group had significantly increased E-wave velocity, LAVI, E/A ratio, E/e', E/(e'×s'), and WMSI (P value <0.05) in comparison to the non-diabetic group. Between the two groups, there was no appreciable variation in MPI (Table 2). At the 3-month follow-up, EF and MPI did not differ substantially, but the diabetes group still had significantly greater LAVI, E/A, E/e', and E/(e'×s') values (P value <0.005). In the diabetic group, EF, WMSI, E wave velocity, s, and e-velocity are still lower (Table 3).

		Diabetic group (n=50)	Non-diabetic group (n=50)	P value
$\Lambda q_{0} (v_{0})$	Mean \pm SD	59.84 ± 10.31	56.14 ± 9.26	0.062^{t}
Age (years)	Range	42 - 89	39 - 86	0.002
Sev	Male	31 (62%)	36 (72%)	0.288 ^{x2}
SCA	Female	19 (38%)	14 (28%)	0.288
Height (m)	Mean \pm SD	172.42 ± 4.25	174.2 ± 5.45	0.072^{t}
fieight (iii)	Range	160 - 181	157 – 182	0.072
Weight (kg)	Mean \pm SD	85.48 ± 8.31	83.38 ± 9.68	0.247^{t}
weight (kg)	Range	65 - 110	60 - 100	0.247
$\mathbf{PSA}(\mathbf{m}^2)$	Mean \pm SD	2.05 ± 0.13	2 ± 0.13	0.064^{t}
DSA (III)	Range	1.67 - 2.31	1.64 - 2.17	0.004
BMI (kg/m^2)	Mean \pm SD	28.82 ± 3.28	27.52 ± 3.52	0.059 ^t
Divit (kg/iii)	Range	22.5 - 36	22.1 - 38.5	0.039
Family History of CAD		14 (28%)	12 (24%)	0.648 ^{x2}
Hypertension		27 (54%)	33 (66%)	0.221^{x^2}
Dyslipidemia		28 (56%)	24 (48%)	0.423^{x^2}
Smoker		38 (76%)	42 (84%)	0.317 ^{x2}
HR (beats/min)	Mean \pm SD	89.52 ± 11.54	85.7 ± 8	0.057 ^t
	Range	69 - 120	65 - 97	
SBP (mmHg)	Mean \pm SD	125.5 ± 19.46	123.8 ± 15.1	0.627 ^t
	Range	70 - 150	100 - 150]
DBP (mmHg)	Mean \pm SD	78.52 ± 10.4	77.44 ± 8.96	0.579 ^t
	Range	60 - 105	60 - 90]

Table 1: Patient characteristics of the study population

HR: Heart rate, SBP: Systolic blood pressure, DBP: Diastolic blood pressure. CAD: Coronary artery disease. BSA: Body surface area, BMI: Body mass index, t: Unpaired student's t-test, x^2 : Chi-square test.

		Diabetic group (n=50)	Non-diabetic group (n=50)	P value	
EF (%)	Mean ± SD	45.08 ± 9.01	48.66 ± 10.44	<0.001* t	
	Range	(27 – 59)	(35 - 66)		
LAV (ml)	Mean \pm SD	68.46 ± 27.25	55.89 ± 10.17		
	Range	45 - 109	29 - 79	<0.001*1	
LAVI (ml/m2)	Mean \pm SD	32.64 ± 33.64	26.11 ± 7.18		
	Range	25 - 45	13.82 - 40.3	0.004* '	
E-velocity (cm/s)	Mean \pm SD	72.37 ± 16.96	65.65 ± 11.11	· · · •	
	Range	43.4 - 104	40.24 - 89.3	0.021**	
A-velocity (cm/s)	Mean \pm SD	59.21 ± 19.8	64.08 ± 13.25	0 151 ^t	
	Range	(27.3 - 90.43)	(34.2 - 92.1)	0.151	
E/A ratio	Mean \pm SD	1.47 ± 0.69	1.09 ± 0.39		
	Range	0.14 - 2.67	0.55 - 2.24	<0.001* ^t	
e` lateral (cm/s)	Mean \pm SD	7.79 ± 1.76	8.52 ± 1.77	0.04 3 * ^t	
	Range	(4 - 10.54)	(4.95 - 12.4)	0.043	
e` septal (cm/s)	$Mean \pm SD$	6.96 ± 1.55	7.59 ± 1.5	0.041* ^t	
	Range	(3.2 - 9.78)	(4.74 - 11.76)		
s`lateral (cm/s)	Mean \pm SD	6.5 ± 1.62	7.43 ± 1.42	0.003* ^t	
	Range	(4.17 – 14)	(4.35 - 10.46)	0.005	
E/ (e'*s') ratio- lateral	Mean \pm SD	1.66 ± 0.8	1.42 ± 1.46	<0.001* ^t	
	Range	(0.09 – 3.54)	(0.46 - 2.59)		
s` septal (cm/s)	Mean \pm SD	6.14 ± 1.52	6.83 ± 1.3	0.016* ^t	
	Range	(4 - 13.4)	(3.96 - 9.2)	0.010	
E/ (e'*s') ratio- septal	Mean \pm SD	1.94 ± 0.85	1.39 ± 0.55	<0.001* ^t	
	Range	(0.11 - 4.01)	(0.7 - 2.83)	<0.001	
E/ (e'*s') ratio-average	Mean \pm SD	1.66 ± 0.77	1.29 ± 0.51	0.005* ^t	
	Range	(0.35 - 3.82)	(0.64 - 2.63)	0.003	
WMSI	Mean \pm SD	1.8 ± 0.5	1.54 ± 0.39	0.005*t	
	Range	(1 - 2.75)	(1 - 2.6)	0.005	
LV MPI	Mean \pm SD	0.75 ± 0.35	0.62 ± 0.17	0 722 ^t	
	Range	(0.28 - 53)	(0.11 - 41)	0.755	

Table 2: Transthoracic echocardiography measurements during first day of admission of the studied groups

*Significantly different as P value ≤ 0.05 , EF: Ejection fraction, LAV: Left atrial volume, LAVI: Left *Significantly different as P value ≤ 0.05 , EF: Ejection fraction, LAV: Left atrial volume, LAVI: Left atrial volume index, E- velocity early diastolic mitral inflow velocity, A-velocity: Late diastolic mitral inflow velocity, e`: early diastolic mitral annulus velocity, WMSI: Wall motion score index, MPI: Myocardial performance index, LVESV: Left ventricle end systolic volume, LVEDV: Left ventricle end diastolic volume, t: Unpaired student's t-test.

		Diabetic group (n=50)	Non-diabetic group (n=50)	P value	
EF (%)	Mean ± SD	44.61 ± 8.54	56.47 ± 8.07	<0.001* ^t	
	Panga	(31 56)	(30 62)		
LAV (ml)	Mean + SD	(31 - 30) 74.94 + 13.75	(30 - 02)		
	Range	(66 - 112)	(35 - 88)	0.049^{*t}	
LAVI (ml/m2)	Mean + SD	35 5 + 12 9	$(33 \ 63)$		
	Range	(20-41)	(13.82 - 40.3)	0.034^{*t}	
E-velocity (cm/s)	Mean + SD	77 16 + 13 18	70.51 ± 14.87		
L-velocity (cill/3)	Range	(40.25 - 87.1)	(50 - 1065)	0.039^{*t}	
A-velocity (cm/s)	Mean + SD	67.32 + 14.29	64.42 + 11.86		
rivelocity (cill/3)	Range	(40 - 91 3)	(382-95)	0.322^{t}	
F/A ratio	Mean + SD	137 ± 0.41	1.17 ± 0.39		
	Range	(0.47 - 1.77)	(0.72 - 2.26)	0.03^{*t}	
e`lateral (cm/s)	Mean ± SD	6.44 ± 2.08	9.46 ± 1.98	<0.001* ^t	
	Range	(4.25 - 10.1)	(6.5 - 12.5)	(0.001	
E/e' ratio (lateral)	Mean ± SD	9.66 ± 2.72	7.77 ± 2.35	0.001.tt	
	Range	(4.43 - 13.94)	(4 - 14.96)	0.001**	
e` septal (cm/s)	Mean ± SD	6 ± 1.59	8.73 ± 1.28	.0.001*t	
	Range	(3.87 - 8.7)	(6.05 - 11)	<0.001**	
E/e' ratio (septal)	Mean ± SD	10.08 ± 2.9	8.31 ± 2.39	0.002*t	
	Range	(4.97 - 14.84)	(4.55 - 15.39)	0.003**	
s`lateral (cm/s)	Mean ± SD	5.27 ± 1.1	8.86 ± 1.43	-0.001* ^t	
	Range	(4.5 - 9.32)	(3.1 - 8.6)	<0.001*	
E/(e'*s') ratio- lateral	Mean ± SD	2.6 ± 1.04	1.23 ± 0.62	<0.001* ^t	
	Range	(0.91 - 4.55)	(0.47 - 3.45)	<0.001	
s` septal (cm/s)	Mean ± SD	5.08 ± 1.16	7.69 ± 1.28	<0.001* ^t	
	Range	(3 - 7.57)	(4.2 - 9.8)	<0.001*	
E/(e'*s') ratio- septal	Mean \pm SD	2.9 ± 1.29	1.14 ± 0.48	<0.001* ^t	
	Range	(1.05 - 5.6)	(0.49 - 2.74)		
E/(e'*s') ratio-average	Mean ± SD	2.17 ± 0.47	0.97 ± 0.28	.0.001*t	
	Range	(1.53 - 3.2)	(0.57 - 1.75)	<0.001**	
WMSI	Mean ± SD	1.84 ± 0.33	1.21 ± 0.23	0.001.41	
	Range	(1.5 - 2.43)	(1 - 2.13)	<0.001*`	
LV MPI	Mean ± SD	0.54 ± 0.21	0.48 ± 0.17	0.12ct	
	Range	(0.22 - 1.09)	(0.13 - 0.84)	0.136	

Table 3: (3-month) Follow up transthoracic echocardiography measurements of the studied groups

Diabetic patients showed worse revascularization outcome indicated by low prevalence & ST segment resolution $\geq 70\%$, moreover had lower TIMI flow score and Myocardial blush grade compare to nondiabetic patients (Table 4). The diabetic group fared worse at the 3-month follow-up, with significantly reduced EF and greater $E/(e'\times s')$. The diastolic and systolic functions were both poorer than the baseline values. The diabetes group displayed significantly lower S and e values (P value <0.05) in comparison to the non-diabetic group. The diabetes group exhibited significantly higher WMSI, E- velocity, LAVI, E/A ratio, and E/e' ratio (P value <0.001) in comparison to the non-diabetic group. (Table 5). With a cut-off value of >1.63, the average $E/(e'\times s')$ ratio may accurately predict systolic dysfunction (P<0.001 and AUC=0.869) with 70.97% sensitivity, 84.06% specificity, 66.7% PPV, and 86.6% NPV. table (6) and figure (1) Average $E/(e'\times s')$ ratio has 80% sensitivity, 74.44%

specificity, 25.8% PPV, and 97.1% NPV, and can strongly predict diastolic dysfunction (P<0.001 and AUC = 0.949) at cut-off >1.68. Figure (2) and Table (7). The myocardial blush grade can be strongly predicted by the average $E/(e'\times s')$ ratio at a cut-off of >1.315 (P<0.001 and AUC = 0.870), with 90.0% sensitivity, 75.71% specificity, 61.4% PPV, and 94.6% NPV (figure (3).

Table 4: ST segment resolution,	TIMI flow score and	myocardial blush	grade of the studied	groups
U ,		2	0	0 1

		Diabetic group (n=50)	Non-diabetic group (n=50)	P value
ST segment resolution≥ 70%	Yes	26 (52%)	42 (84%)	0.001* x2
	Ι	4 (8%)	2 (4%)	
TIMI flow score	II	18 (36%)	8 (16%)	0.036* x2
	III	28 (56%)	40 (80%)	
	Grade 0	2 (4%)	0 (0%)	
Myocardial blush grade	Grade 1	4 (8%)	2 (4%)	0.018* x2
	Grade 2	16 (32%)	6 (12%)	0.010 A2
	Grade 3	28 (56%)	42 (84%)	

*Significantly different as P value <0.05, TIMI: Thrombolysis in myocardial infarction, x2 : Chi-square test.

ble 5 : Transthoracic echocardiograph y measurements in diabetic group

		Baseline group (n=50)	Follow up group (n=50)	P value
EF (%)	Mean \pm SD	45.08 ± 9.01	44.61 ± 8.54	0.001* t
	Range	(27 – 59)	(31 – 56)	
$\mathbf{I} \mathbf{A} \mathbf{V} (\mathbf{m})$	Mean \pm SD	68.46 ± 27.25	74.94 ± 13.75	0.027* t
	Range	45 - 109	(66 – 112)	0.027 t
I A V I (m1/m2)	Mean \pm SD	32.64 ± 33.64	35.5 ± 12.9	0.043* t
LAVI(IIII/III2)	Range	(25 - 40)	(20-41)	0.045 · 1
E valocity (cm/s)	Mean \pm SD	72.37 ± 16.96	77.16 ± 13.18	0.04* t
E-velocity (cm/s)	Range	(43.4 – 104)	(40.25 - 87.1)	0.04 1
A valacity (cm/s)	Mean \pm SD	59.21 ± 19.8	67.32 ± 14.29	0.262 t
A-velocity (cill/s)	Range	(27.3 - 90.43)	(40 - 91.3)	0.202 t
E/A ratio	Mean \pm SD	1.47 ± 0.69	1.37 ± 0.41	0.066 t
E/A latio	Range	(0.14 - 2.67)	0.47 - 1.77	0.900 t
a`lataral (am/s)	Mean \pm SD	7.79 ± 1.76	6.44 ± 2.08	
e lateral (cli/s)				<0.001* t
	Range	(4 - 10.54)	(4.25 - 10.1)	
E/e' ratio (lateral)	Mean \pm SD	9.16 ± 3.44	9.66 ± 2.72	<0.001* t
	Range	(0.81 - 16.73)	4.43 - 13.94	
a contal (am/a)	Mean \pm SD	6.96 ± 1.55	6 ± 1.59	

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		Baseline group (n=50)	Follow up group (n=50)	
	Range	(3.2 - 9.78)	(3.87 - 8.7)	<0.001* t
E/a' ratio (sontal)	Mean \pm SD	10 ± 3.62	10.1 ± 2.9	
E/e Tatio (septar)	Range	(0.94 - 17.83)	4.97 - 14.84	0.002* t
s`lateral (cm/s)	Mean \pm SD	6.5 ± 1.62	5.27 ± 1.1	<0.001* t
s lateral (eni/s)	Range	(4.17 – 14)	(4.5 - 9.32)	
E/(e'*s') ratio- lateral	Mean ± SD	1.66 ± 0.8	2.6 ± 1.04	<0.001* t
	Range	(0.09 - 3.54)	(0.91 - 4.55)	<0.001 t
s`sental (cm/s)	Mean \pm SD	6.14 ± 1.52	5.08 ± 1.16	
s septar (eni/s)	Range	(4 - 13.4)	(3 - 7.57)	<0.001* t
E/(e'*s') ratio- septal	Mean \pm SD	1.94 ± 0.85	2.9 ± 1.29	<0.001* t
	Range	(0.11 - 4.01)	(1.05 - 5.6)	<0.001 t
E/(e'*s') ratio-average	Mean ± SD	1.66 ± 0.77	2.17 ± 0.47	<0.001* t
	Range	(0.35 - 3.82)	(1.53 - 3.2)	<0.001 t
WMSI	Mean ± SD	1.8 ± 0.5	1.84 ± 0.33	<0.001* t
	Range	(1 - 2.75)	(1.5 - 2.43)	\0.001 t
	Mean \pm SD	0.75 ± 0.35	0.54 ± 0.21	
	Range	(0.28 - 53)	(0.22 - 1.09)	0.320 t

* t: Unpaired student's t-test.

Table 6: $E/(e^{2} \times s^{2})$ ratio-average in prediction of systolic dysfunction measured by EF cut point 40%

Cut-off	Sensitivity	Specificity	PPV	NPV	AUC	P value
>1.63	70.97%	84.06%	66.7%	86.6%	0.869	<0.001*

PPV: positive predictive value, NPV: negative predictive value, AUC: area under the curve.



Figure 1: E/(e'×s')ratio-average in prediction of systolic dysfunction

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Figure 2: E/(e'×s')ratio-average in prediction of diastolic dysfunction



Cut-off	Sensitivity	Specificity	PPV	NPV	AUC	P value
>1.68	80%	74.44%	25.8%	97.1%	0.949	<0.001*



Figure3: E/(e'×s')ratio-average in prediction of myocardial blush grade

Discussion

Acute myocardial infarction (AMI) is a medical illness that can be fatal and is characterized by an abrupt onset. The prognosis is intimately correlated with the remodeling of the ventricular geometry in the vicinity of the infarct that occurs after an AMI. The effective treatment for acute myocardial infarction is percutaneous interventional therapy; however, only 30% of patients with severe AMI **El-Dosouky, I., et al** show evidence of ventricular remodeling. Because of this, early detection of those who may undergo LV remodeling after AMI has significant implications for treatment and prognosis [17]. The current study used TDI, which has the characteristics of being easily accessible and reproducible It is extensively employed and proven in the literature to examine myocardial velocities in

order to measure and identify left ventricular systolic and diastolic dysfunction after PPCI in STEMI with T2DM. It's unclear how diastolic function and systolic performance relate to one another. It is preferable to think of relaxation and contraction as a single, continuous cycle because it is conceptually very difficult to distinguish between the two. The long axis motion of the ventricle, which is crucial for both the systolic and diastolic function of the left ventricle, is reflected in the mitral annular velocities. Abnormalities in the diastolic and systolic phases have different impacts on the left ventricular filling pressure in illnesses such congestive heart failure [6]. Based on the demographic data, we were unable to identify any statistically significant differences in gender or age across the research groups. Our results showed that there were no significant differences between the two groups for danger signals, heart rate, SBP, and DBP. LV filling pressure and myocardial damage impact a patient's prognosis when they have STEMI. There is a direct correlation between LA size and the diastolic LV filling pressure. LVEF, LA size, and LV volume indices are valid markers of cardiovascular outcome in ACS patients[18]. $E/(e' \times s')$ is a helpful indicator that evaluates diastolic and systolic parameters and is applicable to positive remodeling evaluation. Compared to other echocardiographic measures, it is simpler to measure. E' and s' velocity measurements can reveal information regarding myocardial perfusion in an oblique manner. $E/(e' \times s')$) is a robust predictor of negative prognosis in a number of cardiac illnesses, regardless of LVEF, according to research by Mornos C et al. Subclinical alterations in LV systolic or diastolic function are brought on by ischemia [6]. Ionac et al. 307 consecutive hospitalizations, $E/(e' \times s')$ prognostic value was

assessed before discharge and six weeks later in

patients with NSTE-ACS and successful PCI. The primary finding, defined as cardiac mortality or

readmission due to myocardial infarction or failure, indicated that $E/(e' \times s')$ before to discharge was a

very reliable independent predictor of the composite

event. It was discovered that 1.63 (74% sensitivity, 67% specificity) was the ideal cut-off value [19].

Our study found that follow-up LV diastolic and systolic function decreased more than baseline in diabetic patients. with EF and S~ getting worse while LAV and LAVI increased and were linked to lower e' (septal and lateral) as well as higher elevation in the septal and lateral E/e' ratios, which suggested worse WMSI and higher elevation in LVEDP. These results were in line with Karayiannides [20]. We found that the left ventricle's poor remodeling is measured by the $E/(e' \times s')$ ratio had greater levels during diabetic patient follow-up than at baseline. This result was consistent with the conclusions drawn by Ionac et al. [19] and Kenar et al. [21]. However, we also observed that the non-diabetic individuals had improved LV systolic function, with improved EF and S velocity, and improved diastolic function, with decreased LAV and LAVI and higher e' wave velocity, which was linked to improved WMSI. The results aligned with the findings of Reinstadler et al. [22].

During the follow-up period, we observed a reduction in the E/(e'×s') ratio among non-diabetic patients, indicating improved remodeling in these individuals. The outcomes matched those of Ionac et al. [19] and Kenar et al. [21]. The majority of studies like Biering-Sørensen et al [18] and Ionac et al [19] it assessed tissue Doppler in patients diagnosed with NSTEMI and STEMI and reported outcomes such as re-infarction, target lesion revascularization, and death; as the main research outcome, we concentrated on the development of HF.

The Cadillac study's conclusion that individuals with diabetes are more likely to have decreased myocardial perfusion which may have unfavorable effects was validated by our data Prasad et al. [23]. We discovered that non-diabetic patients had better TIMI flow scores and myocardial blush grades with a high incidence of ST segment resolution \geq 70%,

which can explain why their function improved over time in comparison to those with diabetes and why their systolic and diastolic function was better at baseline than that of diabetic patients who receive the same intervention (PPCI on time) [24]. It was discovered that the average $E/(e' \times s')$ ratio may accurately predict both systolic dysfunction and diastolic dysfunction at cut-offs of >1.63 (70.97% sensitivity, 84.06% specificity) and >1.68 (80% sensitivity, 74.44% specificity), respectively. The results of Ionac et al. [19] are in line with these findings. Reperfusion therapy aims to minimize the progression of myocardial necrosis via complete and sustained myocardial tissue reperfusion in addition to reestablishing the coronary artery's epicardial flow. This phrase describes а microvascular blockage and decreased cardiac output following the opening of an occluded artery due to microcirculatory damage. According to statistics from the literature, MBG strongly predicts a poor prognosis and has a detrimental effect on the outcome: among 777 patients in the population. When MBG was measured right after PCI, it was discovered to be correlated with the extent of the infarct and a predictor of both long-term mortality and LV function [25]. Measuring the longitudinal shortening velocity of the ventricle using Tissue Doppler imaging may be a more sensitive indicator of subclinical changes in LV performance in diabetes than assessing global function using conventional echocardiography methods, given the increased susceptibility of the subendocardium to ischemia and interstitial fibrosis [26]. With a sensitivity of 90.0% and specificity of 75.71%, we discovered that the $E/(e'\times s')$ index, as determined by TDI at the mitral annulus site, may be a valid predictor of the myocardial blush grade at a cut-off of >1.315.

Ratio and EF showed no significant positive association, while ratio and LAV did. There was a significant negative association found between the two. These results establish a connection between the diastolic and systolic functions of the left

ventricle and lend credence to the idea that positive remodeling in the ventricle can be predicted using the $E/(e' \times s')$ ratio. The $E/(e' \times s')$ ratio, which indicates LV remodeling (24,25&18 chances), and the WMSI (highest odds ratio: 89) were found to be the strongest predictors of LV and diastolic dysfunction. These outcomes are consistent with the findings of Kenar et al [21]. We also discovered that the best predictors of myocardial blush grad 3 were WMSI (53 odds ratio) and $E/(e'\times s')$ ratio. We looked examined this indicator's prognostic power and its relationship to myocardial perfusion in patients who had positive left ventricular remodeling and those who did not. Additionally, we showed how helpful index for individuals the is undergoing percutaneous interventional therapy for an acute myocardial infarction.

The limitations of the study: This study contained a small sample of STEMI patients who were admitted within a predefined window of time and were not contacted again. The analysis was descriptive and was done at a single location. Furthermore, this investigation did not employ more advanced or contemporary echocardiographic methods. It resulted from our efforts to discover a quick and simple method for classifying STEMI patients.

Conclusion:

According to the current research, TDI E/(e'×s'), a substantial independent prognostic index for HF, may strongly predict diastolic dysfunction at cut-off >1.68 and systolic dysfunction at cut-off >1.63. Patients with STEMI who had primary PCI can also be used as a significant predictor of these results. In non-diabetic patients, tissue perfusion measured by MBG and ST segment resolution was better even after successful initial PCI from an angiographic standpoint. A quick and easy way to assess LV diastolic and systolic dysfunction in patients with type 2 diabetes is to use the TDI E/(e'×s').

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