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Post myocardial infarction assessment of left ventricular geometry by echocardiography

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Abstract---BACKGROUND AND AIM: Left ventricular dilatation is a well recognized precursor of 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 subjects after acute MI. METHOD: A hospital based prospective study was conducted at tertiary care center in Uttar Pradesh, India with a total of 30 patients including 17 Males and 13 Females having acute MI along 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 for males/females and calculations were done by using Microsoft Excel 2010 software. RESULTS: The geometrical parameters were compared and analysed. P value was calculated. P value<0.0001 gave highly significant results and <0.05 gave significant results. LVIDd(<0.05), LVIDs(<0.05), PWTd(<0.05), LVM(<0.05), LVMI(BSA)(<0.05), LVMI(Ht^{2.7})(<0.05), 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 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, perhaps by aiding in the identification of subjects with subclinical left ventricular dysfunction. Though further studies are required to document the behavior of L.V. under pharmacological and physiological stresses.

Keywords---myocardial infarction, echocardiography, left ventricular geometry, left ventricular internal dimension, posterior wall thickness, left ventricular mass, left ventricular mass index.

Introduction

Coronary artery disease is the leading cause of mortality and morbidity and acute myocardial infarction (MI) is the commonest mode of its presentation¹. Acute myocardial infarction (AMI) is characterized by regional myocardial damage that may lead to systolic and diastolic dysfunction associated with a subsequent risk of left ventricular (LV) remodeling and dysfunction. The pathophysiology and prognosis of LV geometrical changes after AMI have been the focus of research for several decades.² Acute myocardial infarction triggers structural, geometric, biochemical and functional changes in both the infarcted and non-infarcted regions of the left ventricular (LV) myocardium. Changes in LV structure and function after acute infarction can be recorded echocardiographically a few seconds after coronary artery blockage. These changes form the substrate for the early and late complications of acute infarction. Heart failure, severe arrhythmias, mitral regurgitation and sudden death are common manifestations after acute myocardial infarction³.

Ventricular remodeling, occurs progressively in untreated patients after large myocardial infarction. The term ventricular remodeling refers to alteration in ventricular architecture, with associated increased volume and altered chamber configuration⁴. Left ventricular remodeling is the process by which the heart changes its size, geometry and function over time. Quantitative M-mode transthoracic echocardiography enables characterization of LV remodeling that occurs in myocardial infarction and in a variety of heart diseases. A unique form of remodeling occurs following myocardial infarction due to the abrupt loss of contracting myocytes. Between one-half and one-third of post-infarction patients experience progressive dilatation with distortion of ventricular geometry and mitral regurgitation.⁵ Quantification of cardiac chamber size, ventricular mass and function ranks among the most clinically important and most frequently requested tasks of echocardiography.⁵

Material 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 included total of 30 patients including 17 Males and 13 Females having acute MI between age 30 and 60 years. 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). Both male and female suspected cases of Myocardial infarction coming for admission to cardiac coronary care unit were included.

Conditions that could alter results like old MI, Hypertension, diabetes mellitus, CCF, 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(Ht^{2.7}), LVMI(g/m), RWT was calculated.

Calculations

Body surface area (BSA) [16] $BSA = 0.6 \times \text{height (m)} + 0.0128 \times \text{weight (kg)} - 0.1529$

Body mass index (BMI) = $\text{Weight} / \text{Ht}^2$

Relative wall thickness (RWT) = $2 \times \text{PWd} / \text{LVIDd}$.

Left ventricular mass (LVM) = $0.8 [1.04 (\text{IVS} + \text{LVIDd} + \text{PWT})^3 - (\text{LVIDd})^3] + 0.6$ (Devereux formula)⁶

LVMI (indexed to BSA) = LVM / BSA .

LVMI (indexed to Height^{2.7}) = $\text{LVM} / \text{Ht (m)}^{2.7}$

LVMI (indexed to Height) = $\text{LVM} / \text{Ht (m)}$

Identification of LV geometric pattern based on parameters for structural changes:

- Normal geometry-normal RWT and normal LVMI
- Concentric remodeling- increased RWT and normal LVMI
- Eccentric hypertrophy- normal RWT and increased LVMI
- 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 for each group between males and females by using Microsoft Excel 2010 software. Mean $\pm$ 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. A statistically nonsignificant difference was noted in hemodynamic parameters with lower values noted in female AMI cases as depicted in Table/Fig 1. Table/Fig 2 shows the echocardiographic parameters in both study groups. A statistically significant

difference was noted in all the parameters in both study groups when comparing to normative data.

Table/ Fig 1
Showing baseline parameters (Mean $\pm$ SD) of both study groups

	Male (n 17) (Mean $\pm$ SD)	Female (n 13) (Mean $\pm$ SD)	P value
Age (years)	53.16 $\pm$ 7.91	54.56 $\pm$ 5.53	0.67
Weight (kg)	57.00 $\pm$ 5.13	56.96 $\pm$ 4.68	0.95
Height (cm)	158.10 $\pm$ 6.19	157.64 $\pm$ 6.15	0.50
BMI (kg/m ²)	23.56 $\pm$ 2.01	23.01 $\pm$ 2.44	0.58
BSA (kg/m ²)	1.59 $\pm$ 0.07	1.58 $\pm$ 0.07	0.29
HR (bpm)	70.67 $\pm$ 1.13	69.88 $\pm$ 2.96	0.65
SBP (mmHg)	117.07 $\pm$ 2.12	116.82 $\pm$ 3.97	0.88
DBP (mmHg)	70.04 $\pm$ 3.15	69.30 $\pm$ 4.50	0.46
PP (mmHg)	45.03 $\pm$ 1.18	47.52 $\pm$ 11.32	0.42
MAP (mmHg)	87.71 $\pm$ 6.22	86.15 $\pm$ 7.13	0.62

BMI- body mass index, BSA- body surface area, HR- heart rate, SBP- systolic blood pressure, DBP- diastolic blood pressure, PP- pulse pressure, MAP- mean arterial pressure, $p < 0.05$ is significant

Table/Fig 2
Showing left ventricular geometry indices based on echocardiography (Mean $\pm$ SD) in both study groups

LEFT VENTRICULAR STRUCTURAL PARAMETERS	NORMATIVE DATA	MALE (N=17) MEAN $\pm$ SD	FEMALE (N=13) MEAN $\pm$ SD	P VALUE
LVIDd	44.84 $\pm$ 1.25 ⁸	48.96 $\pm$ 5.93	43.333 $\pm$ 4.61	0.0086
LVIDs	24.94 $\pm$ 0.229 ⁸	33.15 $\pm$ 6.544	28 $\pm$ 3.46	0.0158
PWTd	10.10 $\pm$ 0.44 ⁸	11.09 $\pm$ 1.254	10.16 $\pm$ 1.04	0.0389
IVSTd	10.10 $\pm$ 0.45 ⁸	11.22 $\pm$ 1.373	10.66 $\pm$ 1.52	0.299
LVM	168.1 $\pm$ 39.2 ⁸	210.80 $\pm$ 53.24	157.02 $\pm$ 40.75	0.0053
LVMI(BSA)	93.43 $\pm$ 6.94 ⁸	122.76 $\pm$ 32.46	100.21 $\pm$ 22.74	0.0419
LVMI(Ht ^{2.7})	39.36 $\pm$ 3.22 ⁸	58.25 $\pm$ 31.17	48.81 $\pm$ 22.24	0.0075
LVMI(g/m)	94.17 $\pm$ 5.6 ⁸	98.21 $\pm$ 19.02	92.25 $\pm$ 11.86	0.0131
RWT	0.45 $\pm$ 0.03 ⁸	0.458 $\pm$ 0.064	0.474 $\pm$ 0.080	0.4399

LVIDd- Left ventricular internal dimension(diastole), LVIDs- Left ventricular internal dimension(systole), PWTd- Posterior wall thickness(diastole), IVSTd- Inter ventricular septal wall thickness(diastole), LVM- Left ventricular mass, LVMI(BSA)- Left ventricular mass index(indexed to Body surface area), LVMI(Ht^{2.7})- Left ventricular mass index(indexed to Height^{2.7}), LVMI(g/m)- Left ventricular mass index(indexed to Height), RWT- Relative wall thickness, $p < 0.05$ is significant

Discussion

Subjects were stratified according to quintile of RWT and also according to LV geometrical pattern. Partition values for LVMI (g/m) and RWT were: Indian Asian males—118/0.50 and Indian Asian females—107/0.47. The values of LVM, LVMI (BSA), LVMI ($Ht^{2.7}$), LVMI (g/m) was on higher side in males as compared to females and the normative data. The LV dimensions were increased in male cases as compared to females suggestive of greater effect of MI on males as compared to females. Serial observations of left ventricular dimensions after myocardial infarction suggest that dilatation of the left ventricle is implicated in the development of progressive cardiac dysfunction and congestive heart failure. Left ventricular dimensions emerged as the most important echocardiographic predictor of congestive heart failure.⁹ Systolic and diastolic thinning occurred only in patients with acute infarction. The presence of wall thinning was indicative of an acute ischemic event in our study.¹⁰ The compensatory ventricular hypertrophy is not accomplished during 1st year post-MI, therefore, left ventricular (LV) dilatation and thinning continues progressively both in infarct and noninfarct area due to the volume overload and the increase in global wall tension, according to Laplace's and Frank-Starling law.

The increase of wall stress further exacerbates energy imbalance and ischemia, especially in the subendocardial layer, which causes the additional apoptosis in the affected area. Also, overstretching of the ventricular wall and cardiomyocytes destroy the functional sarcomeres and further impairs contractility. As a consequence of this, the left ventricle undergoes wall thinning, chamber dilatation, and reshaping from the elliptical to spherical form years after onset of MI. Cardiac remodelling is the key mechanism and predictor of the late adverse outcome after myocardial infarction¹¹. Postinfarction remodeling has been arbitrarily divided into an early phase (within 72 hours) and a late phase (beyond 72 hours). The early phase involves expansion of the infarct zone, which may result in early ventricular rupture or aneurysm formation. Late remodeling involves the left ventricle globally and is associated with time-dependent dilatation, the distortion of ventricular shape, and mural hypertrophy¹². In our study it was an early phase (within 72 hours) so there was no increase in LVM as there was no hypertrophy.

The LV chamber dilatation causes increased internal dimensions and further the myocardial thinning and necrosis causes a decrease in the LV mass during early post MI phase. In Early Remodelling Infarct expansion occurs within hours of myocyte injury which results in wall thinning and ventricular dilatation, causing the elevation of diastolic and systolic wall stresses¹². The Late remodeling process is by hypertrophic myocyte elongation in the noninfarcted zone, which results in increased wall mass, chamber enlargement, and a shift from an elliptical to a more spherical chamber configuration⁴.

Conclusion

From the study, it can be concluded that the overall L.V. geometry was affected among Acute Myocardial Infarction patients, markedly in males as compared to females suggestive of greater effect of MI on males as compared to females. An

increase in left ventricular internal dimension is a risk for congestive heart failure. 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. 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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