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Case Report

Surgical Repair of Giant Dissecting Pulmonary Artery Aneurysm Associated with Atrial Septal Defect and Pulmonary Arterial Hypertension: A Case Report

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Abstract

Pulmonary artery aneurysm (PAA) and pulmonary artery dissection (PAD) are rare and potentially fatal conditions that may lead to pulmonary artery (PA) rupture and cardiac tamponade. PAA is often associated with other cardiac and congenital heart diseases, such as atrial septal defect (ASD). We report a case of a patient with ASD and a giant dissecting PAA who underwent surgical repair to prevent potentially fatal outcome and discuss the probable etiologies of this case. We present a rare case of a 50-year-old woman with a secundum ASD and severe pulmonary arterial hypertension (PAH) who developed a giant PAA of 114 mm with dissection. The PAA caused extrinsic compression of the left main coronary artery (LMCA), which was misdiagnosed as coronary artery disease (CAD) at local hospital. Right heart catheterization revealed PAH of 73 mmHg and she was referred to our center for further treatment. After 4 months of lowering PAH treatment, she underwent successful surgical repair of the PAA to release the compression of LMCA, as well as mechanical valve replacement, fenestrated ASD closure and tricuspid valvuloplasty. She had an uneventful recovery and showed significant improvement in pulmonary hemodynamics and clinical symptoms at one-year follow-up. PAA with dissection is a rare complication of ASD and PAH that can potentially be fatal. Patients with large or symptomatic PAA and PAD may require early surgical intervention, particularly if they experience compression of nearby structures or are at risk of rupture. It is crucial to promptly refer and consult with specialists and ensure optimal preoperative hemodynamic management to enhance patient outcomes.

Keywords

pulmonary artery aneurysm; atrial septal defect; pulmonary arterial hypertension; dissection

Introduction

Pulmonary artery aneurysm (PAA) and pulmonary artery dissection (PAD) are rare and potentially fatal conditions that may result from congenital or acquired causes [1]. PAA has a prevalence of 1 in 14,000 individuals, while PAD has been reported in only 0.014% of congenital heart disease (CHD) patients or 1.7% of primary pulmonary arterial hypertension (PAH) patients [2,3]. It is mentioned that atrial septal defect (ASD), ventricular septal defect (VSD), and patent ductus arteriosus (PDA) are the major causes [4,5]. Other causes of PAA with dissection are infection or inflammation, acquired cardiac disease, and iatrogenic factors [5]. Unlike aortic dissection, the hemodynamic alteration and pressure in the pulomonary artery (PA) is scarcely possible to directly cause dissection formation; commonly, the PA would develop into an aneurysm with PAH, but the underlying course is difficult to detect. Moreover, many patients are asymptomatic or have nonspecific symptoms, which may lead to delayed diagnosis and treatment [4,6], especially for inexperienced institutes or hospitals. When the adjacent tissues or structures are compressed by a giant aneurysm that may cause clinical symptoms [7,8], but most patients are diagnosed incidentally. This can worsen the prognosis of CHD patients with chronic PAH and increase the risk of PAA dissection and rupture. Surgical repair is a feasible option for this group of patients who meet the surgical indications. In this case report, we present a rare case of a patient with ASD and PAH who developed a giant dissecting PAA that compressed the left main coronary artery (LMCA). And we successfully performed right ventricular outflow tract (RVOT) reconstruction and ASD repair for this patient after PAH management. The CARE checklist was used when writing this case report in (**Supplementary Table 1**).

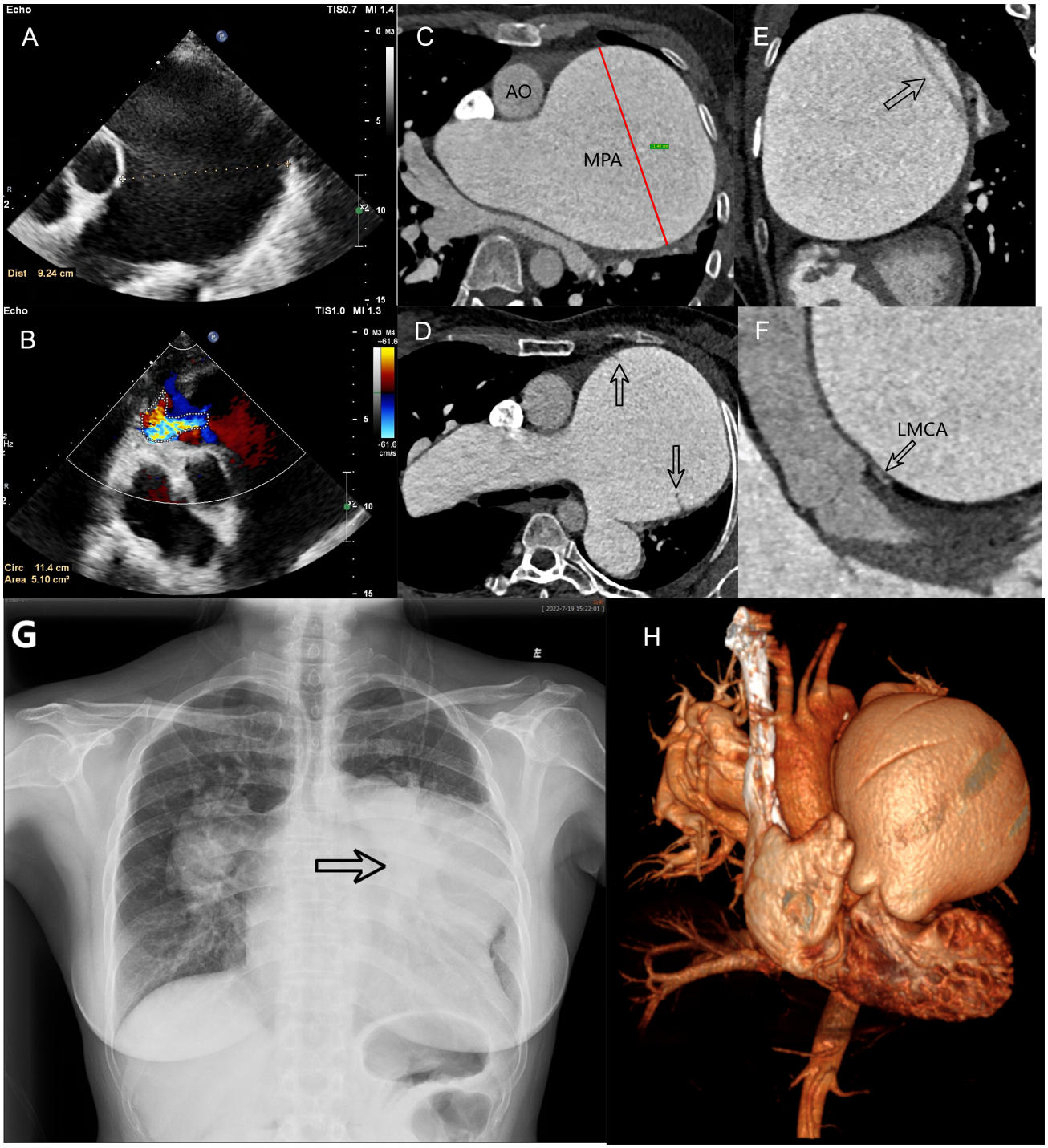

Fig. 1. The preoperative imaging findings. Imaging examinations revealed a giant pulmonary artery aneurysm (PAA) (Panel A, C, G, H) and dissection (arrow, (Panel D, E)) with a computed tomography angiography (CTA) diameter of 114.6 mm and an echocardiography diameter of 92.4 mm, respectively. The PAA also caused extrinsic compression of the left main coronary artery (left main coronary artery (LMCA); arrow, (Panel F)). Echocardiography showed a pulmonary regurgitation (PR) area of 5.1 cm^2 (Panel B).

Case Presentation

A 50-year-old lean woman presented with exertional dyspnea, lower extremity edema, intermittent angina, palpitations, and dizziness for one year. She had not received any medical attention since symptom onset. She was hospitalized at a local hospital when her condition worsened five months later. The patient initially presented with non-specific symptoms and was misdiagnosed with coronary artery disease (CAD) due to the compression of the LMCA by a giant PAA. They planned to perform percu-

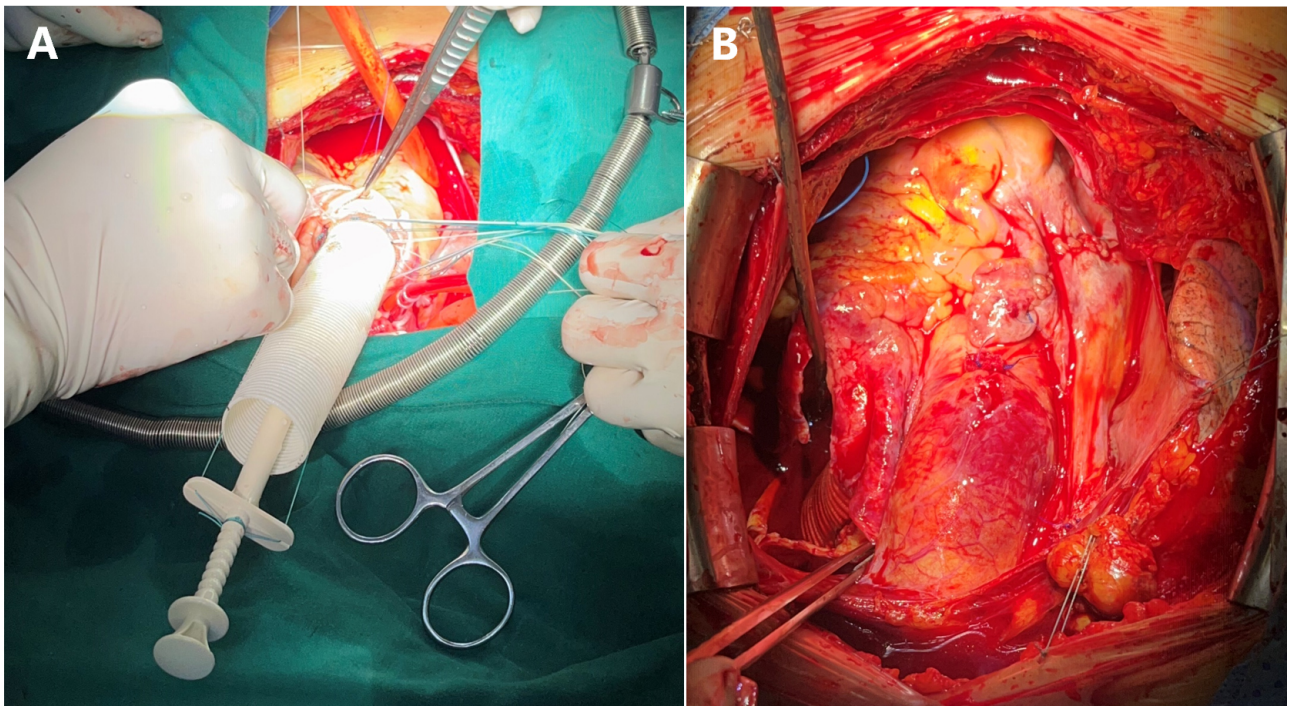

Fig. 2. The homograft replacement procedure. (A) The left picture showed the reconstruction of the main pulmonary artery (MPA) and the replacement of the pulmonary valve (PV) with a homograft with a mechanical valve. (B) The right picture showed the completed homograft replacement.

taneous coronary intervention (PCI) in terms of the results, but further investigations revealed that she had an atrial septal defect (ASD) and pulmonary artery (PA) dilation, with pulmonary arterial hypertension (PAH) confirmed by right heart catheterization (RHC). The RHC showed a pulmonary arterial pressure (PAP) of 73/38 (50) mmHg and a pulmonary vascular resistance (PVR) of 12.2 Wood units. The high pulmonary pressure precluded them from performing PCI and they recommended conservative management with Bosentan. She was then referred to our center for further evaluation and treatment. Considering the risk of PAD rupture, surgery was deemed too risky at that time. We advised the patient to receive targeted therapy (Ambrisentan and Sildenafil) to lower pulmonary pressure and diuretics and to rest adequately to avoid exertion. She was scheduled to return to the hospital for reassessment and further evaluation after 4 months, and then decide on the surgical option. During the follow-up period, the patient adhered to her medication regularly and stayed at home without working. She was admitted to our center with New York Heart Association functional class III after 4 months. We performed the relevant investigations as detailed below. Based on a comprehensive evaluation, we determined that surgery was feasible, as the PAP remained high, but the PVR had significantly decreased compared with the previous result.

The patient had undiagnosed CHD and no treatment. She had no surgical history or family history of cardiovascular diseases and negative syphilis, with a single episode of falling from a height of 3 meters and one normal delivery in her youth. Echocardiography revealed secundum ASD of 25 mm, severe tricuspid regurgitation, severe pulmonary regurgitation (PR), and estimated PAP at 80 mmHg. Computed tomography angiography (CTA) demonstrated a PA diameter of 114 mm, dissecting intimal flap originating from the wall of main pulmonary artery (MPA) without left (LPA) and right pulmonary artery (RPA) involvement, and LMCA compression (Fig. 1). RHC showed PVR decreased to 8.3 wood and PAP to 76/26 (42) mmHg after treatment. The electrocardiograph showed sinus rhythm, premature atrial contractions, and complete right bundle branch block. Pulmonary ventilation test indicated moderate to severe impairment.

After a multidisciplinary discussion, we decided to perform a surgical intervention urgently to prevent the risk of rupture of dissecting PAA. Peripheral cannulation for the bypass was established followed by sternotomy and pericardiotomy, and the PAA was incised longitudinally. During the operative exploration, we observed multiple old fibrotic intimal flaps attached to the dilated segment of the pulmonary arterial wall, without involvement of the pulmonary valve (PV) or branches. However, the pulmonary valvular annulus was markedly dilated and had severe PR, which required PV replacement. The leaflets were excised. Then we performed PA reconstruction using a prosthetic homograft with a mechanical valve (St. Jude Regen Mechanical Heart Valve—25 mm, Graft—28 mm) to replace the main PA and PV, similar to the Bentall procedure (Fig. 2). The orifices of the left and right PA were also dilated and mea-

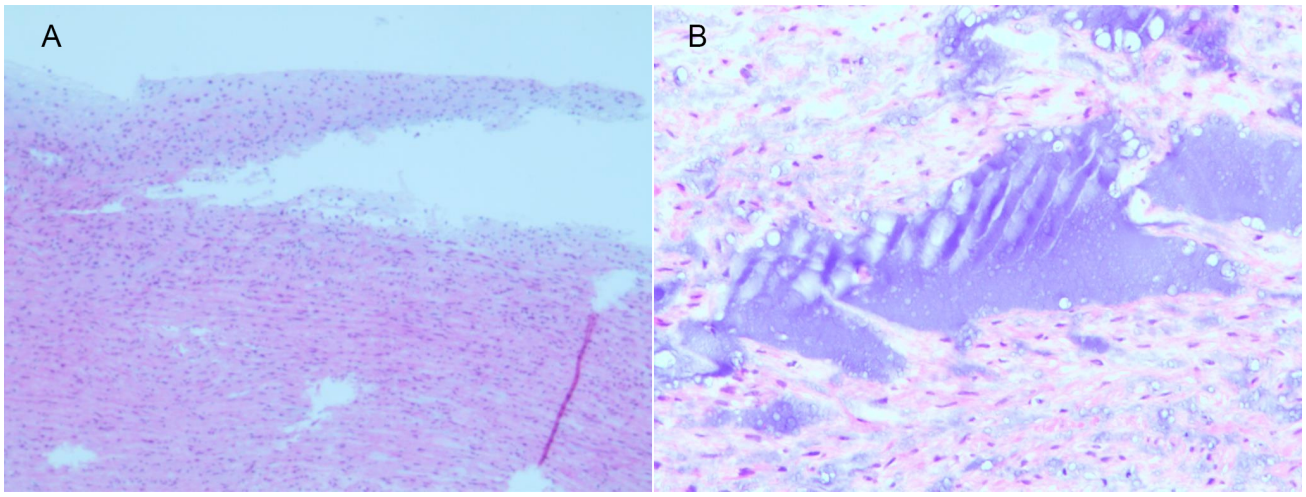

Fig. 3. The results of histopathological observation. (A) The pathological section showed a local intimal tear and dissection formation from the inner wall of the pulmonary artery, with multiple perforations that reduced the elasticity and increased the rigidity of the wall. (B) The picture revealed a series of marked and typical pathological changes, as shown in the picture that include mucoid degeneration of the media caused by vascular smooth muscle cell necrosis, which increased vascular tone and stiffness, and a local thickening of the adventitia that may attributed to inflammation, fibrosis, and vasa vasorum formation, which contributed to vascular remodeling and occlusion.

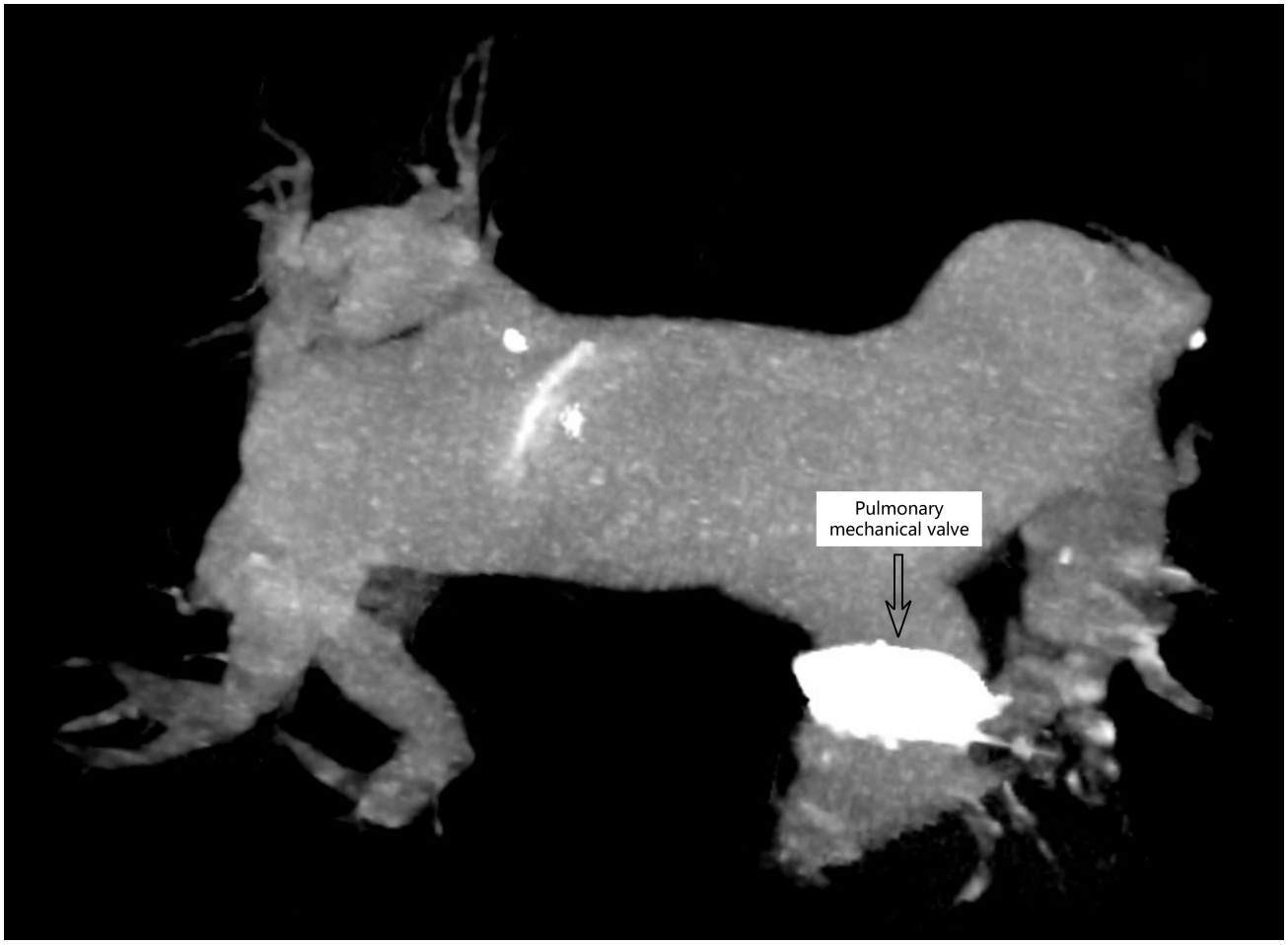

Fig. 4. CTA with 3-dimensional reconstruction showed a 52 mm diameter of the main pulmonary artery after surgery, with a well-functioning pulmonary mechanical valve.

Table 1. Timeline of course.

Time	Events
April 1972	The patient was born with an undiagnosed atrial septal defect (ASD).
May 1995	A fall of about 3 meters accidently.
October 1998	A pregnancy and delivery.
August 2021	Symptoms of exertional dyspnea to onset.
11 February 2022	The patient was presented to the Department of Cardiology at the local hospital. The examinations include coronary arteriography, right heart catheterization, echocardiography, and chest computed tomography to diagnose ASD, pulmonary arterial hypertension (PAH), and dilation of the main pulmonary artery.
28 February 2022	The patient was referred to our center and prescribed oral antihypertensive drugs to lower the pulmonary pressure.
13 July 2022	The patient was admitted to the Department of Cardiology in our hospital, where she received preoperative examinations and evaluations.
3 August 2022	Surgical treatment was carried out. (The operation lasted from 09:00 to 15:15, with an aortic cross-clamp time of 123 minutes and a cardiopulmonary bypass time of 193 minutes. The postoperative ratio of right ventricular pressure to left ventricular pressure (RV/LV) was 0.6, and the central venous pressure was 12 mmHg)
4 August 2022	The patient was weaned from the mechanical ventilation and extubated.
23 August 2022	The patient was discharged from the hospital after postoperative rehabilitation and respiratory function exercise.
10 March 2023	The patient returned to the hospital for a follow-up examination, which showed a successful recovery and improvement of the cardiac function and pulmonary pressure.
20 November 2023	The patient was without symptoms of complications one year after the surgery.

sured at 50 mm and 38 mm, respectively. We trimmed the distal homograft with a wedge shape, connected and sutured it to the bifurcation that covered the orifices, and strengthened the anastomotic stoma with artificial patches. Then we excised the aneurysm wall to the appropriate size and homograft was wrapped with residual wall of PAA. We then performed the ASD closure with a fenestrated patch about 4 mm and tricuspid valvuloplasty (Balance Medical Tricuspid Valvuloplasty Ring—29 mm). After the surgery, the PAP was estimated at 68/4 (26) mmHg. We sent the dissecting aneurysm specimens for pathological examination (Fig. 3).

The patient was extubated one day later and got Treprostinil and oral Ambrisentan for lowering pulmonary pressure. After rehabilitation for atelectasis and PAH reduction, she was discharged smoothly on day 20th. Oral warfarin was prescribed as lifelong anticoagulation, and three antihypertensive drugs (Tadalafil, Macitentan, and Selexipag) were administered to manage PAH. Six-month echocardiograph and CTA showed main pulmonary artery pressure (MPAP) 57mmHg and a well-functioning valve (Fig. 4). She remained asymptomatic and continued to receive medication for PAH management at the one-year follow-up. The Table 1 shows the holistic course of the patient's condition.

Discussion

The PA diameter greater than 40 mm is often defined as a PAA [1], and dissection commonly involves the pulmonary trunk (72–80%) or its branches (4–6%) [5], which varies among individuals and requires personalized and targeted therapeutic strategies. These include surgical correction, conservative treatment, heart-lung transplantation, or surgical repair with lung transplantation (LTx), as shown in Table 2 (Ref. [2,9–21]). Some studies have also proposed the use of endovascular repair, which is more applicable for acute iatrogenic injury [22] and special cases without aneurysmal dilatation of the pulmonary trunk [23] not suitable for this case. The common indication for surgical treatment is a main PA diameter of $\geq$55 mm or a PAA diameter growth of $\geq$5 mm in 6 months [4], but some authors suggest a threshold of $>$75 mm and a growth rate of $>$2 mm per year [1]. A necessary indication for surgical repair of PAA is complicated with signs of dissection and rupture, which requires verification of etiology and specific PAP beforehand. Other indications include clinical symptoms, associated with pulmonary valvular disease, and compression of adjacent structures or tissues by the dilated PAA [4,24]. Sometimes conservative treatment also can be an option for patients with suitable situations but still need a meticulous and comprehensive evaluation [16,18].

In this case, surgical repair was the necessary option for the patient to prevent life-threatening outcomes, apart from the consideration of PAH, especially since there were no indications for heart-lung transplantation after comprehensive evaluation. We fully recognized the risk and urgency of this disease and administered oral antihypertensive drugs before the operation for sufficient window time, which improved pulmonary vascular resistance and right ventricular function to optimize preoperative hemodynamics [25]. As shown in the Table 2, this PAH management

Table 2. Comparison of different treatments and outcomes for PAA with dissection.

Age (years), sex	Etiologies	Clinical symptoms	Imageological examinations	PAA site, size (mm)	Branches involvement	PAP (mmHg)	Treatment	Outcome	Reference
32, male	Pulmonary stenosis, a history of PBV	Thoracic pain, fever, persistent cough	Echo, CTA	MPA, 45	No	Normal	Medication of 3 days, aneurysmectomy + homograft replacement	Well after surgery 3.5 years	Adodo, 2017 [9]
55, male	History of chemotherapy for malignant melanoma (undefined)	Worsen dyspnea	Chest X-ray, echo, CTA	MPA, 99	Both	Estimated at 70	Homograft replacement	Death caused by cardiogenic shock and multiorgan failure	Perrotta, 2015 [10]
55, male	Primary PH	Exertional Dyspnea	Echo, CTA	MPA, 93	No	Estimated at 80	Homograft replacement	Death, cardiogenic shock after surgery 4 days	Malm, 2015 [11]
22, male	PDA	Progressive exertional dyspnea	CTA, Echo, RHC	MPA, 108	No	60	Aneurysmectomy + homograft replacement	Well after surgery 6 months	Tiwari, 2013 [12]
32, female	PDA occlusion	Chest pain	TEE, CTA	MPA, N/A	No	Estimated at 36	Dissection surgical repair by pericardial patch	Well after surgery one year	Zhang, 2023 [13]
25, male	DORV/VSD, CoA, PAH	Asymptomatic	Echo, RHC, CTA	MPA, N/A	No	76	Correction of DORV + angioplasty of the pulmonary artery and valve + ligation of PDA	Survive without follow-up	Ku, 2022 [14]
61, female	Prior pulmonary embolus, chronic right-sided heart failure PAH	Progressive dyspnea on exertion, orthopnea, lower extremity edema	CTA, Echo, RHC	MPA, 61	No	69	Composite valve-tube graft	Well after surgery 2.5 years	Biasi, 2021 [15]
88, female	Chronic PAH	Loss of consciousness, chest pain, dyspnea, cough	CTA, Echo, TEE	MPA, 48	No	Estimated at 75	Medication	Well after discharge 3–6 months	Togo, 2015 [16]
41, female	Repaired ASD, PAH	Unspecified	Chest X-ray, echo, CTA	MPA, 46.6	LPA	Estimated at 113	Medication	Death, progressive heart failure after diagnosis PAD 10 years	Nuche, 2019 [17]
31, male	IPAH	Chest pain	CTA, echo, MRI	MPA, 120	LPA	Estimated at 47	LTx was rejected by the patient and continuing to receive medication	Discharge smoothly without follow-up	Liu, 2018 [18]

Table 2. Continued.

Age (years), sex	Etiologies	Clinical symptoms	Imageological examinations	PAA site, size (mm)	Branches involvement	PAP (mmHg)	Treatment	Outcome	Reference
35, female	IPAH	Chest pain	Chest CT	MPA, 120	No	N/A	Medication for preoperative preparation, bilateral LTx + aneurysmectomy + homograft replacement	Well after surgery 6 months	Ganapathi, 2022 [19]
22, female	IPAH	Chest pain, dyspnea	MRI, echo, RHC	MPA, 90	No	90	Medication for PAH management 6 months, but LTx was rejected by the patient	Sudden death caused by PAA rupture and cardiac tamponade	Florczyk, 2018 [20]
37, female	IPAH	Chest pain	CTA, RHC	MPA with 60 but autopsy showed that PAA with 105	LPA	93	Medication and waiting for LTx	Sudden death caused by PAA rupture and cardiac tamponade	Florczyk, 2018 [20]
49, male	History of d-TGA with the palliated procedure, chronic PH	Chest pain, cough, mild hemoptysis, progressive shortness of breath	Chest X-ray, TTE, MRI, CTA	MPA with 75, RPA with 60	RPA	81	Medication for PH management and waiting for heart-lung transplantation	Stable at 6 months follow-up	Tomasino, 2023 [21]
46, female	PDA, PAH	Substernal pressure radiating into the shoulder, shortness of breath, nausea	Chest X-ray, Echo, CTA	MPA, 62.4	No	N/A	Preparation for heart-lung transplantation	Sudden death caused by PAA rupture and cardiac tamponade	Zhang, 2016 [2]

Abbreviations: PAA, pulmonary artery aneurysm; PAP, pulmonary arterial pressure; PBV, pulmonary balloon valvuloplasty; Echo, echocardiography; CTA, computed tomography angiography; MPA, main pulmonary artery; PH, pulmonary hypertension; PDA, patent ductus arteriosus; RHC, right heart catheterization; TEE, transesophageal echocardiography; DORV, double outlet right ventricle; VSD, ventricular septal defect; CoA, coarctation of aorta; ASD, atrial septal defect; LPA, left pulmonary artery; MRI, magnetic resonance imaging; PAD, pulmonary artery dissection; IPAH, idiopathic pulmonary arterial hypertension; LTx, lung transplantation; d-TGA, d-transposition of the great arteries; RPA, right pulmonary artery.

for preoperative preparation is the common therapeutic strategy that can significantly reduce the risk and the probability of perioperative adverse events. For patients with irreversible PAH and cardiac failure, transitioning to optimal PAH management before heart-lung transplantation may be the only effective option [26,27]. However, the signs of rupture in PAD are difficult to predict and require frequent examinations and evaluations. It is crucial to determine the optimal timing and condition of surgery for each patient [28]. Aneurysmectomy with homograft replacement is a common surgical procedure that usually results in a favorable outcome [9,12–15]. For the consideration of PV replacement, patients with main PAA and PAH who often have significant PR, concurrent PV replacement should be considered during the operation, even if the valve leaflets are not involved in the dissection, to prevent long-term recurrence caused by abnormal hemodynamics [29].

Chronic PAH, whether primary, idiopathic, or secondary, is the most common factor that can cause increased wall stress and hemodynamic changes in the pulmonary artery, leading to a series of pathophysiological alterations and dilation of the vessel finally [4]. These are the main factors for the formation of dissecting PAAs, especially in patients with CHD such as PDA [30] or ASD [31]. Combined with intraoperative findings, we did not observe any blue ecchymotic areas that are typical of acute rupture, nor any signs of vasculitis associated with Behcet disease during the operation or in the specimen results related to connective tissue disease. Instead, the intimal flaps of the dissection mostly adhered to the most severely dilated segment of the pulmonary artery, showing fibrotic changes and loss of elasticity and thickness that corresponded with the pathological changes of chronic PAH and the law of Laplace [4]. The exact timing of these pathological changes is difficult to define, as we lack previous examination evidence and long-term follow-up before admission. Based on the intraoperative findings, we inferred that the PAA had a slow progression and did not rupture immediately. After ruling out other possible factors such as vasculitis and RHC-induced injury, we propose that the PAD formation may be related to the patient's history of falling when she was young, which may have caused or aggravated the injury to the PA wall. This is consistent with previous studies that have reported traumatic injury as a possible cause of PAD [31]. Moreover, the patient reported a low level of physical activity, which may have contributed to the slow rate of progression of the PAA and dissection. We also considered that pregnancy may have increased the cardiopulmonary demand, especially by increasing the right heart preload, thus exacerbating pulmonary hypertension and PA dilation [1,4,5]. These factors may have a synergistic effect on the pathophysiological changes that we speculated, which need more research to verify.

As shown in Table 2, previous case studies have reported that patients with PAA and dissection often present with non-specific symptoms or are asymptomatic, which poses a significant challenge for developing a systematic and personalized treatment plan for this rare and heterogeneous condition, and finding evidence-based medical guidance for these patients. In this case, the patient was initially misdiagnosed with CAD by a local hospital, based on coronary angiography. Further examination revealed that she had a rare condition of PAA with dissection, which compressed the LMCA and caused myocardial ischemia. This may reflect the lack of experience and expertise in managing such cases in underdeveloped regions, which may result in inappropriate treatment strategies and poor outcomes. We need to be vigilant for such scenarios and provide timely referrals and consultation for patients with suspected PAA with dissection. Large-scale screening using echocardiography or chest X-ray for these patients is necessary and helpful especially in in underdeveloped and rural regions [32], which may show the dilated pulmonary artery and pulmonary atelectasis, as well as cardiac enlargement in the shunt type of CHDs. Echocardiography can also quickly identify the size of PAA and the presence of dissection, but care should be taken to distinguish it from the pulmonary valve [2,4]. CTA and RHC can further evaluate the scope of dissection involvement and specific pulmonary pressure, with attention to the possibility of catheter-induced rupture [33]. For this population, it is also essential to provide them with relevant medical knowledge and preventive education. This is an important issue that requires our attention and improvement.

PAA with dissection is a rare but potentially fatal complication of ASD and PAH. Early surgical intervention may be indicated for patients with large or symptomatic PAA and dissection, especially if they have compression of adjacent structures or risk of rupture. Even if the surgical repair is not applicable or necessary for the patient through all-around inspection and evaluation, the patient's condition and alteration of symptoms should be frequently monitored in the follow-up. It's necessary to identify the specific etiologies, and whether or not PAH is present, which are related to the choice of therapeutic strategies. To tackle the problem of management for the patients, indispensable examinations, prompt referral, and consultation, as well as optimal preoperative hemodynamic management, are essential for improving the outcomes of these patients. More research and evidence-based practice are needed to guide the optimal management and treatment of PAA with dissection.

Conclusion

This case report illustrates the complexity of pulmonary artery aneurysm (PAA) with dissection, emphasizing personalized and holistic approaches to treatment. If the patient's condition allows, the surgical repair is a feasible and effective option to prevent fetal complications. Tradi-

tional medical values, such as a holistic view of health and preventive care, align with the discussed strategies. Moreover, the report reveals the challenges of diagnosis and management, especially in underdeveloped regions, and underscores the importance of early detection and referral. Continuous monitoring, a thorough diagnostic approach, and a call for further research demonstrate a commitment to evidence-based practice and ongoing improvement in managing this rare condition.

Author Contributions

JZ and SW designed the study and performed the surgery. WX also designed the study, wrote the first draft of the manuscript, and completed the revision. JM, YZ and HY were in charge of data collection and video recording. SW, YZ, and JZ provided professional suggestions and discussion on the surgery and supervised the research. All authors contributed to editorial changes in the manuscript. All authors read and approved the final manuscript. All authors have participated sufficiently in the work to take public responsibility for appropriate portions of the content and agreed to be accountable for all aspects of the work in ensuring that questions related to its accuracy or integrity.

Ethics Approval and Consent to Participate

The study was approved by Guangdong Provincial People's Hospital Ethics Committee (No. GDREC2019338H(R2)) on 17th September 2019. The written informed consent was obtained from the patient's parents.

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Conflict of Interest

The authors declare no conflict of interest.

Supplementary Material

Supplementary material associated with this article can be found, in the online version, at https://doi.org/10.59958/hsf.6835.

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