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Article

Surgical Outcomes of Right Ventricular Outflow Tract Reconstruction With Biological Valved Conduits in Children: A Retrospective Single-Center Series From Vietnam

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Abstract

Background: Right ventricle-to-pulmonary artery (RV-PA) valved conduits are widely used for right ventricular outflow tract (RVOT) reconstruction in complex congenital heart disease. However, in developing countries, this technique is still limited to a few major centers, and evidence on long-term outcomes remains scarce. This study aimed to report early- and mid-term outcomes of RVOT reconstruction using biological valved conduits in pediatric patients at a Vietnamese tertiary center. **Methods:** We retrospectively reviewed all pediatric patients with complex congenital heart disease who underwent RVOT reconstruction with a biological valved conduit at our institution between March 2017 and August 2022. Patients were described overall and by clinical strata (first conduit for RVOT obstruction vs second conduit and pulmonary valve stenosis (PVS)). Outcomes included perioperative characteristics, in-hospital complications, mortality, echocardiographic assessment of conduit performance, and conduit-related reintervention or replacement. **Results:** A total of 34 children underwent RVOT reconstruction with 38 conduit procedures. The median age was 4.0 years (interquartile range 1.0–10.3), and 55.9% were male. Preoperative malnutrition was present in 38.2%, and prior cardiac surgeryor intervention in 67.6%. The RVOT obstruction subgroup had marked baseline hypoxemia, with a mean peripheral capillary oxygen saturation of 73.8%. Postoperative complications were frequent, particularly pneumonia (42.1%) and low cardiac output syndrome/heart failure (28.9%). Early mortality occurred in two patients (5.9%), and late mortality in one patient (2.9%). No conduit-related interventions were required during the index hospitalization. At discharge, the median systolic conduit gradient was 20 mmHg and increased during follow-up (mean 30.3 mmHg). At follow-up, mild and moderate stenosis were observed in seven (18.4%) and two (5.3%) cases, respectively. During follow-up, four patients underwent surgical conduit replacement at a median of 60 months, with severe valve calcification documented intraoperatively. **Conclusions:** In this single-center Vietnamese series, biological valved conduits enabled RVOT reconstruction with acceptable early outcomes despite substantial preoperative risk burden and frequent postoperative respiratory and cardiac morbidity. Mid-term follow-up demonstrated progressive conduit obstruction in a subset of patients and clinically relevant conduit replacement at approximately 5 years, consistent with structural valve degeneration.

Keywords: congenital heart defects; cardiac surgical procedures; heart valve prosthesis implantation; pulmonary valve; postoperative complications

1. Introduction

Right ventricular outflow tract (RVOT) reconstruction with a valved conduit was an essential component of definitive or staged repair for complex congenital heart defects in which the native RVOT was absent or obstructed, including tetralogy of Fallot variants and pulmonary atresia with ventricular septal defect [1,2]. First introduced by Ross and Somerville [1], by establishing right ventricle-to-pulmonary artery (RV-PA) continuity, this surgery restored normal pulmonary blood flow and provided competent pulmonary valve function in anatomies that could not be managed by patch enlargement alone [2].

Multiple biological conduit types had been adopted for pediatric RVOT reconstruction, including cryopreserved homografts and xenografts such as bovine jugular vein conduits, with selection driven by availability, size range, and surgeon preference [1,3,4]. However, no conduit had demonstrated ideal durability or growth potential in children, and all options remained vulnerable to structural valve degeneration, leaflet calcification, distal or anastomotic stenosis, and patient-prosthesis mismatch as somatic growth progressed [4,5,6]. Long-term series reported a need for reintervention or conduit replacement over time, particularly in younger patients and smaller conduit sizes [2,4,7].

Most outcome evidence for RV-PA biological valved conduits had been generated in developed settings, while data from Vietnam and other developing countries remained limited [4,6,8]. In this limited context, children often presented later with advanced disease, higher comorbidity burdens such as malnutrition, and intercurrent infections, which compounded perioperative risk and complicated postoperative recovery [9,10]. In addition, financial barriers, geographic distance to referral centers, and workforce and infrastructure limitations influenced the timing of intervention, the choice of conduit, follow-up adherence, and the feasibility of timely reintervention [9,11].

Given these context-specific limitations, reporting local surgical outcomes was necessary to guide conduit selection, perioperative management, and follow-up strategies in pediatric practice of developing countries. Therefore, this retrospective study evaluated early and mid-term outcomes of RVOT reconstruction using biological valved conduits in children at a Vietnamese tertiary center, including mortality, major postoperative complications, hemodynamic performance, and freedom from conduit-related reintervention.

2. Materials and Methods

2.1 Study Design

The retrospective study was conducted at the Pediatric Heart Surgery Department, Cardiovascular Center of the participating tertiary center, a tertiary healthcare center in Vietnam. We reviewed all eligible operations performed between March 2017 and August 2022 and abstracted data using a predefined case report form. The reporting of this study adheres to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines for cohort studies [12].

2.2 Participants

We included all children with complex congenital heart disease characterized by pulmonary artery hypoplasia/aplasia and/or pulmonary valve regurgitation who underwent RVOT reconstruction using a biological valved conduit during the study timeframe. Eligibility required complete clinical and paraclinical records sufficient for analysis, with diagnoses confirmed by transthoracic echocardiography and chest computed tomography. No exclusion criteria were applied. The final cohort comprised 34 patients with 38 conduits.

The analytical unit was the conduit procedure (n = 38) for surgical, intraoperative and conduit-related variables, and the individual patient (n = 34) for mortality and patient-level outcomes. Four patients contributed two procedures (index and replacement conduit); these were prespecified to the second-conduit/pulmonary valve stenosis (PVS) stratum to make the conditional survival of re-do cases explicit rather than implicit in the combined analysis.

2.3 Surgical Technique for RVOT Reconstruction With a Biological Valved Conduit

Operations were performed via median sternotomy with pericardiotomy, followed by cardiopulmonary bypass (CPB) using aorto-bicaval cannulation once the activated clotting time exceeded 400 seconds. If present, the aortopulmonary shunt or ductus arteriosus was ligated and divided. Mild-to-moderate hypothermia was maintained, and myocardial protection was achieved with aortic cross-clamping and antegrade cardioplegia.

Subsequently, a right atriotomy and a right ventriculotomy at the infundibulum were performed. The septal and parietal bands were resected, RVOT caliber was assessed using an appropriately sized Hegar bougie, and the ventricular septal defect was closed through the ventriculotomy. Pulmonary artery augmentation was performed as needed, and the pulmonary bifurcation was tailored when required.

Then, an RV-PA valved conduit was inserted, with the valve positioned near the pulmonary bifurcation. After completing the distal anastomosis, the proximal end of the conduit was trimmed and beveled. If necessary, a patch was used to create a hood at the proximal anastomosis. The right atriotomy was then closed, and the aortic cross-clamp was removed. After weaning from cardiopulmonary bypass and performing modified ultrafiltration, cannulas were removed, and hemostasis was achieved. A pericardial drain tube and pacing wires were placed, and the sternum was closed using steel wires.

The biological valved conduits used in this series were xenograft valved conduits: Contegra^{TM} pulmonary valved conduits (Medtronic Inc., Minneapolis, MN, USA), made from glutaraldehyde-fixed bovine jugular vein with a trileaflet venous valve, and Labcor biological valved conduits (Labcor Laboratórios Ltda., Belo Horizonte, Brazil), derived from porcine valve tissue. Conduit sizes ranged from 11 to 21 mm. Conduit size was selected intraoperatively according to body surface area and the diameter of the recipient pulmonary trunk and branches, following the principle of implanting the largest conduit anatomically tolerated.

2.4 Data Collection

Data were collected retrospectively from paper and/or electronic medical records using a prespecified case-report form and cross-checked across operative reports, anesthesia/perfusion records, Intensive Care Unit (ICU) flowsheets, discharge summaries, and outpatient follow-up notes to minimize information bias. Variables included: (i) baseline characteristics and cardiac anatomy (including detailed diagnostic categories and associated lesions), and quantitative imaging indices such as left and right pulmonary artery Z-scores; (ii) intraoperative data, including biological valved conduit implantation and all linked surgical procedures, and CPB and aortic cross-clamp times; (iii) early postoperative outcomes, including duration of me-

chanical ventilation, ICU and hospital length of stay, predefined in-hospital complications (e.g., pneumonia, low cardiac output syndrome/heart failure, pleural or pericardial effusions), reoperation, and early and late mortality; and (iv) conduit performance assessed by echocardiography at discharge and follow-up, follow-up duration, and conduit-related reintervention or surgical replacement, with replacement cases further characterized by interval between conduit implantations and intraoperative findings at reoperation (e.g., severe valve calcification).

Follow-up was conducted at the outpatient pediatric cardiology clinic. Each follow-up visit included clinical assessment, 12-lead electrocardiography and transthoracic echocardiography performed by a pediatric cardiologist; chest radiography and laboratory testing were performed when clinically indicated. Data were captured prospectively in the clinic record and abstracted retrospectively into the case-report form.

Conduit function was assessed by transthoracic echocardiography using continuous-wave Doppler across the conduit. Severity was graded by peak systolic gradient as non-significant (<25 mmHg), mild (25–49 mmHg), moderate (50–64 mmHg), or severe (≥64 mmHg). These thresholds correspond to those applied in widely reported pediatric conduit cohorts in which significant stenosis was defined as a peak gradient ≥50 mmHg [13,14].

Conduit calcification was assessed at two levels: (i) qualitative echocardiographic evaluation during follow-up (presence or absence of leaflet echogenicity) and (ii) direct intraoperative grading at the time of conduit replacement. Patient–prosthesis match was assessed by indexing the implanted conduit diameter to body surface area at the time of implantation to derive a conduit-diameter Z-score, with patient–prosthesis mismatch defined as conduit-diameter Z-score ≤−2; the same definition has been used for RV-PA conduit dysfunction in recent pediatric cohorts [15,16].

The primary endpoint was the composite of conduit-related reintervention or surgical conduit replacement during follow-up. Secondary endpoints were early (in-hospital and ≤30-day) mortality, late mortality, predefined in-hospital complications (pneumonia, low cardiac output syndrome or heart failure, pleural or pericardial effusion, arrhythmia, multi-organ dysfunction, cardiogenic shock, wound infection, pneumothorax, sepsis), conduit hemodynamic performance at discharge and at last follow-up, and conduit stenosis severity at follow-up. Follow-up duration was calculated from the date of conduit implantation to the last available outpatient visit, death, conduit replacement, or the administrative end of the study period, whichever occurred first. The mean follow-up duration was 32.9 ± 6.5 months, with the actual observed follow-up duration ranging from 4 to 66 months.

2.5 Statistical Analysis

All statistical analyses were performed using Stata (version 14.0, StataCorp LLC, College Station, TX, USA) after data entry and cleaning in Excel. Patients were analyzed overall and by prespecified clinical strata (first conduit group with RVOT obstruction vs second conduit group and PVS). Continuous variables were assessed for distributional normality and summarized as mean ± standard deviation (SD) with minimum-maximum for approximately symmetric distributions, or as median with interquartile range (IQR) for skewed distributions. Categorical variables were summarized as counts and percentages, including preoperative status, prior interventions, complications, and conduit stenosis categories. No formal hypothesis testing was performed because this was a descriptive case series.

Key postoperative and follow-up endpoints were reported descriptively, including mechanical ventilation duration, ICU and hospital length of stay, postoperative complications (e.g., pneumonia, low cardiac output syndrome), early and late mortality, and conduit function measures (systolic gradient at discharge and follow-up; conduit stenosis severity). Conduit replacement during follow-up was summarized as frequency and timing (interval between conduit placements) together with operative findings at reoperation.

3. Results

3.1 Participant Characteristics

The study cohort was categorized for analysis according to conduit implantation episode (first-time implantation vs conduit replacement) and the clinical indication/underlying lesion (Fig. 1). Of 34 children who underwent RVOT reconstruction using a biological valved conduit, the median age at surgery was 4 years (IQR 1.0–10.3), and males predominated (55.9%, 19/34). Median body weight was 12.0 kg (IQR 6.8–22.5). Preoperative malnutrition was documented in 13 cases (38.2%) (Table 1).

Table 1. General characteristics of patients (n = 34).

Characteristics	Value
Age, years	4.0 (1.0–10.3)†
-First conduit group with RVOT obstruction	0.3–13.0‡
-Second conduit group and PVS	4.0–14.0‡
Male, n (%)	19 (55.9)
Body weight, kg	12.0 (6.8–22.5)†
- RVOT obstruction group	3.0–27.0‡
- Combined second conduit/PVS group	14.0–46.0‡
Preoperative malnutrition, n (%)§	13 (38.2)

Annotations: †Median (Interquartile range); ‡ Minimum–Maximum; §The malnutrition rate was calculated based on the number of individual patients (n = 34). **Abbreviations**: PVS, pulmonary valve stenosis; RVOT, right ventricular outflow tract.

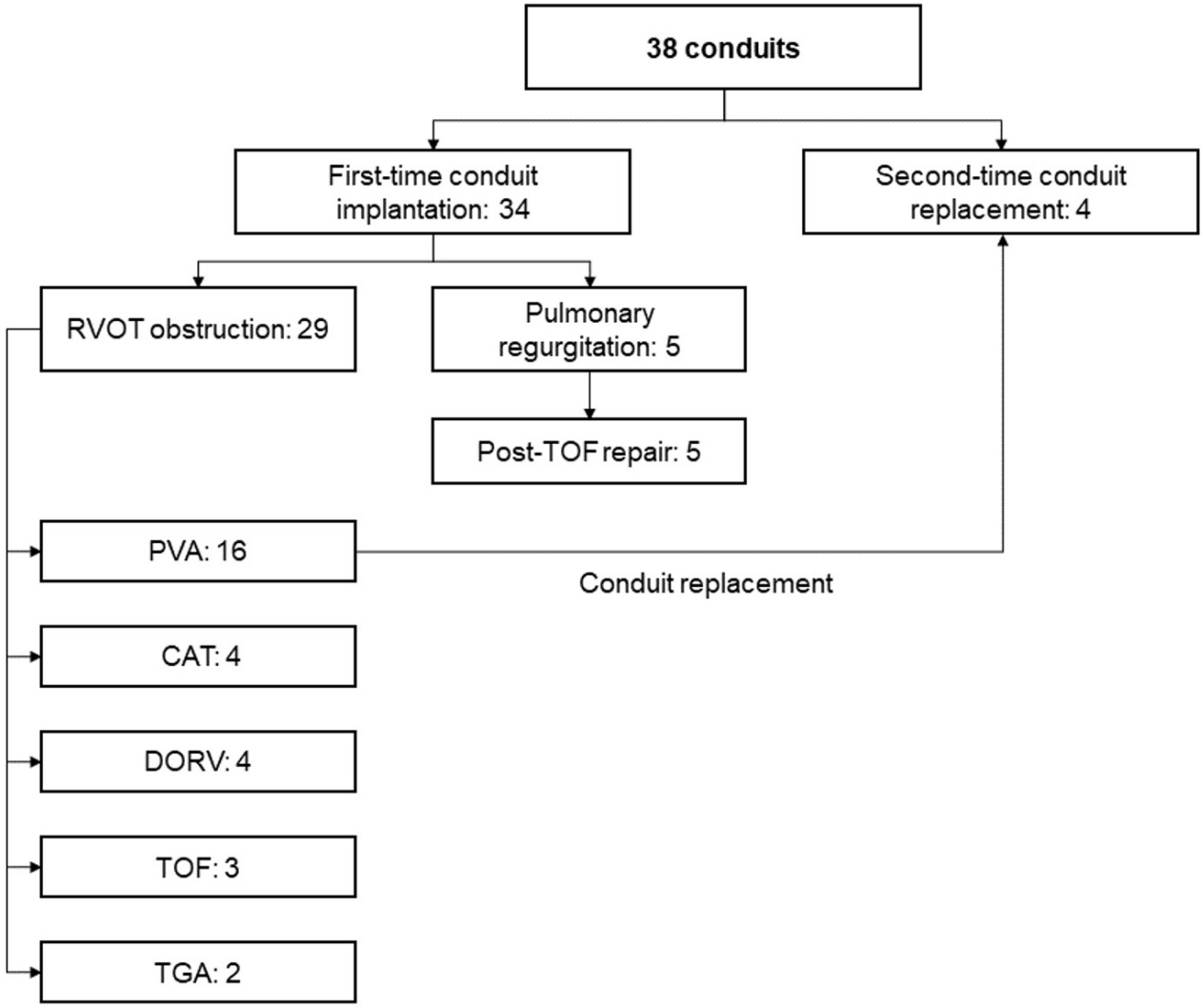

Fig. 1. Grouping by conduit type and underlying cardiac lesion. Flow diagram showing how the study material was organized for analysis by conduit procedure episode and primary indication/diagnosis. Numbers in boxes denote conduit procedures (not unique patients), and arrows indicate the hierarchical grouping used for descriptive and comparative reporting. **Abbreviations**: PVA, pulmonary valve agenesis; CAT, common arterial trunk; DORV, double outlet right ventricle; TOF, tetralogy of Fallot; TGA, transposition of the great arteries; RVOT, right ventricular outflow tract.

3.2 Clinical and Paraclinical Characteristics

Among children receiving a first conduit for RVOT obstruction, baseline peripheral oxygen saturation (SpO_2) averaged 73.8 ± 10.8% (53–96), whereas all patients in the second conduit/PVS group had baseline SpO_2 >95%. A history of prior cardiac intervention or surgery was frequent (23/34; 67.6%), most commonly ductus arteriosus stenting and prior palliative shunt procedures. Ventricular septal defect was present in all patients (Table 2).

Pulmonary artery branch dimensions were generally preserved, with the average recorded branch Z-scores being +0.9 in the RVOT obstruction group and +0.5 in the second conduit/PVS group. All patients underwent concomitant ventricular septal defect (VSD) closure (100%), and patent ductus arteriosus ligation/division was performed in 26.5%. Operative complexity was greater in first conduit group with RVOT obstruction, with longer aortic cross-clamp time (115.5 ± 43.6 versus 74.7 ± 61.2 minutes) and cardiopulmonary bypass time (256.5 ± 86.2 versus 172.6 ± 54.6 minutes) compared with the second conduit/PVS group (Table 3).

3.3 Results of Right Ventricular Outflow Tract Reconstruction Using Biological Valved Conduit

Early postoperative recovery was less favorable in the RVOT obstruction group, with a median ventilation time of 42 hours, ICU stay of 6 days, and postoperative hospital stay of 16 days, compared with 14 hours, 2 days, and 8.3 days, respectively in the second conduit/PVS group. The most frequent complications were pneumonia (42.1%) and low cardiac output syndrome/heart failure (28.9%). Early

Table 2. Clinical characteristics and cardiac anatomy of patients (n = 34).

Characteristics	Value
History of cardiac interventions/surgeries, n (%)	23 (67.6)
Previous transcatheter interventions	12 (35.3)
Ductus arteriosus stenting	9 (26.5)
Atrial septostomy	2 (5.9)
Stent dilation	1 (2.9)
Previous palliative surgeries	13 (38.2)
Modified Blalock–Taussig shunt	7 (20.6)
Pulmonary artery banding	2 (5.9)
Sano shunt	2 (5.9)
Bidirectional Glenn shunt	1 (2.9)
Central shunt	1 (2.9)
Previous total correction	9 (26.5)
Tetralogy of Fallot repair	5 (14.7)
PVA–VSD surgical repair	4 (11.8)
Preoperative hemoglobin (g/dL)	
First conduit group with RVOT obstruction	15.0 ± 3.0 (8.5–22.1)†
Second conduit group and PVS	11.3 ± 1.9 (9.9–15.4)†
Preoperative hematocrit (%)	
First conduit group with RVOT obstruction	47.7 ± 8.0 (36.0–70.6)†
Second conduit group and PVS	36.4 ± 4.2 (31.6–44.0)†
Iron-deficiency anemia	4 (11.8)
Associated anomalies	
Ventricular septal defect	34 (100.0)
Patent ductus arteriosus	9 (26.5)
Atrial septal defect	4 (11.8)
Moderate aortic regurgitation	1 (2.9)
Pulmonary valve agenesis	
PVA–VSD type 1	11 (32.4)
PVA–VSD type 2	4 (11.8)
PVA–VSD type 3	1 (2.9)
Double outlet right ventricle	3 (8.8)
Common arterial trunk type 2	2 (5.9)
Transposition of the great arteries	1 (2.9)
Pulmonary artery stenosis	
Tetralogy of Fallot	3 (8.8)
Transposition of the great arteries	3 (8.8)
Double outlet right ventricle	1 (2.9)
PVS post-TOF repair	
Pulmonary valve preservation	4 (11.8)
Monocusp valve reconstruction	1 (2.9)

Annotations: †Mean ± Standard deviation (Minimum–Maximum). **Abbreviations**: PVA, pulmonary valve agenesis; PVS, pulmonary valve stenosis; RVOT, right ventricular outflow tract; VSD, ventricular septal defect.

and late mortality occurred in 5.9% and 2.9%, respectively, and no conduit-related intervention was required during the hospitalization. At discharge, the median systolic conduit gradient was 20 mmHg at discharge and increased at follow-up to a mean of 30.3 mmHg, with mostly mild-to-moderate stenosis (Table 4).

Following discharge, patients were monitored in outpatient clinics, with a mean follow-up duration of 32.9 ± 6.5 months (range: 4–66 months). Four patients, including two females, underwent surgical conduit replacement later, at ages 5, 5, 11, and 13 years, respectively. All four had an initial diagnosis of pulmonary valve agenesis with VSD, and the interval between the two conduit placements was 60 months in each case. Severe calcification of the conduit valve was documented at reoperation. At reoperation, all four explanted conduits demonstrated severe nodular calci-

Table 3. Paraclinical findings and operative details of patients (n = 34).

Characteristics	Value
Left pulmonary artery Z-score	
First conduit group with RVOT obstruction	0.9 ± 0.7 (–1.5 to +3.3)†
Second conduit group and PVS	0.5 ± 1.1 (–1.3 to +2.0)†
Right pulmonary artery Z-score	
First conduit group with RVOT obstruction	1.0 ± 1.0 (–0.8 to +3.2)†
Second conduit group and PVS	0.9 ± 0.6 (+0.1 to +1.8)†
Surgical procedures performed in addition to conduit placement	
Ventricular septal defect closure	34 (100.0)
Patent ductus arteriosus ligation/division	9 (26.5)
Removal of previous palliative shunt	6 (17.6)
Atrial septal defect closure	4 (11.8)
Pulmonary artery debanding	2 (5.9)
Aortic valve repair	1 (2.9)
Aortic cross-clamp time (minutes)	
First conduit group with RVOT obstruction	115.5 ± 43.6 (60.0–225.0)†
Second conduit group and PVS	74.7 ± 61.2 (0.0–165.0)†
Cardiopulmonary bypass time (minutes)	
First conduit group with RVOT obstruction	256.5 ± 86.2 (180.0–540.0)†
Second conduit group and PVS	172.6 ± 54.6 (110.0–270.0)†

Annotations: †Mean ± Standard deviation (Minimum–Maximum). **Abbreviations**: PVS, pulmonary valve stenosis; RVOT, right ventricular outflow tract.

fication of the leaflets and conduit wall, with reduced leaflet excursion and circumferential thickening; no infective vegetation was identified.

4. Discussion

RV-PA valved conduits remain central to repairing complex congenital heart disease that requires RVOT reconstruction, yet no currently available conduit provides durable performance with growth potential in children [17]. This retrospective series was undertaken to evaluate early and mid-term outcomes of biological valved conduits in a Vietnamese tertiary center. In this cohort, children undergoing first-time conduit implantation for RVOT obstruction presented with a burden of preoperative hypoxemia and malnutrition, while the second conduit/PVS subgroup had near-normal baseline oxygenation. Early outcomes were characterized by frequent pneumonia and low cardiac output syndrome/heart failure, low in-hospital conduit event rates, and a small proportion progressing to severe conduit stenosis requiring surgical replacement during follow-up.

In our study, 67.6% of patients had previously undergone cardiac surgery, consistent with the report by Dearani et al. [2], which documented a rate of 56.6%. This finding indicates that more than half of the patients were not suitable for definitive repair during the initial operation and required placement of a RV-PA conduit as a part of an overall strategy to stabilize physiology, improve pulmonary blood flow, and enable subsequent definitive correction [18]. In the RVOT obstruction group, mean baseline SpO_2 was 73.8%, which is physiologically expected in cyanotic lesions where right-to-left or bidirectional shunting produces systemic venous admixture and reduces arterial oxygen saturation [19]. Chronic hypoxemia in cyanotic congenital heart disease drives compensatory secondary erythrocytosis, explaining for the higher hemoglobin observed in this subgroup [20]. In parallel, the high prevalence of malnutrition in this study was even higher than a systematic review and meta-analysis of Diao et al. [21], which reported a rate of 27.4% (95% confidence interval, 21.7–34.0) for underweight. Children with congenital heart disease frequently presented with undernutrition and that undernutrition adversely affected postoperative recovery through higher infection risk and prolonged ICU and hospital stay [22].

Related to morbidity, postoperative pneumonia represented the most frequent complication. Prior pediatric series have shown that ventilator-associated pneumonia after cardiac surgery increased morbidity and was associated with longer ventilation duration and longer pediatrics ICU stay, due to complex surgery, longer cardiopulmonary bypass exposure, inflammatory status and postoperative lung dysfunction that follow extracorporeal circulation [23,24]. These mechanisms may also contribute to low cardiac output syndrome/heart failure [25]. Early mortality was higher than that reported by Dearani et al. [2], at 5.9% versus 3.7% respectively. This difference is still explainable given the higher physiologic risk profile typical of cohorts in developing countries, including later presentation, chronic hypoxemia, and limited preoperative optimization, which can shift perioperative risk upward even when operative technique is comparable [7,10,11].

Table 4. Outcomes of RVOT reconstruction using biological valved conduits (n = 38).

Characteristics	Value
First conduit group with RVOT obstruction	
Postoperative mechanical ventilation duration (hours)	42.0 (15.0–110.0)†
Postoperative ICU stay (days)	6.0 (5.0–7.0)†
Postoperative hospital stay (days)	16.0 (12.8–20.0)†
Second conduit group and PVS	
Postoperative mechanical ventilation duration (hours)	14.0 ± 7.7 (4.0–22.0)‡
Postoperative ICU stay (days)	2.0 ± 0.9 (1.0–3.0)‡
Postoperative hospital stay (days)	8.3 ± 3.0 (5.0–13.0)‡
Postoperative complications	
Pneumonia	16 (42.1)
Low cardiac output syndrome – heart failure	11 (28.9)
Pleural or pericardial effusion	7 (18.4)
Arrhythmias	3 (7.9)
Multiple organ dysfunction syndrome	3 (7.9)
Cardiogenic shock	3 (7.9)
Early mortality	2 (5.9)
Wound infection	2 (5.3)
Pneumothorax	1 (2.6)
Sepsis	1 (2.6)
Late mortality	1 (2.9)
Reoperation	0 (0.0)
Conduit infection	0 (0.0)
Postoperative conduit function	
Systolic gradient across the conduit at discharge (mmHg)	20.0 (16.0–28.8)†
Systolic gradient across the conduit at follow-up (mmHg)	30.3 ± 38.0 (4.0–80.0)‡
Conduit stenosis at follow-up	
Mild conduit stenosis	7 (18.4)
Moderate conduit stenosis	2 (5.3)
Severe conduit stenosis	1 (2.6)

Annotations: †Median (Interquartile range); ‡Mean ± Standard deviation (Minimum–Maximum). Mortality percentages were calculated using the patient denominator (n = 34); other procedure-related outcomes were calculated using the procedure denominator (n = 38).
Abbreviations: ICU, intensive care unit; PVS, pulmonary valve stenosis; RVOT, right ventricular outflow tract.

In our cohort, conduit hemodynamics were acceptable at discharge (median systolic gradient 20 mmHg) but worsened over follow-up (mean 30.3 mmHg), with mild stenosis in 18.4%, moderate stenosis in 5.3% and severe stenosis in 2.6%, indicating progressive obstruction in a subset despite initially favorable performance. Four patients developed severe conduit stenosis requiring surgical conduit replacement at approximately 60 months, and marked valve calcification was documented at reoperation, supporting structural degeneration as the main mechanism in these cases [26]. In this context, immune-mediated injury and dystrophic calcification contribute to progressive stenosis and the need for conduit replacement [27,28]. These mid-term findings were similar to previous data from large RV-PA conduit series of Lewis et al. [8] showing that conduit-related reoperation (including replacement) accumulated over time, with a rate of 20% at 5 years in mixed pediatric cohorts. Comparable trajectories of conduit dysfunction and the role of conduit-size mismatch have been reported in recent single-center cohorts [13,16,29].

When placed in the broader context in developing settings, these findings supported the practical implication that acceptable perioperative outcomes after RVOT reconstruction with biologic valved conduits were achievable, but they also exposed system-level constraints. Barriers such as delayed diagnosis, financial hardship, geographic distance, and limited longitudinal follow-up infrastructure have contributed to later presentation with cyanosis and malnutrition as well as challenges in timely detection of conduit dysfunction [9,11]. These considerations reinforced the need for structured postoperative surveillance with standardized echocardiographic assessment and clear criteria for referral [30,31].

5. Limitations

This study has several limitations. First, its retrospective single-center design may introduce selection bias related to referral patterns, surgeon preference, and the absence of a standardised regional registry. Second, the modest sample size, heterogeneous lesions and conduit types, and small number of events limited statistical precision and precluded robust multivariable modelling; the available events did not meet the events-per-variable threshold required for stable regression estimates. Third, re-do conduit procedures were conditional on survival after the index operation, which may have slightly underestimated early adverse events; this was addressed descriptively by separating patient-level mortality from procedure-level conduit outcomes. Fourth, follow-up was clinician-driven, and conduit assessment relied mainly on transthoracic echocardiography without routine computed tomography or cardiac magnetic resonance, limiting detection of early calcification and formal evaluation of patient–prosthesis mismatch. Fifth, the mean follow-up of approximately 33 months was adequate for early and mid-term outcomes but insufficient to assess late conduit durability, growth-related mismatch, and cumulative structural valve degeneration. Finally, these findings reflect a Vietnamese tertiary-center case-mix with delayed presentation, chronic cyanosis, and frequent malnutrition, so extrapolation to other settings should be cautious. Multicenter prospective studies with standardised echocardiographic definitions and time-to-event analyses are warranted.

6. Conclusions

In this single-center retrospective series from a Vietnamese tertiary center, right ventricular outflow tract reconstruction using biological valved conduits achieved acceptable early and mid-term outcomes in children with complex congenital heart disease. Postoperative recovery was more prolonged in patients with RVOT obstruction, consistent with greater operative complexity and the need for concomitant intracardiac and pulmonary artery procedures. Pneumonia and low cardiac output syndrome/heart failure were the most frequent postoperative complications. Mid-term echocardiographic follow-up showed progressive conduit obstruction in a subset, and a small proportion required surgical conduit replacement within approximately 5 years, with intraoperative findings consistent with structural valve degeneration. These data support continued use of biological valved conduits for pediatric RVOT reconstruction in Vietnam and highlight the importance of systematic follow-up to detect conduit dysfunction and guide timely reintervention.

Abbreviations

CAT, common arterial trunk; CPB, cardiopulmonary bypass; DORV, double outlet right ventricle; ICU, intensive care unit; IQR, interquartile range; PVA, pulmonary valve agenesis; PVS, pulmonary valve stenosis; RV-PA, right ventricle-to-pulmonary artery; RVOT, right ventricular outflow tract; SD, standard deviation; SpO_2, peripheral oxygen saturation; STROBE, Strengthening the Reporting of Observational Studies in Epidemiology; TGA, transposition of the great arteries; TOF, tetralogy of Fallot; VSD, ventricular septal defect.

Availability of Data and Materials

The datasets used in this study can be obtained from the corresponding author upon a reasonable request.

Author Contributions

Conceptualization: LTKV. Data curation: LTKV, HNV. Investigation: LTKV, HNV, BQT. Methodology: HTB. Formal analysis: LTKV, HTB. Statistical analysis: HTB. Visualization: HTB, BQT. Validation: LTKV, HNV. Writing — original draft: LTKV, HNV. Writing — review & editing: HTB, BQT. Supervision: BQT. Final approval of the manuscript: LTKV, HNV, BQT, HTB. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

The study was carried out in accordance with the guidelines of the Declaration of Helsinki and approved by the Biomedical Research Ethics Committee of Cho Ray Hospital (Decision No. 3678/QĐ-BVCR, dated September 6, 2022). Owing to the retrospective nature of the study and the use of existing medical records, the requirement for informed consent to participate was waived by the Ethics Committee. All methods were conducted in accordance with the relevant ethical guidelines and regulations.

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Conflicts of Interest

The authors declare no conflicts of interest.

Supplementary Material

Supplementary material associated with this article can be found, in the online version, at https://doi.org/10.31083/HSF50637.

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