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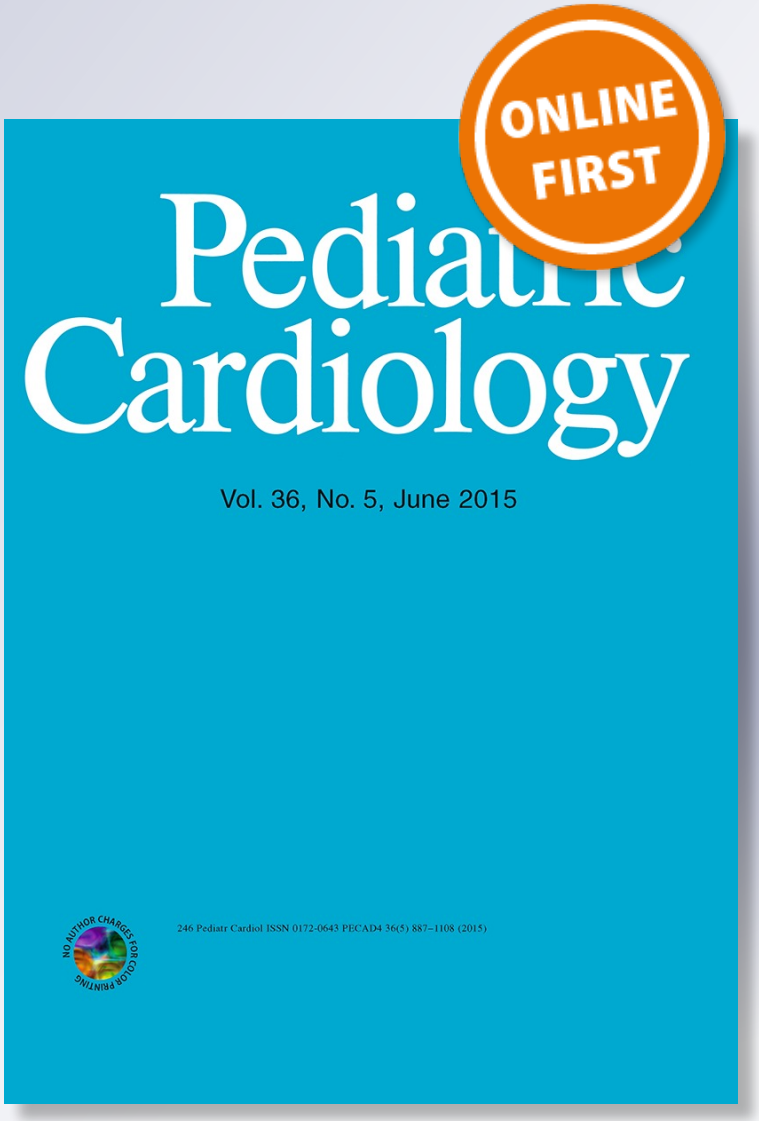

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ORIGINAL ARTICLE

Surgical Results of Anomalous Origin of One Pulmonary Artery Branch from the Ascending Aorta

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Abstract We reviewed our surgical experience with anomalous origin of one pulmonary artery from the ascending aorta (AOPA). From 1989 to 2012, 12 children (five neonates) aged 3–734 days (mean 152 ± 222) with AOPA underwent operations. Eight patients had right AOPA, and four patients had left AOPA. The majority of the patients had elevated right ventricular pressure, with 58 % (7 of 12) demonstrating suprasystemic right ventricular pressure. Surgery was performed by direct anastomosis (group 1) in seven patients and by employing an autologous patch (group 2) in five patients. There were two postoperative mortalities caused by heart failure and pulmonary hypertensive crisis. The mean follow-up duration was 12.6 ± 8 years. Catheterization showed that the right ventricle-to-systemic pressure ratio decreased following operation (preoperative vs. postoperative; 1.13 ± 0.19 vs. 0.48 ± 0.03, $p = 0.043$). There was no difference in the perfusion of the affected lung as measured by the final lung perfusion scan, between the two groups (group 1 vs. group 2; 50.0 ± 10.3 vs. 42.7 ± 28.7 %, $p = 0.158$). Two patients required reoperations for pulmonary regurgitation and pulmonary artery stenosis. There were two catheter-based interventions. At 20 years, survival by the Kaplan–Meier was 91.7 ± 8.0 %, freedom from reoperation was 80.0 ± 17.9 %, and freedom from catheter intervention was 80.8 ± 12.2 %. Early repair of AOPA improves right ventricular pressure and overall hemodynamics with excellent survival and low risk of reintervention. The type of surgical repair did not significantly affect the long-term outcomes (measured via lung perfusion scan).

Keywords Great vessel anomalies · Pulmonary arteries · Aorta · Hemitruncus

Introduction

Anomalous origin of one pulmonary artery (AOPA) from the ascending aorta is rare congenital anomaly, pulmonary artery that originates from the ascending aorta. This should be distinguished from other heart defects associated with an anomalous blood supply to the lungs such as patent ductus arteriosus, major aortopulmonary collateral artery (MAPCA), and truncus arteriosus. This type of cardiac malformation was first described by Fraentzel [8] in 1868. Since then, there have been several case reports and series. Patients were characteristically present with early infantile respiratory distress and heart failure [20].

This anomaly results in a large left-to-right shunt with the entire cardiac output from the right ventricle going to one lung and the other lung receiving blood at a systemic pressure from the aorta. If not corrected early, progressive pathologic changes occur in the lungs with bilateral pulmonary hypertension which can progress to cause severe pulmonary vascular obstructive disease [16]. Surgical treatment is recommended as soon as a diagnosis is confirmed.

Materials and Methods

The retrospective database and chart review were approved by the Institutional Review Board at the Seoul National University Children's Hospital, which waived patient

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consent because of the retrospective data analysis. Clinical records were reviewed to document clinical features, operative procedures, and perioperative courses. All patients had preoperative echocardiogram. Six patients also underwent cardiac catheterization. Computed tomography was used both to confirm diagnosis in four patients and enable the better planning of operations (Fig. 1). These examinations assessed cardiac function and associated anomalies. The following data were obtained from the preoperative echocardiograms: (1) the site of origin of the anomalous pulmonary artery, (2) the presence of additional intracardiac defects, (3) the estimated right ventricular pressure, and (4) the presence or absence of right ventricular dysfunction. Patients' characteristics are detailed in Table 1.

Patients

From June 1989 to April 2012, 12 children, six boys and six girls aged from 3 to 734 days (median age 56 days, mean, 152 ± 222 days), including five neonates, with weights from 2 to 11.6 kg (mean 4.5 ± 2.8 kg), and anomalous origin of pulmonary artery from the ascending aorta (AOPA), underwent surgical repair at Seoul National University Children's Hospital. Common symptoms at presentation were cyanosis and tachypnea in nine and five of the patients, respectively. CATCH 22 was diagnosed in patient 7. Eight patients presented with right AOPA, and four patients presented with left AOPA. All of the AOPA originated from the ascending aorta. The majority of patients had elevated right ventricular pressure, with suprasystemic pressure in seven and systemic pressure in two. Additionally, the right ventricular pressure of two patients with tetralogy of Fallot (TOF) with an absent pulmonary valve and one patient with mild valvular pulmonary stenosis was near systemic. Surgery was performed by direct anastomosis (group 1) in seven patients and by employing an autologous patch (group 2) in five patients.

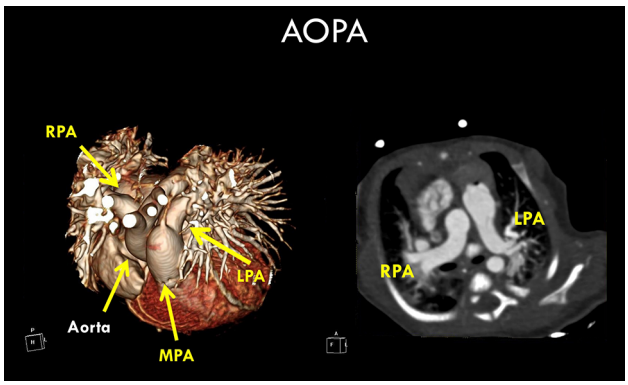

Fig. 1 Preoperative computed tomography image of abnormal origin of the right pulmonary artery from the ascending aorta. *RPA* right pulmonary artery, *LPA* left pulmonary artery, *MPA* main pulmonary artery

Operative Technique

Surgery was undertaken through median full sternotomy, and cardiopulmonary bypass was used in all cases. Ascending aortic and bicaval cannulations were implanted, and mild or moderate hypothermic cardiopulmonary bypass was maintained. Two of twelve patients had repair performed on a beating heart without cross clamping. After excision of the anomalous pulmonary artery with a rim of aortic tissue, all of the patients had primary aorta closures. The implantation of anomalous pulmonary artery to the main pulmonary artery was performed with an emphasis on native tissue-to-tissue anastomosis, which required being aggressive in mobilizing the AOPA and main pulmonary artery with a maximum length and flexible reconstruction. If the distance between the AOPA and the main pulmonary artery was small enough to permit direct anastomosis, an autologous pericardial patch was employed to enlarge the anterior or inferior aspect for anastomosis. Seven patients had direct implantation of anomalous pulmonary artery onto the side of the main pulmonary artery, five patients had glutaraldehyde-fixed autologous pericardial patch augmentation of the anastomosis to maintain a tension-free anastomosis, four patients had an anterior side augmentation (Fig. 2), and one patient had an inferior side augmentation (Fig. 3). In patients 2 and 12, who were diagnosed with TOF with an absent pulmonary valve, total repair and main pulmonary artery angioplasty were performed with pericardial patches, with the left pulmonary arteries being implanted directly to the side of the main pulmonary arteries without the use of additional pericardial math materials. In patient 7, the ascending aorta was transected, just above and beneath the origin of the right AOPA; the superior side of the right pulmonary artery was reconstructed with primary closure of the aortic ring; and an autologous pericardial patch was employed to augment the inferior side of the right pulmonary artery (Fig. 3). In patient 10, following the previous unifocalization of the two left-side MAPCAs, the right and main pulmonary arteries were reconstructed with pericardial patches and a Carpentier–Edward valved conduit (16 mm) for complete repair. Associated procedures undertaken included patent ductus arteriosus (PDA) ligation or division in eight patients, closure of the ventricular septal defect (VSD) in six patients, and coarctoplasty in two patients. The additional defects and surgical details are outlined in Table 1.

Results are reported as the mean ± SD. Probability of survival, freedom from reoperation, and freedom from catheter reintervention were estimated using the Kaplan–Meier method. The SPSS 21 was used for the statistical analysis.

Table 1 Characteristics of the patients and postoperative outcome

Patient no.	1	2	3	4	5	6
Anomalous PA	Right	Left	Right	Right	Left	Left
Age (day)	40	734	87	27	89	310
Weight (kg)	3.2	11.6	3	2	4.1	6.2
Other cardiac defect	VSD (SA), PDA, PFO	TOF (absent pulmonary valve syndrome), PFO	PDA, CoA	Muscular VSD, ASD, PDA	PDA, PFO	VSD, PDA, PFO
RV pressure	Systemic	Near systemic	Suprasystemic	Suprasystemic	Suprasystemic	Near systemic
Procedure for AOPA	Direct implantation	Direct implantation	Direct implantation	Direct implantation	Augmentation (pericardial patch)	Augmentation (pericardial patch)
Associated procedure	VSD closure, PFO closure, PDA division	VSD closure, RVOT widening, monocusp implantation	Coarctoplasty PDA division	VSD closure, PDA division	PFO closure, PDA ligation	VSD closure, PFO closure, PDA ligation
Complication	None	None	None	AV dissociation heart failure, sepsis	None	None
Reoperation, reintervention	None	PVR LPA angioplasty at 14 years postoperatively	RPA ballooning, at 3 years postoperatively	None	None	None
Outcome	Alive	Alive	Alive	Died at 1 month	Alive	Alive
Pre–post catheterization						
Pre-Prv/Psys	1.0	0.94	1.27		1.24	0.9
Post-Prv/Psys	–	0.46	0.45	–	0.5	0.47

Patient no.	7	8	9	10	11	12
Anomalous PA	Right	Right	Right	Right	Right	Left
Age (day)	10	72	3	404	21	28
Weight (kg)	3.1	3.4	2.9	8.4	3	3
Other cardiac defect	PDA, PFO	PDA	CoA, PDA	MAPCAs, VSD	PDA, PFO	TOF (absent pulmonary valve syndrome), ASD
RV pressure	Suprasystemic	Suprasystemic	Suprasystemic	Suprasystemic	Systemic	Near systemic
Procedure for AOPA	Aorta transection and augmentation (pericardial patch)	Direct implantation	Direct implantation	Augmentation (pericardial patch)	Augmentation (pericardial patch)	Direct implantation
Associated procedure	PFO closure, PDA division	PDA division	Coarctoplasty PDA division	VSD baffling, RV-PA conduit reconstruction	PFO closure, PDA ligation	RVOT widening VSD baffling, RPA reduction ASD closure
Complication	Chylothorax	None	None	None	None	None
Reoperation, reintervention	None	None	None	None	RPA ballooning, at 1 year postoperatively	None
Outcome	Alive	Alive	Alive	Alive	Alive	Alive
Pre–post catheterization						
Pre-Prv/Psys	–	–	–	1.3	–	–
Post-Prv/Psys	–	–	–	0.52	0.31	–

VSD ventricular septal defect, *ASD* atrial septal defect, *PDA* patent ductus arteriosus, *PFO* patent foramen ovale, *TOF* tetralogy of Fallot, *CoA* coarctation of aorta, *RVOT* right ventricular outflow tract, *RV* right ventricle, *PA* pulmonary artery, *RPA* right pulmonary artery, *LPA* left pulmonary artery, *AV* aortic valve, *PVR* pulmonary valve replacement, *MAPCA* major aortopulmonary collateral artery, *Pre* preoperative, *Post* postoperative, *Prv/Psys* right ventricle-to-systemic pressure ratio

Results

The mean length of follow-up was 12.6 ± 8 years. There was a single hospital death in 2-kg neonate patient (patient 4) with right pulmonary artery arising from ascending aorta, multiple muscular VSD, large patent ductus arteriosus, and atrial septal defect (ASD). The right pulmonary artery was connected to the main pulmonary artery following PDA division and excision, the aorta defect was closed primarily, the VSD was closed via a right atriotomy,

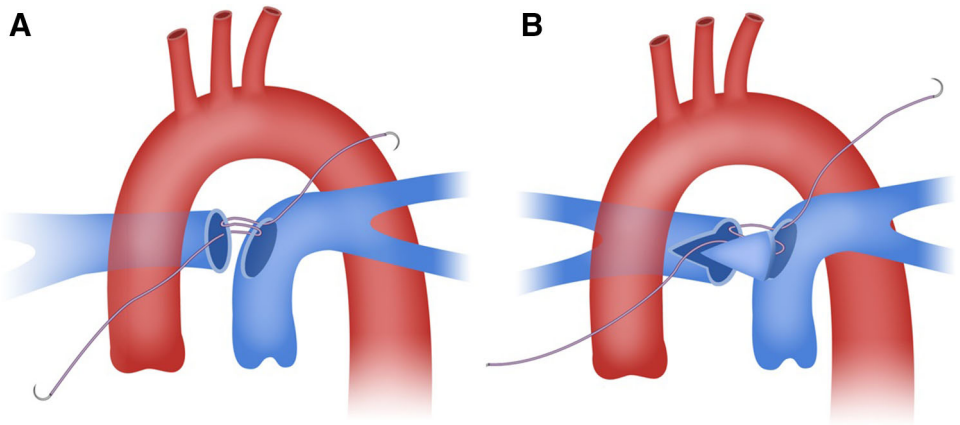

Fig. 2 Operative techniques for anomalous pulmonary artery from the ascending aorta. **a** Direct implantation of the anomalous pulmonary artery onto the main pulmonary artery, **b** anterior side augmentation with an autologous pericardial patch

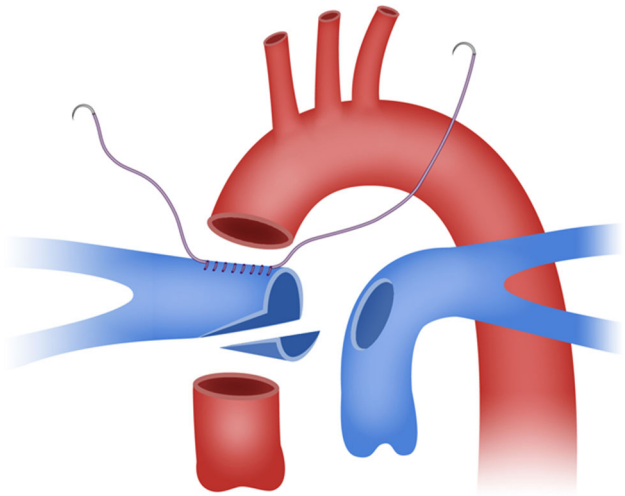

Fig. 3 Ascending aorta transection; superior side reconstruction with an aortic ring and an inferior side augmentation with an autologous pericardial patch in patient 7

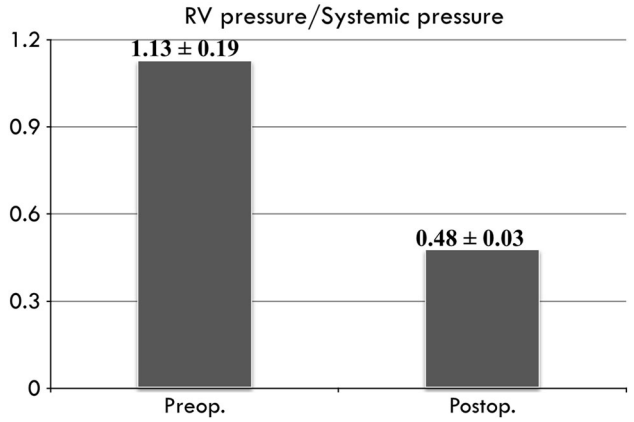

Fig. 4 Preoperative and postoperative right ventricle pressure-to-systemic pressure ratio. *RV* right ventricle

and the ASD was closed primarily. The patient was transferred to an intensive care unit with cardiopulmonary bypass support on postoperative day one. The patient underwent the placement decreased ventricular function, and cardiopulmonary bypass weaning was completed of a permanent pacemaker due to a developed complete heart block, and a delayed sternal closure was completed on postoperative day 9. Patient 4 developed low cardiac output, acute renal failure, sepsis, multi-organ failure and died on postoperative day 30. Early postoperative pulmonary hypertension crisis was identified in patient 3. Chylothorax was identified in patient 7, which was treated by pleural draining and a fat-free diet during the first two postoperative weeks.

There were five patients who were evaluated for preoperative and postoperative cardiac catheterizations (Table 1). Postoperative follow-up cardiac catheterization was completed at a median age of 2 years (range 1.0–11.5 years). The follow-up showed that the elevated right ventricular pressure-to-systemic ratio was significantly decreased after operation in these five patients (preoperative right ventricle-to-systemic ratio vs. postoperative right ventricle-to-systemic ratio = 1.13 ± 0.19 vs. 0.48 ± 0.03, $p = 0.043$) (Fig. 4).

Ten of the twelve patients had lung perfusion scans, with the final lung perfusion scan completed at a median age of 3.6 years (range 8 days to 20.3 years). The overall final lung perfusion of the affected lung was 46.6 ± 20.7 %, and there were no lung perfusion differences between direct implanted group I and the autologous pericardial employing group II [group I ($n = 5$) vs. group II ($n = 5$); 50.0 ± 10.3 vs. 42.7 ± 28.7 %, $p = 0.158$].

One of the twelve patients required reoperation for the left pulmonary artery stenosis implanted site and pulmonary regurgitation 14 years after the initial surgery. This patient was diagnosed with absent pulmonary valve syndrome, and total correction and direct implantation of the anomalous origin of the left pulmonary artery to the main pulmonary artery were completed at the time of the initial operation.

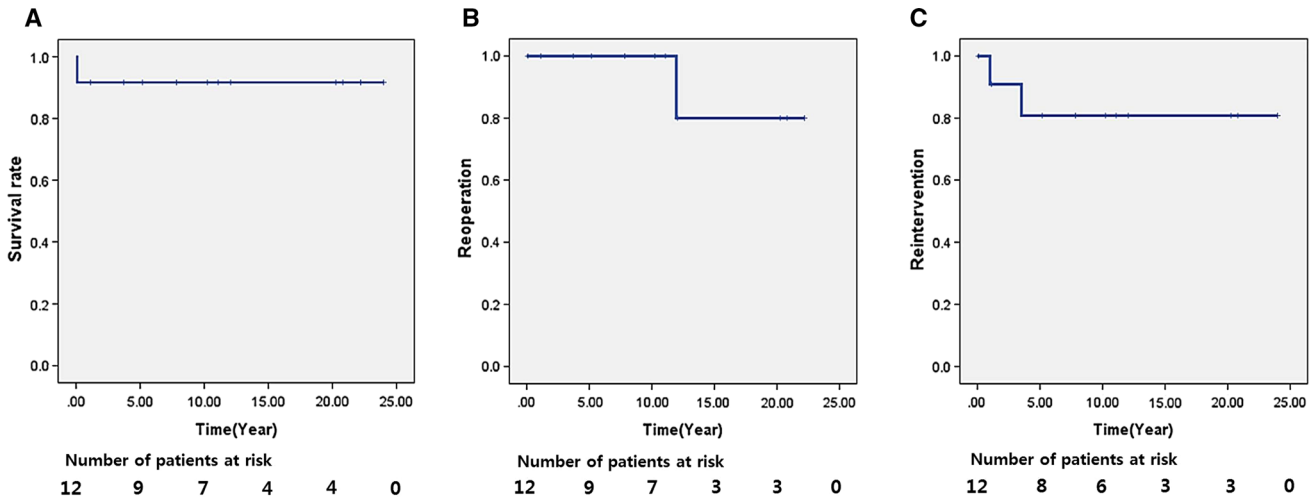

Fig. 5 Kaplan–Meier graph representing probability of freedom from **a** death, **b** reoperation, and **c** catheter-based intervention at 20 years

Two of the twelve patients underwent catheter-based interventions. Both of these patients had balloon dilatations of the pulmonary artery. One patient had direct implantation of the right pulmonary artery to the main pulmonary artery, and the other patient had a pericardial augmentation of the right pulmonary artery to the main pulmonary artery during the initial operation.

The probability of freedom from death at 20 years by the Kaplan–Meier estimate was 91.7 ± 8.0 %, the freedom from reoperation at 20 years was 80.8 ± 17.9 %, and the freedom from catheter intervention at 20 years was 80.8 ± 12.2 % (Fig. 5).

Discussion

Anomalous origin of one pulmonary artery from the ascending aorta is a rare congenital anomaly [12, 13]. This disease entity is distinct from discontinuous pulmonary arteries, where one branch of the pulmonary artery is supplied by a duct-like or other major aortopulmonary collateral. Neural crest cells have been reported to play a role in the development of the third and fourth pharyngeal pouch derivatives as well as the conotruncus and ascending aorta [3]. Origin of the right or left pulmonary artery from the aorta is the result of embryologically distinct processes. It has been hypothesized that a media fusion failure of the AOPA with the main pulmonary artery trunk results in persistence of the aortic sac from which AOPA originates. Additionally, the left pulmonary artery to main pulmonary artery fusion failure, which is due to the absence of the left sixth arch, results in persistence of an aortic sac from which the left pulmonary artery arises [2].

Anomalous origin of the right pulmonary artery from the aorta results from a delay in or abnormal migration of the sixth right aortic arch to the left side [5, 10, 11]. This entity is 4–8 times more common than anomalous origin of the left pulmonary artery from aorta. In our study, eight of the 12 patients (67 %) had anomalous origin of the right pulmonary artery from the ascending aorta [6]. The deletion of chromosomal band 22q11 may cause some degree of disarrangement in the neural crest, suggesting an association with the CATCH 22 syndrome complex including DiGeorge syndrome [4, 6, 14]. However, it has been reported that DiGeorge syndrome is less common with AOPA than with other conotruncal malformations such as truncus arteriosus or an interrupted aortic arch [22]. In our study, there was just one patient with CATCH 22.

The AOPA may be isolated or associated with other congenital heart defects, such as tetralogy of Fallot, isthmic hypoplasia, aortopulmonary window, interrupted aortic arch, VSD, and patent ductus arteriosus [4, 6, 14, 22]. In our study, we had two patients with absent pulmonary valve syndrome, two patients with arch hypoplasia, and one patient with contralateral side MAPCA. There were four patients with isolated AOPA lesions with no other major intracardiac or arch anomalies except for PDA or patent foramen ovale (PFO). Three of these patients presented with right AOPA, and one patient presented with left AOPA.

The pathophysiology resulting from AOPA exposes the pulmonary circulation of both lungs to pressure and volume overload. The full quantity output flows to the single lung attached to the right ventricle, and the other lung is exposed to unrestricted aortic blood flow and pressure. If this abnormal circulation is allowed to continue uncorrected, early development of pulmonary vascular obstructive disease can be expected [7, 16, 24].

Clinically, these patients often present with progressive respiratory distress and evidence of congestive heart failure

early in life. There is often a degree of cyanosis from the right to left shunting through the PFO of PDA and secondary to elevated end-diastolic pressure in the hypertensive, noncompliant right ventricle from increased pulmonary resistance [7, 16]. Almost all patients had systemic or suprasystemic right ventricular pressure in our study. Postoperatively, five patients had diagnostic catheterization, which normalized their right ventricular and pulmonary arterial pressure. Echocardiography and cardiac catheterization have been used for diagnosis. More recently, we used computed tomography angiograms, which being used to image the branch pulmonary artery and confirm suspected cases (Fig. 1). Computed tomography angiogram is noninvasive and allows for better surgical planning via imaging of anatomic details.

Various surgical techniques have been employed for treating AOPA in the past. Direct implantation was introduced by Kirkpatrick et al. [17] in 1967 and was most frequently used in the previous described series [1, 15, 18, 19]. When direct implantation is not feasible, an aortic flap [23], interposition of an autologous pericardial patch, end-to-end anastomosis with a synthetic graft [9, 21], or interposition of a homograft [1, 18] have been successfully employed to increase the AOPA length in specific cases. In our study, direct implantation of the anomalous pulmonary artery in the main pulmonary artery was successful in seven of the 12 patients. Five patients required a fixed autologous pericardial patch augmentation of pulmonary artery implant site. Contrary to other reports [1, 18, 21, 23], there were no differences between direct implantation and autologous pericardial patch augmentation in lung perfusion and reintervention. We believe that aggressive mobilizing vessels can prevent tensioned anastomosis, even when using a pericardial patch for tension-free anastomosis. The principle of native tissue-to-tissue anastomosis with regard to the growth potential of the pulmonary artery contributes to decreased restenosis and maintaining favorable lung perfusion. However, this analysis is limited due to the small number of patients.

Early operative repair is the treatment of choice. Early repair is preferred to avoid persistent pulmonary hypertension and irreversible pulmonary vascular occlusive disease. Hospital mortality has been reported from 0 to 21 % in previous reports [1, 15, 19, 21] and a need for reintervention from 12.5 to 36 % [9]. Only one patient in our study suffered hospital mortality (8 %), and two patients required catheter-based intervention (17 %). Additionally, one patient required reoperation (8 %). We believe that the early diagnosis and prompt surgical repair of AOPA results in excellent survival and avoids the development of pulmonary vascular obstructive disease.

Conclusion

AOPA from the aorta is a rare disease entity. The early repair of AOPA results in decreased right ventricular pressure, prevention of irreversible pulmonary vascular occlusive disease, and excellent survival with low incidence of reintervention or reoperation. The aggressive mobilizing of the pulmonary artery and employing native tissue-to-tissue anastomosis with or without autologous pericardial augmentation appear to be associated with a low frequency of reintervention, but studies with larger numbers of patients are required to confirm these results.

Conflict of interest None.

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