Echocardiographic evaluation of the left ventricular function after acute myocardial infarction in female patients

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ABSTRACT

Background: The study was undertaken to evaluate LV functions (systolic and diastolic) and potential complications by M-mode echocardiography in patients of acute myocardial infarction (AMI). Aim and Objective: The aim of the study was to observe determinants of the left ventricular functions using echocardiography in female AMI cases. Materials and Methods: The present research was done after taking permission from Institutional Ethics Committee and consent was taken from all subjects. A case–control study was conducted with age, sex, and body mass index (BMI) matched 40 normal female healthy controls and 75 cases of AMI were subjected for general examination/history taking and demographic profile assessment. Echocardiographically detailed left ventricular functional indices were evaluated over mitral annulus. The data were analyzed using Microsoft Excel 2010. Results: The major sites of myocardial infarction (MI) were anteroseptal (54.6%), inferior wall (40%), and posterior/lateral wall (4%) in the cases. The baseline and functional parameters were compared and analyzed. Both the study groups were having BMI in overweight area. Hemodynamic parameters were showing statistically significant difference and the AMI cases having lower values. Hemodynamic parameters heart rates (P = 0.002) were significant, systolic blood pressure, diastolic blood pressure and mean arterial pressure were highly significant (P < 0.001). Further, (P < 0.001) statically significant difference was present in stroke volume, cardiac output, and CI with lower values in MI cases as compared to healthy subjects. The reduced ejection fraction and reduced FFS were observed in the cases which were statistically significant (P < 0.001). Further, there was elevation in ESS and EISS which was statistically significant (P < 0.001) in AMI cases. There was decrease in E (P < 0.001), elevation in A (P < 0.001) and reduction in E/A ratio (P < 0.001) which was statistically significant, in AMI patients as compared to healthy subjects in this study. Conclusion: In AMI patients, the LV function is affected and was reduced. Echocardiographic assessment of patients of AMI gives us prognosis of cases which allows us to reduce risks and start treatment to decrease morbidity and mortality. Although researches are to be done to observe the nature of LV, it is treated with stress in physiology and pharmacology.

KEY WORDS: Myocardial Infarction; Echocardiography; Left Ventricular Function

INTRODUCTION

Acute myocardial infarction (AMI) is a major disease globally.[1] An alarming increase of ischemic heart diseases (IHD) and associated mortality has been noted in South Asian sub-continent. By 2020, it is estimated that IHD will be the major cause of death in India.[2,3] Coronary artery disease
is common in South Asian population due to increased prevalence of hypercholesterolemia, hypertriglyceridemia, diabetes mellitus (DM), and obesity.[4]

Acute complications of myocardial infarction (MI) include ischemic, mechanical, arrhythmic, inflammatory, and embolic.[5] It also leads to molecular, cellular, or interstitial alterations which may cause changes in geometry, function, and size of heart, known as cardiac remodeling[6] which can be early phase, that is, before 72 h or lately after 72 h.[7] The remodeling before 72 h helps the heart to retain its function as there is impaired contractility whereas the late phase includes myocyte hypertrophy, cellular elongation, and proliferation continuing for years.[8]

Ejection fraction (EF) is an prominent factor which helps to stratify risk after MI as AMI leads to generalized hypokinesia.[9] MI patients having EF <40% have 5-year survival rate of 65% and end-systolic volume >130 cm³ have 5-year survival rate 52%.[10] Furthermore, LV diastolic dysfunction causes LV dilatation, cardiac failure, and death.[11] Research has proved that early diastolic filling pattern (E/A ratio >1) post-MI in patients after 3–12 weeks and associated with an EF<40% causes serious outcome.[12]

Over the years, echocardiography is popular non-invasive diagnostic procedure. It helps in diagnosing mechanical complications, evaluating hemodynamic parameters, risk stratification, and outcome in AMI patients.[13] This further helps in formulating rationale therapeutic decisions after MI. Considering the lack of available information in this direction, the research was undertaken to evaluate LV systolic and diastolic functions with potential complications by echocardiography in patients of AMI.

MATERIALS AND METHODS

The present research was done after taking permission from Institutional Ethics Committee and consent was taken from all subjects.

Seventy-five female MI patients and 40 controls were selected as subjects after fulfilling the inclusion criteria. A detailed medical history of smoking and alcohol consumption hypertension (HT), DM was taken within 24 h of admission. Clinical examination was done for their anthropometric measurements, blood pressure (BP), and resting heart rates (HR).

It was a case–control study with age, sex, and body mass index (BMI) matched normal healthy controls and confirmed cases of AMI.

Patients with age ranging 30–60 years, having DM, HTN (without LVH), and obesity were included in the study. Conditions that could alter results such as HT (with LVH), old MI, cardiac ailments, CCF, CABG, kidney and respiratory disease, thyroid diseases and athletes were excluded from the study.

Case was defined on the basis of electrocardiographic evidence of MI and estimation of Troponin T.

M-mode echocardiograms were obtained by a trained cardiophysiologist. Maximum velocity of active mitral filling (A), maximum velocity of passive mitral filling (E), ratio (E/A) of passive to active were calculated, Stroke volume (SV) and EF were measured.[14]

Calculations:

\[
\text{BMI} = \frac{\text{Weight}}{\text{Ht}^2}
\]

\[
\text{SV} = \text{LVDd}^3 - \text{LVDs}^3
\]

\[
\text{CO} = \text{HR} \times \text{SV}
\]

\[
\text{Cardiac output (CO) (CI)} = \text{CO} \times \text{BSA}
\]

\[
\text{EF (EF \%) = } \frac{\text{LVDd}^3 - \text{LVDs}^3}{100/\text{LVDd}^3}
\]

Fractional fibre shortening (FS%) = LVDd-LVDs × 100/ LVDd

\[
\text{ESS} = \text{Systolic blood pressure (SBP)} \times \text{LVDs/PWTs} \times (1+\text{PWTs/LVDs}) \times 0.334
\]

\[
\text{EISS} = \text{Diastolic blood pressure (DBP)} \times \text{LVDs/PWTs} \times (1+\text{PWTs/LVDs}) \times 0.334
\]

The data were analyzed in controls and AMI cases using Microsoft Excel 2010 software. Mean ± Standard deviation was calculated. Comparison of all variables in both groups was done using t-test (unpaired). Statistically significant value (\(P \leq 0.05\)) was taken.

RESULTS

BMI was observed to be in overweight category in both the study groups though the study population was age and BMI matched. Hemodynamic parameters were showing statistically significant difference and the AMI cases having lower values as depicted in Table 1. Table 2 shows all the parameters in both study groups having statistically significant difference. The site of MI in patients has been depicted in Figure 1 with 54.6% cases presenting with anteroseptal MI, followed by 40% with inferior wall MI.

DISCUSSION

MI is a prominent issue and causes mortality and morbidity globally. Garg et al. reported that changes in lifestyle caused
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Table 1: Study group’s base parameters

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Controls (n 40) (Mean±SD)</th>
<th>Cases (n 75) (Mean±SD)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>53.33±6.00</td>
<td>54.56±5.53</td>
<td>0.179</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>55.97±11.8</td>
<td>59.21±10.3</td>
<td>0.065</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>155.29±7.9</td>
<td>157.64±8.3</td>
<td>0.0663</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>23.20±2.12</td>
<td>23.20±2.12</td>
<td>0.0618</td>
</tr>
<tr>
<td>BSA (kg/m²)</td>
<td>1.55±0.65</td>
<td>1.60±0.72</td>
<td>0.642</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>74.48±4.48</td>
<td>69.88±12.96</td>
<td>0.002</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>126.20±2.46</td>
<td>116.83±12.97</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>77.45±1.88</td>
<td>69.31±9.16</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>PP (mmHg)</td>
<td>48.75±3.22</td>
<td>47.52±11.32</td>
<td>0.331</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>93.72±1.56</td>
<td>85.16±9.15</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

SD: Standard deviation, BMI: Body mass index, HR: Heart rates, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, MAP: Mean arterial pressure

Table 2: Systolic and diastolic function indices of the left ventricle (Mean±SD) of both study groups

<table>
<thead>
<tr>
<th>Indices</th>
<th>Control Female (n 40) (Mean±SD)</th>
<th>Case Female (n 75) (Mean±SD)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Output assessment parameters</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SV (ml)</td>
<td>82.54±9.32</td>
<td>64.45±24.17</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>CO (l/min)</td>
<td>6.19±0.81</td>
<td>4.53±2.00</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>CI (l/m²)</td>
<td>3.93±0.53</td>
<td>2.88±1.29</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Contractile assessment parameters</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EF (%)</td>
<td>61.81±2.41</td>
<td>32.93±5.64</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>FS (%)</td>
<td>44.55±2.36</td>
<td>24.04±6.72</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>After load assessment parameters</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ESS (dynes/cm²)</td>
<td>122.94±9.74</td>
<td>143.06±19.55</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>EISS (dynes/cm²)</td>
<td>75.74±6.33</td>
<td>84.83±13.16</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Diastolic function assessment of the left ventricle</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>E (cm/sec)</td>
<td>0.66±0.09</td>
<td>0.59±0.11</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>A (cm/sec)</td>
<td>0.57±0.08</td>
<td>0.63±0.06</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>E/A ratio</td>
<td>1.16±0.048</td>
<td>0.93±0.17</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

SD: Standard deviation, SV: Stroke volume, CO: Cardiac output

Obesity which leads to HT and cardiac diseases. As per the Western Pacific Classification of BMI for Asian population given by the WHO in 2000, all the study subjects were falling in the overweight category. In the present study, predominant sites of MI were anteroseptal (54.6%), inferior wall (40%), and posterior/lateral wall (4%). Bhawardj R in their study on 124 patients reported anterior wall MI in 88 patients (70.97%), inferior wall MI in 31 patients (25%), and lateral wall MI in 5 patients (4.03%). Srivastava et al. also noted anterior wall MI in 47.44%, lateral wall MI in 2.18%, and inferior and right ventricular MI in 10% of their study population. Pfeffer and Braunwald have reported that inferior and anterior infarcts are more likely to produce ventricular enlargement and anterior wall infarctions are associated with greater ventricular enlargement and a higher late mortality.

Studies have reported that several hemodynamic alterations are noted after MI which are influenced by the size of the infarcted area. A statistically significant difference (P < 0.001) was noted in SBP, DBP, and mean arterial pressure in the study group with lower values in AMI cases. Similar observations have been made by Master et al. by studying on 538 cases with initial and recurrent MI who reported decrease in the BP during the phase of hospitalization. The most plausible explanation for fall in the SBP is depression of SV due to ineffective contraction of infarcted myocardium. Fries et al. stated in their study that heart rate increased in patients of MI with increasing severity of infarction. On the contrary, we noted a decreased HR as AMI cases were administered β-blockers during hospitalization which are known to cause decrease in heart rate. Astrom et al. and colleagues in their study on AMI cases used sotalol infusion and noted a marked reduction of SBP, heart rate, SV, and CO but not in the group given saline infusion. Further, (P < 0.001) statically significant difference was present in SV, CO, and CI with lower values in MI cases when compared with healthy subjects. Similar observations have been reported by Fries ED and colleagues in their study on hemodynamic alterations in AMI. LV EF is one of the important parameters to evaluate the systolic function of the left ventricle. The reduced EF was observed in the cases which was statistically significant (P < 0.001) when comparison was done with healthy subjects. Out of 75 female AMI cases, six were noted to have mildly depressed (LVEF% 45–54), 59 having moderately depressed (LVEF% 30–44), and 10 had severely depressed EF (LVEF% <30). Alam et al. reported that patients after AMI showed abnormal wall motion and reduced mitral annular motion and reduced EF as against healthy subjects. Moller et al. showed that strong predictor of mortality was
LVEF and wall motion score index measured within 24 h of AMI and then during follow-up of 19 months.[26] Fractional fiber shortening is an index to measure contractile state of myocardium and FFS≤25% is an indicator of systolic dysfunction.[27] FFS was reduced after MI and statistically significant (P < 0.001) difference was there. FFS is an index to measure contractile state of myocardium. Value of FFS≤25% is an indicator of systolic dysfunction. The reason for reduced contractility was damaged myocardium which was due to infarct, destruction of myocytes, and changes due to inflammation resulting in the functional impairment.[28] Further, we noted a statistically significant (P < 0.001) increase in ESS and EISS in AMI cases. Hirose et al. and colleagues in their research on 40 MI cases reported that after the 1 year time of hospital discharge when the baseline was recorded, circumferential wall stresses and the mid-left ventricular end-systolic meridional stresses in patients with an anterior wall infarction were increasing significantly and were progressive. These increasing wall stresses can lead to further changes in the left ventricular function which is representative of alteration in intrinsic contractility if they continue to increase years from the date of infarction.[29] Clerfond et al. after studying on 169 cases concluded that ESS (end systolic wall stress) is an independent predictor of heart failure after ST-segment elevation MI post discharge from the hospital. They also suggested further research in this direction to determine whether systolic wall stress should be target of post-MI therapeutic interventions.[30] Wolk has also stated that ventricular arrhythmias can occur due to myocardial ischemia and a rise in ventricular wall stress coexisting together.[31] Moller et al. and colleagues mentioned in their study that post-AMI, the rate of active relaxation is influenced by microvascular dysfunction, myocardial ischemia causing cell necrosis, and wall motion abnormality. Furthermore, LV chamber stiffness is affected directly by fibrocellular infiltration, interstitial edema, and scar formation.[32] Hence, parameters: Peak velocity of late diastolic filling (A), peak velocity of early diastolic filling (E), and ratio of E/A are important predictors of cardiac mortality and were evaluated in the present study. There was decrease in E (P < 0.001), elevation in A (P < 0.001), and reduction in E/A ratio (P < 0.001) which was statistically significant, occurred in AMI patients as compared to healthy subjects in our study. About 58.6% of AMI cases had diastolic dysfunction in our study. Hadi et al. in their study made similar observations and they reported diastolic dysfunction post-MI in 58% of their cases.[33]

**Strength and Limitations**

Further, to calculate the filling patterns, mitral inflow, deceleration time (DT), and E/A ratio are used: Filling patterns are normal, pseudonormal impaired relaxation, and restrictive filling.[34] As DT was not calculated in our study, so we were unable to classify the study subjects into these filling patterns.

**CONCLUSION**

Echocardiographic assessment of patients of AMI gives us prognosis of cases which allows us to reduce risks and start treatment to decrease morbidity and mortality. Although researches are to be done to observe the nature of LV, it is treated with stress in physiology and pharmacology.

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