Pulmonary Mass in an Immunocompromised Patient: Think Outside the Box

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Introduction

The differential diagnosis of pulmonary masses and nodules is wide, especially in immunocompromised patients, who are prone to various infectious and non-infectious complications. In these patients the diagnosis of pulmonary masses and nodules should proceed systematically and rare conditions should be kept in mind.

Case Summary

A 41-year-old man with a history of acquired immune deficiency syndrome (last CD4 count was 40 cells/mL) presented to the emergency department complaining of sore throat for 2 weeks, which was worse with swallowing. He denied fever, dyspnea, cough, chest pain, hemoptysis, or epistaxis. Initial evaluation found tachypnea and a peripheral oxygen saturation of 90% on room air. Auscultation revealed normal breathing sounds and no additional sounds. There were no skin or mucus-membranes lesions.

He had a history of vitiligo, and his surgical history included repair of an inguinal hernia and removal of a kidney stone. He was on highly active retroviral therapy, dapsone, and azithromycin. He was a smoker and his family history was positive for lung cancer and coronary artery disease.

Chest radiograph showed a left-upper-lobe mass (Fig. 1). Arterial blood gas analysis showed a pH of 7.43, a P_{aCO_2} of 35.5 mm Hg, a P_{aO_2} of 56 mm Hg, and an oxygen saturation of 86%. Contrast-enhanced chest computed tomography (CT) showed an enhancing left-upper-lobe mass with feeding pulmonary blood vessels,

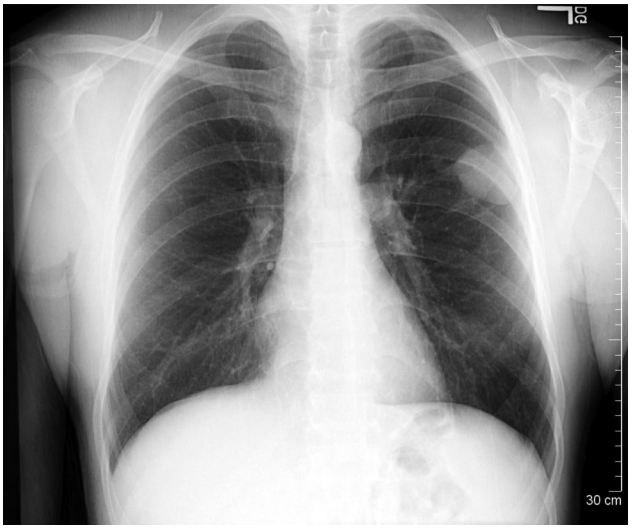

Fig. 1. Chest radiograph shows a left-upper-lobe pulmonary mass

which was diagnostic of a pulmonary arteriovenous malformation (PAVM) (Fig. 2). His hypoxia was not improving with supplemental oxygen. An arterial blood sample taken while he was breathing 100% oxygen showed a pH of 7.39, a P_{aCO_2} of 38 mm Hg, a P_{aO_2} of 86 mm Hg, and an oxygen saturation of 95%. The shunt fraction was calculated at 27%.

Given his symptoms, hypoxemia, and the size of the PAVM, he underwent pulmonary angiography, which confirmed a large PAVM with 2 feeding arteries, which were successfully embolized (Fig. 3). His symptoms improved and he was discharged home. A few weeks after the embolization, chest radiograph showed resolution of the left-upper-lobe mass (Fig. 4), and his arterial blood gas values on 100% oxygen were: pH 7.43, P_{aCO_2} 37 mm Hg, P_{aO_2} 367 mm Hg, and oxygen saturation 97%.

Discussion

Solitary pulmonary nodules ($<$ 3 cm in diameter) and masses ($\geq$ 3 cm in diameter) are common in immunocompromised patients and are caused by various infectious and

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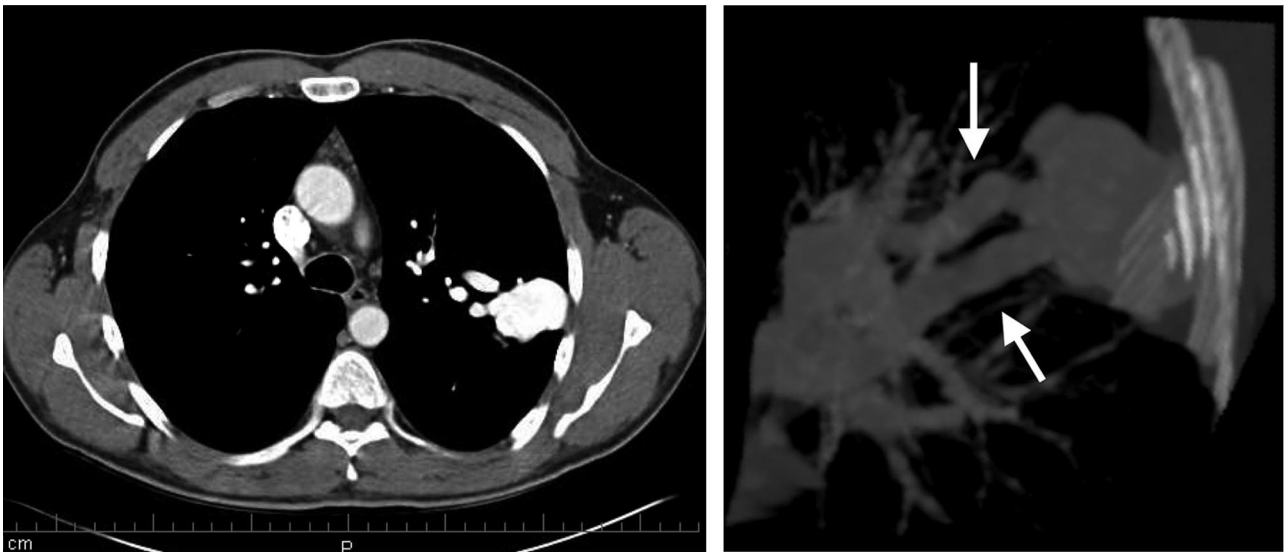

Fig. 2. Left: Contrast computed tomogram shows an intensely enhanced left-upper-lobe mass. There are at least 2 dilated pulmonary vessels close to the mass. Right: Reconstructed image from the computed tomogram, with maximum intensity projection, shows the pulmonary arteriovenous malformation with the feeding and draining pulmonary vessels (arrows).

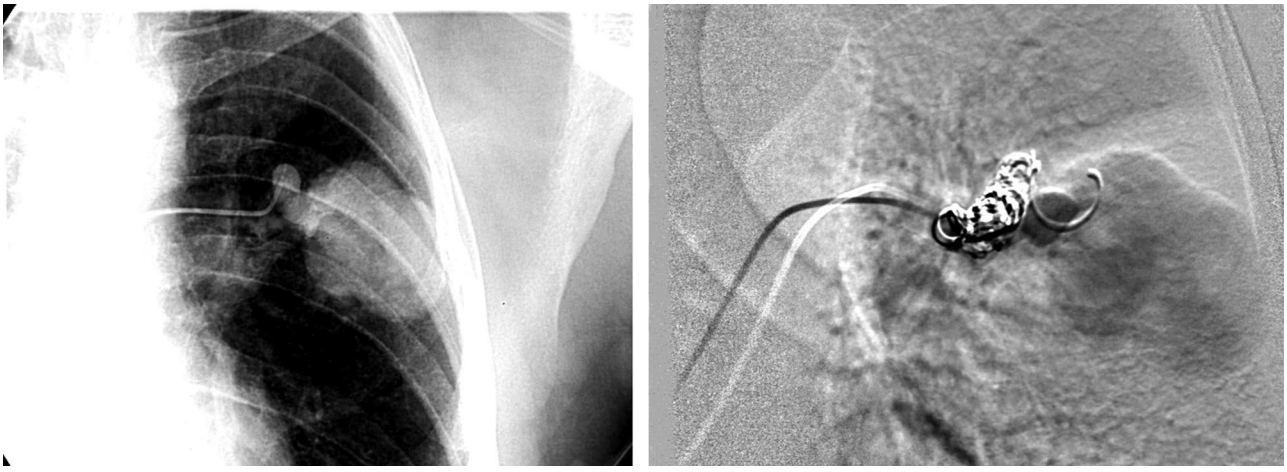

Fig. 3. Left: Pulmonary angiography shows the enhanced left-upper-lobe mass with dilated feeding pulmonary artery, consistent with pulmonary arteriovenous malformation. Right: Obliteration of the pulmonary arteriovenous malformation after multiple coil embolizations.

noninfectious conditions. The most common infectious causes include fungal infections, such as aspergillus, histoplasma, and coccidiomyces. Mycobacterial infections are also important. Bacterial infections may rarely present as round pneumonia. *Pneumocystis jiroveci* has rarely been reported as the cause of a solitary pulmonary mass or nodule.[1] Pulmonary masses in immunocompromised patients may also be caused by noninfectious conditions such as primary lung cancer, lymphoma, metastatic disease, and connective-tissue diseases such as Wegener granulomatosis and rheumatoid nodules. Other conditions, not related to the immunosuppression, such as nodular sarcoidosis and PAVM, should be considered.

In these patients, diagnosis of a pulmonary mass or nodule detected on chest radiograph should follow a systematic approach. Medical history (eg, dyspnea, hemoptysis, fever, smoking, travel, and medications) is important. Severe hypoxemia that is out of proportion to the radiology findings should raise the possibility of PAVM. It is also necessary to obtain old radiographs for comparison. Almost all patients will need a CT, with thin sections, to better characterize the lesion. Features such as number, size, edges, and content are important in differentiating malignant and nonmalignant lesions.[2] The presence of feeding and draining blood vessels and the timing and the intensity of enhancement are diagnostic of PAVM. The

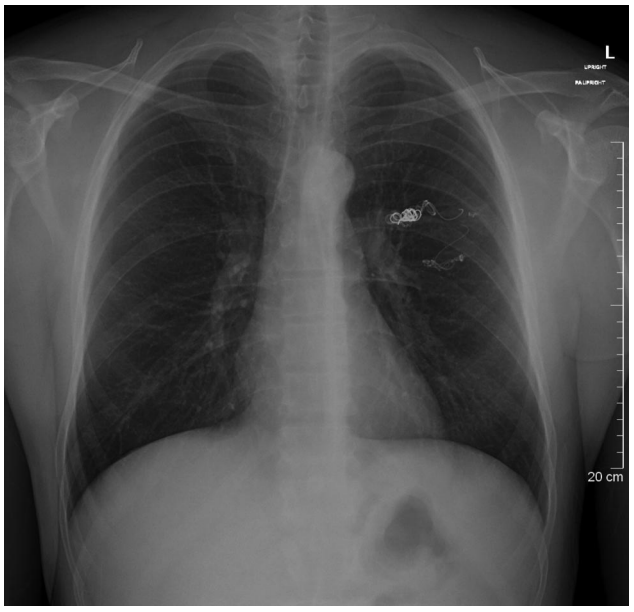

Fig. 4. Chest radiograph following embolization of the pulmonary arteriovenous malformation shows multiple coils and almost complete disappearance of the left-upper-lobe mass.

PAVM diagnosis can also be confirmed by 3-dimensional reconstruction of noncontrast helical chest CT images of the mass.[3] PAVM has to be considered and excluded before attempting tissue diagnosis of the pulmonary mass.

A PAVM is an anomalous communication between pulmonary veins and arteries. PAVMs are commonly seen in patients with hereditary hemorrhagic telangiectasia (Osler-Weber-Rendu syndrome).[4] The diagnosis of hereditary hemorrhagic telangiectasia can be made if the patient has at least three of the following: recurrent epistaxis, telangiectasias, internal arteriovenous malformations, and family history of hereditary hemorrhagic telangiectasia. However PAVM could be an isolated finding or secondary to trauma, mitral stenosis, schistosomiasis, actinomycosis, tuberculosis, bronchiectasis, cardiac surgery, or underlying liver disease as part of the hepatopulmonary syndrome.[5] The PAVM in our patient was idiopathic; we excluded other potential causes such as hereditary hemorrhagic telangiectasia and infections.

PAVM should be suspected in patients who have pulmonary opacities on chest radiograph and dyspnea, hypoxemia, clubbing, recurrent epistaxis, or telangiectasias of the skin or mucus membranes. Symptoms related to PAVM are more likely when the PAVMs are multiple or > 2 cm in diameter.[3]

The degree to which the PAVM contributes to a shunting effect can be estimated with the 100%-oxygen method,[6] which is performed by obtaining and analyzing an arterial blood gases sample while the patient is breathing room air, then placing the patient on supplemental oxygen (via non-rebreather oxygen mask) for 15–20 min, then obtaining and analyzing another arterial blood gases sample. Normally the shunt fraction is around 5%, and a shunt fraction more than that is considered abnormal. In our patient the baseline shunt fraction was 27%. Other tests that confirm the presence of right-to-left pulmonary shunt include contrast echocardiography[7] and radionuclide perfusion lung scanning, the latter of which is more accurate than the 100%-oxygen method but not widely available.[8]

Indications for correction of a PAVM include symptomatic hypoxemia, paradoxical embolization, progressive enlargement of the PAVM, and feeding vessels ≥ 3 mm in diameter.[3] Embolization via pulmonary angiography has replaced surgery as the preferred method for correcting PAVM.[3]

Pulmonary masses and nodules are caused by various infectious and noninfectious conditions, and in the immunocompromised patient these lesions are most commonly related to the underlying disease, but may rarely be unrelated. Clinicians should keep this possibility in mind when evaluating these patients. PAVM should be considered in the differential diagnosis of pulmonary nodules or masses. Computed tomography, with thin sections, is usually diagnostic.

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