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Research Brief

Assessment of right ventricular systolic function by two dimensional speckle tracking echocardiography in rheumatic mitral valve disease

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A B S T R A C T

We assessed the Right Ventricular (RV) systolic function using two dimensional (2D) speckle tracking echocardiography (STE) in Mitral valve disease before and after intervention. 90 patients divided into 3 groups of Mitral stenosis [MS], Mitral regurgitation [MR] and MS with MR were the study subjects. All the patients were subjected to conventional echocardiography and measurement of Right ventricular global longitudinal strain (RVGLS). Prior to intervention, subjects who demonstrated normal RV function by conventional methods, it was found that RVGLS was impaired significantly in the MS and MS with MR groups but normal in the MR group. After intervention, the RVGLS improved significantly in the MS group but not in the other groups. Right ventricular systolic pressure (RVSP) had a significant negative correlation to RVGLS in all 3 groups.

1. Introduction

The RV function is an important determinant of the clinical symptoms, exercise capacity, pre-operative survival and post-operative outcome in the patients with Mitral valve (MV) disease.[1] RV systolic dysfunction occurs early prior to the onset of clinical systemic congestion, therefore early detection has both diagnostic and therapeutic value. Echocardiographic assessment of RV function is challenging due to the complex anatomy of RV, narrow acoustic window, and geometric assumption in volumetric calculations.[2] Conventional methods like TAPSE, FAC and LATERAL S' have significant drawbacks such as angle dependency and analysis in only one dimension. Two dimensional (2D) strain and strain rate imaging by Speckle tracking enables angle independence and 2D assessment of RV function, it has been shown that 2D strain imaging is better than conventional Doppler and TDI in the evaluation of RV function.[2] STE has been developed for the quantitative assessment of global and regional myocardial function based on stress and strain.[3] The primary objective of our study is to compare the RVGLS before and after intervention in Rheumatic Mitral valve disease and to correlate with RVSP, which was measured by tricuspid regurgitation (TR) jet.

2. Methods

The study population consisted of 90 patients of Rheumatic Mitral valve disease with an established diagnosis of MS, MR and MS with MR in NYHA class II or more and no history of diabetes or hypertension who were admitted for either Balloon mitral valvotomy (BMV) or Mitral valve replacement (MVR). They were divided into three groups of 30 cases each: pure MS (group 1), pure MR (group 2) and combined significant MS with MR (group 3). All patients in Sinus rhythm with normal RV function demonstrated by any two conventional methods were included in this study. Patients with atrial fibrillation, RV dysfunction, moderate to severe Aortic valve disease, organic TV disease and LV systolic dysfunction (EF $<$ 50%) were excluded. Written informed consent was obtained from all the patients and the Institutional Ethical Committee accepted the study proposal.

2.1. Echocardiographic measurements

Echocardiography was performed using a 3.5 MHz probe of iE 33 Philips echocardiography machine by a single experienced operator. Patients were monitored by single lead electrocardiogram. The baseline measurements of the left and the right heart were taken

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according to the American society of echocardiography (ASE) guidelines. Measurements of RVSP greater than 40 mmHg defines pulmonary hypertension (PAH). Detailed assessment of RV function was done by measuring the RV Fractional Area Change (RVFAC), TAPSE and TDI Lateral S'.

2.2. Two dimensional strain imaging of RV

2D echocardiography images for speckle tracking were obtained from the modified A4CH view with frame rates of 40–70 frames/s. Digital data were stored and analysed off-line. The average of RVFW and septum were used to calculate the RV global strain. After defining the three points (RV apex, medial and lateral tricuspid annulus) the software automatically traced the endocardial and epicardial borders in modified A4CH view. The speckle tracking width was modified to cover the whole RV wall thickness to obtain strain imaging. After adjusting tracking points manually if required, longitudinal strain obtained. The cut-off values for all the variables were taken according to ASE guidelines (Lang et al 2015).[4]

Prior to intervention, 8 patients were excluded due to suboptimal images. After intervention, 12 patients were excluded, of which 2 were due to complications and 10 were lost in follow up. Patients were subjected to basal study 24 h before intervention, 24 h after BMV,7 days after MVR. Statistical analysis was done by using the software SPSS version 21.0. All the variables were expressed as mean ± standard deviation (SD). For paired data, paired *t*-test was utilized to determine any significant difference in RV strain before and after treatment. A *p* value of <0.05 was considered to be statistically significant. Pearson correlation analysis was done to evaluate the relationship between the variables of RVSP and RV strain before treatment in each group. Correlation is considered significant at the 0.01 level (2-tailed). Intra observer variability of global RV strain before and after treatment was assessed through Cronbach alpha method. We examined the degree of agreement in strain before and after treatment by same observer at two different intervals.

3. Results

The baseline variables are summarized in Table 1. Before intervention, the patients in group 1 had lower RVGLS which increased significantly after BMV (−15 ± 4.7 vs. −19 ± 3.6, *t* value = 4.83, CI = (1.91–4.76), *p* value = < 0.001). Group 2 patients had a normal RVGLS and after surgery there was no significant difference (-20 ± 4.2 vs. −21 ± 3.2, t-value = 1.38, CI = (−0.31–1.56), *p* value = 0.181). Group 3 patients had lower RVGLS at baseline, and Post surgery, there was no significant improvement in RVGLS (−16 ± 4.16 vs. −16 ± 7.80, t-value = −0.18, CI = (−2.94–2.47), *p* value = 0.858) (Fig. 1). All three groups showed significant negative correlation between RVSP and RVGLS with gp-2 showing the most ($r = -0.814$, $p < 0.001$). Sub group analysis of patients with RVSP > 40 mmHg showed significant improvement in post intervention RVGLS in group 1 (*p* value = < 0.001), group 2 (*p* value = 0.011), and group 3 (*p* value = 0.011) but no significant improvement in patients with RVSP < 40 mmHg. Intra observer variability assessed through Cronbach alpha, found greater agreement by Cronbach α of 0.970 and 0.946 in RV strain before and after intervention respectively.

4. Discussion

4.1. RV global longitudinal strain

LA hypertension leads to chronic pulmonary venous congestion followed by PAH, which is ultimately responsible for increased RV afterload and subsequent RV dysfunction. We observed a lower

Table 1
Baseline parameters.

VARIABLES	PURE MS (Mean ± SD)	PURE MR (Mean ± SD)	SIG MS + MR (Mean ± SD)	*p* Value
AGE	34.57 ± 8.85	35.10 ± 15.84	39.80 ± 13.35	0.236
GENDER M/F	7/23	9/21	16/14	0.039
TAPSE(>16 mm)	18.33 ± 2.39	20.57 ± 4.51	18.30 ± 1.97	0.13
FAC (>35%)	40.98 ± 11.27	52.42 ± 9.48	48.39 ± 11.23	<0.001
TDI lateral S' (>9.5 cm/s)	11.39 ± 1.38	13.04 ± 3.0	11.25 ± 5.11	0.116
LA dimension (men -<4.0 cm, women- < 3.8 cm)	4.32 ± 0.48	4.26 ± 0.58	4.89 ± 0.57	<0.001
LV dimension in diastole (men- 4.2–5.8 cm, women −3.8–5.2 cm)	4.36 ± 0.33	5.14 ± 0.36	5.00 ± 0.80	<0.001
LV dimension in systole (men- 2.5–4.0 cm, women- 2.2–3.5 cm)	2.89 ± 0.33	3.23 ± 0.49	3.15 ± 0.64	0.034
LVEF (>50%)	58.9 ± 2.42	60.19 ± 1.72	60.03 ± 1.82	0.044
RVSP (<40 mmHg)	54.46 ± 19.41	48.07 ± 17.31	56.73 ± 26.45	0.019

(LA −Left atrium, LV −Left ventricle, LVEF- Left ventricular ejection fraction, RVSP- Right ventricular systolic pressure, TAPSE- Tricuspid annular plane systolic excursion, FAC- Fractional area change, LATERAL S′- Peak systolic velocity at lateral tricuspid annulus).

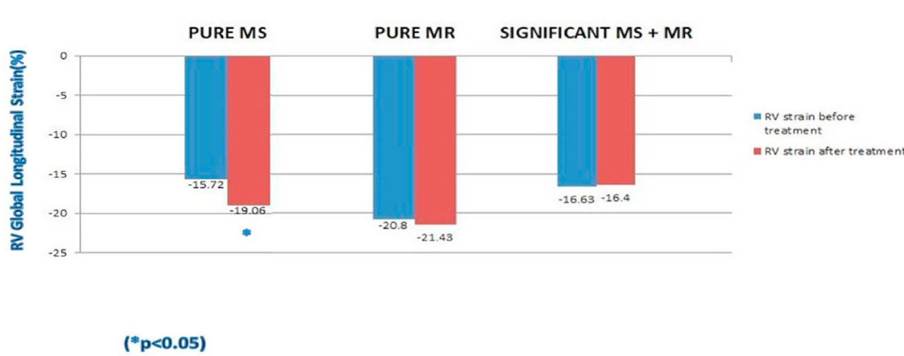

Fig. 1. Comparison of RVGLS before and after treatment in each group.

RVGLS in 83.3% of patients with pure MS before intervention. After intervention, a significant rise in RVGLS was observed. Earlier studies have shown impaired RV systolic function in patients with MS planned for BMV and attributed it to increased afterload.[5] They found a significant rise in global and septal strain, but not in RVFW after BMV, it was concluded that different RV segments are differently affected by different degrees of RV afterload and their function may not improve shortly after BMV. Another study in Moderate - severe MS patients had found lower RVGLS and ascribed it to direct involvement of the RV myocardium by the rheumatic process.[2] Our results are consistent with the earlier studies.

RV dysfunction is the result of a complex interaction between the remodelled and enlarged LV, septal performance and PASP with or without degenerative MR.[6–8] Before intervention, our study demonstrated that the MS with MR group had 65.4% and the pure MR group had 30.8% of patients with lower RVGLS. In both groups, there was no significant improvement in RVGLS after surgery. The lack of improvement in these groups is probably linked to the fact that open sternotomy directly impacts the longitudinal motion of the RV free wall and RV dysfunction may need long followup to show improvement. The pericardiotomy leads to loss of the pericardial assistance of the longitudinal deformation of the RV.[9] Other contributing factors maybe intraoperative ischemia and myocardial stunning secondary to poor protection during cardiopulmonary bypass (CPB).[10] One study had found RV systolic dysfunction by conventional echo in 32% of the patients with chronic degenerative MR undergoing MV repair and they found significant change in postoperative RV global strain only in patients with PASP > 50 mmHg.[11] Sub group analysis of our patients also revealed intervention helped to improve RV Systolic function only in patients with PAH.

4.2. Correlation analysis

We evaluated the correlation of LA, RVSP, RA pressure, RA area, RA major and minor axis, RV at the base and mid-level with RVGLS before intervention. The parameter which correlated most with RVGLS was RVSP in all three groups, which also showed a significant negative correlation with RVGLS. This means that increased RV afterload is the reason for RV systolic dysfunction.

The implications of our study are that patients of MV disease with impaired RVGLS may be taken up early for intervention and be better planned for inotropic support during intraoperative/perioperative period to improve the prognosis. STE can be considered in modifying the guidelines for intervention in valvular heart diseases.

Major limitations were the lack of a control group, lack of validation of reference in RVGLS, and short term followup. The methodological limitation was that we could assess the RV strain in modified A4CH view as we needed to calculate the average of all 6 segments of RV whereas the conventional parameters were measured in normal A4CH and focused A4CH view.

5. Conclusion

Pre intervention with a background of normal RV function by conventional methods, RV systolic function evaluated by RVGLS was impaired significantly in the MS and MS with MR groups but was normal in the MR group. Post intervention RV systolic function improved significantly in the MS group, but not in the MR and MS with MR groups. Subgroup analysis showed an improvement in the RV function in all three groups of patients with PAH.

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Declaration of competing interest

All authors have none to declare.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ihj.2021.01.003.

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