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Original Article

Impact of Isolated Coronary Artery Bypass Grafting on Non-Organic Tricuspid Regurgitation Severity

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Abstract

Background: *Moderate non-organic tricuspid regurgitation (TR) concomitant with coronary artery disease is not uncommon. Whether or not TR improves after pure coronary artery bypass grafting (CABG), however, is unclear. The aim of this study was to evaluate the effect of isolated CABG on moderate non-organic TR.*

Methods: *This study recruited 50 patients (40% female, mean age: 65.38±8.01 years, mean left ventricular ejection fraction (LVEF): 45.74±13.05%) with moderate non-organic TR who underwent isolated CABG. TR severity before and after CABG was compared. Pulmonary arterial systolic pressure (PAPs)>30mmHg and LVEF<50% were considered elevated PAPs (EPAPs) and LV systolic dysfunction, respectively. Presence of Q-wave in leads II, III, and aVF was considered inferior myocardial infarction (inf. MI).*

Results: *Pre-operatively, 81.5% of the patients had EPAPs, 16% right ventricle (RV) dilation, and 50% left ventricle (LV) and 16% RV systolic dysfunction. TR severity improved in 64% after CABG, whereas it remained unchanged or even worsened in others (P value<0.001). Patients with inf. MI showed no improvement in TR, while patients without inf. MI had significant TR regression after CABG (P value=0.050). Improvement of TR severity after CABG was not related to pre-operative RV size and function, LV systolic function, or PAPs reduction.*

Conclusion: *Although TR severity decreased remarkably after isolated CABG, a considerable number of the patients had no TR regression. In addition, only absence of inf. MI was significantly correlated to TR improvement after CABG. Further prospective studies with long-term follow-up are needed to determine the other factors predicting TR regression after isolated CABG.*

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Introduction

Non-organic tricuspid regurgitation (TR) is a common finding which is often ignored until it is moderate or severe.[1,2] In addition to right ventricular (RV) dilation and dysfunction; regardless of pulmonary artery pressure (PAP) and left ventricular ejection fraction (LVEF), moderate or greater TR is associated with worse survival.[3, 4] RV dilation and consequently enlarged tricuspid valve annulus due to RV ischemia or infarction, mitral valve disease, and pulmonary vascular disease is a more common cause of functional TR than primary valvular defect.[5] For many decades, it was thought that functional TR was related to mitral valve disease; consequently, it was suggested that once the left-sided lesions were treated, functional TR would regress spontaneously.[6, 7] Despite the correction of left-

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sided lesions, concomitant functional TR that is surgically ignored can persist or can appear anew.[8, 9] The mechanism of these situations is unclear, however. In addition to the other causes of TR, previous investigators hypothesized that TR could be due to coronary-disease-induced ischemia.[10] In the coexistence of moderate or greater TR with coronary artery disease and the absence of any structural defect in the tricuspid valve (TV), we need to ascertain whether TV should be repaired or ignored during coronary artery bypass grafting (CABG) because there is a dearth of data on the accompaniment of TR with ischemic heart disease.

The aim of this study was to analyze pre- and post-operative TR severity in patients with structurally normal TV leaflets and moderate TR who underwent isolated CABG to find out whether TR severity improved after surgery. In addition, we investigated factors affecting this improvement.

Methods

A total of 50 patients with moderate TR underwent isolated CABG at Tehran Heart Center between February 2003 and September 2008. None of them had concomitant organic TV disease.

Two-dimensional echocardiography was performed with a 3.5-MHZ phased array sector scanner (Vivid 3 GE) twice for each patient, within a week before and during the first month after surgery. The TR severity was observed in all standard views, including the apical 4-chamber, parasternal short axis, RV inflow, and subcostal views. TR was graded as mild, moderate, and severe.[11] LVEF was measured by the modified biplane Simpson method. For the estimation of pulmonary arterial systolic pressure (PAPs), the peak tricuspid regurgitation gradient was obtained from multiple windows and was added to right atrial pressure (RAP) as described, previously.[12] Tricuspid annular plane systolic excursion (TAPSE) and Doppler tissue imaging were used to determine RV systolic function.

LVEF <50 % and PAPs >30 mmHg were considered LV systolic dysfunction and elevated pulmonary arterial systolic pressure (EPAPs),[13] respectively. TAPSE <15 mm and Doppler tissue imaging peak systolic velocity <10 cm/s were deemed as RV systolic function impairment. Presence of Q-wave in leads II, III, and aVF was regarded as inferior myocardial infarction (inf. MI).

All the surgeries were performed with similar cardiopulmonary bypass. The median number of grafts was 3 (range: 2-5); the mean cardiopulmonary bypass time 66.21±23.64 minutes and the aortic clamp time 56.10±28.42 minutes.

The numerical variables were presented as mean±SD, and the categorical variables were summarized by absolute frequencies and percentages. The continuous variables were compared using Student's t-test, and the categorical variables were compared using chi-square or Fisher's exact test, as required.

Before and after surgery, the data were compared using a paired t-test for the continuous, Wilcoxon signed ranks test for the ordinal, and McNemar test for the binary categorical variables. For the statistical analysis, the statistical software SPSS version 13.0 for Windows (SPSS Inc., Chicago, IL) was used. All the P values were 2-tailed, with statistical significance defined by a P value ≤0.05.

Results

The demographic and clinical data are presented in Table 1. The study population consisted of 50 patients at a mean age of 65.38±8.01 years; all the patients had moderate TR before surgery. Overall, TR severity reduced significantly after surgery (P value <0.001). TR severity improved in 32 (64%), worsened in 1 (2%), and did not change in 17 (34%) patients. There was mild and moderate mitral valve regurgitation in two patients, the former with rheumatismal and the latter with degenerative changes. Neither of them underwent concomitant intervention on mitral valve during CABG.

Table 1. Patients' characteristics (n=50)

Age (y)	
Mean±SD	65.38±8.01
Range	47-80
Female (%)	40
LVD (%)	50
EF (%)	
Mean±SD	45.74±13.05
Range	12.5-66
Extent of coronary artery disease (%)	
3-vessel	72
2-vessel	18
1-vessel	10
Number of bypass grafts	
Mean±SD	3.48±0.91
Range	2-5
AF (%)	4

LVD, Left ventricle dysfunction; EF, Ejection fraction; AF, Atrial fibrillation

The frequency and improvement of TR severity after CABG in terms of the presence or absence of RV dilation, LV and RV systolic dysfunction, and inf. MI are illustrated in Table 2.

A comparison of the TR improvement after surgery between the patients with normal and those with dilated RV yielded no statistically significant difference. In addition, post-operative TR changes in relation to RV and LV systolic function was assessed. Similar to RV size, there was no significant relationship between TR improvement and pre-operative LV and RV systolic functions in our patients (Table 2).

Table 2. Frequency of TR severity changes after CABG

Before CABG	After CABG		
	Improved	Not changed or Worsened	P value
RV dilation			
Yes: 16.0 (8)	37.5 (3)	62.5 (5)	NS
No: 84.0 (42)	69.0 (29)	31.0 (13)	NS
RV systolic function			
Normal: 84.0 (42)	66.7 (28)	33.3 (14)	NS
Dysfunction: 16.0 (8)	50.0 (4)	50.0 (4)	NS
LV systolic function			
EF<50% : 50.0 (25)	56.0 (14)	44.0 (11)	NS
EF≥50% : 50.0 (25)	72.0 (18)	28.0 (7)	NS
Inferior MI			
Yes: 10.0 (5)	20.0 (1)	80.0 (4)	0.05
No: 90.0 (45)	68.9 (31)	31.1 (14)	0.05

Data are presented as % (n)
TR, Tricuspid regurgitation; CABG, Coronary artery bypass grafting; RV, Right ventricle; LV, left ventricle; MI, Myocardial infarction; EF, Ejection fraction; NS, Non-significant

After surgery, mean EF increased to 47.10±12.38 mmHg from 45.74±13.05 mmHg but this change was not statistically meaningful. Of the patients with EF >50% before surgery, EF decreased to <50% in 2 patients after CABG; TR severity, however, improved in both.

Inf. MI was detected in 10% of the patients. In patients without inf. MI, TR severity improved significantly after surgery (P value=0.050); while in patients with inferior MI, the change in TR severity was not significant.

This finding encouraged us to investigate the relationship between the presence or absence of right coronary artery (RCA) stenosis and TR improvement after surgery. RCA stenosis was diagnosed in 70.8% of the patients. After CABG, TR severity was ameliorated in 58.8% of the patients with RCA stenosis, whereas 78.8% of the patients without RCA stenosis showed TR improvement, although this difference was not significant.

Prior to surgery, 81.8% of the patients had EPAPs; in 34.4% of whom PAPs fell to ≤30 mmHg after surgery (P value=0.057). Mean PAPs reduced significantly from 41.32±10.99 before surgery to 34.95±7.23 mmHg thereafter (P value <0.001). Nearly 64% of the patients with pre-operative EPAPs and 62.5% of the normal PAPs patients showed TR improvement.

Among the patients in whom PAPs fell to ≤30 mmHg after operation, 72.7% had improvement in TR severity; while the figure was 57.1% for the patients in whom PAPs remained >30 mmHg after CABG. This difference, however, was not statistically remarkable.

Discussion

In our study, the improvement in TR severity in patients with coronary artery disease and moderate non-organic TR was significant after CABG. TR regression appeared in 64% of the patients; and interestingly, it was significantly related to the pre-operative absence of inf. MI.

Many studies have investigated changes in mitral regurgitation severity after pure CABG in patients with concomitant coronary artery disease and mitral valve insufficiency.[14-16] Nonetheless, there are few studies in the existing literature focusing on the changes in TR severity after surgery, with the major focus being on TR improvement after left-sided valve surgery or TV repair. We conducted the present study to delve into the changes in non-organic TR in patients with coronary artery disease undergoing CABG alone.

Matsunaga et al. reported that in the patients undergoing simultaneous CABG and MVR, the post-operative TR did not differ between the groups of patients with and without TV repairs.[17] In their study, 12 patients had moderate or greater TR before surgery; one third of them showed TR improvement and two thirds showed no change in TR severity at follow-up in the group of patients without TV repair.

Formerly, it was demonstrated that TR was associated with RV systolic dysfunction in addition to reactive pulmonary hypertension.[4] We sought to find out the factors influencing TR improvement after isolated CABG. With respect to RV systolic function, TR improvement occurred in about half of the patients with RV systolic dysfunction; while in the patients with normal RV systolic function, this improvement was about 70%. Moreover, 70% of the patients with normal RV size showed improvements in TR after surgery; whereas in the patients with RV dilation, this improvement occurred in about 40%. These findings demonstrated that RV systolic function and size had no significant relationship with the changes in TR severity, although it may be due to a relatively small number of the cases in this study.

It was previously hypothesized that direct myocardial ischemia might have a harmful effect on the RV function and eventually lead to tricuspid regurgitation.[5] We, therefore, investigated the possible relationship between myocardial infarction and TR severity changes after isolated CABG. In this regard, data analysis revealed that in the patients without inf. MI, TR regression was significant after surgery, while the patients with inf. MI did not show significant TR improvement after CABG.

McCarthy et al. reported that TV annuloplasty did not consistently eliminate functional regurgitation, and the risk factors for worsening regurgitation included pre-operative higher regurgitation grade and poor LV function.[18] Also, Trichon et al. suggested that if surgical revascularization was necessitated in the presence of TR among patients with systolic dysfunction, concomitant TV repair should be strongly considered.[5] By contrast, in the present study there was no significant relation between LV systolic function and TR improvement after CABG; 60% of the patients with normal and 60% of those with impaired LV systolic function

showed TR regression after surgery. Thus, consideration of systolic dysfunction as a conclusive reason to perform concomitant TV repair needs more investigation.

Some investigators have shown that severe functional TR with a dilated annulus can be eliminated spontaneously after reducing PAP[19] as opposed to others who have demonstrated no significant determinable role for elevated PAP in functional TR.[20] In our investigation, PAPs reduced significantly after surgery and more than 60% of the patients with pre-operative EPAPs revealed TR regression; improvement occurred mostly in the patients whose PAPs fell to ≤30 mmHg. These results can support the idea of TR regression after PAPs reduction, although the number of the study patients was not probably high enough to reach significant correlation.

The limitations of our study were its retrospective design and relatively small number of cases; non-significant variables may become significant in a larger series. In this study, we used only the immediate post-operative echocardiography data. Longer follow-up periods are required to evaluate the persistence or absence of TR changes.

Conclusion

In this retrospective study, we investigated TR severity changes after isolated CABG in patients with coronary artery disease and moderate non-organic TR. Although TR severity decreased remarkably after surgery, a considerable number of the patients had no TR regression. According to this study, only the absence of inf. MI was significantly correlated to TR improvement after CABG and improvement was not related to pre-operative RV size, RV and LV systolic function, RCA stenosis, EPAPs, or PAPs reduction after surgery. Further prospective studies with long-term follow-up are needed to determine the other factors predicting TR regression after pure CABG.

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