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Left Ventricular Filling Pressure as a Useful Marker of Myocardial Injury Following Elective Percutaneous Coronary Intervention

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Abstract

Background: Advances in Percutaneous Coronary Intervention (PCI) techniques have improved patient outcome. However, occurrence of myonecrosis after elective PCI has been extensively debated.

Aim of Study: We investigated the value of the ratio of early transmittal velocity to tissue Doppler mitral annular early diastolic velocity (E/E') in detecting the occurrence of peri-procedural myocardial injury in patients undergoing elective PCI.

Patients and Methods: Sixty two patients undergoing elective PCI were recruited prospectively. All participants had EF% >50% and sinus rhythm. E/E' ratio was measured immediately before, and within 24 hours after PCI. CK-MB and cardiac troponin (cTn) were measured in the same day after successful PCI to diagnose myocardial injury.

Results: The cohort age was 56.7±9.7 year, and consisted of forty four (71%) males, 49 (27.4%) had ≥ two risk factors for CAD, 60% were symptomatic before PCI. Thirty (48.4%) patients had regional wall motion abnormalities (regional Wall Motion Score Index (WMSI): 0.74±0.29). Myocardial injury was diagnosed by elevated cTn in 39 (62.9%) patients. Patients with predilation showed reduction of LVEDP as estimated by Δ E/E' (8.9±2.9 Vs. 7.9±3.2, p<0.001). Pearson correlation analysis revealed that the E/E' correlated negatively with LV EF% (r=–26, p<0.03), positively with CK-MB and cTn level after PCI (r=.51, p<0.0001), NYHA functional class (r=.72, p<0.001), the number of risk factors, and number of vessel diseased and the number of vessel treated with PCI (r=36, p<0.003 all). Using ROC curve E/E' value ≥6.55 has 68.1% sensitivity and 66.6% specificity to detect myocardial injury after PCI. From all clinical and echo variables, multivariate regression analysis revealed that only E/E' and number of stents were predictors of postprocedural cardiac injury in elective PCI.

Conclusion: These data suggest that the tissue Doppler derived-index of LV filling pressure (E/E') may be a useful indicator for predicting early myocardial injury after successful PCI. Patients with an elevated E/E' after PCI may need more careful and closer follow-up.

Key Words: *Left ventricular filling pressure – Myocardial injury – PCI.*

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Introduction

ROUTINE Percutaneous Coronary Intervention (PCI) appears to be a relatively safe catheterization procedure. Along with technological progress in coronary intervention, post-procedural complications and adverse outcomes have markedly decreased, yet peri-procedural myocardial injury, manifested as myocardial stunning or infarction, is a frequent complication during PCI and is strongly associated with post-procedural cardiovascular morbidity and mortality **[1]**.

According to the World Health Organization one of the criteria for definition of Myocardial Infarction (MI) is cardiac biomarker elevation **[2]**. It has been reported that post procedure creatinine-MB fraction (CPK-MB) elevation occurs approximately in 25% of patients and troponin I (TnI) level rising is observed at least in 50% of those undergoing PCI **[3]**. Several studies demonstrated that tissue Doppler imaging (TDI) parameters were capable of adding prognostic information to predict cardiac death in major cardiac diseases, such as heart failure **[4-6]** acute coronary syndrome **[7,8]** acute myocardial infarction **[9]**, and hypertension **[10]**.

Elevated Left Ventricular (LV) filling pressure may be clinically silent. The early diastolic transmitral velocity/early mitral annular diastolic velocity ratio (E/E') has been proposed as the best single tissue Doppler predictor for evaluating LV filling

pressure [11] and as a good predictor of cardiac death [12].

We hypothesized that myocardial injury during elective PCI may result in changes in LV filling pressure which can be measured by E/E' ratio. In this study we investigated the value of E/E' ratio, in detecting the occurrence of peri-procedural myocardial injury in patients undergoing elective PCI.

Patients and Methods

Study population:

Between September 2017 and May 2018, 450 consecutive patients were referred to Catheterization laboratory of Cardiology Department, Tanta University Hospital, for elective PCI. A total of 62 patients with Coronary Artery Disease (CAD) met the inclusion criteria and were prospectively enrolled in the study. They were examined in a single centre (Cardiology Department, Tanat University, Egypt).

Inclusion criteria: The study investigated clinically stable patients, with documented CAD and without AMI in the past 4 weeks who attempted to undergo elective PCI and had successful procedure and no prior PCI or CABG, have sinus rhythm and with LV ejection fraction ›_50%.

Our investigation is following the principles outlined in the Declaration of Helsinki. The protocol was approved by the Local Research Ethics Committee of Tanta University, and each patient gave written informed consent.

Exclusion criteria:

Patients with one or more of the following criteria were excluded from the study: Failed PCI, patients with ejection fraction <50%, significant valvular dysfunction, history of heart failure, unstable conditions such as acute coronary syndrome, cardiogenic shock, and ventricular aneurysm; and patients with unsatisfactory echocardiograms or another systemic disease (renal failure or respiratory disease).

The patients were assessed a day prior to coronary angiography and a full clinical history was obtained, including information about cardiovascular risk factors [13,14] and ongoing medications. All patients underwent transthoracic echocardiography and tissue Doppler imaging on the same day just before PCI, and within 24 hours after the procedure. Analysis of the echocardiographic data was performed while blinded to the results the hemodynamic data.

Echocardiographic measurements:

Standard imaging was performed in the left lateral decubitus position using a commercially available system, Vivid 5 ultrasound system (GE Vingmed Ultrasound AS, Horten, Norway). Images were obtained using a 2.5-3.5MHz transducer in the parasternal and apical views. Left Ventricular (LV) End-Diastolic (EDD) and End-Systolic (ESD) diameters were determined with M-mode echocardiography under two-dimensional guidance in the parasternal long axis view, according to the recommendations of the American Society of Echocardiography [15]. LV Ejection Fraction (EF%), was calculated from apical four-chamber views, according to the modified Simpson's rule. Pulsed-Wave (PW) Doppler was performed in the apical 4-chamber view to obtain mitral inflow indices to assess LV filling according to the recommendations of the American Society of Echocardiography [15]. Measurements of mitral inflow include the peak early filling (E-wave) and late diastolic filling (A-wave) velocities, the E/A ratio, Deceleration Time (DT) of early filling velocity and the Isovolumic Relaxation Time (IVRT), derived by placing the cursor of CW Doppler in the LV outflow tract to simultaneously display the end of aortic ejection and the onset of mitral inflow. The Left Atrial Volume Index (LAVI) was estimated from the apical 4-and 2-chamber views by the area length method [16-18].

Tissue Doppler measurements:

Tissue Doppler (TD) of the mitral annulus was obtained from the apical 4-chamber view after filters was set to exclude high-frequency signals. A 5-mm sample volume was placed sequentially at the lateral and medial mitral annulus. The resulting velocities were recorded for three consecutive cardiac cycles at a sweep speed of 50mm/s. The following measurements were made from the recordings: Peak systolic velocity (S'), early (E') and late (A') diastolic velocities. Analysis was performed for the E' and A' velocities as the average of the medial and lateral annulus. The E/E' ratio was used to estimate LV filling pressures [19].

Regional Wall Motion Abnormality (RWMA):
LV regional function was evaluated using the standard 16-segment model as suggested by the American Society of Echocardiography and wall motion was scored as normal=1, hypokinetic=2, akinetic=3, or dyskinetic=4. WMSI=Ē Score of 16 segments/16 [17].

Doppler and echocardiographic studies were repeated, within 24 hours after PCI and before patient, is discharged. All echocardiographic measurements were obtained by one investigator who is blind to the cardiac markers levels and to other clinical parameters.

Percutaneous coronary intervention procedure:

The indication for PCI was based on the American College of Cardiology/American Heart Association recommendations and was performed by experienced interventional cardiologists. All patients were in sinus rhythm during left heart catheterization which was performed through the femoral artery.

A patient was considered to have CAD when a stenosed lesion resulting in a 50% or greater reduction in lumen diameter existed in at least one of the coronary arteries. The severity of CAD was evaluated by number of diseased vessels, diseased lesions, treated vessels and implanted stents were recorded by observers who were blinded to the results of laboratory testing and study grouping **[20]**. Prior to PCI, all patients received adequate loading doses of clopidogrel (600mg) or ticagrelol (180mg) and 70IU/kg intravenous bolus of unfractionated heparin. Glycoprotein IIb/IIIa inhibitors were administered (intra-coronary) or after the procedure according to the operator preference. The PCI procedure was performed via the femoral route.

Interventional techniques and further treatment during PCI were chosen at the operators' discretion and according to current standards. Multi-vessel PCI was defined as more than one target vessel PCI at this procedure **[20]**.

Stents were implanted according to current clinical practice. Angiographic success was defined as a final angiographic residual stenosis of <20% by visual estimation **[20]**. Procedural success was considered in cases of angiographic success and absence of any in-hospital major complications (acute myocardial infarction, need for bypass surgery or repeat PCI, or death) **[22]**. Angiographic measurements were performed with an automated computer-based system (Philips Allura FD10/10 Biplane X-ray system) **[22]**. Aspirin (75mg/day), and clopidogrel (75mg/day) or ticagrelol (90mg bid) and statin were prescribed to all patients after procedure.

The occurrence of angiographic complications during PCI was recorded. Angiographic complications included: Minor/major side branch compromise or occlusions; abrupt intra-procedural vessel closure; major or minor arterial dissection; thrombus formation; transient and/or prolonged slow-no reflow; distal embolization **[22]**.

Diagnosis of myocardial injury: Periprocedural myocardial injury was defined as post-procedural cTnI >1x Upper Limit of Normal (ULN) **[21,22]**. The diagnosis of a peri-procedural myocardial infarction is based on either the development of new pathological Q waves in at least two contiguous ECG leads or an elevation of CK-MB > five times the Upper Limit of Normal (ULN) or cTnI >3x ULN **[21,22]**.

Cardiac troponin I (cTnI) levels were determined in venous blood samples within 24 hours (10-20h) after PCI, and in the event of the occurrence of symptoms or signs suggestive of myocardial ischemia. cTnI was analyzed by an immune-chemiluminometric assay (Access AccuTnI, Beckman Coulter, CA). The ULN was defined as the 99th percentile of normal population with a total imprecision of <10%. The ULN of this test was 0.09ng/mL **[21]**. The peak value of cTnI within 24 hours was used for statistical analysis.

Statistical analysis:

Values were presented as means ± SD or as numbers and proportions, as appropriate. The relations between qualitative variables were evaluated by Chi-square test or Fisher's exact test, as indicated. Means were compared with student's test the significance of paired data was evaluated by the paired student's test. ROC analysis was performed to select cut-off values with suitable sensitivity and specificity to detect myocardial injury. Variables that were statistically significant in univariate analysis were introduced in a logistic regression model to detect independent predictors of myocardial injury as indicate by Troponin >0.1. All tests were bilateral and a p-value of 5% was the limit of statistical significance. Analysis was performed by statistical package software IBM-SPSS for MAC, Version 24.

Results

A total of 62 consecutive patients were enrolled in this prospective observational study. Patients who were sent for elective PCI and met the inclusion criteria and with complete angiographic and laboratory data were consecutively included in the analysis without any pre-selection.

Demographic characteristics:

The baseline demographics and clinical characteristics of all participants are presented in (Table 3). The cohort consisted of 44 (70.1%) males and 18 (19.9%) females with mean age 56.7 ±9.7 (ranged from 40 to 72) years. Six (9.8%) patients are young adult (below 45 years of age). All patients were electively referred for PCI.

Regarding patient presentations: 17 (27.4%) patients had chronic stable angina, 29 (46.8%) had unstable angina, 12 (19.4%) had history of prior ST-Elevation Myocardial Infarction (STEMI) and 4 (6.5%) had history of Non ST-Elevation Myocardial Infarction (NSTEMI). According to patient functional status, 27(43.5%) were NYHA II, 10 (16.1%) were NYHA class III while the remaining 25 (40.3%) were asymptomatic.

Patient risk profile:

In studied patients, 26 (41.9%) were diabetic type II, 31 (50%) were hypertensive, 39 (62.9%) were smokers, 33 (53.2%) had dyslipidemia and 31 (50%) were obese. 3 (4.1%) had no risk factors for CAD, 10(16.1%) had a single risk factor, 17 (27.4%) had two risk factors, 14 (22.6) had 3 risk factors, and 16 (25.8%) had 4 risk factors for CAD. Only 2 patients (3.2%) had five risk factors for CAD to be known before PCI.

Table (1): Patient characteristics.

	Minimum	Maximum	Mean	Std. deviation
Age (years)	40	72	56.74	9.7
BMI (Kg/m^2)	25.9	35.4	30.19	2.9
No of risk factors	0	5	2.58	1.26
HR (b/min)	69	89	79	5.6
SBP (mmHg)	100	155	130	12.5
DBP (mmHg)	60	100	80	6.5

	Patients No.	(%)
NYHA I	25	40.3
NYHA II	27	43.5
NYHA III	10	16.1
Diabetes	26	41.9
Hypertension	31	50
Obesity	31	50
Dyslipidemia	33	53.2

Echocardiographic findings in PCI group:

Initial echocardiograms were obtained 2.3 ±0.4 hour after admission and before cardiac catheterization. Repeated measurements were obtained 4-6 hours after the procedure. Two Dimensional (2D) echo-Doppler variables in PCI patients before and after the procedure are presented in (Table 2).

No significant differences between LV wall thickness, LV dimensions or EF%, FS%, Left Atrium (LA) diameter or LA volume index before and after PCI. However, E mitral flow velocity and E/A ratio showed significant increase after PCI procedure (p<.0001, .007) respectively.

Before PCI, 30 (48.4%) patients had RWMA with WMSI ranged between 1-1.77 and mean value: 1.127±0.12. However, no significant changes were observed after PCI.

S' was significantly increased after PCI (p <0.05). In contrast, there was no significant change in E' or A' velocities after PCI. Before PCI E/E' was ranged from 4.2 to 17.5 and mean value was 8.6±2.9. It showed significant reduction after PCI (7.90±3.2) (p<.01) Figs. (1,2).

Table (2): Echocardiographic variables in studied population.

	Before PCI (n=62)		After PCI (n=62)		
	Mean	Std. deviation	Mean	Std. deviation	p-value
IVS (cm)	1.04	0.18	1.03	0.19	.159
LVPW (cm)	1.06	0.17	1.05	0.17	.159
ESD (cm)	3.37	0.50	3.41	0.53	.318
EDD (cm)	4.99	0.56	5.01	0.60	.545
EF%	60.13	7.75	59.7	7.3	.382
FS%	32.53	5.73	32.2	5.37	.423
WMSI	0.743	0.29	0.69	0.27	.532
LAD (cm)	3.65	0.48	3.35	0.22	.645
LAV (ml)	33.4	8.5	32.1	7.4	.432
LAVI (ml/m^2)	36.2	0.98	32.8	0.76	.32
Mitral E (cm/s)	0.62	0.47	0.78	0.17	.000
Mitral A (cm/s)	0.77	0.18	0.79	0.2	.897
E/A	0.81	0.29	0.92	0.31	.007
WMSI	1.127	0.12	1.113	0.01	.134
E' (m/s)	.082	.024	.089	.027	.074
A' (m/s)	.11	.027	.12	.027	.068
S' (m/s)	.082	.019	.086	.019	.045
E/E'	8.61	2.88	7.90	3.21	.013

IVS : Interventricular Septum.
LVPW : Left Ventricular Posterior Wall.
LVESD : Left Ventricular End-Systolic Diameter.
LVEDD : Left Ventricular End-Diastolic Diameter.
FS : Fractional Shortening.
EF : Ejection Fraction.
LAD : Left Atrium Diameter.
LAVI : Left Atrium Volume Index.
E : Early diastolic mitral inflow velocity.
A : Late diastolic mitral inflow velocity.
WMSI : Wall Motion Score Index.
E' : Early diastolic velocity of the mitral annulus.
A" : Atrial diastolic velocity of mitral annulus.
S' : Peak systolic velocity of mitral annulus.

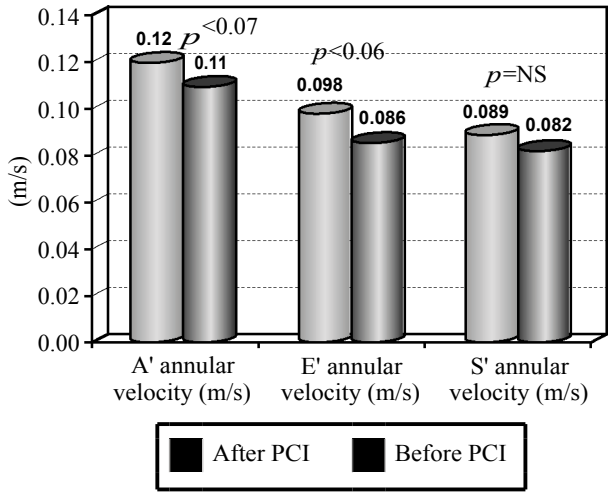

Fig. (1): Tissue Doppler imaging parameters before and after PCI.

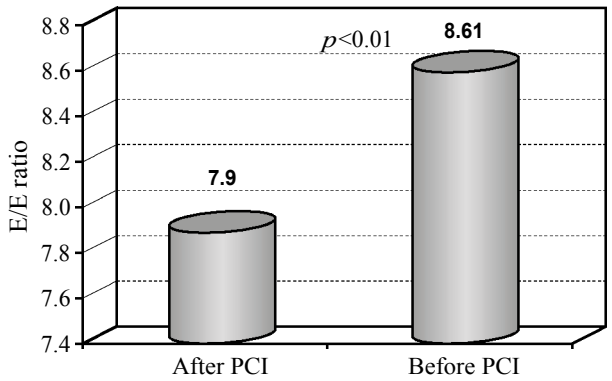

Fig. (2): E/E' before and after PCI in study population.

Angiographic data:

Coronary angiography revealed that 12 (24.2%) patients had single-vessel disease, and 30 (48.4%) had two vessel disease, 13 (21%) had three vessel disease and 4 (6.5%) had multivessel disease. Forty seven (75.8%) patients undergoing elective PCI had significant obstructive lesions. All patients had significant LAD lesions, 30 (48.4%) had LCX diseased and 30 (40.3%) had RCA diseased (Table 3).

During the coronary procedures, 43 (54.8%) patients undergoing PCI had predilation with suitable sized balloon before PCI. 36 (58%) of the number of stents deployed, had balloon dilation before stent implantation.

Forty seven (75.8%) patients had significant LAD disease, 13 (21%) patients had significant diagonal lesions, 25 (40.3%) patients had LCX significant diseases, 6 (9.7%) patients had OM critical lesions and 30 (48.4%) patients had RCA lesions. So, LAD disease frequency (75.8%) was significantly higher compared to RCA affection (48.4%) and LCX disease (40.3%); $p<.0001$ respectively (Table 3).

Also, the number of stents implanted per patient was variable; 24 (38.7%) patients had one stent per procedure, 37 (59.7%) patients had 2 stents implanted, and only one patient had 4 (1.6%) stents implanted. For LAD: 36 (75.8%) had one Drug Eluting Stent (DES) PCI and 10 (16.1%) had 2 DES PCI. While for LCX 13 (21%) patients had one DES and 3 (4.8%) had 2 DES PCI. For RCA 17 (27.4%) had single DES PCI and 2 (3.2%) had 2 DES.

After PCI, CK-MB was elevated to diagnostic level (37.8 ± 19%) of myocardial injury in 47 (75.8%) while cTnI was elevated (1.69 ±3.58) to diagnostic level in 39 (62.9%). In the current study we considered only the 39 (62%) patients with cTnI >1xULN in statistical analysis as the patient group with myocardial injury.

Table (3): Angiographic findings in study population.

	Frequency	Number of vessels treated	
		One	Two
Number of vessels diseased:			
1	15 (24.2)	37	25
2	30 (48.4)	(59.7%)	(40.3%)
3	13 (21.0)		
4	4 (6.5)	Pre-dilation	
No. of stents:		No	Yes
1	24 (37.8%)		
2	37 (59.7)	28	34
4	1 (1.6)	(45.2%)	(54.8%)

Prevalence of significant lesions				
LAD	Diagonal	LCX	OM	RCA
47 (75.8%)	13 (21%)	25 (40%)	6 (9.7%)	30 (48.4%)

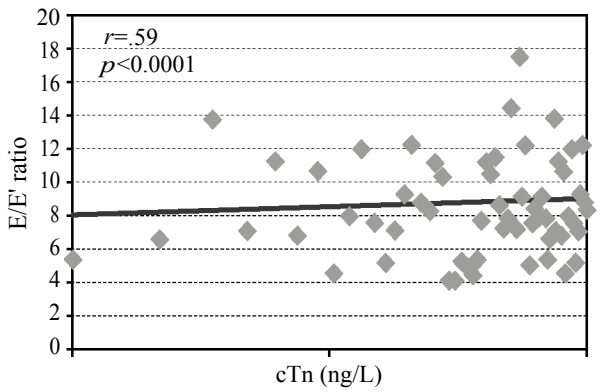

Fig. (3): Correlation of E/E' post PCI to cardiac troponin.

Correlation of E/E' to clinical and laboratory data:

On analyzing the relationship of E/E', before and after PCI and the magnitude of change (A), we demonstrated that E/E' before and after PCI was directly correlated to CK-MB and cTn & NYHA class (p<0.0001). E/E' before PCI was correlated to number of vessels diseased (p<0.05) and number of risk factors (p<0.001), meanwhile E/E' after PCI and A change were directly correlated to number of vessel treated (p<0.001) and inversely related to LV EF% (p<.001, <.002) respectively (Table 4), Figs. (4-7).

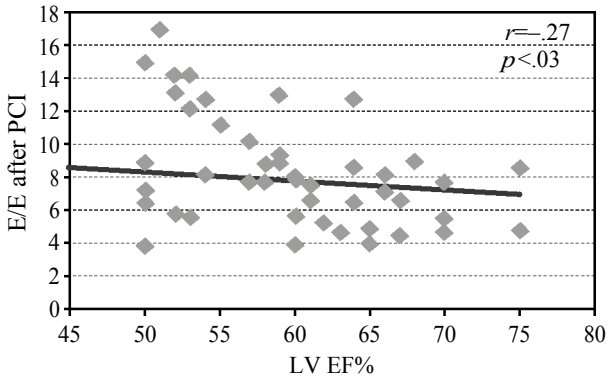

Fig. (4): Correlation between E/E' after PCI and EF%.

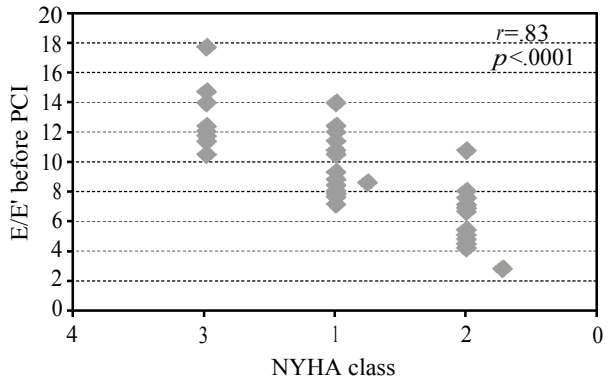

Fig. (5): Correlation between E/E' before PCI and NYHA functional class.

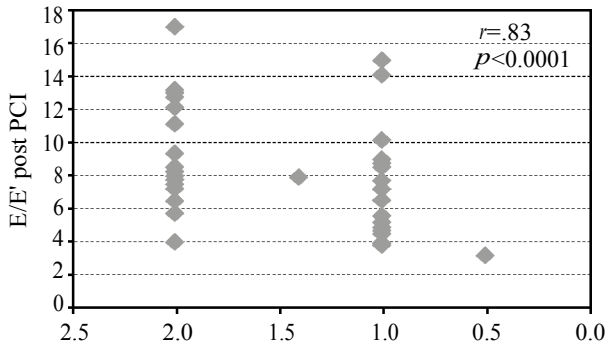

Fig. (6): Correlation of E/E' after PCI and the number of vessels treated.

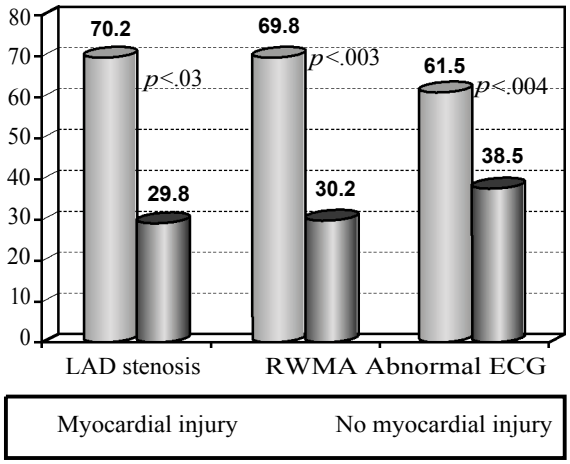

Fig. (7): Prevalence of RWMA, abnormal ECG and LAD stenosis in PCI with and without myocardial injury.

Table (4): Relationship of E/E' to laboratory and angiographic data.

	E/E' (Before PCI)	E/E' (After PCI)	4 E/E'
CK-MB:			
r	.519**	.511**	.082
p	.000	.000	.527
Troponin:			
r	.585**	.587**	.089
p	.000	.000	.492
No of risk factors:			
r	.347**	.226	–.149
p	.006	.077	.246
EF%:			
r	–.002	–.263*	–.382**
p	.986	.039	.002
NYHA:			
r	.803**	.729**	.017
p	.000	.000	.893
No of vessels diseased:			
r	.261 *	.172	–.086
p	.041	.181	.507
WMSI:			
r	–.038	–.040	–.037
p	.770	.759	.776
No of vessels treated:			
r	.340*	.360**	–.001
p	.010	.004	.995
Number of stents:			
r	.063	.097	.064
p	.628	.452	.621

EF% : Ejection Fraction.
NYHA : New York Heart Association.
WMSI : Wall Motion Score Index.

Table (5): Comparison between patients with and without myocardial injury in clinical and angiographic before PCI.

	No myocardial injury n=23	Myocardial injury n=39	*p*-value		No myocardial injury n=23	Myocardial injury n=39	*p*-value
Male (n=44)	17 (38.6%)	27 (61.4%)	.436	Age (years)	55.57±11.7	57.44±8.93	.032
Female (n=18)	6 (33.3%)	12 (66.7%)					
Diabetes mellitus	11 (42.3%)	15 (57.7%)	.324	BMI (kg/m^2)	29.64±3.10	30.52±2.84	.547
Hypertension	11 (35.5%)	20 (64.5%)	.500	NYHA	1.65±.647	1.82±.756	.508
Smoking	13 (33.3%)	26 (66.7%)	.298	No of RF	2.35±1.40	2.72±1.17	.461
Dyslipidemia	10 (33.3%)	23 (69.7%)	.176	HR (b/min)	79.78±12.6	77.05±8.86	.023
Obesity	9 (29%)	22 (7 1%)	.146	SBP (mmHg)	124±18	150±15	.286
Angina	9 (52.9%)	8 (47.1%)	.230	DBP (mmHg)	80±10	82±8.9	.269
UA	7 (24.1%)	22 (75.9%)	.225	IVS (cm)	1.05±.18	1.028±.19	.911
STEMI	5 (41.7%)	7 (58.3%)	.733	LVPW (cm)	1.10±.16	1.04±.17	.860
NSTEMI	2 (50.0%)	2 (50.0%)	0	ESD (cm)	3.32±.47	3.40±.52	.498
NYHA:							
I (n=25)	10 (40.0%)	15 (60)	.473	EDD (cm)	4.91±.63	5.05±.52	.191
II (n=27)	11 (40.7%)	17 (59.3%)	.446	EF%	60.9±5.4	59.7±8.9	.025
III (n=10)	2 (20%)	8 (80%)	.372	WMSI	.734±.34	1.06±.27	.020
Sinus Rhythm	23 (37.1%)	39 (62.9%)	.620	LA (cm)	3.69±.5	3.62±.47	.882
Abnormal ECG	10 (38.5)	16 (61.5%)	.004	E (m/s)	.65±.17	.69±.17	.885
RWMA	13 (30.2)	30 (69.8%)	.003	A (m/s)	.75±.17	.79±.18	.900
LAD	14 (29.8%)	33 (70.2%)	.031	E/A	.88±.22	.92±.33	.259
Ramus	0 (0%)	2 (100%)	.392	E' (m/s)	.09±.02	.08±.03	.435
Diagonal	4 (30.8%)	9 (69.2%)	.424	A' (m/s)	.11±.02	.12±.03	.149
LCX	7 (28.0%)	18 (72.0%)	.171	S' (m/s)	.09±.02	.08±.02	.224
OM	2 (33.3%)	4 (66.7%)	.607	E/E'	7.83±2.6	9.1±2.9	.530
RCA	11 (36.7%)	19 (63.3%)	.578				
Pre-dilation	16 (47.1%)	18 (52.9%)	.765	No of stents	1.17±.58	1.87±.52	.265
LAD PCI:							
1 DES (n=36)	12 (33.3%)	24 (61.7%)	.172	No. vessel diseased	1.83±.78	2.26±.85	.899
2 DES (n=10)	2 (20%)	8 (80%)	.128				
LCX PCI:							
1 DES (n=13)	3 (23.1%)	10 (76.9%)	.160	No. vessel treated	1.17±.39	1.54±.51	.000
2 DES (n=3)	0	3 (100%)	.093				
OM:							
1 DES (n=1)	0	1 (100%)	.629				
RCA PCI:							
1 DES (n=12)	8 (66.6)	4 (33.4%)	.072				
2 DES (n=2)	0	2 (100%)	.063				

UA : Unstable Angina.
LAD : Left Anterior Descending.
LCX : Left Circumflex.
OM : Obtuse Marginal.
RCA : Right Coronary Artery.
BMI : Body Mass Index.
NYHA : New Yourk Heart Association.
HR : Heart Rate.
SBP : Systolic Blood Pressure.
DBP : Diastolic Blood Pressure.
ESD : Left Ventricular End-Systolic Diameter.
LVEDD : Left Ventricular End-Diastolic Diameter.
FS : Fractional Shortening.
EF : Ejection Fraction.
LVPW : Posterior Wall Thickness.
IVS : Interventricular Septum.
S' : Peak myocardial systolic velocity.
E' : Early diastolic annular velocity.
A' : Atrial annular diastolic velocity.
E : Early diastolic flow.
A : Late diastolic flow.
WMSI : Wall Motion score index.

PCI patients were categorized according to cTnI levels into: Patients with myocardial injury (n=39) and no myocardial injury (n=23).

Patients with myocardial injury showed more prevalence of abnormal ECG (p<0.004), more RWMA (p<0.003) and higher incidence of LAD disease (p<0.03) Fig. (7).

Regarding clinical and echocardiographic variables before PCI, patients with myocardial injury where older in age (p<0.03) had lower heart rate (p<0.02), lower EF% (p<0.03), FS% (p<0.003) and higher WMSI (p<0.02), and larger number of vessels treated (p<0.0001) (Table 6). However, no significant difference was observed between E/E' measured before PCI when patients had myocardial

injury was compared to those without evidence of myocardial injury, Fig. (8).

Table (6): Comparison between patients with & without myocardial injury in clinical and echocardiographic post PCI.

	Myocardial injury N=39	No myocardial injury N=23	*p*-value
IVS (after PCI) (cm)	1.02±.19	1.05±.18	.555
LVPW (after PCI) (cm)	1.03±.17	1.10±.16	.133
ESD (after PCI) (cm)	3.47±.537	3.29±.504	.190
EDD (after PCI) (cm)	5.11±.56	4.87±.65	.134
EF% (after PCI)	59.0±8.02	60.9±5.81	.333
FS% (after PCI)	32.2±6.023	32.2±4.16	.993
Mitral E (after PCI) (m/s)	.64±.16	.58±.173	.203
Mitral A (after PCI) (m/s)	.80±.19	.73±.13	.180
E/A (after PCI)	.87±.36	.82±.21	.556
E' (after PCI) (m/s)	.082±.029	092±.020	.162
A' (after PCI) (m/s)	.123±.02	113±.025	.186
S' (after PCI) (m/s)	.084±.021	.091±.017	.154
E/E' (after PCI)	8.77±3.60	6.45±1.59	.005
Δ E/E'	–.34±2.35	–1.46±1.94	.060

ESD : End Systolic Diameter.
EDD : Left Ventricular End-Diastolic Diameter.
FS : Fractional Shortening.
EF : Ejection Fraction.
LVPW : Posterior Wall Thickness.
E : Early diastolic flow.
A : Late diastolic flow velocity.
WMSI : Wall Motion Score Index.
E' : Early diastolic velocity of the mitral annulus.
E : Early mitral inflow velocity.
A" : Atrial diastolic annular velocity.
S' : Peak systolic velocity.

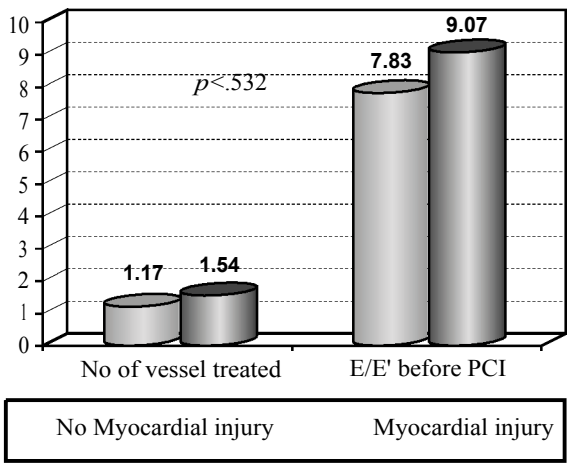

Fig. (8): E/E' before PCI and number of vessels treated in patients with and without myocardial injury.

In contrast, after PCI there was no significant difference between patients with evidence of myocardial injury and those without myocardial injury in any of the echocardiographic variables except the value of E/E' ratio. Patients with myocardial injury had significantly higher E/E' compared to non-injury group (8.77±3.6 versus 6.45±1.5, p <0.005) respectively (Table 6), Fig. (9).

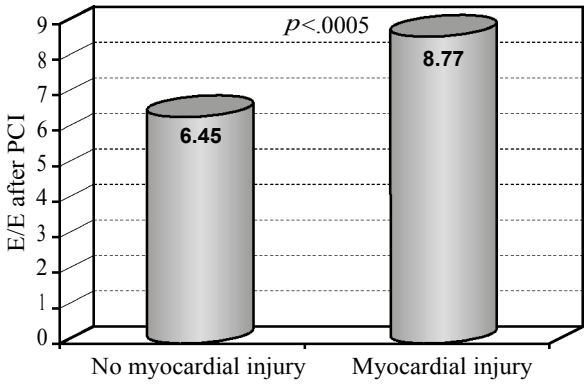

Fig. (9): Comparison of E/E' ratio between myocardial injury and non myocardial injury group.

Roc curve for E/E' to diagnose myocardial injury after PCI:

To explore the value provided by E/E' post PCI in patients with CAD after treated electively to diagnose myocardial injury; we constructed ROC curves according to the cut-off value of cTn ≥0.1 ng/ml. A value of 6.55 or higher for E/E' ratio has 66.7% sensitivity and 52.2% specificity to detect myocardial injury as indicated by Troponin ≥0.1 units. The area under the ROC curve was 0.692 and confidence interval from 0.593 to 0.822 (p <0.01) Fig. (10).

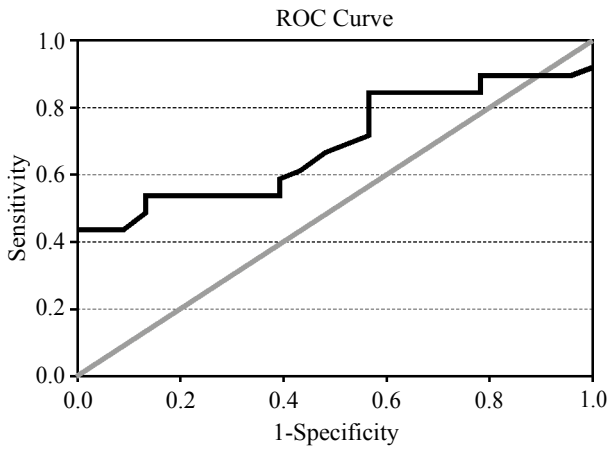

Fig. (10): Roc curve of E/E' to diagnose myocardial injury after PCI.

Multivariate logistic regression analysis:

The variables which showed significant difference in comparing patients with myocardial injury versus those with no evidence of myocardial injury were introduced into multivariate regression analysis. These variables are shown in (Table 7).

From all these clinical and echocardiographic variables (ECG, No of vessels diseased, No. of

vessels treated, No of stents, E/E') only number of stents [odd ratio: 33.368 and 95% CI: 3.601-309.2, $p<.002$] and E/E' post PCI [odd ratio: 1.926, 95% CI: 1.137-3.265, $p<.01$] were independent predictors for myocardial injury in patients sent for elective PCI (Table 7).

Table (7): Multivariate logistic regression analysis.

	B	S.E.	Wald	df	Sig.	Odds ratio	95% C.I. of OR	
							Lower	Upper
Step 1A:								
Number of stents	2.452	.621	15.596	1	.000	11.616	3.439	39.230
Constant	–3.247	.986	10.835	1	.001	.039		
Step 2B:								
Number of stents	2.826	.737	14.688	1	.000	16.885	3.979	71.655
E/E' (after PCI)	.391	.145	7.274	1	.007	1.479	1.113	1.965
Constant	–6.734	1.826	13.597	1	.000	.001		
Step 3C:								
ECG changes			.896	10	1.000			
Number of stents	3.508	1.136	9.534	1	.002	33.368	3.601	309.220
E/E' (after PCI)	.656	.269	5.931	1	.015	1.926	1.137	3.265
Constant	–33.332	28420.740	.000	1	.999	.000		

A: Variable(s) entered on step 1: Number of stents.
B: Variable(s) entered on step 2: E/E' (after PCI).
C: Variable(s) entered on step 3: ECG changes.

Discussion

In the current study TDI-derived estimations of left ventricular diastolic filling pressure (E/E') obtained with transthroacic echocardiography after elective PCI notably predicted incident periprocedural myocardial injury. E/E' showed a strong relationship with cardiac enzymes after PCI, and their values in addition to the number of vessels treated during PCI were the only variables that predict myocardial injury.

Research into the pathology of CAD have demonstrated the usefulness of measuring concentrations of biomarkers released from the injured cardiac muscle and that they can aid the diagnosis of diseases caused by myocardial ischemia. Since the mid-1950s, successively better biochemical markers have been described in research publications and applied for the clinical diagnosis of acute ischemic myocardial injury **[23]**.

After the discovery that cardiac troponins I and T have the desired specificity in diagnosing myocardial injury and infarction,they have replaced the cytosolic enzymes in the role of diagnosing myocardial ischemia and infarction. The use of the troponins provided new knowledge that led to revision and redefinition of ischemic myocardial injury as well as the introduction of biochemicals for estimation of the probability of future ischemic myocardial events. These markers, known as cardiac risk markers, evolved from the diagnostic markers such as CK-MB or troponins, but markers of inflammation also belong to these groups of diagnostic chemicals.

Percutaneous Coronary Intervention (PCI) is frequently accompanied by cardiac marker elevation after the procedure (also known as periprocedural myocardial injury), especially with the use of high-sensitivity troponin **[23,24]**. A large body of data demonstrated that postprocedural troponin elevation was associated with a worse clinical outcome **[25,26]**.

Recently, several studies demonstrated the clinical efficacy of TDI echocardiographic examination of left ventricular longitudinal motion in various pathologic conditions such as acute myocardial infarction and non-valvular atrial fibrillation **[27]**. If TDI echocardiography offers additional information beyond the clinical judgment and baseline systolic function during the setting of cardiac intervention, it too could be used to identify potential at-risk patients during PCI, and thus, may be a very useful non-invasive bedside tool for patient monitoring.

The ratio of trans mitral E wave velocity to TDI measured mitral annular velocity (E/E') relates early transmitral left ventricular filling to myocardial relaxation and is used to estimate mean left atrial pressure, **[28]** which may be elevated in various cardiac pathologies, and specifically in advanced diastolic dysfunction. Although E' and E/E' measurements alone are not diagnostic of diastolic dysfunction per se, they aid in the assess-

ment of its severity when combined with other measurements [29]. Both E' and E/E' are related to the intrinsic pathophysiology of diastolic dysfunction; specifically, reduced myocardial relaxation and elevated LV filling pressures are reflective of physiologic derangements that may indicate more advanced diastolic heart disease.

In the estimation of elevated LV diastolic pressure, E/E' values >15 represent elevated LV filling pressure, and <8 reflect normal filling pressure [28]. Unlike conventional mitral and pulmonary venous flow velocity indices of LV filling pressures (e.g., a short mitral deceleration time (<140ms) and/or an increased E/A ratio (>2.5), the accuracy of E/E' in estimating LV filling pressures is relatively independent of rate and rhythm abnormalities (such as sinus tachycardia and atrial fibrillation), LV hypertrophy, and functional mitral regurgitation [30].

However, there are several caveats to TDI measurements that warrant mention. Importantly, patients with relatively normal hearts with higher baseline tissue Doppler velocities will have more preload dependence compared to patients with marked impairment in myocardial relaxation [31,32]. Age influences TDI values as well; E' decreases in a linear fashion with increasing age, and thus E/E' increases with age [33,34]. Although E' is used to relate to global indices of LV relaxation, it is a regional index. Errors can, therefore, occur in patients with regional wall motion abnormalities at the Doppler sampling site, unless an average of two sites is provided. Another important consideration when using TDI is the position of the sample area within the wall. Diastolic velocities measured in the lateral wall are higher than velocities measured in the septum, as the septum is tethered to the right ventricle and other structures in the middle of the heart [35].

Up to our knowledge, no data exists about the association of LV filling pressure as estimated by E/E' with periprocedural myocardial infarctionor injury following elective PCI. The present study demonstrated a significant relationship between elevated E/E' and cardiac biomarkers, cTn and CK-MB, that accurately diagnose myocardial infarction and myocardial injury.

The value of E/E' showed significant reduction after the successful PCI procedure from 8.61 ±2.88 to 7.90±3.2 which can be explained in our study by reduction of early mitral inflow velocity (E) and not due to improved myocardial relaxation (E'). The hemodynamic changes and drop of LV end diastolic pressure as reflected here by decreased E/E' after the procedure are the earlier changes to happen after revascularization and even before myocardial relaxation is improved. However, the patients who developed myocardial injury still had higher values of E/E' despite the successful procedure.

The peak rate of LV diastolic filling E is the first variable to decreasewith improved diastolic function after successful PCI but progressively increased in response to elevation in LA pressure when myocardial injury occurs. However, the peak E' wave takes time to increase with improvement ofmyocardial relaxation. Thus E' (but not E) accurately reflects the slow progressive improvement of LV relaxation as coronary perfusion improve after PCI [35,36].

Other interesting results in our study include the ability of E/E' to reflect small injury during the procedure. The values of E/E' after the procedure become correlated directly with cTn and CK-MB which is the corner stone in diagnosing myocardial injury or infarction. Furthermore, the patients who developed myocardial injury retained a higher E/E' (8.77±3.6) compared with patients who had no myocardial injury (6.5 ± 1.9).

This is concordant with the findings of Groban et al., [37] who determined the predictive value of E/E' post operatively in predicting the in-hospital outcome after cardiac surgery. They elucidated the relationship of intra-operative E/E' to the use of inotropic support, duration of Mechanical Ventilation (MV), length of Intensive Care Unit stay (ICU-LOS) and total hospital stay (H-LOS) in 205 patients requiring cardiac surgery. They analyzed the relation between intraoperative E/E' or LVEF and early post-operative morbidity, hospital long stay [H-LOS, ICU-LOS and MV] and the probability that a patient would require inotropic support. They demonstrated that E/E' maintained a significant relationship between ICU-LOS and the use of inotropic support. The magnitude of this relationship was substantial; in particular, they found that those patients with moderately elevated (E/E' ≥8 and ≤ 15) and severely elevated (E/E' >15) tissue-Doppler derived filling pressures spent on average between 9 and 30h longer in the ICU than those with an E/E' ratio <8.

The higher values of E/E' in Groban et al., study (≥8) that predict early morbid events after cardiac surgery versus 6.66 in our study that predict myocardial injury could be explained by the more complex environment accompanying the cardiac

surgery and more advanced myocardial diastolic dysfunction. In Gorban et al., study, 48% of the severely elevated E/E' group and 36% in the moderately elevated received postoperative inotropic support, were compared to only 11% of those with E/E' <8 **[37]**.

Taken together, these findings suggest that the TDI-derived index of left ventricular diastolic filling pressure may provide additional prognostic information from that of baseline systolic function.

In the present study E/E' ratio showed direct correlation with NYHA functional class, the number of risk factors, the number of vessels diseased and the number of vessels treated. Moreover, patients with myocardial injury after PCI had higher E/E'.

In a cohort with dilated cardiomyopathy with similar systolic function, Galrinho et al., **[38]** found that patients with an elevated E/E', was more symptomatic and also E/E' was a powerful predictor of clinical outcome.

Terzi et al., observed that E/E' was correlated with cardiopulmonary exercise capacity in patients with LV systolic dysfunction, and Index of the E/E' ratio was found to be the most powerful predictor of peak oxygen uptake **[39]**.

The value of the mitral annular TDI has been demonstrated in many pathological situations. Wang et al., showed the relationship of mitral annular velocity TDI parameters to mortality in patients with HF **[40]**. Saso et al.; determined prognostic value of the Tei index combining systolic and diastolic myocardial performance in patients with acute myocardial infarction treated by successful primary angioplasty **[41]**. Kim et al.; reported that early diastolic but not systolic annular velocity correlated well with exercise time in patients with chronic mitral regurgitation **[42]**. However, Witte et al., have recently reported that TDI mitral annular diastolic velocity correlated significantly with peak VO_2, but the systolic indices (S') were very strongly correlated to exercise capacity **[42]**. In addition, they found a weaker correlation between peak oxygen consumption and LV diastolic inflow than in those studies previously reported.

Combining transmitral flow velocity with annular velocity has been reported to be an alternative method to correct the transmitral velocity for the influence of relaxation. The ratio of E/Ea was found to be correlated well with LV filling pressure. Consistent with previous studies, our results demonstrated that the E/E' ratio was increased in patients with myocardial injury compared with subjects without injury and this ratio tended to be higher in more severe damage. These results confirm the previous observation that the ratio of E/E' is strongly correlated with diastolic dysfunction in patients with coronary artery disease **[43,44]**.

Study limitations:

There are several limitations in the present study. First, this was a small, prospective study of patients who were referred for elective PCI. However, the current group of patients was well selected after the exclusion of all criteria and confounders that may alter E/E' like EF% <50%, prior PCI or CABG or patients with significant mitral regurge. Second, inclusion of two categories of patients with prior history of ACS, in addition to those with chronic stable angina. But, this decision was made in an attempt to examine the utility of E/E' in our overall daily practice and real life patient presentation. Third, there was no direct measurement of LVEDP during the procedure and its relation to E/E'. However, evident myocardial injury may occur hours after PCI which makes direct measurement during the procedure inaccurate.

Conclusion:

The present study adds to the growing body of evidence indicating that E/E' is of clinical relevance in the percutaneous coronary intervention setting. The tissue-Doppler derived surrogate of diastolic filling pressure, E/E', obtained by transthoracic may be a useful indicator for predicting early myocardial injury and possible occurrence of morbid events after PCI that may even change the beneficial effect of coronary re-canalization.

E/E' correlated well with cardiac troponin in catheterization laboratory and could be used as a simple non-invasive bedside test for patient risk stratification after successful PCI. Importantly, patients with an elevated pre-operative E/E' may need more careful peri-and postprocedural management than those patients with E/E' >6.66.

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ضغط إمتلاء البطين الآيسر كعلامة مفيدة للتنبؤ بإصابة عضلة القلب بعد تداخل الشرايين التاجية الإختيارى

أصبحت عمليات توسيع الشرايين التاجية من خلال إجراء القسطرة القلبية آمناً نسبياً وقد أدى التقدم التكنولوجى فى توسيع الشرايين التاجية التداخلى إلى إنخفاض ملحوظ فى المضاعفات التى تحدث فيما بعد.

الهدف من البحث: فى هذه الدراسة تم التحقق من قيمة نسبة سرعة السريان خلال الميترالى إلى سرعة النسيج الحلقى الميترالى بإستخدام الدوبلر النسيجى فى توقع حدوث إصابات بعضلة القلب بعد إجراء توسيع الشرايين التاجية التداخلية الإختيارى.

طرق البحث: تم إجراء البحث على مرضى الشرايين التاجية الذين يتعرضون للتوسيع التداخلى الإختيارى وهم فى حالة إكلينيكية مستقرة. وتم مطابقة التحقيق للمبادئ الواردة فى إعلان هلسنكى والموافقة على البروتوكول من قبل لجنة أخلاقيات البحوث العلمية وقد كان لكل مريض إقرار موافقة على البحث.

بعد عمل تقييم إكلينيكى، وفحص بالأشعة الموجات فوق الصوتية تم إستبعاد المرضى اللذين يعانون من: قصور بوظيفة القلب الإنقباضية (أقل من ٥٠٪) خلل وظيفى مؤثر بالصمامات (المترالى أو الأورطى)، هبوط بعضلة القلب، توسيع تداخلى سابق للشرايين التاجية، تغيير للشرايين التاجية بعملية القلب المفتوح، أو قصور حاد بالشرايين التاجية تم عمل قياسات الموجات فوق الصوتية على القلب وفقاً لتوصيات الجمعية الأمريكية للموجات فوق الصوتية تم تشخيص وجود إصابة بعضلة القلب، عن طريق وجود تغيرات مرضية برسم القلب، أو إرتفاع فى إنزيمات القلب أكثر من ٥ أضعاف الحد الأعلى الطبيعى تم عمل هذه الأبحاث قبل وبعد القسطرة القلبية بقسم القلب بمستشفيات جامعة طنطا.

النتائج: كان عمر مجموعة المرضى ٥٦.٧±٩.٧ سنة، وتتألف من ٤٤ (٧١٪) من الذكور، ٤٩ (٢٧.٤٪) وكان هناك إثنين من عوامل الخطر لأمراض الشرايين التاجية، ٦٠٪ كانوا من أعراض قبل توسيع الشرايين بالقسطرة I. ثلاثون (٤٨.٤٪) من المرضى الذين يعانون من تشوهات بجدار القلب (معامل تحرك جدار القلب: ٠.٧٤±٠.٢٩). إصابة عضلة القلب cTn فى ٣٩ (٦٢.٩٪) من المرضى. إرتفاع الضغط الإنبساطى للبطين الآيسر كما هو مقدر بواسطة (٨.٩±٢.٩) 'E/E Δ مقابل ٣.٢±٧.٩، ($p<0.001$). وكشف تحليل الإرتباط بيرسون أن 'E/E يرتبط سلباً مع (LV ٪ $p<0.003$)، بشكل إيجابى مع CK-MB ومستوى cTn بعد (PCI $p<0.0001$)، فئة وظيفية (NYHA $p<0.001$)، وعدد عوامل الخطر، وعدد الدعامات المستخدمة. بإستخدام قيمة R/E لمنحنى ROC ≥6.55 يكون ٦٨.١٪ حساسية و٦٦.٦٪ خصوصية للكشف عن إصابة عضلة القلب بعد PCI.

الخلاصة: تشير هذه البيانات إلى أن التصوير النسيجى بالدوبلر يمكن إستخدامها فى التنبؤ بحدوث إحتشاءه بعضلة القلب عند توسيع الشرايين التاجية بالقسطرة التداخلية.