Case Report

Stubbornly preserving native leaflets is not always right: a case of tricuspid valve re-operation

Lijie Jiang, Xueshan Zhao, Jiao Li, Zhong Wu

Abstract

Tricuspid valve replacement is becoming more and more popular at various medical centres due to the increase in numbers of patients with tricuspid regurgitation. We report on a case of a 59-year-old man who had undergone tricuspid valve replacement with preservation of the native leaflets two years earlier, and developed early prosthetic dysfunction, which may have been caused by fusion of the native valve leaflets with the prosthetic valve leaflets. The experience of this case informs us that preserving the subvalvular apparatus may impede the motion of the prosthesis, and that adapting the individual morphology of the native tricuspid valve during tricuspid valve replacement could benefit the patient and avoid re-operation.

Keywords: cardiac surgical procedures, heart valve prosthesis implantation, re-operation

Submitted 21/3/21, accepted 14/4/21
Cardiovasc J Afr 2021; 32: online publication www.cvja.co.za
DOI: 10.5830/CVJA-2021-019

With the increase in morbidity rate of tricuspid regurgitation, tricuspid valve replacement (TVR) is becoming more and more popular. However, the pathogenesis of prosthetic valve dysfunction after TVR is not entirely understood. From experience, the main cause is pannus formation on the prosthetic cusps on the side of the right ventricle. At the same time, the reason that the native valve leaflets attach to the prosthesis should not be neglected. We present a case of early decay of the tricuspid prosthesis due to preserving the native valve leaflets during the first operation, which restricted the closure of the prosthesis.

Case report

A 59-year-old man developed dyspnoea on exertion of one year duration. Transthoracic echocardiography showed severe tricuspid regurgitation. He was then diagnosed with rheumatic heart disease and underwent TVR at another hospital with the choice of a bioprosthetic valve (Carpentier–Edwards PERIMOUNT tricuspid bioprosthetic valve, 31 mm) on the basis of his age. On the 12th day after the operation, he was discharged from hospital and six months of oral anticoagulant therapy was prescribed.

However, the patient developed worsening dyspnoea on exertion two years after the operation and was admitted to our hospital. Physical examination showed a high-pitched, pansystolic murmur in the fourth intercostal space of the parasternal region. Transesophageal echocardiography revealed poor closing of the bioprosthetic valve, causing severe regurgitation from the valve orifice rather than perivalvular leakage.

Transesophageal echocardiography (Fig. 1) demonstrated a regurgitant jet from the anterior cusp of the bioprosthetic valve. Coronary arteriography showed a healthy coronary artery system.

Re-operation was done via a routine median sternotomy, and cardiopulmonary bypass was set up through the ascending aorta and superior/inferior vena cava after separating the pericardium from the heart. Cross clamping and cardioplegic cardiac arrest were routinely performed and the tricuspid valve was exposed with a right atriotomy. The bioprosthetic valve was in situ, closely

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Fig. 1. Transoesophageal echocardiography: the regurgitant jet from the anterior cusp of the bioprosthetic valve.
attached to the annulus, and the annulus maintained a good shape without expanding.

After incision of the bioprosthetic valve, we were surprised to found that, while the native posterior and septal leaflets were connected to the chordae tendineae and in a normal state, the native anterior leaflet, preserved in the previous operation, was adhering tightly to the bioprosthetic valve leaflets (Fig. 2). The leaflets of the bioprosthetic valve maintained good plasticity, but were restricted from closing by the native anterior tricuspid leaflet. The anterior leaflet of the bioprosthetic valve did not meet with the other two (Fig. 3), causing severe tricuspid regurgitation.

The bioprosthetic valve was therefore removed and most of the native tricuspid valve leaflets were excised, keeping only a small portion of the native leaflet, which was plicated to the annulus. The bioprosthetic valve was replaced with a new mechanical prosthetic valve (31-mm St Jude Medical prosthesis), according to the patient’s own choice before the re-operation, and the heart and sternotomy were successfully sutured closed.

The patient recovered well. Transthoracic echocardiography showed good functioning of the new tricuspid mechanical prosthesis.

Discussion
Preservation of the subvalvular apparatus and creating valve–ventricular interaction has been advocated in the past decades, causing this to become a routine procedure in TVR operation. It maintains better right heart function and has a lower probability of myocardial rupture. However, the phenomenon of bioprosthetic valve regurgitation in this case alerted us to the fact that stubbornly preserving the native leaflets may cause restrictive motion and early dysfunction of the prosthetic valve, which can lead to re-operation.

Since the first operation was performed at another hospital, we could only guess why the native tricuspid valve leaflet had adhered to the bioprosthetic valve. The patient may have suffered from pulmonary hypertension after the first operation, which would have caused the native anterior leaflet to come into contact with the bioprosthetic valve and eventually fuse.

Prosthetic valve regurgitation induced by preserving the native valve leaflets is not rare in the left heart system. Tomoki et al.4 reported a similar case of the aortic valve, in which the left coronary leaflet of the bioprosthetic valve had attached and fused to the wall of the sinus of Valsalva, causing deformation of the valve leaflet and a commissural gap between the left and right coronary leaflets. They hold the view that a mismatch of the prosthesis size was the main reason for aortic regurgitation in that case.

This was not so in our case because the same size prosthesis (both 31 mm) was used in both operations, and we observed the tricuspid annulus to be in good shape with no expansion. Some cases may need a second TVR due to thrombosis in the prosthetic valve,5 however anticoagulant therapy was routinely performed in our case and ultrasonic cardiography carried out in the third and 12th month after the first operation had revealed the bioprosthesis was functioning well.

Conclusion
The experience from this case indicates that preserving the subvalvular apparatus may hamper the motion of the prosthetic valve. However one need not abandon the valve-preserving principle. The decision to keep or remove the native tricuspid valve during TVR depends on its morphology. If the individual morphological features of the native tricuspid valve and right ventricular structure are in good shape, the subvalvular
apparatus can be preserved and valve–ventricular interaction created. However, an expanded tricuspid annulus should be excised during tricuspid valvoplasty, and the native valve leaflets, which may hamper the prosthesis from working properly, should be excised and the rest should be plicated to the annulus of the tricuspid valve, rather than preserving all the leaflets.

Pre-operative echocardiography should be carefully executed to acquire a better view of the native valve, and postoperative echocardiography can be used to check the surgical outcome.

Lijie Jiang, Xueshan Zhao and Jiao Li contributed equally to the article.

References