Pericardiectomy for constrictive pericarditis with postoperative increase of tricuspid regurgitation

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Abstract

We report a case of tricuspid regurgitation (TR) that increased markedly after pericardiectomy for constrictive pericarditis. Preoperative mild-to-moderate TR increased to severe following surgery. The patient was asymptomatic, and gradual regression of TR was observed. Eighteen months postoperatively, a left atrial thrombus formed, and a second surgery consisting of thrombectomy and tricuspid annuloplasty was performed. The increase in TR after pericardiectomy was thought to be due to dilatation of the right chambers and the annulus of the tricuspid valve. Several studies that entail mitral regurgitation after pericardiectomy are discussed.

Keywords: Pericardiectomy, constrictive pericarditis, tricuspid regurgitation, mitral regurgitation

Introduction

Pericardiectomy for constrictive pericarditis is not a complicated procedure; it involves peeling off the hard shell of calcification. However, it is not always a safe procedure and has a 5-15% perioperative mortality [1]. Longstanding disease may induce myocardial atrophy or fibrosis; mediastinal flammation and fibrosis may produce recurrent cardiac compression, and inadequate or incomplete resection may result in no improvement [1]. Tricuspid regurgitation (TR) is another factor, which may complicate pericardial constriction [2]. Recently, we encountered a surgical case of pericardial constriction in which TR increased postoperatively. Although the patient tolerated the early-phase procedure, tricuspid annuloplasty was subsequently performed. The relationship between pericardiectomy and regurgitation of the atrio-ventricular valves is discussed in this report.

Case presentation

A 68-year-old woman was referred to our hospital due to acute heart failure. She had been diagnosed with constrictive pericarditis with extensive calcification15 months previously. After treating the acute heart failure with intravenous furosemide, her symptoms abated; however, she required a high dosage of oral furosemide (120 mg/day) and spironolactone (50 mg/day). She was scheduled for surgery in June 2009. Preoperative transthoracic echocardiography revealed diastolic dysfunction with a mild to moderate TR. The left ventricular diastolic dimension (LVDd) was 40 mm, the left ventricular systolic dimension (LVDs) was 27 mm, and the left ventricular ejection fraction (LVEF) was 60.0%. The deceleration time was 127 msec, indicating diastolic dysfunction. (**Table 1**) presentsthe results of echocardiograms in order from preoperative to recent dates.

Cardiac catheterization revealed a dip and plateau of right and left ventricular pressures. Computed tomography (CT) revealed severe calcification of the pericardium.

The first surgery was a pericardiectomy, via amedian sternotomy, and cardiopulmonary bypass(CPB). The anterior pericardium was incised between the phrenic nerves and the inferior pericardium. The harmonic scalpel was effective in resecting the calcified pericardial shell. Since immediate preoperative echocardiography revealed only mild TR, tricuspid repair was not performed. Postoperative echocardiography revealed severe TR due to annular dilatation. The LVDd increased to 47 mm, the LVDs increased to 34 mm, and the LVEF decreased to 54.9%. Although she still required the same doses of oral diuretics, she had no cardiac failure symptoms, and received routine follow-up. (**Figure 1**) shows that TR increased after the first surgery. (**Figure 2**) shows the preoperative enhanced CT and the postoperative plain CT. The annular diameter of the tricuspid valve increased from 33.8 mm to 36.9 mm postoperatively. After pericardiectomy, the direction of the apex rotated dorsally, and the right heart chambers moved anteriorly.

A pericardial biopsy revealed no signs of active inflammation and no signs of granuloma formation. Therefore, the etiology of the pericarditis was unknown. Furthermore, cultures were negative for bacterial infection and negative for tuberculous and nontuberculous mycobacterial infection.

The preoperative serum creatine kinase (CK)-MB of the referral day when acute cardiac failure occurred was only 18 IU/l. In addition, the serum CK-MB on the first postoperative day was only 26 IU/l. These data indicated that with the first procedure there was no evidence of myocardial involvement pre- and post-surgery.

In December 2010, echocardiography revealed moderate TR

Table 1. Echocardiography.

	Pre 1st op	Post 1st op	Pre 2nd op	Post 2nd op	Recent
LVDd (mm)	40	47	49	65	55
LVDs (mm)	27	34	31	52	37
LVEF (%)	60.0	54.9	66.8	40.7	59.7
TR	mild-moderate	severe	moderate	trivial	mild
MR	trivial	trivial	trivial	trivial	mild

LVDd: Left Ventricular Diastolic Dimension
LVDS: Left Ventricular Systolic Dimension
LFEF: Left Ventricular Ejection Fraction
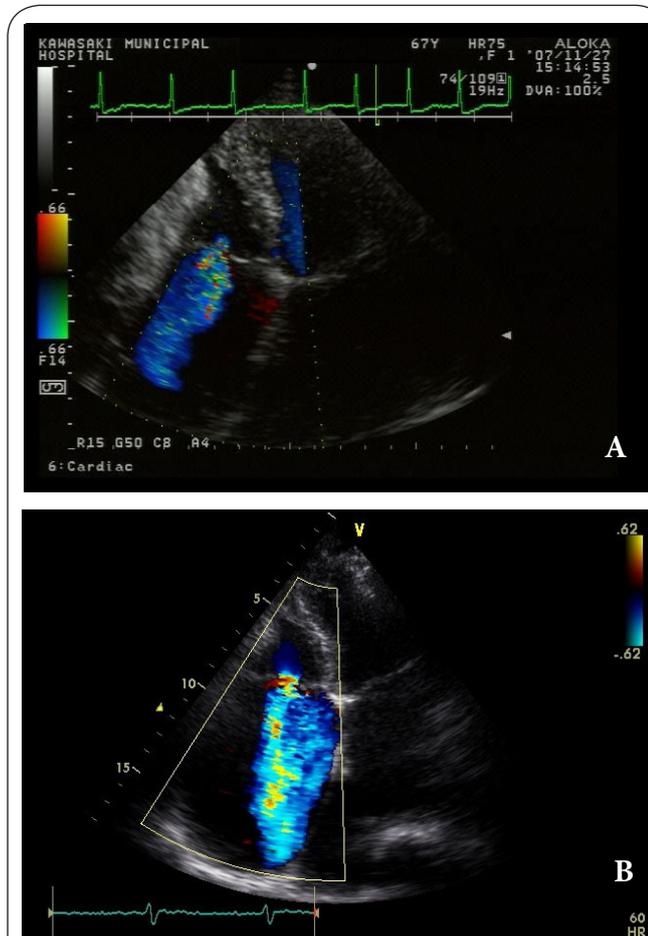TR: Tricuspid Regurgitation
MR: Mitral Regurgitation

Figure 1. (**A**) Preoperative color Doppler echocardiography showing mild to moderate tricuspid regurgitation (TR). (**B**) Postoperative color Doppler echocardiography showing severe TR.
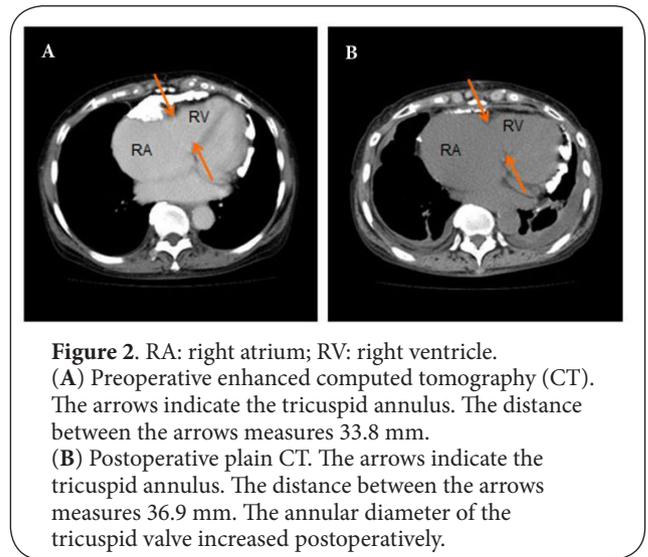
Figure 2. RA: right atrium; RV: right ventricle.
(**A**) Preoperative enhanced computed tomography (CT). The arrows indicate the tricuspid annulus. The distance between the arrows measures 33.8 mm.
(**B**) Postoperative plain CT. The arrows indicate the tricuspid annulus. The distance between the arrows measures 36.9 mm. The annular diameter of the tricuspid valve increased postoperatively.

control (PT-INR 1.20). The LVDd was 49 mm, the LVDs was 31 mm, and the LVEF was 66.8%. The second surgery consisted of a tricuspid annuloplasty using a 34 mm Cosgrove ring and a thrombectomy. Postoperatively, transient renal failure occurred, and echocardiography revealed trivial TR. The LVDd increased to 65 mm, the LVDs increased to 52 mm, and the LVEF decreased to 40.7%.

The patient is currently doing well; echocardiography reveals mild TR, the LVDd is 55 mm, the LVDs is 37 mm, and the LVEF is 59.7%. During the course of follow-up, mitral regurgitation (MR) has remained trivial, except for the recent echocardiography, which revealed mild MR.

Discussion

There are two standard approaches for a pericardiectomy: anantero-lateral thoracotomy or a median sternotomy [3]. We usually select a median sternotomy with routine use of CPB. We believe that it safer to use CPB because of the potential risks of hemodynamic deterioration with this disease. Under a median sternotomy, a nanterior excision of the pericardium between the phrenic nerves, and the inferior pericardium is performed. This primarily relieves the surface of the right chambers and some of the left chambers, which may lead to annular dilatation and regurgitation of the tricuspid valve.

Gongora et al., [2] evaluated 261 cases of constrictive pericarditis. Among those with moderate/severe TR, operative mortality was similar whether or not repair was undertaken. They reported only 29% improvement in TR if repair was not performed. Their conclusion was to consider repair when moderate/severe TR exists to reduce symptoms. TR may worsen after pericardiectomy [2,4]. Our case exhibited only mild TR immediately before surgery, and mild cases were not discussed by Gongora et al.

Mantri et al., [5] studied 14 cases of pericardiectomy for

with a 3 cm thrombus in the left atrium. The etiology of the thrombus formation was suspected to be the enlarged left atrium (63 mm), atrial fibrillation, and suboptimal warfarin

constrictive pericarditis with TR or MR without repairing them. Long term follow-up resulted in regression of the regurgitation in half the cases. They concluded that if an adequate pericardiectomy is done at an earlier stage, the regurg-itations will regress. There were no patients in whom these regurgitations worsened after pericardiectomy.

Johnson et al., [4] described two cases of pericardiectomy in which TR worsened postoperatively. They suggested that the deterioration was a result of postoperative right ventricular dilatation. Expansion of the right ventricle results in severe TR. In our case as well, expansion of the right ventricle due to decortification anteriorly and the annular dilatation of the tricuspid valve occurred concomitantly.

We did not perform tricuspid annuloplasty because the echocardiography taken immediately before the first surgery showed only mild TR. Since severe TR developed postoperatively, tricuspid annuloplasty was probably indicated in the first surgery. Before the second surgery, TR had decreased to a moderate level. Since the second surgery was indicated due to a left atrial thrombus, concomitant tricuspid annuloplasty was performed. There are cases in which MR after pericardiectomy subsided due to compensation and did not require reoperation [6,7]. Similarly in our case, the possibility exists that the TR might have regressed further without the second surgery.

Nakamura et al., [8] reported a case of severe MR posto-peratively that required surgical management three weeks after the pericardiectomy. Their case was primary mitral disease; thus, it differs from that of ours. They stated that in MR after pericardiectomy, the change in the left ventricular geometry and shift of the papillary muscles [6], increased mobility of the lateral wall, and increased the left ventricular volume [7], are the hypotheses for the cause. Although our case was a TR, the same hypotheses may be applied to the right ventricle.

After pericardiectomy, initial clinical and hemodynamic responses are not always dramatic and continued improve-ment may occur over many months [9]. This is consistent with the fact that the left ventricular dimension progressively increases and the systolic function remains stable [9]. Since the left ventricle is located in the postero-lateral side where the thick pericardium is not resected, it is likely that the left ventricle gradually dilates. In our case, the most recent left ventricular dimension was larger than the preoperative data from the first procedure. This may also explain why the MR recently increased from trivial to mild with probable annular dilatation.

Conclusions

TR increased markedly after pericardiectomy for constrictive pericarditis. The etiology is thought to be the dilatation of the right chambers with annular dilatation of the tricuspid valve. In treating constrictive pericarditis, care must be taken to evaluate TR, even if it is mild. Even if TR deteriorates to a severe level, it may regress due to compensation.

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