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Original Research Article

LEFT VENTRICULAR GEOMETRICAL ANALYSIS IN MALE PATIENTS POST MYOCARDIAL INFARCTION BY ECHOCARDIOGRAPHY

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ABSTRACT

Background: LV enlargement after myocardial infarction is associated with decreased survival from congestive heart failure, arrythmia and increased risk of death. Present study is to evaluate the Left ventricular geometry and the determinants of L.V. geometry by M-mode Echocardiography in male subjects after acute MI.

Materials and Methods: A hospital based prospective study was conducted at tertiary care center in Uttar Pradesh, India with a total of of 125 male cases of Myocardial Infarction who fulfilled the inclusion criteria and 60 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.

Results: 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), LVM (<0.001), LVMI (BSA) (<0.001), LVMI (Ht2.7) (<0.001), LVMI (g/m) (<0.001) were highly significant respectively.

Conclusion: From the study, it can be concluded that the overall L.V. geometry was changed among Acute Myocardial Infarction patients. Arrhythmias and congestive heart failure can occur due to increase in left ventricular internal dimension. Knowledge of the left ventricular dimension controls the risk of congestive heart failure assumed due to traditional risk factors. 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, Left ventricular mass, Left ventricular mass index.

INTRODUCTION

In industrialized world Acute Myocardial Infarction (AMI) is a major health issue and most common disease in industrialized world.[1] People are exposed to adverse IHD risk factors irrespective of their habitat (rural vs. urban) due to changing lifestyles.[2] Increased prevalence of metabolic abnormalities like high triglyceride concentration, increased total cholesterol and high-density lipoprotein ratio, type 2 diabetes mellitus, and central or visceral obesity has increased incidence of coronary artery disease in South Asian population.[3] Heart architecture, mass, geometric pattern, function and size is altered after cardiac injury due to molecular, cellular and interstitial alterations, which are manifested clinically. This phenomenon is known as cardiac remodeling.[4] Post infarction remodeling has been divided into early phase i.e., within 72 hours and late phase beyond 72 hours.[5]

Also, AMI is associated with different remodeling patterns with varied prognostic implications. Verma et al,[6] observed four distinct geometrical patterns in patients post myocardial infarction which are having left ventricular dysfunction: concentric hypertrophy

(12.6%), eccentric hypertrophy (18.6%) concentric remodeling (18.2%) and normal pattern (50.6%) in their landmark VALIANT echocardiographic study. They reported concentric LV hypertrophy has greatest risk of adverse cardiovascular events including death.

Over the past decades, for the assessment of patients with AMI echocardiography has made major advancements in cardiology. Besides helping in guiding therapy in patients after MI, it also provides substantial information regarding risk stratification, hemodynamic assessment, detection of mechanical complications, and long-term prognosis in AMI.[7] Left ventricular mass can be assessed using Quantitative M-mode transthoracic echocardiography. Distorted ventricular geometry, ventricular dilation and mitral regurgitation is experienced by one-half to one-third patients approximately.[8]

To stratify risk of morbidity and mortality, left ventricular geometrical pattern can be defined which is based upon the LVMI and RWT.

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 125 male cases of Myocardial Infarction who fulfilled the inclusion criteria and 60 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) BSA = 0.6 × height (m) + 0.0128 × weight (kg) –0.1529.[16]

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).[9]

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.[10]

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 highly significant difference and the AMI cases having lower values as depicted in [Table 1].

Table 1: Both study group's base parameters

	Controls Male (n=60) (Mean ± SD)	Cases Male (n=125) (Mean ± SD)	P value
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 (m2)	1.74±1.53	1.62±1.42	0.061
HR (bpm)	80.43±2.24	70.67±11.60	<0.001
SBP (mm Hg)	126.43±1.99	117.07±10.73	<0.001
DBP (mm Hg)	77.96±2.13	73.04±8.45	<0.001
PP (mm Hg)	48.46±3.11	44.03±9.09	<0.001
MAP (mm Hg)	94.11±1.53	87.74±8.21	<0.001

In [Table 2] Except for the parameters like, PWT (diastolic), IVST (diastolic), LAD statistically highly significant difference (P<0.001) was observed between the two study groups. Difference in Relative wall thickness (mm) was significant P=0.013.

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

Variable	Control Male (n=60) (Mean ± SD)	Case Male (n=125) (Mean ± SD)	P value
For assessing the structural changes in heart			
LVM (gm)	162.72±21.10	181.48±30.59	<0.001
LVM/ BSA (gm/m2)	95.75±13.04	114.40±20.09	<0.001
LVM/height2.7(gm/m2)	40.42±5.67	51.46±10.56	<0.001
LVMI (gm/m)	97.13±12.78	113.65±20.0	<0.001
RWT (mm)	0.43±0.04	0.42±0.05	0.013
LVIDs (mm)	25.16±1.52	34.86±3.83	<0.001
LVIDd (mm)	46.04±2.06	48.42±3.37	<0.001
LVPWTd (mm)	10.05±0.89	10.14±1.08	0.526
IVSTd (mm)	10.02±0.97	10.27±1.27	0.090
LAD (mm)	29.13±3.50	29.43±4.33	0.321

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

Left Ventricular Geometry	Control Male (n=60)	Case Male (n=125)
Normal Geometry	57(95%)	77(61.6%)
Concentric Remodelling	1(1.6%)	5(4%)
Eccentric Hypertrophy	1(1.6%)	39(31.2%)
Concentric Hypertrophy	1(1.6%)	4(3.2%)

DISCUSSION

Several alterations are observed at molecular and cellular level after cardiac inury which, are manifested as alterations in heart architecture, mass, geometric pattern, function and size cinically. These changes play a key role in pathophysiology of ventricular dysfunction after MI, and are referred to as cardiac remodeling.[4] So after acute Myocardial Infarction it becomes necessary to investigate the patients for any cardiac remodeling.[2] These changes can be evaluated by using Echocardiography which is available in most of the tertiary care centers in the country as a reliable and non-invasive investigation. The present study was taken up with an aim to study the effect of MI on left ventricular mass and left ventricular geometry and function.

Studies have reported that the size of the infracted area influences several hemodynamic alterations which are seen after MI.[11] As shown in table 1, a statistically highly significant difference was noted in SBP, DBP, PP and MAP (p<0.001) in the male study group.

Master AM et al,[12] in their study on 538 patients with initial and recurrent MI during the phase of hospitalization reported fall in the blood pressure. Gibson TC et al,[13] described a considerable fall in blood pressure early after myocardial infarction, without considering the possible effects of heart failure, diuretic therapy, sedatives and narcotics. The most plausible explanation for fall in the blood pressure is depression of stroke volume due to ineffective contraction of the infarcted myocardium. Elevation of SBP can exacerbate the clinical manifestations of MI and have adverse effect on the prognosis, as SBP is a major contributor to myocardial oxygen demand14. Severe pump dysfunction can occur if this hypotensive state persists for a long time post MI, on the contrary.

In the present study a statistically highly significant difference (p<0.001) 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,[11] in their study reported that with increasing severity of infarction heart rate increased in patients. 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.[11]

Diastolic heart dysfunction is strongly associated with increased LV mass hence to assess LV Hypertrophy, left ventricular mass was calculated

using Devereux formula.[9] A study has mentioned that there is decreased survival from congestive heart failure due to LV enlargement post myocardial infarction and LV size is direct related to increased risk of death.[15]

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

Pandey AK et al,[16] in their study in overweight and obese Indian subjects found LVIDd- 45.9±6.5 mm and LVIDs- 28.6±3.7 mm and it is matching to our observations in control group which also included obese subjects. Our results are also in agreement with experimental study of Gao XM and colleagues,[17] who in their study noted a progressive increase in LVIDd and LVIDs where as there was a decline in fractional shortening, during repeated echocardiographic evaluation of left ventricular dimensions and function post myocardial infarction in mice.

LV hypertrophy is a strong risk factor for cardiovascular morbidity and mortality. Several physiological factors like age, sex, height, BMI and SBP influence LV mass. Height, diverse allometric height adjustments, weight, body surface area, body mass index, and free-fat mass are the indexes for body size correction that have been suggested.[18] There is still controversy for normalization of LV mass 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/Ht 2.7 was higher in the AMI cases and the difference was statistically highly significant ($p<0.001$). Increase in LV mass in AMI cases indicated increased haemodynamic load.

Studies have reported that myocardial oxygen consumption is increased in LVH, causing myocardial ischemia or diastolic dysfunction leading to heart failure. Also, LVH reflect a prolonged exposure to other cardiovascular risk factors, such as hypertension and it may be a marker of atherosclerosis.[19]

Following AMI one of the most harmful consequences of the complex process of ventricular remodeling is the development of LV dilatation, due to alterations in architecture and function of the left ventricle. Ventricular remodeling is considered as one of the major determinants of poor outcome and it involves both the infarcted and non-infarcted zone. Pfeffer MA and Braunwald E,[20] showed that a greater degree of chamber remodeling was associated with a greater degree of myocardial injury over time in a rodent MI model.

In the present study we noted that in control group the geometrical patterns seen were: NG 57, CR 1, EH 1, CH 1; in male cases: NG 77, CR 5, EH 39, CH 4; 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 al6 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, post MI patients with any of the patterns of LV remodeling had a greater risk of cardiovascular death, heart failure, stroke, or resuscitated cardiac arrest as compared with patients without any evidence of LV remodeling.

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

Levy D et al,[21] reported that concentric LVH was strongly associated with increased over all cardiovascular mortality and eccentric LVH has been associated more strongly with ventricular arrhythmias.

CONCLUSION

In our study group LV geometry showed changes of eccentric hypertrophy, concentric remodelling and concentric hypertrophy in patients of MI. There is a need to identify the structural and functional changes in heart post AMI so that rationale therapeutic interventions can be instituted timely and prevent short and long term complications. Though further studies are required to document the behavior of L.V. under pharmacological and physiological stresses.

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