ORIGINAL ARTICLE

ECHOCARDIOGRAPHIC EVALUATION OF LEFT VENTRICULAR FUNCTION AFTER ACUTE MYOCARDIAL INFARCTION

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Background: The study was taken up to evaluate the left ventricular systolic and diastolic function after acute myocardial infarction (AMI) in male patients. **Method:** A hospital based study was conducted at tertiary care centre in UP, India with 60 male healthy controls and 125 confirmed cases of AMI admitted to the coronary care unit who fulfilled the inclusion criteria. After a clinical examination/history taking, left ventricular (LV) functional parameters were assessed by echocardiography in M-mode. The data obtained was analyzed using Microsoft Excel2010 software. **Results:** The predominant sites of myocardial infarction (MI) were antero-septal MI (57.6%), with inferior wall MI (28%), and lateral wall MI (4.8%). Body mass index (BMI) was in overweight category in both study groups. A statistically significant difference was noted in haemodynamic parameters with lower values noted in AMI cases. Statistically significant differences with lower values in AMI cases were noted in Stroke volume ($p<0.001$), cardiac output ($p<0.001$), Ejection fraction ($p<0.001$) and Fractional fibre shortening ($p<0.001$) when compared with healthy subjects. A statistically significant decrease in E ($p=0.015$), increase in A ($p<0.001$) and decrease in E/A ratio ($p=0.015$) was noted in AMI cases when compared with healthy controls. **Conclusion:** The overall LV function was decreased among acute myocardial infarction patients. Echocardiographic assessment provides prognostic information which helps to stratify risks and initiate rationale therapeutic measures to reduce morbidity and mortality associated with it.

Keywords: Acute Myocardial Infarction, Echocardiography, Left Ventricular Function, E/A ratio, Fractional fibre shortening

INTRODUCTION

Acute Myocardial Infarction (AMI) is a major health problem globally.[1] Over the past two decades an alarming increase in the prevalence of ischemic heart diseases (IHD) and associated mortality has been noted in South Asian sub-continent. It has been estimated that by 2020, IHD will be the largest cause of disability and death in India, with 2.6 million Indians predicted to die due to IHD.[2,3] Prevalence of coronary artery disease in this region has been reported to be high due to increased prevalence of hypercholesterolemia, hyper-triglyceridemia, diabetes mellitus (DM) and obesity.[4]

Acute complications of MI include ischemic, mechanical, arrhythmic, inflammatory and embolic.[5] MI also leads to molecular, cellular or interstitial alterations which may manifest clinically as alterations in heart architecture, mass, geometric pattern, function and size. This phenomenon is known as cardiac remodeling.[6] Cardiac remodelling is a progressive change starting within few hours after MI and continuing for years. The early phase remodelling 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.[7]

Left ventricular failure is a well-recognised mechanical complication of AMI. The degree of dysfunction depends on site and extent of MI. Left ventricular ejection fraction (LVEF) is an important factor which helps to stratify risk after MI as AMI leads to generalised hypokinesia.[8] It has been reported that post-MI patients with an Ejection fraction (EF) of less than 40% and End-systolic volume (ESV) of more than 130 cm^3 had a 5-year survival rate of 65% and 52%, respectively.[9] The presence of LV diastolic dysfunction is an important predictor of outcome following AMI as it is related to progressive LV dilatation, heart failure and cardiac death.[10] A predominantly early diastolic filling pattern (E/A ratio>1) 3 to 12 weeks after MI in patients with an EF<40% is associated with adverse outcome.[11]

Over the years, echocardiography has made major advancements in the field of non-invasive diagnostic cardiology. It helps in haemodynamic assessment, detection of mechanical complications, risk stratification and long term prognosis in AMI patients.[12] This further helps in formulating rationale therapeutic decisions after MI. Considering the lack of available information in this direction, the present study was undertaken to assess LV systolic and diastolic function and potential complications by M-mode echo-cardiography in patients of AMI.

MATERIAL AND METHODS

The present study was conducted at tertiary care centre in Uttar Pradesh, India. The study was approved by Institutional Ethics Committee and informed written consent was obtained from each subject. It was a case-

control study with age, sex and BMI matched normal healthy controls and cases of AMI. One hundred and twenty-five male confirmed cases of AMI (based on electrocardiographic evidence and estimation of Troponin T) admitted to the coronary care unit who fulfilled the inclusion criteria, and 60 controls were selected as study subjects. Within 24 hours of admission, a detailed medical history including history of hypertension (HTN), DM, smoking and alcohol consumption was taken. Clinical examination was carried out to record their anthropometric measurements, blood pressure (BP) measurements and resting heart rates (HR).

Patients with age ranging 30–60 years, having DM, HTN (without Left Ventricular Hypertrophy, LVH) and obesity were included. Conditions that could alter results like old MI, hypertension (with LVH), CCF, valvular lesions, arrhythmias, cardiomyopathy, left bundle-branch block, coronary artery bypass grafting, respiratory disease, kidney disease, thyroid disorder and athletes were excluded.

Two-dimensional M-mode echocardiograms (Siemens Acuson P300) of all participants were obtained by a trained cardiologist. Stroke volume (SV) and ejection fraction (EF) were measured from apical four chamber view using the monoplane area-length method. Pulsed Doppler measurements were obtained in the apical four chamber view, with beam aligned as perpendicularly as possible to the plane of the mitral annulus. Sample volume was placed between the tips of the mitral leaflets during diastole and maximum velocity of passive mitral filling (E), maximum velocity of active mitral filling (A), ratio of passive to active velocity (E/A) were calculated.[13]

Calculations:

Body surface area[14] (BSA)= 0.6×height (m)+0.0128×weight (Kg)-0.1529
Stroke volume[15] (SV)= (LVDd)³-(LVDs)³
Cardiac output[15] (CO)= SV×HR
Cardiac output[15] (CI)= CO×BSA
Ejection fraction[15] (EF%)= LVDd³-LVDs³×100/LVDd³
Fractional fibre shortening[15] (FS%)= LVDd-LVDs×100/LVDd
End systolic Stress[15] (ESS)= 0.334×SBP×LVDs/PWTs×(1+PWTs/LVDs)
End Isovolumetric-Systolic stress[15] (EISS)= 0.334×DBP×LVDs/PWTs× (1+PWTs/LVDs)

The data was analysed using Microsoft Excel 2010. Mean±SD were calculated. Comparison of all variables in both groups was done by using unpaired Student's *t*-test and *p*≤0.05 was considered as statistically significant.

RESULTS

BMI was in overweight category in both the study groups. In AMI group, 6 were noted to be hypertensive and 3 were diabetic. A statistically significant difference

was noted in hemodynamic parameters with lower values noted in AMI cases (Table-1).

The site of MI in patients has been depicted in Table-2 with 57.6% cases presenting with antero-septal MI, followed by 28% with inferior wall MI.

Figure-1 shows the echocardiographic parameters in both study groups. A statistically significant difference was noted in all the parameters in both study groups.

Table-1: Baseline parameters of study groups (Mean±SD)

	Controls (n=60)	Cases (n=125)	*p*
Age (years)	51.50±6.53	52.16±7.91	0.488
Weight (Kg)	65.31±22.7	59.77±21.3	0.070
Height (Cm)	167.56±31.5	159.96±29.8	0.062
BMI (Kg/m²)	23.26±1.84	23.45±2.77	0.543
BSA (Kg/m²)	1.74±1.53	1.62±1.42	0.061
HR (bpm)	80.43±2.24	70.67±11.60	<0.001*
SBP (mmHg)	126.43±1.99	117.07±10.73	<0.001*
DBP (mmHg)	77.96±2.13	73.04±8.45	<0.001*
PP (mmHg)	48.46±3.11	44.03±9.09	<0.001*
MAP (mmHg)	94.11±1.53	87.74±8.21	<0.001*

*Significant

Table-2: Left ventricular systolic and diastolic function indices based on echocardiography in study groups (Mean±SD)

	Control Male (n=60)	Case Male (n=125)	*p*
Parameters for output assessment			
SV (ml)	82.09±11.21	71.27±24.83	<0.001*
COP (l/min)	6.66±1.03	5.09±2.12	<0.001*
CI (l/m²)	11.21±1.57	3.21±1.36	<0.001*
Parameters for contractile assessment			
EF (%)	61.02±2.02	31.96±6.05	<0.001*
FS (%)	45.32±2.76	27.76±8.60	<0.001*
Parameter for after load assessment			
ESS (dynes/cm²)	124.22±11.69	137.01±19.36	<0.001*
EISS (dynes/cm²)	76.63±7.74	85.35±12.62	<0.001*
For assessing the diastolic functions of the left ventricle			
E (cm/sec)	0.65±0.09	0.61±0.14	0.015*
A (cm/sec)	0.56±0.08	0.64±0.12	<0.001*
E/A ratio	1.17±0.06	0.98±0.36	<0.001*

SV=stroke volume, COP=cardiac output, CI=cardiac index, EF=ejection fraction, FS=fractional shortening, ESS=End systolic stress, EISS=End Isovolumetric Systolic Stress, E=peak velocity of early diastolic filling, A=peak velocity of late diastolic filling, *Significant
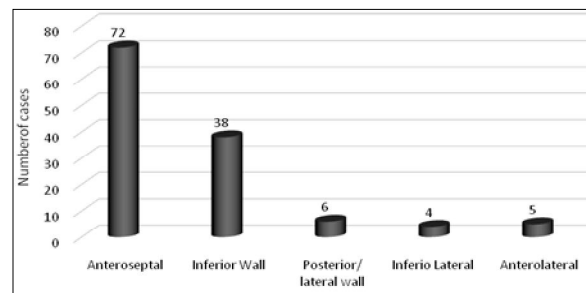

Figure-1: Case profile with different sites of MI

DISCUSSION

Myocardial infarction is a major public health issue and a leading cause of mortality and morbidity globally. Garg *et al* reported that marked shift in lifestyle has resulted in an increasing trend of obesity which

predisposes to ailments like hypertension and cardiovascular diseases.[16] As per the Western Pacific Classification of BMI for Asian population given by WHO in 2000, all the study subjects were falling in the overweight category.

In the present study predominant sites of MI were antero-septal (57.6%), inferior wall (30.4%) and lateral wall (4.8%). Bhardwaj 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%).[17] 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.[18]

Studies have reported that several haemodynamic alterations are noted after MI which are influenced by the size of the infarcted area.[19] A statistically significant difference was noted in SBP ($p<0.001$), DBP ($p<0.001$), PP ($p<0.001$) and MAP ($p<0.001$) in the study group with lower values in AMI cases. Similar observations have been made by Master *et al* in their study on 538 patients with initial and recurrent MI who reported decrease in the blood pressure during the phase of hospitalization.[20] The most plausible explanation for fall in the SBP is depression of stroke volume due to ineffective contraction of infarcted myocardium.[21] Fries ED *et al* in their study reported that heart rate increased in patients of MI with increasing severity of infarction.[19] 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 M and colleagues in their study on AMI cases used Sotalol infusion and noted a significant reduction in heart rate, systolic blood pressure, cardiac output and stroke volume compared to placebo group given saline infusion.[22] Further, a statistically significant difference with lower values in MI cases were noted in SV ($p<0.001$), CO ($p<0.001$) and CI ($p<0.001$) when compared with healthy subjects. Similar observations have been reported by Fries ED and colleagues in their study on haemodynamic alterations in AMI.[19]

LVEF is one of the important parameters to assess the left ventricular systolic function. A statistically significant reduction in ejection fraction was noted in the AMI cases ($p<0.001$) when compared with healthy controls. Out of 125 male AMI cases, 7 were noted to have mildly depressed (LVEF% 45–54), 88 had moderately depressed (LVEF% 30–44) and 30 had severely depressed ejection fraction (LVEF% <30). Alam M, *et al*, in their study on 202 AMI patients reported that following AMI patients showed reduced ejection fraction, abnormal wall motion and decreased mitral annular motion as compared to healthy subjects.[23]

Moller, *et al*, in their study on 767 patients with acute MI demonstrated that echocardiographically determined LVEF and wall motion score index at 1 day after admission for acute MI were powerful predictor of all-cause mortality during a median follow-up of 19 months.[24] Fractional fibre shortening is an index to measure contractile state of myocardium and FFS ≤25% is an indicator of systolic dysfunction.[25] In the present study we found that the FFS was significantly reduced after MI and the difference was statistically significant ($p<0.001$). In 8 (6.4%) cases FFS was ≤14 % indicating severely abnormal, 15 (12%) cases FFS was between 15–19% indicating moderately abnormal, 13 (10.4%) cases FFS was between 20–24% indicating mildly abnormal systolic function. Normal systolic function as indicated by FFS between 25–43% was noted in 89 (71.2%) AMI cases. The reason for reduced contractility was infarct-related myocardium, loss of myocytes and inflammatory changes resulting in the functional impairment.[26] Further, we noted a statistically significant ($p<0.001$) increase in ESS and EISS in AMI cases. Hirose K, and colleagues in their study on 40 MI cases reported that mid-left ventricular end-systolic meridional and circumferential wall stresses in patients with an anterior wall infarction increased progressively and significantly during the first year from the baseline recorded at the time of hospital discharge. These wall stresses if continue to increase remote from the date of infarction, then may lead to further alterations in left ventricular performance representative of changes in intrinsic contractility.[27] Clerfond G, *et al*, in their study on 169 patients concluded that end systolic wall stress is an independent predictor of post discharge heart failure after ST-segment elevation MI. They also suggested further research in this direction to determine whether systolic wall stress should be target of post MI therapeutic interventions.[28]

Moller JE, and colleagues in their study mentioned that after AMI, myocardial ischemia, cell necrosis, microvascular dysfunction, and regional wall motion abnormalities influence the rate of active relaxation. Also, interstitial oedema, fibro-cellular infiltration, and scar formation directly affect LV chamber stiffness.[29] Hence, pulsed-wave Doppler imaging parameters: peak velocity of early diastolic filling (E), peak velocity of late diastolic filling (A), E/A ratio become important predictors of cardiac mortality and these were assessed in the present study. A statistically significant decrease in E ($p=0.015$), increase in A ($p<0.001$) and decrease in E/A ratio ($p=0.015$) was noted in our study in AMI cases when compared with healthy controls. 58.4% of AMI cases had diastolic dysfunction in our study. Similar observations have been made by Hadi A, *et al*, in their study who reported post MI diastolic dysfunction in 58% of their cases.[30] Mitral inflow E/A ratio and deceleration time are used

to identify the filling patterns: normal, impaired relaxation, pseudo-normal and restrictive filling. As deceleration time was not noted in our study we could not classify the study subjects into these filling patterns.

CONCLUSION

Echocardiographic assessment of patients of AMI provides prognostic information which leads us to stratify risks and initiate rationale therapeutic measures to reduce morbidity and mortality associated with it. Further studies are required to document the behaviour of LV under pharmacological and physiological stresses.

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