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A Study of Left Ventricular Dyssynchrony After ST Elevated MI

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Abstract

Background: To evaluate left ventricular dyssynchrony after ST elevated MI.

Materials & methods: A total of 80 subjects were enrolled. 40 were with acute STEMI and 40 healthy controls. The complete history was taken, and 12-lead electrocardiography (ECG) and echocardiographic examination was done. $P < 0.05$ was considered statistically significant.

Results: LVEDD and LVESD, were significantly higher in the STEMI group than in the control group (5.4cm vs. 4.6cm, P = 0.0007; and 3.5cm vs. 2.8cm, P = 0.001 respectively).

Conclusion: Patients with acute STEMI showed significant LV dyssynchrony.

Keywords: Left Ventricular Dyssynchrony, Myocardial Infarction, ST Elevation MI.

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 assessed using tissue Doppler imaging (TDI), strain rate imaging, or tissue synchronization imaging (TSI).[4] Dyssynchrony of left ventricular (LV) contraction is an indicator of a temporal discrepancy in myocardial contraction among different segments of the left ventricle. The concept of dyssynchrony has been more frequently proposed since the introduction of cardiac resynchronization therapy (CRT) for intractable heart failure.[5,6] Various parameters representing dyssynchrony have been evaluated for validation as predictors of response to CRT;[6] however, the clinical implications of dyssynchrony after an acute myocardial infarction (AMI) have not been fully evaluated.

Left ventricular mechanical dyssynchrony can occur in patients with coronary artery disease who had no prior myocardial infarction and narrow QRS complexes.[7] Significant coronary artery stenotic lesions have been associated with left ventricular dyssynchrony.[8] Timely identification is more important for left main coronary artery stenosis (LMCAS) than for other coronary stenotic lesions in patients with non-ST-segment elevation myocardial infarction (NSTEMI). Unprotected left main coronary disease in acute coronary syndrome is associated with high mortality.[9] However, the accuracy of the diagnosis of LMCAS usually depends on

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invasive coronary angiography. Thus far, only a few studies focusing on noninvasive diagnostic techniques for LMCAS have been conducted. Lead aVR ST segment elevation in 12-lead electrocardiograms has been suggested to be a marker of left main coronary artery disease.[10] Hence, this study was done to evaluate left ventricular dyssynchrony after ST elevated MI.

MATERIALS & METHODS

Study Design

This is an observational cross-sectional study conducted in the Department of Cardiology, Government medical college, Kota. The study was conducted for 1 year. A total of 80 subjects were enrolled. 40 were with acute STEMI and 40 healthy controls. The complete history was taken, and 12-lead electrocardiography (ECG) and echocardiographic examination was done. Evaluation of patients with acute onset of chest pain should begin with an electrocardiogram (ECG) and troponin level. The American College of Cardiology, American Heart Association, European Society of Cardiology, and the World Heart Federation committee established the following ECG criteria for ST-elevation myocardial infarction (STEMI):[4]

- New ST-segment elevation at the J point in 2 contiguous leads with the cutoff point as greater than 0.1 mV in all leads other than V2 or V3
- In leads V2-V3 the cutoff point is greater than 0.2 mV in men older than 40 years old and greater than 0.25 in men younger than 40 years old, or greater than 0.15 mV in women

Measurement of LV end-diastolic diameter (LVEDD) and LV end-systolic diameter (LVESD) was done. Two-dimensional echocardiography was done. The results were analysed using SPSS software. Student's t-test was used to compare quantitative data between the STEMI and healthy control groups. $P < 0.05$ was considered statistically significant.

RESULTS

LVEDD and LVESD, were significantly higher in the STEMI group than in the control group (5.4cm vs. 4.6cm, P = 0.0007; and 3.5cm vs. 2.8cm, P = 0.001 respectively).

There was a highly significant correlation between LVESD and all segments SD (P = 0.0001). The correlation between Ejection fraction and all segment SD with p- value 0.001.

Table 1: Echocardiographic parameters in patients with anterior myocardial infarction (MI)

	STEMI group	Control group	P –value
LVESD (cm) mean	3.5	2.8	0.001 (Significant)
LVEDD (cm) mean	5.4	4.6	0.007 (Significant)
Ejection fraction mean	46.6	66.4	0.001 (Significant)

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

Table 2: Correlation between conventional echocardiographic parameters and tissue synchronization imaging parameters in ST elevation myocardial infarction patients

Correlation (Pearson's)	All segment SD (ms) (p-value)
LVEDD (cm)	0.08 (Non- Significant)
LVESD (cm)	0.001 (Significant)
Ejection fraction (%)	0.001 (Significant)

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.[11] 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.[12,13] In fact, LV

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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.[14] Hence, this study was done to evaluate left ventricular dyssynchrony after ST elevated MI.

In the present study, LVEDD and LVESD, were significantly higher in the STEMI group than in the control group (5.4cm vs. 4.6cm, P = 0.0007; and 3.5cm vs. 2.8cm, P = 0.001 respectively). In a study by Azazy AS et al[15], 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. −6.0ms, P = 0.036; and 42.7 vs. 23.94ms, 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. 95.45ms, P = 0.013; and 44.47 vs. 26.45ms, 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. 15.83ms, P = 0.038; 57.44 vs. 19.06ms, P = 0.040; 138.11 vs. 100.0ms, P = 0.035; and 45.44 vs. 32.50ms, 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.[15]

In the present study, there was a highly significant correlation between LVESD and all segments SD (P = 0.0001). The correlation between Ejection fraction and all segment SD with p-value of 0.001.

Another study by Nucifora G et al[16], showed that all patients underwent primary percutaneous coronary intervention. Real-time 3-dimensional echocardiography and myocardial contrast echocardiography were performed to assess LV function, LV dyssynchrony, and infarct size. LV dyssynchrony was defined as the SD of the time to reach the minimum systolic volume for 16 LV segments, expressed in percent cardiac cycle (systolic dyssynchrony index [SDI]). Myocardial perfusion at myocardial contrast echocardiography was scored (1 = normal/homogenous; 2 = decreased/patchy; 3 = minimal/absent) using a 16-segment model; a myocardial perfusion index, expressing infarct size, was derived by summing segmental contrast scores and dividing by the number of segments. SDI in patients with AMI was 5.24 + or - 2.23% compared to 2.02 + or - 0.70% of controls (p <0.001). Patients with AMI and LVEF < 45% had significantly higher SDI compared to patients with LVEF ≥ 45% (4.29 + or - 1.44 vs 6.95 + or - 2.40, p <0.001). At multivariate analysis, SDI was independently related to LVEF; in addition, the impact of SDI on LV systolic function was incremental to infarct size and anterior location of AMI (F change 16.9, p <0.001). In conclusion, LV synchronicity is significantly impaired soon after AMI. LV dyssynchrony is related to LVEF and has an additional detrimental effect on LV function, beyond infarct size and the anterior location of AMI.[16]

The various predictors for LVR after an AMI have been tested for validation.[17,18] The current established predictors are infarction size, involvement of the left anterior descending artery, and the presence of hypertension.[19,20] Among the known predictors for LVR, infarction size, which is estimated by the peak level of cardiac enzymes, was in agreement with the findings in the current study. Even though the incidence of LAD involvement and hypertension had a higher tendency in the LVR group than the group without LVR, there were no significant differences between the two groups. The lack of differences in the incidence of LAD involvement and hypertension may reflect the small population number; enrollment of a larger number of patients in a future study may clarify the discrepancy. The assessment of LV dyssynchrony has been performed using various echocardiographic techniques. Currently,

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tissue Doppler imaging is the major modality for estimation of myocardial contractile performance. The maximum difference in the peak systolic time, as measured by a color-coded tissue Doppler imaging technique, and the standard deviation of the time to peak radial strain in speckled tracking radial strain analysis are two mainstays for the assessment of dyssynchrony.[21]

CONCLUSION

Patients with acute STEMI showed significant LV dyssynchrony.

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ISSN: 0975-3583,0976-2833 VOL14, ISSUE 06, 2023

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