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Outcome of Adults with Repaired Tetralogy of Fallot

Outcome of adult patients with repaired tetralogy of Fallot (TOF) was studied with emphasis on postrepair problems. A retrospective review of clinical, echocardiographic, catheterization, and surgical data was performed for 48 patients who underwent corrective repair of TOF after 15 years of age. All patients survived total repair and have been followed up from 3 months to 11 years (median 4.6 years). Postoperatively, 81.3% of patients were in functional class I and 85.4% had normal right ventricular function. One patient (2.1%) died during follow-up. There were 6 reoperations (12.5%) in 5 patients. The indications for reoperation included residual ventricular septal defect (VSD) (n=1), right ventricular outflow obstruction with VSD (n=4), and pulmonary regurgitation (n=1). The 10-year actuarial survival rate was 97.1%, and the 10-year freedom from reoperation was 81.3%. Aortic regurgitation was seen preoperatively in 6 patients (12.5%) and there were 2 newly developed aortic regurgitations after operation, one of which was caused by infective endocarditis. Corrective repair of TOF can be recommended in this patient group since the survival rate, postrepair functional status and hemodynamics are acceptable. Continued close follow-up, however, is essential for early identification and correction of postrepair problems.

Key Words: *Tetralogy of Fallot; Reoperation; Cardiovascular Abnormalities; Heart Defects, Congenital*

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INTRODUCTION

Since corrective surgery for tetralogy of Fallot (TOF) has been available for over 40 years (1), excellent operative results and the long-term survival of patients with TOF have been achieved by the technical development of myocardial protection and postoperative management. Recently, the 32-year actuarial survival rate is reported to be 86%, compared with an expected rate of 96% in normal population (2). Thus, this disease entity is no longer limited to pediatric patients but is extended to the patients who have survived into adulthood. Nonetheless, in addition to scanty data regarding grown-up congenital heart (GUCH) disease, there are fewer centers having GUCH clinic in our country than in western countries (3).

Accordingly, we followed up a total of 48 adult patients with repaired TOF and studied retrospectively their clinical outcome with emphasis on postrepair residual problems, and thereby intend to provide our own basic data for grown-up congenital heart disease.

MATERIALS AND METHODS

Patients

The patients consisted of 48 patients (30 males and 18 females) who underwent corrective repair of TOF after 15 years of age at Sejong Heart Institute between August 1984 and July 1994. Patients were excluded who had TOF with atrioventricular septal defect, pulmonary atresia, absent pulmonary valve, or associated with complex heart disease. Their ages at the time of corrective repair ranged from 15.1 to 43.3 years with a median of 21.2 years (Table 1).

Methods

Clinical, echocardiographic, catheterization, and surgical data for 48 patients were reviewed retrospectively. Reoperation included only surgical treatment, and excluded were balloon angioplasty for branch pulmonary artery stenosis after initial repair and valve replacement

Table 1. Age and sex distribution in patients at the time of repair

Age (yrs)	Male (No.)	Female (No.)	Total (No.)
15-20	12	9	21
21-30	8	6	14
31-40	6	3	9
41≤	4	0	4
Total	30	18	48

for severe regurgitation caused by infective endocarditis in the reoperation category.

Using echocardiography, single pass or gated blood pool radionuclide study and catheterization, postrepair right ventricular (RV) function was graded as good if right ventricular ejection fraction (RVEF) is greater than 45% and right ventricular end-diastolic pressure (RVEDP) is less than 10 mmHg; fair, if RVEF is 35-45% and RVEDP is 10-20 mmHg; poor, if RVEF is less than 35% and RVEDP is greater than 20 mmHg. Pulmonary regurgitation was graded as mild, moderate, and severe according to the definition of Zahka et al. (4), and the severity of aortic regurgitation was graded by the method of Perry et al. (5).

Statistical analysis

Using PC-SAS (version 6.04), all data are expressed as mean±SD, median, or percentage. Comparisons between the groups were assessed by paired t test or Fisher exact test. The probability of survival and freedom from reoperation were estimated by the Kaplan-Meier method. A p value of <0.05 was considered significant.

RESULTS

Palliative surgery prior to corrective repair was performed in 2 patients. Palliative RV outflow tract reconstruction 6 months before corrective repair was performed in one patient and palliative RV outflow reconstruction following Blalock-Taussig shunt 11 months prior to repair in another. Surgical approach and RV outflow patch at the time of corrective repair were summarized in Table 2. Concomitant procedures at the time of tetralogy repair were performed in 9 patients (Table 3). The mean postrepair ratio of the peak RV to left ventricular (LV) pressure ($P_{RV/LV}$) measured after the termination of cardiopulmonary bypass was 0.55±0.12 (range 0.29 to 0.71) for a cohort of 40 patients.

Table 2. Surgical approach and method of right ventricular outflow reconstruction

Repair approach	Pulmonary outflow patch	No. of patients
Trans RA-PA	Without TAP	3
	With limited TAP	1
Trans RV	No patch	2
	Right ventricular patch	26
	TAP	16
Total		48

TAP, transannular patch; Trans RA-PA, transatrial-transpulmonary; Trans RV, transventricular

There were no hospital deaths, which occurred within the first 30 days after operation or before hospital discharge. All survivors have been followed up postoperatively from 3 months to 11 years with a median of 4.6 years and functional status, pulmonary regurgitation and RV function were assessed. Functional status was determined at the time of the latest follow-up. Thirty-nine patients (81.3%) were in New York Heart Association (NYHA) functional class I; 8 (16.7%) were in class II, and one (2.1%) was in class III. Thirty-six patients (75%) were graded as mild pulmonary regurgitation, 11 (22.9 %) as moderate, and one as severe. The incidence of significant (moderate to severe) pulmonary regurgitation was higher in patients repaired with transannular patch than in those repaired without (37.6% vs 14.2%, $p<0.001$), and also higher in patients who showed RV aneurysm than in those who did not (59.3% vs 28.7%, $p<0.001$). Forty-one patients (85.4%) had good RV function; 5 (10.4%) fair, and 2 (4.2%) poor. One of the 2 patients who had poor RV function underwent corrective repair at 36.2 years of age, and underwent reoperation 2 years after initial repair for a residual ventricular septal defect (VSD), RV outflow obstruction, and mitral regurgitation. Severe pulmonary and tricuspid regurgitation developed 4.5 years after reoperation and led to RV dysfunction with RVEF of 23% and RVEDP of 21 mmHg. The other patient underwent corrective repair with concomitant aortic valve replacement at 42 years of age, but a residual VSD, paravalvular leak, and RV failure occurred postoperatively, and died of biventricular dysfunction 2.6 years after operation.

There were 6 reoperations (12.5%) in 5 patients. Their

Table 3. Concomitant procedures at the time of repair

Procedure	No. of patients
PA angioplasty	3
ASD closure	3
AVR	2
AVP	1
Total	9

ASD, atrial septal defect; AVP, aortic valvuloplasty; AVR, aortic valve replacement; PA, pulmonary artery

Table 4. Causes for reoperation and time interval from initial repair

Years after repair	VSD	RVOO+VSD	PR
≤1	1		
1-5		4*	
≥5			1†

Total=6 in 5 patients; 1 patient had 2 reoperations.
*Associated with tricuspid regurgitation, mitral regurgitation in 1 each
†Associated with tricuspid regurgitation
PR, pulmonary regurgitation; RVOO, right ventricular outflow obstruction; VSD, ventricular septal defect

ages at the time of reoperation ranged from 26.1 to 43 years with a median of 30.2 years, and time interval between initial repair and reoperation ranged from 6 months to 6.7 years with a median of 2 years (Table 4). The actuarial freedom from reoperation 6 months, 1 year, 5 years, and 10 years after initial repair was 97.8%, 95.4%, 87.5%, and 81.3%, respectively (Fig. 1). The indications for reoperation included residual VSD (n=1), RV outflow obstruction with VSD (n=4), and pulmonary regurgitation (n=1). There were no hospital deaths related to reoperation. Residual VSD was present in all 5 patients undergoing reoperation. The mean value of pulmonary to systemic flow ratio (Qp/Qs) was 2.1±0.5. The location for a residual VSD was in the ventriculoinfundibular fold (n=2), anterosuperior quadrant, posteroinferior quadrant, and perimembraneous area in 1 each. Residual RV outflow obstruction, the main cause for reoperation, was at the infundibulum (n=1) and midcavity (n=3). The mean peak systolic pressure gradient at the level of obstruction measured by catheterization was 57±3 mmHg.

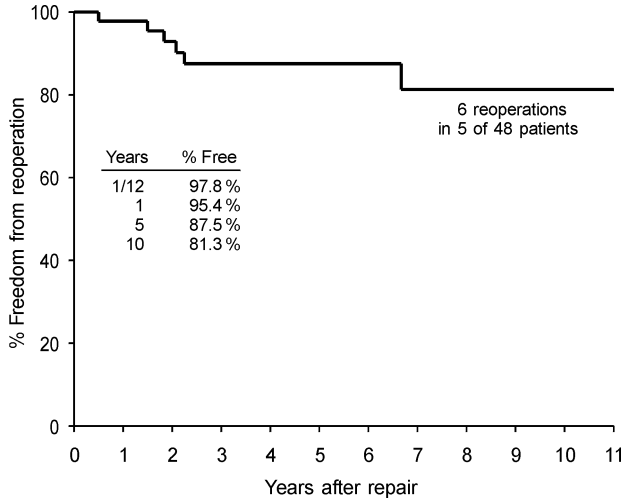

Fig. 1. Actuarial freedom from reoperation after initial repair of tetralogy of Fallot.

Pulmonary valve replacement for severe pulmonary regurgitation was performed in one patient. He underwent corrective repair using RV outflow patch at the age of 36.2 years. Reoperation was performed to repair a residual VSD, RV outflow obstruction and mitral regurgitation 2 years later. Severe pulmonary and tricuspid regurgitation developed 4.5 years after reoperation. Finally, he underwent pulmonary valve replacement using Carpentier-Edwards 31 mm with tricuspid valvuloplasty as the second reoperation at the age of 42.9 years. After pulmonary valve insertion, echocardiography demonstrated a reduction in RV dilation and cardiothoracic ratio decreased from 0.68 to 0.53 at the 10-month follow-up.

Six patients (12.5%) had aortic regurgitation preoperatively: 2 were graded as IV, 1 as III, 1 as II, and 2 as I. In addition to tetralogy repair, aortic valve replacement or valvuloplasty was performed in 3 patients with severe (grade IV or III) regurgitation. Of the 3 patients with grade I or II regurgitation, 2 did not show any change in the severity of aortic regurgitation after repair, while there was a mild increase in the severity in one. Two patients had newly developed aortic regurgitation after repair: one had mild aortic regurgitation caused by the surgical repair itself; the other had severe regurgitation by infective endocarditis (Fig. 2). There was one (2.1%) infective endocarditis during follow-up. Infection occurred at the aortic valve, leading to severe aortic regurgitation 1.4 years after corrective repair. Aortic valve replacement was performed in this patient.

The common electrocardiographic abnormalities after repair included right bundle branch block, ventricular ectopic beat, and bifascicular block, etc. (Table 5). Post-

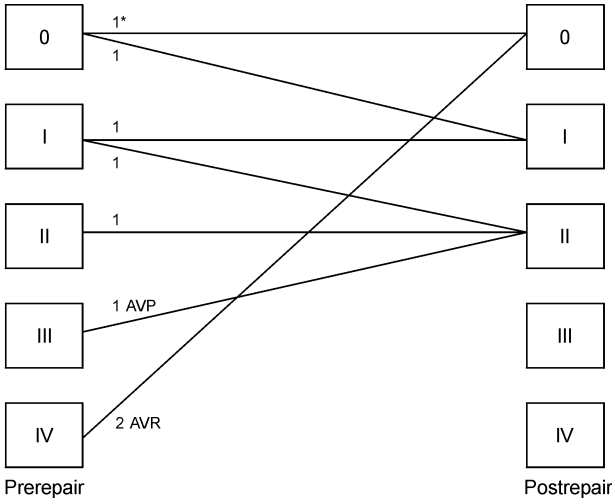

Fig. 2. Changes in the severity of aortic regurgitation (AR) after repair of tetralogy of Fallot, with the number of patients shown near each line. *Severe AR caused by infective endocarditis developed 1.4 years postrepair, leading to aortic valve replacement (AVR). AVP, aortic valvuloplasty

Table 5. Postoperative electrocardiographic findings (n=48)

ECG findings	No. of patients (%)
RBBB	26 (54.2)
Normal sinus rhythm	9 (18.8)
Ventricular ectopy	8 (16.7)
RBBB+LAH	6 (12.5)
RBBB+1° AVB	4 (8.3)
Atrial ectopy	3 (6.3)
RBBB+LAH+1° AVB	1 (2.1)
1° AVB	1 (2.1)

AVB, atrioventricular block; LAH, left anterior hemiblock; RBBB, right bundle branch block

operative complete heart block was not seen in our patient group.

Of the 18 female patients, 2 patients had two live births each, 5 and 8 years after corrective repair, and the other 2 had a live birth each, 4 years after repair. There were no live births with congenital heart disease.

There was one (2.1%) late death. The patient, aged 42 years at the time of correction, underwent aortic valve replacement using Carbomedics 25 mm for severe regurgitation in addition to tetralogy repair. But a residual VSD, paravalvular leak and postrepair RV failure developed into biventricular dysfunction, leading to death 2.6 years after operation. Autopsy was not performed. Thus, the 10-year actuarial survival rate estimated by the Kaplan-Meier method was 97.1% for a total of 48 patients (Fig. 3).

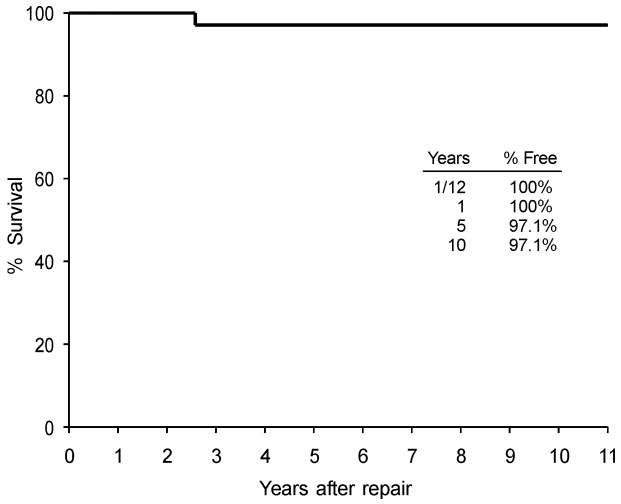

Fig. 3. Actuarial survival of 48 patients with repaired tetralogy of Fallot.

DISCUSSION

There are approximately 500,000 adults with congenital heart disease in the United States, and each year another 10,000 children who have undergone surgical repair reach adulthood (6). Among these patients, TOF is one of the major diagnostic category. This study involved the clinical outcome of grown-ups with repaired TOF and focused on postrepair problems.

Although the majority of patients with repaired TOF are reported to be excellent with respect to survival, functional status and hemodynamics by the long-term follow-up studies (2, 7-9), a small group of patients have residual problems or sequelae that require medical or surgical therapy. The major problems include conduction disorders, RV outflow obstruction, pulmonary regurgitation, or RV dysfunction. Our study showed that the 10-year survival rate following repair was 97.1%, 81.3% of patients were in good functional status, 85.4% have maintained normal RV function, and the incidence of reoperation was 12.5% during a mean follow-up of 4.6 years.

Among the residual lesions after corrective repair, RV outflow obstruction is the major cause for reoperation (9). It may be the sole residual defect, or more often, it is associated with additional defects. RV outflow obstruction may be at the infundibulum, pulmonary valve or annulus, main or branch pulmonary arteries (9, 10), while in this study, it was at the infundibulum or midcavity. It is speculated that the residual obstruction may be caused either by incomplete resection of muscle within the RV cavity or by the development of double-chambered right ventricle due to displacement of the hypertrophied moderator band with subsequent progressive hypertrophy even after corrective repair (11), as the mean subpulmonary gradient by Doppler echocardiography increased from 18±3 to 62±9 mmHg ($p<0.05$).

Pulmonary regurgitation commonly occurs after repair of TOF. Zahka et al. (4) demonstrated that pulmonary regurgitation was present in 78% of patients with tetralogy repair, with 59% of patients graded as mild and 19% as moderate. Mild pulmonary regurgitation was present in 75% of our patients and was well tolerated. Considerable controversies, however, exist regarding the effects of moderate to severe pulmonary regurgitation on the long-term RV function (7-9, 12-16). The studies supporting the benign nature of pulmonary regurgitation have been criticized, because the assessment of RV function was based primarily on the presence or absence of clinical symptoms of ventricular failure during a relatively short period of follow-up. In addition, many of these studies focused on the effects of isolated pulmonary regurgitation and fail to consider the influences of the frequently coexisting other lesions such as RV outflow obstruction, residual VSD, or RV aneurysm in exaggerating the adverse effects of pulmonary regurgitation (13, 14). Bove et al. (13) reported improved RV function following

pulmonary valve replacement for severe regurgitation or stenosis in 7 of 11 patients, and Ilbawi et al. (14) defined residual pulmonary stenosis, a large transannular patch, and residual shunts as risk factors for exaggerating the deleterious effects of pulmonary regurgitation on RV function, and recommended early pulmonary valve insertion in patients with such risk factors for preservation of normal RV function. Our study also suggests that significant pulmonary regurgitation may be associated with use of a transannular patch, or RV aneurysm. Conclusively, the influences of pulmonary regurgitation on RV function have become increasingly recognized not to be benign by many studies (12-16), and if residual shunts coexist with pulmonary regurgitation, RV volume overload accelerates ventricular dilation frequently present with valvular insufficiency. Ventricular dilation, in turn, leads to secondary tricuspid regurgitation and a vicious cycle between valvular insufficiency and ventricular dilation. The combination of pulmonary and tricuspid regurgitation compromises RV forward output significantly and results in severe failure (13, 14). Therefore, pulmonary valve insertion should be done before the onset of clinical symptoms, i.e., prior to the development of irreversible RV dysfunction (12-15, 17), and a prosthetic tissue valve such as a porcine valve is usually recommended for its freedom from thromboembolic complications and long durability (13-15, 17, 18).

Aortic regurgitation is an acquired complication that may be missed before repair, that may be caused by the surgical repair itself, that may slowly progress after repair, or that may be caused by infective endocarditis (19). It was seen preoperatively in 12.5% of patients in our study, as compared with 6.7% to 24.3% in other series (20, 21). Also there were 2 patients who had newly developed aortic regurgitation after operation. One may be caused by the repair itself and another was caused by infective endocarditis. Aortic regurgitation can be based on two different mechanisms in patients with TOF unassociated with repair, i.e., prolapse of an aortic cusp or central dilation (22). Aortic regurgitation caused by cusp prolapse is analogous to that associated with a doubly committed subaortic VSD. Dilation of aortic root appears to be more common etiology of aortic regurgitation (4, 22). It could be secondary to increased flow through the aortic valve. If increased flow is the basis for development of aortic regurgitation, it would follow that the older the patients, the greater the likelihood of aortic incompetence (22). In our study, aortic regurgitation before repair was considered to be caused by central dilation in all 6 cases.

Infective endocarditis may occur despite corrective repair of TOF. Li and Somerville (23) described that after complete repair, the incidence of infective endocarditis lessens, but tends to occur only with residual lesions such as regurgitant aortic valve, or residual VSD. Especially, if infective endocarditis involves regurgitant aortic valve in adult patients with TOF, it may induce acute severe aortic regurgitation, leading to death. According to the recently revised recommendations of prevention of bacterial endocarditis by the American Heart Association, TOF belongs to the high risk category and reparative procedure does not modify the patient's long-term risk for infective endocarditis, which continues indefinitely (24). Accordingly, as we can see one patient who acquired infective endocarditis during follow-up, it is very important to continue prevention of bacterial endocarditis indefinitely, even though there is no definitive hemodynamic abnormality after corrective repair.

With respect to pregnancies and births associated with repaired TOF, Lillehei et al. (8) reported that there were 43 (89.5%) live births among 48 pregnancies in 22 female patients with repaired TOF, and the incidence of congenital heart disease (7%) was considerably higher than that in the normal populations of about 1%, but they concluded that there is no reason for these patients not to have children. Labor and delivery have been well tolerated, and the small increase in the risk of a child with congenital heart disease is offset by the fact that almost all possible lesions are readily correctable at an early age.

Independent predictors of long-term survival following TOF repair were reported to be older age at operation and high $P_{RV/LV}$ (2, 9, 25). One late death in this study was related to biventricular dysfunction. Although the left ventricle is not directly involved anatomically in TOF, patients who have undergone corrective repair of this lesion seem prone to develop LV dysfunction as well as RV dysfunction. Borow et al. (26) noted that latent LV dysfunction may occur in the older TOF patients but not in the patients repaired during infancy. Waien et al. (27) also found a progressive decrease in LV ejection fraction with time at both and exercise in adults with repaired TOF. The compromise in LV function could be explained by the fact that fibrosis or hypertrophy of the interventricular septum or the presence of a prosthetic patch could prevent the LV chamber from changing shape or accommodating an increased preload during diastolic filling (16, 26, 27). Whether postrepair LV dysfunction is related to age at operation needs further evaluation.

As this is a retrospective study, there are many limitations. First of all, it was insufficient to analyze arrhythmias associated with postoperative TOF. In addition, balloon angioplasty for branch pulmonary artery stenosis after repair of TOF (28), compatible with surgical option in effectiveness, was excluded in the reoperation category. Finally, the small number of patients and short-term

follow-up in this study do not ensure that these data are representative of a large group of patients.

Despite the limitations, on the basis of our experience, corrective repair of TOF can be recommended even in patients above the age of 15 years since the survival rate, postrepair functional status and hemodynamics are acceptable. However, as residual anatomic and functional abnormalities exist postoperatively, continued close follow-up is essential for early identification and correction of these problems to minimize further functional deterioration affecting long-term outcome.

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