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Palen, R.L.F. van der; Ouden, A. den; Knobbe, I.; Bouma, B.J.; Koolbergen, D.R.; Jongbloed, M.R.M.; Hazekamp, M.G.

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CONGENITAL DISEASE

The arterial switch operation for transposition of the great arteries with left ventricular outflow tract obstruction and ventricular septal defect: clinical outcomes and specimen study

Roel L.F. van der Palen [a,*], Abe den Ouden [a], Ingmar Knobbe [b], Berto J. Bouma [c], David R. Koolbergen [d,e], Monique R.M. Jongbloed [f,g] and Mark G. Hazekamp [e]

[a]Department of Pediatrics, Division of Pediatric Cardiology, Leiden University Medical Center, Leiden, Netherlands
[b]Department of Pediatrics, Division of Pediatric Cardiology, Amsterdam UMC, University of Amsterdam, Amsterdam, Netherlands
[c]Department of Cardiology Amsterdam UMC, University of Amsterdam, Amsterdam, Netherlands
[d]Department of Cardiothoracic Surgery Amsterdam UMC, University of Amsterdam, Amsterdam, Netherlands
[e]Department of Cardiothoracic Surgery, Leiden University Medical Center, Leiden, Netherlands
[f]Department of Cardiology, Leiden University Medical Center, Leiden, Netherlands
[g]Department of Anatomy & Embryology, Leiden University Medical Center, Leiden, Netherlands

*Corresponding author. Tel: +31-715262835; e-mail: r.vanderpalen@lumc.nl (R.L.F. van der Palen).

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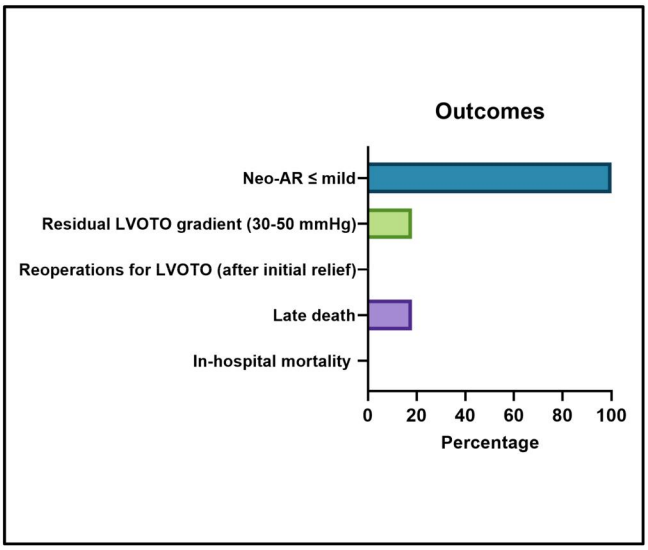
The arterial switch operation for transposition of the great arteries with left ventricular outflow tract obstruction and ventricular septal defect: clinical outcomes and specimen study

Summary

This retrospective study assessed patients with TGA-VSD-LVOTO who underwent arterial switch operation (ASO). LVOTO mechanisms were multifactorial, and multilevel in 55% of patients. In selected cases, ASO is feasible, yielding good long-term outcomes with preserved neo-aortic valve function and no reoperations for LVOTO after initial relief.



Legend: LVOTO: left ventricular outflow tract obstruction; Neo-AR: neo-aortic regurgitation

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Abstract

OBJECTIVES: In selected patients with transposition of the great arteries (TGA), ventricular septal defect (VSD) and left ventricular outflow tract obstruction (LVOTO), the arterial switch operation (ASO) may be the procedure of choice. This study reviews the clinical outcomes of TGA–VSD–LVOTO patients after ASO and compares mechanisms of LVOTO in this patient group to a historical series of cardiac specimens.

METHODS: This retrospective analysis included all cases with TGA–VSD–LVOTO who underwent ASO between January 1977 and December 2023. Additionally, a series of non-operated cardiac specimens with TGA–VSD–LVOTO was selected and examined for morphological comparison.

RESULTS: Eleven patients with TGA–VSD–LVOTO underwent ASO. Eight of them had TGA–VSD, and three had Taussig–Bing anomaly. LVOTO mechanisms were multifactorial, including posteriorly deviated infundibular septum and fibrous tissue masses. Median age at ASO was 0.4 (0.07–1.8) years. Ten patients underwent primary LVOTO relief during ASO; no in-hospital mortality occurred. Two patients died >30 days post-ASO at 3.1 months and 6.0 years. Median follow-up was 19.0 (11.1–26.8) years, all survivors in NYHA class I. The patient without initial LVOTO relief did require reoperation during follow-up for progressive LVOTO at 3.5 months post-ASO. Two patients had moderate residual LVOTO at latest follow-up (gradient 30–50 mmHg). No significant neoaortic valve regurgitation was observed. From the anatomical specimen series, 10 of 33 TGA–VSD–LVOTO specimen were deemed eligible for ASO, revealing similar LVOTO mechanisms as the clinical cases.

CONCLUSIONS: ASO is feasible in selected patients with TGA–VSD–LVOTO showing good long-term outcomes with preserved neoaortic valve function and no reoperations for LVOTO after initial relief.

Keywords: transposition of the great arteries • congenital heart disease • left ventricular outflow tract obstruction • arterial switch operation

ABBREVIATIONS

ALM	Anterolateral muscle bundle
ASO	Arterial switch operation
LVOTO	Left ventricular outflow tract obstruction
Neo-AR	Neoaortic regurgitation
TGA	Transposition of the great arteries
VSD	Ventricular septal defect

INTRODUCTION

Transposition of the great arteries (TGA) can be associated with additional anatomical anomalies, such as left ventricular outflow tract (LVOT) obstruction and ventricular septal defect (VSD). LVOT obstruction (LVOTO) is present at birth in ∼15% of infants with TGA, and its occurrence is almost always associated with a VSD [1–3]. These obstructions can be complex and may occur at multiple levels, complicating the surgical approach.

Historically, the presence of LVOTO in TGA patients was considered a contraindication for the arterial switch operation (ASO), leading surgeons to explore alternative procedures like the Rastelli, Réparation a l'Etage (REV), and Nikaidoh procedures. These alternatives were often selected due to concerns that ASO, when performed in the presence of LVOTO, might lead to suboptimal long-term outcomes, including an increased risk of reoperation and a higher incidence of postoperative neoaortic regurgitation (neo-AR). However, more recent studies have shown that ASO can be successfully performed in selected patients with TGA and LVOTO, eliminating the need for prosthetic reconstruction of the right ventricular outflow tract [3–6].

The decision to perform ASO in patients with TGA and LVOTO remains complex, requiring careful consideration of the specific anatomical and haemodynamic characteristics of each case. Despite the critical nature of this issue, relatively few studies document the mid- or long-term outcomes of ASO in the presence of LVOTO [4, 5, 7–9]. Patients with TGA, VSD, and LVOTO who are eligible for ASO with LVOTO resection represent a distinct group and can usually not be compared to those requiring a Rastelli, REV, or Nikaidoh procedure. The aim of our study was to review the clinical outcomes of TGA–VSD–LVOTO patients after ASO and to compare the spectrum of LVOTO with a historical series of non-operated cardiac specimens with TGA–VSD–LVOTO in which ASO was considered feasible.

METHODS

All patients with TGA, VSD, and LVOTO who underwent ASO with LVOTO relief at Leiden University Medical Center between January 1977 and December 2023 were identified from the local database. LVOTO was defined as an anatomic abnormality of the LVOT or atrioventricular (AV) valve apparatus apparatus leading to outflow obstruction in the uncorrected patient with TGA–VSD requiring surgical intervention either as part of the primary ASO or shortly thereafter. Data were collected on demographics, cardiac morphology, surgical characteristics, postoperative course and follow-up (including survival, need for reoperations or catheter interventions, systolic left ventricular function, presence of residual LVOTO, degree of neo-AR and occurrence of arrhythmias) using hospital and outpatient records. Coronary artery anatomy was categorized based on the Leiden Convention coronary coding system [10]. Early mortality was defined as mortality occurring within 30 days or before hospital discharge following ASO, while all other deaths were categorized as late mortality. Systolic left ventricular ejection fraction was evaluated by echocardiography at the latest follow-up and categorized as follows: normal (≥55%), mildly reduced (≥40–54%) or moderately to severely reduced (≤40%). The degree of residual LVOTO was evaluated based on echocardiographic peak gradient at latest follow-up and classified as: normal (≤10 mmHg), mild (10–30 mmHg), moderate (30–50 mmHg) or severe (≥50 mmHg). Neo-AR severity was assessed semi-quantitatively

by evaluating the colour Doppler regurgitation jet width at the level of the neoaortic valve in the parasternal long-axis view, as previously described [11, 12]. The severity of neo-AR was classified as none–trivial, mild, moderate or severe.

Cardiac specimen

Cardiac specimens from the Leiden collection of congenital cardiac malformations with TGA–VSD–LVOT that were eligible for ASO were reviewed. These hearts have been collected from autopsies since the early 1950s and preserved in an ethanol/glycerine solution. Our prior study identified that out of 200 specimens with TGA, 33 had TGA–VSD–LVOTO, and 10 of these were deemed eligible for ASO [13]. The anatomical details of these specimens were reviewed, photographed and used for illustration and comparison with the patient series.

Ethic statement

This study complied with the principles of the Declaration of Helsinki. The clinical part of this study was approved by the local scientific committee board, and the need for written informed consent was waived (reference number 23-3069). The specimen study was approved by the local scientific committee board and conducted in accordance with the guidelines of the local biobank of the Leiden University Medical Center and Dutch regulations governing the proper use of human tissue for medical research.

Surgical procedure

Our general approach is as follows: when the pulmonary valve is sufficiently large and functional (pulmonary annulus Z-score >-2.0), and the LVOTO is resectable, an ASO is possible. In cases of pulmonary valve hypoplasia or severe valvular dysfunction, the ASO is no longer a viable treatment option. In such cases, the left ventricle must either be tunnelled to the aorta (Rastelli procedure) or the aorta translocated to the LVOT (Nikaidoh procedure). Details of these treatment strategies, along with their advantages and disadvantages, have been described previously in our review [14]. Additionally, a detailed description of the ASO procedure at our centre and the criteria for a two-stage correction for TGA have been previously published [15, 16].

Regarding LVOTO, resection of the obstruction and/or repair of a stenotic pulmonary valve is performed if these interventions facilitate an ASO, minimize the risk of significant residual LVOTO and avoid the need for early pulmonary (neoaortic) valve replacement. LVOTO can be multifactorial, involving one or more obstructive substrates, each carefully assessed for resectability and size before correction. Obstructive lesions of the LVOT include a subpulmonary fibrous ridge or membrane, resectable posterior deviation of the ventricular septum, accessory tissue originating from the mitral or tricuspid valve, and valvular pulmonary stenosis, either alone or in combination.

In cases of valvular pulmonary stenosis caused by fused commissures, valvulotomy can be performed to address the stenosis. LVOTO relief can often be effectively achieved via the pulmonary valve; however, in some cases, complete removal of the deviated outlet septum through a right ventriculotomy may be necessary to fully desobstruct the LVOT. A bicuspid pulmonary valve with good function (no stenosis or insufficiency) is not considered a contraindication for ASO. Conversely, ASO is considered not feasible in cases of a hypoplastic pulmonary valve annulus or non-resectable LVOTO. In our practice, the anatomy of the coronary arteries is never a contraindication for performing an ASO.

Statistical analysis

This study is descriptive in nature. Statistical analysis was performed using IBM SPSS Statistics 29.0. Continuous variables were expressed as median (interquartile range), while categorical variables are presented as counts and percentages (%). Kaplan–Meier analysis was used to estimate overall survival. For the estimation of follow-up time, the inverse Kaplan–Meier method was used, i.e., patients were censored at the time of the event (death) and for patients without an event, the last follow-up time was counted as an event.

RESULTS

Patient characteristics

Between January 1977 and December 2023, a total of 532 patients at our institution underwent the ASO for TGA. TGA morphology among these patients included: TGA with intact ventricular septum in 322 (60.5%), TGA with VSD in 156 (29.3%) and double outlet right ventricle with overriding pulmonary trunk and subpulmonary VSD (i.e. Taussig–Bing anomaly) in 54 (10.2%) patients. Eleven patients with TGA, VSD, and LVOTO underwent ASO, with initial LVOTO relief in 10 of these patients. Baseline and surgical characteristics of these patients are shown in Table 1.

Of the 11 patients with LVOTO, 8 had TGA–VSD and 3 had Taussig–Bing anomaly. One patient in the latter group also presented with coarctation and aortic arch hypoplasia. Tricuspid valve straddling type B was present in two patients with TGA–VSD. Mechanisms of LVOTO were multifactorial and included posteriorly deviated outlet septum, fibrous tissue masses from or attached to the tricuspid or mitral valve or subpulmonary fibrous ring/membrane. These conditions existed either individually or in combination; in 6/11 (54.5%) patients, LVOTO was a multilevel problem (Table 1). The pulmonary valve was bicuspid in 1 patient and tricuspid in 10 patients. Among those with tricuspid pulmonary valve anatomy, three had asymmetrical tricuspid valves and one had thickened pulmonary valve leaflets.

Eight patients had prior surgery before ASO, including a modified Blalock–Taussig shunt in six patients (three of whom also underwent atrioseptectomy), coarctectomy in one patient and pulmonary artery banding at the age of 6.5 months to reduce pulmonary flow in another patient (performed in a low-resource setting abroad). In this patient, LVOTO developed over time while awaiting the ASO. The median age at ASO was 0.4 (0.07–1.8) years. Ten patients underwent primary LVOTO relief during ASO as specified in Table 1. In one patient, the LVOT was considered acceptably open during ASO based on visual inspection, and the outflow obstruction was not primarily addressed. A Lecompte manoeuvre was performed in nine patients. Median cardiac bypass time was 220 (194–254) min, and the median aortic cross-clamp time was 133 (129–175) min.

Table 1: Baseline characteristics, cardiac anatomy, LVOTO aetiology and surgical relief

No.	Sex	Year of ASO	Age at ASO	Weight at ASO (kg)	Diagnosis	Associated lesions	Coronary anatomy[a]	Position great arteries	Pulmonary valve	Previous surgery	Aetiology LVOTO	Surgical relief LVOTO
1	Male	1983	1.1 years	7.1	TGA–VSD	–	1LCx-2R	Ao anterior to PA	Asymmetric tricuspid	mBTT+ atrioseptectomy	Fibrous shelf	Resection fibrous shelf
2[c]	Female	1987	32 days	2.5	TBA	Hypoplastic arch with coarctation	1LCx-2R	Side by side	Bicuspid	Coarctectomy	Deviated muscular outlet septum	Incision anterior side of outlet septum to enlarge the LVOT
3	Female	1995	2.4 years	8.5	TGA–VSD	Straddling TV type B	1LCx-2R	Ao anterior to PA	Tricuspid, thick leaflets	mBTT+ atrioseptectomy	Malalignment and hypertrophy of outlet septum + subpulmonary fibrous membrane attached to MV and outlet septum	Resection of the outlet septum and enucleation of subvalvular fibrous membrane
4	Male	1998	54 days	4.5	TGA–VSD	–	1 l-2CxR	Side by side	Tricuspid	mBTT	Aneurysmatic membranous septum	Reefing of aneurysmatic membranous septum and retraction to the right ventricle
5[b,c]	Female	1998	1.8 years	8.7	TGA–VSD	–	1LCx-2R	Ao anterior to PA	Tricuspid	mBTT	Malalignment outlet septum[d]+ accessory subpulmonary fibrous tissue related to the small VSD	Resection of the left-posterior deviated outlet septum + fibrous tissue
6	Female	2001	27 days	3.5	TGA–VSD	–	1LCx-2R	Ao anterior to PA	Tricuspid	–	Subpulmonary accessory tissue attached to MV	Resection of accessory tissue
7	Female	2005	119 days	5.0	TGA–VSD	Juxtaposition of right atrial appendage	1LCx-2R	Side by side	Tricuspid	mBTT+ atrioseptectomy	Redundant "cauliflower-like" TV tissue	Reefing of accessory tissue
8	Female	2008	8 days	2.6	TBA	–	1LCx-2R	Ao left anterior to PA	Tricuspid	–	Malalignment/bulging outlet septum+ accessory subpulmonary fibrous TV tissue	Reefing of accessory fibrous tissue
9	Male	2011	9 days	3.8	TBA	–	1 l-2R, no Cx	Side by side	Tricuspid	–	Crest of the trabecular ventricular septum + circular fibrous ring (post-ASO)	3.5 months post-ASO: enucleation of subvalvular fibrous ring + myectomy of the crest of the trabecular ventricular septum
10	Female	2013	2.3 years	10.0	TGA–VSD	LSVC to CS	1R-2LCx	Ao right anterior to PA	Tricuspid	PAB+ atrioseptectomy	Subvalvular membrane, abnormal LVOT shape with prominent outlet septum	Enucleation of subvalvular membrane
11	Male	2018	0.7 years	7.5	TGA–VSD	Straddling TV type B	1LCx-2R	Ao right anterior to PA	Asymmetric tricuspid	mBTT	Fibrous mass from the TV attached to the smallest PV leaflet	Removal of fibrous mass

Ao: aorta; CoA: aortic coarctation; CS: coronary sinus; IVS: intact ventricular septum; LSVC: left superior caval vein; LVOT: left ventricular outflow tract; mBTT: modified Blalock–Thomas–Taussig shunt; MV: mitral valve; PA: pulmonary artery; PAB: pulmonary artery banding; PV: pulmonary valve; TGA: transposition of the great arteries; TBA: Taussig–Bing anomaly; TV: tricuspid valve; VSD: ventricular septal defect.

[a]according to the Leiden Convention coronary coding system [10].

[b]Epicardial pacemaker for complete atrioventricular block.

[c]Late death.

[d]Not infundibular septum.

Outcome

No in-hospital mortality occurred. Two patients required an epicardial pacemaker for complete AV block. Two patients died after hospital discharge (>30 days) post-ASO (Fig. 1): one patient, who required an epicardial pacemaker on postoperative day 18 for complete AV block, died at 3.1 months post-ASO (Table 1, patient 5; exact cause of death unknown, pacemaker dysfunction could possibly be a factor), and another patient died 6.0 years post-ASO for an unknown reason (Table 1, patient 2). Median follow-up was 19.0 (11.1–26.8) years, with all survivors being in NYHA class I.

No reoperation for LVOTO was necessary after primary LVOTO relief during ASO. However, one patient, in whom LVOTO was not initially addressed during ASO, required surgery for progressive LVOTO at 3.5 months post-ASO. Myectomy of the anterior part of the ventricular septum, along with enucleation of a fibrous ring under the neoaortic valve, was performed. Another patient required re-operative surgery after ASO at the age of 27.9 years, undergoing valve-sparing root replacement and mitral valve plasty for neoaortic root dilatation (diameter 51 mm) and residual mitral regurgitation. A relation with the pre-existing LVOTO was considered as being unlikely. No catheter interventions were necessary. No arrhythmias occurred.

Two patients (Table 1, patients 3 and 9) had moderate residual LVOTO at latest follow-up, with peak gradients of 40 and 48 mmHg, respectively. Systolic left ventricular function was normal in six patients and mildly reduced in three patients, but all had a left ventricular ejection fraction ≥45%. No significant neo-AR was observed; regurgitation was graded as none–trivial in four patients and mild in five patients.

Cardiac specimen

The ASO with VSD closure and LVOTO resection was considered feasible in 10 specimens. The detailed anatomy of these specimens and the optimal approach for surgical LVOTO relief are summarized in Supplementary Material, Table S1. Two specimens exhibited accessory fibrous tissue connected to the mitral valve (specimens no. 3 and 9), which was estimated to be feasible for resection to achieve an unobstructed LVOT (Fig. 2A). In one specimen, LVOTO was due to an aneurysmatic membranous septum extending into the LVOT (Fig. 2B and C), which could be either tightened or resected. In two specimens, a bicuspid pulmonary valve was identified either as an associated lesion (specimen no. 1) or as the sole cause of LVOTO (specimen no. 10). Valve repair was considered necessary and feasible, with an acceptable outcome expected after ASO. Of note, pulmonary valve repair should only be attempted when there is high confidence in its safety and effectiveness. If the valve is accidentally damaged, this may become apparent until after the ASO and clamp removal.

One specimen had a fibromuscular ridge along with a prominent anterolateral muscle bundle (ALM) (Fig. 3A), where resection of fibromuscular tissue would relief the LVOTO. In five other specimens (specimens no. 1, 2, 5, 7 and 8), resection of obstructive muscular tissue was required to relieve the LVOTO. Three of these specimens had obstruction caused by malalignment of the outlet septum (Fig. 3B and C), and partial resection of the outlet septum was deemed feasible through the pulmonary valve.

Specifically, for the specimens with Taussig–Bing anomaly (specimens no. 3 and 6), also addressing the subaortic right ventricular outflow tract in addition to LVOTO relief is necessary to prevent future right ventricular outflow tract obstruction. The surgical approach would involve resecting part of the outlet septum and the muscles extending from the outlet septum to the anterior right ventricular free wall [17].

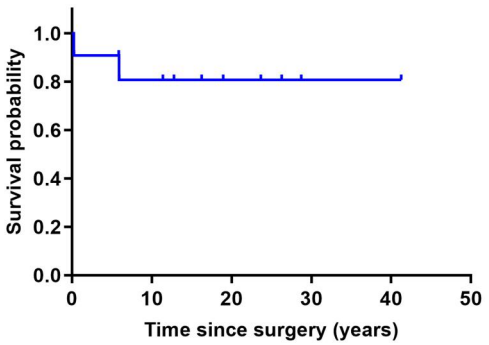

Figure 1: Kaplan–Meier estimates of overall survival after ASO in the clinical patient group

DISCUSSION

LVOTO is present in ∼10–15% of all patients with TGA [3]. In these complex cases, particularly when a VSD is also present, various surgical procedures, such as the Rastelli, REV and Nikaidoh, or the ASO, can be considered. The decision on the procedure depends on the specific advantages and disadvantages of each, with the resectability of the obstruction and the size of the pulmonary valve being key determinants when considering ASO. The results of our study show that ASO, combined with effective LVOTO relief, can be a viable option for selected cases, leading to favourable long-term outcomes, including low early mortality, acceptable reoperation rates and preserved neoaortic valve and left ventricular function. These findings support ASO as being a more favourable surgical option compared to alternatives like the Rastelli, REV or Nikaidoh.

Our patient cohort exhibited a variety of LVOTO mechanisms, including posterior deviation of the infundibular septum, fibrous tissue masses attached to the tricuspid or mitral valves and subpulmonary fibrous rings. This variability highlights the need for a tailored surgical approach to LVOTO relief during ASO. Notably, 55% of the patients had multilevel LVOTO, consistent with the literature reporting 40–70% of multileveled cases [8, 18, 19]. The absence of in-hospital mortality and the low rate of reoperations for LVOTO (one case) suggest that our surgical strategy was effective in achieving satisfactory immediate outcomes. It is well established that the anatomical complexity of obstruction and the size of the pulmonary valve are critical factors influencing postoperative outcomes. Previous studies have identified a lower pulmonary valve Z-score (i.e. below Z −1.8) and AV-valve-related LVOTO as independent predictors of LVOTO recurrence and the need for reoperation [8, 18]. Additionally, literature indicates that patients with initial multilevel LVOTO generally have significantly smaller pulmonary valve Z-scores [8, 18]. Of note, the presence of a VSD or the preoperative echo peak gradient

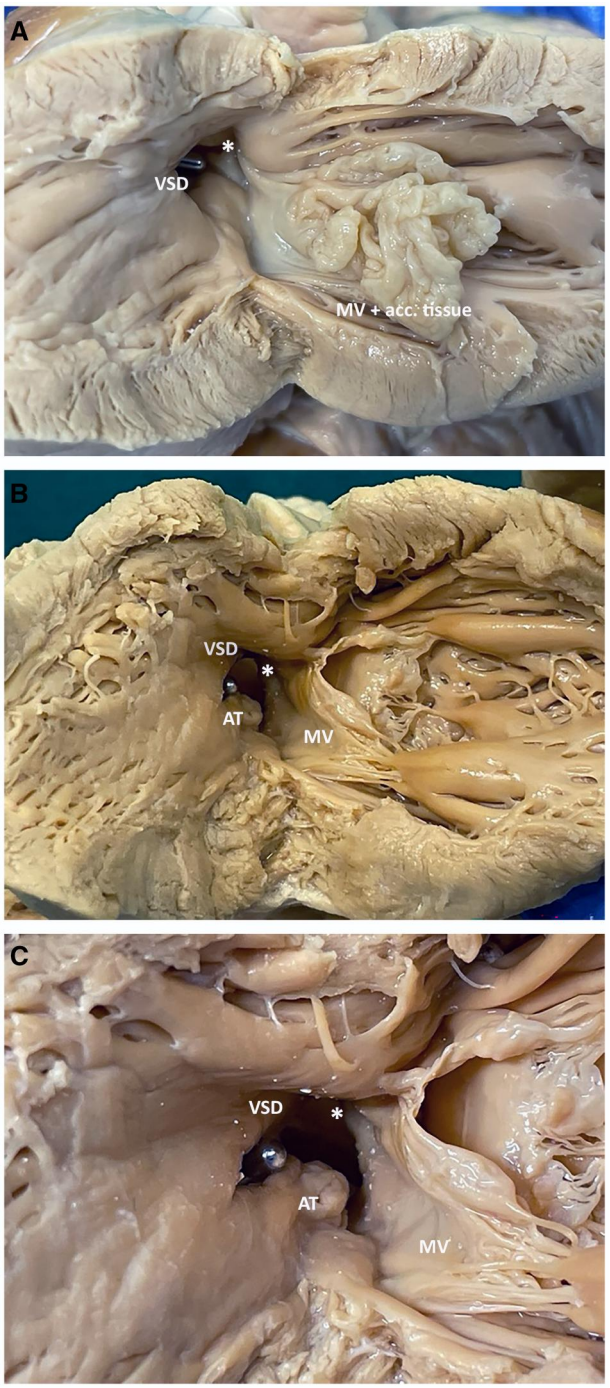

Figure 2: (**A**) Specimen no. 3: opened left ventricle with a probe from right ventricle to left ventricle through the VSD. The mitral valve has accessory tissue (acc. tissue) attached to the anterior leaflet that obstructs the LVOT. (**B**) Specimen no. 4: opened left ventricle with a probe from right ventricle to left ventricle through the VSD. Aneurysmatic tissue (AT) of the membranous septum in the left ventricular outflow tract (LVOT) can be seen. (**C**) Zoom-in of specimen no. 4 Asterisk (*) indicates the LVOT. MV: mitral valve; VSD: ventricular septal defect

have not been reported as risk factors for recurrent obstruction [3, 5, 8].

Some studies have reported on an LVOTO complexity score, which could be useful for determining the timing of early versus late ASO repair and for decision-making between late ASO and the Nikaidoh procedures [18]. However, surgical preference and strategy also play a significant role in determining the optimal timing, as ASO at a later stage can be a suitable option. The results from our series, in which 55% of patients underwent ASO with LVOTO relief beyond neonatal (14 days) or early infantile period (>8 weeks), support this approach. In our opinion, the final decision on the surgical approach may sometimes only be determined after direct inspection in the operating room. If a planned ASO unexpectedly proves unfeasible at that time, alternative procedures such as the Rastelli or Nikaidoh procedures can be considered (if coronary artery anatomy permits).

Although late mortality was relatively high in this cohort for reasons that remain unclear, most survivors experienced a relatively uneventful follow-up. At a median follow-up of 19 years, all surviving patients were in NYHA class I, indicating excellent functional status. Only two patients had moderate residual LVOTO at the latest follow-up, and reoperation for LVOTO relief was needed only in the patient whose LVOT was not primarily addressed during the ASO. This aligns with the findings of Emani *et al.* [4], who reported a higher rate of LVOT reintervention in patients undergoing ASO alone compared to those who had concomitant ASO and LVOT intervention. Similarly, other studies based on small series of TGA–VSD–LVOTO patients with mid-term follow-up after ASO report a relatively low rate of LVOT reoperation, ranging from 4% to 10% [4–9, 18, 20].

The low incidence of significant neo-AR in our cohort further supports the durability of ASO in this setting, addressing concerns raised by Sharma *et al.* [21] regarding postoperative neoaortic valve function. Mild neo-AR was observed in five of nine late survivors (56%), which is slightly higher than the incidence reported in our previous study, where 36% of the TGA–VSD cohort and 54% of the Taussig–Bing population experienced mild or more severe neo-AR [12]. The longer median follow-up period for the TGA–VSD–LVOTO subgroup (19.0 [range 5.9–41.3] years) compared to the entire TGA population after ASO (12.2 [range 1.0–39.0] years) may account for this difference. Importantly, none of the nine survivors had more than mild neo-AR. Potential factors contributing to this outcome could include pre-operative turbulent subpulmonary blood flow, prior pulmonary arterial banding or the surgical relief of subpulmonary obstruction and/or VSD closure through the native pulmonary valve during ASO. Similar studies have not reported significant neo-AR in this patient group after ASO in mid-term follow-up studies [4, 5, 7–9, 18]. However, Sharma *et al.* observed a relatively higher incidence of mild neo-AR at discharge (38%) in patients with preoperatively LVOTO compared to those without. Additionally, 50% of these patients progressed from mild neo-AR at discharge to moderate or severe regurgitation within a 6-year follow-up period [21].

The comparison with anatomical specimens from the collection of congenital cardiac malformations provided further insights into the surgical feasibility of performing an ASO in TGA–VSD–LVOTO cases. The detailed anatomical review highlighted key areas for potential obstruction. Obstructive fibromuscular tissue or accessory fibrous tissue attached to the mitral valve can usually be effectively removed without a high risk of recurrence, provided the LVOT geometry is favourable. Deviation of the outlet septum through the VSD often contributes to significant LVOTO, which may occur anterior to the pulmonary orifice or posteriorly as part of a subpulmonary conus. In such cases, resection of the muscular obstruction (e.g., malaligned outlet septum or muscular subpulmonary conus) can facilitate the successful execution of the ASO. These anatomical findings align with other clinical studies that

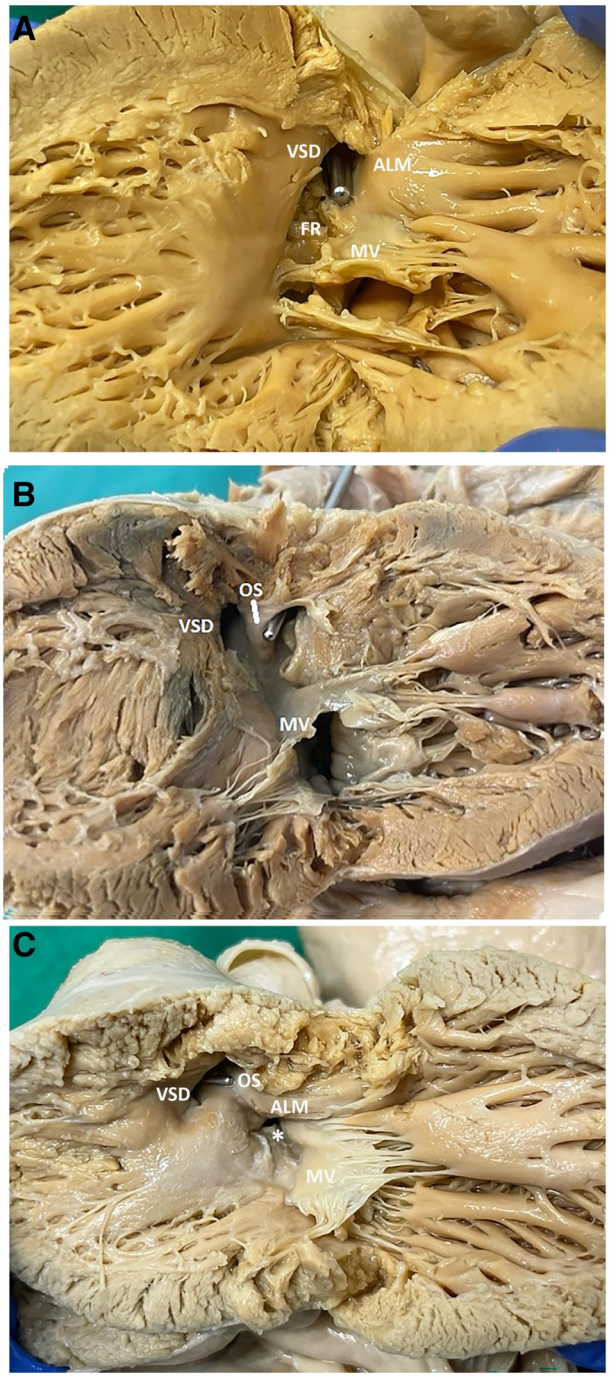

Figure 3: (**A**) Specimen no. 6: opened left ventricle with a probe in the left ventricular outflow tract (LVOT). Narrowing of this tract is due to the anterolateral muscle bundle (ALM) and a fibrous ridge (FR). (**B**) Specimen no. 1: opened left ventricle with a probe from the pulmonary artery in the LVOT. Deviation of the outlet septum (OS) to the left structurally narrows the LVOT. (**C**) Specimen no. 8: opened left ventricle with malalignment VSD (anterior subaortic) with deviation of the OS to the left through the VSD towards the LVOT. The subpulmonary infundibulum is encased by the outlet septum, along with the ALM, ventricular septum and the mitral valve. Asterisk (*) indicated the LVOT. MV: mitral valve.

emphasize the importance of individualized surgical planning based on precise anatomical assessments.

A less common described morphological factor that may contribute to LVOTO is the ALM (also referred to as the muscle bundle of Moulaert) [22], a component of the anterolateral left ventricular wall. Although this factor was not identified as a cause of LVOTO in our clinical patient group, it was present in three of the cardiac specimens and assessed as a contributing factor to LVOT narrowing. The ALM separates the mitral valve's anterior leaflet from the anterior border of the left ventricle, as previously described in specimen with TGA–VSD and pulmonary stenosis [23]. Under normal anatomical circumstances, it is located adjacent to the left coronary semilunar cusp and extends into the anterolateral wall of the left ventricle, being present in ∼40% of normal heart specimens [22]. In hearts with ventriculoarterial discordance, the ALM borders the left pulmonary semilunar cusp [23]. While a discrete node or muscle of the free wall may be amenable to resection, a very radical procedure may not be, and the ALM could still contribute to (multilevel) LVOTO.

Limitations

The findings of this study are limited by its retrospective design, small sample size, patient heterogeneity and the inclusion of a wide range of surgical eras, all of which may introduce bias in the evaluation of outcome. Additionally, because patients from different surgical periods were included, pulmonary annular Z-scores were unavailable for most patients before corrective surgery, and thus, these scores were not reported.

CONCLUSION

Consistent with previous data, this long-term follow-up study of a small series of TGA–VSD–LVOTO patients after ASO demonstrates that patients can achieve favourable clinical outcomes with preserved neoaortic valve function. Reoperations for LVOTO are necessary in only a small subset of cases, specifically when concomitant LVOTO relief was not performed during the initial ASO. Postmortem cardiac specimens illustrate the anatomical diversity of LVOTO and highlight the potential of ASO as an effective surgical solution for these cases, whether addressed early or later. Based on our findings, ASO remains the preferred surgical option for TGA–VSD–LVOTO patients when anatomically feasible.

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None declared.

SUPPLEMENTARY MATERIAL

Supplementary material is available at *ICVTS* online.

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Conflict of interest: none declared.

DATA AVAILABILITY

The datasets generated during and/or analysed during the current study are available from the corresponding author on reasonable request.

Author contributions

Roel L.F. van der Palen: Conceptualization; Data curation; Formal analysis; Methodology; Project administration; Visualization; Writing—original draft. **Abe den Ouden:** Data curation; Formal analysis; Writing—original draft; Writing—review & editing. **Ingmar Knobbe:** Data curation; Writing—review & editing. **Berto J. Bouma:** Data curation; Writing—review & editing. **David R. Koolbergen:** Data curation; Writing—review & editing. **Monique R.M. Jongbloed:** Data curation; Methodology; Visualization; Writing—review & editing. **Mark G. Hazekamp:** Conceptualization; Data curation; Formal analysis; Methodology; Supervision; Writing—review & editing.

Reviewer information

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