Mini-Review

Pulmonary artery aneurysm: computed tomography (CT) imaging findings and diagnosis

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Abstract: Pulmonary artery aneurysm (PAA) is a rare pulmonary vascular disease with nonspecific symptoms and various etiologies. As the disease progresses, in addition to the dilation of the pulmonary arteries, it may be accompanied by remodeling of the cardiac structure and changes in the morphology of the aorta. Recognizing the cause of PAA is therefore a clinically challenging task. In this review article, we provide an overview of various causes of PAA with the support of corresponding imaging findings on computed tomography pulmonary angiography (CTPA) examination. Firstly, from the perspective of hemodynamics, a logical diagnosis is provided according to whether the main pulmonary artery (MPA) is dilated, and whether the PA is dilated locally or diffusely. Secondly, for the imaging examination of vascular wall lesions, due to the limitations of ultrasound examination and interventional procedures, the irreplaceability of dual-phase CTPA examination in disease assessment is especially emphasized. Finally, for highly suspected disorders, it is necessary to comprehensively check with the patient whether there is a family history or past medical history. For patients with PAA, especially those with Marfan syndrome (MFS) or arteritis, adequate preoperative imaging evaluation, regular postoperative radiographic follow-up, and concurrent treatment of the underlying disease (if necessary) are crucial, which are related to the prognosis and long-term quality of life of such patients. Despite the nonspecific features of PAA presentation, a thorough examination of the patient's clinical history and imaging characteristics will play an important role in diagnosing PAA and planning patient management strategies.

Keywords: Cardiovascular disease; pulmonary artery aneurysm (PAA); pulmonary hypertension (PH); computed tomography (CT); diagnosis

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Introduction

Pulmonary artery aneurysm (PAA) is an extremely uncommon, low-incidence pulmonary vascular disease, with a prevalence of 1 in 14,000 individuals according to a previous study based on an analysis of 109,571 autopsies (1). Pulmonary artery (PA) dilatation is increasingly reported in individuals with congenital heart disease (CHD) due to the frequent use of imaging methods (2). The causes of PAA, as summarized in previous literature reviews, can be congenital, acquired, or idiopathic (3,4). CHDs with increased pulmonary blood flow, such as patent ductus arteriosus (PDA), ventricular septal defect (VSD), and atrial septal defect (ASD), are the most prevalent congenital causes. Pulmonary hypertension (PH), autoimmunity-associated vasculitis, infection, malignancy, iatrogenic manipulation, and trauma are acquired causes of PAAs.

In general, PAA is caused by a multitude of variables with complicated interactions, resulting in various alterations in the morphological structure of the aorta and heart during the course of the illness. Therefore, although it is not difficult for radiologists to detect PAAs or PA dilatation based on morphological abnormalities in the PA, many causes of PAAs are easily neglected. The majority of PAA cases are asymptomatic and are often discovered by accident during imaging tests; however, if a PAA ruptures, it is exceedingly hazardous and may lead to life-threatening consequences, including massive hemoptysis and cardiac tamponade. Furthermore, therapy may be delayed if the cause of PA dilatation is unknown. Although echocardiography has advantages in assessing cardiac morphology and function, valve motion, and pressure measurement, it is limited in its ability to evaluate the left and right PA trunks and intrapulmonary vascular branches, as well as vascular wall lesions, intravascular lesions, and perivascular walls lesions. Right heart catheterization is a useful technique for cardiopulmonary hemodynamic diagnosis and monitoring. Interventional imaging procedures offer excellent visualization of pulmonary vascular lesions; however, their assessment of vascular and perivascular wall lesions is severely restricted. Chest imaging, particularly computed tomography pulmonary angiography (CTPA) is the primary test used for the clinical detection of PAA. Radiologists should have logical diagnostic thinking when searching for the origin of PAAs. In their review, Gupta *et al.* analyzed 248 cases of PAA, with details on classification and demographics (3). Duijnhouwer *et al.* conducted a comprehensive review of the current literature and identified 38 original studies and reviews, 169 case reports, 2 guidelines, and 1 conference abstract (4). Although authors have reviewed the current literature on PAA, there is a lack of imaging findings corresponding to each type of PAA. In this review article, we provide an overview of the clinical diagnosis and imaging findings in patients with PAA. We believe that our review will serve as a useful learning source for clinicians to diagnose and manage patients with suspected PAA.

Definition

PAA is defined as the enlargement of the artery's local diameter, although there is currently no agreed-upon definition of the standard vascular diameter criteria for the clinical diagnosis of PAA. Some authors define PAA as expansion greater than 1.5× the upper limit of the normal PA diameter, and PAA formation is considered when the upper limit of PA diameter is greater than 43 mm for men and 40 mm for women (3). Others define PAAs as focal dilation of the PA, arguing that local dilation exceeding the maximum normal diameter of the PA can be defined as PAA. Duijnhouwer *et al.*, meanwhile, categorized PA with a diameter >40 mm as PAA, without taking into account individual variations or sex-related considerations (4,5). PAAs are classified as true or pseudoaneurysms based on the integrity of the 3-layer structure of blood vessels. True aneurysms have intact tunica intima, tunica medium, and tunica externa and often exhibit spindle dilatation on imaging, whereas pseudoaneurysms have incomplete 3-layer structures and a greater risk of rupture and frequently show cystic dilation on imaging.

Imaging findings and diagnosis

Dilatation of the main pulmonary artery (MPA)

PH in relation to PAA

PH is a group of pathophysiological syndromes caused by heterogeneous diseases and different pathogeneses, characterized by increased pulmonary vascular resistance (6). PH is defined as a resting mean PA pressure >20 mmHg (7). Symptoms of PH are primarily related to right ventricular dysfunction and the early signs of PH are frequently associated with activity. Dyspnea following exercise, fatigue and rapid exhaustion, dyspnea or palpitations during forward bending, hemoptysis, and syncope are the main

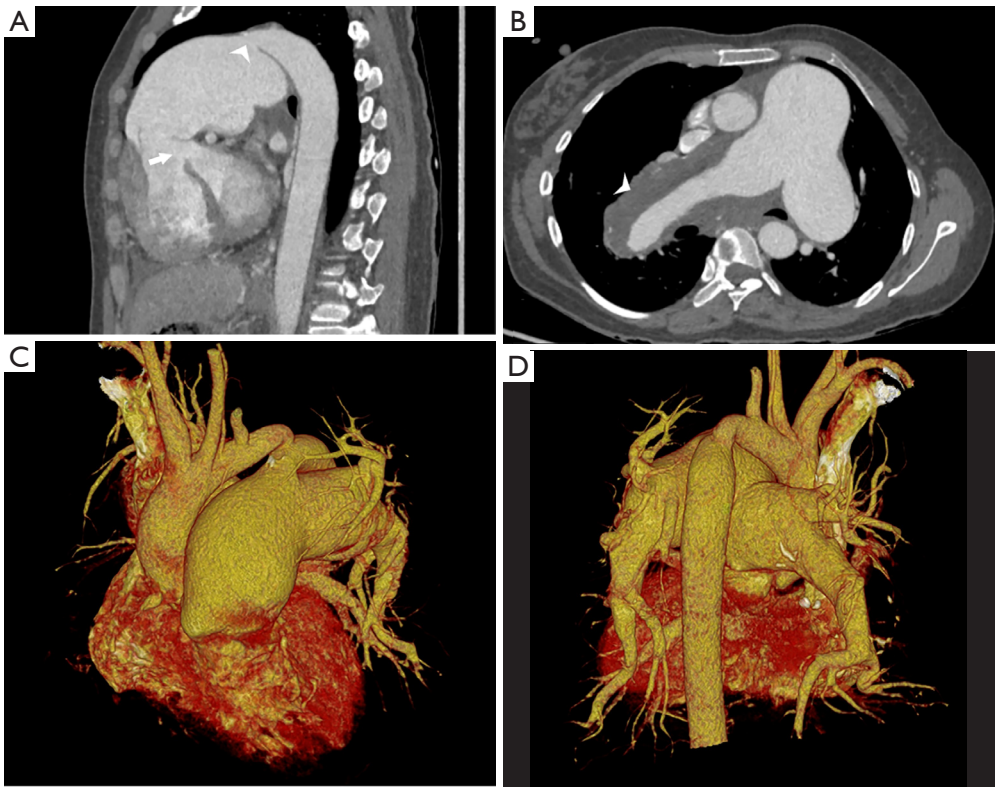

Figure 1 PDA with ventricular septal defect. (A) MPR reconstruction shows PDA (arrowhead) and ventricular septal defect (arrow). (B) MPR reconstruction shows mural thrombus (arrowhead) in the main trunk of the right pulmonary artery. (C,D) VR reconstruction shows diffuse dilation of the main-pulmonary artery-bilateral pulmonary arteries. PDA, patent ductus arteriosus; MPR, multiplanar reformation; VR, volume rendering.

clinical manifestations. Exercise-induced chest pain or hoarseness (dysphonia) may occur when the PA dilates and dynamically pressurizes the left coronary artery trunk or recurrent laryngeal nerve. Bronchial compression may cause shortness of breath, wheezing, and coughing and may be associated with lower respiratory tract infection, atelectasis, and other symptoms.

PH is reportedly present in 66% of patients with PAA (8), showing a close association between the degree of PH and PAA occurrence. A previous study showed that the ratio of the diameter of the MPA to the ascending aorta ≥1.0 has high specificity for the diagnosis of PH (9). Therefore, PH should be suspected when a corresponding change in PA morphology is observed on computed tomography (CT). It can present simultaneously with right atrial and ventricular enlargement, thickening of the right ventricular wall, and flattening or deviation of the interventricular septum. In the late stages of PH, vascular wall remodeling may occur. In individuals with Eisenmenger syndrome, CT may reveal calcification of the PA wall or mural thrombus in the vascular lumen. When the branches of the patient's MPA and both PAs are found to be diffusely dilated during an imaging examination, it is critical to determine the cause of the patient's pulmonary hyperemia in clinical diagnosis, and the first thing to be extremely watchful for is whether there is PH caused by shunting (left-to-right shunt). Left-to-right shunt intracardiac procedures, including PDA, ASD, and VSD, are the most frequently performed procedures in clinical practice (*Figure 1*). The absence of an obvious intracardiac left-to-right shunt during a chest examination cannot be used as evidence that the patient does not have a left-to-right shunt. Instead, it is necessary to recommend that the patient continue to undergo extra-examination of the abdominal blood vessels to determine whether an extracardiac left-to-right shunt exists (*Figure 2*).

Additionally, if the morphological changes in the PA are consistent with the imaging findings of PH and no strong evidence of intra- or extra-cardiac left-to-right shunts is

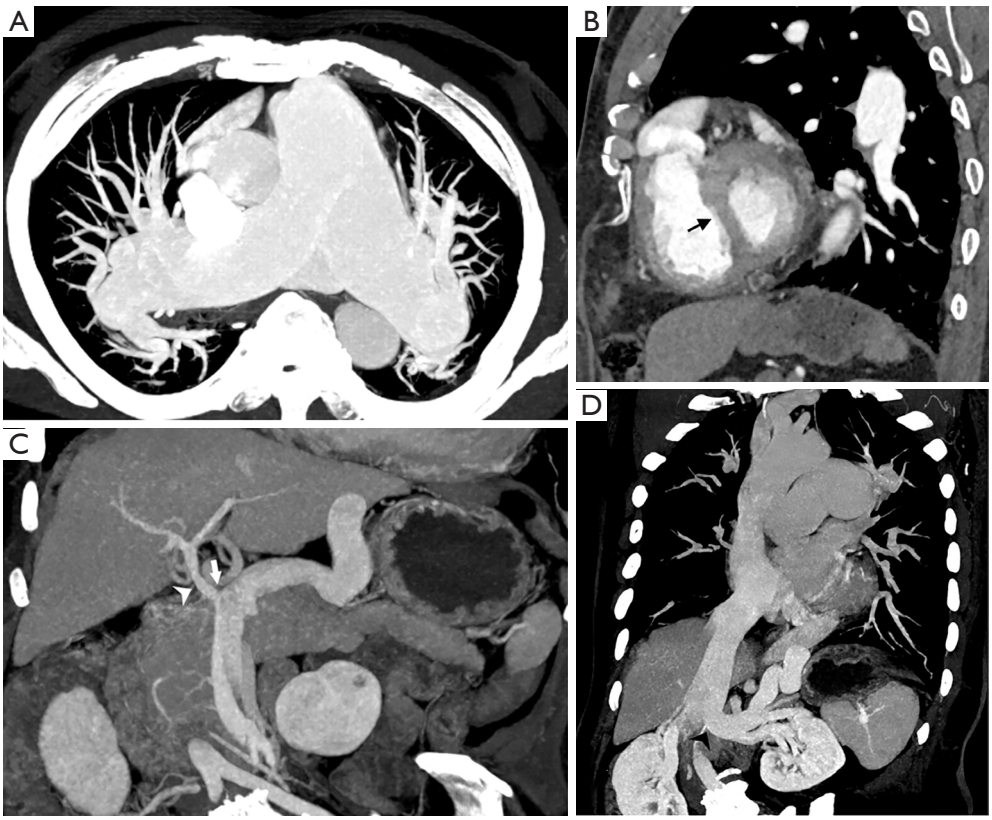

Figure 2 CEPS. (A) MIP reconstruction shows diffuse dilatation of the main pulmonary artery and bilateral pulmonary arteries at all levels. (B) MPR reconstruction shows enlarged right ventricle and flattened interventricular septum (arrow) leading to 'D-shaped' left ventricle, demonstrating severe pulmonary hypertension. (C,D) Supplemental abdominal examination shows CEPS, the intrahepatic portal vein is dysplastic, and the main trunk of the portal vein and its branches are small (arrowhead). Part of the splenic vein and superior mesenteric vein joins into the portal vein (arrow), and the other part joins with the left renal vein into the inferior vena cava (black arrowhead). This patient eventually died of acute right heart failure due to pulmonary hypertension. CEPS, congenital extrahepatic portosystemic shunt; MIP, maximum intensity projection; MPR, multiplanar reformation.

identified, other causes of PH (e.g., chronic pulmonary embolism with PH) should be considered. Chronic pulmonary embolism is characterized by sudden reduction and truncation of the PA on CTPA, and poor filling of the distal PA, which is not clearly observed in images. It may also present with segmental and subsegmental PA diameter reduction compared with the accompanying bronchi. An organized thrombus forms an eccentric wall-filling defect in the PA that is obtuse to the PA wall and may be accompanied by calcification. Recanalization of thromboses appears on CTPA images as a reticular filling defect in the PA lumen, which is more common in the segmental and subsegmental PAs. In concomitant PH, the unaffected PAs may appear diffusely dilated or dilated after stenosis (*Figure 3*).

Since PH has a variety of causes, in addition to the morphological changes of the PAs, if there is a primary disease and related morphological changes, close attention should also be paid to the pulmonary vessels (patency, lesions in the lumen, wall, or perivascular), lungs, heart (morphology, structure, and function), mediastinum, and hilar structures in the image, which can help to identify and evaluate the cause of PH.

Pulmonary valve-related PAA

When MPA dilation is accompanied by unilateral PA trunk dilation, particularly left PA (LPA) dilation, the clinical diagnosis must be made carefully to rule out the existence or absence of valve anomalies. Previous studies have shown that pulmonary valve stenosis (3,4), regurgitation,

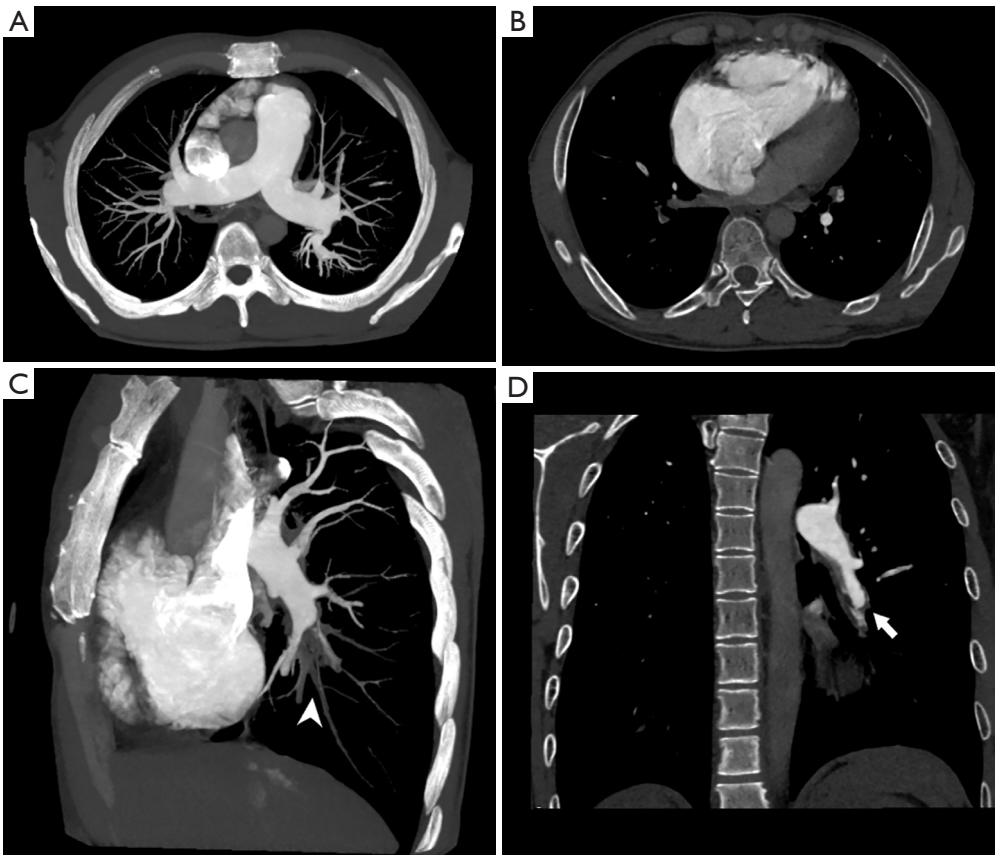

Figure 3 Chronic thromboembolic pulmonary hypertension. A 35-year-old male patient presented with pulmonary hypertension 2 years after thrombolysis for pulmonary embolization. (A) MIP reconstruction showing dilatation of the main and bilateral pulmonary arteries. (B) Chambers of the right ventricle and right atrium are increased. (C,D) MIP and MPR reconstruction show filling defects in both the bilateral lower segmental and subsegmental pulmonary arteries (arrowhead). Partial thrombus recanalization is grid-shaped (arrow). MPR, multiplanar reformation MIP, maximum intensity projection.

or absence can result in PA dilatation (*Figures 4-6*). All PAAs caused by anomalies in the pulmonary valve develop proximally and present considerable dilatation of the main PA, either with or without LPA or right pulmonary artery (RPA) dilatation. It is mostly produced by high-speed blood flow beams striking the vascular wall via the restricted pulmonary valve orifice or by increased blood volume stress on the right side of the heart due to pulmonary valve issues, leading to MPA dilation. Furthermore, the LPA is preferentially opened to the MPA rather than to the right side, leading to preferential shunting of the blood flow to the left and causing the LPA diameter to expand. Since the structural abnormality of the pulmonary valve is easily missed in standard transection CT-enhanced examination, radiologists must focus on the reconstruction and observation of the pulmonary valve through the post-processing workstation in patients with this typical change in the morphology of the PA, because stenosis or regurgitation of the pulmonary valve orifice, or even if the pulmonary valve is absent, may cause the formation of PAA.

PPA caused by Marfan syndrome (MFS)

MFS is an autosomal dominant connective tissue condition that often manifests in youth, with 75% of patients having a family history of MFS. Pathogenic variations in fibrillin-1, the primary structural element of the extracellular matrix that supports connective tissue, particularly in structures such as arteries and periocular areas, cause MFS (10). Diagnosing MFS is challenging because its clinical symptoms are age-related and overlap with those of other connective tissue illnesses. Clinical symptoms, family history, and *FBN1* mutations are used to diagnose MFS.

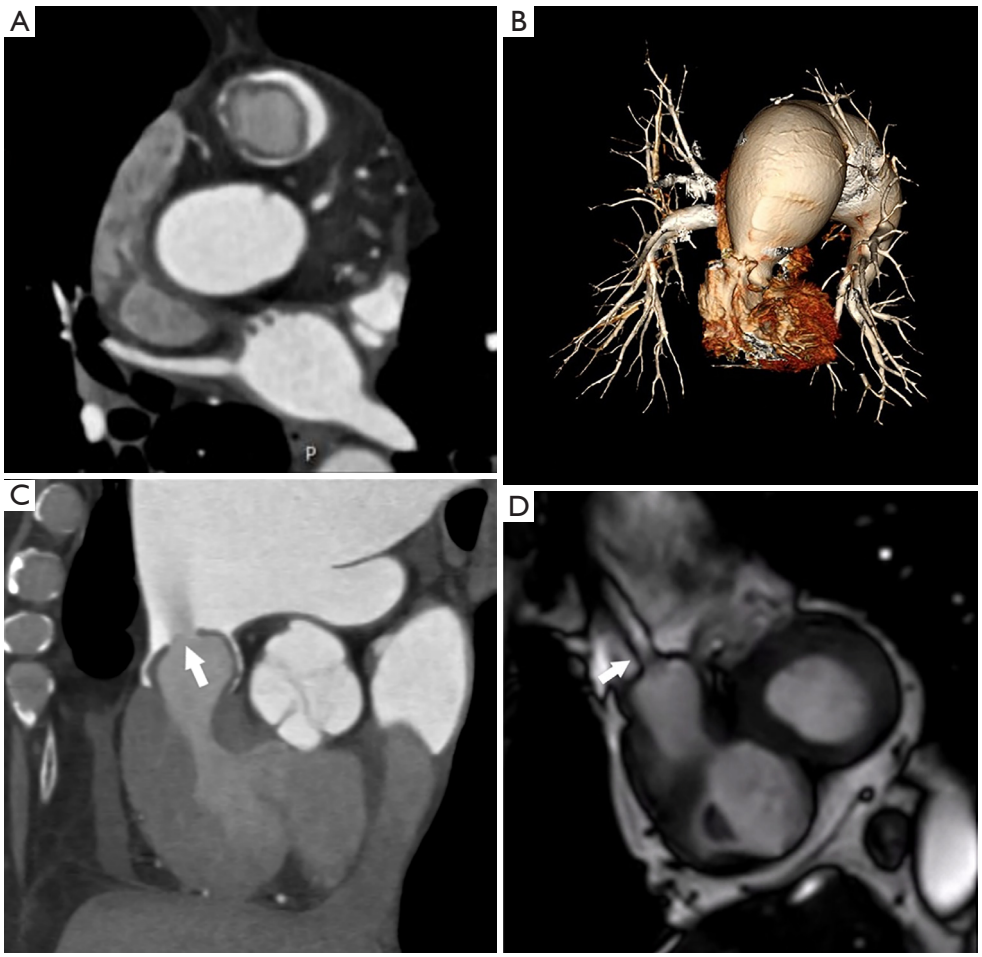

Figure 4 Divalvular malformation of the pulmonary valve. (A) Transpulmonary valve-level MPR reconstruction shows pulmonary valve stenosis due to bivalve malformation of the pulmonary valve. (B) VR reconstruction shows obvious dilation of the main and left trunk of pulmonary artery. (C,D) MPR reconstruction and cardiac magnetic resonance cine shows high-velocity blood flow beams ejecting from the pulmonary artery stenosis valve (arrows). VR, volume rendering; MPR, multiplanar reformation.

The signs of skeletal abnormalities include acetabular protrusion, chest wall deformities (pectus carinatum and pectus excavatum), and scoliosis. Physical examination may reveal thin limbs, reduced subcutaneous fat, slender fingers and toes, and excessive joint mobility.

FBN1 mutation causes reduced elastic fibrin, decreased elasticity of the blood vessel wall, and mucus degeneration of the middle structure of the blood vessel wall, which finally leads to aneurysms and dissections. The main radiographic features of MFS involving the cardiovascular system are ascending aortic dilatation involving the aortic sinus and development of aortic dissection aneurysms. Aortic root dilation may result in aortic annular stretching, which leads to leaflet misalignment and aortic valve regurgitation, often accompanied by aortic valve cusp prolapse and commissural fenestration.

Children and adults with MFS may have PA dilatation, which is linked to aortic root dilatation, prior aortic root surgery, decreased left ventricular ejection fraction, and increased systolic blood pressure in the PAs (11-14). According to a literature review, MFS combined with PA dilatation is uncommon, and we believe that the cause of PA dilatation in patients with MFS may be due to reduced vascular wall elasticity, which may not necessarily be combined with PH. If simultaneous dilatation of the PA and thoracic aorta is observed on the image, especially in the aortic and PA segments closest to the heart, the possibility of MFS should be considered in close

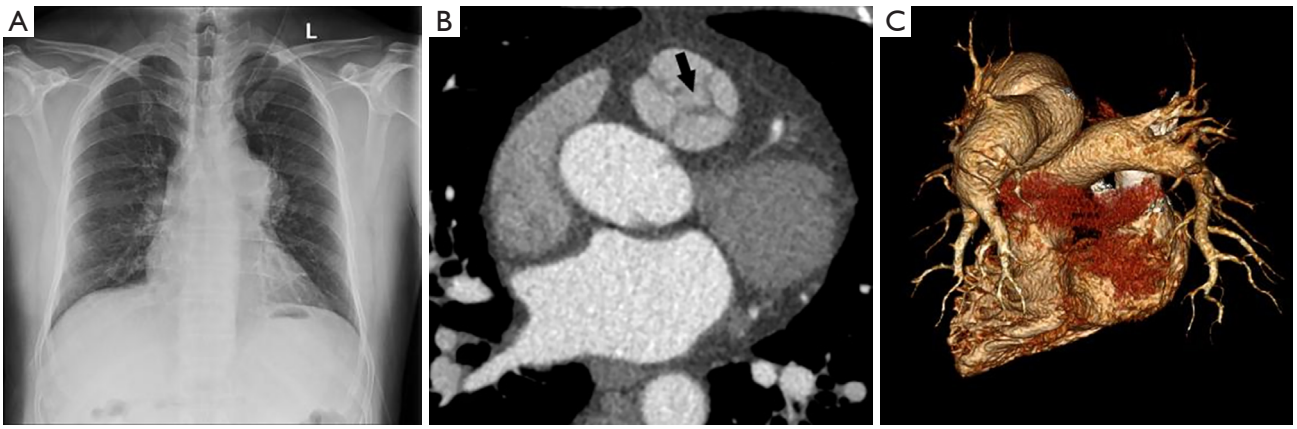

Figure 5 Quadricuspid pulmonary valve. (A) Chest X-ray shows enlarging of the pulmonary trunk and slender blood vessel shadow of the peripheral lung field. (B) Transpulmonary valve-level MPR reconstruction shows the quadricuspid pulmonary valve and incomplete central closure of the valve opening leading to pulmonary valve regurgitation (arrow). (C) VR reconstruction shows dilation of the main and left trunk of the pulmonary artery but no definite dilation of the distal branches. MPR, multiplanar reformation; VR, volume rendering.

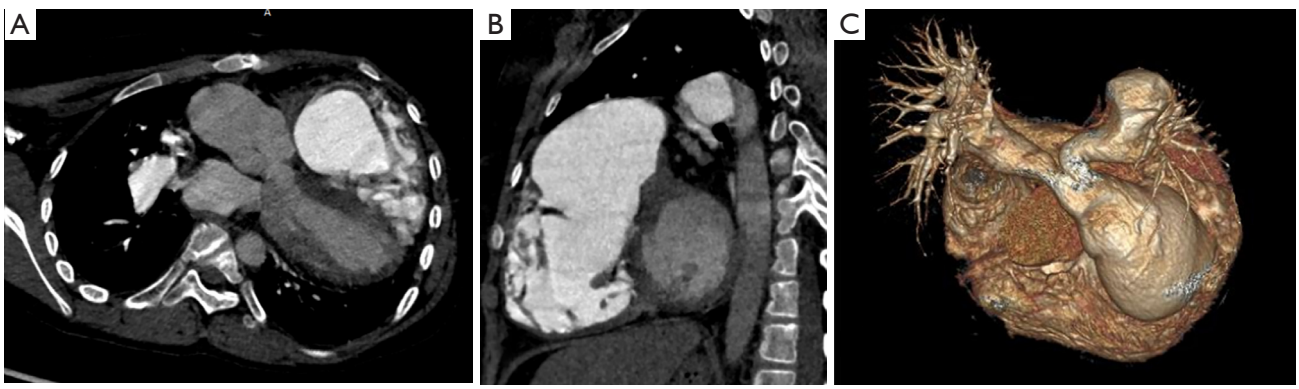

Figure 6 Absent pulmonary valve. (A) MPR reconstruction at the pulmonary valve level shows absence of the pulmonary valve. (B) The main pulmonary artery is markedly dilated. (C) VR reconstruction shows bilateral pulmonary artery aneurysmal dilation, and the distal branches are small universally. MPR, multiplanar reformation; VR, volume rendering.

combination with the patient's clinical manifestations and medical history (*Figure* 7).

Idiopathic PA dilatation in association with PAA

When PAAs due to blood flow factors and vascular wall abnormalities are excluded in a close context with clinical history, PA dilatation with no proven etiology is categorized as idiopathic PA dilation (*Figure 8*). Greene *et al.* established 4 criteria for idiopathic pulmonary aneurysm (15): single dilation of the PA trunk, with or without expansion of adjacent PA branches, absence of abnormal intracardiac or extracardiac shunts, no chronic heart or lung disease, and no arterial disease, such as arteritis or pulmonary arteriosclerosis due to syphilis.

Dilation of the lobar or segmental PA

PAA caused by Behcet's disease (BD)

BD is a systemic inflammatory disease of unknown etiology. The mouth-eye-genital triad is the most prevalent clinical manifestation of BD, with recurrent mouth ulcers being the most common clinical sign. Patients may also develop ophthalmitis (e.g., uveitis and optic neuropathy) or skin lesions (e.g., erythema nodosum). The geographical distribution of BD is distinct. It is more common in Mediterranean and East Asian nations such as Japan, Korea,

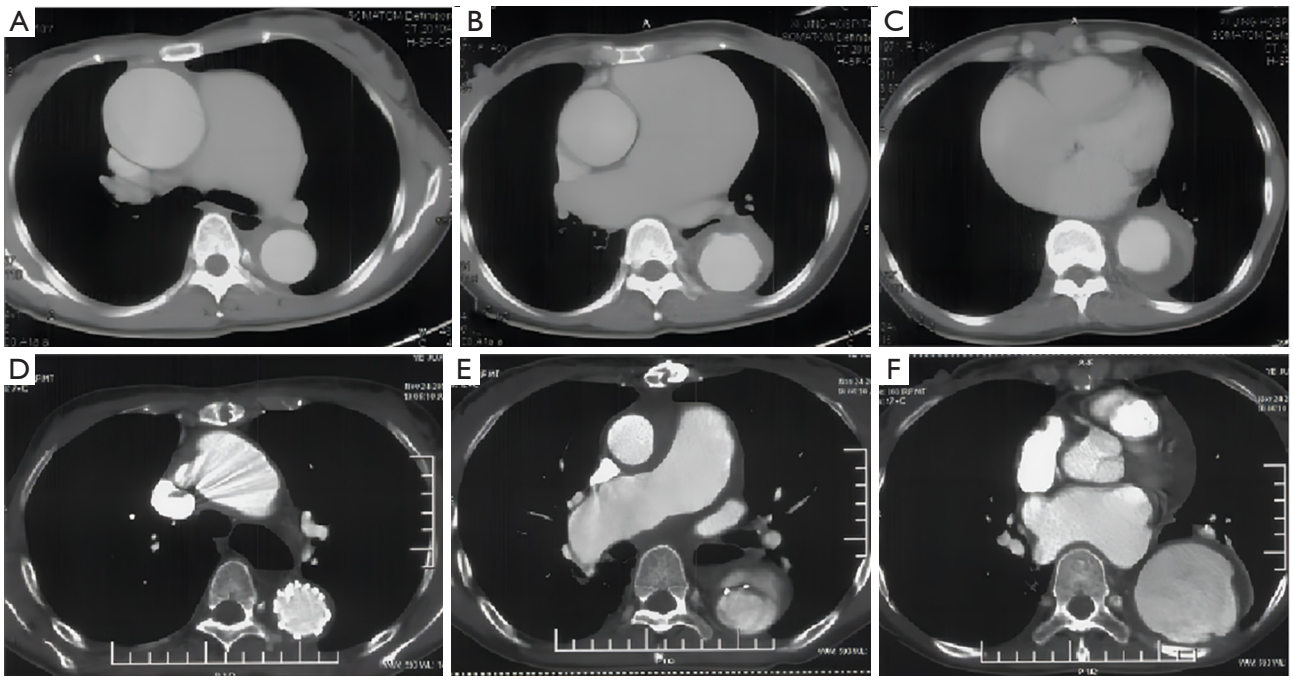

Figure 7 Marfan syndrome. (A-C) Contrast-enhanced CT examination in patients with Marfan syndrome shows simultaneous dilation of the ascending aorta and the main pulmonary artery and intramural hematoma of the descending aorta. (D-F) CT reexamination 3 months after thoracic aortic arch stenting shows that the descending aorta tube diameter is significantly wider than that reported the previous time with dissection. CT, computed tomography.

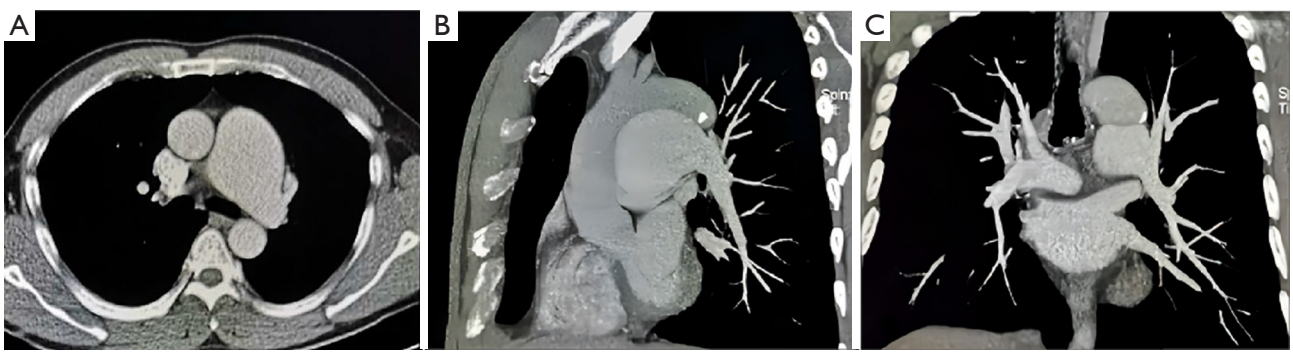

Figure 8 Idiopathic pulmonary hypertension in a 38-year-old male. Physical examination revealed enlargement of the pulmonary artery. The patient had a history of military service for 3 years. Follow-up for 3 years revealed no relevant clinical symptoms. (A-C) CTPA imaging and MPR reconstruction show dilation of the main and left trunk of the pulmonary artery but no dilation of the distal branch. CTPA, computed tomography pulmonary angiography; MPR, multiplanar reformation.

and China.

In the advanced stages, BD manifests in the thoracic region, including venous thrombosis, pulmonary infarction and hemorrhage, recurrent pneumonia, organizing pneumonia, and pleural effusion. Radiographic observations of single or multiple pulmonary aneurysms with thicker walls, non-smooth inner walls, and *in situ* neighboring thrombosis are prevalent when BD affects pulmonary arteries (16). They are usually multiple, bilateral, saccular, and partly or totally thrombosed, in the majority of patients (*Figure 9*). Patients with BD may also have thickened bronchial arteries around the afflicted PA, as well as

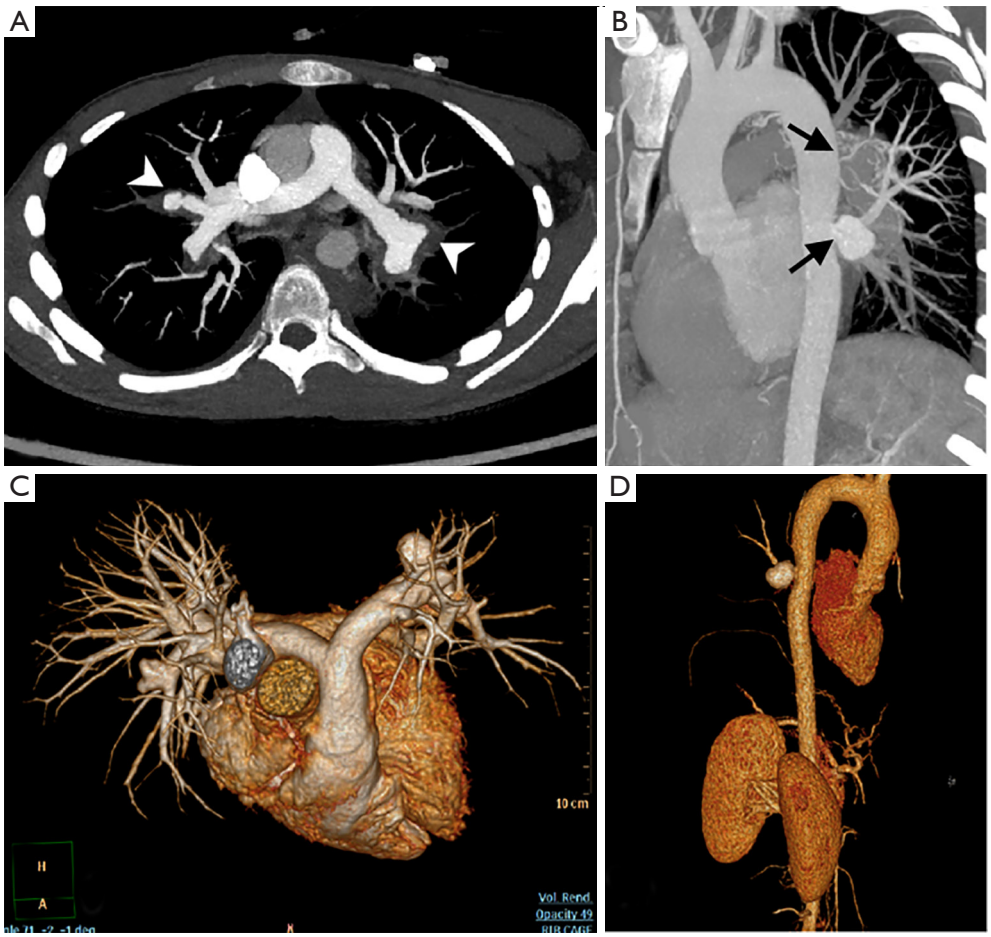

Figure 9 Behcet's disease. An 18-year-old male complained of hemoptysis. (A) MPR shows bilateral multiple PAA with thickened walls (arrowhead) and occlusion of the distal pulmonary arteries caused by thrombosis. (B) MIP reconstruction shows descending aorta pseudoaneurysm and thickened bronchial arteries around the pulmonary artery (arrows). (C,D) VR reconstruction shows multiple pseudo-PAA and pseudoaneurysm of the descending aorta. MPR, multiplanar reformation; PAA, pulmonary artery aneurysm; MIP, maximum intensity projection; VR, volume rendering.

blockage of the distal branch of the PAA. Localized regions of consolidation or ground-glass opacities may suggest pulmonary bleeding due to PA pseudoaneurysm (PAP) rupture or vasculitis. Medical treatment with cytostatic agents and corticosteroids can induce regression. However, varying degrees of repeated hemoptysis are common and are one of the leading causes of death (17). The rupture of an aneurysm with erosion into the bronchus and the development of *in situ* thrombosis from active vasculitis have been postulated as causes of hemoptysis (18). Therefore, it is clinically vital to rule out BD in conjunction with pertinent clinical symptoms when the patient has no apparent history of trauma and an imaging scan reveals a PA branch aneurysm.

Infection-related PAA

Syphilis, tuberculosis (TB), purulent bacterial infection, fungal pneumonia, and other diseases may damage the local PA wall, resulting in pleuropulmonary aneurysms, pulmonary bleeding, and life-threatening hemoptysis. Patients often have or have had pulmonary infections in the past, and non-contrast chest CT may easily miss or conceal the existence of a localized pulmonary pseudoaneurysm in patients with atelectasis, cavitation, lung abscess, or lung consolidation.

Rasmussen aneurysms, which are pseudoaneurysms of the PA resulting from pulmonary TB, are often located in the upper lung lobe in patients with recurrent active TB. The lung window reveals pulmonary cavity lesions

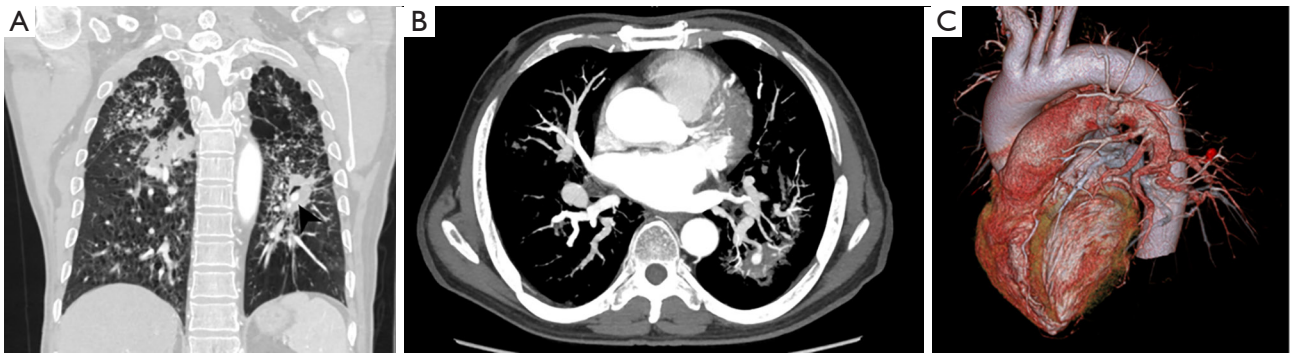

Figure 10 Rasmussen aneurysms. (A) Multiple tuberculosis foci are visible in both lungs in the chest CT lung window. A significantly enhanced wall nodule is seen within the cavity in the left inferior lobe (arrowhead), suggesting the formation of PAA. (B,C) MIP and VR reconstructions are more intuitive for observing the relationship between the PAA and PA. CT, computed tomography; PAA, pulmonary artery aneurysm; MIP, maximum intensity projection; VR, volume rendering; PA, pulmonary artery.

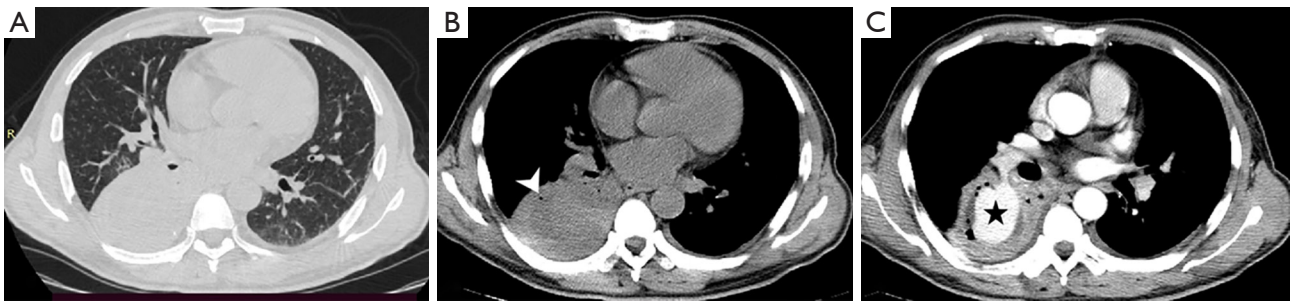

Figure 11 Mucor infection with PAA. (A) Lung window shows a mass in the lower lobe of the right lung. (B) A small amount of gas (arrowhead) can be observed within this lesion in the mediastinal window. (C) Contrast-enhanced scan shows a significantly enhanced lesion (star) communicating with the adjacent pulmonary artery. The patient was eventually diagnosed with mucor infection. PAA, pulmonary artery aneurysm.

with wall nodules, which were markedly accentuated on an enhanced scan (*Figure 10*). Segmental artery dilatation occurs as the elastic fibers in the blood vessel wall break down and are replaced by granulation tissue. This occurs throughout the healing phase of the disease and explains the delayed onset of PAP. In active TB cases, the bronchial arteries cause more hemoptysis than in PAP. Nonetheless, if hemoptysis develops, pulmonary circulation should be assessed, particularly if bronchial angiography findings are normal (19).

Bacterial and fungal pulmonary pseudoaneurysms are the most common among intravenous drug users and are often associated with infective endocarditis and septic embolism. When such patients are immunocompromised, especially those secondary to blood-borne bacterial infections (e.g., simple pulmonary eosinophilic infiltration, septic pulmonary embolism) or airway spread fungal infections (e.g., mucor infection, invasive *Aspergillus* infection), the clinical manifestations are secondary to infection-induced fever and hemoptysis, and clinicians must be highly vigilant to the formation of infection-related pulmonary pseudoaneurysms (*Figures 11*,*12*).

Iatrogenic-related PAA

Patients with iatrogenic-related PAAs have a clear history of invasive medical operations. The clinical manifestations are massive hemoptysis and pulmonary hemorrhage, and the characteristic imaging manifestation is a pulmonary pseudoaneurysm, all of which occur in the relevant lung area where medical operations have been performed in the past. PAPs secondary to various invasive medical procedures have been reported in the literature (20), such as Swan-

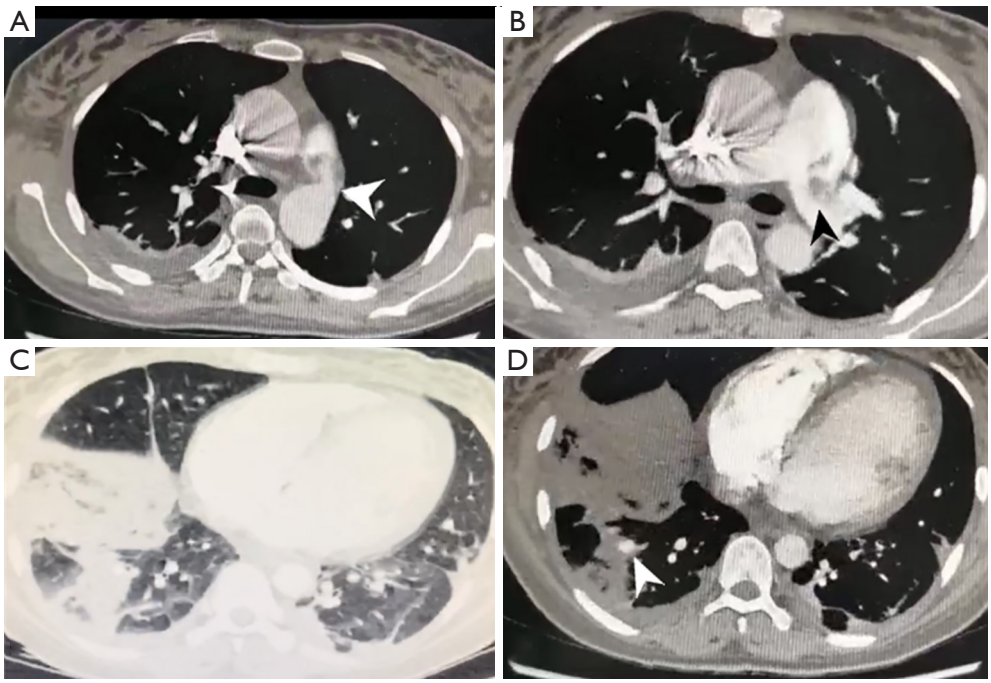

Figure 12 PDA combined with infective endocarditis and PAA. (A,B) CTPA images show PDA (arrowhead) and dilation of the main and left trunk of the pulmonary artery with bacterial thrombi formation (black arrowhead). (C,D) Lung and mediastinal window shows multiple inflammatory lesions of both lungs, and small nodules with obvious enhancement (arrowhead) can be seen in the inflammatory lesions of the right lower lobe on enhanced examination. PDA, patent ductus arteriosus; PAA, pulmonary artery aneurysm; CTPA, computed tomography pulmonary angiography.

Ganz catheter placement, conventional angiography, needle biopsy, chest drain placement, and pulmonary wedge resection. Displaced Swan-Ganz catheters are an increasingly common cause of iatrogenic pulmonary pseudoaneurysms (5). Insertion of the distal tip of the catheter erodes the PA wall, causing the PA to weaken and dilate, possibly forming a PAA. PAAs may also form by distal catheter displacement during balloon deflation, retraction, or hyperbaric inflation. Clinical history is critical for diagnosis and interventional coil embolization is the first-line treatment.

PAAs caused by neoplasms

PAAs may develop as a result of direct invasion and destruction of the natural structure of the PA wall by primary lung malignancies and metastases (e.g., myxoma and choriocarcinoma; *Figure 13*). Focal and aneurysmal dilatations may also result from primary malignancies originating in the PA, such as leiomyosarcoma and angiosarcoma. Clinically, there is usually a history of sudden hemoptysis. During follow-up, CTPA may be used to assist in identifying the development of a pulmonary pseudoaneurysm and differentiate the border between the tumor tissue and the pseudoaneurysm.

Trauma-related PAAs

These patients have a well-documented history of trauma, such as a fall from a great height, vehicle accident, knife wound, or gunshot wound. By reviewing past case reports (20), in addition to mediastinal hematoma and lung contusion, imaging features of patients who had experienced car accidents and height falls revealed that pulmonary aneurysms occur primarily in the area entering and leaving the pericardium because of the relatively fixed position of the inner segment of the PA pericardium and the thoracic shear force, particularly in the left MPA. Pulmonary pseudoaneurysms arise around the knife or bullet entry channels in individuals with knife or gunshot wounds. Connectivity between the normal PA and the pulmonary aneurysm may be observed on CTPA or angiography.

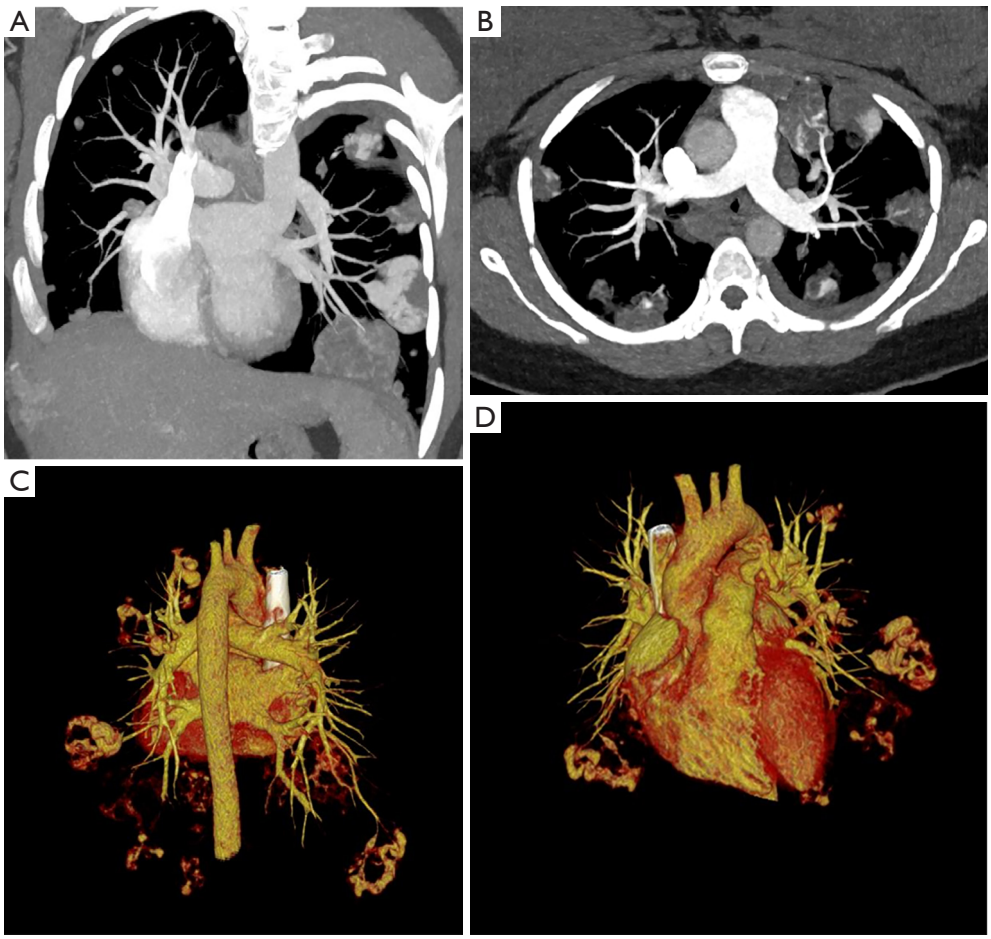

Figure 13 Uterine chorionic carcinoma with multiple PAA. This patient was diagnosed with uterine chorionic carcinoma and experienced sudden hemoptysis. (A-D) MIP and VR reconstruction show multiple metastases in both lungs with PAA, and pulmonary arteriovenous fistulas are seen in some metastases. PAA, pulmonary artery aneurysm; MPR, multiplanar reformation; VR, volume rendering.

Conclusions

Previous studies have defined pulmonary aneurysms primarily based on etiology, including congenital, acquired, and idiopathic. Various locations of PA dilation are discussed and analyzed in this review (*Figure 14*). First, the role of the blood flow factors was considered. Based on the characteristic morphological changes in the expansion of the MPA and its branches, patients were targeted for observation of CHD with increased pulmonary blood, hilar-body venous shunt, or pulmonary valve abnormality. Second, to study and infer the etiology of pulmonary aneurysms, it is necessary to carefully integrate the clinical history and features of patients and pay attention to local PA wall morphological alterations and peripheral PA abnormalities. If the previous hypotheses fail to identify a definite cause of PA dilation, these 4 criteria must be used to consider the possibility of idiopathic PA dilation.

Additionally, we believe that when a patient has massive hemoptysis that necessitates immediate surgical intervention, it is critical to determine whether the primary cause of hemoptysis is a dilated bronchial artery or a ruptured pulmonary aneurysm, and it is recommended that dual-phase CTPA be performed prior to interventional procedures to identify the primary cause of hemoptysis before proceeding.

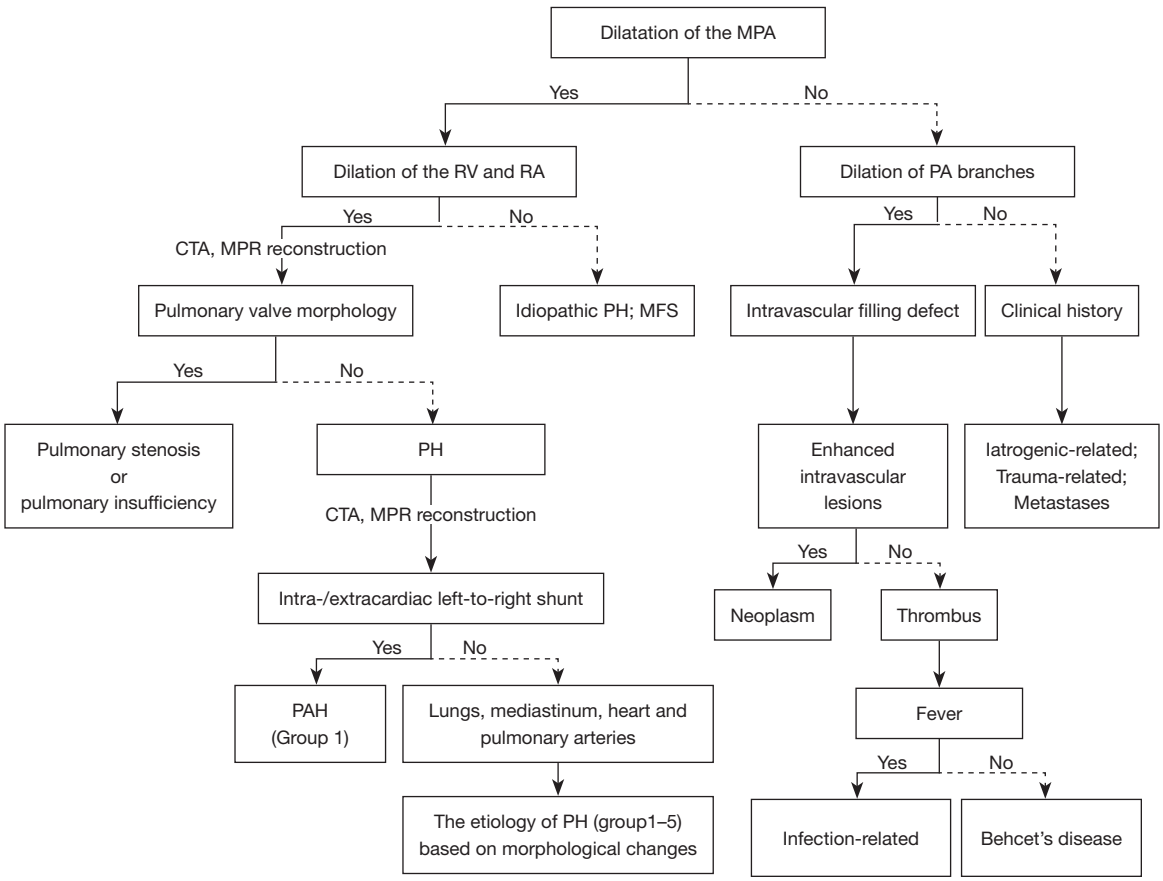

Figure 14 Diagnostic flowchart of diagnosing pulmonary artery aneurysm. MPA, main pulmonary artery; RA, right atrium; RV, right ventricle; PA, pulmonary artery; CTA, computed tomography angiography; MPR, multiplanar reformation; PH, pulmonary hypertension; MFS, Marfan syndrome; PAH, pulmonary arterial hypertension.

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Footnote

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://qims.amegroups.com/article/view/10.21037/qims-24-462/coif). Z.S. serves as an unpaid associate editor of *Quantitative Imaging in Medicine and Surgery*. The other authors have no conflicts of interest to declare.

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