

Original article:

Tricuspid regurgitation and mitral stenosis: a retrospective study

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Abstract

Background: Rheumatic heart disease is common in India. Severity of mitral stenosis is the key factor in deciding for mitral valve surgery. Mitral stenosis is found to be associated with tricuspid regurgitation (TR) depending on its severity. We studied the severity and incidence of TR in patient of mitral stenosis and was managed accordingly.

Methods: This retrospective study was conducted at G B Pant Hospital from June 2021 to May 2022. Cases of Rheumatic heart disease with mitral stenosis were diagnosed clinically. 2D echocardiography was used to find severity of mitral stenosis. Data was entered into SPSS-17.0 and results were recorded and analysed. Pearson's two tailed correlation was used to find the correlation between presence of tricuspid regurgitation in patients with severe mitral stenosis, p was <0.05. All cases of severe mitral stenosis were selected for study. Patient having LA clot were excluded from study. Preoperative workup done.

Results: A total 28 patients with pure mitral stenosis were included in study, out of which 13 were male and 15 were females. Mean age in males was 50±10 years while in females it was 30±15 years. Thirteen out of 28 (46.42%) patients had severe tricuspid regurgitation while ten out of 28 (35.71%) had mild, 5 patient had no tricuspid regurgitation. Mean (MVA) mitral valve area in patients with tricuspid regurgitation was 0.8±0.3 cm² while mean (MVA) mitral valve area in patients mild and without tricuspid regurgitation was 1.4±0.4 cm². Mean left atrial (L.A) size was 42.50±1.5 mm² in patients with tricuspid regurgitation, while it was 40.13±5.14 mm² in patients without tricuspid regurgitation. Mean RSVP was 55mmHg in patients with tricuspid regurgitation while RSVP could not be calculated in patients without tricuspid regurgitation.

Conclusions: It was concluded that tricuspid regurgitation was strongly associated with severe mitral stenosis as almost all patients with severe mitral stenosis had tricuspid regurgitation and none of the patients with mild mitral stenosis had tricuspid regurgitation. Those who had severe mitral stenosis with moderate to severe tricuspid regurgitation, mitral valve replacement and de Vega tricuspid annuloplasty or tricuspid ring annuloplasty was performed.

Keywords: Rheumatic heart disease, Mitral Stenosis, Mitral Valve Area, Right Ventricular Systolic Pressure; Tricuspid regurgitation.

Introduction:

Rheumatic heart disease is common in India. Severity of mitral stenosis is the key factor in deciding for mitral valve surgery. Mitral stenosis is found to be associated with tricuspid regurgitation (TR) depending on its severity. We studied the severity and incidence of TR in patient of mitral stenosis and was managed accordingly.

Methods:

This retrospective study was conducted at G B Pant Hospital from June 2021 to May 2022 .Cases of Rheumatic heart disease with mitral stenosis were diagnosed clinically. 2D echocardiography was used to find severity of mitral stenosis. Data was entered into SPSS-17.0 and results were recorded and analysed. Pearson's two tailed correlation was used to find the correlation between presence of tricuspid regurgitation in patients with severe mitral stenosis, p was <0.05. All cases of severe mitral stenosis were selected for study. Patient having LA clot were excluded from study. Preoperative workup done. All Routine blood investigation was done. Electrocardiogram was done. Echocardiography (Fig1, Fig2) was done and assessment of mitral valve area, tricuspid annulus area, left atrial size. To rule out coronary artery disease, patient aged greater than 40yrs, coronary angiography was done. Patients were operated on cardiopulmonary bypass via median sternotomy through trans-septal approach. Transesophageal echocardiography was done. On table assessment of tricuspid regurgitation was done by finger insinuation method. Tricuspid annulus size was divided by patient BSA, and assessment of intervention for de Vegas and tricuspid annuloplasty was done.

Inclusion criteria- All male and female patients aged 18-60

Exclusion criteria – Patients with comorbidity diabetes and hypertension, hypothyroidism

Concomitant other valvular heart disease

Mitral stenosis with left atrial clot

Results:

A total 28 patients with pure mitral stenosis were included in study, out of which 13 were male and 15 were females. Mean age in males was 50±10 years while in females it was 30±15 years. Thirteen out of 28 (46.42%) patients had severe tricuspid regurgitation while ten out 28 (35.71%) had mild, 5 patient had no tricuspid regurgitation. Mean (MVA) mitral valve area in patients with tricuspid regurgitation was 0.8±0.3 cm² while mean (MVA) mitral valve area in patients mild and without tricuspid regurgitation was 1.4±0.4 cm². Mean left atrial (L.A) size was 42.50±1.5 mm² in patients with tricuspid regurgitation, while it was 40.13±5.14 mm² in patients without tricuspid regurgitation. Mean RSVP was 55mmHg in patients with tricuspid regurgitation while RSVP could not be calculated in patients without tricuspid regurgitation.

Table 1: Study of demographic characteristics

s.no	Demographic characteristic	Range	Mean±SD	Median(IQR)
1	Age(yrs)	18-60	37.60±11.69	37(17)
2	Gender Male	13	46.42%	
	Female	15	53.57%	
3	Height(cm)	150-171	159±8.04	160(18)
4	Weight(kg)	30-55	45.46±8.39	45(15)
5	BSA	1.15-1.64	1.43±0.168	1.44(0.31)

Table 2: Study of clinical characteristics

Sl.no	Clinical	
1	History of joint pain	12 patients
2	Penicillin prophylaxis	13 patients
3	Duration of clinical symptoms	5-10yrs

Table 3: Study of objective of the study

Studied parameters	
ECG Sinus rhythm	18 patients
Atrial Fibrillation	10 patients
Echocardiography	
Ejection fraction	50-60%
Left atrial size(mm)	44-45.27
Mitral valve area (2D)	0.8-1.8cm ²
Tricuspid annulus size	3.2-4.5
TAPSE	0.7-1.3
RVSP	(35-55)+RAP
Mitral valve replacement	15 patients
Mitral valve replacement+ de Vegas tricuspid annuloplasty	8 patients
Mitral valve replacement+ tricuspid ring annuloplasty	5 patients

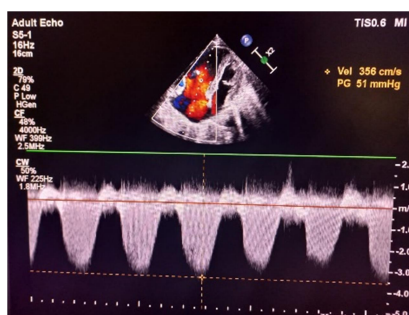


Fig1. 2D ECHO severe TR

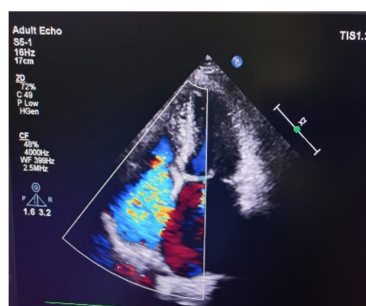
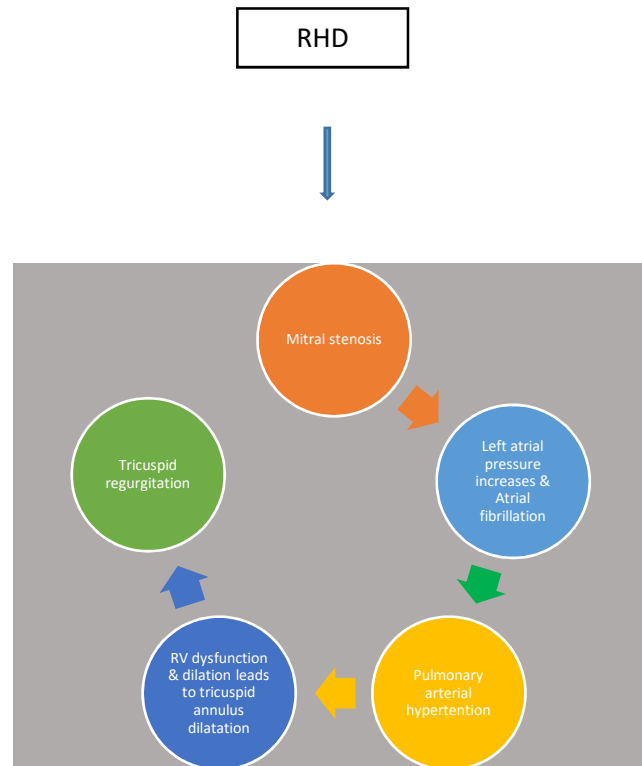


Fig2. Showing colour flow across TV

Discussion

Tricuspid regurgitation is frequently present in patients with MV disease, and more than one-third of the patients with mitral stenosis have at least moderate TR (5, 6). Patients who have severe TR at the time of MV surgery should obviously have their TV repaired at the time of the initial MV surgery (2,3). In patients with less than severe TR, however, TR might progress after surgery if the TV is left untreated. Matsuyama et al. (4) reported significant TR (at least grade 3) on echocardiography performed late after MVR in 37% of the patients with

grade 2 TR before surgery. Tricuspid regurgitation (TR) in patients with mitral valve (MV) disease is associated with poor outcome and predicts poor survival, heart failure, and reduced functional capacity. It is common if left untreated after MV replacement mainly in rheumatic patients, but it is also common in patients with ischemic mitral regurgitation. It is less common, however, in those with degenerative mitral regurgitation. It might appear many years after surgery and might not resolve after correcting the MV lesion. Late TR might be caused by prosthetic valve dysfunction, left heart disease, right ventricular (RV) dysfunction and dilation, persistent pulmonary hypertension, chronic atrial fibrillation, or by organic (mainly rheumatic) tricuspid valve disease. Most commonly, late TR is functional and isolated, secondary to tricuspid annular dilation. Outcome of isolated tricuspid valve surgery is poor, because RV dysfunction has already occurred at that point in many patients. MV surgery should be performed before RV dysfunction, severe TR, or advanced heart failure has occurred. Patients who have severe TR at the time of MV surgery should obviously have their TV repaired at the time of the initial MV surgery (2,3,4). In patients with less than severe TR, however, TR might progress after surgery if the TV is left untreated. Patients with mitral stenosis and moderate or severe TR before MVR are more likely to have class III or IV heart failure (6). Patients with TR after MVR have reduced exercise capacity compared with patients without TR. The pathogenesis of TR in MV disease is complex and multifactorial. Most often TR is functional, secondary to RV dilation and dysfunction and tricuspid annular dilation. Mitral valve disease (usually rheumatic or ischemic) leads to mitral stenosis or regurgitation, which in turn leads to increased left atrial pressure and, if severe enough, to secondary pulmonary hypertension. Long-standing pulmonary hypertension might lead to RV dysfunction and remodeling, which leads to TA dilation, papillary muscle displacement, and tethering of the TV leaflets, leading to TR. Tricuspid regurgitation itself leads to further RV dilation and dysfunction, more TV annular dilation and tethering, and worsening TR. With increasing TR the RV dilates and eventually fails, causing increased RV diastolic pressure and a shift of the interventricular septum toward the LV. Because of ventricular interdependence, this might compress the LV, causing restricted LV filling and increased LV diastolic and pulmonary artery pressure. This phenomenon was named “restriction dilation syndrome” by Antunes and Barlow (10).



Increased left atrial size and pressure might also cause AF, which in turn causes right atrial dilation leading to further tricuspid annular dilation. Atrial fibrillation has been recognized as an important risk factor for the development of TR in patients with MV disease as well as for the persistence or occurrence of TR after MV surgery. In patients with rheumatic valve disease, organic TV involvement might also cause TR. Tricuspid valve leaflet thickening and restriction had been reported in about one-third of the patients with moderate or severe TR after MVR, but the true incidence of organic TV involvement might be higher (7). Tricuspid annular dilation is probably the most important factor in the development of late TR, and it is also the target for intervention. The normal TA is saddle-shaped, with the highest points located in an anteroposterior orientation and the lowest points in a mediolateral orientation. With the development of functional TR, the TA becomes dilated and more planar and circular. Antunes and Barlow (10) suggested that in rheumatic patients direct involvement of the TA by the rheumatic process might weaken the annulus and cause it to dilate. Tricuspid regurgitation was more likely to improve in patients with the following characteristics: 1) younger age; 2) functional (as opposed to organic) TR; 3) smaller MV area; 4) severe pulmonary hypertension; 5) larger resolution of pulmonary hypertension after valvotomy; and 6) no Atrial fibrillation. Because late TR in MV disease is usually due to TA dilation (although rheumatic patients might have organic leaflet disease as well) the results of repeat surgery for isolated late TR are poor and carries significant morbidity and mortality, because, concomitant TV repair with an annuloplasty ring should be performed at the time of the initial MV surgery. Tricuspid valve annuloplasty adds little time and complexity to MV surgery and results in very few complications (8,9,11,12). TV repair is probably better than replacement in patients with non-severe organic TV disease. Tricuspid valve repair with an annuloplasty ring resulted in significantly improved long-term survival, event-free survival, and survival free of recurrent TR compared with De Vega suture annuloplasty. Tricuspid valve ring annuloplasty was also an

independent predictor of long-term survival in that study. All TV repair techniques had an immediate failure rate (grade 3 TR or more) of approximately 14%. Whereas patients who had a ring annuloplasty with a semi-rigid ring (Carpentier-Edwards) had no progression of TR, more than 30% of the patients who had a De Vega procedure. Good results with a modified De Vega repair with pledgeted sutures in patients with non severe organic TV involvement (10). The best evidence for the utility of TV ring annuloplasty during MV surgery and the importance of TA diameter as a criterion for TV repair comes from the study of Dreyfus et al. (8). The TV annulus diameter was measured intraoperatively with a ruler, from the anteroseptal commissure to the anteroposterior commissure. The TV repair with a Carpentier-Edwards ring was performed regardless of the degree of TR, if the TA diameter was ≥ 7 cm (equivalent to 4 cm by echocardiography Groves et al. (1) suggested a threshold of 2.1 cm/m² (equivalent to 3.6 cm for an average person). On the basis of the available information, we believe that prophylactic TV repair should be performed in patients undergoing MVR regardless of TR severity whenever the TV annulus is ≥ 3.5 cm, especially in rheumatic TR. Early TV repair before the occurrence of irreversible RV dysfunction is probably also appropriate in patients with isolated TR secondary to TA dilation without left-sided heart disease. Treatment of patients who develop late isolated TR after MVR like the patient we described is difficult (2, 3). Aggressive antifailure therapy with loop diuretic drugs and spironolactone is the mainstay of therapy and might retard TR progression.

Current guidelines for TV repair and replacement. Both the American College of Cardiology/American Heart Association and the European Society of Cardiology (ESC) guidelines give a class I recommendation for TV repair in patients with severe TR undergoing MV surgery (2,3). The ESC guidelines give a class IIa recommendation for concomitant TV repair in patients with a TA diameter ≥ 40 mm or >21 mm/m² or moderate TR, whereas the American College of Cardiology/American Heart Association gives a more vague, class IIb recommendation for patients with less than severe TR.

Recommendations

Detailed TV assessment, including measurement of the TA diameter, is mandatory in patients with MV disease. A TV annuloplasty with a ring is the best procedure to correct or prevent TR in most cases. It improves survival, prevents late TR and heart failure, and should therefore be performed at the time of the initial MV surgery. The TA diameter is the best guide to select patients for TV repair, and the echocardiographic cutoff should be somewhere around 3.5 cm regardless of TR severity and might be lower for rheumatic patients. Patients with significant TR who are older and have longstanding MV disease, AF, nonsevere pulmonary hypertension, and organic TV disease will benefit more from MV surgery and TV repair.

Conclusion

It was concluded that tricuspid regurgitation was strongly associated with severe mitral stenosis as almost all patients with severe mitral stenosis had tricuspid regurgitation and none of the patients with mild mitral stenosis had tricuspid regurgitation. Those who had severe mitral stenosis with moderate to severe tricuspid regurgitation, mitral valve replacement and de Vega tricuspid annuloplasty or tricuspid ring annuloplasty was performed.

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