Original article

Comparison of tissue doppler derived systolic strain between different severities of left ventricular dysfunction in patients having rheumatic severe mitral regurgitation

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ABSTRACT

Background: Tissue doppler derived systolic strain detects the subtle changes in left ventricular (LV) function. This new modality can detect LV dysfunction in severe rheumatic mitral regurgitation (MR) at earlier stages. The present study was conducted to compare tissue doppler derived peak systolic strain between patients with different severities of LV dysfunction in rheumatic severe MR and to look for the negative correlation between LV dysfunction and peak systolic strain.

Subjects and methods: A descriptive correlational study was conducted from January 2017 to March 2018 at Punjab Institute of Cardiology, Lahore. Fifty healthy controls taken as group-I and 150 asymptomatic patients of rheumatic severe MR were divided into Group-II (with ejection fraction [EF] ≥60%) and left ventricular end systolic dimension (LVESD) ≤40mm), Group-III (with EF ≥60% and LVESD 41-50mm) and Group-IV (with EF <60%). Longitudinal peak systolic strain (PSS) of the groups were measured by tissue doppler imaging and compared. The correlation between systolic dysfunction and PSS was further studied.

Results: Out of 200 subjects, 91 (45.5%) were male and 109 (54.5%) female. Mean age of study subjects was 31± 9.5 years. Moving from group-I to group-IV, mean LVESD increased from 23.3±2.4 to 49.3±3.0, mean LVEDD (left ventricle end diastolic dimension) increased from 46.4±3.4 to 64.0±3.6, ejection fraction decreased from 63.6±2.1to45.7±6.7 and average PSS decreased from 17.8±0.53 to 8.31±0.52. A Significant difference was found in average PSS between these groups (p<0.001).There was a significant (p<0.001) negative correlation (correlation coefficient = -0.968) between average PSS and LV dysfunction (i.e. group number).

Conclusion: In rheumatic severe mitral regurgitation, tissue doppler derived peak systolic strain decreases with increase of LV dysfunction showing a significant negative correlation between the two.

Keywords:

Mitral regurgitation, Systolic strain, Tissue doppler

INTRODUCTION

Rheumatic heart disease (RHD) is one of the most common causes of valvular heart disease in this country. Its prevalence between 6 to 15 years of age has been described as 14.6 per 900 Pakistani patients and mitral regurgitation (MR) is found the most common (56%) valvular lesion to occur in rheumatic heart disease, even more common than mitral stenosis (20.3%).[1,2] Mitral valve surgery is indicated in severe MR if patient is symptomatic or if left ventricular ejection fraction (LVEF) drops to ≤ 60% or left ventricular end systolic dimension (LVESD) reaches ≥ 40mm.[3] Although LVEF was the first predictor to be used to sort out asymptomatic patients of severe MR for surgery, it does not drop during a long compensated phase of chronic severe MR and hence mortality is high if surgery is delayed till LVEF drops below 60%. The left ventricular end systolic dimension (LVESD) emerged as a more sensitive indicator of surgery in MR patients. Even waiting for the LVESD to reach ≥40 mm is independently associated with poorer postoperative medium and long term survival in severe MR.[3] Better parameters are still needed to refer patients of MR for surgery at proper time for better postoperative outcomes. Systolic strain and strain rate are the parameters that can detect left ventricular (LV) dysfunction earlier than conventional echocardiographic parameters.[4] Even subtle change in LV function is detected by the strain imaging. The peak systolic strain (PSS) is the measure of LV function

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which is quantified in echocardiography lab by tissue doppler method and by speckle tracking method.[5,6] There have been studies in which strain imaging detected subclinical LV dysfunction at earlier stages than the conventional echocardiographic parameters in patients with severe MR due to dilated cardiomyopathy, due to ischemia, after mitral valve (MV) surgery and after clipping of MV.[7-10] There are very few studies in this respect regarding MR due to rheumatic heart disease as this is not now a common disease of the developed nations. There is no local study available on this topic in Pakistan in the published indexed literature although rheumatic heart disease remains a major health problem of this country. This study compares the peak systolic strain between patients of different severities of LV dysfunction in severe rheumatic MR to develop a new parameter of LV function as an indictor of LV dysfunction at the earlier stages of the disease.

SUBJECTS AND METHODS

It was a descriptive correlational study in which non-probability purposive sampling was done between January 2017 and March 2018 from Punjab Institute of Cardiology, Lahore. One hundred and fifty asymptomatic patients of rheumatic heart disease with chronic severe MR and fifty healthy controls (all subjects having 15-60 years age) were included in the study after taking informed consent. Exclusion criteria were non-rheumatic MR, ischemic heart disease (based on history, clinical examination or segmental wall motion abnormalities on echocardiography) and more than moderate degree of mitral stenosis, aortic stenosis, or aortic regurgitation. All patients and controls underwent echocardiography using Vivid-7 dimensions machine by GE company serial number 11607V7L. Severe MR was defined as area of regurgitant jet being >50% of the area of left atrium and having a vena contracta of >0.7 cm. Parameters measured on echocardiography were left ventricular end diastolic dimension (LVEDD) and left ventricular end systolic dimension (LVESD)after placing M-mode on left ventricle in parasternal long axis view. Left ventricular ejection fraction (LVEF) was also measured by using Simpson's biplane method. Region of interest (ROI) of tissue doppler imaging (TDI) was placed on left ventricle (LV) in apical 4-chamber view and longitudinal peak systolic strain (PSS) at basal lateral and basal medial walls of LV were measured. The average of both strains was also taken. The angle of incidence between doppler beam and LV wall was kept <20° and minimal optimal gain of tissue doppler imaging (TDI) was used to decrease aliasing. Strain sample length was kept 12mm and Q-analysis function was used to assess strain after frame by frame manual tracking. In case of atrial fibrillation, the PSS of ten consecutive cycles was averaged to get the PSS of corresponding myocardial area. Fifty healthy controls were taken as Group-I. One hundred and fifty patients of MR were divided into three further groups according to severity of LV dysfunction i.e. Group-II (with EF ≥60% and LVESD ≤40mm), Group-III (with EF≥ 60% and LVESD 41-50mm) and Group-IV (with EF <60%). Data was analysed using SPSS version 22.0. Variables under study were age, gender, LVESD, LVEDD, EF, group number, peak systolic strain (PSS)at medial and lateral walls as well as average peak systolic strain. The groups were compared regarding peak systolic strain using ANOVA test and correlation between systolic dysfunction (i.e. group number) and peak systolic strain was observed using spearman rank correlation coefficient test. A p-value of ≤0.05 was taken as significant.

RESULTS

Out of 200 subjects, 91 (45.5%) were males and 109 (54.5%) were females. Mean age of the study subjects was 31± 9.5 years. Regarding echocardiographic parameters, mean values of LVESD, LVEDD, LVEF and average PSS were 37.6±10.3, 58.6±7.9, 59.5±8.9 and 12.9±3.6 respectively. Table 1 summarizes the general characteristics of study subjects.

Dividing into the groups I, II, III and IV, each group comprised fifty subjects. Comparing groups, mean LVESD progressively increased from 23.3±2.4 in group-I to49.3±3.0 in group-IV patients and mean LVEDD progressively increased from 46.4±3.4 in group-I to 64.0±3.6in group-IV. Similarly, ejection fraction of LV decreased significantly from group-I (63.6±2.1) to group-IV (45.7±6.7) with p-value of <0.001 (ANOVA). Table 2 depicts the group-wise characteristics of each group.

Table 1. General characteristics of the study subjects

	N (%)	Age (years)	LVESD	LVEDD	EF	Average peak systolic strain (%)
Male	91(45.5%)	32.5±9.4	37.7±10.6	58.8±8.6	59.3±9.2	12.9±3.7
Female	109(54.5%)	29.7±9.5	37.4±10.0	58.5±7.4	59.7±8.8	12.9±3.6
Total	200(100%)	31± 9.5	37.6±10.3	58.6±7.9	59.5±8.9	12.9±3.6

Table 2. Group-wise characteristics of the study subjects

Characteristics	Group-I	Group-II	Group-III	Group-IV	p-value (ANOVA)
Gender					
Male	24 (48%)	23 (46%)	20 (40%)	24 (48%)	
Female	26 (52%)	27 (54%)	30 (60%)	26 (52%)	
Age	31.2±9.3	30.7±9.9	29.7±9.4	32.4±9.7	0.582
LVESD	23.3±2.4	34.0±3.2	43.6±2.0	49.3±3.0	**<0.001**
LVEDD	46.4±3.4	62.3±3.8	61.8±3.3	64.0±3.6	**<0.001**
LVEF	63.6±2.1	65.8±2.8	63.0±1.8	45.7±6.7	**<0.001**

Table 3. Comparison of peak systolic strain between different groups

Peak Systolic strain (PSS)	Group-I	Group-II	Group-III	Group-IV	p-value (ANOVA)	Spearman rank	
						Correlation coefficient	p-value
Medial wall (PSS-Med)	17.6±0.53	14.62±0.45	10.78±0.48	8.16±0.53	<0.001	-0.969	<0.001
Lateral wall (PSS-Lat)	17.9±0.54	14.88±0.42	11.03±0.47	8.46±0.52	<0.001	-0.896	<0.001
Average PSS (PSS-Avg)	17.8±0.53	14.75±0.43	10.9±0.47	8.31±0.52	**<0.001**	**-0.968**	**<0.001**

Comparing medial, lateral and average peak systolic strains (PSS) between different groups, PSS at medial LV wall decreased progressively from group-I to group-IV being 17.6±0.53 in group-I and 8.16±0.53 in group-IV. Similarly, lateral PSS decreased from 17.9±0.54 in group-I to 8.46±0.52 in group-IV and average PSS decreased from 17.8±0.53 to 8.31±0.52 between group-I and group-IV respectively. Using ANOVA to compare average peak systolic strain between different groups of LV dysfunction showed significant difference in average PSS between these groups with p value of <0.001. Spearman rank correlation coefficient test revealed a significant negative correlation between LV dysfunction (i.e. group number) and average PSS (correlation coefficient= -0.968, p<0.001). Spearman rank correlation test further showed the significant negative correlations between LV dysfunction and PSS at medial wall (coefficient= -0.969, p<0.001) as well as LV dysfunction and PSS at lateral wall (coefficient= -0.896, p<0.001). Table 3 compares the peak systolic strain between different groups and Figures 1 reflects the graphic representation between group number and average peak systolic strain.

DISCUSSION

Mitral regurgitation (MR) is one of the commonest lesions found in rheumatic heart disease. One Pakistani study showed MR was found in 56% patients of rheumatic heart disease out of which 8% were having severe MR.[2] During the long compensated phase of severe rheumatic MR, left ventricle ejection fraction (EF) does not decrease as left ventricle goes on dilating to accommodate the regurgitant volume. Waiting for EF to decrease delays the surgery to irreversible loss. The previous study articles have shown that peak systolic strain (PSS) can detect LV dysfunction at very early subclinical stages.[11,12] Also the longitudinal PSS helps in an early detection of LV dysfunction in patients with preserved EF in settings of severe MR as well as during postoperative period after mitral valve surgery.[9,13-15] This study evaluated the correlation between LV dysfunction and average longitudinal PSS using the tissue Doppler method.[5] This new sensitive measure can be used to early detect LV dysfunction in patients with severe MR. Peak systolic strain can be measured by tissue doppler method or by speckle tracking method.[5,16] This study compared the longitudinal PSS of LV between different severity groups of LV dysfunction. The study exclusively included mitral regurgitation due to rheumatic heart disease as it is the disease of our region and so the lesser number of studies have been conducted in the west on this specific cause of mitral regurgitation. Tissue doppler method was used to measure PSS although previous studies on other causes of MR had used speckled tracking method to measure PSS.[7,17,18]

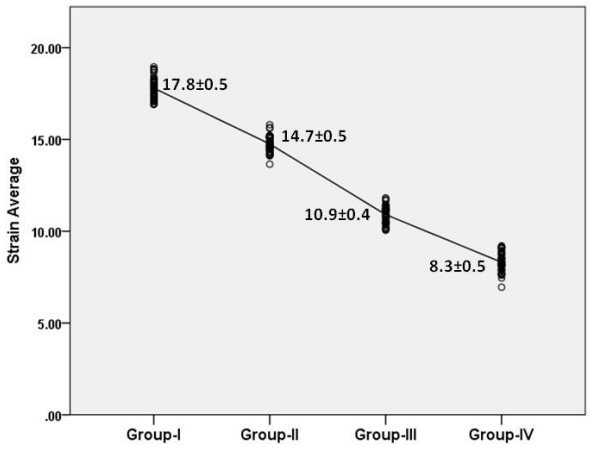

Figure 1. Scatter graph between group number (LV dysfunction) and average peak systolic strain

Speckled tracking method uses three different echocardiographic views to get PSS which is not possible in atrial fibrillation as cycle length varies.[6] Patients with severe rheumatic MR frequently have atrial fibrillation so we averaged the PSS taken in 10 consecutive cycles by tissue doppler method. Moreover, speckle tracking method needs a good quality grayscale image as the machine in this method tracks the speckles of grayscale image to give the systolic strain. This is not a necessity in tissue doppler method which can even get PSS at high frame rate and so has a high temporal resolution. Comparison between the groups revealed that moving from group-I to group-IV, means of both LVESD and LVEDD increased while mean LVEF decreased (ANOVA $p<0.001$) but this was because groups were defined such that with increasing group number LV dysfunction had to increase. So the grouping served the purpose of the study. Now comparing each of the longitudinal PSS at medial basal wall, lateral basal wall and the average PSS between the groups revealed that moving from group-I to group-IV i.e. with increasing LV dysfunction, the PSS also decrease progressively. Comparison of means with ANOVA test showed this change to be significant ($p <0.001$). Moreover, there was a significant negative correlation between LV dysfunction i.e. group number and average PSS (spearman's rank correlation coefficient -0.968, $p <0.001$). A previous study by Gunjan and coauthors compared the PSS between different severities of LV dysfunction in patients with severe rheumatic MR. The study found the similar negative correlation between LV dysfunction and systolic strain which was significant at p value of<0.05.[19] Another study compared speckle tracking derived 3D global strain and radial strain between the patients of asymptomatic severe MR and the healthy controls. They found significant difference between 3D global and radial strains of these groups. However, authors did not include severe MR patients with decreased EF in their study nor they compared the strain between different severities of LV dysfunction in MR patients as we did in the present study.[18] Yurdakul and colleagues compared global longitudinal PSS between healthy controls and patients of moderate to severe MR with normal EF.[20] It was found that longitudinal PSS was significantly decreased in moderate to severe MR compared with controls ($p=0.0001$). This decrease of PSS was even more significant on 12 months follow-up. Still in this study comparison between patients having LV dysfunction and those not having LV dysfunction was not made like our study. Moreover, the PSS by Yurdakul and coworkers was measured by velocity vector imaging (VVI) while we used tissue Doppler imaging (TDI) to measure longitudinal PSS.[20] Kamperidis and group used speckle tracking method to measure the global longitudinal strain (GLS) in patients with severe MR and mild MR and found a significant association between severe MR and impaired GLS ($p=0.03$) but the study was conducted on non-ischemic, dilated cardiomyopathy patients. Moreover, they compared the patients with different severities of MR while present study was conducted on comparison of longitudinal strain between different severities of LV dysfunction in patients having severe MR only.[7]

In present study, when each of the PSS at medial wall and lateral wall is considered separately and compared both these strains separately with LV dysfunction, a significant negative correlation between LV dysfunction and each of these peak systolic strains ($p<0.001$ with spearman rank test in both medial as well as lateral walls) was observed. This shows that taking peak systolic strain at only medial or lateral wall can also serve the purpose of demonstrating LV dysfunction in these patients. There had been very small number of studies on the assessment of LV function by strain imaging in severe MR that was caused by rheumatic heart disease specifically. This is because the rheumatic heart disease is not now common in the west and is now a big problem of the South Asian region including India and Pakistan. Gunjan and associates studied LV function assessment by strain imaging in MR specifically caused by rheumatic heart disease.[19] Other authors included patients with mixed causes of MR including degenerative, mitral valve prolapse and rheumatic patients.[18,20] Kamperidis and colleague studied exclusively the secondary MR due to non-ischemic dilated cardiomyopathy.[7] Limitations of present study were that tissue doppler imaging like all other doppler modalities is angle dependent and thus angle must be <20 degree to get better results. Moreover, tissue Doppler method gives systolic strain of a local area of myocardium where sample volume of tissue Doppler is placed as compared to global peak systolic strain given by speckle tracking method. Although in this study authors have compared LV dysfunction in MR patients using peak systolic strain as a measure of LV function, previous studies have also used strain rate and tissue velocities to compare LV dysfunction between patients of severe MR.[19] This domain still needs further research.

CONCLUSION

There is a significant negative correlation between left ventricular dysfunction and Doppler derived longitudinal peak systolic strain in asymptomatic patients with rheumatic severe mitral regurgitation.

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