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European Journal of Cardio-thoracic Surgery 29 (2006) 162–167

EUROPEAN JOURNAL OF CARDIO-THORACIC SURGERY

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Mid-term follow-up of neoaortic regurgitation after the arterial switch operation for transposition of the great arteries☆

Ho Young Hwang [a], Woong-Han Kim [a,*], Jae Gun Kwak [a], Jeong Ryul Lee [a], Yong Jin Kim [a], Joon Ryang Rho [a], Eun Jung Bae [b], Chung Il Noh [b]

[a] Department of Thoracic and Cardiovascular Surgery, Clinical Research Institute, Seoul National University, College of Medicine, Seoul National University Children's Hospital, 28 Yongon-dong, Jongro-gu, Seoul 110-744, South Korea
[b] Department of Pediatrics, Clinical Research Institute, Seoul National University, College of Medicine, Seoul National University Children's Hospital, 28 Yongon-dong, Jongro-gu, Seoul 110-744, South Korea

Received 31 August 2005; received in revised form 27 October 2005; accepted 7 November 2005

Abstract

Objective: The aim of this study was to determine the outcome of the neoaortic valve after the arterial switch operation for transposition of the great arteries. **Methods:** A retrospective review of arterial switch operations that were performed during the period from 1991 to 2003 was conducted. We followed patients with echocardiography. When regurgitation of the neoaortic valve was observed we analyzed the risk factors. **Results:** One hundred and three patients underwent a successful arterial switch operation. Eighty-one males and 22 females participated in the study. Follow-up period was 77 ± 42 months. The age and body weight at the time of the arterial switch operation were 1.4 ± 2.8 months and 3.8 ± 1.0 kg, respectively. Preoperative pulmonary valve regurgitation was found in six patients (two patients had grade I and four patients had grade II). In the postoperative echocardiography, 52 patients demonstrated neoaortic valve regurgitation (26 patients had grade I, 25 patients had grade II, and 1 patient had grade III). At the last follow-up visit, 61 patients demonstrated neoaortic regurgitation (18 patients had grade I, 37 patients had grade II, 5 patients had grade III, and 1 patient had grade IV). Neoaortic valve regurgitation increased progressively with follow-up (p-value < 0.01). The size discrepancy between the aorta and the pulmonary artery was correlated with neoaortic valve regurgitation (p-value = 0.02). The age and body surface area, relationship of the great arteries, coronary arterial pattern, pulmonary artery banding, use of trap-door technique, myocardial ischemic time, use of total circulatory arrest, and existence of ventricular septal defect were not significant risk factors. **Conclusions:** Neoaortic valve regurgitation progressed after the arterial switch operation. The degree of regurgitation was more severe in patients with a size discrepancy between the aorta and the pulmonary artery preoperatively.
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Keywords: Neoaortic valve regurgitation; Arterial switch operation; Size discrepancy

1. Introduction

Since the first success of the arterial switch operation (ASO) was introduced in 1975 by Jatene et al. [1], it has become the treatment of choice for neonates with transposition of the great arteries (TGA) in the absence of an obstructed left ventricular outflow tract (LVOTO). The ASO has the advantages of (1) systemic location of the left ventricle and mitral valve and (2) the maintenance of sinus node function. There have been many reports that have shown excellent outcomes after the ASO [2–4]. Nevertheless, there are also disadvantages known to be associated with this surgery including coronary transfer, repositioning of the pulmonary valve and root into the systemic position, and distortion of the distal pulmonary arteries. Long-term follow-up studies of outcomes of the ASO for TGA have revealed potential problems including coronary insufficiencies, left ventricular dysfunction, ventricular outflow tract obstruction, and neoaortic valve regurgitation [5–8]. Recently, neoaortic regurgitation (NAR) has been the subject of investigation. However, the incidence, exact cause, and risk factors associated with NAR remain to be solved. The aim of this study was to determine the mid-term fate of the neoaortic valve after ASO for TGA.

2. Materials and methods

2.1. Study population

From July 1991 to December 2003, one hundred and forty consecutive patients underwent ASO for TGA. There

☆ Presented at the joint 19th Annual Meeting of the European Association for Cardio-thoracic Surgery and the 13th Annual Meeting of the European Society of Thoracic Surgeons, Barcelona, Spain, September 25–28, 2005.

* Corresponding author. Tel.: +82 2 2072 3637; fax: +82 2 3672 3637.
E-mail address: woonghan@snu.ac.kr (W.-H. Kim).

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doi:10.1016/j.ejcts.2005.11.027

were 20 early deaths and three late deaths. And we lost follow-up in 14 patients. A total of 103 survived patients who had at least one echocardiogram were included in this study. The patients enrolled were 81 males and 22 females. The hospital records of included patients were reviewed retrospectively. Investigators extracted details about patient characteristics, operative data, and the hospital course.

2.2. Operative technique

All operations were performed under the conditions of moderately hypothermic cardiopulmonary bypass (CPB). In the early cases, a total circulatory arrest (TCA) was used. The Lecompte maneuver was performed in all patients. A variety of surgical techniques were employed for the PA reconstruction. For the initial cases, we used two free pericardial patches to reconstruct the sinus portion of the neopulmonary artery. Alternatively, a direct anastomosis was made without any patch material. Beginning in 1996, a single pantaloon patch was used in almost all patients in the study. Repair of a ventricular septal defect (VSD) was usually performed through the right atrium using standard techniques. When tension in the coronary anastomosis was suspected, the trap-door technique was applied.

2.3. Echocardiographic evaluation

Echocardiographic evaluations were performed preoperatively, immediate postoperatively, and before discharge. A follow-up echocardiography was scheduled for between 6 months and 1 year postoperatively. Thereafter, echocardiographic follow-up was performed on an annual basis.

The presence and quantity of AR were evaluated by color Doppler imaging and graded as none (0), trivial (I), mild (II), moderate (III), or severe (IV) depending on the ratio of the width of the regurgitant jet to the diameter of the left ventricular outflow tract. Size discrepancy of the great arteries was evaluated preoperatively. We measured the internal diameter of the great arteries at the level of the annulus, using echocardiography.

2.4. Statistical analysis

Statistical analysis was performed using SPSS 11.0 software (SPSS Inc., Chicago, IL, USA). All descriptive data were expressed as mean values ± SD. The discrete variables were analyzed by χ^2-test and Fisher's exact test. The continuous variables were analyzed with the Student's *t*-test and linear logistic regression test. Multivariate analysis was performed with the logistic regression test. A *p*-value less than 0.05 was considered to be statistically significant.

3. Results

3.1. Clinical data

Preoperative data is given in Table 1. Age, body weight, and body surface area at the time of the ASO were 1.4 ± 2.8 months, 3.8 ± 1.0 kg, and 0.2 ± 0.1 m^2, respectively. Mean age at ASO was 18 ± 21 days in TGA with intact ventricular septum (IVS) and 68 ± 118 days in TGA with VSD group. The preoperative diagnosis was TGA with IVS in 56 patients and TGA with VSD in 47 patients. Only one patient was identified as {I.L.L.} type. Three patients had combined aortic arch abnormalities. They were interrupted aortic arch (IAA) in two and coarctation of the aorta (CoA) in one. Eighty-four patients had normal coronary artery pattern. Eight patients required palliative procedures before ASO. The palliative procedures were pulmonary artery banding (PAB) with or without modified Blalock–Taussig shunt at the mean age of 44 ± 52 days. The mean CPB and aorta cross-clamp (ACC) time were 153 ± 43 min and 84 ± 25 min, respectively. Forty-eight patients required circulatory pause in early series and the mean TCA time was 20 ± 34 min. Usually we did wedge resection at the neoaortic wall to transfer the coronary buttons. The trap-door technique was utilized in 28 (27%) patients.

Table 1
Preoperative patient characteristics

Variables	Value
Age (day)	41 ± 85
Body weight (kg)	3.8 ± 1.1
Body surface area (m^2)	0.2 ± 0.1
Sex (male:female)	81:22
TGA types	
TGA with IVS	56 (54%)
TGA with VSD	47 (46%)
Associated malformations	
Aortic coarctation	1 (1%)
Interrupted aortic arch	2 (2%)
Previous palliations	
PAB	3 (3%)
PAB + BT shunt	5 (5%)
Coronary artery patterns	
1LCx; 2R	84 (82%)
1LCxR	5 (5%)
1L2RCx	5 (5%)
1L2RCx	3 (3%)
2LCxR	3 (3%)
1R2LCx	1 (1%)
1Cx2RL	1 (1%)
1RCx2L	1 (1.%)

TGA, transposition of the great arteries; IVS, intact ventricular septum; BAS, balloon atrial septostomy; PAB, pulmonary artery banding; BT, Blalock–Taussig.

In the study period, we had 20 (14%) early mortality. But almost all of these occurred in the early period of our experience and we had only one early mortality since 2000.

3.2. Echocardiographic results

Preoperative echocardiographic evaluations showed pulmonary valve regurgitation (PR) in only six patients (6%). Two of these patients had grade I regurgitation and four had grade II. The ratio of diameter of the pulmonary artery to the aorta was 1.2 ± 0.3 (0.7–2.5). The postoperative echocardiography was performed at 16 ± 9 postoperative days. We found 52 patients (51%) with NAR. Although most of these were grade I (n = 26) or II (n = 25), a grade III NAR was identified in one patient. The last follow-

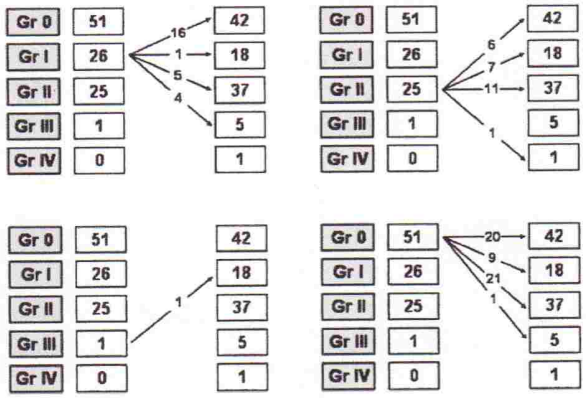

Fig. 1. Change of neoaortic regurgitation from postoperative to last follow-up echocardiography.

up echocardiography was performed at 52 ± 40 (3–149) months postoperatively. The total clinical follow-up duration was 77 ± 42 months. Among the patients who had the diagnosis of NAR at discharge, eight patients had downgrading of their NAR and 22 patients experienced total resolution of their NAR. Twelve patients had no change in their regurgitation grade throughout the follow-up period (grade I in 1 and grade II in 11). The remaining 10 patients had progressive valve diseases. They advanced from grade I to II in five patients, grade I to III in four, and grade II to IV in one. On the other hand, among 51 patients who had no regurgitation at postoperative echocardiography, new onset of NAR was diagnosed in 31 patients (grade I in 9, grade II in 21, and grade III in 1) (Fig. 1).

3.3. Reoperation

During the entire follow-up period, 4 (4%) patients required reoperations. The mean duration of time to reoperation was 49 ± 36 (16–96) months. The procedures performed at reoperation are described in Table 2. There was no patient who underwent reoperation for NAR as the primary indication for surgery although one patient showed grade IV NAR at the last follow-up evaluation. This patient underwent ASO for TGA with IVS in 1997 when he was 5 days old. The predischarge echocardiography revealed a grade II NAR; this progressed to a grade IV NAR over the 6 years of follow-up period. Reoperation has not yet been considered in this patient because his left ventricular dimension has remained in the upper normal range, his left ventricular function has been preserved, and his functional class has not decreased. Only one patient underwent neoaortic commissuroplasty. But the primary indication of his reoperation was left coronary artery (LCA) os occlusion. This patient underwent ASO in 1995 and reoperation in 2003. Echocardiography and cardiac angiography prior to reoperation revealed LCA occlusion, neoaortic root dilatation, and grade II NAR. During surgery, the patient had left main ostioplasty, neoaortic sinus reduction, and aortic commissuroplasty. The pathologic finding of an aortic root biopsy revealed a decrease in medial smooth muscle cells and medial fibrosis (Fig. 2).

3.4. Risk factors of neoaortic valve regurgitation

The preoperative variables considered as possible risk factors were age at operation, body surface area, coronary

Table 2
Reoperations: causes and procedures performed

Number	Diagnosis	Main procedure	Additional procedures
1	LCA occlusion, AAE, AR (II)	LCA ostioplasty	Neoaortic sinus reduction, AV commissuroplasty
2	Supravalvar PS, RVOTO	MPA angioplasty	Infundibulectomy
3	RPA stenosis, supravalvar AS	RPA angioplasty	Aortoplasty
4	Supravalvar PS	MPA angioplasty	–

LCA, left coronary artery; AV, aortic valve; PS, pulmonary stenosis; MPA, main pulmonary artery; RPA, right pulmonary artery; AS, aortic stenosis; RVOTO, right ventricular outflow tract obstruction.

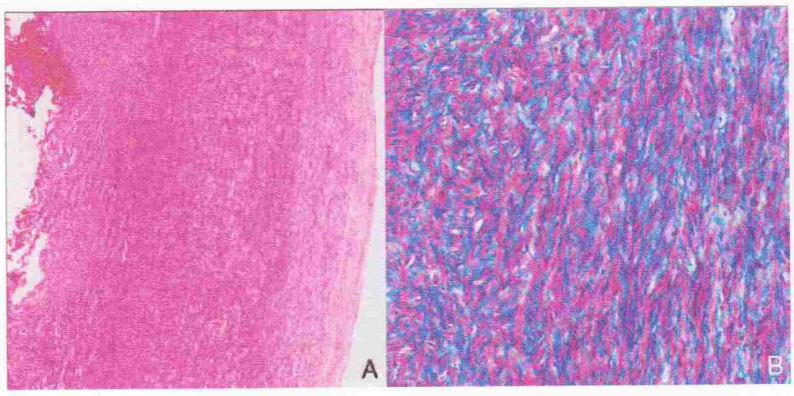

Fig. 2. Neoaortic wall biopsy. (A) Hematoxylin and eosin stain (×40) showed decrease of medial smooth muscle cells. (B) Masson's trichrome stain (×100) showed medial and adventitial fibrosis.

Table 3
Risk factors for neoaortic regurgitation after arterial switch operation

Variable	p-value		Variable	p-value	
	Univariate	Multivariate		Univariate	Multivariate
Age at operation	0.06	ns	Preoperative PR	0.88	–
Body surface area	0.70	–	Trap-door technique	0.84	–
Coronary artery pattern	0.56	–	CPB time	0.84	–
Previous PAB	0.62	–	ACC time	0.91	–
Size discrepancy	0.02	0.03	Use of TCA	0.53	–
VSD	0.27	ns	Year of operation	0.51	–

PAB, pulmonary artery banding; VSD, ventricular septal defect; PR, pulmonary regurgitation; CPB, cardiopulmonary bypass; ACC, aortic cross-clamp; TCA, total circulatory arrest.

arterial pattern, previous pulmonary artery banding, aorta-pulmonary artery size discrepancy, presence of a VSD, and preoperative pulmonary valve regurgitation (Table 3). The operative factors considered were as follows: use of the trap-door technique, CPB time, ACC time, use of TCA as well as the year of operation (before 1998). Among these variables studied, the size discrepancy of the great arteries was the only significant risk factor identified for development of neoaortic regurgitation in univariate ($p = 0.02$) and multivariate ($p = 0.03$) analyses. In addition, we analyzed whether the duration of follow-up was related to the grade of NAR. Follow-up duration did show a significant correlation with the grade of NAR ($p < 0.01$). Among eight patients who received palliative PAB before definitive correction, NAR developed in five patients and their NAR grade progressed during follow-up, as observed in other patients. However, there was no significant difference between the two groups ($p = 0.62$). Although the presence of a VSD itself had no statistical significance in the development of NAR, the size discrepancy between the aorta and pulmonary artery was significantly greater in cases that had a VSD ($p = 0.02$).

4. Discussion

The two main findings from this study are as follows: first, the size discrepancy between the aorta and the pulmonary artery significantly affected the development of neoaortic valve regurgitation; second, neoaortic valve regurgitation progressively increases with follow-up.

These observations raised concern in regard to the early and long-term function of the neoaortic valve after operation. Previous reports have shown that the neoaortic root increases in size and that the pattern of this increment is suggestive of early passive dilatation followed by normal active growth over a period of 6–10 years [9]. In our study, postoperative echocardiography performed before discharge demonstrated NAR in 52 patients. Among these patients, 30 (58%) patients experienced a decrease in or resolution of NAR during the follow-up period. This transient AR could be explained by an immediate postoperative dilating phase that is then followed by the final normal growth of the vessel. Many authors have analyzed the risk factors associated with NAR. They include existence of VSD, previous PAB, and use of the trap-door technique [7,10–13]. However, in our study, we could not identify a significant relationship between NAR and any of these suggested risk factors. In our analysis, size discrepancy was the only significant risk factor observed ($p = 0.02$). Although a report suggested that VSD closure through the pulmonary artery (neoaorta) was a risk factor for the occurrence of NAR [14], the existence of VSD itself was not found to be a significant risk factor in our series. Instead, its hemodynamic effect might contribute to create a size difference between aorta and pulmonary artery. We found this size discrepancy to be greater in the cases that had a VSD ($p = 0.02$). The size mismatch can contribute to NAR in two ways [6,11]. First, the size mismatch makes the neo-aortic reconstruction more difficult and second, it can cause more turbulence and possibly do harm to the neoaortic valve leaflets and their coaptation. These factors may result in the development of NAR and its progression.

In this study, NAR increased progressively with follow-up ($p < 0.01$). This finding is supported by other reports [6,7,12,15]. This could be explained by the size discrepancy, resultant turbulence, and other risk factors suggested. In addition, some investigators have observed histologic abnormalities in the great arterial wall. A previous report [16] revealed structural abnormalities of the great arterial walls in congenital heart disease. The ascending aorta in complete TGA is usually of normal size. However, all eight neonates included in the study had high-grade medial abnormalities, gaps in continuity along the elastic fibers, or complete loss of fibers. Because these abnormalities were found even in neonatal patients, they suggested that some of the medial abnormalities in congenital heart disease, including ASO, may be inherited. Another report [17] also identified histologic abnormalities in the great arteries of 20 untreated TGA patients. They found smooth muscle cell down-regulation in patients with TGA without VSD over 3 months. This cannot be explained by press- or flow-mediated vascular remodeling. The authors concluded that the findings might imply that the pulmonary artery in TGA is predetermined to develop differently than the pulmonary artery in a normal heart. These two reports suggested that there is a genetic predisposition for the development of NAR that should be considered in addition to acquired factors like the size discrepancy and resultant flow disturbance. In our patient who underwent reoperative procedure including neoaortic sinus reduction and neoaortic commissuroplasty, the histologic features were similar to the latter report mentioned above. It is unclear whether the findings are a result of genetic predisposition, impaired flow of the vasa vasorum due to LCA occlusion, or a combination of both.

Although the finding of NAR after ASO is not an infrequent finding, most investigators report a very low reoperation rate

(0–1.1%) [6,7,9–11,18]. In consideration of the fact that NAR increases progressively over time, further study is required with a larger population and longer follow-up period to determine the natural history and clinical significance of this finding.

In conclusion, neoaortic regurgitation after the arterial switch operation for TGA develops and increases progressively over time as a result of multiple causes including acquired factors such as surgical technique, turbulence due to size-discrepancy as well as possible predisposing factors noted in the pathology of the ascending aorta and pulmonary trunk (neoaortic root). Further study is required with a larger population and long-term follow-up.

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Appendix A. Conference discussion

Dr G. Stellin *(Padova, Italy)*: In how many of your 61 patients, in which you have found aortic regurgitation, there was an associated ventricular septal defect, and in how many the VSD was closed through the pulmonary valve?

Dr Hwang: Almost all of our patients do closure through right atrium, so VSD closure with pulmonary artery was not related to either.

Dr Stellin: All of them underwent ventricular septal defect closure through the tricuspid valve?

Dr Hwang: Yes.

Dr R. Jonas *(Washington, DC, USA)*: Your paper has very important implications, I think, for the Ross operation, where, of course, in addition to asking the pulmonary valve to work at systemic pressure, you're also placing a suture line underneath and very close to the valve leaflets.

I do have a technical question for you. I think one of the important points about doing an arterial switch is to understand that because the aortic valve is on a subaortic conus in transposition, when you transfer the coronary arteries, they should be above the sinotubular junction of the neoaortic or pulmonary valve. Therefore it's very important to stay out of the sinuses of the pulmonary valve, which are thin and prone to bleeding, but also will upset the normal mechanics of the neoaortic valve.

At what level did you implant your coronary arteries?

Dr Hwang: Usually we transfer coronary artery at the sinotubular junction level.

Dr Jonas: Do you mean the bottom of the button is above the sinotubular junction? I'm not clear on what you mean.

Dr Hwang: No, below the sinotubular junction.

Dr Jonas: Below?

Dr Hwang: Yes.

Dr Jonas: I think that may have something to do with your findings being somewhat different from the experience of others, because this is the highest incidence that I have seen of aortic regurgitation after the arterial switch.

Dr D. Metras *(Marseilles, France)*: You mentioned the aorto-pulmonary discrepancy as a risk factor for developing aortic insufficiency. What do you call discrepancy? Because, in general, there is always some discrepancy. So where do you put your limit to no discrepancy or discrepancy?

Dr Hwang: There is no point to decide the discrepancy or not. So we just calculated the ratio of the pulmonary artery to aorta. And the range is 0.7–2.5. So we analyzed this as a continuous variable and we get statistical significance. There is no cutoff point of size discrepancy.

Dr R. Pretre *(Zurich, Switzerland)*: You had a fairly large number of patients who showed significant improvement of the aortic valve function over time. How do you explain that?

Dr Hwang: Improvement of aortic regurgitation?

Dr Pretre: Yes.

Dr Hwang: The incidence of postoperative failure was higher in our study. But as you pointed out, many of these are transient and disappeared or decreased in 30 patients.

Previous reports suggest that neoaortic growth after arterial switch procedure have three phase. First is immediate postoperative dilate phase, so we think this transient AR is related to the immediate postoperative phase. And it disappeared in final normal closed phase, the third phase.

Dr V. Tsang *(London, United Kingdom)*: How many arterial switches did your unit do between 1991 and 2003?

Dr Hwang: 140 patients.

Dr Tsang: Because I think Dr Jonas has got a very good point here. 140 patients, and you looked at 103 successful arterial switch. What happens to the 33 patients?

Dr Hwang: Total early mortality was 20 and 3 late death.

Dr Tsang: I am following what Dr Jonas said. I find it quite difficult to transfer the coronary arteries below the commissural level because of the intrinsic nature of the aortic valve sitting on the RV infundibulum. The coronary arteries are already quite high. And I just wonder whether your technique may have some implications in your so-called successful number of procedures.

Dr Hwang: I beg your pardon?

Dr Tsang: The coronary transfer is vital to have successful arterial switch, and I believe the coronary transfer should be higher in the neoaorta than usual because of the aortic valve sitting on top of the muscular sleeve of the right ventricle. And your coronary transfer may have implications in terms of the high incidence of your neoaortic valve regurgitation. And I'm only just trying to speculate why your incidence of neoaortic regurgitation is very high. We don't want you to make a wrong comment on the basis of rather unusual data or surgical technique.

Dr Hwang: Yes, we think surgical technique was one of the factors in aortic valve regurgitation. But we think it's multifactorial. And as we showed in this slide, pathology was different with the normal population. So these congenital factors, and other factors like surgical technique and disturbance of flow by the size discrepancy itself, and so this is the factor for neoaortic regurgitation I see.

Dr E. Belli (*Le Plessis-Robinson, France*): A comment to avoid a misunderstanding. The trapdoor in the coronary valsalva sinus cannot be considered as a reason for aortic valve insufficiency, because the left coronary artery naturally comes at the level of the commissures. And of course, if you try to avoid to put into the valsalva sinus, you will be obliged to stretch, or to mobilize very extensively, in order to put in a higher level. Our current practice in Marie Lannelongue for the left coronary artery consisted in a trap door incision more or less at level of the valsalva sinus.

In our recent retrospective study concerning the aortic valve regurgitation after arterial switch, which will appear in the Journal of American College of Cardiology, the two risk factors were demonstrated creating subsequent valve regurgitation: when the VSD closure was performed through the PA, and the concomitant coarctation repair.