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Original Research Article

GEOMETRICAL ASSESSMENT OF LEFT VENTRICLE IN FEMALE POST MYOCARDIAL INFARCTION PATIENTS BY ECHOCARDIOGRAPHY

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ABSTRACT

Background: Left ventricular dilatation leads to ventricular dysfunction and congestive heart failure after myocardial infarction. Present study is to evaluate the Left ventricular geometry and the determinants of L.V. geometry by M-mode Echocardiography in female subjects after acute MI. **Material and Methods:** A hospital based prospective study was conducted at tertiary care center in Uttar Pradesh, India with a total of 75 female cases of Myocardial Infarction who fulfilled the inclusion criteria and 40 age and BMI matched controls with thorough General Examination / history taking and Demographic profile assessment. Detailed L.V. geometrical parameters were assessed by Echocardiography at lateral and septal sides of L.V. at the level of mitral annulus on 1st day of admission. Data was analyzed and calculations were done by using Microsoft Excel 2010 software. **Result:** The geometrical parameters were compared and analysed. P value<0.001 gave highly significant results and <0.05 gave significant results. LVIDd (<0.001), LVIDs (<0.001), LVPWTd (0.041), VM (<0.05), LVMI (BSA) (0.191), LVMI ($Ht^{2.7}$) (<0.001), LVMI (g/m) (<0.05) were significant respectively. **Conclusion:** From the study, it can be concluded that the overall L.V. geometry was affected among Acute Myocardial Infarction patients. An increase in left ventricular internal dimension is a risk for arrhythmias and congestive heart failure. Knowledge of the left ventricular dimension improves predictions of the risk of congestive heart failure made on the basis of traditional risk factors. Though further studies are required to document the behavior of L.V. under pharmacological and physiological stresses.

INTRODUCTION

Acute myocardial infarction (MI) is the frequent coronary artery disease which is the leading cause of mortality and morbidity.[1] Acute myocardial infarction (AMI) is characterized by regional myocardial damage causing left ventricular (LV) remodeling that may lead to systolic and diastolic dysfunction. The pathophysiology and consequences of LV geometrical changes post AMI have been the centre of research for some decades.[2]

There are structural, geometric, biochemical and functional changes in both the infarcted and non-infarcted areas of the left ventricular (LV) myocardium post AMI.

Echocardiographically LV structural and functional changes can be recorded after Acute myocardial infarction. These changes are responsible for the early and late complications such as Heart failure, severe arrhythmias, mitral regurgitation and sudden death after acute myocardial infarction.[3]

Ventricular remodeling, occurs after massive myocardial infarction. The term ventricular remodeling involves alteration in ventricular architecture, along with increased volume of chamber with altered configuration.[4] The heart changes its size, geometry and function in Left ventricular remodeling. Quantitative M-mode transthoracic echocardiography helps in demonstration of LV remodeling due to damage of contracting myocytes. Approximately one-half to one-third patients experience distorted ventricular geometry, ventricular dilation and mitral regurgitation.[5]

Quantification of cardiac chamber size, ventricular mass and function are the most clinically important and frequently urged task of echocardiography.[5] Based upon the LVMI and RWT left ventricular geometrical pattern can be defined to stratify risk of morbidity and mortality.

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MATERIALS AND METHODS

The present study was conducted at tertiary care center in Uttar Pradesh, India. The study was approved by institutional ethics committee and informed written consents were obtained from all study subjects.

The study population consisted of 75 female cases of Myocardial Infarction who fulfilled the inclusion criteria and 40 age and BMI matched controls.

Patients with age ranging 30-60 years, having DM, HTN (without LVH),and obesity were included in the study.

A detailed medical record including history of hypertension with or without medications, diabetes mellitus, non-essential habits like smoking, alcohol consumption, chewing tobacco, physical activity including past and family history were noted. Clinical examination included the record of their height, weight, blood pressure (SBP, DBP), and resting heart rate (HR).

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

Conditions that could alter results like old MI, CCF, HTN (with LVH) valvular lesions, arrhythmias, cardiomyopathy, left bundle branch block, age >60 years &<30 years, coronary artery bypass grafting, respiratory disease, kidney disease, thyroid disorder and athletes were excluded.

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

Two dimensional M-mode echocardiograms (Siemens Acuson P 300, Germany) of all participants were obtained, assisted by technician. Left ventricular dimensions were obtained at lateral and septal sides of L.V. at the level of mitral annulus on 1st day of admission, with measurement of interventricular septal thickness (IVST), LV internal dimension in diastole (LVIDd), LV internal dimension in systole (LVIDs) and LV posterior wall thickness (PWT) according to guidelines of American Society of Echocardiography. Devereux formula was used to calculate LVM and then LVMI (BSA), LVMI(Ht2.7), LVMI(g/m), RWT was calculated.

Calculations

Body surface area (BSA),[16] BSA = 0.6 × height (m) + 0.0128 × weight (kg) –0.1529

Body mass index (BMI) = Weight /Ht2

Relative wall thickness (RWT) = 2 × PWd /LVIDd.

Left ventricular mass (LVM) = 0.8 [1.04 (IVS +LVIDd +PWT)3- (LVIDd)3+0.6 (Devereux formula)6

LVMI (indexed to BSA) = LVM/ BSA.

LVMI (indexed to Height2.7) = LVM/Ht (m) 2.7

LVMI (indexed to Height) = LVM/Ht (m)

Identification of LV geometric pattern based on parameters for structural changes

1. Normal geometry-normal RWT and normal LVMI
2. Concentric remodeling- increased RWT and normal LVMI
3. Eccentric hypertrophy- normal RWT and increased LVMI
4. Concentric hypertrophy- increased RWT and increased LVMI.

The pattern of LV remodeling will be determined using LVMI [LVM indexed to height (g/m)] and Relative wall thickness [RWT (mm)].

Indian Asian males- 118/0.50 and Indian Asian females-107/0.47.[7]

The data were analyzed in controls and cases by using Microsoft Excel 2010 software. Mean ± SD was calculated and unpaired student's t-test was applied. P-value of ≤0.05 was considered as statistically significant.

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, except PP and the AMI cases having lower values as depicted in Table 1.

Table 1: Both study group's base parameters

	Controls (n 40) (Mean ± SD)	Cases (n 75) (Mean ± SD)	P value
Age (yrs)	53.33±6.00	54.56±5.53	0.179
Weight (kg)	55.97±11.8	59.21±10.3	0.065
Height (cm)	155.29±7.9	157.64±8.3	0.0663
BMI (kg/m^2)	23.20±2.12	23.20±2.12	0.0618
BSA (kg/m^2)	1.55±0.65	1.60±0.72	0.642
HR (bpm)	74.48±4.48	69.88±12.96	0.002
SBP (mmHg)	126.20±2.46	116.83±12.97	<0.001
DBP (mmHg)	77.45±1.88	69.31±9.16	<0.001
PP (mmHg)	48.75±3.22	47.52±11.32	0.331
MAP (mmHg)	93.72±1.56	85.16±9.15	<0.001

In Table 2. Except for the parameters like, PWT (diastolic), IVST (diastolic), LVMI (Index to height), and LVMI (Index to BSA), statistically highly significant difference (P<0.001) was observed between the two groups. Differences in PWT (diastolic), LVMI (Index to height) was statistically significant P=0.041 and 0.029 respectively.

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Table 2: This shows the echocardiographic parameters of females in both study groups

Variable	Control Female (n=40) (Mean ± SD)	Case Female (n=75) (Mean ± SD)	P value
For assessing the structural changes in heart			
LVM (gm)	165.85±21.02	176.97±28.18	0.005
LVM/BSA (gm/m^2)	108.94±14.83	112.31±17.86	0.191
LVM/ $height^{2.7}$(gm/m^2)	50.66±7.13	52.07±9.30	<0.001
LVMI (gm/m)	106.86±13.87	112.35±17.89	0.029
RWT (mm)	0.43±0.039	0.41±0.051	<0.001
LVIDs (mm)	25.67±1.47	36.47±3.05	<0.001
LVIDd (mm)	46.30±1.73	48.17±3.85	<0.001
LVPWTd (mm)	10.16±0.856	9.91±0.681	0.041
IVSTd (mm)	10.02±0.802	10.21±0.827	0.138
LAD (mm)	29.45±3.72	27.89±3.85	0.03

Table 3: This shows the pattern of Left Ventricular geometry in both study groups

Left Ventricular Geometry	Control Female (n=40)	Case Female (n=75)
Normal Geometry	25(62.5%)	23(30.6%)
Concentric Remodelling	0	5(6.6%)
Eccentric Hypertrophy	15(37.5%)	47(62.6%)
Concentric Hypertrophy	0	0

DISCUSSION

Alterations in heart architecture, mass, geometric pattern, function and size are manifested clinically due to alterations at molecular and cellular level after cardiac injury. These changes are considered as cardiac remodeling and after MI play a key role in pathophysiology of ventricular dysfunction.[8] So post Acute Myocardial Infarction it becomes necessary to investigate the patients for any cardiac remodelling.[9] These changes can be assessed by using Echocardiography which is a reliable and non-invasive investigation available in most of the tertiary care centers in the country. The present study was taken up with an aim to study the impact of MI on left ventricular mass and left ventricular geometry.

Studies have reported that the size of the infracted area influenced several hemodynamic alterations which are noted after MI.[10] As shown in table 1 statistically significant difference was noted in SBP, DBP and MAP (p<0.001) and not in PP in the female study group.

Master AM et al,[11] in their study on 538 patients with initial and recurrent MI reported decrease in the blood pressure during the phase of hospitalization. Gibson T et al,[12] described that early after myocardial infarction, without taking into consideration the possible effects of heart failure, diuretic therapy, sedatives and narcotics a considerable decrease in blood pressure was seen. The most plausible explanation is that the depression of stroke volume due to ineffective contraction of the infarcted myocardium causes fall in the blood pressure. As SBP is a major contributor to myocardial oxygen demand, elevation of SBP can exacerbate the clinical manifestations of MI and have adverse effect on the prognosis.[13] But on the contrary, there is severe pump dysfunction if this hypotensive state persists for a long duration after MI.

In the present study a statistically significant difference (p=0.002) was noted in heart rate and relative bradycardia was seen in MI cases. The reason for the decrease in heart rate was administration of β-blockers which are known to cause decrease in heart rate. On the contrary, Freis ED et al,[10] in their study reported that with increasing severity of infarction the heart rate also increased in patients of myocardial infarction. The average HR mentioned in their study was 92 bpm in mild cases, 101 bpm in moderately severe cases and 128 bpm in severe cases.[10]

Increased LV mass is strongly associated with the development of diastolic heart dysfunction hence to assess LVH, left ventricular mass was calculated using Devereux formula.[6] A study has mentioned that LV enlargement after myocardial infarction is associated with congestive heart failure leading to decreased survival and the risk of death increases in direct relation to LV size.[14]

In the present study we observed that left ventricular internal dimensions during systole and diastole showed a statistically significant increase in MI cases (LVIDs: p<0.001, LVIDd: p<0.001).

Pandey AK et al,[15] in their study in overweight and obese Indian subjects found LVIDd- 45.9±6.5 mm and LVIDs- 28.6±3.7 mm in overweight subjects which is similar to our observations in control group which also constituted of obese subjects. Our results are also in agreement with experimental study of Gao XM and colleagues,[16] who in their study of serial echocardiographic assessment of left ventricular dimensions and function after myocardial infarction in mice noted a progressive increase in LVIDd and LVIDs where as there was a decline in fractional shortening.

LV hypertrophy is a strong risk factor for cardiovascular morbidity and mortality. LV mass is influenced by several physiological factors like age, sex, height, BMI and SBP. Several indexes for body size correction have been proposed, such as height, diverse allometric height adjustments, weight, body surface area, body mass index, and free-fat mass.[17] The best way for normalization of LV mass is still

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controversial but as per the literature available, in the present study indexing to height, BSA and height 2.7 was done.

It was observed that LVM, LVM/BSA, LVM/Ht, LVM/Ht2.7was higher in the AMI cases and the difference was statistically significant ($p=0.005$, $p=0.1$, $p=0.02$, $p<0.001$ respectively). Increase in LV mass in AMI cases is indicative of the fact that hemodynamic load was increased.

Studies have reported that LVH increases myocardial oxygen consumption, which may lead to myocardial ischemia or heart failure due to diastolic dysfunction. Also, LVH reflect a prolonged exposure to other cardiovascular risk factors, such as hypertension and atherosclerosis.[18]

Following AMI the development of LV dilatation is one of the most feared consequences of the complex process of ventricular remodeling, due to alterations in architecture and function of the left ventricle. Ventricular remodeling involves both the infarcted and non-infarcted zone, and is considered as one of the major determinants of poor outcome. Pfeffer MA and Braunwald E,[19] in a rodent MI model showed that a greater degree of myocardial injury was associated with a greater degree of chamber remodeling over time.

In the present study we noted that in control group the geometrical patterns seen were: NG 25, CR 0, EH 15, CH 0; and in cases: NG 23, CR 5, EH 47, CH 0. On further analysis, we found that EH was more common in older subjects with higher BMI and SBP and CH was seen in still older age group subjects having highest SBP.

Verma A et al,[20] in their study on MI cases reported 12.6% had CH, 18.6% had EH, 18.2% had CR and 50.5% had NG. Also, they found that, as compared with patients without evidence of LV remodeling, patients with any of the patterns of LV remodeling had a greater risk of cardiovascular death, heart failure, stroke, or resuscitated cardiac arrest.

In the present study we noted eccentric hypertrophy was the most common geometrical pattern observed in AMI study group. The importance of this finding is higher association with ventricular arrhythmias.

Levy D et al,[21] reported that eccentric LVH has been associated more strongly with ventricular arrhythmias whereas concentric LVH shows a strong association with increased over all cardiovascular mortality.

CONCLUSION

In our study group LV geometry showed changes of eccentric hypertrophy, concentric remodelling and concentric hypertrophy in patients of MI. The present study brought into light several structural changes after acute myocardial infarction. This can help to identify the patients who have greater risk of morbidity and mortality post acute MI. These changes if identified early can help the clinician to institute appropriate therapeutic measures. Though further studies are required to document the behavior of L.V. under pharmacological and physiological stresses.

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Conflict of Interest: Further studies are required to document the behavior of L.V. under pharmacological and physiological stresses.

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