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ORIGINAL ARTICLE

Impact of elective PCI on left intraventricular mechanical dyssynchrony in patients with chronic stable angina (tissue Doppler study)

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KEYWORDS

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Abstract *Aim:* To detect the impact of elective PCI on left intraventricular mechanical dyssynchrony in patients with chronic stable angina.

Methods: 100 patients with chronic stable angina were included and divided into two groups according to LV systolic and diastolic mechanical dyssynchrony measured by TSI 12-segments SD and Te-SD. 24 h then 1 month after PCI, patients with dyssynchrony were reclassified into improved vs persistent LV mechanical dyssynchrony.

Results: At baseline 72% had LV systolic mechanical dyssynchrony. Patients with LV systolic mechanical dyssynchrony were significantly older (58.42 ± 4.617 vs. 54.64 ± 3.456, respectively, $p < 0.001$), diabetic (36.11% vs. 14.3% p value < 0.05), higher prevalence of pseudo-normal and restrictive filling patterns (p value < 0.05), significantly larger LVESV (39.88 ± 13.67 vs. 32.93 ± 9.79 ml, $p < 0.05$), lower EF% (54.13 ± 6.69% vs. 58.54 ± 6.4%, p value < 0.05) and greater WMSI (1.3 ± 0.25 vs. 1.15 ± 0.13, p value < 0.05). 24 h after PCI, 16 (22.22%) improved. 1 m after PCI 61 (84.72%) improved from baseline. Latest activated segment improved in 21.02% after 24 h and 41.69% improved after 1 m. Age was only variable independently associated with non-improvement of LV systolic mechanical dyssynchrony on multivariate analysis. Of the 23 patients with normal EF and WMSI, 13 had systolic dyssynchrony at baseline, 6 improved after 24 h and all improved at 1 m. Diastolic dysfunction improved in 18 (28.125%) 24 h after PCI. After 1 m improved in 50 (78.125%) and remained unchanged in 14 (21.875%), and was closely correlated to the grades of LV diastolic dysfunction.

Conclusion: PCI had significant effect on LV systolic and diastolic mechanical intraventricular dyssynchrony.

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1. Introduction

Mechanical dyssynchrony is increasingly used to describe the mechanical effects of asynchronous ventricular contraction and relaxation, which may or may not be associated with electrical conduction delay. Although LV dyssynchrony was initially recognized as a phenomenon related only to electrical conduction delay in systolic heart failure with widened QRS complexes, previous studies have reported that it also exists in approximately 30–40% of patients with a normal QRS duration[1] and in a significant number of patients with heart failure and preserved ejection fraction.[2]

Coronary artery disease (CAD) is one of the commonest causes of heart failure with preserved EF; however, there are limited results about mechanical dyssynchrony in CAD patients with preserved EF. Acute myocardial ischemia leads to delayed onset and slower rate of contraction and relaxation in regional myocardial segments and thus may generate LV mechanical dyssynchrony,[3] which may in turn compromise LV systolic and diastolic performance and lead to clinical HF.

Until now however, evaluation of systolic function of the myocardium has mainly depended on detection of global function or analyze changes in time and the conduction function of the heart. New approaches have been developed to investigate local myocardial conduction and systolic function such as Doppler tissue imaging, strain rate imaging and especially TSI.[4]

Schiller et al. used the TSI method to evaluate the myocardial dyssynchrony before and after cardiac pacemaker treatment. They reported that the TSI method is an easy and applicable method in the quantitative detection of regional dyssynchrony.[5]

The aim of the present study was to detect the impact of elective PCI on left intraventricular mechanical dyssynchrony (both systolic and diastolic) in patients with chronic stable angina.

2. Patients and methods

This prospective single center study was conducted at the cardiology department of "Benha University Hospital" from March 2012 to February 2014. The study included 132 patients with chronic stable angina scheduled for elective percutaneous coronary intervention (PCI). Thirty-two patients were excluded from the study due to incomplete follow-up so the final study population comprised 100 patients.

Consent from the patients and the approval from the ethics committee were obtained.

According to the presence of LV systolic mechanical dyssynchrony at baseline measured by TSI 12-segments SD, patients were classified into two groups. The first group included patients with LV systolic mechanical dyssynchrony. The second group included patients without LV systolic mechanical dyssynchrony. One month after PCI patients of the first group were reclassified into patients with improved LV systolic mechanical dyssynchrony and patients with persistent LV systolic mechanical dyssynchrony.

According to the presence of LV diastolic mechanical dyssynchrony at baseline measured by Te-SD, patients were classified into two groups. The first group included patients with LV diastolic mechanical dyssynchrony. The second group included patients without LV diastolic mechanical dyssynchrony. One month after PCI, patients of the first group were reclassified into patients with improved LV diastolic mechanical dyssynchrony and patients with persistent LV diastolic mechanical dyssynchrony.

2.1. Exclusion criteria

Patients with:

(1) Prior myocardial infarction.
(2) Bundle branch block (BBB).
(3) Atrial fibrillation (AF) or flutter.
(4) Previous pacemaker implantation.
(5) Restrictive or dilated cardiomyopathy.
(6) Rheumatic heart disease.
(7) Prosthetic valves.
(8) Severe mitral annular calcification.

2.2. Methods

The following data were collected:

(1) Patient characteristics:
- Demographics data (age and sex) and admission details as Risk factors (DM, HTN, smoking, dyslipidemia and family history of CAD).
- History of ischemic chest pain.
- Physical examination including heart rate and rhythm, systolic and diastolic blood pressure, neck veins, and chest and heart auscultation.

(2) Investigations
- *ECG (electrocardiography)* which was done to detect the presence of:
  - o Ischemic changes in the form of Q waves, ST depression and T wave changes.
  - o AF.
  - o Bundle branch block.
- *Laboratory tests:* Including electrolytes (Na and K), serum creatinine and Lipid profile (cholesterol, triglyceride, HDL and LDL).
- Transthoracic echocardiography:
  Echocardiography was done before PCI, 24 h and one month after elective PCI. All patients were examined in the left lateral position using (Vivid 7, Vingmed-General Electric, Horten, Norway) machine with multi-frequency transducer equipped with DTI software.

I- Conventional echocardiography
- o *Global LV function* was assessed by measuring LV end-diastolic volume (LVEDV), LV end-systolic volume (LVESV) and LVEF from the conventional apical 2- and 4-chamber images, using the biplane Simpson's method.[5]
- o *Wall motion score index (WMSI):* The American Society of Echocardiography has recommended a 16-segment model. This model consists of six seg-

ments at both the basal and mid-ventricular levels and four segments at the apex.[6] The attachment of the right ventricular wall to the left ventricle defines the septum, which is divided at basal and mid-left ventricular levels into antero-septum and infero-septum. Continuing counterclockwise, the remaining segments at both basal and mid-ventricular levels are labeled as inferior, infero-lateral, antero-lateral and anterior. The apex includes septal, inferior, lateral and anterior segments. Each segment was analyzed and scored based on its motion and systolic thickening. Segments were scored as normal (>40 percent thickening with systole) = 1, hypokinesis (10–30 percent thickening) = 2, akinesis (<10 percent thickening) = 3, dyskinesis = 4 and aneurysmatic = 5. WMSI was derived as the sum of all scores divided by the number of segments visualized.

$$\text{WMSI} = \frac{\text{Sum of wall motion scores}}{\text{Number of segments visualized}}$$

- o *Left ventricular diastolic function:* Pulsed-wave mitral inflow Doppler was obtained by placing the Doppler sample volume between the tips of the mitral leaflets. LV diastolic dysfunction was graded according to the diastolic filling pattern.
  - Grade 2 was differentiated from the normal pattern through a decrease in preload, by having the patient perform the Valsalva maneuver, may be able to unmask the underlying impaired relaxation of the LV, decreasing the E/A ratio by more than 0.5.
  - Grade 3 and 4 were differentiated by the Valsalva maneuver which may reverse the restrictive filling pattern to a grade 1 to 2, indicating the reversibility of high filling pressure (grade 3 diastolic filling).

II- *Tissue Doppler imaging (TDI)* was obtained for long axis motion from the apical 4-chamber, 2-chamber and apical long axis views. Two-dimensional echocardiography with color TDI optimized for pulse repetition frequency, color saturation, sector size and depth was obtained to maximize the frame rate to 100 Hz or higher. At least 3 consecutive beats in sinus rhythm were analyzed offline using a workstation.

- 2D color-TDI:
- o The 2D color-TDI cine loops of multiple beats were stored digitally for offline analysis. Myocardial velocity curves were reconstituted offline using the six-basal/six-mid-segmental model, which consisted of septal, lateral, anteroseptal, posterior, anterior and inferior segments at both basal and mid-levels of the left ventricle. The basal segments were sampled just above the mitral annulus, and the mid-segments were sampled at the mid-level of LV.
- o The timing of the beginning and the end of LV ejection (aortic valve opening and closure) and filling (mitral valve opening and closure) periods by continuous-wave Doppler of the aortic forward flow and pulsed-wave Doppler of the mitral inflow was measured.
- o Ts (time to peak myocardial systolic velocity during the ejection period) and Te (time to peak myocardial early diastolic velocity during the filling period) were measured with reference to the onset of the QRS complex.[7]
- o Mechanical dyssynchrony index (Ts-SD or Yu index) is the standard deviation of the time-to-peak systolic velocity (Ts) of the twelve basal and mid segments of the LV. It is normally less than 30 ms and the cut-off value was 33 ms.[7]
- o Te-SD (the standard deviation of the time-to-peak early diastolic velocity "Te" of the twelve basal and mid segments of the LV).[7]

III- Tissue synchronization imaging (TSI)

- TSI Portrays regional dyssynchrony on 2D images by transforming the timing of regional peak positive velocity of TDI data into color codes, which allows immediate visual identification of regional delay in systole by comparing the color mapping of orthogonal walls.[8] The color-coding ranges from green (earliest) to red (latest). The normal myocardium, which achieved Vp (peak velocity) at an early stage of contraction, was coded green, which meant no delay in motion (Tp "time to peak velocity" 20–150 ms). The myocardium showing delayed contraction was coded yellow or red according to the degree of delayed time in Vp. The myocardium that achieved Vp at an advanced stage of contraction or diastolic phase was coded yellow, for a mild to moderate delay (Tp, 150–300 ms) or red for a severe delay (Tp, 300–500 ms).[9]
- The following parameters were measured:
  1- TSI 12 segments SD.
  2- TSI 12 segments maximal difference.
  3- TSI opposing wall delay.

- *Coronary angiography:* All patients underwent standard left heart catheterization and coronary angiography via the femoral approach using standard technique with 7Fr arterial sheath. The extent of C-AD whether single-vessel, 2-vessel or multi-vessel disease was determined. The coronary stenosis severity in the vessel to be monitored during PCI was assessed visually. Coronary angioplasty and stenting were performed using standard techniques. Stents used were either drug eluting stents or bare metal stents.

2.3. Statistical analysis

Data were tabulated and statistically analyzed to evaluate the difference between the groups under the study as regards different parameters. Calculations were done using statistical software package namely (SPSS 19) special package for special sciences. Continuous variables were expressed as means ± SD. Student's t-test was used to compare two groups of unpaired data. Comparisons among patient groups and among various grades of diastolic dysfunction were performed using Pearson chi-square test. Univariate analysis was performed for all clinical and echocardiographic variables. Variables with p value < 0.1 on univariate analysis were tested in the multivar-

iate logistic regression with the forward stepwise method to identify independent associations with LV dyssynchrony.

3. Results

The study sample consisted of 100 patients; 57 patients "57%" were males and 43 patients "43%" were females. Their age ranged from 45 to 69 years with mean age (57.36 ± 4.6) years.

Table 1 Patients' demographic and clinical data.

Age (Mean ± SD)	57.36 ± 4.6 years
Gender	
Males	57 (57%)
Females	43 (43%)
Cardiac risk factors	
Diabetes mellitus	30 (30%)
Hypertension	75 (75%)
Smoking	38 (38%)
Dyslipidemia	44 (44%)
Family history of CAD	7 (7%)
Patients' clinical data	
Heart rate (Mean ± SD)	74.38 ± 9.05 bpm
Systolic blood pressure (Mean ± SD)	125 ± 11.76 mmHg
Diastolic blood pressure (Mean ± SD)	81.65 ± 10.52 mmHg

(Table 1 outlined the clinical parameters for the patients at baseline).

Electrocardiography was normal in 26 patients (26%) and showed ischemic changes in the form of combined ST depression and T wave inversion in 36 patients (36%), down-sloping ST depression in 16 patients (16%), horizontal ST depression in 5 patients (5%) and symmetrical T wave inversion in 17 patients (17%).

Conventional echocardiographic parameters. At baseline, the mean LVEDV and LVESV were 73.13 ± 20.47 ml and 37.93 ± 13.03 ml, respectively. Mean LVEF was 55.36 ± 6.87% and mean WMSI was 1.26 ± 0.23. As regards the LV diastolic function, 12 patients (12%) had normal LV diastolic function, 61 patients (61%) had grade I diastolic dysfunction, 14 patients (14%) had grade II diastolic dysfunction and 13 patients (13%) had grade III diastolic dysfunction.

3.1. Systolic mechanical dyssynchrony

Patients were then classified according to the presence of LV systolic mechanical dyssynchrony at baseline measured by TSI 12-segments SD into patients with LV systolic mechanical dyssynchrony (n = 72, 72%) and without LV systolic mechanical dyssynchrony (n = 28, 28%). Patients with LV systolic mechanical dyssynchrony were significantly older than those without LV systolic mechanical dyssynchrony (58.42 ± 4.617 vs. 54.64 ± 3.456, respectively, $p < 0.001$) (Table 2).

Table 2 Baseline data of patients with versus without LV systolic dyssynchrony.

Variable	LV systolic dyssynchrony (n = 72)	No LV systolic dyssynchrony (n = 28)	P value
Age (Mean ± SD)	58.42 ± 4.617	54.64 ± 3.456	0.001
Gender			
Male	41 (56.9%)	16 (57.1%)	1.0
Female	31 (43.1%)	12 (42.9%)	
Cardiac risk factors			
Diabetes mellitus	26 (36.11%)	4 (14.3%)	0.082
Hypertension	56 (77.8%)	19 (67.9%)	0.304
Smoking	29 (40.3%)	9 (32.1%)	0.452
Dyslipidemia	34 (47.2%)	10 (35.7%)	0.298
Family history of CAD	5 (6.9%)	2 (7.1%)	0.07
Hemodynamics			
Heart rate	75.76 ± 9.55 bpm	70.82 ± 6.49 bpm	0.013
Systolic blood pressure	126.39 ± 12.138 mmHg	121.43 ± 10.08 mmHg	0.058
Diastolic blood pressure	83.4 ± 10.87 mmHg	77.14 ± 8.1 mmHg	0.007
Echocardiographic			
LVEDV (Mean ± SD)	84.9 ± 21.65 ml	78.57 ± 16.54 ml	0.17
LVESV (Mean ± SD)	39.88 ± 13.67 ml	32.93 ± 9.79 ml	0.016
LVEF (Mean ± SD)	54.13 ± 6.69%	58.54 ± 6.4%	0.003
WMSI	1.3 ± 0.25	1.15 ± 0.13	0.002
LV diastolic function			
Normal	7 (9.7%)	5 (17.9%)	0.002
Grade I	38 (52.8%)	23 (82.1%)	
Grade II	14 (19.4%)	0 (0%)	
Grade III	13 (18.1%)	0 (0%)	
Angiographic			
Single vessel disease	37 (51.39%)	25 (89.29%)	0.002
2-Vessel disease	23 (31.94%)	3 (10.71%)	
Multi-vessel disease	12 (16.67%)	0 (0%)	

Table 3 Prevalence of LV systolic and diastolic mechanical dyssynchrony before and 24 h after PCI.

Variable	Before PCI (%)	24 h after PCI (%)
Systolic		
TSI opposing wall delay	68	47
TSI 12 segments maximal difference	70	47
TSI 12 segments SD	72	56
Ts SD	70	45
Ts maximal difference	67	27
Diastolic		
Te-SD > 34	64%	46%

Variable	Pre-PCI	24 h after PCI
LV systolic and diastolic dyssynchrony parameters before and 24 h after PCI		
TSI all segments SD	50.79 ± 27.13ms	36.26 ± 21.75ms
Ts-SD	51.98 ± 27.59ms	34.27 ± 19.11ms
Te-SD	40.40 ± 17.53ms	34.34 ± 15.91ms

Table 4 Demographic and clinical data of patients with improved versus persistent LV systolic dyssynchrony one month after PCI.

Variable	Improved LV systolic dyssynchrony (n = 61)	Persistent LV systolic dyssynchrony (n = 11)	P value
Age (Mean ± SD)	57.64 ± 3.98	62.73 ± 5.66	<0.001
Gender			
Male	34 (55.74%)	7 (63.64%)	>0.05
Female	27 (44.26%)	4 (36.36%)	
Cardiac risk factors			
Diabetes mellitus	15 (24.59%)	11 (100%)	0.001
Hypertension	45 (73.77%)	11 (100%)	0.26
Smoking	24 (39.34%)	5 (45.45%)	0.05
Dyslipidemia	26 (42.62%)	8(72.72%)	0.06
Patients' clinical data			
Heart rate	74.34 ± 9 bpm	83.64 ± 9.01 bpm	0.002
Systolic blood pressure	125.557 ± 11.48 mmHg	130.91 ± 15.14 mmHg	0.058
Diastolic blood pressure	82.38 ± 10.55 mmHg	89.09 ± 11.36 mmHg	0.05

No differences were observed between patients with and without LV systolic mechanical dyssynchrony as regards the risk factors except for DM which was more prevalent among patients with LV systolic mechanical dyssynchrony (26 patients "36.11%" of those with LV systolic mechanical dyssynchrony vs. 4 patients "14.3%" of those without, p value < 0.05) (Table 2).

Coronary angiography and elective PCI were performed as clinically determined and no complications were observed during hospital stay. 62 patients (62%) had single-vessel disease, 26 patients (26%) had 2-vessel disease and 12 patients (12%) had 3-vessel disease.

Patients without LV systolic mechanical dyssynchrony had coronary angiograms showing single-vessel disease 25 patients (89.3%) and only 3 patients (10.7%) had 2-vessel disease (Table 2).

Patients with triple-vessel disease had significantly higher TSI 12 segments SD (65.33 ± 15.6 compared with 48.81 ± 27.81 ms; P < 0.05), Ts-SD (72.5 ± 14.5 compared with 49.18 ± 27.82 ms; P < 0.001) and Te-SD (64.83 ± 13.47 compared with 37.07 ± 15.27 ms; P < 0.001) than those with single or double-vessel diseases suggesting the important role of myocardial ischemia in the pathogenesis of mechanical dyssynchrony in patients with CAD.

Diastolic dysfunction was more severe in patients with LV systolic mechanical dyssynchrony, who had a higher prevalence of pseudo-normal and restrictive filling patterns (p value = 0.002) (Table 2).

Twenty-four hours after PCI all the patients were assessed again with further echocardiography. Twenty-four hours after elective PCI, the latest activated segment improved by 21.02% from baseline values. (Table 3 *outlines the prevalence and parameters of systolic and diastolic dyssynchrony before and 24 h after PCI*).

One month after PCI latest activated segment improved by 41.69% from baseline values.

Pre- and 1 month after PCI time to peak systolic velocity (Ts) values were measured in 12 segments by TSI. The Ts values of all segments were significantly decreased in post-PCI measurements (p value < 0.001).

One month after elective PCI patients were then reclassified according to the presence of LV systolic mechanical dyssynchrony measured by TSI 12-segments SD into patients with improved LV mechanical dyssynchrony (n = 61, 84.72%)

Table 5 Demographic and clinical data of patients with improved versus persistent LV diastolic dyssynchrony one month after PCI.

Variable	Improved LV diastolic dyssynchrony (n = 50)	Persistent LV diastolic dyssynchrony (n = 14)	P value
Age (Mean ± SD)	57.48 ± 4.21	62.43 ± 3.3	0.001
Gender			
Male	27 (54%)	9 (64.3%)	>0.05
Female	23 (46%)	5 (35.7%)	
Cardiac risk factors			
Diabetes mellitus	12 (24%)	11 (78.6%)	<0.001
Hypertension	37 (74%)	13 (92.9%)	0.08
Smoking	20 (40%)	8 (57.1%)	0.058
Dyslipidemia	24 (48%)	6 (42.9%)	0.09
Patients' clinical data			
Heart rate	75.62 ± 8.97 bpm	76.86 ± 10.82 bpm	0.66
Systolic blood pressure	125.4 ± 11.64 mmHg	133.57 ± 10.82 mmHg	0.02
Diastolic blood pressure	81.4 ± 10.5 mmHg	90.7 ± 8.29 mmHg	0.003
Echocardiography			
LVEDV (Mean ± SD)	83.8 ± 18.33 ml	104.36 ± 18.81 ml	0.001
LVESV (Mean ± SD)	38.74 ± 11.19 ml	53.57 ± 10.31 ml	0.001
LVEF (Mean ± SD)	54.06 ± 5.22%	48.86 ± 3.46%	0.001
WMSI	1.27 ± 0.2	1.55 ± 0.26	0.001

and patients with persistent LV mechanical dyssynchrony (n = 11, 15.28%) (Table 4).

3.2. Patients with normal EF and WMSI

At baseline there were 23 patients with normal EF and WMSI; 13 patients had LV systolic dyssynchrony based on TSI 12 segments SD, 24 h after PCI, 4 patients had LV systolic dyssynchrony and 1 month after PCI, none of the patients had LV systolic dyssynchrony. And 84 patients had normal EF in spite of WMA; of them 56 patients had LV systolic dyssynchrony based on TSI 12 segments SD. 24 h after PCI, 43 patients had LV systolic dyssynchrony. 1 month after PCI, 5 patients had LV systolic dyssynchrony.

Several variables were associated with non-improvement of LV systolic mechanical dyssynchrony on univariate analysis including the age, the presence of DM, heart rate, echocardiographic parameters (LVEDV, LVESV, EF, WMSI and LV diastolic function) and the presence of multi-vessel disease. Age was only variable independently associated with non-improvement of LV systolic mechanical dyssynchrony on multivariate analysis.

3.3. LV diastolic mechanical dyssynchrony

24 h after elective PCI; 46% of patients had evidence of significant LV diastolic dyssynchrony based on Te-SD. One month after elective PCI, 14% of patients had evidence of significant LV diastolic dyssynchrony based on Te-SD. Te-SD decreased significantly post-PCI (40.4 ± 17.5 vs. 25.64 ± 12.5 ms, p value < 0.001). One month after PCI, patients were divided into patients with improved diastolic dyssynchrony (n = 50, 78.1%) and patients with persistent diastolic dyssynchrony (n = 14 (21.9%)) (Table 5).

One month after PCI, most of the patients with improved LV diastolic mechanical dyssynchrony had coronary angiograms showing single-vessel disease 28 patients (56%), 16 patients (32%) had 2-vessel disease and 6 patients (12%) had multi-vessel disease. Most of the patients with persistent LV diastolic mechanical dyssynchrony had coronary angiograms showing 2-vessel disease in 7 patients (50%) and multi-vessel 6 patients (42.9%) but only 1 patient (7.1%) had single-vessel disease.

Patients with persistent LV diastolic dyssynchrony 1 month after PCI had more severe LV diastolic dysfunction (P value < 0.001) (Table 5).

Several variables were associated with non-improvement of LV diastolic mechanical dyssynchrony on univariate analysis including the age, the presence of DM, systolic and diastolic blood pressure, echocardiographic parameters (LVEDV, LVESV, EF, WMSI and LV diastolic function) and the presence of 2- and multi-vessel disease.

4. Discussion

The aim of the present study was to detect the impact of elective PCI on left intraventricular mechanical dyssynchrony (both systolic and diastolic) in patients with chronic stable angina.

In the present study, systolic mechanical dyssynchrony was highly prevalent in CAD patients. At baseline, significant LV systolic dyssynchrony was present in 72% of the patients based on TSI 12 segments SD, a finding consistent with previous reports by Lee et al.[10]

In our study 23 patients had normal EF and WMSI 13 of them had systolic dyssynchrony at base line; of them 6 improved 24 h after PCI and all improved at 1 m and 84 patients had normal EF, 56 of them had systolic dyssynchrony at baseline, 13 patients improved after 24 h and 38 improved at 1 m.

This is due to the important role of myocardial ischemia in the pathogenesis of mechanical dyssynchrony in CAD. As changes in timing of regional mechanical events may precede local motion abnormalities during myocardial ischemia,

mechanical dyssynchrony could be present when LVEF is relatively preserved.

Also, Lee et al.[11] assessed LV mechanical dyssynchrony in heart failure with preserved ejection fraction complicating acute coronary syndrome. They found that Ts-SD was increased in both ACS groups (patients with and without heart failure) compared with controls.

Similarly, Ng et al.[12] reported that left ventricular dyssynchrony was present in a significant proportion of patients early after acute myocardial infarction in the absence of congestive heart failure.

In the present study no significant difference in LVEDV was observed between patients with and without LV systolic mechanical dyssynchrony. Patients with LV systolic mechanical dyssynchrony had larger LVESD, lower EF and greater WMSI.

This is consistent with Ng et al.[12] who found that patients with evidence of LV dyssynchrony on all four dyssynchrony parameters had the largest LVESV and lowest LVEF at all-time intervals.

In the current study diastolic dysfunction assessed by pulsed-wave mitral inflow Doppler was more severe in patients with LV systolic mechanical dyssynchrony, who had a higher prevalence of pseudonormal and restrictive filling patterns.

This is in agreement with the results reported by Tanaka et al.[13] They found that patients with mechanical dyssynchrony had greater diastolic dysfunction and prevalence of restrictive filling.

In the present study diastolic mechanical dyssynchrony was highly prevalent in CAD patients. At baseline, 64% of patients had evidence of significant LV diastolic dyssynchrony based on Te-SD.

As myocardial ischemia is often a regional phenomenon, it is possible that regional delay in relaxation leads to diastolic mechanical dyssynchrony during IHD. Among patients with chronic coronary artery disease and preserved LV systolic function, diastolic mechanical dyssynchrony has been shown to predict exercise induced ischemia with resultant impairment of early ventricular filling.[14]

Of patients with LV systolic mechanical dyssynchrony, 37 patients (51.39%) had single-vessel disease, 23 patients (31.94%) had 2-vessel disease and 12 patients (16.67%) had multi-vessel disease. Patients with triple-vessel disease had significantly higher TSI 12 segments SD, Ts-SD and Te-SD than those with single or double-vessel diseases suggesting the important role of myocardial ischemia in the pathogenesis of mechanical dyssynchrony in patients with CAD.

This is in agreement with the results reported by Lee et al.[11] They found that patients with triple-vessel or left main diseases had significantly higher Ts-SD (48.9 ± 12.9 compared with 21.6 ± 9.7 ms; $P < 0.001$) and Te-SD (36.2 ± 19.1 compared with 21.7 ± 6.2 ms; $P < 0.001$) than those with single or double-vessel diseases. In another clinical study, the time to peak systolic velocity measured by TDI was found to be associated with coronary stenosis in patients with chest pain who had no apparent ventricular wall motion abnormalities on echocardiography.[14]

In the current study, all the patients were assessed again with further echocardiography twenty-four hours and one month after PCI. Twenty-four hours after elective PCI, the latest activated segment improved by 21.02% from baseline values. One month after PCI, it improved by 41.69% from baseline values.

Pre- and one month after PCI time to peak systolic velocity (Ts) values were measured in 12 segments. The Ts values of all segments were significantly decreased in post-PCI measurements ($p < 0.001$).

This is consistent with Inci et al.[15] who reported that in the anterior AMI group, the Ts values of the basal septal, basal anterior and mid anterior segments were significantly decreased in post-PCI measurements (p value < 0.01). No significant difference was observed in the Ts values of the basal lateral, mid-lateral, mid-septal, basal inferior, or the mid-inferior segments in post-PCI measurements as PCI was done to the left anterior descending artery (infarct related artery). In the inferior AMI group, Ts values of the basal septal, mid-septal, basal inferior and mid-inferior segments significantly decreased in post-PCI measurements (p value < 0.01). No statistical significance was observed in the Ts values of the basal lateral, mid-lateral, basal anterior, or the mid-anterior segments in post-PCI measurements as PCI was done to the right coronary and the left circumflex artery.

In the current study, twenty-four hours after elective PCI, significant LV systolic dyssynchrony was evident in 56% of the patients based on TSI 12 segments SD. One month after elective PCI, significant LV systolic dyssynchrony was evident in 11% of the patients based on TSI 12 segments SD.

The prevalence of LV systolic mechanical dyssynchrony and its improvement after elective PCI in patients with chronic stable angina were firstly evaluated in this study. In previous studies, there were no pre- or post-PCI comparisons made. 24 h after elective PCI, LV systolic mechanical dyssynchrony measured by TSI 12-segments SD decreased in 16 patients (22.22%) and remained unchanged in 56 patients (77.78%). One month after elective PCI, LV systolic mechanical dyssynchrony measured by TSI 12-segments SD decreased in 61 patients (84.72%) and remained unchanged in 11 patients (15.28%).

This is similar to Babaee Beigi et al.[16] who evaluated left ventricular dyssynchrony after coronary artery bypass grafting in patients with ischemic left ventricular dysfunction and reported that LV dyssynchrony decreased in 24 patients (80%) and remained unchanged in 7 patients (20%). Also, Inci et al.[15] reported that there was a significant decrease in LV dyssynchrony after primary PCI. Myocardial ischemia is one of the major causes of LV dyssynchrony and so correction of ischemia by revascularization (PCI or CABG) may resynchronize LV contraction.

Also, LV diastolic mechanical dyssynchrony pre- and post-PCI in patients with chronic stable angina was firstly assessed in this study. Twenty-four hours after elective PCI, LV diastolic mechanical dyssynchrony measured by Te-SD decreased in 18 patients (28.125%) and remained unchanged in 46 patients (71.875%). One month after elective PCI, LV diastolic mechanical dyssynchrony measured by Te-SD decreased in 50 patients (78.125%) and remained unchanged in 14 patients (21.875%).

Predictors of non-improvement of LV mechanical dyssynchrony one month after PCI were assessed. Several variables were associated with non-improvement of LV systolic mechanical dyssynchrony on univariate analysis including the age, the presence of DM, heart rate, echocardiographic parameters (LVEDV, LVESV, EF, WMSI and LV diastolic function) and the presence of multi-vessel disease. The age was the only variable independently associated with non-improvement of LV systolic mechanical dyssynchrony on multivariate analysis.

Predictors of non-improvement of LV diastolic mechanical dyssynchrony on univariate analysis included the age, the presence of DM, systolic and diastolic blood pressure, echocardiographic parameters (LVEDV, LVESV, EF, WMSI and LV diastolic function) and the presence of 2- and multi-vessel disease.

5. Conclusion

PCI had significant effect on LV systolic and diastolic mechanical dyssynchrony.

Conflict of interest

We have no conflict of interest to declare.

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