Bioprosthetic tricuspid valve stenosis: a case series

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Background	Bioprosthetic tricuspid valve stenosis is a late sequela of tricuspid valve replacement (TVR); however, detailed information regarding its clinical picture is lacking.
Case summary	Thirty-one patients with bioprosthetic TVR (mean age: 60.5 ± 16.6 years, male/female: 11/20) were followed-up for 79.5 ± 49.1 months (14–188 months). Eleven patients developed bioprosthetic tricuspid valve stenosis (mean tricuspid gradient >5 mmHg) at a median interval of 96 months (interquartile range: 61–114 months). The mean tricuspid gradient at the time of tricuspid valve stenosis diagnosis was 10.9 ± 3.9 mmHg. Although the mid-term tricuspid valve stenosis-free survival was favourable (92.4% at 60 and 78.7% at 84 months), it had declined steeply to 31.5% by 120 months. Ten out of 11 tricuspid valve stenosis patients showed signs of right heart failure (RHF) as manifested by oedema and elevated jugular venous pressure, requiring moderate-to-high doses of diuretics. Diastolic rumble was audible in 10 patients. Five of the 11 tricuspid valve stenosis patients required redo TVR as a result of refractory RHF. Examination of the five excised bioprostheses showed pannus in four, fusion of the commissure in three, native valve attachment in two, and sclerosis in one. Detailed clinical pictures and pathology of the explanted valves in three cases that underwent surgery are presented in this case series.
Discussion	Bioprosthetic tricuspid valve stenosis is not uncommon after 8 years. Tricuspid valve replacement performed at the second surgery was associated with a higher incidence of bioprosthetic tricuspid valve stenosis.
Keywords	Tricuspid valve stenosis • Bioprosthetic valve dysfunction • Right heart failure • Case series

Learning points

- Bioprosthetic tricuspid stenosis (TS) is not rare after 8 years of tricuspid valve replacement.
- Pannus (host tissue overgrowth) at the base of the leaflets, fusion of the commissure, calcification of the free margin of the leaflets appeared to be an important causes of TS.
- Oedema, elevated jugular venous pressure and diastolic rumble were frequently seen in patient with bioprosthetic TS.

Introduction

Tricuspid stenosis (TS) is a rare condition and is usually caused by rheumatic heart disease.[1] Less common causes include carcinoid syndrome, congenital heart disease, infective endocarditis with large vegetations, and pacemaker lead insertion across the tricuspid valve.[1] Another important cause of TS is bioprosthetic tricuspid valve stenosis, which is a serious, late sequela after tricuspid valve replacement (TVR) with a bioprosthetic valve. However, the clinical picture and pathophysiology of bioprosthetic TS have been poorly defined.

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Timeline

Clinical characteristics at index TVR and timeline of bioprosthetic TS patients

Case no.	Sex	Age at index TVR	Underlying disease at index TVR	Reason for index TVR	Index TVR at 1st surgery or ≥2nd	Concomitant surgery @ index TVR	Valve at index TVR (size in mm)
1	Female	54	Rheumatic MS, S/P OMC	TR	2	MVR	CE 29
2	Female	71	Rheumatic MS	TSR	1	MVR	CE 29
3	Male	16	TOF, S/P VSD closure, and TVP	TR	2	VSD closure, PVR, RVOT plasty	CE 29
4	Female	15	Ebstein, ASD, VSD, PS	Ebstein	1	Repair of VSD/ASD	CE 27
5	Female	64	Rheumatic ASR, MS S/P AVR, and MVR	TR	2	None	CE 27
6	Female	67	Rheumatic MS, TR, TVP	TR	2	None	CE 29
7	Male	39	S/P repair of sinus of Valsalva aneurysm-RA fistula	TR	2	MVP, Maze	CE 29
8	Female	46	Traumatic TR	TR	1	None	CE 31
9	Female	74	Rheumatic MSR AR, S/P AVR, MVR, and TVP	TR	2	None	CE 27
10	Female	60	Type A aortic dissection, RV infarction, S/P ascending aortic replacement, and CABG to RCA	TR	2	None	CE 27
11	Female	57	Sarcoidosis, DCM, S/P MVP, and CRTD	TR	2	MVR	SJM 27

Case no.	Index TVR-TS (months)	Treatment for TS	Index TVR-redo TVR (months)	Mean TVG (mmHg)	Severity of TR	Main symptoms	Diastolic rumble	Elevated JVP	Oedema	Diuretics (mg)
1	180	Medical		7.1	Mild	Chest pain (due to CAD)	3/6 mid-diastole	Yes	No	F 10, S 25
2	129	Medical		12.6	Moderate	Oedema, abdominal distension	2/6 mid-diastole	Yes	Yes	F 20, S 25
3	88	Redo TVR	89	14.2	Mild	Oedema	4/6 pan-diastole	Yes	Yes	F 20
4	114	Redo TVR	128	7.6	None	Oedema, weight gain	2/6 mid-diastole	No	Yes	T 3.75, F 30, S 50
5	114	Balloon×2, Redo TVR	125	20.0	None	Oedema, dyspnoea	3/6 pan-diastole	Yes	Yes	T 15, F 60, S 50
6	77	Medical		11.2	Moderate	Oedema	3/6 pan-diastole	Yes	Yes	T15, F 60, S 50
7	61	Medical		7.1	Moderate	Oedema	3/6 pan-diastole	Yes	Yes	F 40. E 75
8	101	Redo TVR	109	10.4	Mild	Oedema, palpitation	3/6 pan-diastole	Yes	Yes	F 20
9	45	Medical		12.5	Mild	Haemolysis, oedema, dyspnoea	3/6 mid-diastole	Yes	Yes	F 40, S 25, B 4
10	96	Redo TVR	98	8.5	Severe	Oedema	NA	Yes	Yes	A 30, S 25, T 3.75
11	26	Medical		10.1	Mild	Oedema, dyspnoea	3/6 pan-diastole	Yes	Yes	T 15, F 80, S 50, B 4

Case numbers were assigned in order of the date of index TVR.

A, Azosemide (the dose in mg); ASD, atrial septal defect; ASR, aortic valve stenosis and regurgitation; AVR, aortic valve replacement; B, Benzylhydrochlorothiazide; CABG, coronary aorta bypass surgery; CAD, coronary artery disease; CE, Carpentier-Edwards; CRTD, cardiac resynchronization therapy with defibrillator; DCM, dilated cardiomyopathy; E, Eplerenone; F, Furosemide; JVP, jugular venous pulse; MS, mitral stenosis; MSR, mitral stenosis and regurgitation; MVP, mitral valve plasty; MVR, mitral valve replacement; NA, not available; No., number; OMC, open mitral commissurotomy; PS, pulmonic valve stenosis; PVR, pulmonary valve replacement; RCA, right coronary artery; RV, right ventricle; RVOT, right ventricular outflow tract; S, Spironolactone; SJM, St Jude Medical (numbers after manufacturer represent the size of the valve in mm); S/P, status post; T, Tolvaptan; TOF, Tetralogy of Fallot; TR, tricuspid regurgitation; TSR, tricuspid stenosis and regurgitation; TVG, tricuspid gradient at diagnosis of TS; TVP, tricuspid valve plasty; TVR, tricuspid valve replacement; TVR-redo TVR, interval between index TVR and redo TVR; TVR-TS, interval between index TVR and diagnosis of TS; VSD, ventricular septal defect.

Among 36 patients who underwent bioprosthetic TVR from 2000 to 2015, 31 patients survived the surgery and were followed-up for 79.5 ± 49.1 months (14–188 months). The clinical characteristics at the time of the index TVR are summarized in *Table 1*. The mean age was 60.5 years, and the male-to-female ratio was 11/20. The Carpentier–Edwards (CE) bovine pericardial valve was used in 20 patients, and the St. Jude Medical (SJM) tissue valve was used in 11 patients (after 2012). The most common aetiology was rheumatic valvular disease in 16 patients, followed by congenital heart disease in 4 (*Table 1*).

Bioprosthetic TS was defined as when the mean tricuspid valve gradient (TVG) was greater than 5 mmHg (measurements from at least five cardiac cycles were averaged).[1] During follow-up, 11 patients developed bioprosthetic TS at a median interval of 96 months (interquartile range: 61–114 months). The clinical picture and timeline of the 11 bioprosthetic TS patients are summarized in timeline. The mean TVG at the time of diagnosis of TS was 10.9 ± 3.9 mmHg. In addition to TS, one patient had severe tricuspid regurgitation (TR) and three had moderate TR. Among 20 patients without TS, severity of TR was moderate in four and mild in four. There was no patient with severe TR requiring redo TVR in patients without TS. Although the mid-term TS-free survival was favourable (92.4% at 60 months and 78.7% at 84 months), it had declined steeply to 31.5% by 120 months (*Figure 1*). TVR at the second (or later) surgery was the only significant risk factor for bioprosthetic TS as assessed by the log-rank test ($P = 0.047$), whereas age, sex, aetiology, valve manufacturer, concomitant surgery, rhythm, pulmonary hypertension, and kidney function at the index TVR were not (Supplementary material online, *Table S1* and *Figure S1*). Ten out of 11 TS patients showed signs of right heart failure (RHF) as manifested by oedema and elevated jugular venous pressure (JVP), requiring moderate-to-high doses of diuretics. Diastolic rumble was audible in 10 patients, and five of 11 TS patients required redo TVR as a result of refractory RHF. The definition and mechanism for valve dysfunction in the five cases requiring redo TVR was summarized in Supplementary material online, *Table S2*. Detailed analysis of the three explanted valves was available, and the pathophysiology of the prosthetic valve TS is discussed in this case series.

Case presentation

Case 5

A 74-year-old woman was referred to us for refractory heart failure and perioperative management of a left femoral neck fracture. Her past history was remarkable for mitral and aortic valve replacement (Hall–Kaster mechanical valves) for severe rheumatic mitral stenosis and aortic regurgitation at the age of 49 years. She developed RHF because of TR and had isolated TVR with a 27-mm CE bovine pericardial valve at the age of 64 years (15 years after the first surgery). She had been stable for 10 years; however, she developed refractory leg oedema at 74 years of age. She was treated medically in another hospital. Three months before the referral, she suffered a left femoral neck fracture in a fall; nevertheless, surgical treatment was declined in another hospital because of high operative risks. However, she
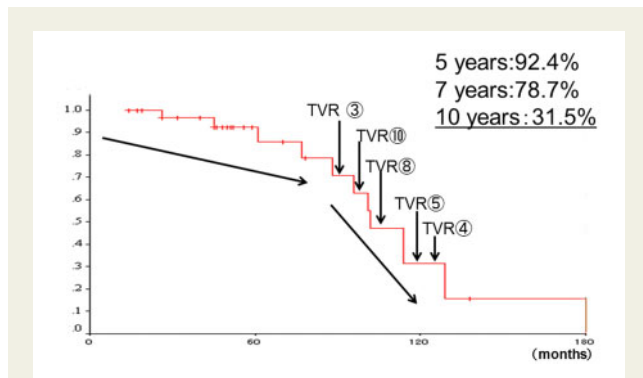
Table 1 Clinical characteristics at index TVR (n = 31)

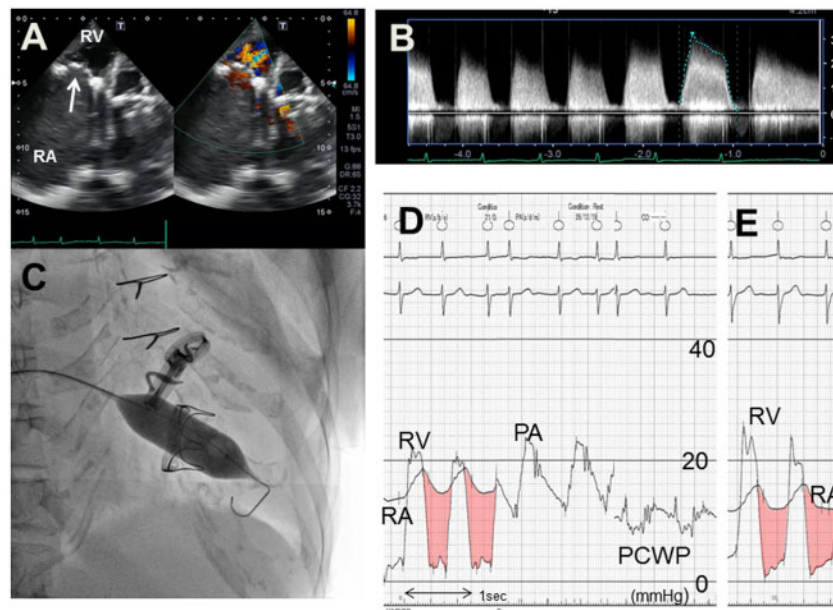|	
Age	
Mean ± SD (years)	60.5 ± 16.6
Sex	
Male (*n*)	11
Female (*n*)	20
Manufacturer	
Carpentier-Edwards	20
St Jude Medical	11 (after 2012)
Aetiology	
Rheumatic	16
Non-rheumatic	15
Congenital	4
Degenerative	2
Secondary	2
Trauma	1
Infective endocarditis	1
Sarcoidosis	1
Right ventricular infarction	1
Unknown	3
Isolated TR	3
With other valvular disease	28
Operative procedures	
Isolated TVR	12
TVR with concomitant procedures	19
Indication of TVR	
TR	28
TSR	2
Epstein	1
Timing of TVR	
1st	13
≥2nd	18
Rhythm	
Normal sinus rhythm	6
Atrial fibrillation	24 (1 with VVI)
DDD (CRTD)	1
Anticoagulation with warfarin	
Yes	30
No	1
Left ventricular function	
Normal	25
Reduced	6
Pulmonary hypertension (>25 mmHg)	
Yes	19
No	12
eGFR	
≥60	16
<60	15

CRTD, cardiac resynchronization therapy with defibrillator; DDD, DDD pacing mode; eGFR, estimated glomerular filtration rate; SD, standard deviation; TR, tricuspid regurgitation; TSR, tricuspid stenosis and regurgitation; TVR, tricuspid valve replacement; VVI, pacing with VVI mode.

strongly desired surgical treatment and was, therefore, referred to us. The initial evaluation revealed an emaciated patient with severe leg oedema and elevated JVP up to her jaw level in the seated

Figure 1 Tricuspid stenosis-free survival. The numbers in the circles represent the case numbers in timeline. Arrows indicate the timing of tricuspid valve replacement. TVR: tricuspid valve replacement.

position. Pan-diastolic rumble was audible at the 4th left sternal border. Laboratory data revealed severe hypoalbuminaemia of 2.8 g/dL. Evaluation revealed protein-losing gastroenteropathy because of high venous pressure. Echocardiography revealed severe bioprosthetic TS with TVG as high as 20 mmHg (*Figure 2A* and *B*). Because of poor nutrition, redo TVR was deemed to be too risky. Therefore, the patient was treated with the less invasive balloon tricuspid valvuloplasty before the hip replacement surgery, and we expected improvements in her haemodynamic and nutritional condition (*Figure 2C*). The right atrial pressure decreased from 18 to 13 mmHg, and her general condition improved (*Figure 2D* and *E*). After the balloon valvuloplasty, she underwent successful left hip replacement surgery without complication. However, she developed refractory RHF because of recurrent worsening of TS 8 months later. After the second balloon valvuloplasty (requested by the cardiac surgeons as a bridge, *Figure 3*), redo TVR with a SJM tissue valve was finally performed. Examination of the explanted valve revealed the following findings (*Figure 6A*). X-ray examination showed moderate calcification along the free margin of the leaflets. Heavy pannus (host tissue overgrowth) was observed in both atrial and ventricular aspects. All the commissures appeared to have been fused by the pannus, and the fused commissures were partially split by the balloon valvuloplasty.

Figure 2 An echocardiography and cardiac catheterization in Case 5. (*A*) An echocardiogram showing the thickened leaflets of the bioprosthetic tricuspid valve (white arrow) with restricted opening and turbulent flow across the tricuspid valve. (*B*) Continuous Doppler examination showing accelerated flow velocity and a markedly high mean tricuspid valve gradient of 14–22 mmHg. (*C*) Balloon valvuloplasty with a 20-mm Hopkinton Z-MED II balloon, through a 12-Fr sheath. To measure simultaneous right ventricle and right atrial pressures, an 8-Fr Mullins sheath was advanced to the right atrium and a 6-Fr wedge catheter was inserted through the Mullins sheath. Right atrial pressure was obtained from the tip of the Mullins sheath, and right ventricular pressure was obtained from an end-hole of the wedge catheter simultaneously. (*D*) Before the balloon valvuloplasty, right atrial pressure was markedly elevated to 18 mmHg, and tricuspid valve gradient (shaded area) was as high as 15 mmHg. Pulmonary capillary wedge pressure was 11 mmHg, and the pulmonary arterial pressure was 22/13 (17) mmHg, indicating pure right heart failure caused by severe tricuspid stenosis. (*E*) After the balloon valvuloplasty, right atrial pressure and tricuspid valve gradient decreased to 13 and 11 mmHg, respectively, indicating the partial effectiveness of the balloon valvuloplasty. PA, pulmonary artery; PCWP, pulmonary capillary wedge pressure; RA, right atrium; RV, right ventricle; TVG, tricuspid valve gradient.
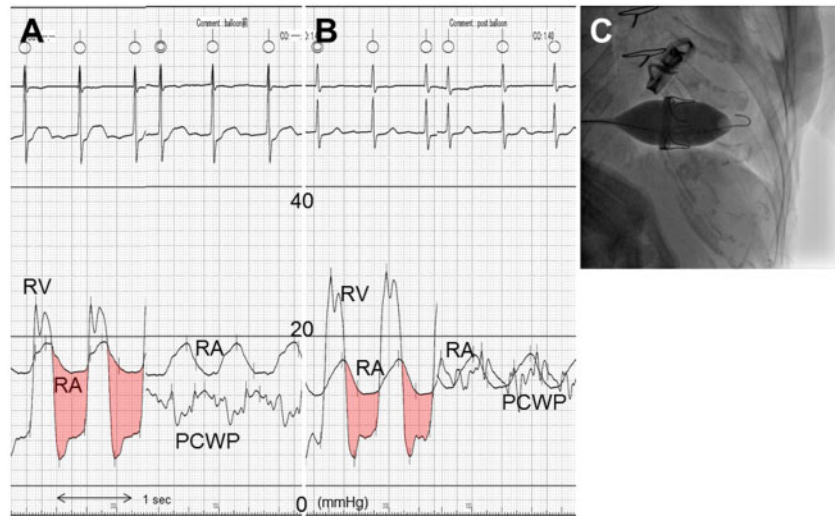
Figure 3 Pressure tracings before and after the second tricuspid balloon valvuloplasty in Case 5. (A) Before the valvuloplasty, the intracardiac pressures were as follows: RA = 17 mmHg, PCWP = 11 mmHg, and TVG (shaded area) = 12 mmHg. (B and C) After the valvuloplasty with 25-mm Hopkinton Z-Med II balloon, right atrial pressure had decreased to 14 mmHg, pulmonary capillary wedge pressure had increased to 14 mmHg, and tricuspid valve gradient had decreased to 10 mmHg. PA, pulmonary artery; PCWP, pulmonary capillary wedge pressure; RA, right atrium; RV, right ventricle; TVG, tricuspid valve gradient.
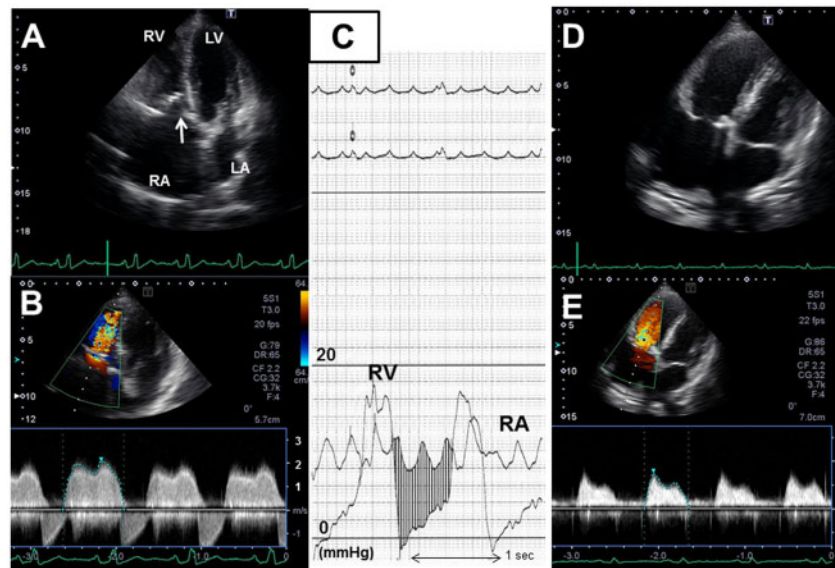
Figure 4 Echocardiographic findings before (A, B) and after redo tricuspid valve replacement (D, E), and cardiac catheterization before redo tricuspid valve replacement (C) in Case 8. (A) A four-chamber view showing thickened prosthetic tricuspid valve leaflets with restricted opening (white arrow). (B) A colour Doppler showing turbulent flow across the tricuspid valve; the mean TVG increased up to 10.4 mmHg as measured by continuous-wave Doppler. (C) Simultaneous right ventricular and right atrial pressure tracing during cardiac catheterization revealed significantly increased tricuspid valve gradient (shaded area) up to 8 mmHg. Note that the rhythm at the time of catheterization was atrial flutter. Occasional Cannon A waves were clearly seen when the F-wave coincided with ventricular systole. There was no pulmonary hypertension, and pulmonary capillary wedge pressure was lower than right atrial pressure. CO = 3.45 L/min (CI = 2.16 L/min/m^3), RA: 12 mmHg, RV: 18/0–5 mmHg, PA: 17/8 (12) mmHg, PCWP = 8 mmHg, aorta: 85/50 (64) mmHg, left ventricle: 85/3–9 mmHg, TVG = 8 mmHg. (D) After tricuspid valve replacement, the leaflets opened well, and the size of the right atrium decreased. (E) The flow pattern across the tricuspid valve normalized and tricuspid valve gradient decreased to 3.5 mmHg. PA, pulmonary artery; PCWP, pulmonary capillary wedge pressure; RA, right atrium; RV, right ventricle; TVG, tricuspid valve gradient.
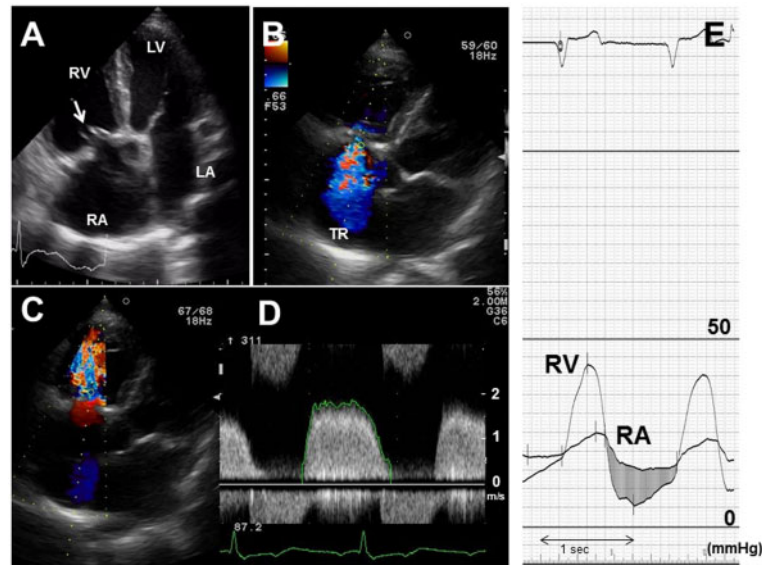
Figure 5 Echocardiography and cardiac catheterization in Case 10. (*A*) A four-chamber view from the apex, showing thickened leaflets and restricted opening of the leaflets of the tricuspid prosthesis. (*B*) Severe tricuspid regurgitation was also noted. (*C* and *D*) A colour Doppler showing turbulent diastolic flow across the tricuspid valve (*C*); the mean tricuspid valve gradient was 7.6 mmHg as measured by continuous wave Doppler (*D*). (*E*) Tricuspid valve gradient (shaded area) at the time of cardiac catheterization was 7.1 mmHg. PA, pulmonary artery; PCWP, pulmonary capillary wedge pressure; RA, right atrium; RV, right ventricle; TR, tricuspid regurgitation; TVG, tricuspid valve gradient.
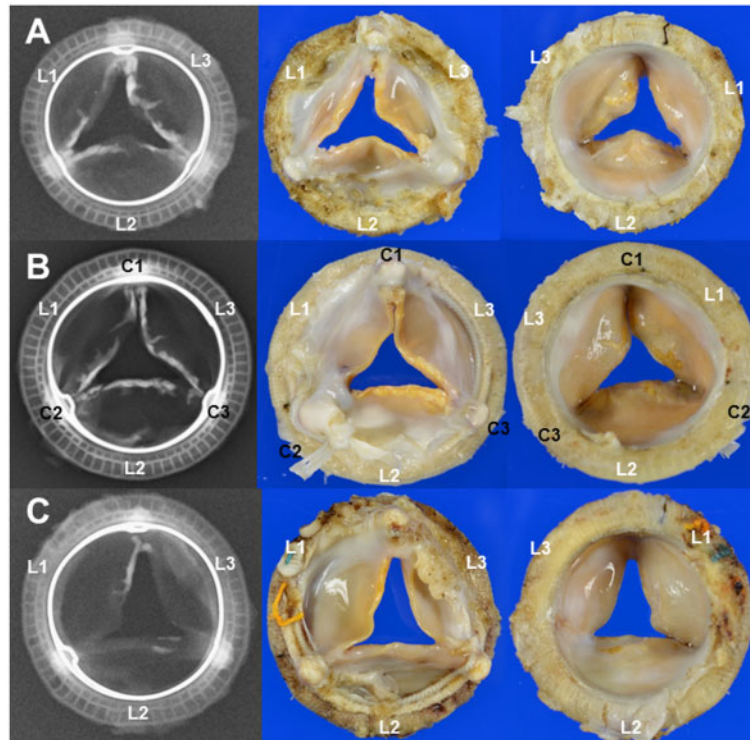
Case 8

The patient underwent TVR with a 31-mm CE bovine pericardial valve at the age of 46 years for refractory RHF because of traumatic TR. RHF improved after the surgery, and her condition remained stable until she was 55 years old, which was 8.4 years (101 months) after the first TVR, when she started to develop palpitation, fatigue, shortness of breath, and swelling of the legs. Her vital signs at presentation were as follows: blood pressure of 110/75 mmHg, heart rate of 75 b.p.m., and respiratory rate of 18 b.p.m. Her JVP was elevated up to the height of 4–5 cm above the clavicle in a seated position, with a prominent A wave, and Kussmaul's sign was also noted. Auscultation of the heart revealed Grade 3 pan-diastolic rumble best heard at the 4th left sternal border. Lung sounds were normal; however, there was moderate leg oedema. A chest roentgenogram showed cardiomegaly without lung congestion. A 12-lead electrocardiogram (ECG) showed normal sinus rhythm, first-degree atrioventricular (AV) block, and right bundle branch block. An echocardiogram showed thickened leaflets of the bioprosthetic tricuspid valve, with increased TVG up to 10 mmHg (*Figure 4A* and *B*). Mild TR was also noted. The size and function of the left ventricle was normal, but the right ventricle and the right atrium were enlarged (*Figure 4A*). The aortic, mitral, and pulmonic valves appeared to be normal. Prosthetic TS was diagnosed, and the patient was started on diuretics. Cardiac catheterization and redo TVR were recommended; however, the patient did not agree. She was then treated medically, but her RHF finally worsened 8 months later. An ECG revealed atrial flutter with AV block (rate was 50/min). She was hospitalized and treated with intravenous dobutamine and diuretics. Cardiac catheterization showed an elevated mean TVG of 8 mmHg, confirming the diagnosis of TS

(*Figure 4C*). Redo TVR with an SJM tissue valve was performed 109 months after the first TVR. X-ray examination of the explanted tricuspid bioprosthesis revealed moderate calcification at the margin of all three leaflets (*Figure 6B*). Visual inspection showed moderate-to-heavy pannus in both atrial and ventricular aspects in all three leaflets. Pannus was more prominent in the ventricular aspect and encroached onto the leaflets, with a distance of 6–15 mm. Additionally, the leaflets were fused by 3–5 mm. The remnants of the chordae were attached to the sewing ring of the valve. There was no degeneration of the valve prosthesis itself. The prosthetic tricuspid valve stenosis was caused by the pannus formation, fusion of the commissure and the calcification at the leaflets' margin. After the redo TVR, the signs of RHF improved and TVG decreased to 3.5 mmHg (*Figure 4D* and *E*).

Case 10

A 69-year-old woman developed refractory oedema. Her past medical history was remarkable for Type A aortic dissection complicated by right coronary artery (RCA) malperfusion, resulting in inferior wall and right ventricular infarction at the age of 60 years. She underwent emergency ascending aortic replacement and coronary artery bypass surgery to the RCA using a saphenous vein graft. She developed signs of RHF, including oedema and elevated JVP 10 months after the surgery. Echocardiography and cardiac catheterization showed severe TR and hypokinesis of the anterior wall of the right ventricle. Left ventricular systolic function was preserved (ejection fraction = 59%), and there was no elevation of the left sided pressures. She was diagnosed with severe TR because of right ventricular infarction, and TVR with a 27-mm CE bovine

Figure 6 X-ray examination and inspection of the explanted valve in Case 5 (*A*), Case 8 (*B*), and Case 10 (*C*). Left: X-ray, Centre: Ventricular aspects, Right: Atrial aspects. (*C*) Commissure, L: Leaflet. (*A*) X-ray examination showing moderate calcification along the free margin of the leaflets. Heavy pannus was observed in both atrial and ventricular aspects. All the commissures appeared to have been fused by the pannus, and the fused commissures partially split after the balloon valvuloplasty. (*B*) X-ray showing moderate calcification along the free margin of the leaflets. Heavy pannus was observed in both atrial and ventricular aspects. Commissures 2 and 3 (C2, 3) were fused by pannus. A remnant of the native valve was attached to the sewing ring at C2. (*C*) X-ray examination showing only mild calcification. Heavy pannus was observed in both atrial and ventricular aspects. All the commissures were fused by pannus. Leaflets 2 and 3 were rolled towards the ventricular side.

pericardial valve was performed. After the TVR, she was stable for 8 years. However, she developed refractory leg oedema at the age of 69 years. Echocardiography and cardiac catheterization showed both TR and TS without pulmonary hypertension (*Figure 5A–D*). Tricuspid valve gradient was 7.1 mmHg, and the estimated tricuspid valve area was 0.74 cm^2. The saphenous vein graft to RCA was patent, and left ventricular function was preserved. The patient underwent redo TVR with a 31-mm SJM tissue valve. X-ray examination of the excised valve showed only mild calcification (*Figure 6C*). Heavy pannus was observed in both atrial and ventricular aspects and caused fusion in all the commissures and stiffness of the base of the leaflets. Leaflets 2 and 3 were deformed and were rolled towards the ventricular side. These changes resulted in both severe TR (*Figure 5B*) and TS.

Discussion

Conflicting long-term results have been reported after TVR with a bioprosthetic valve. Previous studies reported 10-year survival rates between 33% and 68.7%, indicating unsatisfactory long-term results of TVR using bioprosthesis,[2–9] unlike those of aortic and mitral valve replacement.[10] Preoperative liver dysfunction, residual pulmonary hypertension, and decreased right ventricular ejection are associated with poor long-term outcomes.[3,7,8] In our case series, although the long-term survival was favourable (70.5% at 10 years), TS-free survival declined steeply after 8 years (*Figure 1*). Five out of 11 TS patients underwent redo TVR after 8 years. The incidence of bioprosthetic TS after TVR has been poorly defined. Among 84 patients who survived the initial TVR, Nakano *et al.*[4] reported that 12 patients required redo TVR for bioprosthetic dysfunction, nine of which were TS. Only two cases showed structural valve deterioration such as significant calcification or degeneration after evaluation of the explanted valves. Non-structural dysfunction such as pannus formation and native valve attachment were more common causes of the dysfunction and were seen in seven and five cases, respectively.[4] They also reported that subclinical valve dysfunction became increasingly common after 5 years with careful evaluation using echocardiography and redo TVR rates increased significantly after 8 years.[4,5] They emphasized the pannus formation on the ventricular side of the cusp as an important cause of TS. In our cases, the stiffened base of the leaflets caused by the pannus was an important cause of stenosis in all

three explanted valves. Additionally, pannus formation also resulted in commissure fusion, leading to restricted opening of the valves. One patient (Case 10) developed both TS and severe TR. The severity of TR was below moderate in the remaining 10 cases (timeline). The deformity of the tip of the leaflets as well as the fixed semi-open position caused by the heavy fusion of the commissure can result in both stenosis and regurgitation, as seen in Case 10 in the present series. A remnant of the native valve was attached in the sewing ring in two of our cases, but its contribution to valve stenosis was not clear.

The interval between the initial TVR and TS development appeared to be somewhat early in our series compared with previous reports.[2] However, Nakano et al.[5] reported that the mean interval from the initial TVR to the redo TVR was 85.9 months, which was shorter than in our study (mean: 93.7 months, median: 96 months). They suggested that subclinical TS may be more common than previously thought.[4,5] The diagnosis of bioprosthetic TS in our case series was driven by the clinical symptoms of RHF. However, early diagnosis can be made with serial echocardiographic examination even before overt RHF develops.

Regarding treatment, there are few case reports of successful percutaneous balloon dilatation for prosthetic TS;[11] however, the long-term results of percutaneous therapy have been poorly defined. Balloon valvuloplasty may be temporarily and partially effective by splitting the fused commissure. However, it may not be effective on a stiffened valve base caused by heavy pannus or valve calcification itself, as observed in Case 5. Redo TVR is usually necessary in the majority of the cases for definite treatment.[5] In future, percutaneous valve-in-valve insertion or other transcatheter interventional procedures may become feasible.[12]

In conclusion, pannus formation (host tissue overgrowth) is an important cause of tricuspid valve dysfunction in addition to structural valve deterioration such as calcification.

Lead author biography
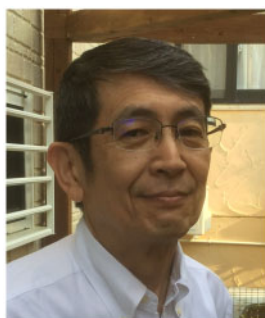
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Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

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