Article published online: 2021-08-09

ORIGINAL ARTICLE

Left ventricular dyssynchrony assessment using tissue synchronization imaging in acute myocardial infarction

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Access this article online

Website: www.avicennajmed.com

DOI: 10.4103/ajm.AJM_168_18

Quick Response Code:

ABSTRACT

Objectives: To assess left ventricular (LV) dyssynchrony in patients with ST elevation myocardial infarction (STEMI). **Background:** Mechanical synchronization disorder leads to a decrease in LV ejection fraction (LVEF) and stroke volume, an abnormal distribution of wall tension, and increase in workload during cardiac contraction. **Methods:** We enrolled 56 participants, 36 with acute STEMI and 20 healthy controls. The automatically color-coded time to peak myocardial velocity was measured using a 6 mm sample volume, manually positioned within the two-dimensional-tissue strain image of the 12 basal and middle LV segments. **Results:** A significant delay was found between the septal-lateral and septal-posterior walls in patients with STEMI compared to patients in the control group (36.36 vs. –6.0 ms, $P = 0.036$; and 42.7 vs. 23.94 ms, $P = 0.042$, respectively). Furthermore, all segment maximum differences and all segment standard deviation (SD; dyssynchrony index) were found to be significantly higher in the STEMI group (131.28 vs. 95.45 ms, $P = 0.013$; and 44.47 vs. 26.45 ms, $P = 0.001$, respectively). A significant delay between the septal-lateral walls and septal-posterior walls, all segment maximum difference, and all segment SD (dyssynchrony index) were found in patients with complicated STEMI (70.89 vs. 15.83 ms, $P = 0.038$; 57.44 vs. 19.06 ms, $P = 0.040$; 138.11 vs. 100.0 ms, $P = 0.035$; and 45.44 vs. 32.50 ms, $P = 0.021$, respectively). There was a significant negative correlation between tissue synchronization imaging parameters and LVEF, and a positive correlation with LV end systolic dimension. **Conclusion:** Patients with acute STEMI showed significant LV dyssynchrony, which was an independent predictor of inhospital complications.

Key words: Left ventricular dyssynchrony, myocardial infarction, tissue synchronization imaging

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.[1] Left ventricular (LV) dyssynchrony is observed in 30–40% of patients with a normal QRS duration[1] and in a significant number of patients with heart failure (HF) and preserved LV ejection fraction (LVEF).[2] Coronary artery disease (CAD) is one of the most common causes of HF with preserved left ventricular ejection fraction (LVEF); however, there are limited results about mechanical dyssynchrony in patients with CAD with preserved LVEF.[3] Acute myocardial infarction leads to a delayed onset and slower rate of contraction and relaxation in regional myocardial segments and may cause LV mechanical dyssynchrony and subsequent clinical HF.[3] Local myocardial conduction and systolic function may be

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Cite this article as: Azazy SA, Soliman M, Yaseen R, Mena M, Sakr H. Left ventricular dyssynchrony assessment using tissue synchronization imaging in acute myocardial infarction. Avicenna J Med 2019;9:48-54.

assessed using tissue Doppler imaging (TDI), strain rate imaging, or tissue synchronization imaging (TSI).[4] 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.[4] Approaches have been developed to investigate local myocardial conduction and systolic function such as Doppler tissue imaging, strain rate imaging, and especially TSI.[4] We aimed to assess LV dyssynchrony in patients with ST elevation myocardial infarction (STEMI) using TSI.

MATERIALS AND METHODS

This study included 36 patients that presented with acute STEMI who were recruited from the coronary care unit at a university hospital. The control group comprised 20 age and sex-matched healthy individuals.

Patients with a wide QRS complex (≥120 ms), myocardial diseases (hypertrophic cardiomyopathy, restrictive cardiomyopathy, or dilated cardiomyopathy), paced rhythm, rheumatic heart disease with significant valvular lesions, previous open heart surgery, and poor echocardiographic windows were excluded from the study. Written informed consent for participation was obtained, and the hospital ethics committee approved the protocol.

All participants in the study were subjected to the following: full history taking, thorough clinical examination, 12-lead electrocardiography (ECG) and echocardiographic examination. Transthoracic echocardiographic examination was performed using a commercially available system (Vivid 9; GE Vingmed Ultrasound AS, Horten, Norway) equipped with a 1.7–4 MHz phased-array transducer with simultaneous ECG tracing. Echocardiography was performed within 24 h of the admission in the left lateral position, according to the recommendations of the American Society of Echocardiography.[5]

Conventional echocardiography

M-mode echocardiography

M-mode echocardiography was performed using the parasternal long axis view with the M-mode cursor perpendicular to the interventricular septum and posterior wall at the level of the mitral valve tip for measurement of LV end-diastolic diameter (LVEDD), LV end-systolic diameter (LVESD), interventricular septal thickness in diastole, posterior wall thickness in diastole, fractional shortening, and LVEF.

Two-dimensional echocardiography

Two-dimensional (2D) echocardiography for assessment of wall motion abnormality and estimation of EF was performed using Simpson's method.

Doppler echocardiography

For measurement of peak E (early diastolic) wave velocity, peak A (late diastolic) wave velocity, and the E/A ratio with pulsed Doppler sample volume, the transmitral Doppler probe was placed in the middle of the LV inflow tract 1 cm below the plane of the mitral annulus in the apical four-chamber view. Trans-aortic Doppler in the apical five-chamber and three-chamber views was used for measuring aortic valve opening and closure.

Tissue strain imaging

The automatically color-coded time to peak myocardial velocity (Ts) was measured using a 6 mm sample volume manually positioned within the 2D TSI image for 12 LV segments. The 12 segments included 6 basal and 6 mid-wall segments of opposing LV walls in apical two-, three-, and four-chamber views. At least three consecutive beats on TSI were stored and the images were analyzed offline using a customized software package (EchoPAC for PC; GE Vingmed Ultrasound) [Figure 1]. To prevent the TSI system from measuring peak systolic velocities outside the ejection phase, the event-timing tool was used to manually adjust start and end times of the aortic valve ejection. Parameters of systolic dyssynchrony were computed using the software. The parameters included standard deviation (SD) of Ts of the 12 LV segments (dyssynchrony index), septal-lateral delay, septal-posterior delay, and all segmental maximum difference. The dyssynchrony index is the most widely used parameter for LV dyssynchrony, which is defined as a dyssynchrony index >34.4 ms on TSI.[6] LV systolic dyssynchrony is defined as septal-lateral delay or septal-posterior delay ≥2 SDs above the control.[7]

STEMI complications

All patients were followed up for any inhospital post-STEMI complications including death, cardiogenic shock, pulmonary edema, arrhythmia, stroke, acute kidney injury, severe mitral regurgitation, ventricular septal rupture, and complete heart block.

Statistical analysis

Statistical analysis was performed using IBM-SPSS (version 20; IBM Corp, Armonk, NY) software. Mean and SD were computed for all quantitative variables. Student's *t*-test was used to compare quantitative data between the STEMI and healthy control groups, patients with and without anterior STEMI, and patients with and without STEMI complications. The chi-square test was used to compare qualitative data. The Pearson correlation test was used to analyze the TSI parameters correlated with LVEF, LVESD, and LVESD in the STEMI group. $P < 0.05$ was considered statistically significant.

RESULTS

Baseline characteristics

The cohort comprised 36 patients presenting with acute STEMI (mean age, 54 ± 10.8 years) and 20 age and sex-matched healthy volunteers who represented the control group (mean age, 49.1 ± 7.5). Among the patients in the STEMI group, 41.7% were hypertensive, 33.3% were diabetic, 33.3% had dyslipidemia, and 55.6% were smokers [Table 1].

In the STEMI group, 47% (n = 17) of patients developed post-STEMI complications: 35.2% (6) had pulmonary edema,11.7% (2) had cardiogenic shock, 11.7% (2) had stroke, 17.6% (3) had ventricular tachycardia, 17.6% (3) had heart block, and 5.7% (1) had ventricular septal rupture.

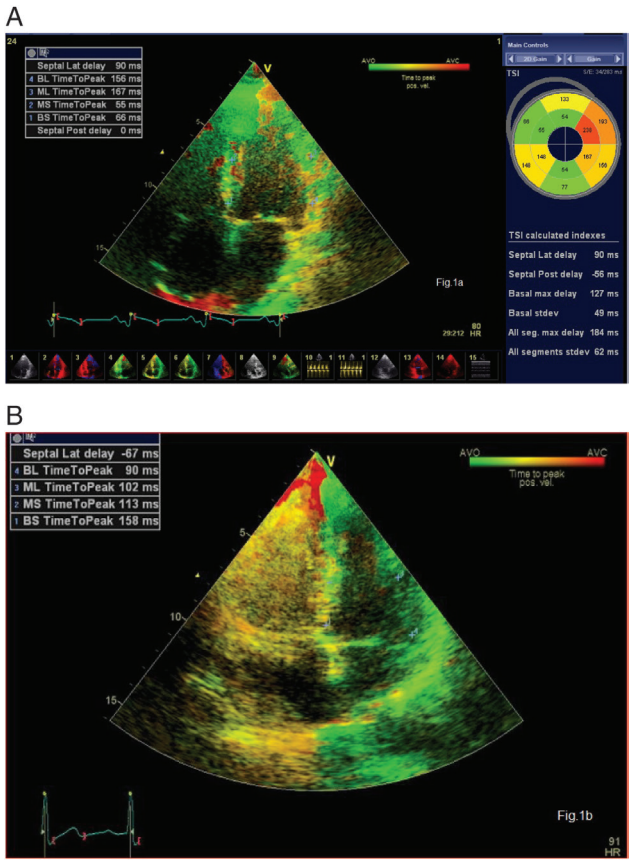

Figure 1: (A) Tissue synchronization imaging (TSI) of the LV in patient with anterior myocardial Infarction (apical four-chamber view) demonstrating cursor placement for auto-TSI analysis; the bulls-eye diagram for time to peak velocity measurements showed LV dyssynchrony as dyssynchrony index was 62 ms. (B) TSI of the LV in control (apical four-chamber view)

Table 1: Demographic data of the studied groups

	Patients group (N = 36)
Woman, n (%)	8 (22.2%)
Men, n (%)	28 (77.8%)
Age (years), Mean ± standard deviation	54.06 ± 10.81
Hypertension n (%)	15 (41.7%)
Diabetes mellitus, n (%)	12 (33.3%)
Dyslipidemia, n (%)	12 (33.3%)
Smoking, n (%)	20 (55.6%)

Conventional echocardiographic parameters in the STEMI and control groups

The left atrial (LA) diameters, LVEDD, LVESD, and A-wave velocity were significantly higher in the STEMI group than in the control group (3.79 cm vs. 3.31 cm, P = 0.0001; 3.74 cm vs. 2.97 cm, P = 0.001; and 5.27 cm vs. 4.94 cm, P = 0.006, respectively). On the other hand, the E/A ratio and LVEF were significantly lower in the STEMI group (1.01 vs. 1.30, P = 0.002; and 49.03% vs. 68.45%, P = 0.0001, respectively) [Table 2].

TSI parameters in the STEMI and control groups

Significantly longer septal-lateral and septal-posterior delays were found in the STEMI group than in the control group (36.36 ms vs. −6.0 ms, P = 0.036; and 42.7 ms vs. 23.94 ms, P = 0.042, respectively). In addition, all segment maximum differences and all segment SD (dyssynchrony index) were found to be significantly higher in the patients with STEMI than in the controls (131.28 ms vs. 95.45 ms, P = 0.013; and 44.47 ms vs. 26.45 ms, P = 0.001, respectively) [Table 3; Figure 1].

Conventional echocardiographic parameters in patients with and without anterior STEMI

We found that LVEDD, LVESD, and A-wave velocity were significantly higher in patients with anterior STEMI (5.28 cm vs. 4.98 cm, P = 0.012; 3.67 cm vs. 3.19 cm, P = 0.043; and 0.73 m/s vs. 0.58 m/s, P = 0.020 respectively). However, the LVEF and E/A ratio were significantly lower in patients with anterior STEMI (46.25% vs. 54.58%, P = 0.018; and 0.88 vs. 1.53, P = 0.0001, respectively). Interestingly, LA dimension and E-wave velocity were not significantly different (P = 0.724 and P = 0.317, respectively) [Table 2].

TSI parameters in patients with and without anterior STEMI

Septal-lateral and septal-posterior wall delays, all segment maximum difference, and all segment SD (dyssynchrony index) were significantly higher in patients with anterior STEMI than in those without (57.21 ms vs. −5.82 ms, P = 0.022; 55.75 ms vs. 16.67 ms, P = 0.001; 148.29 ms vs. 97.25 ms, P = 0.005; and 47.21 ms vs. 36.91 ms, P = 0.017, respectively) [Table 3].

TSI parameters in patients with and without complicated STEMI

A significant increase in delay between the septal-lateral walls and septal-posterior walls, all segment maximum difference, and all segment SD (dyssynchrony index) was

observed in patients with complicated STEMI (70.89 ms vs. 15.83 ms, $P = 0.038$; 57.44 ms vs. 19.06 ms, $P = 0.040$; 138.11 ms vs. 100.0 ms, $P = 0.035$; and 45.44 ms vs. 32.50 ms, $P = 0.021$, respectively) [Table 4].

Correlation between conventional echocardiographic and TSI parameters in STEMI group

Across the entire study population, we found that there was a highly significant negative correlation between LVEF and TSI parameters, such as septal-lateral wall delay ($r = -0.665$; $P = 0.0001$), septal-posterior wall delay ($r = -0.978$; $P = 0.0001$), all segments maximum difference ($r = -0.557$; $P = 0.0001$), and dyssynchrony index ($r = -0.608$; $P = 0.0001$). In contrast, there was a positive correlation between LVESD and septal-lateral wall delay ($r = 0.250$; $P = 0.001$), septal-posterior wall delay ($r = 0.068$; $P = 0.001$), all segments maximum difference ($r = 0.257$; $P = 0.001$), and dyssynchrony index ($r = 0.523$; $P = 0.001$) [Table 5; Figure 2].

DISCUSSION

After myocardial infarction (MI), LV global contraction is asynchronous due to the partial reduction or even the loss of infarct myocardial contractility, which ultimately results in LV global remodeling and dysfunction. Furthermore, MI occurring in different segments is associated with variable effects on LV function and clinical prognosis.[7]

Among the various echocardiographic techniques, TDI has gained acceptance by virtue of the ability to define regional timing and contractility as well as its reproducibility. Recently, TDI has evolved into another technical modality,

Table 2: Comparison between echocardiographic parameters in the studied groups (A) and comparison of echocardiographic parameters between patients with anterior myocardial infarction (MI) and non-anterior MI (B)

	A			B		
	STEMI group (*n* = 36)	Control group (*n* = 20)	*P*-value	Anterior (*n* = 24)	Non-anterior (*n* = 12)	*P*-value
Aortic opening (cm), mean ± SD	2.69 ± 0.17	2.59 ± 0.18	0.070	3.17 ± 0.37	2.98 ± 0.45	0.175
Left atrium (cm), mean ± SD	3.79 ± 0.56	3.31 ± 0.16	<0.0001	3.81 ± 0.62	3.74 ± 0.43	0.724
LVESD (cm), mean ± SD	3.74 ± 0.83	2.97 ± 0.75	0.001	3.67 ± 0.84	3.19 ± 0.88	0.043
LVEDD (cm), mean ± SD	5.27 ± 0.47	4.94 ± 0.31	0.006	5.28 ± 0.49	4.98 ± 0.31	0.012
Ejection fraction (%), mean ± SD	49.03 ± 10.13	68.45 ± 3.68	<0.0001	46.25 ± 8.19	54.58 ± 11.64	0.018
E m/s, mean ± SD	0.68 ± 0.20	0.71 ± 0.11	0.603	0.66 ± 0.20	0.73 ± 0.18	0.317
A m/s, mean ± SD	0.68 ± 0.17	0.57 ± 0.06	0.009	0.73 ± 0.17	0.58 ± 0.12	0.020
E/A, mean ± SD	1.01 ± 0.34	1.30 ± 0.27	0.002	0.88 ± 0.29	1.53 ± 0.62	<0.0001

LVESD = left ventricle end-systolic diameter, LVEDD= left ventricle end-diastolic diameter, SD = standard deviation, STEMI = ST elevation myocardial infarction

Table 3: Comparison of tissue synchronization imaging (TSI) parameters in studied groups (A) and comparison of TSI parameters between patients with anterior myocardial infarction (MI) and non-anterior MI (B)

	A			B		
	STEMI group (*n* = 36)	Control group (*n* = 20)	*P*-value	Anterior (*n* = 24)	Non-anterior (*n* = 12)	*P*-value
Septal lateral delay (ms), Mean ± SD	36.36 ± 75.89	−6.00 ± 59.98	0.036	57.21 ± 66.45	−5.82 ± 82.87	0.022
Septal post delay (ms), Mean ± SD	42.72 ± 35.40	23.94 ± 15.19	0.042	55.75 ± 29.08	16.67 ± 33.30	0.001
All segment maximum difference (ms) Mean ± SD	131.28 ± 53.73	95.45 ± 41.58	0.013	148.29 ± 54.12	97.25 ± 34.13	0.005
All segment SD (ms) (Dyssynchrony index) Mean ± SD	44.47 ± 15.82	26.45 ± 7.06	<0.001	47.21 ± 19.03	36.91 ± 8.89	0.017

SD = standard deviation, STEMI = ST elevation myocardial infarction

Table 4: Comparison of tissue synchronization imaging parameters between complicated patients and noncomplicated patients

	Complicated STEMI (*n* = 17 [47%])	Noncomplicated STEMI (*n* = 19 [53%])	Independent *t*-test	
			t	*P*-value
Septal lateral delay (ms) Mean ± SD	70.89 ± 78.05	15.83 ± 74.64	2.163	0.038
Septal post delay (ms) Mean ± SD	57.44 ± 50.96	19.06 ± 56.83	−2.134	0.040
All segment maximum difference (ms) Mean ± SD	138.11 ± 62.38	100.00 ± 39.07	2.197	0.035
All segment SD (ms) Mean ± SD	138.11 ± 62.38	32.50 ± 13.26	2.422	0.021

SD = standard deviation, STEMI = ST elevation myocardial infarction

TSI. Tissue imaging portrays regional asynchrony on 2D echocardiography by transforming the timing of regional peak velocity into color codes. This allows for immediate visual identification of regional delay in systole by comparing the color mapping of orthogonal walls. In addition, quantitative measurement of regional delay is possible. However, the ability of TSI to assess systolic asynchrony and predict a positive response to cardiac resynchronization therapy has not been explored.[8-10]

LV mechanical dyssynchrony leads to a decrease in ejection fraction and stroke volume, an abnormal distribution of wall tension, and increased workload during cardiac contraction.[11-13] In fact, LV systolic function failure is a grave complication after MI. Thus, an accurate and detailed assessment of LV remodeling and systolic dyssynchrony carries significant implications for clinical management and prognosis. LV dyssynchrony includes both mechanical and electrical dyssynchrony, and the former has been commonly accepted as a direct indicator of LV systolic dyssynchrony.[9]

In the current study, we found that patients with STEMI had significant LV systolic dyssynchrony compared to controls. Furthermore, we found that LV dyssynchrony is more common in patients with anterior STEMI. Ng *et al.*[14] reported similar results, where LV dyssynchrony was present in a significant proportion of patients early after acute MI in the absence of congestive HF. In the current study, LVESD was significantly higher in patients with STEMI than in controls, whereas LVEDD was not significantly different. Similarly, Mollema *et al.*[15] found that the incidence of LV dilatation after acute MI was not markedly increased; however, LVESV was significantly larger in patients who died from a cardiac cause than in survivors.

LA diameter in our study was significantly larger in the patients with STEMI than in the controls, which can be

Table 5: Correlation between conventional echocardiographic parameters and tissue synchronization imaging parameters in ST elevation myocardial infarction patients

		Septal lateral delay (ms)	Septal post delay (ms)	All segment maximum difference (ms)	All segment SD (ms)
LVEDD (cm)	*r*	0.228	−0.125	0.262	0.283
	P value	0.181	0.468	0.123	0.094
LVESD (cm)	*r*	0.250	0.068	0.257	0.523
	P value	**0.002**	**0.002**	**0.001**	**0.001**
Ejection fraction (%)	*r*	−0.665	−0.978	−0.557	−0.608
	P value	**0.0002**	**0.0001**	**0.0001**	**0.0001**
Aortic opening (cm)	*r*	0.058	0.211	0.08	0.151
	P value	0.742	0.217	0.642	0.378
Left atrium (cm)	*r*	0.213	0.003	−0.11	0.222
	P value	0.220	0.984	0.525	0.193
E	*r*	0.173	−0.048	0.312	0.296
	P value	0.319	0.780	0.064	0.080
A	*r*	0.441	0.180	0.166	0.201
	P value	0.008	0.293	0.334	0.239
E/A	*r*	−0.308	−0.262	−0.114	−0.083
	P value	0.072	0.122	0.509	0.631

SD = standard deviation, LVEDD = left ventricle end-diastolic diameter, LVESD = left ventricle end-systolic diameter
P < 0.05

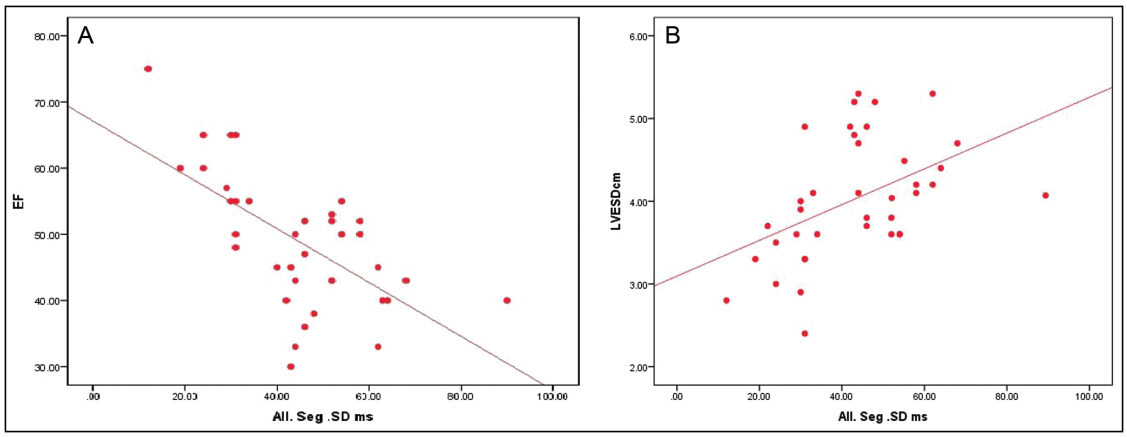

Figure 2: Correlation chart in STEMI between (A) EF and dyssynchrony index and (B) LVESD and dyssynchrony index. LVESD = left ventricular end-systolic diameter, STEMI = ST elevation myocardial infarction

explained by the increase in the LA pressure as a result of post-MI diastolic dysfunction, higher LV filling pressure, and/or mitral regurgitation.[16] Our findings agree with those of Meris *et al.*[17] who concluded that early LA remodeling and size after MI is an independent predictor of death or hospitalization for HF in patients with high-risk MI. In addition, they found that the risk appears to be continuous even in patients with a slightly larger LA.[17,18]

The patients with anterior STEMI in our study had a significantly lower LVEF, higher LV volumes, and frequent LV systolic dyssynchrony than those with a non-anterior STEMI. Moreover, inhospital complications, especially HF, were more frequent in patients with anterior STEMI. This may be due to the extensive myocardial necrosis and greater myocardial damage, which leads to decreased LV systolic dysfunction in anterior MIs.[19]

The findings of our study were consistent with those of Zhang *et al.*,[20] who found that peak systolic velocity durations were significantly longer in patients with acute MI, especially those with anterior infarcts. However, their study employed cardiac magnetic resonance to assess the size and location of the infarction. Of note, our study showed a significant negative correlation between all TSI parameters and LVEF. This was in agreement with Zhou *et al.*[21] who evaluated the relation between LVEF and LV dyssynchrony, and reported that the LV dyssynchrony occurred more often in patients with cardiac dysfunction after MI, and was significantly related to LVEF. Furthermore, our study found a significant positive correlation between all TSI parameters and LVESD, which was consistent with the results of Mollema *et al.*[15] and Ng *et al.*[14]

The clinical implication of our study is to draw attention to the importance of assessment of LV dyssynchrony in patients with acute myocardial infarction, which necessitates early aggressive treatment and longer follow-up.

LIMITATIONS OF THE STUDY

Our study is limited by its small sample size. Therefore, larger numbers of patients with longer follow-ups should be recruited in subsequent studies. Another limitation is that LV dyssynchrony is affected by other factors, such as hypertension and diabetes mellitus. However, because numerous patients with STEMI exhibit these risk factors, we did not exclude them from our study. Further studies focusing on the effect of ischemia itself on LV dyssynchrony are needed. Finally, we did not study the effect of percutaneous coronary intervention on LV dyssynchrony.

CONCLUSION

Patients with acute STEMI, particularly anterior infarcts, showed significant LV dyssynchrony, which is an independent predictor of inhospital complications and is closely related to the size of the myocardial infarction. Aggressive treatment is highly recommended in such patients.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

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