Original Article

Prospective Hospital-Based Evaluation of Left Ventricular Dyssynchrony Following ST-Elevation Myocardial Infarction at a Tertiary Care Centre

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Abstract

Background: Mechanical systolic LV dyssynchrony is associated with poor outcomes in patients with cardiac failure. However, its role in predicting prognosis after acute MI has been remotely explored. Therefore, this study was conducted to assess left ventricular dyssynchrony following ST elevated myocardial infarction. **Material and Methods:** 50 patients with acute STEMI who were selected from the cardiology department at SMS Medical College in Jaipur over the course of a year participated in this hospital-based observational study. Fifty healthy individuals of the same age and gender made up the control group. Echocardiography was performed on each patient, and their baseline data were recorded. **Results:** In the STEMI group, 56% of patients smoked, 42% had hypertension, 32% had diabetes, and 34% had dyslipidaemia. LVEDD, LVESD, A-wave velocity, and left atrial (LA) diameters were significantly greater in the STEMI group than in the control group (3.76 cm vs. 3.28 cm, P <0.001; 3.70 cm vs. 2.93 cm, P = 0.001; and 5.32 cm vs. 4.92 cm, P <0.001, respectively). In contrast, the STEMI group had significantly lower LVEF and E/A ratios (49.34% vs. 65.48%, P <0.001; 1.02 vs. 1.32, P <0.05). **Conclusion:** Left ventricular dyssynchrony may be used to identify patients at high risk for the development of LV remodeling after infarction which can help in providing early intervention and thereby decreasing the cardiac morbidity.

Keywords: STEMI, LVEDD, LVESD, LA, LV Dyssynchrony.

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Introduction

Acute myocardial infarction (AMI) is one of the primary causes of illness and death among older adults. It typically arises from a sudden decrease or total stoppage of blood flow to the coronary arteries due to the rupture of an atherosclerotic plaque, which is followed by the formation of a thrombus.[1] This sudden loss of blood supply results in ischemia and death of heart muscle tissue. The degree and location of the myocardial damage significantly affect the patient's outcome, including the risk of complications related to heart mechanics and electrical functioning. Changes in the electrocardiogram, such as elevation of the ST segment, new left bundle branch block, or abnormal Q waves, serve as crucial diagnostic indicators. Additionally, cardiac biomarkers, including troponins, verify the presence of myocardial damage.[2]

An increasing number of people are using the term "mechanical dyssynchrony" to describe the mechanical effects of asynchronous ventricular contraction and relaxation, which may or may not be related to electrical conduction delay.[3] A considerable proportion of patients with heart failure and intact left ventricular ejection fraction,[4] have left ventricular (LV) dyssynchrony as do 30–40% of those whose QRS length is normal,[1] Coronary artery disease is one of the most common causes of heart failure with preserved left ventricular ejection fraction.; yet, little is known about mechanical dyssynchrony in individuals with both conditions.[5] Certain parts of the heart muscle may contract and relax more slowly after an acute myocardial infarction, potentially causing left ventricular mechanical dyssynchrony and eventually resulting in clinical heart failure.[5] Methods such as tissue synchronisation imaging, strain rate imaging, or tissue Systolic function and local myocardial conduction can be evaluated using Doppler imaging (TDI).[6] When distinct portions of the left ventricle contract at different times, this is referred to as dyssynchrony in left ventricular (LV) contraction. Since the creation of cardiac resynchronisation therapy (CRT) for the treatment of severe heart failure, the concept of dyssynchrony has received more attention.[7,8] The impact of dyssynchrony after an acute myocardial infarction is still unknown, despite the fact that several dyssynchrony measurements have been investigated to see if they may predict how patients would react to CRT.

Patients with coronary artery disease may experience left ventricular mechanical dyssynchrony even in the absence of a previous myocardial infarction and when the narrow QRS

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complexes are narrow.[9] There has been a connection between left ventricular dyssynchrony and significant coronary artery stenotic lesions.[10] In individuals with non-ST-segment elevation myocardial infarction (NSTEMI), prompt detection of left main coronary artery stenosis (LMCAS) is more important than other coronary stenotic lesions. High mortality in acute coronary syndrome is associated with unprotected left main coronary disease.[11] However, invasive coronary angiography is typically required for the diagnosis of LMCAS to be accurate. Noninvasive diagnostic methods for LMCAS have been the subject of very few studies thus far. It has been proposed that lead aVR ST segment elevation in 12-lead ECGs is a sign of left main coronary artery disease.[12]

In order to evaluate left ventricular dyssynchrony after ST raised myocardial infarction, this study was carried out.

Materials and Methods

In this hospital-based observational study, 50 patients with acute STEMI were selected over the course of a year from the cardiology department at SMS Medical College in Jaipur. Fifty healthy, age-and sex-matched people made up the control group.

Patients having a large QRS complex (≥120 ms), paced rhythm, rheumatic heart disease with severe valve lesions, myocardial disorders (hypertrophic, restrictive, or dilated cardiomyopathy), prior open-heart surgery, and poor echocardiographic windows were not included in the study.

A 12-lead electrocardiogram (ECG) and an echocardiogram were performed in addition to taking a full medical history. An electrocardiogram (ECG) and troponin level should be the first tests performed on patients who have experienced sudden chest discomfort. The World Heart Federation committee, American College of Cardiology, American Heart Association, and European Society of Cardiology created the following ECG criteria for ST-elevation myocardial infarction (STEMI):[6]

- A new ST-segment elevation in two consecutive leads at the J point, with a cutoff point greater than 0.1 mV in all leads except V2 or V3
- The cutoff point for leads V2-V3 is greater than 0.15 mV for women, greater than 0.25 mV for males under 40, or higher than 0.2 mV for men over 40.

LV end-diastolic diameter (LVEDD) and LV end-systolic diameter (LVESD) measurements were made. An echocardiogram in two dimensions was performed. SPSS software was used to analyse the data. Quantitative data from the STEMI and healthy control groups were compared using Student's t-test. P less than 0.05 was regarded as statistically significant.

Results

The cohort included 50 patients with acute STEMI (mean age, 57.86±9.24 years) and 50 healthy volunteers of the same age and sex who served as the control group. Table 1 shows that 42% of the patients in the STEMI group had hypertension, 32% had diabetes, 34% had dyslipidaemia, and 56% smoked. Left atrial (LA) diameters, LVEDD, LVESD, and A-wave velocity were significantly greater in the STEMI group than in the control group (3.76 cm vs. 3.28 cm, P <0.001; 3.70 cm vs. 2.93 cm, P = 0.001; and 5.32 cm vs. 4.92 cm, P <0.001, respectively).

However, The STEMI group had significantly lower LVEF and E/A ratios (49.34% vs. 65.48%, P <0.001 and 1.02 vs. 1.32, P <0.05, respectively) [Table 2].

Table 1: Demographic data of studied group

Demographic variables	Number of patients (N=50)	Percentage
Age (Mean±SD) (yrs)	57.86±9.24	
Male	40	80%
Female	10	20%
Hypertension	21	42%
Diabetes mellitus	16	32%
Dyslipidemia	17	34%
Smoking	28	56%

Table 2: Echocardiographic parameter comparisons between the groups under study (A) and between patients with anterior and non-anterior myocardial infarction (MI) (B)

Variables	STEMI group (N=50)	Control group (N=50)	P – value	Anterior (N=35)	Non-anterior (N=15)	P-value
Aortic opening (cm), mean ± SD	2.63±0.19	2.55±0.16	>0.05	3.23±0.34	2.96±0.48	>0.05
Left atrium (cm), mean ± SD	3.76±0.52	3.28±0.15	<0.001	3.84±0.58	3.72±0.41	>0.05
LVESD (cm), mean ± SD	3.70±0.78	2.93±0.65	0.001	3.68±0.83	3.20±0.91	<0.05
LVEDD (cm), mean ± SD	5.32±0.42	4.92±0.33	<0.05	5.26±0.44	5.03±0.36	>0.05
Ejection fraction (%), mean ± SD	49.34±9.54	65.48±3.62	<0.001	46.42±8.21	54.63±11.66	>0.05
E m/s, mean ± SD	0.67±0.19	0.72±0.13	>0.05	0.68±0.22	0.71±0.19	>0.05
A m/s, mean ± SD	0.67±0.15	0.58±0.05	>0.05	0.75±0.16	0.60±0.15	>0.05
E/A, mean ± SD	1.02±0.33	1.32±0.26	<0.05	0.92±0.26	1.54±0.64	<0.01

Discussion

Both locally and regionally, myocardial ischaemia impacts LV systolic function. Focal hypokinesis, or reduced systolic thickening, happens seconds after myocardial ischaemia begins, prior to chest discomfort and ECG abnormalities. When compared to nearby perfused segments, this pathognomonic result will emerge as a hinge point in the area of the left and/or right ventricle supplied by the impaired artery (at least 70% stenosis). Another sign of ischaemia is a segment's delayed contractility.[13] The single metric with the highest prognostic and clinical value during and after MI is global LVEF,

which shows the total size and location of the infarct. In actuality, a serious consequence following MI is LV systolic function failure. Therefore, a precise and thorough evaluation of systolic dyssynchrony and LV remodeling has important consequences for clinical therapy and prognosis. Both mechanical and electrical dyssynchrony are included in LV dyssynchrony, and the former is widely recognised as a clear sign of LV systolic dyssynchrony.[14]

According to our research, the STEMI group had substantially greater left atrial (LA) diameters, LVEDD, LVESD, and A-wave velocity than the control group (3.76 cm vs. 3.28 cm, P <0.001; 3.70 cm vs. 2.93 cm, P = 0.001; and 5.32 cm vs. 4.92 cm, P <0.001, respectively). On the other hand, the STEMI group had significantly lower LVEF and E/A ratios (49.34% vs. 65.48%, P <0.001; 1.02 vs. 1.32, P <0.05). Ng et al,[15] reported similar results, where a considerable percentage of individuals had LV dyssynchrony in the early stages following acute MI without congestive heart failure. In the current study, there was no significant difference in LVEDD between patients with STEMI and controls, although LVESD was considerably higher. Similarly, Mollema et al,[16] found that while LVESV was considerably higher in individuals who died from a cardiac aetiology than in survivors, the prevalence of LV dilatation following acute MI was not noticeably elevated.

Because of increased LV filling pressure, mitral regurgitation, and/or post-MI diastolic dysfunction, in our study, the STEMI patients' LA diameter was significantly larger than that of the controls.[17] Our results are consistent with those of Meris et al,[18] who found that in patients with high-risk MI, early LA remodeling and size following MI are an independent predictor of death or hospitalisation for heart failure. Furthermore, they discovered that even in patients with a somewhat larger LA, the danger seemed to be ongoing.[18,19] Compared to patients with non-anterior STEMI, individuals with anterior STEMI in our study had significantly lower LVEF, larger LV volumes, and more frequent LV systolic dyssynchrony. Furthermore, patients with anterior STEMI had higher rates of in-hospital sequelae, including heart failure. This could be because anterior MIs had less LV systolic dysfunction because of the widespread myocardial necrosis and increased myocardial damage.[20]

The small population size may be the reason for the lack of variations in the incidence of hypertension and LAD involvement; enrolling more patients in a subsequent investigation may help to explain the disparity. Several echocardiographic methods have been used to evaluate LV dyssynchrony. The primary method for estimating cardiac contractile function at the moment is tissue Doppler imaging. The standard deviation of the time to peak radial strain in speckled tracking radial strain measurement and the biggest variation in the peak systolic time as assessed by a colour-coded tissue Doppler imaging method are two important indicators for assessing dyssynchrony.[21]

Conclusion

Significant LV dyssynchrony was seen in patients with acute STEMI. Patients who are at high risk of developing left ventricular remodelling following an infarction can be identified using left ventricular dyssynchrony, which can aid in early management and reduce cardiac morbidity.

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Conflicts of interest

There are no conflicts of interest.

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