Pediatric Cardiology (2022) 43:1277–1285
https://doi.org/10.1007/s00246-022-02849-5

ORIGINAL ARTICLE

Long-Term Outcomes of Surgical Repair for Ventricular Septal Defect in Adults

Jae Hong Lim[1] · Sungkyu Cho[2] · Chang-Ha Lee[1] · Eung Re Kim[1] · Yong Jin Kim[1]

Received: 16 December 2021 / Accepted: 7 February 2022 / Published online: 18 February 2022

Abstract

Data of the outcomes of ventricular septal defect (VSD) closure in adults are limited to establish recommendations. Therefore, we reviewed our experience with surgical VSD closure in adult patients. We retrospectively reviewed 152 patients who underwent surgical VSD closure between January 1996 and April 2020. The median age of the patients was 30.5 [interquartile range (IQR) 23.1–42.7] years. The median follow-up duration was 10.9 (IQR 4.8–16.1) years. VSDs were classified according to the Society of Thoracic Surgeons classification as type 2 ($n=66$, 43.4%), type 1 ($n=59$, 38.8%), and type 4 ($n=27$, 17.8%). Aortic cusp prolapse ($n=86$, 56.6%) and aortic valve regurgitation (AR, $n=75$, 49.3%) were the most common indications for surgical closure. Four patients underwent late reoperation (2.6%) due to AR, infective endocarditis and residual VSD. In the log-rank test, preoperative trivial or more degree of AR ($P=0.004$) and coronary cusp deformity ($P=0.031$) was associated with late moderate or greater degree of AR. Preoperative moderate or greater AR was associated with reoperation ($P=0.047$). Only concomitant aortic valve (AV) repair at the time of VSD closure was a significant risk factor for late significant AR progression in the multivariable analysis. VSD closure in adults can be performed with low mortality and morbidity rates. AR can progress after VSD closure because the aortic cusp may have irreversible damage from long-standing shunt flow exposure. We conclude that VSD with AV deformity or AR in adults should be treated aggressively before disease progression with irreversible damage occurs.

Keywords Ventricular septal defect · Adult congenital heart disease · Aortic valve regurgitation

Introduction

Isolated ventricular septal defect (VSD) is a common congenital heart defect in children. The long-term outcomes of surgical VSD closure in infancy are excellent and are well documented in the literature in terms of survival, morbidity, and quality of life [1–3]. However, spontaneous and surgical closure is less common in adults. The natural history of VSDs persisting beyond childhood has been reported in some studies [4–6]. A study has reported that surgical management was required in approximately one-third of patients with VSDs initially managed conservatively [5]. Neumayer et al. reported that 47% of adult patients with unrepaired VSD reported no long-term complications; however, 25% of patients had serious complications, such as infective endocarditis, progressive aortic valve regurgitation (AR), and arrhythmia [4].

Previous studies on surgical repair of adult VSDs were early experiences or included a small number of cases; thus, contemporary outcome data on which recommendations can be based are limited in the literature [1, 7, 8]. Furthermore, information about the fate of the aortic valve (AV) after surgical repair of VSDs in adulthood is limited. Therefore, to bridge this gap in knowledge, this study investigated the long-term outcomes of surgical repair of adult VSDs and examined the prognosis of the AV.

Meeting presentation This article was presented at the 34th Annual Meeting of the European Association for Cardio-Thoracic Surgery, October 8–10, 2020.

✉ Sungkyu Cho
csk1022@hanmail.net

[1] Department of Thoracic and Cardiovascular Surgery, Sejong General Hospital, Bucheon, South Korea

[2] Department of Thoracic and Cardiovascular Surgery, Seoul National University Hospital, Seoul, South Korea

Methods

Patient Selection

Patients with VSD who were 18 years of age or older at the time of surgical VSD closure were included in this study. The exclusion criteria were the presence of any of the following: double inlet or double outlet ventricle, atresia of any valve, conotruncal anomaly, transposition of the great arteries, atrioventricular septal defect, or Ebstein anomaly. The database and chart review were approved by the Institutional Review Board of Sejong General Hospital. The requirement for informed consent was waived because of the retrospective nature of the analysis. A retrospective study was conducted on a consecutive series of 152 patients (95 men, 57 women) who underwent surgery for congenital VSD after the age of 18 years between January 1996 and April 2020. Preoperative and postoperative data were collected from patients' medical records. The median age of the patients at the time of surgical closure was 30.5 [interquartile range (IQR) 23.1–42.7] years.

Preoperative Evaluation

Diagnosis was made based on transthoracic echocardiography and direct intraoperative findings. Cardiac catheterization was performed in 52 patients who required hemodynamic evaluations. If the patient had a VSD with a large amount of shunt flow suspicious for pulmonary hypertension on echocardiography, cardiac catheterization was performed. For other patients, we estimated pulmonary hypertension on echocardiography using tricuspid valve regurgitation (TR) velocity, right ventricle (RV) morphology, and pulmonary regurgitation velocity. These values are sufficient to estimate pulmonary hypertension in patients with a low risk of severe pulmonary hypertension. VSDs were classified according to the Society of Thoracic Surgeons (STS) classification system [9]. Follow-up was initiated on the day of the surgery. The early postoperative period was defined as 30 days after surgery or surgical hospitalization, based on whichever was longer. The last follow-up details were obtained from the last outpatient department visit and the national healthcare insurance data.

Operation

After establishment of a standard bicaval cardiopulmonary bypass with moderate hypothermic and cardioplegic arrest, the VSD was closed with a patch in 136 patients with primary closure in 16 patients. Glutaraldehyde-fixed autopericardium, fresh autopericardium, bovine pericardium, and Dacron were used for patch closure. Surgical VSD closure was performed via transatrial and trans-pulmonary approaches. Aortotomy was performed when the AV required exploration or repair.

Echocardiography

Two-dimensional (2D) echocardiography and Doppler echocardiography were used to obtain preoperative clinical information. Left ventricle (LV) size and ejection fraction were assessed with 2D echocardiographic guidance by visual estimates or the Simpson biplane method. TR and AR were assessed semi-quantitatively as trivial, mild, moderate, or severe [10]. The diameter of the VSD was measured using 2D imaging. The peak flow velocity through the VSD was measured using continuous-wave Doppler ultrasound.

Statistical Analysis

Statistical analysis was performed using the IBM SPSS statistical software (version 23.0; IBM, Inc., Armonk, NY, USA). Descriptive statistics for categorical variables are presented as frequency and percentage, whereas continuous variables are presented as mean ± standard deviation or median and IQR. Overall survival, freedom from significant valve regurgitation, and reoperation were evaluated with the Kaplan–Meier survival analysis with log-rank test to compare factors. Multivariable Cox proportional hazards models were used to identify the risk factors for moderate AR, TR, and reoperation. Statistical significance was set at $P < 0.05$.

Results

Patient Characteristics

The patient demographics are summarized in Table 1. STS type 2 VSDs were the most common. The most common associated lesion was coronary cusp prolapse in 86 patients (56.6%), among whom, sinus of Valsalva aneurysmal rupture was present in 12 patients (7.9%). In addition, 40 patients had right ventricular outflow tract (RVOT) obstruction (26.3%), 20 had atrial septal defect or patent foramen ovale (13.2%), 9 had significant mitral regurgitation (5.9%), 2 had patent ductus arteriosus (1.3%), 2 had left ventricular out-track obstruction (1.3%), and 11 had arrhythmia (7.2%, Table 1).

Preoperative Echocardiographic and Catheterization Data

Preoperative echocardiographic data were available for all the patients, and preoperative catheterization was

Table 1 Patients characteristics ($n = 152$)

Characteristics	Value
Demographics	
Male:female, *n* (%)	95 (62.5%):57 (37.5%)
Age at operation (years, median, IQR)	30.5 (23.1–42.7)
STS type of VSD	
Type 1 (subarterial)	59 (38.8%)
Type 2 (membranous)	66 (43.4%)
Type 3 (inlet)	0 (0)
Type 4 (muscular)	27 (17.8%)
Associated lesions	
Coronary cusp prolapse	86 (56.6%)
Aortic valve regurgitation	75 (49.3%)
Trivial	24 (15.8%)
Mild	30 (19.7%)
Moderate	15 (9.9%)
Severe	6 (3.9%)
Tricuspid valve regurgitation ≥ mild	52 (11%)
Right ventricular outflow track obstruction (double-chambered right ventricle, pulmonary stenosis)	40 (26.3%)
Atrial septal defect, patent foramen ovale	20 (13.2%)
Mitral regurgitation ≥ moderate	9 (5.9%)
Patent ductus arteriosus	2 (1.3%)
Left ventricular outflow track obstruction	2 (1.3%)
Arrhythmia	11 (7.2%)
NYHA	
I	72 (47.4%)
II	61 (40.1%)
III	18 (11.8%)

VSD ventricular septal defect, *NYHA* New York Heart Association functional class

Table 2 Preoperative echocardiography and catheterization data

	Value	*N*
Echocardiography		152
LVEDd (mm)	54.7 ± 8.5	134
LVEF (%)	64.5 ± 7.6	133
Mean V_{max} of shunt flow (m/s)	4.7 ± 0.9	113
Mean shunt diameter (mm)	7.9 ± 5.3	139
TR ≥ mild (moderate)	52 (11)	152
AR ≥ mild (moderate)	51 (21)	152
Catheterization		52
Q_p/Q_s	2.0 ± 0.8	52
LVEDP (mmHg)	14.3 ± 5.4	45
RV systolic pressure (mmHg)	74.4 ± 32.9	47
Mean PA pressure (mmHg)	40.0 ± 21.0	47
PVR (U/m^2)	2.7 ± 2.1	27

LVEDd left ventricle end-diastolic dimension, *LVEF* left ventricle ejection fraction, *TR* tricuspid valve regurgitation, *AR* aortic valve regurgitation, *LVEDP* left ventricle end-diastolic pressure, *RV* right ventricle, *PA* pulmonary artery, *PVR* pulmonary vascular resistance

performed in 52 patients (Table 2). The mean VSD shunt diameter was 7.9 ± 5.3 mm. The LV was enlarged in most patients (mean LV end-diastolic diameter 54.7 ± 8.5 mm, IQR 49.9–59 mm). Systolic function was preserved or

mildly impaired (mean left ventricular ejection fraction 64.5% ± 7.6%; range 37–70%). Overall, 86 patients reported aortic cusp prolapse (56.6%). Not all patients with aortic cusp prolapse had AR. There were 11 patients with only aortic cusp prolapse without AR. AR and TR were present in 75 (49.3%) and 52 (11%) patients, respectively (Table 1). The peak velocity of flow across the VSD was reported in 152 patients (4.7 ± 0.9 m/s, IQR 4.2–5.1 m/s). Coronary cusp deformity was defined as cusp thickening and distortion on echocardiography.

The surgical indications for VSD closure in adult patients included coronary cusp deformity or prolapse, AR, dual chamber RV, $Q_p/Q_s \geq 1.5$, LV volume overload, pulmonary hypertension, sinus of Valsalva aneurysm or rupture, infective endocarditis, and LV dysfunction (Table 3). Coronary cusp deformity and coronary cusp prolapse were the primary motivations for surgery ($n = 88$ and 86; 57.9% and 56.6%, respectively). Overall, 75 patients (49.3%) had AR.

Surgical Data

Most patients underwent patch closure ($n = 134$, 88.2%, Table 3); 16 patients (10.5%) underwent VSD primary closure and 2 (1.3%) underwent VSD patch fenestration. The most common approach for VSD closure included the right atrium ($n = 126$, 82.9%). A total of 110 patients (72.4%) underwent concomitant repair for other cardiac defects (Table 3).

The concomitant repair included the following: RVOT enlargement in 47 patients (30.9%), atrial septal defect or patent fossa ovalis closure in 39 (25.7%), tricuspid valve (TV) repair in 23 (15.1%), AV repair in 21 (13.8%), AV replacement in 5 (3.3%), sinus of Valsalva repair in 10 (6.6%), resection of subaortic obstructing fibrotic tissue in 5 (3.3%), mitral valve repair in 5 (3.3%), the Maze procedure in 4 (2.6%), pulmonary artery angioplasty in 2 (1.3%), patent ductus arteriosus closure in 2 (1.3%), mitral valve replacement in 1 (0.7%), pulmonary valve repair in 1 (0.7%), and partial anomalous pulmonary venous return repair in 1 (0.7%). Surgical atrial septal defect was created in 3 patients (2%) who had high pulmonary vascular resistance (more than 5 Wood unit on cardiac catheterization) but had responded to the pulmonary vasodilator test. Therefore, VSD was closed with atrial septal defect creation for postoperative stabilization.

Table 3 Indications for surgery and operative data

Indication for surgery	*N* (%)
Coronary cusp deformity	88 (57.9)
Coronary cusp prolapse	86 (56.6)
Aortic regurgitation ≥ trivial	75 (49.3)
Double chamber right ventricle	28 (18.4)
$Q_p/Q_s \geq 1.5$	25 (16.4)
LV volume overload	19 (12.5)
Pulmonary hypertension	15 (9.9)
Sinus Valsalva rupture	12 (7.9)
Sinus Valsalva aneurysm	1 (0.7)
Infective endocarditis	10 (6.6)
LV dysfunction	1 (0.7)
Others	21 (13.8)
Type of closure	***N* (%)**
Primary closure	16 (10.5)
Patch closure	136 (88.2)
Patch closure with fenestration	2 (1.3)
Glutaraldehyde-fixed autopericardium	117 (77.0)
Fresh autopericardium	1 (0.7)
Bovine pericardium	13 (8.6)
Dacron	5 (3.3)
Surgical approach	
Right atrial	126 (82.9)
Pulmonary artery	87 (57.2)
Aorta	31 (20.4)
Left atrial	7 (4.6)
Right ventricular	7 (4.6)
Concomitant procedures	110 (72.4)
RVOT enlargement	47 (30.9)
Atrial septal defect closure	39 (25.7)
Tricuspid valve repair	23 (15.1)
Aortic valve repair	21 (13.8)
Aortic valve replacement	5 (3.3)
Sinus of Valsalva repair	10 (6.6)
Subaortic fibrotic tissue resection	5 (3.3)
Mitral valve repair	5 (3.3)
Maze procedure	4 (2.6)
Atrial septal defect creation	3 (2)
Pulmonary artery angioplasty	2 (1.3)
PDA closure	2 (1.3)
Mitral valve replacement	1 (0.7)
Pulmonary valve repair	1 (0.7)
PAPVR repair	1 (0.7)

LV left ventricle, *RVOT* right ventricular outflow tract, *PDA* patent ductus arteriosus, *PAPVR* partial anomalous pulmonary venous return

Outcomes and Mortality

There were two early mortalities in this study. One patient died of massive intracranial hemorrhage on postoperative day 19. The other patient, who had Down syndrome, died because of severe right ventricular failure with pulmonary hypertension on postoperative day 22. The patient's

pulmonary vascular resistance was 5.1 Wood unit and the pressure was 33 mmHg.

There was one case of a third-degree atrioventricular block that required postoperative permanent pacemaker implantation. Postoperative morbidity also included postoperative bleeding requiring reoperation, cerebral infarction, pneumonia, intracranial hemorrhage, and VSD patch detachment requiring reoperation, which occurred in one patient each.

There were four late mortalities (Table 4). The survival rates at 10 and 20 years after surgery were 97.0% and 91.7%, respectively [Fig. 1, 95% confidence interval (CI) 22.1–23.7]. Five patients (3.3%) developed atrial fibrillation on late follow-up. There were four patients who underwent late reoperation (2.6%): two patients underwent Bentall operation and AV repair, one patient underwent reoperation due to infective endocarditis, and one patient underwent reoperation 8.3 months after the initial operation due to residual VSD. Preoperative moderate or greater degree of AR was associated with reoperation in the log-rank test ($P=0.047$, Fig. 3F), but a preoperative moderate or greater degree of TR was not associated with reoperation ($P=0.604$, Fig. 2C). The rate of freedom from reoperation at 5 and 20 years was 98.5% and 96.6%, respectively. The multivariable Cox proportional hazard model could not identify any risk factors for reoperation (Table 5).

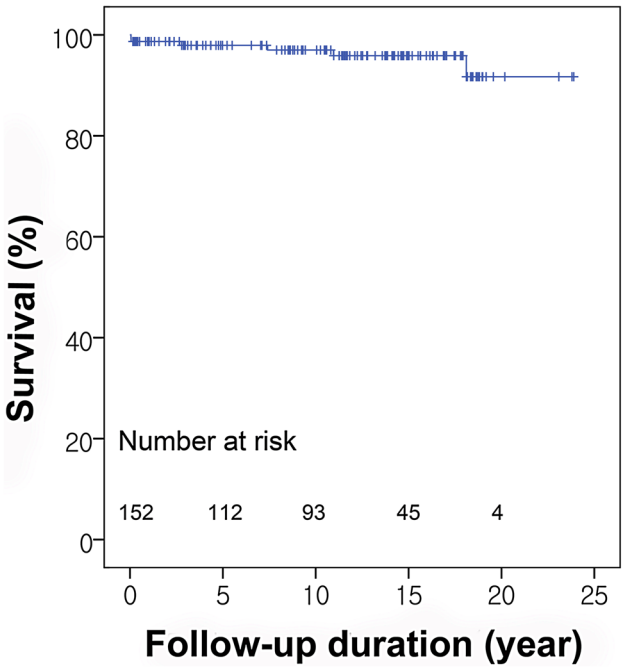

Fig. 1 Overall survival after VSD closure

Table 4 Outcomes after surgical VSD closure

	n	Value
Follow-up (years)	152	10.9 (IQR 4.8–16.1)
Early mortality	152	2 (1.3%)
Late mortality	152	4 (2.6%)
New York Heart Association functional class I–II	142	142
High-grade atrioventricular block	152	1 (0.7%)
Permanent pacemaker	152	1 (0.7%)
Atrial fibrillation	152	5 (3.3%)
Atrial flutter	152	1 (0.7%)
Supraventricular tachycardia	152	1 (0.7%)
Residual VSD	110	5 (3.3%)
Late reoperation	152	4 (2.6%)
Echocardiogram at latest follow-up	110	
Time after surgery, years (median, IQR)	110	5.1 (IQR 0.9–8.7)
Left ventricular end-diastolic diameter, mm (median, IQR)	100	48.6 (IQR 45.0–51.9)
LVEF, % (median, IQR)	102	60.3 (IQR 55.8–65.0)
TR ≥ mild	110	36 (32.7%)
TR ≥ moderate	110	9 (8.2%)
Moderate TR duration, year (median, IQR)	9	4.6 (IQR 1.1–13.3)
AR ≥ trivial	110	63 (57.3%)
AR ≥ moderate	110	10 (9.1%)
Moderate AR duration, year (median, IQR)	10	6.7 (IQR 0.2–11.5)

VSD ventricular septal defect, *IQR* interquartile range, *LVEF* left ventricle ejection fraction, *TR* tricuspid valve regurgitation, *AR* aortic valve regurgitation

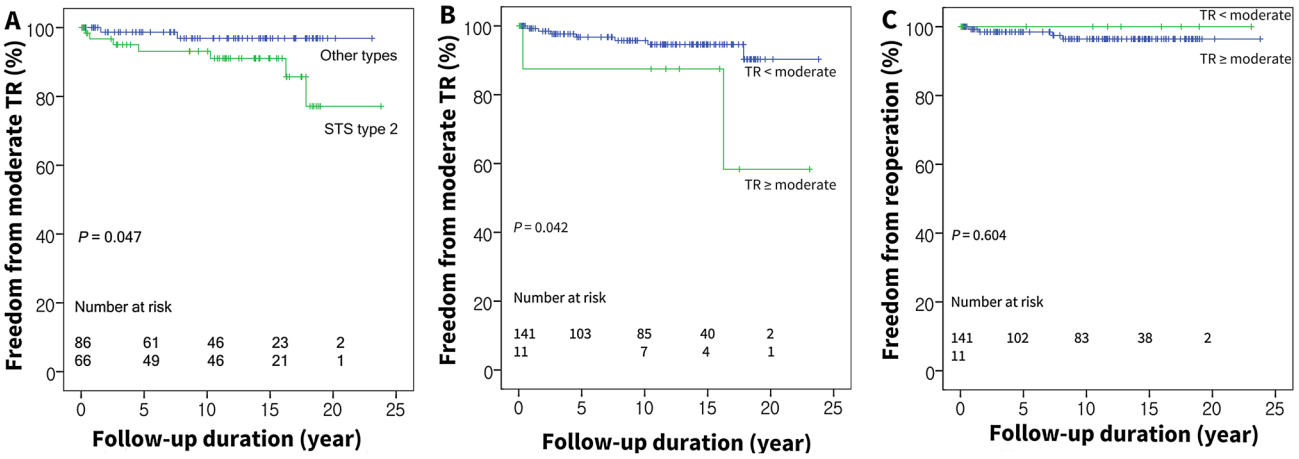

Fig. 2 Tricuspid valve analysis. **A** Freedom from moderate tricuspid valve regurgitation (TR, STS type 2 VSD). **B** Freedom from moderate TR (preoperative moderate or more degree of TR). **C** Freedom from reoperation (preoperative moderate or more degree of TR)

Table 5 Univariate and multivariable Cox proportional hazard model for risk factors of late moderate or greater degree tricuspid valve and aortic valve regurgitation and reoperation

Risk factors of moderate or greater degree of TR ($n=9$)	*N*	Univariate		Multivariable	
		HR	*P*-value	HR	*P*-value
STS type 2 VSD	66	0.591 [0.107–3.258]	0.546	0.762 [0.103–5.612]	0.526
Preoperative TR ≥ moderate	11	1.044 [0.198–5.506]	0.959	0.338 [0.034–3.386]	0.356
Preoperative TR ≥ mild	51	0.513 [0.093–2.832]	0.443		
Tricuspid valvuloplasty	23	0.406 [0.094–1.751]	0.227	2.460 [0.571–10.595]	0.227
TV detachment	30	3.932 [0.647–23.890]	0.137		
Risk factors of moderate or greater degree of AR ($n=10$)	*N*	Univariate		Multivariable	
		HR	*P*-value	HR	*P*-value
STS type 1 VSD	59	6.489 [1.377–30.571]	0.018	1.843 [0.278–12.222]	0.526
Preoperative LVOTO	2	10.510 [1.284–86.014]	0.028		
Aortic valve repair	21	8.119 [2.284–28.865]	0.001	4.546 [1.199–17.235]	0.026
Sinus of Valsalva repair	10	7.368 [1.836–29.569]	0.005	1.851 [0.373–9.170]	0.451
Preoperative AR ≥ trivial	110	10.536 [1.331–83.397]	0.026	5.922 [0.680–51.568]	0.107
Coronary cusp deformity	88	6.753 [0.854–53.408]	0.070	0.548 [0.021–14.014]	0.716
Risk factors of reoperation ($n=4$)	*N*	Univariate		Multivariable	
		HR	*P*-value	HR	*P*-value
Coronary cusp deformity	88	0.795 [0.112–5.641]	0.818	0.795 [0.112–5.641]	0.818
Preoperative AR ≥ trivial	110	1.196 [0.138–8.499]	0.858	1.735 [0.132–22.863]	0.675
Preoperative AR ≥ moderate	21	5.759 [0.811–40.891]	0.080		
Aortic valve repair	21	5.121 [0.720–36.423]	0.103		

VSD ventricular septal defect, *TR* tricuspid valve regurgitation, *HR* hazard ratio, *AR* aortic valve regurgitation, *TV* tricuspid valve, *LVOTO* left ventricular outflow tract obstruction

Five residual VSDs were identified in the late echocardiographic examination, all of which were hemodynamically insignificant, except for the VSD in one patient mentioned above.

Tricuspid Valve

During late follow-up, a mild or greater degree of TR was noted in 36 of the 110 patients who were assessed by

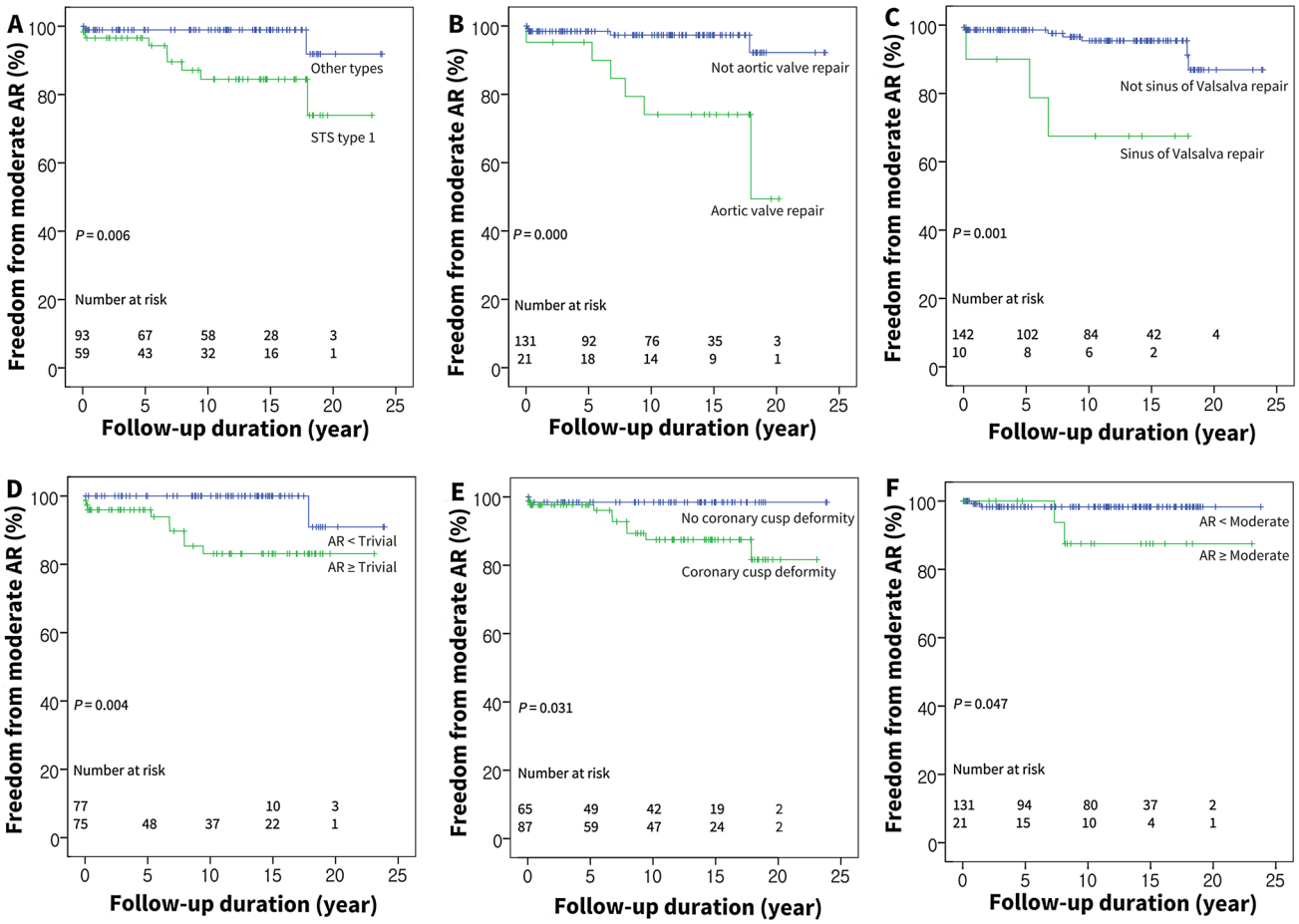

Fig. 3 Aortic valve analysis. **A** Freedom from moderate aortic valve regurgitation (AR) (STS type 1 VSD). **B** Freedom from moderate AR (aortic valve repair). **C** Freedom from moderate AR (sinus of Valsalva repair). **D** Freedom from moderate AR (preoperative AR ≥ trivial). **E** Freedom from moderate AR (coronary cusp deformity). **F** Freedom from reoperation (preoperative AR ≥ moderate)

echocardiography (Table 4). TR was moderate or greater in 9 patients (8.2%) at late echocardiographic follow-up (median 4.6 years; IQR 1.1–13.3 years). Among the 9 patients with moderate or greater TR at late follow-up (Table 5), 7 had an STS type 2 VSD and 2 had an STS type 4 VSD. In the log-rank test, STS type 2 VSD and a preoperative moderate or greater degree of TR were associated with moderate or greater TR at late follow-up ($P = 0.047$ and 0.042, respectively) (Fig. 2A, B). However, the multivariable Cox proportional hazard model did not reveal the statistical significance of all the risk factors (Table 5).

Aortic Valve

AR was noted in the preoperative echocardiographic data of 75 of 152 patients, including trivial AR (Table 1). Ten patients (9.1%) had a moderate or greater degree of AR on the last echocardiography (median 6.7 years; IQR 0.2–11.5 years). Among these 10 patients, 8 had an STS type 1 VSD and 2 had an STS type 2 VSD. In the log-rank test, STS type 1 VSD, AV repair, and sinus of Valsalva repair were associated with moderate or greater degrees of AR ($P = 0.006$, 0.000, and 0.001, respectively, Fig. 3A–C). The multivariable Cox proportional hazard model revealed that AV repair was the only significant risk factor for late moderate or greater degree of AR (hazard ratio, HR 4.546 [1.199–17.235], $P = 0.026$, Table 5).

In the log-rank test, any degree of preoperative AR and coronary cusp deformity with or without prolapse was associated with a late moderate or greater degree of AR ($P = 0.004$ and 0.031, respectively, Fig. 3D, E).

Discussion

This study describes the long-term surgical treatment of VSDs in a large series of adults patients [1, 4, 7]. Primarily, this study was conducted to analyze the course of repaired VSDs in adults who already have cardiac structural changes caused by long-standing VSDs. Most adult patients with

VSD underwent surgery not because of a large amount of shunt flow but due to structural complications that occurred in the natural course of unrepaired VSDs. These complications include subaortic stenosis, double-chambered RV, TR, and AR, some of which can cause serious complications such as infective endocarditis and sinus of Valsalva rupture. Nevertheless, our results concurred with the findings of Mongeon et al., who reported excellent outcomes of surgical VSD closure in adults and indicated that aortic and TR require life-long follow-up [1]. We analyzed our patients according to the VSD type and the preoperative changes caused by VSDs. Most of the enrolled patients had restrictive VSDs without a large shunt flow. Therefore, this study included a relatively small number of patients with pulmonary hypertension.

The surgical outcomes of VSD closure were excellent, and the survival rates at 10 and 20 years after surgery were 97.0% and 91.7%, respectively. Preoperative AR and coronary cusp deformity with or without prolapse were associated with a moderate or greater degree of AR at the late follow-up. AV repair was the only risk factor for late moderate or greater AR. Echocardiography revealed the prevalence of AR in 63 of 110 patients (57.3%) who had late follow-up. Other studies have reported the prevalence of late AR to be 40–82% [1, 8, 11]. STS type 1 VSD, sinus of Valsalva rupture, and AV or sinus of Valsalva repair are known risk factors for the development of moderate AR at follow-up [1, 12, 13]. Other studies have reported that the surgical closure technique, VSD size, and VSD leakage are risk factors for the progression of moderate AR [13–15]. In the log-rank test, we determined that STS type 1 VSD, sinus of Valsalva rupture, and AV or sinus of Valsalva repair were associated with late moderate AR progression (10/110, 9.1%). In addition, we revealed that preoperative AR of even a trivial degree and coronary cusp deformity with or without prolapse were associated with the progression of late moderate AR. In the multivariable analysis, concomitant AV repair was a risk factor for late significant AR. These results suggest that irreversible AV damage before surgical VSD repair is a poor prognostic factor for AR. Based on our findings, we suggest that surgical VSD repair be actively considered before irreversible AV damage.

The progression of AR in an unrepaired VSD can be explained by the Venturi effect and drag forces [16–18]. However, the progression of AR after VSD closure cannot be explained easily, given that it is affected by multiple factors that are difficult to identify.

It could be assumed that long-term exposure to the Venturi effect in untreated adults causes permanent histologic degeneration compared with that noted in pediatric patients, leading to progression of AR even after VSD closure. This explains the progression of AR after surgical repair, even in patients without prior AR. For patients with an STS type 2 VSD with AV prolapse, Cheung et al. correlated the degree of coronary cusp deformity, which they categorized into three grades based on the extent of prolapse, with the degree of AV prolapse [13].

A greater than moderate degree of AR can be an indication of AV surgery, but there is a higher risk for reoperation even in patients who have only coronary cusp deformity without AR or trivial AR because postoperative AR can progress continuously and slowly. Therefore, careful long-term follow-up is necessary.

Our study reported the follow-up data for TR and AR. The number of patients with a moderate or greater degree of TR was relatively small (9 of 110 patients, 8.2%) according to the last echocardiographic follow-up data. Transatrial VSD repair can affect TV competence, but a careful transatrial approach can avoid TV complications. Some studies have reported the outcomes of transatrial VSD repair using the TV leaflet detachment technique [19–21]. These studies have reported the safety of transatrial VSD closure and excellent exposure through TV detachment. STS type 2 VSD and a preoperative moderate or greater degree of TR were risk factors for late moderate TR. Mongeon et al. also reported the potential association of STS type 2 VSD with a moderate or greater degree of TR [1]. Despite the low prevalence of moderate TR, TV also requires long-term follow-up because a preoperative moderate or greater degree of TR is a risk factor for late TR according to our data.

Limitations

This study has some limitations. First, this was a retrospective, nonrandomized, single-center study. Second, late follow-up echocardiographic data were available only for 110 patients (72.4%). Third, the degree of aortic cusp prolapse could not be assessed because, to the best of our knowledge, there are no standard criteria for assessing aortic cusp prolapse, despite the fact that aortic cusp prolapse can be evaluated easily. Fourth, the multivariable Cox proportional hazard model revealed that only AV repair was a risk factor for late moderate or greater degree or AR. Fifth, we could not identify the cause of death in three of the four late mortality patients because we used national health care insurance data.

Conclusions

Surgical VSD closure in adults can be performed with low short- and long-term mortality and morbidity rates. Most indications for surgical VSD closure in adult patients differ from those for surgical VSD closure in pediatric patients. AR and TR are potential complications during long-term follow-up. Our data report that patients who underwent AV repair at the time of VSD closure had a significantly higher

incidence of late postoperative AR of a moderate or higher degree. AR could progress to a moderate or greater than moderate degree, even in cases with successful AV repair at the time of VSD closure. AR progression may be due to permanent degenerative damage of the aortic cusp prior to surgical VSD closure. If the VSD is associated with AV deformity and regurgitation, VSD repair should be actively considered because it can worsen progressively.

Acknowledgements Statistical analysis was supported by the Seoul National University Hospital Medical Research Collaborating Center.

Funding None.

Declarations

Conflict of interest There are no disclosures to report.

Ethical Approval This study was approved by the Institutional Review Board of Sejong General Hospital (2020-0537).

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