Radiology Case Reports 16 (2021) 128–131

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Case Report

Multiple pulmonary artery mycotic aneurysms and septic emboli in a patient with tricuspid valve vegetation and infective endocarditis ☆,☆☆

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ARTICLE INFO

Article history:
Received 28 September 2020
Revised 27 October 2020
Accepted 2 November 2020

Keywords:
Pulmonary artery
Mycotic aneurysm
Septic emboli

ABSTRACT

Mycotic pulmonary artery aneurysms are rare infectious aneurysmal dilatations of the pulmonary arteries in patients with risk factors of intravenous drug use, endocarditis, or congenital heart disease. Timely diagnosis is crucial given high mortality rate associated with this condition. We present a rare case of a 24-year old male with history of intravenous drug use who presented with fever, hypoxia, and bacteremia. The patient was subsequently diagnosed with infective endocarditis with septic vegetations of the tricuspid valve. Computed tomography angiogram demonstrated multiple bilateral mycotic pulmonary artery aneurysms and associated pulmonary septic emboli in this patient with infective endocarditis. Treatment options for mycotic pulmonary artery aneurysms are variable and include conservative management, endovascular coil intervention, or surgical resections. Presence of hemoptysis and increasing aneurysm size may warrant aggressive intervention.

Introduction

Mycotic aneurysms are aneurysmal dilatations of blood vessels due to infections of vessel walls, usually due to hematogenous seeding from septic emboli and endocarditis. Common causative organisms of mycotic aneurysms are *Staphylococcus* and *Streptococcus* species. Most common sites of aneurysms in the body include the aorta, visceral arteries, and cerebral arteries [1-2]. However, mycotic aneurysm involving the pulmonary artery is much less common and only small number of cases have been reported in literature [3-5]. Multiple bilateral mycotic pulmonary artery aneurysms within a single patient is even more rare. Here we present a case of multiple bilateral pulmonary artery mycotic aneurysms with associated septic emboli in a patient with known bacterial endocarditis of the tricuspid valve.

Case report

A 24-year-old male with a history of intravenous drug use (IVDU) presented with 2 weeks of low-grade fever, cough,

☆ Competing Interests: None.
☆☆ The submitted article does not constitute "Redundant Publication."
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https://doi.org/10.1016/j.radcr.2020.11.006

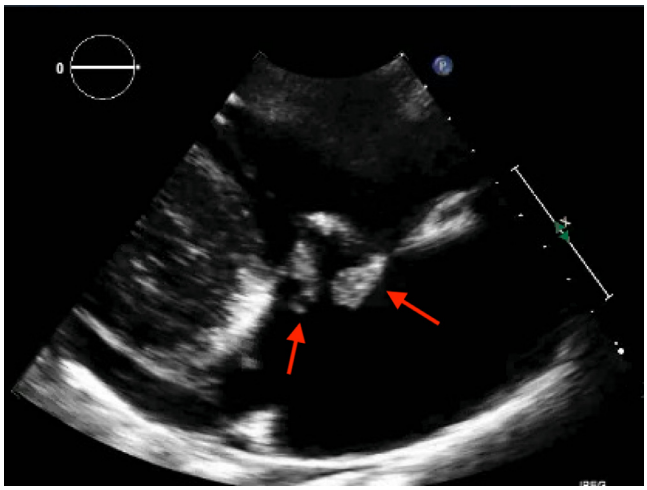

Fig. 1 – Echocardiogram demonstrated multiple large mobile echogenic masses on the tricuspid valve (red arrows).

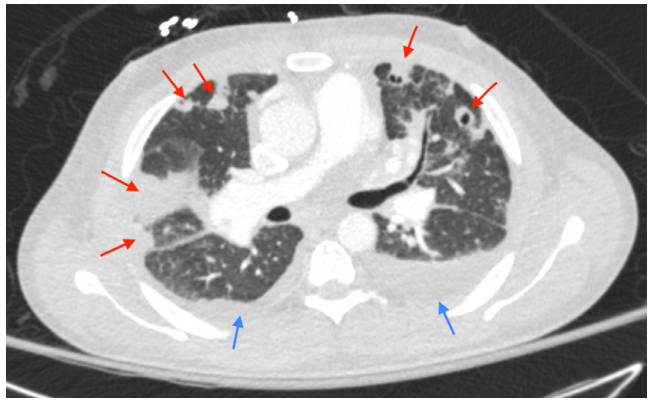

Fig. 2 – Axial CT of the chest on lung windows demonstrated numerous cavitary and noncavitary pulmonary nodules and masses throughout the bilateral lungs (red arrows). Bilateral, left greater than right pleural effusions were also noted (blue arrows).

sore throat, and malaise. On admission, patient was found to be febrile to 102°F with leukocytosis, elevated CRP, and methicillin-sensitive *Staphylococcus aureus* (MSSA) bacteremia. Coronavirus disease (COVID-19) polymerase chain reaction was initially negative. Echocardiogram demonstrated multiple large mobile echogenic masses on the tricuspid valve (Fig. 1) with evidence of valve perforation. Patient's overall condition was consistent with MSSA tricuspid valve infective endocarditis and treatment with intravenous antibiotics was started. Additionally, repeat COVID-19 polymerase chain reaction performed on hospital day 5 was positive and patient was also treated with remdesivir and dexamethasone. However, patient continued to be symptomatic despite being on antibiotics and a follow up echocardiogram showed a persistent 1.0 cm tricuspid vegetation (not shown).

Computed tomography angiogram of the chest performed on hospital day 20 demonstrated numerous cavitary and noncavitary pulmonary nodules and masses throughout the bilateral lungs (Fig. 2), compatible with septic emboli. In addition, multifocal aneurysmal dilatations of the bilateral lobar, segmental, and subsegmental pulmonary arteries were noted (Figs. 3A and B), compatible with mycotic aneurysms of the pulmonary arteries. Multiple eccentric filling defects were noted in the aneurysmal portions of the right lower lobar pulmonary artery and left lower lobe segmental pulmonary arteries (Figs. 4A and B), which were attributed to septic or bland pulmonary emboli. Bilateral, left greater than right pleural effusions were also noted (Fig. 2, Figs. 4A and B), which were atypical for COVID-19. No commonly reported finding of COVID-19 pneumonia was present on CT.

The patient subsequently underwent successful open surgical bioprosthetic tricuspid valve replacement. The valve vegetation culture was positive for MSSA. The right pleural effusion (shown on Figs. 2 and 4) was found to be a loculated empyema intraoperatively and was also positive for MSSA. Conservative approach for mycotic pulmonary artery aneurysms was pursued, and the patient was placed on a 6-week course of intravenous antibiotics postoperatively.

Discussion

Mycotic aneurysms arising from pulmonary arteries are rare infectious dilatations of the pulmonary artery vessel walls. Mechanisms of development include (1) intraluminal septic thromboembolus from systemic hematogenous infection that subsequently extends into vessel wall, (2) direct extension of a pulmonary infectious focus into an adjacent pulmonary artery, or (3) direct hematogenous seeding of the vasa vasorum by infectious microemboli. All of these result in a common final pathway of progressive arterial wall weakening and dilatation [1].

Staphylococcus and *Streptococcus* species are the most common causative organisms of mycotic pulmonary artery aneurysm. Less common organisms include *Mycobacterium tuberculosis* or fungal organisms such as Candida, Aspergillosis, Mucor [2]. Risk factors include IVDU, infective endocarditis, or congenital cardiac disease. Endocarditis is thought to be the primary predisposing factor [4]. Our patient had the risk factors of IVDU and *Staph Aureus* endocarditis involving the tricuspid valve with aneurysmal dilatation of the pulmonary artery walls likely due to extension of pulmonary septic thromboemboli from tricuspid valve vegetations into pulmonary artery vessel walls.

Clinical manifestations usually reflect underlying endocarditis and pulmonary septic emboli. However, direct clinical signs of mycotic pulmonary artery aneurysms are often occult in absence of serious complication such as aneurysmal rupture, which would manifest as massive hemoptysis [2]. Our patient did not have any direct clinical signs of mycotic pulmonary artery aneurysm throughout his hospital course. Thus, it is important to consider mycotic aneurysms in differential diagnosis of patients with the appropriate risk factors and appropriate image findings in order to enable rapid diagnostic workup, especially given the potential catastrophic complication of aneurysm rupture.

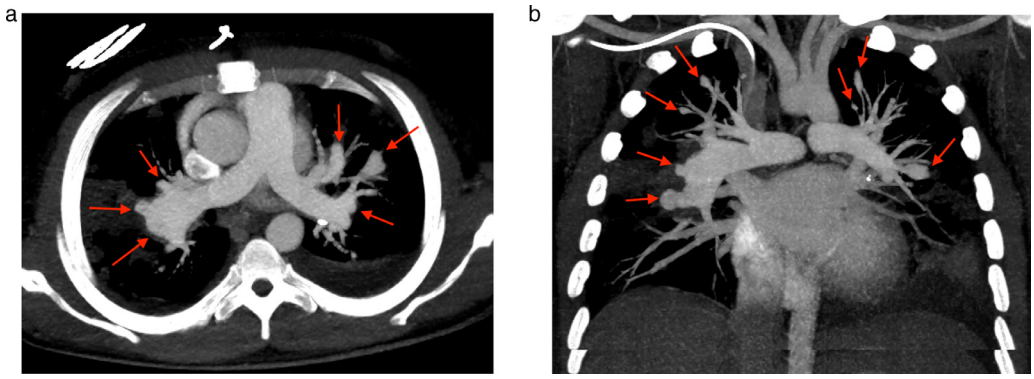

Fig. 3 – A and B: Axial (3A) and coronal (3B) maximal intensity projections (MIP) of CT angiogram of the chest demonstrated multifocal aneurysmal dilatations of the bilateral lobar, segmental, and subsegmental pulmonary arteries (red arrows).

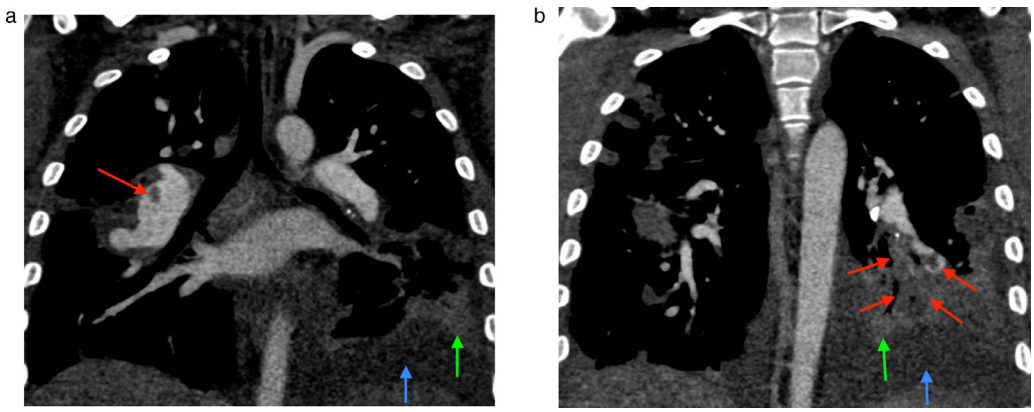

Fig. 4 – A and B: Coronal CT angiogram of the chest demonstrated multiple eccentric filling defects within the aneurysmal portions of the right lower lobar pulmonary artery (red arrow, 4A) and left lower lobe segmental pulmonary arteries (red arrows, 4B), left pleural effusion (blue arrows) and subjacent atelectasis of the left lower lobe (green arrows).

Computed tomography angiogram is the most reliable imaging modality for diagnosis and most often demonstrates one or more aneurysmal dilatations of the pulmonary arteries. Given these findings, an important imaging differential consideration is Rasmussen aneurysm, a pulmonary artery aneurysm adjacent to or within a tuberculous pulmonary cavity [3]. Mycotic and Rasmussen aneurysms share similar risk factors and can have indistinguishable clinical and image findings. Thus, clinical context such as established infective bacterial endocarditis and direct microbiology evidence are important factors in differentiating between the 2 entities.

Given the high mortality rate associated with pulmonary artery mycotic aneurysms due to high risk of rupture, treatment options tend to favor endovascular embolization or various surgical interventions such as lobectomy, pneumonectomy, or aneurysmectomy [4]. Successful conservative treatments with intravenous antibiotics alone have also been documented in selected patients, usually when the size of aneurysm is small and stable, there is no clinical evidence of aneurysm rupture or when the patient is not a candidate for endovascular or surgical interventions [5,6]. Our patient was treated with conservative management given the more urgent need for surgical repair of the infected and perforated tricuspid valve, a high-risk procedure that would increase the risk of any subsequent aggressive intervention for mycotic pulmonary artery aneurysm. In summary, our case highlights a rare complication of multiple bilateral mycotic pulmonary artery aneurysms with septic emboli in a patient with tricuspid valve vegetation and infective endocarditis.

Patient Consent Statement

Patient consent was not needed for the present case report as no patient identifiers are present, it is not a rare diagnosis and all images have been deidentified.

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