Original Article

Reoperation for Tricuspid Regurgitation after Total Correction of Tetralogy of Fallot

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Background: The aim of this study is to review the outcome of reoperation for severe tricuspid regurgitation after repair of tetralogy of Fallot .
Methods: Between 1972 and 2000, 12 patients underwent reoperation on the tricuspid valve after total correction of tetralogy of Fallot. The mean age at the time of reoperation was 17 years (range, 1 to 39 years). The mean interval between the initial correction and the reoperation was 7.8 years (range, 10 days to 19 years). The functional class was New York Heart Association class II in 2 patients and class III or IV in 10. Six patients underwent tricuspid valve repair, and the others underwent tricuspid valve replacement.
Results: Hospital mortality was 16.7% (2/12). Three patients (30%, 3/10) required a second reoperation 1.6, 9.2, and 15.6 years after the most recent reoperation with no deaths. The reasons for second reoperation were failure of the tricuspid valve repair in two and a thrombosed valve in one. There were two late deaths. Mean overall event-free actuarial survival at 10 years was 46.3%.
Conclusion: Reoperation for severe tricuspid regurgitation after total correction of tetralogy of Fallot was associated with a high operative mortality and disappointing long-term results. Tricuspid regurgitation after corrective surgery for tetralogy of Fallot must be diagnosed promptly and cured, as tolerance is poor because of postoperative right ventricular insufficiency. (Ann Thorac Cardiovasc Surg 2002; 8: 199–203)

Key words: tetralogy of Fallot, tricuspid regurgitation, reoperation

Introduction

Functional results have been excellent following correction of tetralogy of Fallot (TOF).[1-3] However, tricuspid regurgitation can develop as an early or late complication.[4] Tricuspid regurgitation may occur early as a result of tricuspid valve distortion as a consequence of ventricular septal defect or late repair, as a sequela of the progression of right heart failure. The management and indications for tricuspid valve surgery after correction of TOF remains controversial, particularly as to the timing of surgical treatment. The outcome of tricuspid valve surgery in this setting has not been well studied and followed.

This study reviews the early and late results of surgery for tricuspid regurgitation in patients who present with severe tricuspid regurgitation after correction of TOF.

Methods

Between 1972 and 2000, 12 (2.4%, 9 males and 3 females) of the 507 patients who had corrective surgery for TOF at the Sapporo Medical University (between 1955 and 1999) required reoperation for severe tricuspid regurgitation. The mean age at the time of reoperation was 17.3±11.1 years (range, 1 to 39 years). The mean interval between the initial correction and the reoperation was 7.8±7.2 years (range, 10 days to 19 years). The diagnosis

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Table 1. Clinical data

Patient	Sex	Age (yr) at TV operation	Age (yr) at initial repair	NYHA	CVP (mmHg)	Cause of tricuspid regurgitation
1	F	21	15	IV	20	Fixation of the tricuspid septal leaflet
2	F	23	4	IV	11	Chordal rupture
3	M	10	10	IV	18	Fixation of the tricuspid septal leaflet
4	M	16	6	IV	17	Fixation of the tricuspid septal leaflet
5	F	5	3	III	15	Chordal rupture
6	M	39	34	III	14	Annular dilatation
7	M	31	12	III	22	Annular dilatation
8	M	6	5	III	8	Fixation of the tricuspid septal leaflet
9	M	1	1	IV	13	Fixation of the tricuspid septal leaflet
10	M	21	11	III	18	Annular dilatation
11	M	18	3	II	8	Fixation of the tricuspid septal leaflet
12	M	18	6	II	8	Fixation of the tricuspid septal leaflet

TV: tricuspid valve, NYHA: New York Heart Association, CVP: central venous pressure

of tricuspid regurgitation was made by echocardiographic and cardiac catheterization data. Patients with tricuspid regurgitation were considered for reoperation when their symptomatic status deteriorated or right heart dysfunction, including the onset of ventricular or supraventricular arrhythmia, was suspected.

Four patients had previously undergone a palliative operation: the Blalock-Taussig shunt in 4, the Waterston shunt in 2, and the Brock procedure in 1 patient. The functional class was New York Heart Association (NYHA) class II in 2 patients and class III or IV in 10. The mean central venous pressure was 14.3±4.8 mmHg (range, 8 to 22 mmHg). Five patients had associated residual ventricular septal defect, 5 had severe or mild pulmonary valve regurgitation, 2 had residual right ventricular outflow tract (RVOT) obstruction, 2 had pulmonary artery stenosis, 2 had atrial septal defect, 1 had mitral valve insufficiency, and 2 had left ventricle-right atrium communication. RVOT obstruction was considered significant when the systolic pressure gradient was more than 50 mmHg. Three patients underwent reoperation for isolated tricuspid regurgitation due to annular dilatation (Table 1).

Standard hypothermic (28 to 32°C) cardiopulmonary bypass with bicaval cannulation was performed in all patients. Cold blood cardioplegia was used for myocardial protection with topical cold saline solution infused into the pericardium. The tricuspid valve was approached through the right atrium. The tricuspid valve apparatus was inspected to verify that the lesions were concordant with the clinical findings and to allow the surgeon to decide the appropriate type of repair or replacement. The annulus was dilated in all patients, and this dilatation was solely responsible for regurgitation in 3 patients. The other lesions contributing to the tricuspid regurgitation were distortion and fixation of the septal leaflet (7 patients) and chordal rupture of the anterior leaflet (2 patients).

Six patients underwent tricuspid valve repair. The surgical treatments included repair of a ruptured chordae with autopericardium, removal of one stitch on the septal leaflet, Kay's annuloplasty of the anteroseptal commissure and suture of the anteroposterior commissure, Kay's annuloplasty of the anteroseptal commissure, partial annular plication (Modified Davila's annuloplasty) and suture of the anteroseptal commissure, Carpentier ring (Baxter Healthcare, Irvine, CA, USA) annuloplasty and suture of anteroseptal commissure. The other 6 patients underwent tricuspid valve replacement. A Hancock II bioprosthesis (Medtronic Heart Valve, Inc., Santa Ana, USA) was selected in 3 patients (33-, 31-, and 29-mm valves), a Carpentier-Edwards bioprosthesis (Baxter Healthcare, Irvine, CA, USA) in 1 (31-mm valve), a St. Jude Medical prosthesis (St. Jude Medical, Inc., St. Paul, USA) in 1 (33-mm valve), and a Wada-Cutter prosthesis in 1 (34-mm valve). Concomitant procedures were performed in 9 patients. These operations included repair of a ventricular septal defect in 5 patients, pulmonary valve replacement in 2, repair of an atrial septal defect in 2, closure of a left ventricle-right atrium fistula in 2, angioplasty of a distal pulmonary artery with a pericardial patch in 2, reconstruction of the RVOT with a monocuspid patch in 1, pulmonary valvotomy in 1, and mitral valve annuloplasty in 1 (Table 2).

Results

The hospital mortality rate was 16.7% (2/12). One pa-

Table 2. Profile of the 12 patients who required reoperation for severe tricuspid regurgitation after correction of TOF

Patient	TV operation	Concomitant procedure	Outcome	Late follow-up
1	TVR (34-Wada-Cutter)	Re VSD closure + LV-RA fistula repair	Re TVR (29-SJM)	28 yr
2	Repair of the ruptured chordae	Re VSD closure + PV valvotomy	Alive	22 yr
3	Removal of one stitch on the septal leaflet	Re VSD closure + ASD closure	TVR (31-Hancock II)	17 yr
4	TVR (29-Hancock II)	Re VSD closure + PVR	Alive	13 yr
5	Kay's annuloplasty + commissuroplasty	–	Alive	11 yr
6	TVR (31-Carpentier-Edwards)	Re Reconstruction of RVOT	Alive	11 yr
7	TVR (33-Hancock II)	LV-RA fistula repair	Late death	–
8	Kay's annuloplasty	Re VSD closure + ASD closure + distal PA plasty	Late death	–
9	Modified Davila's annuloplasty + commissuroplasty	–	Re TV repair	3 yr
10	TVR (31-Hancock II)	MAP (Kay's annuloplasty)	Died 2 days postoperatively	–
11	TVR (33-SJM)	PVR + distal PA plasty	Died 16 days postoperatively	–
12	Carpentier ring annuloplasty + commissuroplasty	–	Alive	4 m

TOF: tetralogy of Fallot, TV: tricuspid valve, TVR: tricuspid valve replacement, SJM: St. Jude Medical, VSD: ventricular septal defect, LV: left ventricle, RA: right atrium, PV: pulmonary valve, ASD: atrial ventricular defect, PVR: pulmonary valve replacement, RVOT: right ventricular outflow tract, MAP: mitral valve annuloplasty, PA: pulmonary artery

tient had difficulty coming off cardiopulmonary bypass, received percutaneous cardiopulmonary support, and died 2 days later. The other patient developed postoperative mediastinitis and died on the 16th postoperative day. Postoperative complications induced chronic complete atrioventricular block in 2 patients and a vegetative state following sudden cardiac arrest postoperatively in another.

The 10 surviving patients were followed for 4 months to 28 years (mean, 11.1 years) after the reoperation. Three patients (30%) required a second reoperation on the tricuspid valve 1.6, 9.2, and 15.6 years after the most recent reoperation with no deaths. The reasons for second reoperation were failure of the tricuspid valve repair in 2 and thrombosis of the mechanical valve in 1. One of the two patients who had underdone a repair initially underwent repeat tricuspid valve repair, artificial chordae implantation on the anterior leaflet and suture of the anteroseptal and anteroposterior commissures, and the other patient underwent tricuspid valve replacement with Hancock II bioprosthesis (31-mm valve) because the valve was torn and had degenerated at the septal and anterior cusps. In the final patient, the Wada-Cutter prosthesis was replaced with a St. Jude Medical prosthesis (29-mm valve). There were 2 late deaths. One occurred 1.5 years after reoperation when the patient was in a vegetative state. The other late death which occurred 2 years after reoperation, was sudden, and happened at home. The cause remains unknown, although we suspect it was arrhythmia-related.

Five patients were alive without tricuspid reoperation at follow up, and 7 reached an end-point (3 reoperations and 4 deaths). The mean overall event-free actuarial survival at 10 years was 46.3% (Fig. 1). The frequency of late death and reoperation was determined using the Kaplan-Meier method.

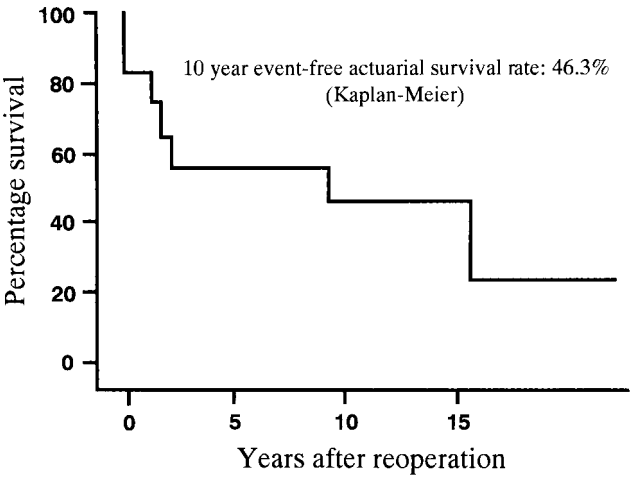

Fig. 1. Kaplan-Meier event-free actuarial analysis for patients after reoperation.

Discussion

Increasing numbers of adults with repaired TOF have late complications, such as arrhythmias, heart failure, and sudden death. These complications result from tricuspid and pulmonary regurgitation, right ventricular dysfunction, ventricular septal patch leaks, and stenosis of the

right ventricular outflow tract and branch pulmonary arteries.[5,6] Reoperation for recurrent or residual intracardiac defects following initial correction of TOF yields excellent hemodynamic and functional results over both short- and long-term follow-up.[7-9] Oechslin and coworkers[7] reported no perioperative mortality at reoperation after correction of TOF, and the actuarial 10-year survival was 92±6%. Results in this study (reoperation for severe tricuspid regurgitation) are less encouraging. There were 2 perioperative deaths, and the mean overall event-free actuarial survival at 10 years was 46.3%. Although tricuspid regurgitation is a minor complication after the initial correction of TOF, it becomes serious when liver dysfunction and right ventricular dysfunction occur.

Surgical criteria for tricuspid valve surgery after correction of TOF are controversial.[10] Patients with congestive heart failure and correctable residual lesions generally are considered good candidates. The most common causes of tricuspid regurgitation are intraoperative injury to the tricuspid valve or its attachments, distortion of the valve as a result of anchoring the ventricular septal defect patch to the septal leaflet, and less frequently, annual dilation due to myocardial distension or marked volume overload. Fixation of the septal leaflet interferes with the bulging of the leaflet at the end of ventricular diastole. Restraining the septal leaflet decreases the right ventricular end-diastolic capacity, resulting in an elevation of the right ventricular end-diastolic pressure. Furthermore, this restraint has been proposed as one etiology of tricuspid regurgitation in the late period.[3] A number of patients in this study developed severe tricuspid regurgitation due to technical problems with the closure of ventricular septal defect. In this tricuspid regurgitation, the deterioration of right ventricular function usually is due to the volume overload caused by the tricuspid regurgitation itself over time. Chronically elevated central venous pressure and volume overload can result in hepatic dysfunction and failure, as in one patient (case 7). Kurosawa and associates[11] noted that closure of the ventricular septal defect in the conotruncal repair for TOF should use a membranous flap instead of the septal leaflet of the tricuspid valve. This technique is advantageous because it leaves the septal leaflet intact, a very important consideration during the initial correction of TOF.

On the other hand, secondary tricuspid regurgitation usually is caused by right ventricular failure. Right ventricular failure may produce passive dilatation of the valve annulus and cause abnormal tension on the chordae, spontaneous chordal rupture and impairing coaptation of the leaflets. Generally, tricuspid annuloplasty is performed in cases of secondary tricuspid regurgitation.[12,13] However, tricuspid valve replacement is required in patients with severe tricuspid regurgitation and a markedly dilated tricuspid annulus with serious cardiac failure. This result creates the impression that this tricuspid regurgitation from right ventricular failure carries a worse prognosis than does malformation of the tricuspid valve.

In the absence of severe structural damage to the native valve, repair or annuroplasty should always be attempted. Prosthetic ring annuloplasties appear to achieve competence without significantly altering valve anatomy. However, the use of prosthetic rings poses unique problems in children. Growing children require a longitudinal management strategy to account for growth that is not a consideration in adult cardiac surgery. Therefore, every effort should be made to repair the native valve in children, even when it is severely malformed.

Patients with mild tricuspid regurgitation are at risk of developing congestive heart failure. Patients must be followed closely. Tricuspid regurgitation after corrective surgery for TOF must be diagnosed and cured promptly, as tolerance is poor because of postoperative right ventricular insufficiency. This study found that reoperation for tricuspid regurgitation after total correction of TOF is associated with a high operative mortality rate and disappointing long-term results. The report by Oechslin and coworkers[7] was in fact no perioperative mortality at reoperation after correction of TOF, and only three of 60 patients had severe tricuspid regurgitation. Furthermore, the majority of the patients (62%) in that series were in Class I or II, compared to only 2 of 12 in this series. Tricuspid regurgitation that develops after repair of TOF must be managed by reoperation on the tricuspid valve and correction of other residual lesions before severe regurgitation develops. We recommend that every patient who has evidence of tricuspid regurgitation receive a full evaluation, looking for correctable pathology.

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