Coronary Artery Problems Late After Arterial Switch Operation for Transposition of the Great Arteries

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Background: The incidence of late coronary artery abnormalities after arterial switch operation (ASO) for d-loop transposition of the great arteries may be underestimated.

Methods and Results: We retrospectively reviewed coronary artery morphology in 40 of 97 patients who survived the first year after ASO. Seven asymptomatic patients developed significant late coronary artery abnormalities. One patient died suddenly at home with severe left coronary artery (LCA) ostial stenosis at age 3.8 years. The second patient collapsed during exercise at age 9.6 years due to ventricular fibrillation and severe LCA ostial stenosis despite prior negative exercise stress test (EST) and myocardial perfusion imaging (MPI). The third patient was found to have moderate ostial stenosis of the LCA with negative EST and MPI. The fourth patient with exercise-induced ST-T depression and myocardial perfusion defect was shown to have complete LCA occlusion with collateral vessel formation. Three other patients had complete proximal obliteration of either of the coronary arteries with collateral supply. An additional 4 asymptomatic patients had trivial-mild narrowing of the LCA on routine selective coronary angiogram.

Conclusions: Incidence of late coronary stenosis or occlusion was not infrequent after ASO (11.3%) and presented usually without preceding symptoms and often after negative non-invasive screening. We advocate routine coronary imaging in all patients after ASO before they participate in competitive sports. (*Circ J* 2015; **79**: 2372–2379)

Key Words: Arterial switch operation; Coronary ostial stenosis; Myocardial ischemia; Sudden death; Transposition of the great arteries

The long-term prognosis of d-loop transposition of the great arteries (d-TGA) significantly improved after the introduction of arterial switch operation (ASO) in the 1970s.^{1–6} Transfer of the coronary arteries during ASO is a critical surgical step that influences early postoperative outcome. Whereas coronary complications are regarded as a major risk factor for early postoperative morbidity and mortality after ASO,^{7–10} the incidence of late coronary problems after ASO is largely unknown. These patients remain generally asymptomatic, and the first manifestation of the coronary abnormality may be circulatory collapse or unexpected sudden death.^{11–13} Patients with severe coronary artery stenosis may be totally asymptomatic with no evidence of myocardial ischemia at rest or with exercise.¹⁴ In the majority of retrospective large cohort studies, the incidence of late coronary artery abnormalities was relatively low, <3%,^{4,15–17} but others reported a higher incidence of 5–8%, after ASO.^{7,18–21} The incidence and pathogenesis of late coronary artery abnormalities after ASO are poorly understood.

After successful ASO, patients with d-TGA are routinely evaluated in the outpatient clinic for growth and development, symptoms, exercise tolerance, and hemodynamic assessment

with non-invasive tests including electrocardiogram (ECG) and echocardiogram. Exercise stress test (EST) and myocardial perfusion imaging (MPI) may be performed to assess aerobic fitness level and myocardial perfusion status. Cardiac catheterization and other imaging modalities, such as computed tomography (CT) and magnetic resonance imaging (MRI), may be indicated for assessment of existing hemodynamic and anatomical abnormalities including stenosis of anastomotic sites, branch pulmonary artery (PA) stenosis, neo-aortic root enlargement, neo-aortic valve regurgitation, and possible coronary artery abnormalities.²² There is no consensus, however, as to how and when coronary artery status can be reliably assessed as a long-term follow-up strategy in asymptomatic patients with d-TGA after ASO.

To investigate the reliability of clinical assessment and non-invasive tests in detecting significant coronary stenosis or total obliteration, we reviewed the medical records of all patients who were followed at Nemours Cardiac Center of Alfred I. duPont Hospital for Children, Wilmington, Delaware, USA after neonatal ASO. We examined clinical presentation at each outpatient encounter, non-invasive cardiac studies, and specific

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Table 1. Patient Clinical Profile

	n=97	%
Male	63	64.9
Anatomical classification		
d-TGA/IVS	66	68.0
d-TGA with VSD	23	22.7
d-TGA with VSD and CoA/arch hypoplasia	3	3.1
Taussig-Bing anomaly (DORV)	5	5.2
d-TGA with single coronary artery	3	3.1
Years of follow-up		
1–2	13	13.4
2–5	18	18.6
5–10	29	30.0
10–15	18	18.6
>15	19	19.6
Tests		
ECG and echocardiogram	97	100
EST	30	30.9
MPI (and EST)	24	24.7
cMRI	29	29.9
Cardiac catheterization	70	72.2
Coronary angiogram	39	40.2
Autopsy	1	1.0

cMRI, cardiac magnetic resonance imaging; CoA, coarctation of aorta; d-TGA, d-transposition of the great arteries; DORV, double outlet right ventricle; ECG, electrocardiogram; EST, exercise stress test; IVS, intact ventricular septum; MPI, myocardial perfusion imaging; VSD, ventricular septal defect.

imaging for coronary morphology, and assessed the incidence and presentation of late coronary abnormalities in these patients.

Methods

Retrospective chart review was performed for all 97 patients who underwent ASO for d-TGA and double outlet right ventricle (DORV; Taussig-Bing anomaly) in the newborn period and who were followed for at least 12 months at the Nemours Cardiac Center of Alfred I. duPont Hospital for Children from 1998 to 2013. Approval for the study was obtained from the Institutional Review Board at Nemours/Alfred I. duPont Hospital for Children. Seventy-one ASO were performed at the Nemours Cardiac Center (73.2%), whereas 26 (26.8%) were done elsewhere. The LeCompte maneuver was used in all patients to establish RV-PA continuity. The coronary ostia were excised with a large “button” of surrounding aortic wall and then reimplanted to the neo-aorta except in the patients with single coronary artery who underwent Takeuchi procedure. Patients who did not survive beyond the first birthday were excluded. These 97 patients were followed regularly in the outpatient cardiology clinic with ECG and echocardiogram for more than 1 year. Echocardiograms were reviewed and finalized by each ordering cardiologist or the Echocardiography Laboratory attending of the day (overseen by J.M.B., Director of Echocardiography Laboratory). Additional diagnostic tests were performed including EST, MPI, MRI, CT/CT angiography, and/or cardiac catheterization, as clinically indicated. EST was performed using a bicycle ergometer or treadmill under the Bruce protocol. The study was reviewed and finalized by a group of cardiologists assigned to the Exercise Laboratory (overseen by B.W.R., Director of Exercise

Table 2. Indications for Coronary Investigation

Indications	n=40	%
Investigation of abnormal hemodynamics on echocardiogram	24	60.0
Routine assessment (sports clearance)	7	17.5
Symptoms	4	10.0
Chest pain	1	
Palpitations (patient 5)	1	
Sudden death-autopsy* (patient 1)	1	
Cardiac arrest/VF* (patient 2)	1	
Unusual coronary anatomy	2	5.0
Abnormal EST or MPI (patients 4, 6)	2	5.0
ASD closure	1	2.5

ASD, atrial septal defect; VF, ventricular fibrillation. *Asymptomatic at outpatient visits. Other abbreviations as in Table 1.

Laboratory). MPI was performed with technetium-99m sestamibi injection, and perfusion imaging was obtained before and after exercise. Studies were read by a radiologist specialized in nuclear medicine. All cardiac catheterizations and angiograms were reviewed by W.R., Director of the Cardiac Catheterization Laboratory. All study results and original images were presented and carefully discussed in a weekly Multidisciplinary Case Conference at Nemours Cardiac Center for final consensus among the group, which included all attending cardiologists, intensivists, surgeons, and anesthesiologists prior to any critical decisions being made.

Results

The demographic features of the 97 patients are listed in **Table 1**. Sixty-five percent of the cohort was male, and nearly 70% had d-TGA with intact ventricular septum. Total duration of follow-up ranged from 1.0 to 25.6 years (median, 8.25 years). All patients had ECG and echocardiogram at regular outpatient clinic visits. Seventy patients (72.2%) underwent more than 1 cardiac catheterization after ASO for hemodynamic assessment and/or interventional procedures. Thirty patients had EST, and 24 patients had both EST and MPI.

Coronary artery anatomy was investigated in 40 patients with angiography (n=39) or autopsy (n=1). Of the 39 patients, 24 underwent selective coronary angiography and 15 underwent neo-aortic root angiography if deemed adequate for coronary assessment. The major indications for cardiac catheterization included (1) significant hemodynamic abnormalities detected on echocardiogram, (2) routine assessment of hemodynamic status and coronary arteries prior to allowing competitive sports in early adolescence, and (3) heart-related symptoms (**Table 2**). Only 2 of the 97 patients complained of symptoms during routine outpatient follow up: 1 patient with chest pain was identified to have non-cardiac chest pain (musculoskeletal chest pain), and the other patient with palpitations was demonstrated to have benign isolated monomorphic ventricular ectopies that were suppressed by exercise (patient 5). Two life-threatening events occurred unexpectedly outside the hospital. None of these 40 patients was receiving cardiac medications.

Eleven patients (11.3%) were found to have coronary artery abnormalities. Seven patients (patients 1–7; **Tables 3,4**) had significant coronary artery abnormalities including the 2 patients who presented with unexpected sudden life-threatening events; 1 died suddenly at home (patient 1) and the other unexpectedly

Table 3. Development of Significant Coronary Abnormalities (n=7)

Patient ID no.	Sex	Age (years) at diagnosis of coronary lesion	Symptoms	Initial diagnosis	Original coronary anatomy	Initial operation	Acquired coronary abnormalities	Coronary revision
1	M	3.8	Sudden death*	d-TGA/IVS	Usual	ASO/LM	Severe stenosis of LCA ostium	NA
2	F	9.6	Cardiac arrest/VF*	d-TGA, VSD	Usual	ASO/LM; VSD closure	Severe stenosis of LCA ostium	Yes
3	M	12.6	None	d-TGA/IVS	Usual	ASO/LM	Moderate stenosis of LCA ostium	Yes
4	M	9.5	None	d-TGA/IVS	LCX from RCA	ASO/LM	Complete obliteration of proximal LAD; Retrograde filling by RCA via collateral vessels	Yes
5	M	15	Palpitation (Isolated PVC)	d-TGA/IVS	Dominant RCA; Collateral flow to hypoplastic LCX, small LAD	ASO/LM	Total obliteration of LCX	No
6	M	0.6	None	d-TGA/IVS	Usual	ASO/LM	Total obliteration of LCA	No
7	M	10.3	None	d-TGA/IVS	Single coronary artery	ASO/LM; Takeuchi procedure†	Obliteration of proximal RCA RCA supplied by collaterals from proximal and distal LCA	No

*Asymptomatic at outpatient visits. †Creation of an aortopulmonary window and intra-PA baffling connecting the aorta to the single coronary artery. ASO, arterial switch operation; LCA, left coronary artery; LCX, left circumflex artery; LM, LeCompte maneuver; NA, not applicable; PA, pulmonary artery; PVC, premature ventricular complex; RCA, right coronary artery. Other abbreviations as in Tables 1,2.

Table 4. Test Results and Outcome of Significant Coronary Abnormalities (n=7)

Patient ID no.	ECG	Echocardiogram	EST	MPI	MRI	Outcome
1	NSR, WNL	Normal RV, LV function; Branch PA not well visualized	ND	ND	Slight narrowing of RPA; Coronary not delineated (3.25 years)	Sudden death (3.75 years)
2	NSR, WNL	Mild RV enlargement, Moderate neo-PI; Mild neo-Ao dilatation	Normal	Normal	ND	Alive
3	NSR, RBBB	Mild RV enlargement, mild branch PA stenosis, mild-moderate neo PI	Normal	Normal	Significant narrowing of LCA orifice	Alive
4	NSR, RBBB	Trivial supralvalvar neo-PS	Max $\dot{V}O_2$ 65% predicted ST-T depression (7 mm) in lateral leads	Reversible perfusion defect	ND	Alive
5	NSR, 1°AVB; Isolated PVC	Mild-moderate neo-AI	Max $\dot{V}O_2$ 72% predicted isolated PVC suppressed by exercise	Normal	Mild dilatation of neo-Ao, mild biventricular dilatation, LCA not well seen	Alive
6	NSR, RVH	Mild MPA stenosis, mild neo-AI	ND	ND	ND	Lost to follow-up
7	NSR, IRBBB	Free neo-PI (s/p transannular patch augmentation of RVOT at 3 months)	ST-T depression (2 mm) in lateral leads	Normal	Moderate RV dilation, mild proximal RPA narrowing, delayed Gd enhancement of LV endocardium	Alive

1°AVB, first-degree atrioventricular block; Gd, gadolinium; IRBBB, incomplete right bundle branch block; LV, left ventricle; MPI, myocardial perfusion imaging; ND, not done; neo-AI, neo-aortic insufficiency; neo-Ao, neo-aorta; neo-PI, neo-pulmonary insufficiency; neo-PS, neo-pulmonary stenosis; NSR, normal sinus rhythm; RBBB, right bundle branch block; RPA, right pulmonary artery; RV, right ventricle; RVH, right ventricular hypertrophy; RVOT, right ventricular outflow tract; s/p, status post; WNL, within normal limits. Other abbreviations as in Tables 1–3.

developed ventricular fibrillation (VF) during exercise (patient 2). Four patients (patients 8–11), all asymptomatic, were found to have only minor coronary anomalies on selective coronary angiogram (Table S1). Nine patients were totally asymptomatic at the routine outpatient visits, but 5 of these were found to have significant coronary artery abnormalities on coronary

angiogram (patients 3–7). Four patients had complete obliteration of one of the coronary artery branches (left anterior descending artery [LAD], n=2; left circumflex artery [LCX], n=1; and right coronary artery [RCA], n=1) with retrograde filling via collateral vessels from the contra-lateral side. One asymptomatic patient had EST and MPI abnormalities (patient

Figure 1. Cross-sectional micrographs of the left coronary artery ostium in patient 1: (A) hematoxylin-eosin staining and (B) Masson's trichrome staining. There is extensive intimal thickening with prominent intimal fibrosis (B, blue) with severely obliterated vascular lumens (arrows). Scale bar, 500 μ m.

Figure 2. Aortic root injection in patient 2 at (A) 4 months and (B) soon after the patient developed near-fatal ventricular fibrillation, at 9.6 years. The orifice of the left main coronary artery is severely narrowed (arrow).

4), and 4 patients (patients 2,3,5,7) had negative EST and MPI despite significant stenosis or total occlusion of one of the major coronary arteries (Table 4). MRI and CT/CT angiogram were performed mainly to assess the anatomy of great vessels inadequately visualized on echocardiogram. MRI detected significant narrowing of the left coronary artery (LCA) orifice in patient 3.

Patient 1 underwent ASO with no significant postoperative complications. At 3 years of age, he presented at the outpatient clinic with normal physical examination without symptoms and normal echocardiographic findings except for mild bilateral branch PA stenosis. MRI confirmed mild narrowing of the PAs but could not delineate coronary artery anatomy. Seven months after this visit, he died suddenly at home after complaining of acute onset of abdominal pain followed by a generalized seizure. Autopsy demonstrated severe ostial stenosis

of the LCA with near-complete luminal obliteration by severe intimal fibrosis (Figure 1).

Patient 2 had ASO and patch repair of perimembranous type ventricular septal defect in the newborn period. At 4 months of age, she underwent surgical revision of residual right ventricular outflow tract (RVOT) obstruction. Aortic root injection at preoperative cardiac catheterization showed unobstructed coronary arteries (Figure 2A). After the surgical RVOT revision, she did well clinically with no symptoms. Echocardiography showed mild residual pulmonary stenosis (PS) and insufficiency with mild RV dilatation. At 8.7 years of age, she had negative EST and MPI with excellent fitness level. Ten months later, however, she had a cardiac arrest during a lacrosse game due to VF. Aortic root injection showed severe stenosis of the LCA ostium (Figure 2B). Intraoperative observation showed that the LCA ostium was severely stenotic and par-

Figure 3. Elective left ventriculogram and selective coronary angiogram at (A) 4 months and (B) 10.9 years, respectively, in patient 3. Although there was no narrowing at the proximal left main coronary artery (LCA) earlier, there was moderate stenosis of the proximal LCA later on (arrow).

Figure 4. Coronary angiogram of patient 4 at 9 years of age. (A) On aortic root injection, the right coronary artery (RCA) is shown to give rise to left circumflex artery (LCX; arrow), but the left anterior descending artery (LAD) is not filled from the aorta, suggesting possible complete obliteration of the proximal LAD. (B) Selective RCA angiogram showing that the LAD (arrows) is filled retrograde from the LCX via collateral vessels.

tially covered with a flap of intima. She underwent pericardial patch augmentation of the left main coronary artery without significant postoperative complications. There was no recurrence of VF after the coronary artery revision.

Patient 3 had surgical revision of supra-aortic PS (mean pressure gradient on echocardiogram, 30 mmHg) and supra-aortic stenosis (mean pressure gradient, 28 mmHg) 4 months after ASO. Cardiac catheterization at 4 months of age showed mild bilateral residual branch PS with no abnormal findings in the coronary arteries (Figure 3A). EST and MPI at 11 years of age showed no abnormal findings with good exercise effort and normal aerobic fitness level for his age. Routine

cardiac catheterization at 12 years of age, however, indicated moderate ostial stenosis of the LCA on selective coronary angiogram (Figure 3B). The coronary lesion was thought to be progressive when compared with the previous angiogram (Figure 3A). The patient underwent coronary revascularization surgery with pericardial patch augmentation of left main ostial stenosis with an uneventful postoperative course.

Patient 4 had an LCX originating from the RCA. After initial ASO, he underwent homograft patch augmentation of the main PA and right PA at 9 months of age. Left ventricle injection suggested unobstructed coronary arteries, but aortic root or selective coronary injection was not performed. The patient

had routine EST and MPI at 9 years of age prior to participating in competitive sports. There were no symptoms induced by exercise, but his fitness level was mildly diminished. At the peak of exercise, ECG showed significant exercise-induced ST-T depression (7 mm) in the lateral leads with simultaneous MPI showing exercise-induced perfusion defect at the apex and the anteroapical wall. Subsequent selective coronary angiography showed complete obliteration of the proximal LAD with collateral supply from small distal LCX and RCA (Figure 4). He underwent revascularization surgery of the occluded proximal LAD at another institution. Three other asymptomatic patients (patients 5, 6, and 7) were found to have total obliteration of 1 coronary artery supplied by collateral vessels originating from the other coronary artery.

None of these 7 patients had specific symptoms referable to myocardial ischemia. The significant coronary abnormalities were not detected until coronary angiography was performed or the patient presented with life-threatening conditions. MRI had limited value in determining coronary abnormality in the present series (Tables 3,4).

Discussion

Coronary artery abnormalities, including stenosis, significant obstruction, or complete obliteration, are known as early and late complications after ASO. The incidence of late coronary abnormalities has not been well documented, because the reported incidences tend to depend upon the intensity of the specific coronary investigations. Patients with late coronary artery abnormalities after ASO are commonly asymptomatic, with no evidence of myocardial ischemia on routine outpatient testing.^{7,18,23} Only sporadic cases of angiogram-proven coronary abnormalities or unexpected sudden death with postmortem evidence of coronary artery occlusion have been reported.^{11,13,21,24} This may not reflect the entire clinical spectrum of abnormal coronary artery changes occurring late after the initial ASO. Herein, we report on 11 (11.3%) of 97 post-ASO patients who developed variable degrees of late coronary artery abnormalities; of these, 7 (7.2%) had significant coronary vascular changes including total obliteration. These numbers are probably underestimating the true incidence, because only 40% of the present post-ASO patients underwent diagnostic coronary angiography (39 of 97).

Several mechanisms have been implicated as contributing factors to the development of coronary artery occlusion, including fibrosis of the suture line surrounding the coronary ostium, mechanical kinking and stretching of the transplanted vessels, and reactive injury related to the surgical procedure.¹⁷ The spatial relationship between the coronary arteries and great vessels after ASO may predispose the coronary arteries to the development of late vascular stenosis.²⁵ The presence of intimal thickening after ASO suggests a specific underlying condition that favors development of vascular remodeling.^{26,27} Pedra et al reported that 50% of 22 children were found to have proximal eccentric intimal thickening on intravascular ultrasound after ASO.²⁷ We observed similar pathological findings in the present patients 1 and 2, which may represent extreme-spectrum post-ASO vascular remodeling. The underlying pathogenesis of intimal thickening remains poorly understood. Acute angle of the coronary origin has been observed frequently with neo-aortic root dilatation,²⁸ particularly in adults late after ASO.²⁹ The underlying mechanisms of coronary artery obstruction after ASO are multifold.

The present patients represent a wide variation in the clinical spectrum of acquired coronary artery problems after ASO,

from unexpected sudden death at rest and near-fatal exercise-induced VF to totally asymptomatic condition with trivial abnormality in coronary arterial architecture. All of the present 7 patients with significant coronary abnormalities were clinically asymptomatic at the outpatient follow up visit. Importantly, none of these coronary abnormalities was detected on history and/or non-invasive studies including ECG and echocardiogram, and only 1 patient had positive screening on EST and MPI (patient 4). Although Khairy et al recently reported a relatively low incidence of late coronary complications after ASO in a larger single-center study,³⁰ the present results suggest that an occurrence of late coronary events including sudden cardiac death is not insignificant and consistent with the reports by other groups.^{18,20,31} Whereas the clinical significance of trivial-mild coronary abnormalities is not entirely clear, some coronary vascular changes may be progressive.²⁰ Post-ASO coronary artery abnormalities may not be detected reliably without specific coronary imaging.¹⁶ It is misleading to assume that lack of clinical symptoms affirms absence of potentially lethal coronary abnormalities that may lead to myocardial ischemia, catastrophic ventricular arrhythmia, or even sudden death. Based upon our experience, we have changed our practice to include assessment for coronary abnormalities after ASO, regardless of physical symptoms. We now routinely perform coronary angiography (preferably selective coronary angiogram) in addition to EST and/or MPI before patients reach the age of participation in competitive sports (usually before age 12). We also frequently perform coronary angiography (or at least neo-aortic root injection) in patients who are undergoing diagnostic or interventional cardiac catheterization for other residual hemodynamic abnormalities.

We found that EST as a screening test for coronary insufficiency after ASO is neither sensitive nor specific. One near-fatal false-negative result was seen in patient 2, whereas 3 false-positive EST changes were confirmed on coronary angiogram (data not shown), given that ST-T segment depression does not necessarily indicate myocardial ischemia in children.³² EST is a useful non-invasive tool in assessing aerobic fitness level and in identifying the nature of exercise-induced symptoms and/or arrhythmias, but its reliability in detecting occult myocardial ischemia after ASO appears to be limited in children and adolescents. Perfusion defects on MPI have been reported in asymptomatic patients after ASO, but the clinical significance has not been well determined in children.³³ Although MPI was true positive for myocardial ischemia in patient 4, the negative studies cannot always rule out the presence of silent myocardial ischemia or significant coronary abnormalities (stenosis/occlusion) in the pediatric population (patients 2, 3). MPI has a limited role in detecting underlying coronary abnormalities.³⁴ Based upon the present data, we emphasize that coronary imaging, especially selective coronary angiography, may be considered as a routine follow-up investigation for late coronary abnormality, as advocated by other groups.^{12,14} At a minimum, those patients with significant coronary abnormalities merit either further coronary revascularization intervention or closer outpatient monitoring under certain exercise restriction.

Although coronary angiography is regarded as the gold standard in assessing coronary artery anatomy, there are several technical challenges for delineation of coronary anatomy after ASO. The location of coronary artery orifices varies after ASO and is not in the position for which the standard coronary catheters are designed. In addition, mismatch between aortic root and ascending aorta sizes complicates catheter curve selection. In most cases, non-standard coronary catheters and/

or manual reshaping are necessary. Proper catheter position and injection techniques are critical for optimum opacification of the orifice where most stenoses are located.²³ Projection angles have to be customized to delineate the orifice and proximal coronary artery segment. These crucial points should be addressed properly to be able to generate a reliable coronary angiogram. CT angiography may be a good alternative for coronary angiography in patients with appropriate heart rates and when state-of-the-art multislice scanners are available. Ou et al. addressed certain advantages of CT angiography over coronary angiography: it allows (1) visualization of topographic context of adjacent structures, (2) multiple viewing angles, and (3) better visualization of coronary ostia or proximal segments.^{25,35}

Overall myocardial perfusion status after ASO may depend on the degree of vascular occlusion and simultaneous collateral vessel formation. Two of the present patients with severe LCA stenosis presented with serious life-threatening conditions, whereas patients with total LCA occlusion remained asymptomatic in the setting of collateral vessel formation. These asymptomatic patients with complete obliteration of 1 vessel and concomitant collateral vessel formation may still be at risk at times of extreme myocardial demand and/or when atherosclerotic changes develop.¹⁷ Surgical intervention may be indicated for those who are symptomatic or who have clinical evidence of myocardial ischemia. The clinical significance of trivial-mild coronary abnormalities after ASO remains unknown. It is possible that this is a static minor vascular abnormality, but it could also represent as an early phase of progressive vascular remodeling.²⁰ Although these relatively minor coronary abnormalities are unlikely to develop into lethal events, we recommend careful long-term follow-up of patients who develop coronary artery problems after ASO.

Study Limitations

This was a retrospective chart review in a single center, and thus, the number of patients studied was limited. In addition, during this period of time, cardiologists had varied practices for assessing patients with d-TGA after ASO. Also, use of cardiac catheterization and coronary angiography was not consistent among the cardiologists at Nemours Cardiac Center. Only a limited number of asymptomatic patients had routine coronary angiography. Thus, the true incidence of acquired coronary abnormalities may be higher in the present patients following ASO.

Conclusions

Late coronary artery abnormality is not a rare complication after ASO. Clinical presentation may be altered by the presence or absence of collateral vessels and the status of myocardial sympathetic denervation/reinnervation after transection of the great arteries during ASO.³⁶ Patients with this abnormality may be asymptomatic until they present with catastrophic life-threatening events. Based upon our experience, and as advocated by others,^{10,20} we recommend coronary angiography or coronary CT angiography to rule out preclinical coronary abnormalities after ASO. Whether repeated coronary imaging at regular intervals is warranted remains to be determined.

Conflict of Interest

None.

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Supplementary Files

Supplementary File 1

Table S1. Clinical presentation in 4 patients with minor coronary abnormalities

Please find supplementary file(s);
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