

Tricuspid Valve Repair: Devega's Tricuspid Annuloplasty in Moderate Secondary Tricuspid Regurgitation

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Citation

Pradhan S, Gautam NC, Singh YM, Shakya S, Timala RB, Sharma J, Koirala B. Tricuspid Valve Repair: Devega's Tricuspid Annuloplasty in Moderate Secondary Tricuspid Regurgitation. *Kathmandu Univ Med J* 2011;33(1)64-8.

ABSTRACT

Background

Moderate secondary tricuspid incompetence has variable natural history if left unattended during mitral valve surgery. Recent data suggest progression of the secondary tricuspid incompetence over time. Secondary moderate tricuspid regurgitation in rheumatic mitral valve disease may regress after mitral valve surgery without direct intervention.

Objectives:

The present retrospective comparative hospital based study was done to assess early result of DeVega tricuspid valve annuloplasty amongst those with moderate tricuspid regurgitation due to rheumatic mitral valve disease.

Methods:

Group I (mitral valve replacement with tricuspid repair) and Group II (mitral valve replacement only) were compared regarding functional class, heart rate, rhythm, cardiac dimensions, function and valve pathology. The two groups were followed up at three months post-operatively and evaluated for their functional class and echocardiography variables. The data was analyzed with SPSS 16.0

Results:

There were 43 patients who underwent mitral valve replacement with moderate tricuspid regurgitation. Twenty three underwent mitral valve replacement with tricuspid repair group (Group 1). Most of the patients were women (28/43). The mean age was 31.4 + 14.8 and 25.13 + 9.4 years. Group I had 21(91.3%) and Group II had 17 (85%) in NYHA class III & IV. The pre-operative echocardiographic cardiac left ventricular and left atrial dimensions, left ventricular function and valve lesions were statistically similar for both groups, except PASP was higher amongst tricuspid repair (Group 1: 38.60 + 12.75mmHg, Group 2: 61.52 + 19.76mmHg; p= <0.05). At three month's review after surgery, four patients were in NYHA II amongst those without tricuspid repair (Group II), whilst the rest were in NYHA I. Left ventricular dimensions, Left Ventricular function and valve prosthetic valve function were similar between groups. Eleven (47.8%) patients in Group I and only five (25%) of Group II had trace or less TR at the follow-up (p < 0.05). There were 7 (16.2%) patients who had persistent moderate TR. Higher PASP and larger LV dimensions at three months were predictive of persistent moderate TR.

Conclusion

Mitral valve replacement does decrease the severity of tricuspid regurgitation amongst those with secondary moderate tricuspid regurgitation by at least one grade, but DeVega's annuloplasty confers a better repair result.

Key Words

tricuspid valve, tricuspid annuloplasty; DeVega's annuloplasty; secondary tricuspid regurgitation.

INTRODUCTION

Secondary tricuspid regurgitation is observed in 8% to 30% of those with left sided valve disease.¹ The “functional” tricuspid regurgitation due to left sided valve lesion is attributed to annular dilatation as the result of pressure overload on the right ventricle following raised left atrial pressure. This early pressure overload is accentuated by volume overload once the regurgitation sets in. It is well accepted that mild or lesser degrees of tricuspid regurgitation will regress after correction of the left sided pathology, while a higher grade of regurgitation may need to be addressed at the time of left sided valve surgery. The 2006 ACC/AHA Guidelines Pertaining to the Surgical Management of Tricuspid Valve Disease/Regurgitation suggests Tricuspid annuloplasty for less than severe TR in patients undergoing MV surgery when there is pulmonary hypertension or tricuspid annular dilatation as a class II b indication with level C evidence.² Partial annular plication and DeVega type annuloplasty perhaps have been the most popular tricuspid repair techniques. DeVega annuloplasty offers a readily available, technically less demanding and cheap alternative to the annuloplasty ring.

The present study was carried out to assess the effectiveness of De Vega’s annuloplasty in reducing the tricuspid incompetence amongst those with moderate tricuspid regurgitation, a population where the regurgitation severity has the potential to regress without added intervention.

METHODS

The study is a retrospective comparative study. Patients with secondary moderate tricuspid regurgitation who underwent mitral valve replacement with and DeVega tricuspid annuloplasty (Group I) were compared with those who did not undergo tricuspid repair (Group II). The data of those who were operated between June 2009 and June 2010 was collected from the admission records, operation records and discharge summaries. There were twenty three patients who underwent mitral valve replacement and DeVega type tricuspid annuloplasty (Group I) during the period and twenty (Group II) others who underwent mitral valve replacement for the same degree of tricuspid regurgitation at the same period.

The NYHA functional class and ECG variables were recorded at the time of admission. The echocardiogram was done taking the standard views and the tricuspid severity was graded according to the jet area and jet area to right atrial area ratio. The tricuspid regurgitation severity was graded as none, trace, mild, moderate and severe. Moderate tricuspid regurgitation was defined as jet area 5-10cm² and jet area to right atrial area ratio of 20-40% in apical four chamber view. The pulmonary artery systolic pressure was determined using the modified Bernoulli’s formula with 10mmHg added for right atrial pressure. All patients underwent mitral valve replacement under

cardio-pulmonary bypass and cold blood cardioplegic arrest, through a vertical left atriotomy just lateral to Waterston’s groove. DeVega’s tricuspid valve annuloplasty was done through a right atriotomy. For the annuloplasty, a double row of pledgeted 3-0 braided polyester stitch starting at the septo-anterior commissure was continued along the antero-lateral tricuspid annulus to reach the postero-septal commissure; here, the two ends were tied over another pledget. The tricuspid competence was checked with saline instillation into the right ventricle. The patients were followed up at three months after surgery. The functional class, ECG and echocardiogram variables were recorded. The data is presented as mean + S.D. and range for continuous variables and categorical variables as counts and percentages. The two groups were compared using independent sample’s t-test for continuous variables and Mann-Whitney U test for categorical variables. The Chi-square test of independence was used to assess for significant difference between the two groups. The two groups were reassessed after sub-grouping them into trace and less tricuspid regurgitation (Group A) and mild and more tricuspid regurgitation (Group B). The patients with persistent moderate tricuspid regurgitation were identified and evaluated for predisposing variables.

RESULTS

The population in our study was young and mostly comprised of women. All patients had rheumatic mitral valve disease with secondary tricuspid regurgitation of moderate severity. There was similar number of patients in atrial fibrillation between the groups. Most of the patients were in NYHA III functional class. Though the pre-operative mean ejection fraction was slightly lower for Group I, it was not significantly different from that of Group II. The patients in Group I had significantly elevated pre-operative pulmonary artery systolic pressure as compared to Group II.

The cardio-pulmonary bypass and aortic cross-clamp times were slightly prolonged for Group I, but were not significantly different from that of Group II. The DeVega annuloplasty did not significantly prolong the aortic cross clamp time as compared to those with mitral valve replacement only. The mediastinal bleed and ICU stay were also similar between the two groups. One patient in Group I was re-explored for excessive mediastinal bleed.

When the patients were followed up at three months, four patients were with NYHA II symptoms in Group II, otherwise, the rest were with NYHA I symptoms. There were more patients in atrial fibrillation amongst Group I patients. Though the left ventricular end-diastolic diameter was larger in Group I, the left ventricular ejection fraction was not significantly different between groups. The post-operative echocardiographic left atrial size, mean transprosthetic mitral valve gradient, tricuspid regurgitation pressure gradient and pulmonary artery pressure gradient

were not statistically different between the groups.

Table 1. Preoperative Variables.

Variable	Group I	Group II	p- value
Age (years)	25.13 + 9.4 (13-43)	31.40 + 14.8 (11-47)	0.101
Female	14(65.2%)	13 (65%)	0.988
NYHA (Class II-IV)	21 (91.3%)	17 (85%)	0.266 (MW-U)
AF	11 (47.8%)	7(35%)	0.091
Heart Rate (/min)	83.80+ 13.29	80.25 +5.35	0.259
LVIDd (cm)	5.70+1.09 (3.8-7.8)	5.82+1.15 (4.0-8.8)	0.71
LVIDs (cm)	4.03+0.86 (2.5-5.5)	3.94+0.99 (2.5-6.7)	0.76
EF	57.65+7.99 (35-70)	61.15+5.41 (50-70%)	0.10
MS (mod-sev)	12/23 (52.17%)	10/20 (50%)	0.88 (MW-U)
MR (mod-sev)	20/23 (86.95%)	15/20 (75%)	0.32 (MW-U)
PASP	71.52+19.76 (44-117)	48.60+12.75 (30-70)	<0.05
LA size	6.37+1.20 (4.5-9.3)	5.87+1.10 (4.1-7.9)	0.15
AR	0.70+0.7	0.20+0.61	0.058 (MW-U)

MW-U: Mann-Whitney U Test

Table 2. Peri-operative variables.

Variable	Group I	Group II	p- value
CPB	89.86+14.98 (65-125)	81.60+26.70 (47-142)	0.24
AoX	62.39+11.09 (45-90)	56.95+18.46 (30-108)	0.25
Mediastinal bleed	413.91+239.54 (120-1010)	334.75+175.30 (160-840)	0.22
ICU stay	3.30+1.60 (1-8)	3.25+2.17 (1-9)	0.92

Table 3. Variables at 3months post-operative follow-up.

Variable	Group I	Group II	p- value
NYHA I	23	16	0.026 (MW-U)
AF	14	8	0.042
LVIDd (cm)	5.11+0.45	4.76+0.63	0.04
LVIDs (cm)	3.60+0.60	3.37+0.68	0.13
EF	56.52+9.03	53.95+8.73	0.35
LA	4.90+0.76	4.80+0.93	0.71
TVMG	5.90+3.01	5.50+3.60	0.69
PASP	34.04+10.47	30.70+7.22	0.23
TR	1.61+0.89	1.75+0.91	0.45(MW-U)

MW-U: Mann-Whitney U Test

Most of the patients had at least one grade reduction in severity of tricuspid regurgitation. There were no patients with severe tricuspid regurgitation. As a group, the reduction in TR severity was not significant as per non-parametric test. To assess for difference, the groups were further sub-divided into those with trace and less TR, and those with mild and more TR at three months. Patients who underwent Tricuspid repair had less severe tricuspid regurgitation (TR < I: 47.8%) than those who did not undergo DeVega’s repair (TR < I: 25%). This was found to be significant with p-value of <0.05 and one degree of freedom (Table 6) using the Chi-square test for independence.

Table 4. Post-operative change in TR severity in Group I after surgery. (n=23)

TR Severity	Post-operative
0	2
I	9
II	8
III	4
IV	0

Table 5. Post-operative change in TR severity in Group II after surgery. (n=20)

TR Severity	Post-operative
0	3
I	2
II	12
III	3
IV	0

Table 6. Subgroup analysis of TR severity.

Variable	TR ≤ Trace	TR ≥ Mild
Group I	11 (47.8%)	12 (52.2%)
Group II	5 (25.0%)	15 (75.0%)

Table 7. Predictors of persistent moderate TR at 3 months.

Variable	TR < II	TR > III	p-value
LVIDd	4.84+0.50	5.48+0.59	0.005
LVIDs	3.42+0.61	4.08+0.58	0.012
EF	56.08+8.23	51.42+11.70	0.209
LA	4.79+0.87	5.17+0.58	0.285
TRPG	20.53+8.36	32.57+6.16	0.001
PASP	30.53+8.36	42+6.16	0.001
TMVG	5.84+3.35	5.07+3.01	0.579
HR	81.97+13.23	84.29+15.47	0.682
AF/ Sinus Rhythm	17/36	5/7	0.241

The patients with moderate tricuspid regurgitation were assessed to identify the predictors among the recorded variables. Larger left ventricular diameter and higher pulmonary artery systolic pressure were found to predict persistence of moderate tricuspid regurgitation. Other variables like atrial fibrillation, post-operative ejection fraction, left atrial size and trans-prosthetic mitral valve

gradient were not found to predict the persistence of moderate tricuspid regurgitation.

DISCUSSION

Regression of secondary tricuspid regurgitation after mitral valve surgery is a well accepted phenomenon. The initial documentation of TR regression by Braunwald et al³ amongst those undergoing mitral commissurotomy suggested that TR would heal by itself, and TR repair was done for those with severe regurgitation. The impact of moderate and severe tricuspid regurgitation on survival was documented by Nath et al amongst Veterans Administration patients.⁴ Dreyfus et al advocated pre-emptive repair of tricuspid valve to prevent late progression of TR.⁵ Despite the outcome studies, the tricuspid repair rate for less than severe secondary tricuspid regurgitation remains low.

In our study, the patients were relatively young (11-47yrs), and all had rheumatic mitral valve disease resulting in secondary moderate tricuspid regurgitation. The study population of Calafiore et al & Dreyfus et al. were a mix of various etiologies for secondary tricuspid regurgitation, with majority suffering from non-rheumatic mitral valve pathology.⁵⁻⁶ The group that underwent tricuspid repair had a higher pulmonary artery systolic pressure. The duration of cardiopulmonary bypass and aortic cross clamp was not significantly different between the two groups. Thus, DeVega annuloplasty can be undertaken quickly enough without prolonging the myocardial ischemia time. The post-operative course between the two groups was comparable, though one patient from the repair group had to be re-explored for excessive mediastinal bleed, and three required amiodarone to control their heart rate. The regression of pulmonary artery hypertension was independent of tricuspid repair and is attributable to resolution of elevated left atrial pressure following the mitral valve replacement as well as individual response to decrease in left atrial pressure. At three month's follow-up, most of the patients were in NYHA class I except for four patients who were in NYHA II functional class. There were more patients with atrial fibrillation in Group I. This group had a larger left atrium to start with and despite the size regression at three month's follow up, was marginally larger than that of Group II. The left ventricular internal diameter at end-diastole, trans-mitral gradient, tricuspid regurgitation pressure gradient and pulmonary artery systolic pressure were similar for both the groups. The left ventricular end systolic diameter was significantly larger for Group I, but the ejection fractions were similar. The mean tricuspid valve regurgitation was similar for both groups. This resolution can be attributed to the younger patient population, severe pulmonary hypertension, and larger resolution of pulmonary artery hypertension after mitral valve surgery as outlined by Hannoush et al.⁷ The 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 I patients were found to have a greater resolution with more patients with trace and less TR as compared to Group II.

There were altogether seven (16.27%) patients with moderate tricuspid regurgitation. There were four (7.39%) patients in Group I and three (15.5%) in Group II. The patients with moderate tricuspid regurgitation were found to have a larger left ventricular diameter at both end-diastole and end-systole. Fukuda et al proposed that altered left ventricular geometry directly affected the right ventricular geometry and function through the interventricular septum or pericardium.⁸ Thus, the enlarged left ventricle may have had a negative impact on the right ventricular function and the tricuspid competence, by the aforementioned mechanism. The left ventricular ejection fraction, though not significantly different, was found to be slightly less than those with persistent moderate tricuspid incompetence. Since the left ventricular ejection fraction was preserved in both the groups, it was not predictive of persistence of TR. The left atrium was also larger amongst those with persistent moderate TR, but was not found to be statistically significant. The pulmonary artery systolic pressure (PASP), as a derivative of the tricuspid regurgitation pressure gradient was found to be significantly more amongst those with moderate tricuspid regurgitation. Though, the PASP was elevated, it was not above 60mmHg, as identified by Kobayashi et al amongst post-repair patients of Tetralogy of Fallot.⁹ The attribution of pulmonary artery hypertension to recurrence TR was documented by Fukuda et al also.⁸ Atrial fibrillation was not found to be a determinant of tricuspid regurgitation in the present study. The persistence of moderate tricuspid regurgitation could not be determined by the pre-operative variables evaluated in the present study.

This study is a non-randomized study. The patients who underwent tricuspid annuloplasty were compared with a similar group of patients who underwent mitral valve replacement only during the same period. The quantification of tricuspid regurgitation was semi-quantitative and determinants of recurrent tricuspid regurgitation were not specifically evaluated. 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 DeVega's repair.

CONCLUSION

Mitral valve replacement does decrease the severity of tricuspid regurgitation amongst those with secondary moderate tricuspid regurgitation by at least one grade, but DeVega's annuloplasty confers a better repair result. Therefore, given the availability and cost of DeVega's annuloplasty, it should be considered as an alternative to other annuloplasties amongst those with moderate secondary tricuspid regurgitation.

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