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Original Research

Two decades after the arterial switch operation: stable right ventricular function but reduced exercise capacity

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ABSTRACT

Background: Right ventricular (RV) function and exercise capacity predict prognosis in transposition of the great arteries (TGA) after arterial switch operation (ASO). We aim to longitudinally evaluate RV dimensions, global function and exercise capacity after ASO, comparing patients with and without RV pressure overload.
Methods: This retrospective study included TGA patients post-ASO with two cardiovascular magnetic resonance (CMRs) examinations at the University Medical Center Utrecht between 2004 and March 2024. Cardiac volumes, function, strain, and vessel dimensions were measured. Patients were categorized by RV pressure overload. Repeated exercise tests were performed in a subset. The first and second CMR were compared.
Results: The cohort (111 patients, 22 ± 8 years; 71% male (79/111)) underwent the first CMR at median 13 [11–19] years post-ASO (mid-term follow-up) and the second at 21 [16–26] years post-ASO (long-term follow-up). RV volumes, function, and strain remained stable during long-term follow-up. Aortic root dimensions showed no progression during long-term follow-up (diameter: 23 ± 5 mm/m^2 vs. 20 ± 4 mm/m^2, $p < 0.001$). 50% (56/111) underwent exercise testing, revealing a VO2peak decline, with 25% (14/56) having reduced VO2peak at mid-term follow-up and 46% (26/56) at long-term follow-up (mean age 21 ± 7 years) ($p = 0.012$). This was not related to peak heart rate or chronotropic index (peakHR: $R = 0.115$, $p = 0.413$; chronotropic index: $R = 0.099$, $p = 0.484$). No differences were observed between patients with and without RV pressure overload.
Conclusion: Long-term exercise capacity is impaired in a significant portion of TGA patients. RV volumes, global function, strain, and aortic root dimensions remained unchanged during long-term follow-up post-ASO.

1. Introduction

Transposition of the great arteries (TGA), corrected by arterial switch operation (ASO) often combined with the Lecompte maneuver, was first performed by Dr. Jatene in 1974. It is now the procedure of choice with most patients reaching adulthood [1–3]. However, up to 25% require reinterventions, often due to pulmonary artery (PA) stenosis, mainly caused by PA traction and compression by a dilated aortic root, leading to increased right ventricular (RV) afterload, RV dysfunction, and decreased exercise capacity [4–6]. RV systolic function is an independent predictor of prognosis [7]. Mid-term follow-up studies (< 20 years after ASO) show relatively preserved RV function and

Abbreviations: ASO, arterial switch operation; CMR, cardiovascular magnetic resonance; CPET, cardiopulmonary exercise testing; LV, left ventricle; PA, pulmonary artery; PBFM, pulmonary blood flow maldistribution; RV, right ventricle; TGA, transposition of the great arteries; IVS, interventricular septum; FWGLS, free wall global longitudinal strain; VO2peak, peak oxygen uptake; Wpeak, peak workload; HRpeak, peak heart rate; VSD, ventricular septal defect

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exercise capacity within the lower normal limits [8–12]. However, RV function and exercise capacity have never been assessed during long-term follow-up. The aim of this study is to longitudinally evaluate RV dimensions, RV function, and exercise capacity after ASO using cardiac magnetic resonance (CMR) imaging and cardiopulmonary exercise testing (CPET). Additionally, RV remodeling, function and exercise capacity are compared between patients with and without RV pressure overload.

2. Methods

2.1. Study population

This retrospective longitudinal cohort study included TGA patients with two CMR follow-up examinations after ASO at the University Medical Center Utrecht between July 2004 and March 2024. Patients were divided into subgroups with or without RV pressure overload, defined by increased RV systolic pressure (> 30 mmHg) or interventricular septal (IVS) post-systolic flattening using echocardiography, CMR, or right heart catheterization [13]. Outcomes were evaluated for the entire study population, as well as for patients with and without RV pressure overload, by comparing the first CMR with the second CMR. This study was approved by the Institutional Ethics Committee of the University Medical Center Utrecht and due to the extensive design of this study, the right of no objection was used (23U-0357).

2.2. Cardiac magnetic resonance imaging

CMR was performed on a 1.5 Tesla scanner (Philips Medical Systems, Best, the Netherlands). Biventricular volumes, function, and strain were post-processed using Circle Cardiovascular Imaging (CVI42, version 5.12.4, Calgary, Alberta, Canada) by manual delineation of the endocardial and epicardial ventricular borders (Fig. 1) [6]. Papillary muscles and trabeculae were included in the ventricular wall mass (method 1) and as part of ventricular volume (method 2). RV dysfunction was defined as RV ejection fraction (RVEF) < 48% [10]. RV remodeling was defined as RV hypertrophy or RV dilatation, which were indicated by increased RV mass, relative RV wall thickness, and RV end-diastolic volume. The feature tracking module of the software was used to measure RV free wall global longitudinal strain (RV FWGLS) from the 4-chamber cine images and LV global strain from the 2-, 3-, and 4-chamber cine images. Biventricular circumferential strain values were derived from a stack of short-axis cine images. Aortic root diameters were measured from cusp-to-cusp using the PACS workstation (ISD7®, Sectra). Radiology reports provided PA and aortic dimensions and flow, obtained as described before [14]. All diameters, volumes, and masses were indexed (i) for body surface area (BSA) according to the Dubois formula. In case of ovoid vessel shape, the longest and shortest perpendicular dimensions were averaged, according to institutional agreements. Pulmonary blood flow maldistribution (PBFM) was defined as the discrepancy in flow from the normal distribution (LPA 45% vs. RPA 55%).

2.3. Cardiopulmonary exercise testing

A subgroup underwent two CPETs using an electronically braked cycle ergometer (Lode Corrival, Lode BV, Groningen, The Netherlands) according to the Godfrey ramp protocol for children and stepwise 20 W/min incremental protocol for adults [14,15]. Maximum effort was defined as a peak respiratory exchange ratio (RER) > 1.0 for children and > 1.1 for adults. Outcomes included rest and peak systolic blood pressure, peak heart rate (HRpeak, bpm), peak oxygen uptake (VO2-peak, mL/min/kg) and peak workload (Wpeak, W). Reference values and Z-scores were calculated as previously described [16]. Reduced exercise capacity was defined as reduced VO2peak with Z-score < −2. Heart rate reserve was calculated as the difference between peak and resting heart rates. The chronotropic index was calculated as (HRpeak – resting heart rate) / (190 – age – resting heart rate) in case < 18 years old and (HRpeak – resting heart rate) / (220 – age – resting heart rate) for ≥18 years old, with a value below 0.8 indicating chronotropic incompetence [16,17].

2.4. Statistical analysis

Statistical analysis was performed using SPSS Statistics (version 29.0, IBM, Armonk, New York), and figures were created with Graphpad Prism (version 10.2.0, San Diego, California). Variables were presented as mean ± SD, median and IQR, or frequencies (%). Comparisons were made using independent sample t-tests, paired-samples t-tests, chi-square tests, Fisher's exact tests, or McNemar tests. Changes between mid-term and long-term follow-up across subgroups were analyzed using independent sample t-tests. Correlations were assessed using Pearson's correlation coefficient. Results were deemed statistically significant at a two-tailed p-value < 0.05.

3. Results

3.1. Study population

We included 111 TGA patients, predominantly male (71%, (79/

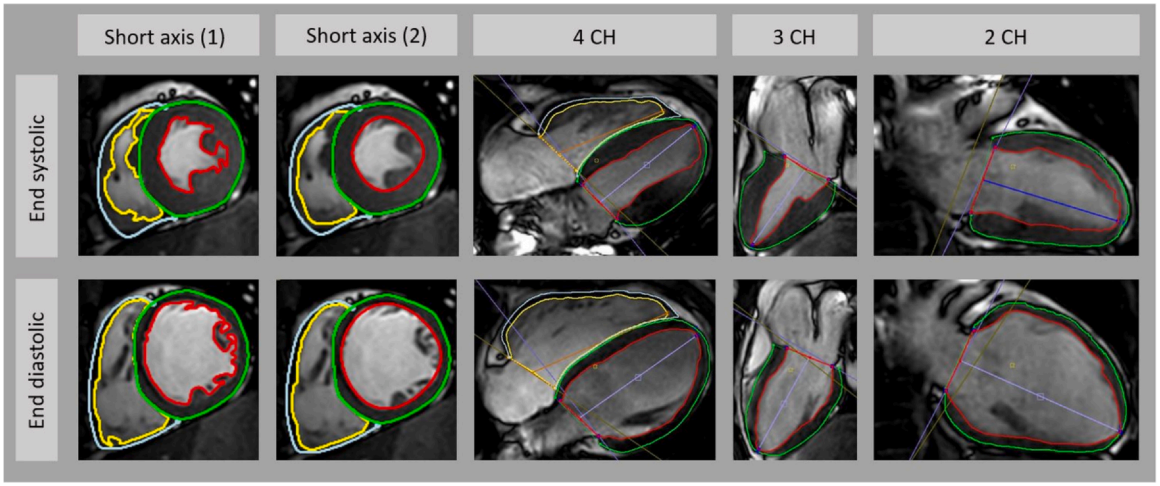

Fig. 1. Example of CMR contouring. Including papillary muscles and trabeculae in the ventricular wall mass (method 1) and as part of the ventricular volume (method 2). *CMR* cardiovascular magnetic resonance

Table 1
Patient characteristics.

	TGA patients (n = 111)
Male, n (%)	79 (71)
Age CMR at mid-term follow-up (yrs), mean ± SD	15 ± 6
BSA CMR at mid-term follow-up (m^2), mean ± SD	1.5 ± 0.4
Resting heart rate CMR at mid-term follow-up (bpm), mean ± SD	72 ± 14
Diagnosis, n (%)	
• TGA-intact ventricular septum	66 (59)
• TGA-VSD	40 (36)
• Taussig-Bing anomaly	5 (5)
Age at ASO (days), median [IQR]	8 [6–13]
Weight at ASO (kg), mean ± SD	3.6 ± 0.5
Lecompte, n (%)	104 (94)
Two stage ASO, n (%)	8 (7)
Aortic valve/root intervention before, n (%)	4 (4)
• AoV plasty	2 (50)
• Dacron prosthesis between AoV and AoAsc	1 (25)
• AoV replacement with prosthesis	1 (25)
PA intervention between, n (%)	13 (12)
Coronary artery intervention between, n (%)	NA
Aortic valve/root intervention between, n (%)	4 (4)
• Bentall procedure	1 (25)
• Yacoub procedure	1 (25)
• Valve-sparing aortic root replacement with prosthesis	1 (25)
• Valve-sparing ascending aorta replacement with prosthesis	1 (25)
Time between mid-term and long-term follow-up CMR (yrs), mean ± SD	7 ± 3
Age CMR at mid-term follow-up (yrs), mean ± SD	15 ± 6
Age CMR at long-term follow-up (yrs), mean ± SD	22 ± 8
RV overload, n (%)	
• No RV overload	87 (78)
• RV pressure overload	24 (22)

AoAsc = ascending aorta; *AoV* = aortic valve; *ASO* = arterial switch operation; *BSA* = body surface area; *CMR* = cardiac magnetic resonance; *IQR* = interquartile range; *IVS* = intact ventricular septum; *NA* = not applicable; *RV* = right ventricle; *SD* = standard deviation; *TGA* = transposition of the great arteries; *VSD* = ventricular septum defect.
± = plus or minus.

111)) with a mean age of 15 ± 6 years (Table 1). Fifty-nine percent (66/111) had simple TGA with intact ventricular septum , whereas 36% (40/111) had TGA with a ventricular septal defect (VSD) and 5% (5/111) had a Taussig-Bing anomaly. ASO was performed at a mean age of 8 days [6–13] and weight of 3.6 ± 0.5 kg. Most patients (94%, (104/111)) underwent the Lecompte maneuver, and 5% (8/111) had a two-stage ASO. The first CMR, conducted at a median of 13 [11–19] years post-ASO, was defined as mid-term follow-up. The second CMR, conducted at a median of 21 [16–26] years post-ASO, was defined as long-term follow-up. These time intervals reflect the median values for the entire cohort, although individual variations may exist. Few aortic root interventions occurred before (n = 4) and between (n = 4) CMRs, with no coronary interventions. PA interventions were performed in 13 out of 111 patients (12%, (13/111)) between CMRs, involving the MPA (n = 1), bifurcation (n = 1), LPA (n = 4), RPA (n = 4), and bilateral (n = 3). Among the cohort, 78% (87/111) did not have RV pressure overload, the remaining 22% did (24/111) (n = 13 diagnosed based on invasive RV pressures during right heart catheterization and n = 11 diagnosed based on RV pressures or post-systolic IVS flattening using non-invasive imaging).

3.2. Biventricular dimensions and function

A total of 222 CMR's in a cohort of 111 TGA patients were examined. During mid-term and long-term follow-up, RV volumes and global RV function remained stable (all $p \geq 0.05$) (Table 2, supplemental Fig. 1A-B). RV FWGLS decreased over time (−21 ± 3% vs. −20 ± 3%, p = 0.011). Cardiac output, RV mass, RV relative wall thickness, and RV trabecular mass increased between mid-term and long-term follow-up (all $p < 0.05$). LV dimensions and function were preserved and stable over time (all $p \geq 0.05$). Results were consistent regardless of papillary muscle and trabeculation inclusion (Supplemental Table 1).

3.3. Great vessels

PA diameters and PA stroke volume (difference between forward and backward flow) were available in 83 out of 111 patients (75%) and remained stable during long-term follow-up. PBFM showed a trend toward a decrease in patients with RV pressure overload between mid-term and long-term follow-up, while it remained stable in those without RV pressure overload (RV pressure overload: 16 ± 13% vs. 10 ± 7%, p = 0.067; no RV pressure overload: 6 ± 5% vs. 6 ± 5%, p = 0.985) (supplemental Table 2). Images for aortic root dimensions were available for 94 out of 111 patients (85%). There was no progression in aortic root diameter during follow-up (aortic root diameter: 23 ± 5 mm/m^2 vs. 20 ± 4 mm/m^2, $p < 0.001$). No aortic dissections were reported. Aortic valve regurgitation remained minimal and stable (4 ± 6% vs. 5 ± 9%, p = 0.062). Two patients had a bicuspid aortic valve (1.8%, (2/111)). Eight distinct patients underwent aortic valve/root interventions prior to mid-term follow-up CMR and between CMRs (Table 1). Indications for aortic root interventions were severe aortic valve regurgitation (n = 6), severe aortic root dilatation (n = 1) and impaired exercise capacity, reduced LV function and LV hypertrophy (n = 1). There were no differences among subgroups for RV pressure overload.

3.4. Cardiopulmonary exercise testing

Maximal effort CPETs were conducted in 56 of 111 patients (50%) (Table 3). The first CPET was conducted at a median age of 14 [11–20] years and the second CPET at a median of 20 [17–27] years. RV function and remodeling were similar in patients with and without CPET (data not shown). In the group of RV pressure overload, 2 patients underwent a PA intervention between the CPETs and 10 did not. Wpeak, HRpeak, VO2peak and the chronotropic index decreased during follow-up (Z-score Wpeak: −0.41 ± 1.42 vs. −1.60 ± 2.02, $p < 0.001$; Z-score HRpeak: −0.70 ± 1.24 vs. −1.09 ± 1.53, p = 0.022; Z-score VO2peak: −0.81 ± 1.70 vs. −1.81 ± 1.38, $p < 0.001$; chronotropic index: 0.99 ± 0.1 vs. 0.93 ± 0.2, p = 0.016) (supplemental Fig. 1C). Reduced exercise capacity was found in 46% of the patients (26/56) at long-term follow-up and not associated with heart rate reserve, HRpeak, and weakly associated with chronotropic index (HR reserve: R = 0.194, p = 0.167; Z-score HRpeak: R = 0.116, p = 0.424; chronotropic index: R = 0.331, p = 0.017). Chronotropic incompetence was found in 18% of the patients (10/56) during long-term follow-up. All patients had New York Heart Asso class I. There were no differences across RV pressure overload subgroups.

4. Discussion

This study is the first to examine the longitudinal course of RV function, dimensions, and exercise capacity in TGA patients after ASO. Key findings are (1) RV volumes, global function, and strain remain preserved during long-term follow-up post-ASO, with similar outcomes in patients with and without RV pressure overload, (2) aortic root dimensions stay stable without clinical sequalae; (3) exercise capacity is

Table 2
Longitudinal biventricular CMR parameters in subgroups RV pressure overload.

	All TGA (n = 111)			No RV pressure overload (n = 87)			RV pressure overload (n = 24)		
	Mid-term FU	Long-term FU	p	Mid-term FU	Long-term FU	p	Mid-term FU	Long-term FU	p
Left ventricle									
LVEDV (mL/m^2), mean ± SD	85 ± 18	85 ± 18	0.57	85 ± 17	86 ± 18	0.416	83 ± 19	79 ± 18	0.180
LVESV (mL/m^2), mean ± SD	33 ± 10	34 ± 12	0.198	33 ± 10	36 ± 13	**0.040**	32 ± 9	30 ± 10	0.312
LVSV (mL/m^2), mean ± SD	51 ± 11	51 ± 9	0.367	52 ± 10	51 ± 9	0.470	50 ± 14	49 ± 10	0.549
CO, (l/min/m^2), mean ± SD	5.4 ± 1.6	6.5 ± 1.6	< **0.001**	6.1 ± 1.3	5.9 ± 1.6	0.271	5.1 ± 2.0	6.1 ± 1.4	**0.005**
LVM (g/m^2), mean ± SD	62 ± 14	69 ± 16	< **0.001**	63 ± 13	71 ± 16	< **0.001**	58 ± 15	64 ± 18	0.053
LV wall thickness (g/mL), mean ± SD	0.74 ± 0.17	0.83 ± 0.19	< **0.001**	0.8 ± 0.2	0.8 ± 0.2	< **0.001**	0.7 ± 0.2	0.8 ± 0.3	**0.007**
LVEF (%), mean ± SD	61 ± 7	60 ± 7	0.388	61 ± 6	60 ± 7	0.052	61 ± 8	63 ± 5	0.263
LV GLS (%), mean ± SD	−17 ± 3	−17 ± 3	0.135	−17 ± 3	−16 ± 3	0.273	−18 ± 2	−17 ± 4	0.293
LV GCS (%), mean ± SD	−18 ± 2	−18 ± 2	0.112	−18 ± 2*	−17 ± 2*	**0.002**	−17 ± 3*	−18 ± 3*	0.306
Right ventricle									
RVEDV (mL/m^2), mean ± SD	84 ± 17	83 ± 15	0.738	84 ± 15	84 ± 14	0.828	83 ± 24	82 ± 17	0.782
RVESV (mL/m^2), mean ± SD	35 ± 10	36 ± 10	0.498	35 ± 9	36 ± 10	0.552	36 ± 13	36 ± 12	0.754
RVSV (mL/m^2), mean ± SD	48 ± 11	48 ± 9	0.426	49 ± 10	48 ± 8	0.322	47 ± 13	48 ± 12	0.810
RVM (g/m^2), mean ± SD	21 ± 5	22 ± 6	**0.005**	20 ± 4	22 ± 5	**0.008**	22 ± 7	24 ± 7	0.263
RV wall thickness (g/mL) mean ± SD	0.25 ± 0.1	0.27 ± 0.1	**0.002**	0.2 ± 0.0	0.3 ± 0.1	**0.003**	0.3 ± 0.1	0.3 ± 0.1	0.172
RV trabeculation (%), mean ± SD	9 ± 7	11 ± 8	**0.047**	8 ± 7	10 ± 6	0.054	10 ± 9	12 ± 11	0.453
RVEF (%), mean ± SD	58 ± 7	57 ± 7	0.273	58 ± 6	58 ± 7	0.453	58 ± 8	56 ± 7	0.432
RV FWGLS (%), mean ± SD	−21 ± 3	−20 ± 3	**0.011**	−21 ± 3	−20 ± 3	**0.032**	−22 ± 4	−20 ± 2	0.176
RV GCS (%), mean ± SD	−16 ± 3	−16 ± 3	0.346	−16 ± 3	−15 ± 3	0.101	−16 ± 3	−16 ± 3	0.487
Ratio RV FWGLS: RV CS, mean ± SD	1.4 ± 0.3	1.4 ± 0.3	0.401	1.4 ± 0.3	1.4 ± 0.3	0.976	1.4 ± 0.4	1.3 ± 0.2	0.130

Papillary muscles and trabeculations included in ventricular mass

CMR = cardiac magnetic resonance; *CO* = cardiac output; *EDV* = end-diastolic volume; *EF* = ejection fraction; *ESV* = end-systolic volume; *FU* = follow-up; *FWGLS* = free wall global longitudinal strain; *GCS* = global circumferential strain; *GLS* = global longitudinal strain; *LV* = left ventricle; *M* = mass; *RV* = right ventricle; *SD* = standard deviation; *SV* = stroke volume; *TGA* = transposition of the great arteries

Bold values indicates significant value of $p < 0.05$.

± = plus or minus.

* $p < 0.05$ change between no RV pressure overload and RV pressure overload.

Table 3
Longitudinal exercise measurements in subgroups RV pressure overload.

	All TGA (n = 56)			No RV pressure overload (n = 44)			RV pressure overload (n = 12)		
	Mid-term FU	Long-term FU	p	Mid-term FU	Long-term FU	p	Mid-term FU	Long-term FU	p
SBP rest (mmHg), mean ± SD	120 ± 16	126 ± 14	0.381	121 ± 16	126 ± 15	0.503	119 ± 17	123 ± 12	0.416
SBP peak (mmHg), mean ± SD	174 ± 27	188 ± 26	0.052	175 ± 26	188 ± 28	0.078	166 ± 29	188 ± 18	0.436
HRpeak (bpm), mean ± SD	183 ± 11	178 ± 15	**0.007**	184 ± 11	180 ± 15	**0.031**	178 ± 9	172 ± 11	0.115
HRpeak (Z-score), mean ± SD	−0.70 ± 1.24	−1.09 ± 1.53	**0.022**	−0.6 ± 1.27	−0.96 ± 1.51	0.065	−1.01 ± 1.1	−1.58 ± 1.57	0.196
HRpeak < −2 Z-score, n (%)	7 (13)	10 (18)	0.453	6 (14)	7 (16)	1.000	1 (8)	3 (25)	0.500
HR reserve (bpm), mean ± SD	105 ± 19	99 ± 16	0.082	105 ± 20	100 ± 16	0.192	103 ± 15	96 ± 16	0.080
Chronotropic index, mean ± SD	0.99 ± 0.1	0.93 ± 0.2	**0.016**	1.01 ± 0.2	0.95 ± 0.2	**0.032**	0.92 ± 0.1	0.87 ± 0.1	0.293
VO2peak (mL/min/kg), mean ± SD	42 ± 10	35 ± 8	< **0.001**	43 ± 10	36 ± 8	< **0.001**	37 ± 7	32 ± 8	0.065
VO2peak (Z-score), mean ± SD	−0.81 ± 1.70	−1.81 ± 1.38	< **0.001**	−0.65 ± 1.77	−1.64 ± 1.25	< **0.001**	−1.48 ± 1.20	−2.42 ± 1.71	0.092
VO2peak < −2 Z-score, n (%)	14 (25)	26 (46)	**0.012**	10 (23)	17 (39)	0.077	4 (33)	9 (75)	0.125
Wpeak (W), mean ± SD	176 ± 66	204 ± 59	**0.001**	184 ± 66	210 ± 61	**0.005**	149 ± 58	179 ± 48	0.093
Wpeak (Z-score), mean ± SD	−0.41 ± 1.42	−1.60 ± 2.02	< **0.001**	−0.30 ± 1.32	−1.64 ± 2.16	< **0.001**	−0.80 ± 1.76	−1.43 ± 1.43	0.088
Wpeak < −2 Z-score, n (%)	10 (18)	24 (43)	**0.039**	6 (14)	17 (39)	0.065	4 (33)	7 (58)	1.000

FU = follow-up; *HR* = heart rate; *HRpeak* = peak heart rate; *RV* = right ventricle; *SBP* = systolic blood pressure; *SD* = standard deviation; *TGA* = transposition of the great arteries; *VO2peak* = peak oxygen uptake; *Wpeak* = peak workload
± = plus or minus.

generally normal during mid-term follow-up but significantly decreases in a subset of patients during long-term follow-up.

4.1. Ventricular volumes and function

RV function remains preserved during long-term follow-up after ASO, aligning with findings from mid-term follow-up studies [8–10,18]. RV mass, RV relative wall thickness and RV trabecular mass increased between mid-term and long-term follow-up in the entire study population. Patients without RV pressure overload showed significant increases in RV mass, relative wall thickness and a trend toward higher trabecular mass, while those with RV pressure overload only showed trends. This is likely multifactorial. The small sample size of patients with RV pressure overload may explain the observed trends rather than significant findings. Additionally, TGA patients may already have some RV pressure overload, possibly due to PA stretching after the Lecompte maneuver, while not visible yet on conventional imaging, as conventional imaging often underestimates RV pressures compared to invasive measurements in these patients [19]. This study pioneers the longitudinal assessment of RV strain in TGA patients using CMR feature tracking. Of interest, RV FWGLS remained within normal limits during mid-term and long-term follow-up, indicating preserved RV function with robust techniques [6,8]. RV strain has superior prognostic value in congenital heart disease compared to conventional imaging parameters like RVEF measured on CMR, but studies after ASO are limited and lack disease-specific reference values. No differences were observed in RV volumes, function and strain across subgroups for RV pressure overload, even when accounting for PA interventions. This might be attributed to small sample sizes or limited RV pressure overload.

4.2. Aortic root diameter

Patients showed no progression in aortic root dimensions during follow-up, consistent with previous studies [20,21]. However, controversy about progression of aortic root dimensions during follow-up exists, likely due to measurement variations and adjustments for BSA or Z-scores [22,23]. Factors like PA banding contribute to aortic root dilatation in TGA patients [20, 21, 23–25]. In the present study, PA banding was not associated with aortic root dimensions, possibly due to a low number of PA banding cases.

4.3. Exercise capacity

TGA patients had exercise capacity within the lower normal range during mid-term follow-up after ASO, consistent with prior research [11, 12, 26, 27]. However, data on long-term exercise performance are scarce and controversial. Exercise capacity decreased during long-term follow-up, with 46% of the patients (26/56) showing impaired exercise capacity, indicating that longer follow-up predicts lower VO2peak [28]. This is concerning due to its association with reduced quality of life and adverse prognosis. No significant changes were found across subgroups, likely due to the small sample sizes or the effects of PA interventions. Longer follow-up, reduced physical activity, abnormal coronary flow reserve, chronotropic incompetence, and intrinsic ventricular function may contribute to reduced VO2peak [12, 29, 30]. Chronotropic incompetence was found in 18% of our patients (10/56), consistent with previous studies, but likely affects exercise capacity in only a subset [11, 18, 26]. The weak correlation between HRpeak and VO2peak suggests that reduced VO2peak may be more related to stroke volume limitations rather than heart rate response. It is suggested that intrinsic ventricular function is a more significant determinant of maximal cardiac index than any variation in chronotropy [31]. RV intrinsic contractility may already be impaired after ASO, although not evident on conventional CMR [6]. Further investigation is needed to fully understand its impact on exercise capacity.

5. Limitations

The study's main limitations include its retrospective design and missing CPET data. Selection bias is present due to inclusion of patients with two CMRs and CPETs during follow-up. Additionally, data from radiology reports and post-processing may introduce inter-observer variability. PA interventions between mid-term and long-term follow-up could have affected results, and larger sample sizes are needed to address this.

5.1. Clinical implications

Using conventional CMR, we observed stable RV volumes, global function, strain, and aortic root dimensions as TGA patients mature into adulthood. However, exercise capacity declined and was impaired in a substantial portion of the patients, potentially linked to impaired stroke volume during exercise rather than chronotropic incompetence. Further research should investigate how intrinsic ventricular function influences reduced exercise capacity in this population.

6. Conclusion

Exercise capacity declines over time and is significantly impaired in a substantial portion of TGA patients during long-term follow-up after ASO. RV volumes, global function, strain, and aortic dimensions remained stable. This highlights a concerning trend that requires further attention.

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Author contributions

Renée S. Joosen: Writing – original draft, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Marielle C. van de Veerdonk:** Writing – review & editing, Writing – original draft, Supervision, Conceptualization. **Anneloes E. Bohte:** Writing – review & editing, Conceptualization. **Tim Takken:** Writing – review & editing, Conceptualization. **Abraham van Wijk:** Writing – review & editing. **Michael G. Dickinson:** Writing – review & editing. **Gregor J. Krings:** Writing – review & editing. **Michiel Voskuil:** Writing – review & editing, Writing – original draft, Supervision. **Johannes M.P.J. Breur:** Writing – review & editing, Writing – original draft, Supervision, Funding acquisition, Conceptualization.

Data availability statement

Due to the nature of the data and the right of no objection, the anonymous dataset will only be available after a granted collaboration with the corresponding author.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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None.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.jocmr.2025.101899.

References

[1] Jatene AD, Fontes VF, Paulista PP, Souza LC, Neger F, Galantier M, et al. Anatomic correction of transposition of the great vessels. J Thorac Cardiovasc Surg 1976;72(3):364–70.
[2] Lecompte Y, Neveux JY, Leca F, Zannini L, Tu TV, Duboys Y, et al. Reconstruction of the pulmonary outflow tract without prosthetic conduit. J Thorac Cardiovasc Surg 1982;84(5):727–33.
[3] Engele LJ, van der Palen RLF, Joosen RS, Sieswerda GT, Schoof PH, van Melle JP, et al. Clinical course of TGA after arterial switch operation in the current era. JACC Adv 2024;3(2):100772.
[4] van der Palen RLF, Blom NA, Kuipers IM, Rammeloo LAJ, Jongbloed MRM, Konings TC, et al. Long-term outcome after the arterial switch operation: 43 years of experience. Eur J Cardiothorac Surg 2021;59(5):968–77.
[5] Morgan CT, Mertens L, Grotenhuis H, Yoo SJ, Seed M, Grosse-Wortmann L. Understanding the mechanism for branch pulmonary artery stenosis after the arterial switch operation for transposition of the great arteries. Eur Heart J Cardiovasc Imaging 2017;18(2):180–5.
[6] Joosen RS, Voskuil M, Krings GJ, Handoko ML, Dickinson MG, van de Veerdonk MC, et al. The impact of unilateral pulmonary artery stenosis on right ventricular to pulmonary arterial coupling in patients with transposition of the great arteries. Catheter Cardiovasc Inter 2024;103(6):943–8.
[7] Egbe AC, Miranda WR, Stephens EH, Anderson JH, Andi K, Goda A, et al. Right ventricular systolic dysfunction in adults with anatomic repair of d-transposition of great arteries. Am J Cardiol 2023;192:101–8.
[8] Schuwerk R, Freitag-Wolf S, Krupickova S, Gabbert DD, Uebing A, Langguth P, et al. Ventricular and atrial function and deformation is largely preserved after arterial switch operation. Heart 2021;107(20):1644–50.
[9] Voges I, Boll C, Caliebe A, Gabbert D, Uebing A, Krupickova S. Reference values for ventricular volumes and pulmonary artery dimensions in pediatric patients with transposition of the great arteries after arterial switch operation. J Magn Reson Imaging 2021;54(4):1233–45.
[10] Shepard CW, Germanakis I, White MT, Powell AJ, Co-Vu J, Geva T. Cardiovascular magnetic resonance findings late after the arterial switch operation. Circ Cardiovasc Imaging 2016;9(9).
[11] Giardini A, Khambadkone S, Rizzo N, Riley G, Pace Napoleone C, Muthialu N, et al. Determinants of exercise capacity after arterial switch operation for transposition of the great arteries. Am J Cardiol 2009;104(7):1007–12.
[12] de Koning WB, van Osch-Gevers M, Ten Harkel AD, van Domburg RT, Spijkerboer AW, Utens EM, et al. Follow-up outcomes 10 years after arterial switch operation for transposition of the great arteries: comparison of cardiological health status and health-related quality of life to those of the a normal reference population. Eur J Pedia 2008;167(9):995–1004.
[13] Feltes TF, Bacha E, Beekman 3rd RH, Cheatham JP, Feinstein JA, Gomes AS, et al. Indications for cardiac catheterization and intervention in pediatric cardiac disease: a scientific statement from the American Heart Association. Circulation 2011;123(22):2607–52.
[14] Baggen VJ, Driessen MM, Meijboom FJ, Sieswerda GT, Jansen NJ, van Wijk SW, et al. Main pulmonary artery area limits exercise capacity in patients long-term after arterial switch operation. J Thorac Cardiovasc Surg 2015;150(4):918–25.
[15] Adami A, Sivieri A, Moia C, Perini R, Ferretti G. Effects of step duration in incremental ramp protocols on peak power and maximal oxygen consumption. Eur J Appl Physiol 2013;113(10):2647–53.
[16] Bongers BC vBM, Hulzebos EHJ, Takken T. Pediatric norms for cardiopulmonary exercise testing: in relation to sex and age. 's-Hertogenbosch: Uitgeverij BOXPress B.V.,; 2014.
[17] Wilkoff BL, Miller RE. Exercise testing for chronotropic assessment. Cardiol Clin 1992;10(4):705–17.
[18] Khairy P, Clair M, Fernandes SM, Blume ED, Powell AJ, Newburger JW, et al. Cardiovascular outcomes after the arterial switch operation for D-transposition of the great arteries. Circulation 2013;127(3):331–9.
[19] Joosen RS, van der Palen RLF, Udink Ten Cate FEA, Voskuil M, Krings GJ, Bokenkamp R, et al. 30 years' experience in percutaneous pulmonary artery interventions in transposition of the great arteries. JACC Adv 2024;3(11):101327.
[20] McMahon CJ, Ravekes WJ, Smith EO, Denfield SW, Pignatelli RH, Altman CA, et al. Risk factors for neo-aortic root enlargement and aortic regurgitation following arterial switch operation. Pediatr Cardiol 2004;25(4):329–35.
[21] Schwartz ML, Gauvreau K, del Nido P, Mayer JE, Colan SD. Long-term predictors of aortic root dilation and aortic regurgitation after arterial switch operation. Circulation 2004;110(11 1):II128–32.
[22] van der Bom T, van der Palen RL, Bouma BJ, van Veldhuisen SL, Vliegen HW, Konings TC, et al. Persistent neo-aortic growth during adulthood in patients after an arterial switch operation. Heart 2014;100(17):1360–5.
[23] van der Palen RLF, van der Bom T, Dekker A, Tsonaka R, van Geloven N, Kuipers IM, et al. Progression of aortic root dilatation and aortic valve regurgitation after the arterial switch operation. Heart 2019;105(22):1732–40.
[24] Sievers HH, Lange PE, Arensman FW, Radley-Smith R, Yacoub MH, Harms D, et al. Influence of two-stage anatomic correction on size and distensibility of the anatomic pulmonary/functional aortic root in patients with simple transposition of the

great arteries. Circulation 1984;70(2):202–8.
[25] Baruteau AE, Vergnat M, Kalfa D, Delpey JG, Ly M, Capderou A, et al. Long-term outcomes of the arterial switch operation for transposition of the great arteries and ventricular septal defect and/or aortic arch obstruction. Inter Cardiovasc Thorac Surg 2016;23(2):240–6.
[26] Kuebler JD, Chen MH, Alexander ME, Rhodes J. Exercise performance in patients with D-loop transposition of the great arteries after arterial switch operation: long-term outcomes and longitudinal assessment. Pedia Cardiol 2016;37(2):283–9.
[27] van Wijk SW, Driessen MM, Meijboom FJ, Doevendans PA, Schoof PH, Breur HM, et al. Left ventricular function and exercise capacity after arterial switch operation for transposition of the great arteries: a systematic review and meta-analysis. Cardiol Young 2018;28(7):895–902.
[28] Pasquali SK, Marino BS, McBride MG, Wernovsky G, Paridon SM. Coronary artery pattern and age impact exercise performance late after the arterial switch operation. J Thorac Cardiovasc Surg 2007;134(5):1207–12.
[29] Massin MM, Hovels-Gurich HH, Gerard P, Seghaye MC. Physical activity patterns of children after neonatal arterial switch operation. Ann Thorac Surg 2006;81(2):665–70.
[30] Bengel FM, Hauser M, Duvernoy CS, Kuehn A, Ziegler SI, Stollfuss JC, et al. Myocardial blood flow and coronary flow reserve late after anatomical correction of transposition of the great arteries. J Am Coll Cardiol 1998;32(7):1955–61.
[31] Mahle WT, McBride MG, Paridon SM. Exercise performance after the arterial switch operation for D-transposition of the great arteries. Am J Cardiol 2001;87(6):753–8.