



SURGICAL CONSIDERATION REGARDING MANAGEMENT OF TRICUSPID REGURGITATION IN PATIENTS UNDERGOING MITRAL VALVE SURGERY.

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Conflicts of Interest: Nil

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Abstract:

Background: Severity of tricuspid regurgitation (TR) increases after mitral valve surgery in many patients who do not undergo surgical intervention, causing functional limitation. A redo-cardiac surgery for late severe Function Tricuspid regurgitation is uniformly associated with poor prognosis and high early and late mortality.

Concomitant tricuspid repair with mitral valve surgery is recommended and accepted in cases of severe functional TR but its utility for moderate TR remains a dilemma. In such cases, the surgical management is controversial primarily because of uncertainty over the long-term course and clinical sequelae of tricuspid regurgitation after left sided valve surgery; and the diagnostic challenges presented by this very dynamic dysfunction. Thus, this study was done to assess the usefulness of tricuspid annuloplasty concomitant with mitral valve surgery in patients of moderate functional tricuspid regurgitation secondary to rheumatic mitral valve disease.

Methods: A Prospective randomized analytical study was done on 50 patients who were divided into two groups. In 25 patient (group 1) mitral valve replacement and tricuspid annuloplasty was done. In 25 patient (group 2) only mitral valve replacement was done. Preoperative, intraoperative, post-operative and follow up parameters in two group was tabulated in excel sheet. Descriptive statistics was analyzed with Statistical Package for Social Sciences (SPSS) version 21.0. $p < 0.05$ was taken as significant.

Results: At one year follow up, group 1 patients were symptomatically better than group 2. In group 1, no patient had severe tricuspid regurgitation at any follow up. In group 2, 13.04% of patient had severe tricuspid regurgitation at 1 year follow up ($p = 0.019$). One year survival rate was comparable in both the groups ($p > 0.05$).

Conclusion: Moderate functional tricuspid regurgitation with rheumatic mitral valve disease can be managed by concomitant tricuspid repair and mitral valve replacement.

Keywords: tricuspid regurgitation, mitral valve, concomitant.

Introduction

Tricuspid regurgitation (TR) after mitral valve surgery is progressive and cause significant morbidity. In the past, surgeons have shown a fairly conservative approach to tricuspid regurgitation, surgical correction was considered unnecessary when Tricuspid regurgitation was mild, moderate or only detected intermittently. However it has become increasingly apparent that severity of tricuspid regurgitation increases in many patients who do not undergo surgical intervention and become the main cause of functional limitation. A redo-cardiac surgery for late severe Function Tricuspid regurgitation is uniformly associated with poor prognosis and high early and late mortality, varying widely between 11 and 69%.¹

Functional tricuspid regurgitation (FTR) incidence was reported approximately in 30% of patients with both

mitral stenosis and mitral regurgitation.² Late Function Tricuspid regurgitation predicts a 24% lower event-free survival at 15 years, even in the absence of LV dysfunction and pulmonary hypertension, diminished overall long-term survival, and a worse long-term functional status.³ Thus, Surgery for correction of severe Function Tricuspid regurgitation becomes very important towards improved patient survival.⁴ Dreyfus et al advocated pre-emptive repair of tricuspid valve to prevent late progression of TR.⁵

The severity of the TR helps in deciding whether to go for simultaneous tricuspid valve (TV) surgery or not. Severe TR has class I recommendation for TV surgery,⁶ but evidences for surgical intervention in patient whose Tricuspid regurgitation is less than severe is debatable. Up to 50% of mild tricuspid regurgitation cases that are not corrected at the time of surgery become severe after 5 years,⁷ whereas in

other 50% cases, the untreated mild tricuspid regurgitation decreases or does not increase.⁸ The major therapeutic dilemma lies with moderate Tricuspid regurgitation and whether it should be corrected along with the correction of left sided valvular pathology or not.

Thus, the study was conducted to analyze the outcome of a strategy of conservative and one stage surgical management of moderate TR in patient undergoing mitral valve surgery.

Materials and Methods:

A prospective randomized case control study was conducted between April 2012 and May 2015, where a total of 50 subjects with rheumatic mitral valve disease and concomitant moderate functional tricuspid regurgitation were included. Patients with organic tricuspid valve disease or concomitant aortic valve disease; patients with abnormal coagulation mechanism with recent surgery or bleeding history; and patients with failure to follow-up; were excluded from the study.

The 50 subjects were randomised by computer generated random numbers into two groups of 25 in each, according to the treatment:

Group 1: Study group, 25 patients with moderate tricuspid regurgitation in which tricuspid repair with ring annuloplasty was done.

Group 2: Control group, 25 patients with moderate tricuspid regurgitation which was left untreated at mitral valve surgery.

This study was approved by the Ethics Committee of the Hospital. A Signed written informed consents was obtained from all participants.

Pre-, intra- and postoperative data were prospectively recorded for every patient and analyzed.

Methods:

The same surgery and nursing team completed all the studies according to standard medical procedures. Operations were performed through median sternotomies with cardiopulmonary bypass. Myocardial protection was achieved with antegrade cold blood hyperkalemic root cardioplegia. Mitral valve was approached in group 1 through right atriotomy and atrial septotomy approach and in group 2 through left atrial approach. Mitral valve replacement was done in all patients using

interrupted pledgetted suture technique with appropriately sized bileaflet mechanical mitral prosthesis. Following closure of atrial septotomy in group 1 tricuspid valve was examined and sized. In all patients (group1) rigid Edwards MC3 tricuspid ring annuloplasty was performed to correct TR by interrupted suture technique. The tricuspid competence was checked with saline instillation into the right ventricle.

Data collection:

Patients thus recruited in the study were evaluated thoroughly. Details regarding demographic characteristics, symptoms with duration, past history of drug intake (digoxin, diuretics, ACE inhibitors), history of previous interventions (BMV, CMV) were recorded. Signs of valvular heart disease, congestive cardiac failure, pulmonary artery hypertension, pulmonary venous hypertension were noted. NYHA class was determined for each patient.

After preliminary evaluation, investigations comprising of ECG, Chest X ray, transthoracic 2 D Echocardiography and Coronary angiography were done in patients with age more than 40 years.

Right Ventricular systolic function was assessed with Tricuspid annulus plane systolic Excursion (TAPSE). TAPSE < 16 mm indicated RV systolic dysfunction. Pulmonary artery pressure (PAP) was calculated by TR jet method.⁹

Details of intraoperative parameters viz. CPB duration, Aortic cross clamp duration, were analysed. Similarly post-operative parameter namely, inotrope requirement, complication, blood and blood products requirement, ECG changes, Chest X ray, ICU and hospital stay duration was analysed.

Follow Up:

Follow up was done at one week, one month, three month, six month and one year after discharge. Patient symptomatology, NYHA functional class, RA/RV/IVC Size, Progression of TR, TAPSE, Systolic PA pressure were followed up after surgery. Data collection was done from patient attending CTVS OPD, OPD file records.

Statistical analysis:

Categorical variables were presented in number and percentage (%) and continuous variables were presented as mean \pm SD and median. Quantitative variables were compared using Unpaired t-test (when

the data sets were not normally distributed) between the two groups and Paired T test (for non parametric data). Qualitative variables were compared using Chi-Square test. Kaplan meier survival curve with log rank test was used to compare survival of two groups.

A p value of <0.05 was considered statistically significant.

The data was entered in MS EXCEL spreadsheet and analysis was done using Statistical Package for Social

Sciences (SPSS) version 21.0 (IBM SPSS Statistics, Armonk, USA).

Results

Fifty patients were evaluated for the study: Twenty five patients in group 1(study group) and twenty five patients in group 2 (control group). The demographic data has been shown in Table 1.

Table 1: Demographic characteristics:

Patient variables	Group 1 (n=25)	Group 2 (n=25)	P-value
Mean Age (in years)	44.84 ± 13.74	51.56 ± 17.16	0.133
Gender n (%)			0.208
Males	5(20%)	9(36%)	
Females	20(80%)	16(64%)	

Most commonly patient was admitted with complaint of exertional dyspnoea in both the groups associated with cough, chest pain and palpitation in few patients. Congestive heart failure (CHF) was a common finding in both the groups. (Table 2)

Table 2: Clinical symptoms and Signs

Symptoms	Group 1 (n=25)	Group 2 (n=25)	P-value
Dyspnoea n(%)	22 (88%)	25 (100%)	0.235
Palpitation	8(32%)	0(0%)	0.004*
Chest pain	3(12%)	2(8%)	1.000
Cough	5(20%)	6(24%)	0.733
Congestive heart failure	15(60%)	19(76%)	0.225

*P<0.05: Significant

Preoperatively, Transthoracic 2D echocardiographic examination findings has been shown in Table 3. There was a significant difference in the jet density and contour among the two groups.

Table 3: Transthoracic 2 d echocardiography: Preoperative parameters

Severity assessment parameters		Group 1(n=25)	Group 2(n=25)	P value
RA/RV/IVC Size	Dilated	9 (36.00%)	8 (32.00%)	0.765
	Normal	16 (64.00%)	17 (68.00%)	
Jet area – central jet(cm ²)	Mild	1 (4.00%)	0 (0.00%)	1
	Moderate	24 (96.00%)	25 (100.00%)	
Vena contracta	Mild/Moderate	25 (100.00%)	25 (100.00%)	1
	Severe	0(0%)	0(0%)	
Jet density and contour (cont. wave)	Dense/incomplete	7 (28.00%)	0 (0.00%)	0.009*
	Dense/parabolic	17 (68.00%)	25 (100.00%)	
	Dense/variable	1 (4.00%)	0 (0.00%)	
Hepatic vein flow	Systolic Blunting	4 (16.00%)	2 (8.00%)	0.158
	Systolic dominance	21 (84.00%)	20 (80.00%)	
	Systolic reversal	0 (0.00%)	3 (12.00%)	

*P<0.05: Significant

Preoperatively, the median value of TAPSE in both group was 15mm; systolic pressure in group 1 was 48.24 ± 8.15 mmHg and in group 2 it was 47.04 ± 8.6 mmHg (p =0.615)

Intraoperative characteristics:

The most common rhythm after weaning off CPB in the intraoperative period was normal sinus rhythm 56% in group 1 and 60% in group 2, p =0.198), followed by atrial fibrillation (32% in both group). External pacemaker support was required in 9 patients (36%) in group 1 and 8 patients (32%) in group 2 (p =0.765). Intraoperative

CPB duration in group 1 and group 2 were 92.56 ± 22.24 min. and 89.12 ± 23.26 respectively ($p = 0.595$) There difference in the aortic cross clamp and CPB duration was significantly more in Group 1 as compared to Group 2. ($p < 0.05$) It is shown in Table 4.

Table 4: Intraoperative characteristics

Intra-operative parameters		Group 1(n=25)	Group 2(n=25)	P- value
Intraoperative rhythm (weaning off CPB)	AF	32%	32%	0.198
	VT-D/C SHOCK	0%	8%	
	NSR	56%	60%	
	VF	12%	0%	
Intraoperative external pacemaker requirement	Pacemaker requirement.	9 (36.00%)	8 (32.00%)	0.765
Aortic cross clamp and CPB duration	Aortic cross clamp duration			0.145
	Median	73	54	
	Min-Max	41-93	32-105	
	Inter quartile Range	56.250 - 84.250	48 - 76.250	
	CPB duration			0.595
	Mean \pm Stdev	92.56 ± 22.24	89.12 ± 23.26	
	Median	91	90	
Difference between aortic cross clamp and CPB duration	Mean \pm Stdev	30.04 ± 14.44	19.2 ± 10.06	0.004*
	Median	30	17	
	Min-Max	10-74	5-45	
	Inter quartile Range	18.000 - 39.000	12.000 - 21.500	

* $P < 0.05$: Significant; AF: Atrial fibrillation, VT: Ventricular tachycardia, D/C: Direct cardioversion, NSR: Normal sinus rhythm, CPB: Cardio pulmonary bypass.

Postoperative characteristics:

16 patient (64%) in group 1 and 19 patient (76%) in group2 had no complication in the post operative period ($p = 0.274$) Thus more patient in group 2 had complication. The most common complication in both group was tachyarrhythmia, it occurred in 36% of patient in group 1 and 16 % of patient in group 2.

There was no death in group 1 but in group 2 there were two deaths in immediate post-operative period. One patient had cardiac tamponade on first postoperative day for which re-exploration was done and patient expired on 6th postoperative day. Other patient died of refractory tachyarrhythmia on 9th post-operative day. In fact, both 30-day mortality and one-year mortality was more in group 2 than in group 1. Other post-operative parameters and requirements have been shown in Table 5.

Table 5: Post-operative characteristics

Post operative characteristics		Group 1(n=25)	Group 2(n=25)	P-value
Complications	Congestive heart failure	0%	4%	0.274
	Arrhythmias(AF)	36%	16%	
	Pleural effusion	0%	4%	
Inotrope requirement	Mild	13 (52.00%)	21 (84.00%)	0.016*
	Moderate	12 (48.00%)	3 (12.00%)	
	High	0 (0.00%)	1 (4.00%)	
Pacemaker requirement		6 (24.00%)	10 (40.00%)	0.765
Post operative total drainage (ml)	Median	210	230	0.93
	Min-Max	90-500	70-850	
	Inter quartile Range	170 - 275	157.5 - 317.5	
Blood/ Blood product	PRBC(median value)	1.5	2	0.334
	Platelet(median value)	3	2	0.246
	FFP(median value)	2	2	0.52
Post operative Mechanical				0.028*

ventilation duration(in Hr)	Median	12	14	
	Min-Max	6-20	4-216	
	Inter quartile Range	8 - 14	10 - 20	
	Post operative ICU stay (days)	4	5	0.636
	Post operative hospital stay (days)	7	7	0.494

*P<0.05: Significant, PRBC: packed Red blood cells, FFP: Fresh frozen plasma.

Follow up:

During the follow-up, at one month and 6 months, there were significantly more number of patients in NYHA class I in Group 1 as compared to Group 2. (p<0.05) The follow-up NYHA classification status of the patients is shown in Table 6.

Table 6: NYHA functional class

	Pre operative	one week	one month	three months	6 months	one year
Group 1						
I	0 (0.00%)	4 (16.00%)	8 (32.00%)	8 (32.00%)	19 (79.17%)	16 (66.67%)
II	2 (8.00%)	17 (68.00%)	14 (56.00%)	14 (56.00%)	5 (20.83%)	5 (20.83%)
III	14 (56.00%)	4 (16.00%)	3 (12.00%)	2 (8.00%)	0 (0.00%)	3 (12.50%)
IV	9 (36.00%)	0 (0.00%)	0 (0.00%)	1 (4.00%)	0 (0.00%)	0 (0.00%)
Group 2						
I	0 (0.00%)	4 (17.39%)	1 (4.35%)	6 (26.09%)	9 (39.13%)	13 (56.52%)
II	2 (8.00%)	14 (60.87%)	15 (65.22%)	12 (52.17%)	9 (39.13%)	5 (21.74%)
III	16 (64.00%)	5 (21.74%)	7 (30.43%)	5 (21.74%)	1 (4.35%)	1 (4.35%)
IV	7 (28.00%)	0 (0.00%)	0 (0.00%)	0 (0.00%)	4 (17.39%)	4 (17.39%)
P value	0.826	0.853	0.03*	0.449	0.021*	0.152

*P<0.05: Significant

The follow-up ECG findings were comparable among the two groups as shown in Table 7.

Table 7: Follow-up ECG findings

	Pre operative	Post operative	one week	one month	three months	6 months	one year
Group 1							
AF	17 (68.00%)	21 (84.00%)	19 (76.00%)	18 (72.00%)	17 (70.83%)	12 (50.00%)	15 (62.50%)
Group 2							
AF	16 (64.00%)	18 (72.00%)	16 (69.57%)	11 (47.83%)	11 (47.83%)	13 (56.52%)	12 (52.17%)
P value	0.765	0.496	0.616	0.087	0.108	0.654	0.474

At follow-up, TAPSE in group 1 decreased from 15.12 ± 1.81 mm to 14.48 ± 1.23 mm till one month follow up suggestive of improved RV function there after it increased to 15 ± 1.29 mm till one year follow up . Whereas in group 2 TAPSE had increased slightly from 14.72 ± 1.46 mm to 14.91 ± 0.95 mm during one year follow up suggestive of deteriorating RV function. Difference in TAPSE had remained insignificant throughout the study as shown in Table 8.

Table 8: Follow-up TAPSE

RV DYSFUNCTION (TAPSE in MM)	Pre operative	Post operative	1 week	1 month	3 months	6 months	1 year
Group 1							
Mean \pm Stdev	15.12 ± 1.81	14.64 ± 1.66	14.64 ± 1.5	14.48 ± 1.23	14.6 ± 1.22	15 ± 1.14	15 ± 1.29
Median	15	15	15	14	15	15	15
Min-Max	12-18	12-18	12-18	13-18	12-17	13-18	12-18
Inter quartile Range	14 - 16	14 - 16	14 - 15.250	13.750 - 15	14 - 15.250	14 - 15.5	14 - 16
Group 2							
Mean \pm Stdev	14.72 ± 1.46	14.92 ± 1.38	14.87 ± 1.29	14.96 ± 1.22	15.04 ± 1.07	15 ± 0.85	14.91 ± 0.95
Median	15	15	15	15	15	15	15
Min-Max	13-17	13-17	13-17	13-17	13-17	14-16	13-16
Inter quartile Range	13 - 16	14 - 16	14 - 16	14 - 16	14 - 16	14 - 16	14 - 16
P value	0.375	0.606	0.604	0.173	0.245	0.867	0.876

Systolic pulmonary artery pressure had been progressively decreasing in group 1 during one year follow up. It decreased from 48.24 mmHg preoperatively to 40.5 mm Hg at one year follow up. In group 2 systolic pulmonary artery pressure decreased from 47.04 mm Hg preoperatively to 40.91 mmHg at three months follow up but there after there was increasing trend till one year follow up. Thus there is no significant impact on RV function and pulmonary artery pressure immediately, but it appears that tricuspid annuloplasty has favourable effect on RV function. (Table 9)

Table 9: Follow up Systolic Pulmonary artery pressure

SYSTOLIC PA PRESSURE (in mmHg)	Pre operative	Post operative	one week	one month	3 months	6 months	1 year
Group 1							
Mean ± Stdev	48.24 ± 8.15	47.68 ± 8.06	46.64 ± 7.34	45.68 ± 7.56	44.56 ± 7.78	42.92 ± 7.51	40.5 ± 7.42
Median	48	48	46	46	46	43	40
Min-Max	35-61	36-66	34-58	30-58	30-56	30-56	30-54
Inter quartile Range	40.750 – 56	40 – 53	40 – 52	40 – 52	37.5 – 52	37 – 50	32 – 46
Group 2							
Mean ± Stdev	47.04 ± 8.6	46.24 ± 8.07	44.96 ± 7.06	41.04 ± 7.05	40.91 ± 7.54	41.39 ± 7.47	42.17 ± 7.65
Median	48	50	46	40	40	40	40
Min-Max	28-58	28-60	30-56	24-56	24-58	24-58	30-58
Inter quartile Range	40 – 55.250	40 – 52	40 – 50	40 – 43.5	36.5 – 45	38 – 46	36.5 – 46
P value	0.615	0.653	0.423	0.047*	0.138	0.38	0.45

*P<0.05: Significant, PA:Pulmonary artery.

In group 1 all patient had mild tricuspid regurgitation up to 1 month, there after mild tricuspid regurgitation was present in 96% of the patient at 3 month and 91.67% of the patient at 6 month and one year follow up. In group 2 no patient had mild tricuspid regurgitation up to 1 week follow up, thereafter 26.09%, 30.43%, 47.83% and 52.17% had mild tricuspid regurgitation at 1month, 3 month, 6 month and 1 year follow up respectively. No patient in group 1 had moderate tricuspid regurgitation up to one month follow up, there after moderate tricuspid regurgitation was present in 4% patient at 3 month follow up and 8.33% at 6 month and one year follow up. In group 2 72%, 73.91%, 65.22%, 60.87%, 39.13% and 26.09% of patient had moderate tricuspid regurgitation at immediate postoperative , 1 week, 1 month, 3 month, 6 month and 1 year period respectively. In group 1 no patient had severe tricuspid regurgitation at any follow up. In group 2 at immediate postoperative , 1 week, 1 month, 3 month, 6 month and 1 year follow up 28%, 26.09%, 8.7%, 8.7%, 8.7%, and 13.04% of patient respectively had severe tricuspid regurgitation. The difference in the severity of tricuspid regurgitation is significant at each follow up (p<0.05). (Table 10)

Table 10: Comparison of Severity of TR on Follow-up

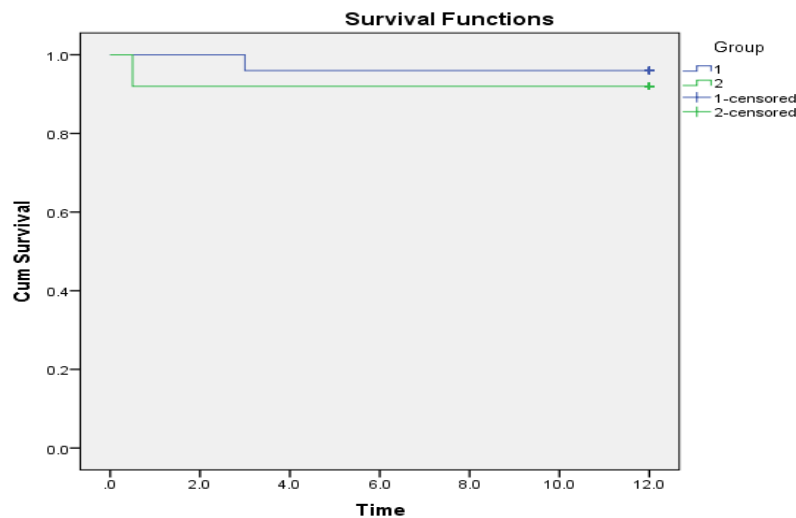
	Pre operative	Post operative	one week	one month	three months	6 months	one year
Group 1							
Mild	0 (0.00%)	25 (100.00%)	25 (100.00%)	25 (100.00%)	24 (96.00%)	22 (91.67%)	22 (91.67%)
Moderate	25 (100.00%)	0 (0.00%)	0 (0.00%)	0 (0.00%)	1 (4.00%)	2 (8.33%)	2 (8.33%)
Severe	0 (0.00%)	0 (0.00%)	0 (0.00%)	0 (0.00%)	0 (0.00%)	0 (0.00%)	0 (0.00%)
Trace	0 (0.00%)	0 (0.00%)	0 (0.00%)	0 (0.00%)	0 (0.00%)	0 (0.00%)	0 (0.00%)
Group 2							
Mild	0 (0.00%)	0 (0.00%)	0 (0.00%)	6 (26.09%)	7 (30.43%)	11 (47.83%)	12 (52.17%)
Moderate	25 (100.00%)	18 (72.00%)	17 (73.91%)	15 (65.22%)	14 (60.87%)	9 (39.13%)	6 (26.09%)
Severe	0 (0.00%)	7 (28.00%)	6 (26.09%)	2 (8.70%)	2 (8.70%)	2 (8.70%)	3 (13.04%)
Trace	0 (0.00%)	0 (0.00%)	0 (0.00%)	0 (0.00%)	0 (0.00%)	1 (4.35%)	2 (8.70%)
P value	-	<.0005*	<.0005*	<.0005*	<.0005*	0.011*	0.019*

*P<0.05: Significant

One year survival in group 1 was 96% in group1 and 92% in group 2. (p = 0.545) The survival estimates at different follow-up times was comparable among the two groups. (p>0.05) It has been shown in Table 11 and Figure 1.

Table 11: Survival follow-up

Group	Mean				Log Rank (Mantel-Cox)	One week OS	One month OS	Three months	six months	One year OS
	Estimate	Std. Error	95% Confidence Interval							
			Lower Bound	Upper Bound						
1	11.640	.353	10.949	12.331	0.545	100%	100%	96%	96%	96%
2	11.080	.624	9.857	12.303		100%	92%	92%	92%	92%
Overall	11.360	.361	10.653	12.067						

**Figure 1:** Kaplan Meier survival curve.

Discussion

We found that concomitant repair of TR with mitral valve surgery offers a better well being to the patients immediately and over a follow up period. The NYHA class and ECG findings were better in patients treated with concomitant TR repair.

The age and sex distribution between two groups were comparable, with Female sex predominating in both the groups. This may have important association with regard to progression of functional tricuspid regurgitation as shown by Vargas Abello *et al*.¹⁰

We included patients with only moderate functional tricuspid regurgitation. Preoperatively, all parameters used for assessing severity of functional tricuspid regurgitation were comparable among the two groups except for preoperative jet density and contour ($p=0.009$).

During weaning of CPB, although heart rate in group 1 was more controlled than group 2 ($p=0.019$) with 8% patient in group 2 requiring DC cardioversion for ventricular tachycardia, and total frequency of tachyarrhythmia was slightly more in group 1 than group 2. Intraoperative pacemaker requirement and

inotrope requirement did not differ significantly between the two groups.

Postoperative atrioventricular block is always an important concern to cardiac surgeons. Performing tricuspid annuloplasty permits avoiding the area of atrioventricular conduction tissue. Zhu *et al* reported that incidence rate of pacemaker requirement in the tricuspid repair group ranged from 1.6 to 5.4%, and no statistical significance was demonstrated between the two groups.¹¹

Aortic cross clamp duration and cardiopulmonary bypass duration was more in group 1 than group 2 but the difference was not significant (ACC – 0.145, CPB – 0.595). Additional tricuspid annuloplasty will certainly increase the total surgical time, as well as the CPB and aortic clamping times.^{12,13} In contrast, one study found that there were no statistically significant differences in surgical times between a tricuspid repair group and non-repair group.¹⁴

The mean aortic cross-clamping times were 57– 83 min without associated tricuspid repair and 62– 100 min with tricuspid repair, and CPB times without and with repair were 82– 124 and 90–174 min, respectively. Our findings were in line with other

studies,^{15,16} who documented that the mean difference of CPB time between the two groups basically reflected the increasing time cost, which was about 20 min (8–50 min). Thus, tricuspid annuloplasty can be undertaken quickly enough without significantly prolonging the myocardial ischemia time.

In our study, frequency of atrial fibrillation was more in group 1 (36%) than group 2 (12%) as was seen by Chikwe *et al*.¹⁷ While there was no death in group 1, there were two post operative death in group 2. One expired due to mediastinal bleeding and other due to refractory tachyarrhythmia. Perioperative mortality is the main quality marker analysed in adult valvular surgery. Dreyfus *et al*.⁵ reported an in-hospital mortality rate of 0.6% in the tricuspid valve plasty (TVP) group and 1.8% in the non-TVP group ($P = 0.36$). Smid *et al*.¹⁸ also showed slightly less perioperative deaths with a concomitant TV procedure, although not statistically significant ($P = 0.55$). Calafiore *et al*.¹⁹ in a study on 298 patients, of whom 131 patients received isolated left-sided valve surgery and 167 received concurrent TV repair; found more atrial fibrillation (AF), but less 30-day mortality in the repair group (8.4 vs 4.2%, $P = 0.13$). These results advocate the advantages regarding perioperative mortality in the TVP group, which reflects the potential benefit to restore right ventricular function when concurrently correcting TR.

In this study, post-operative pacemaker requirement were less in group 1 than group 2 ($p = 0.225$) In contrast, another study found that Tricuspid valve repair may be associated with a higher rate of pacemaker requirement but it is difficult to ascribe this to the tricuspid valve repair with certainty, as many patients have also had multivalvular surgery and atrial fibrillation ablation, which also predispose to heart block.²⁰

In this study, inotrope requirement in group 1 was significantly less than group 2 ($p = 0.016$). It might be a significant morbidity of not doing tricuspid repair. This finding has rarely been documented in the literature. Mediastinal drainage and blood product requirement perioperatively didn't differ significantly between the two group in this study. This again, suggests that additional tricuspid repair does not increase the probability of post-operative bleeding.

Results also suggests that duration of mechanical ventilation in group 1 is significantly less than group 2 ($p=0.028$). This may have better impact on

postoperative pulmonary rehabilitation. These aspects of concomitant tricuspid repair with mitral valve replacement have been discussed inadequately in literature. ICU stay and hospital stay too does not appear to be affected by tricuspid repair. ($p>0.05$) This is consistent with the reported results of another study.²¹

Data on the post-operative course and clinical sequelae of TR are conflicting.^{16,5} The reason may be, in part, that most previous studies reported on groups with heterogeneous mitral disease etiology or surgical management. In a study that focused exclusively on patients with Rheumatic disease who were undergoing concomitant tricuspid repair with mitral valve surgery, Pradhan *et al*.²² reported that at three months follow up, 20% patients were with NYHA II symptoms in non tricuspid repair group and the rest were with NYHA I symptoms. However, in our study, there were more number of patient in NYHA I/II in group 1 than group 2 at 1 month ($p = 0.03$) and 6 month ($p = 0.021$).

Thus, tricuspid annuloplasty improves symptomatic outcome in short term but this needs further follow up. Change in prevalence of atrial fibrillation were unremarkable in both the group during follow up. There were insignificant difference in frequency of atrial fibrillation between the two group at each follow up. Atrial fibrillation was managed pharmacologically and ablation therapy was not required. This suggests that adding tricuspid repair to mitral valve surgery does not appear to influence the occurrence of atrial fibrillation. Atrial fibrillation and Functional TR relationship is well established following mitral valve replacement.³ Occurrence of AF may result in elevation of left atrial pressure, and also induce enlargement of the right atrium and tricuspid annular dilatation, all potentially important factors in causing worsening of TR.²³

Rhythm restoration with MVR simultaneously prevents FTR progression. In study by Ariyoshi *et al*.²¹ only AF was identified by multivariate analysis as a significant predictor of late phase worsening of TR in the non tricuspid repair group. They observed improvement of TR in 95.8% of patients without AF, a value twofold greater than in patients with AF. Such a mechanism may in addition lead to significantly larger left atrium diameter in patients with AF than in those without AF.

Our study reveals that higher pre-operative pulmonary artery pressure, and reduction of

pulmonary artery systolic pressure after surgery, points towards a preserved right ventricular function, and therefore a higher likelihood of regression of tricuspid regurgitation in this population. But despite the impact of mitral valve replacement on tricuspid regurgitation, Group 1 patients were found to have a greater resolution with significantly more patients with mild TR as compared to Group 2.

The strong correlation between TR and RV dysfunction has clinical implications beyond surgical technique and focuses attention on the indication for and timing of surgery, postoperative management, and prognosis. It suggests that earlier surgery should be performed before RV function deteriorates. Surgeons must also develop greater awareness of the implications of RV dysfunction in postoperative management. TV repair might be of little help if the RV has limited capacity to take advantage of a competent valve. Such a scenario offers an explanation for TR as a predictor of early and late mortality in patients undergoing operation for left-sided heart disease.²⁴

Although preoperative pulmonary hypertension is one of the important causes of secondary TR, it has been reported that it does not serve as a predictor of worsening of TR in the late phase after mitral valve surgery. Pulmonary hypertension was also not identified as a predictor of worsening of TR in the late phase by Ariyoshi *et al*²¹ in the non tricuspid annuloplasty group. This may have resulted from the fact that PAP is generally anticipated to decrease after mitral valve surgery. However, we assume that the involvement of the right ventricle may also play a role.

Reduction in right ventricular function may also aggravate TR by inducing tricuspid annular dilatation and right ventricular remodeling through diminished pumping ability it may also reduce the severity of pulmonary hypertension in some cases, and contribute to the complex role played by preoperative pulmonary hypertension as a predictor of worsening of TR.²⁵

In this study group 2 also showed a statistically significant decrease in TR, the most probable cause was resolution of the left heart lesion brought about a subsequent reduction in pulmonary hypertension. About 13% of the patients in the group 2 showed TR progression to severe category combined with dilatation and decreased right ventricle function and the percentage may increase on further follow up.

Thus concomitant tricuspid annuloplasty not only decreases the progression of tricuspid regurgitation but also favorably affects RV reverse remodelling.

The 30 day mortality was comparable among the two groups as seen by Pradhan *et al*.²² At the end of one year Tricuspid annuloplasty was not found to be associated with significantly increased mortality in multivariate analysis ($p = 1$). Estimated 1 year survival was also comparable among the two groups (11.64 month vs 11.080 month, $p > 0.05$).

This study reveals that a strategy of routine repair of moderate TR at the time of MV replacement in patients with rheumatic disease reduces the severity of TR without incremental risk. Importantly, concomitant tricuspid valve repair achieved superior freedom from TR, improved RV function, and better survival compared with MV repair only up to one year follow up. Correcting the mitral valve lesion is insufficient in many patients, in whom progression of tricuspid regurgitation postoperatively is associated with decreased survival and functional status.

Limitations of the study

The main limitation of the study was that the follow up period was short compared to the natural history of progression of tricuspid regurgitation, and the repair result only reflects on the early result of ring annuloplasty repair. Another important drawback is the relatively small sample size. Assessment of tricuspid regurgitation using echocardiography is problematic because this method still depends upon preload and afterload conditions at the time of measurement.

Conclusion

Tricuspid annuloplasty is a safe, reproducible and reliable treatment for moderate functional tricuspid regurgitation, and should be performed at the time of mitral valve surgery in any patient with moderate tricuspid regurgitation.

Acknowledgement: We thank Dr ketan garg for assistance in medical writing and editing.

References

1. Kwak JJ, Kim YJ, Kim MK, Kim HK, Park JS, Kim KH, *et al*. Development of tricuspid regurgitation late after left sided valve surgery : a single centre experience with long term echocardiographic examination . *Am Heart J*. 2008;155(4):732-7.

2. Matsunaga A, Duran CM. Progression of tricuspid regurgitation after repaired functional ischemic mitral regurgitation. *Circulation*. 2005;112(9 Suppl):I453-7.
3. Song H, Kim MJ, Chung CH, Choo SJ, Song MG, Song JM, et al. Factors associated with development of late significant tricuspid regurgitation after successful left-sided valve surgery. *Heart*. 2009;95:931-6.
4. Lee JW, Song JM, Park JP, Lee JW, Kang DH, Song JK. Long-term prognosis of isolated significant tricuspid regurgitation. *Circ J*. 2010;74:375-80.
5. Dreyfus GD, Corbi PJ, Chan KM, Bahrami T. Secondary tricuspid regurgitation or dilatation: which should be the criteria for surgical repair?. *Ann Thorac Surg*. 2005;79:127-132.
6. Nishimura RA, Otto CM, Bonow RO, Carabello BA, Erwin JP 3rd, Guyton RA, et al. 2014 AHA/ACC Guideline for the management of Patients With Valvular Heart Disease. *Journal of the American College of Cardiology*. 2014. doi:10.1016/j.jacc.2014.02.536.p100.
7. Shiran A, Sagie A. Tricuspid regurgitation in mitral valve disease: incidence, prognostic implications, mechanism, and management. *J Am Coll Cardiol*. 2009;53(5):401-408.
8. Colombo T, Russo C, Ciliberto GR, Lanfranconi M, Brushi G, Agati S, et al. Tricuspid regurgitation secondary to mitral valve disease: tricuspid annulus function as a guide to tricuspid valve repair. *Cardiovascular surgery*. 2001;9(4):369-377.
9. Silver MD, Lam JH, Ranganathan N, Wigle ED. Morphology of the human tricuspid valve. *Circulation*. 1971;43:333-348.
10. Abello VLM, Klein AL, Marwick TH. Understanding right ventricular dysfunction and functional tricuspid regurgitation accompanying mitral valve disease. *J Thorac Cardiovasc Surg*. 2013;145:1234-41.
11. Zhu TY, Wang JG and Meng X. Does concomitant tricuspid annuloplasty increase perioperative mortality and morbidity when correcting left-sided valve disease? *Interact Cardiovasc Thorac Surg*. 2015;20:114-119.
12. Benedetto U, Melina G, Angeloni E, Refice S, Roscitano A, Comito C, et al. Prophylactic tricuspid annuloplasty in patients with dilated tricuspid annulus undergoing mitral valve surgery. *J Thorac Cardiovasc Surg*. 2012;143(3):632-638.
13. Chan V, Burwash IG, Lam BK, Auyeung T, Tran A, Mesana TG, et al. Clinical and echocardiographic impact of functional tricuspid regurgitation repair at the time of mitral valve replacement. *Ann Thorac Surg*. 2009;88:1209-15.
14. Calafiore AM, Gallina S, Iaco AL, Contini M, Bivona A, Gagliardi M, et al. Mitral valve surgery for functional mitral regurgitation: should moderate or more tricuspid regurgitation be treated? A propensity score analysis. *Ann Thorac Surg*. 2009;87:698-703.
15. Dunning J, Prendergast B, Mackway-Jones K. Towards evidence-based medicine in cardiothoracic surgery: best BETS. *Interact CardioVasc Thorac Surg*. 2003;2:405-9.
16. Filsoofi F, Salzberg SP, Coutu M, Adams DH. A three-dimensional ring annuloplasty for the treatment of tricuspid regurgitation. *Ann Thorac Surg*. 2006;81:2273-8.
17. Chikwe J, Itagaki S, Anyanwu A, Adams DH. Impact of concomitant tricuspid annuloplasty on tricuspid regurgitation, right ventricular function, and pulmonary artery hypertension after repair of mitral valve prolapse. *J Am Coll Cardiol*. 2015;65: 1931-8.
18. Smid M, Cech J, Rokyta R, Roucka P, Hajek T. Mild to moderate functional tricuspid regurgitation: retrospective comparison of surgical and conservative treatment. *Cardiol Res Pract*. 2010;2:143878. doi:10.4061/2010/143878
19. Calafiore AM, Iaco AL, Romeo A, Scandura S, Meduri R, Varone E, et al. Echocardiographic-based treatment of functional tricuspid regurgitation. *J Thorac Cardiovasc Surg*. 2011;142:308-13.
20. Anyanwu AC, Chikwe J, Adams DH. Tricuspid Valve Repair for Treatment and Prevention of Secondary Tricuspid Regurgitation in Patients Undergoing Mitral Valve Surgery. *Curr Cardiol Rep*. 2008;10:110-117.
21. Ariyoshi T, Hashizume K, Taniguchi S, Miura T, Matsukuma S, Nakaji S, et al. Which type of secondary tricuspid regurgitation accompanying mitral valve disease should be surgically treated? *Ann Thorac Cardiovasc Surg*. 2013;19:428-34.
22. Pradhan S, Gautam NC, Singh YM, Shakya S, Timala RB, Sharma J, et al. Tricuspid valve repair—DeVega's tricuspid annuloplasty in moderate secondary tricuspid regurgitation. *Kathmandu Univ Med J*. 2011; 9:64-8.
23. Henry WL, Morganroth J, Pearlman AS, Clark CE, Redwood DR, Itscoitz SB, et al. Relation between echocardiographically determined left atrial size and atrial fibrillation. *Circulation*. 1976;53:273-9.
24. Braunwald NS, Ross J, Jr, Morrow AG: Conservative management of tricuspid regurgitation in patients undergoing mitral valve replacement. *Circulation*. 1967;35(4 Suppl):I63-I69.
25. Kwon DA, Park JS, Chang HJ. Prediction of outcome in patients undergoing surgery for severe tricuspid regurgitation following mitral valve surgery and role of tricuspid annular systolic velocity. *Am J Cardiol*. 2006; 98: 659-61.