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Case Report

Delayed systemic arterial-pulmonary arterial shunt simulating pulmonary embolism: An unusual case ☆

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ABSTRACT

This case report describes the imaging findings of an older-aged male presenting with infectious respiratory symptoms. Evaluation with routine contrast-enhanced CT of the chest demonstrated pulmonary artery filling defects initially treated as a pulmonary embolism. However, short-term repeat imaging during pulmonary angiographic and delayed phases demonstrated retrograde filling through a systemic arterial-pulmonary arterial shunt. Given the high prevalence of pulmonary embolism which is also the leading cause of pulmonary angiographic filling defects, this case highlights the importance for clinicians to maintain a differential diagnosis and consider alternative etiologies.

Introduction

Pulmonary embolism (PE) is believed to result from a combination of hypercoagulability, vascular endothelial injury, and stasis, with a wide range of inciting factors including congenital or acquired coagulation disorders, hormone replacement therapy or oral contraceptives, critical illness, recent surgery, or chronic heart and lung disease [1]. Additionally, PE maintains a high incidence and precludes a high rate of morbidity and mortality [1,2]. PE is also the most common cause of filling defect within the pulmonary arterial system on CT pulmonary angiogram (CTPA) which is the current gold standard for diagnosis [3]. However, much less common etiologies of pulmonary angiographic filling defects are more prevalent in patients with chronic inflammatory pulmonary disease which may lead to a false positive diagnosis [4]. We report a case of a patient with an acquired systemic arterial-pulmonary arterial (SAPA) shunt that was initially diagnosed as a PE.

Case report

A 74-year-old male with a history significant for treated laryngeal carcinoma and recurrent aspiration initially presented

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

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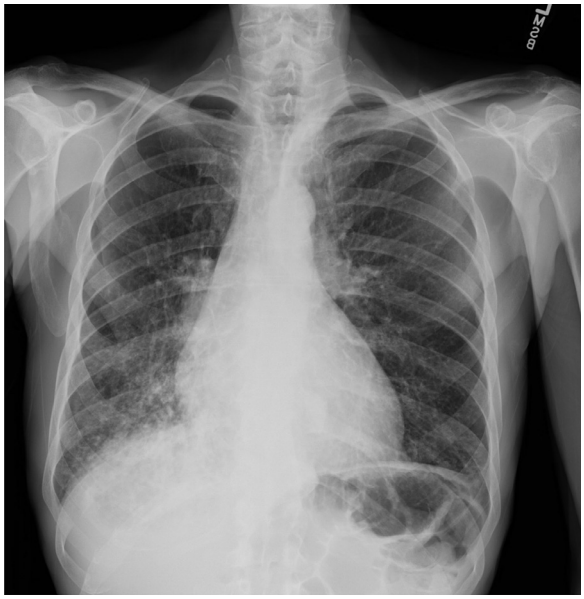

Fig. 1 – Chest radiograph shows right basilar reticulonodular opacities concerning for pneumonia.

with fatigue and shortness of breath. Clinical findings included no adventitious lung sounds on auscultation with the patient normoxic and afebrile. Mild hypercarbia was noted on venous blood gas with serum markers otherwise insignificant. A chest radiograph showed right basilar opacity concerning for pneumonia (Fig. 1) for which the patient was started on antibiotics.

Due to developing hypoxemia at an outpatient follow-up appointment, a routine contrast-enhanced CT of the chest was performed. Images demonstrated right middle and right lower lobe bronchiectasis with scattered nodular and consolidative opacities consistent with acute on chronic infection (Figs. 2A and B). Additionally, a large filling defect in the distal right main pulmonary artery extending into the right middle and right lower lobar branches was seen suggestive of PE (Figs. 3A and B). He was then referred to the emergency department (ED) for urgent medical management. Assessment in the ED revealed a heart rhythm of atrial fibrillation with rapid ventricular rate, depressed left ventricular function, and elevated serum cardiac enzymes. Blood count and serum chemistries were normal and the patient remained hemodynamically stable. The patient was admitted to the medical floor for further management of cardiac dysfunction and was started on heparin.

The PE response team was consulted for evaluation of presumed PE management. However, after collaborative discussion with the interventionalist and medical management teams there was concern for possible artifact degrading diagnostic accuracy of the presumed PE and therefore repeat imaging was recommended. CTPA with additional 30-second delayed images was repeated which again yielded filling defects in the distal right main, right middle, and right lower lobar pulmonary arteries on CTPA images (Figs. 4A and B) which were subsequently shown to fully opacify on the delayed phase images (Figs. 5A and B); an area of suspected col-

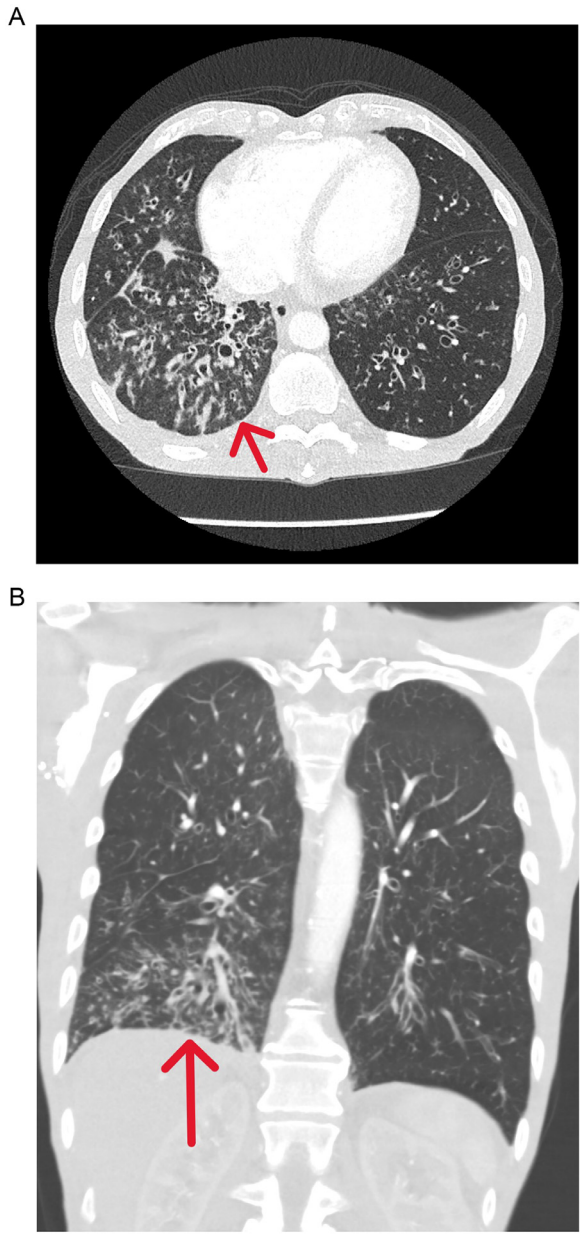

Fig. 2 – (A) Axial image shows bibasilar predominant bronchiectasis with patchy airspace consolidations in the right lower lobe consistent with acute on chronic infection. (B) Coronal maximum intensity projection (MIP) also demonstrates basilar predominant bronchiectasis bilaterally with airspace opacities in the right lower lobe.

lateralization from the adjacent systemic bronchial arterial system was noted (Figs. 6A-C). Additionally, no evidence of deep vein thrombosis was seen on lower extremity Doppler.

Discussion

The pulmonary arterial filling defect in this case was a result of the right pulmonary artery blood flow supplied by a sys-

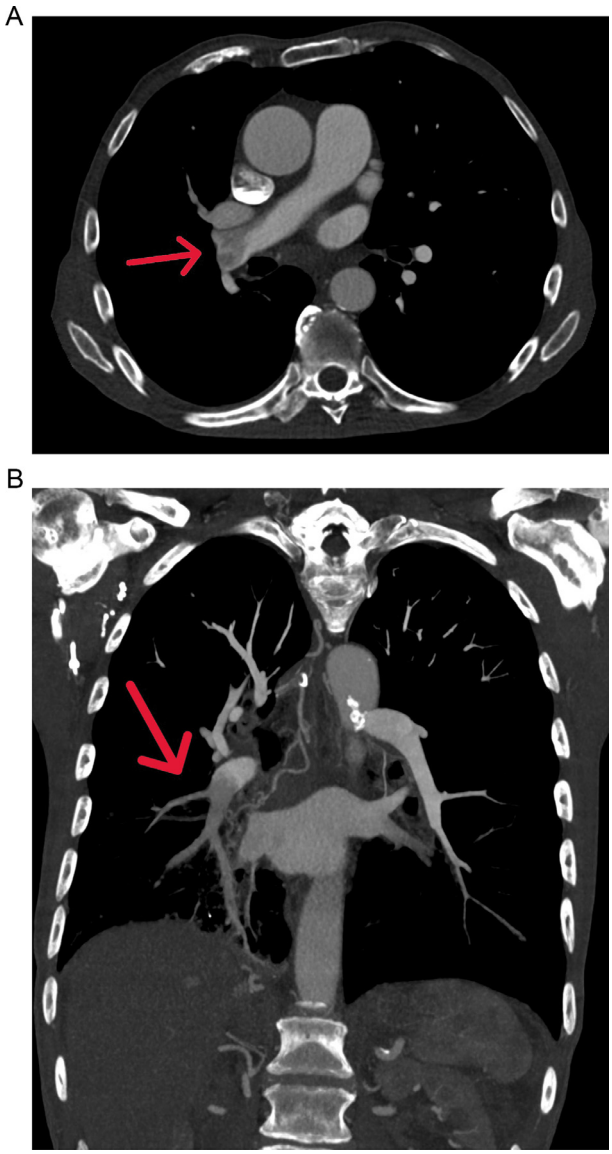

Fig. 3 – (A) Axial image during routine contrast administration reveals a filling defect in the distal right main pulmonary artery extending into the right middle lobar branches. (B) Coronal MIP image during routine contrast administration again demonstrates a filling defect in the distal right main pulmonary artery and right lower lobar branches.

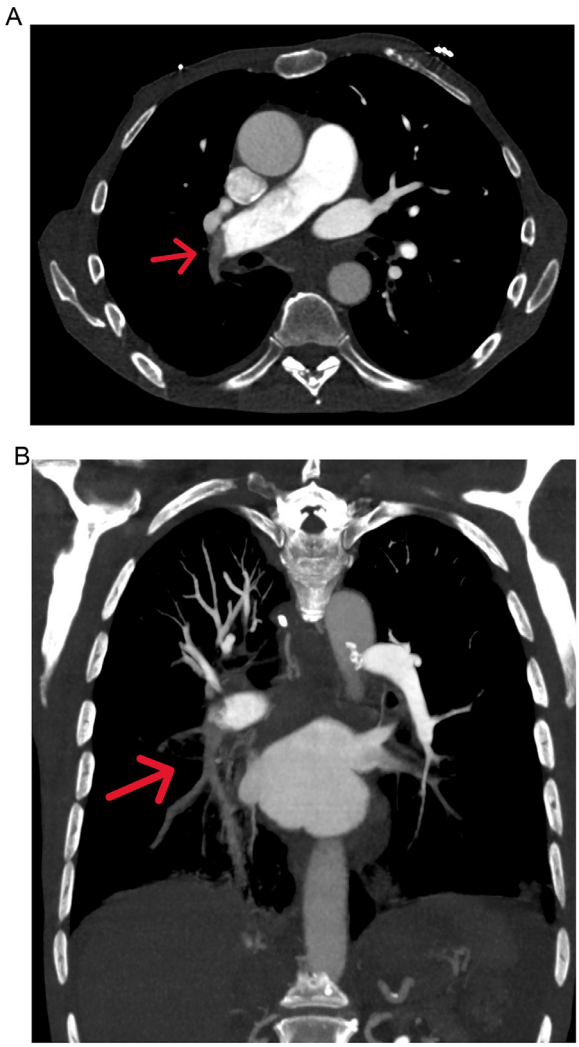

Fig. 4 – (A) Axial slice during CTPA shows an irregular filling defect within the right main pulmonary artery with a more avid, homogeneous filling defect within the distal main pulmonary artery extending into the right lower lobe. (B) Coronal MIP image during CTPA shows the filling defect extending into the right lower lobar branches.

temic bronchial artery from a presumed acquired SAPA shunt creating delayed contrast enhancement of the pulmonary arterial system. CTPA is timed to visualize contrast enhancing the pulmonary arteries after normal physiologic blood flow of contrast administered peripherally travels from the systemic veins, into the right heart and then into the pulmonary arteries. Therefore, the SAPA shunt in this case was not opacified on CTPA since contrast had not yet reached the systemic arterial system via the left heart. Conversely, the initial routine, nontimed contrast-enhanced CT was acquired after the SAPA aberrant connection had already redirected contrast through the shunt and progressed downstream via low-flow pulmonary artery physiology, therefore not highlighting the anomalous anatomy [2].

SAPA shunts are rare entities but have been known to develop as a result of chronic pulmonary inflammation typically in the setting of parenchymal damage requiring collateral blood supply, often neovasculature arising from adjacent vessels regardless of their origin. These anastomoses often divert blood from the high-pressure systemic system to the low-flow pulmonary circuit that may flow antegrade or retrograde depending on the degree of pulmonary artery bed obstruction [5]. Though often asymptomatic, due to the aberrant anatomy and physiology of this connection these vessels are prone to injury with hemoptysis. This common presentation may also be an initial symptom of PE, where evaluation with CTPA yields the first indication of a SAPA shunt usually with a pulmonary arterial filling defect [5,6]. Other less common

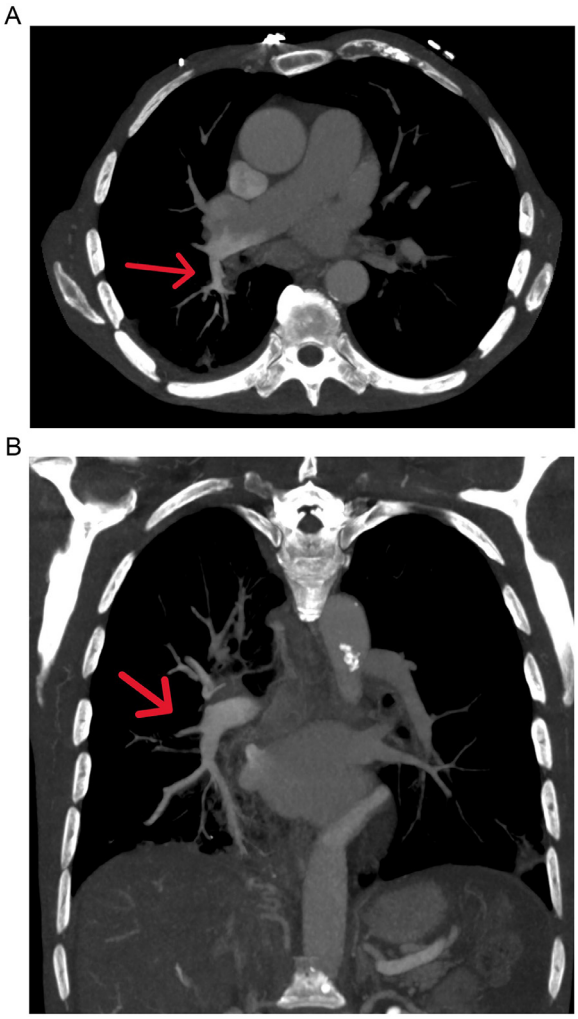

Fig. 5 – (A) Axial MIP slice during 30 second delayed CTPA shows homogeneous contrast opacification of the distal right main pulmonary artery and visualized right lower lobar branches. (B) CTPA 30 second delayed coronal MIP demonstrates clear enhancement of the distal right main pulmonary artery as well as right lower lobar branches.

sources of pulmonary artery filling defects are respiratory motion, flow-related, or partial volume averaging artifacts as well as the presence of pulmonary arterial catheters. More rarely, pulmonary artery sarcoma or secondary tumor thrombus may also simulate filling defects [4]. Tumor thrombus was initially considered upon the first diagnosis of PE but felt less likely as there was no adjacent mass invading the pulmonary vasculature or a known distal tumor thrombus that would embolize centrally.

Bronchiectasis is a known result of chronic pulmonary inflammation, classically associated with dysregulated immunity in the setting of asthma, chronic obstructive pulmonary disease, cystic fibrosis, and tuberculosis leading to recurrent infections [7]. This chronic inflammation leads to alveolar and bronchiolar wall damage, particularly to the elastic and muscular layers resulting in abnormal dilation of the airways which is readily apparent on CT. This airway injury increases

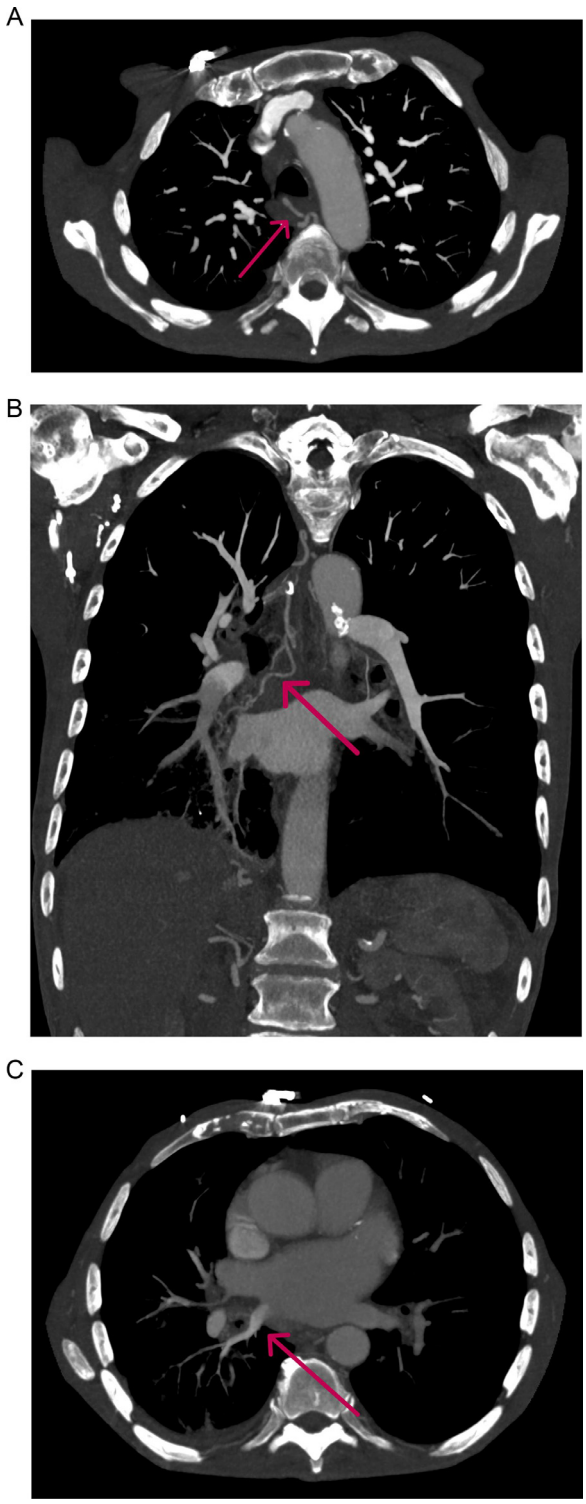

Fig. 6 – (A) CTPA axial MIP demonstrates an enlarged bronchial artery. (B) Routine contrast enhanced coronal MIP shows an enlarged and tortuous systemic bronchial artery extending to the right lower lobar pulmonary artery consistent with SAPA shunt. (C) CTPA 30 second delayed phase axial image shows delayed enhancement of the right inferior pulmonary vein in keeping with slow right lower lobe venous outflow from the SAPA shunt.

susceptibility to pulmonary infection that then leads to further airway damage in a spiraling cycle of infection [8], often with profound lung parenchymal distortion in the setting of severe diseases such as cystic fibrosis or infiltrative microbiota such as *Mycobacteria*. This case is unusual in the fact that this patient developed a SAPA shunt in the setting of relatively mild chronic lung inflammation, where most cases reported depict patients with more advanced lung inflammation and architectural distortion.

Conclusion

SAPA shunts are rare entities usually as a result of chronic pulmonary disease that may present with filling defects on CTPA simulating PE. Clinicians and radiologists should be aware of this pathology in the appropriate patient demographic, as this may prevent the unnecessary use of anticoagulants and unneeded healthcare expenses. Furthermore, this case report depicts the importance for radiologists to maintain a diagnostic differential when interpreting imaging studies including those deemed as routine procedures.

Patient consent

A written and informed consent was obtained from the patient for publication of this case report and accompanying images.

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