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# Repair of Rheumatic Tricuspid Valve Stenosis with Autologous Pericardium

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## ABSTRACT

The aim of the present study was to describe a successful method for treating rheumatic tricuspid valve stenosis with autologous pericardium, commissurotomy, and a Kalangos ring without replacing the tricuspid valve.

## INTRODUCTION

Rheumatic diseases mostly involve mitral and aortic valves; in contrast, involvement of the tricuspid valve (TV) is less common. Functionally important TV disease is not rare; however, it has been reported in 10% to 20% of patients in association with other valve diseases. Common pathologies leading to a mixed state of stenosis and regurgitation are thickening of the leaflets, fibrosis, and commissural fusion. When the TV disease is not severe, surgical treatment can be successful in combination with commissurotomy and annuloplasty; in severe forms of the disease, valve replacement is recommended. Although authors of previous studies have claimed that mechanical prosthetic valves in the tricuspid position have a higher risk of complications than aortic and mitral prosthetic valves, mechanical prosthetic valves are preferred over bioprosthetic valves in the tricuspid position because of their hemodynamic characteristics and durability [Kaplan 2002].

Repair of severe TV stenosis with an autologous pericardial patch for both leaflets (anterior and septal) is very rarely performed. This report describes a case of severe TV stenosis that was repaired successfully with autologous pericardium without the need for TV replacement.

## CASE REPORT

A 55-year-old woman was admitted with known atrial fibrillation (AF) and severe progressive dyspnea (New York Heart Association class III). A physical examination revealed a systolic murmur at the apex, a loud second heart sound, diminished respiratory sounds at the basal zone of both lungs, hepatosplenomegaly, and pitting edema of the lower extremities. Chest radiography revealed moderate cardiomegaly. A preoperative echocardiography examination revealed severe mitral stenosis (valve area, 0.9 $cm^2$), critically restricted mitral valve leaflets, severe pure TV stenosis (valve area, 0.9 $cm^2$; transvalvular diastolic mean gradient, 10 mm Hg), moderate pulmonary hypertension (40 mm Hg), a dilated left ventricle with reduced systolic ventricular function, thrombus at the left atrial appendage, a dilated left atrium (62 mm), and a dilated right atrium (120 mm). A coronary angiography evaluation showed no significant stenosis of the coronary arteries. An operation was carried out after optimization of heart failure therapy.

After a median sternotomy was performed, cardiopulmonary bypass was established via aortic and bicaval cannulation. The right atrium was severely dilated (Figure 1A). Following antegrade cold blood cardioplegia, a standard right atriotomy was performed. The mitral valve was observed through a transseptal incision.

The posterior and anterior mitral valve leaflets appeared thickened. Total resection of the anterior leaflet and subtotal resection of posterior leaflet of the mitral valve were performed. A 31-mm St. Jude mechanical valve prosthesis (St. Jude Medical, St Paul, MN, USA) was implanted. The TV was inspected after mitral valve replacement. The motion of the TV leaflets was severely restricted owing to commissural fusions. To increase the effective area of the valvular orifice, we performed commissurotomies for the anteroseptal and posteroseptal commissural fusions. We evaluated TV competency by rapidly injecting a saline solution through a flow-directed catheter inserted through the TV. We observed leakage between the anterior and septal leaflets. To eliminate the malcoaptation between the shrunk anterior and septal leaflets, we made an incision slightly concave to the annulus (2-3 mm from the annulus) along the anterior leaflet between the commissures and made a similar incision along the septal leaflet. We simultaneously harvested an autologous pericardial patch, bathed it in 0.2% glutaraldehyde solution for 8 minutes, and rinsed the patch with saline solution. The free edges of the anterior and septal leaflets were retracted to the opposite side and stretched to the annulus. The width and length of the deficit were measured for both the anterior and septal leaflets.

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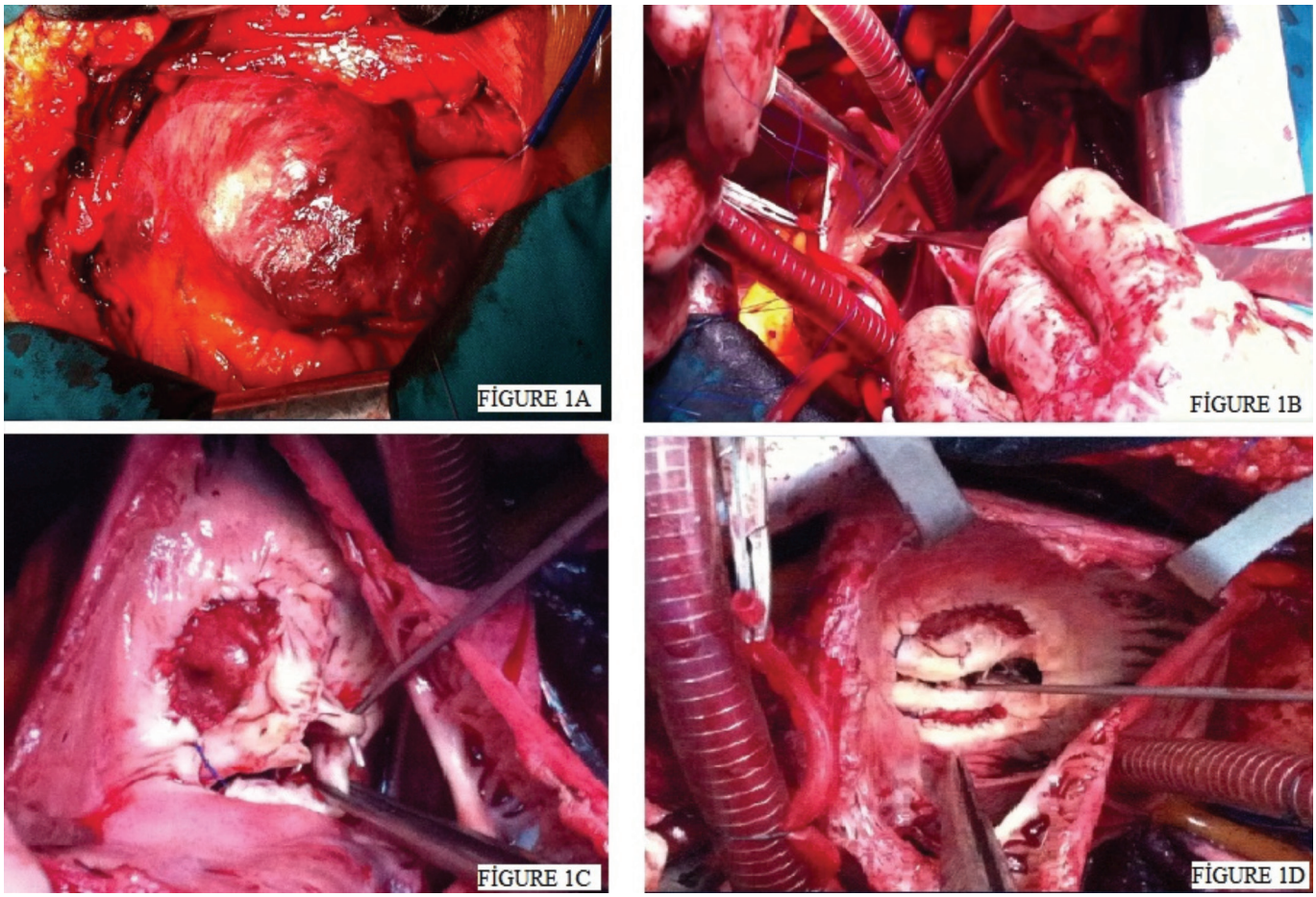


Figure 1. Intraoperative view of the dilated right atrium (A). Glutaraldehyde-treated pericardial patches were sewn via a continuous stitch with 6-0 polypropylene suture (B, C, D).

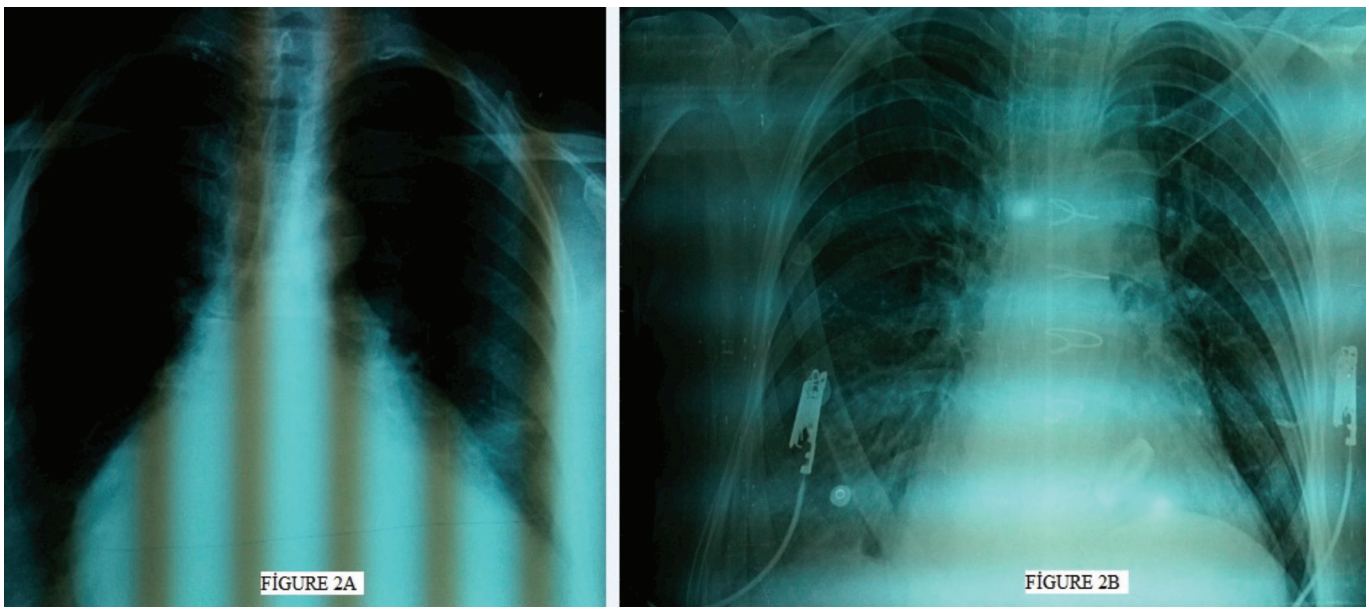


Figure 2. A preoperative chest radiograph (A) and an early postoperative chest radiograph (B).

The pericardial patch was shaped into an elliptical strip (patch for anterior leaflet, 1.1 cm × 2.5 cm; patch for septal leaflet, 1.4 cm × 2.4 cm). The pericardial patches were sewn into the 2 leaflets with a continuous stitch of 6-0 polyproplylene suture (Figure 1B–1D). Finally, we performed tricuspid annuloplasty with the Kalangos Biodegradable Tricuspid Ring (Kalangos Biodegradable Tricuspid Ring; Bioring, Lonay, Switzerland). An intraoperative saline test revealed no fluid leakage, and we easily weaned the patient from cardiopulmonary bypass.

The patient's early postoperative course was uneventful. The patient was anticoagulated with warfarin and was discharged 8 days after the operation. Two days after the operation, a chest radiograph proved the absence of pulmonary edema, and the cardiothoracic index decreased to 0.45 (Figure 2B). A 3-month follow-up echocardiography evaluation showed a normally functioning TV with no stenosis and regurgitation of grade 1. The left ventricular dimension and function were improved. After 3 months of follow-up, the patient was symptom free.

## DISCUSSION

Tricuspid valve stenosis (TS) is a rare clinical condition, and more rarely is it an isolated disorder. Usually TS is accompanied by mitral stenosis and TV regurgitation, both of which contribute to a greater increase in right atrial pressures. Rheumatic heart disease accounts for most of the cases. Other rare causes of TS include congenital TS, carcinoid syndrome, endomyocardial fibrosis, endocarditis, and extracardiac tumors [Bruce 2009]. TS is considered severe when the valve area is <1.0 $cm^2$, but the actual degree of stenosis and its severity are evaluated primarily by continuous wave Doppler echocardiography and calculating the mean diastolic pressure gradient and the transvalvular velocity [Baumgartner 2009]. In clinical practice, the medical therapy for symptomatic TS is usually insufficient, and AF is a particularly common clinical finding. If AF is present, controlling the ventricular rate promotes diastolic filling and improves symptoms. Medical therapy, including diuretic therapy, anticoagulation, and antiplatelet therapy, may aid in the prevention of thromboembolic complications and pulmonary edema. Eventually, the medical therapeutic options for treatment of symptomatic severe TS are exhausted.

In the present case, an intraoperative evaluation showed that the valve leaflet's motion and the effective orifice area were severely restricted because of the commissural fusion. Although the malcoaptation between the anterior and septal leaflets could be seen clearly, the subvalvular involvement was limited, and leaflet motions were sufficiently good for valve repair. We performed commissurotomies to increase the area of the valvular orifice. We then used glutaraldehyde-treated autologous patches to augment both the anterior and septal leaflets and subsequently performed a tricuspid annuloplasty with the Kalangos Biodegradable Tricuspid Ring.

Doss at al. [2005] used similar valve-repair techniques for mitral and aortic valves and reported that glutaraldehyde-treated autologous pericardial patches are more prone to calcification. On the other hand, these investigators also stated that using autologous fresh pericardium might cause fibrotic retraction [Doss 2005]. The other concern about valve-repair techniques that use pericardium is that dehiscence of the pericardial patch may occur, although previously published reports have not described patch dehiscence. In our setting, we chose to use autologous glutaraldehyde-treated pericardial patch for the TV, as Tang at al. described for their case [Tang 2009]. We also used an oversized glutaraldehyde-treated pericardial patch, which might prevent retraction of the leaflets and maintain a safety margin for the leaflet area over the long term. In our case, we chose to use glutaraldehyde-treated pericardium instead of fresh pericardium; however, more randomized controlled trials are needed to compare these techniques.

In conclusion, the use autologous pericardial patch to augment retracted leaflets in combination with other conventional repair techniques may produce good early and midterm results in selected cases and should be considered in the setting of rheumatic TS.

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