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Case Report

Pulmonary air embolism associated with proximal bland thrombus

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ABSTRACT

We report a case of pulmonary air embolism associated with proximal bland thrombus seen on computed tomography pulmonary angiography in a 49-year-old man, who developed acute hypoxia following removal of central venous catheter.

Introduction

Pulmonary air embolism (PAE) is a known, albeit infrequent complication of central venous catheterization. Intraluminal air may lead to pulmonary arterial blood flow obstruction which, in theory, increases the risk of pulmonary arterial thrombosis. Here we report a case of pulmonary air embolism associated with proximal bland thrombus, following removal of central venous catheter. To our knowledge, this is the first report of pulmonary air embolism associated with a thrombus. We hypothesize that the pulmonary thrombus has resulted from blood flow obstruction secondary to air embolism.

Case presentation

A 49-year-old man, admitted for investigation of idiopathic rhomboencephalitis, developed acute hypoxia and hypotension following removal of an internal jugular venous catheter. A stat portable chest radiograph demonstrated no significant abnormality. Gas in the right ventricle was seen on cardiac echography performed at the bedside. Computed tomography (CT) pulmonary angiogram performed shortly thereafter, showed a right lower lobar pulmonary artery thrombus containing central gas (Fig. 1). Intra-arterial gas was also present in multiple distal segmental and subsegmental pulmonary

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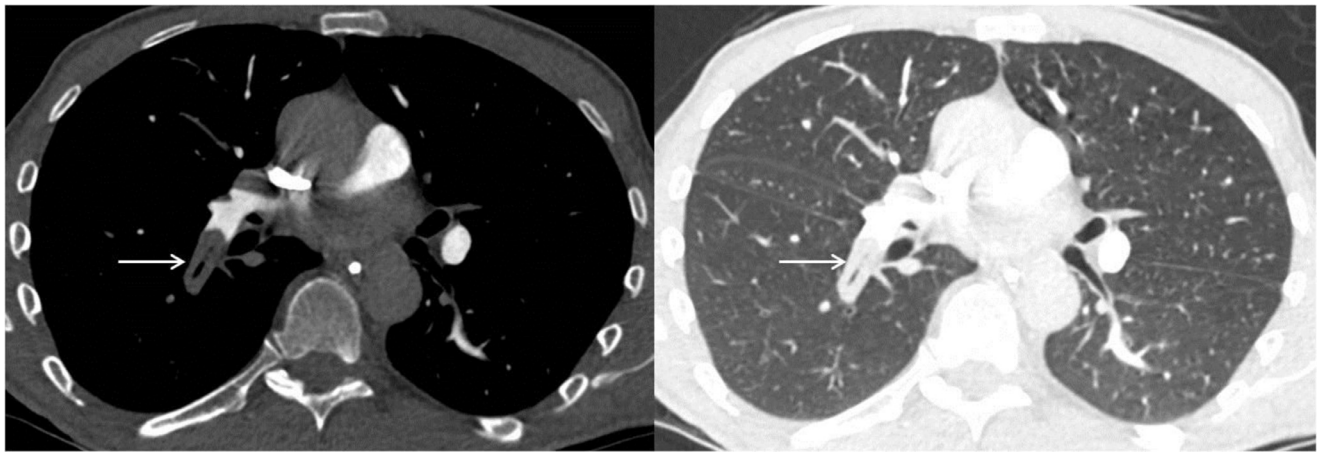

Fig. 1 – CT pulmonary angiogram in mediastinal and lung windows demonstrates a filling defect in the right lower lobar pulmonary artery with central gas (arrows).

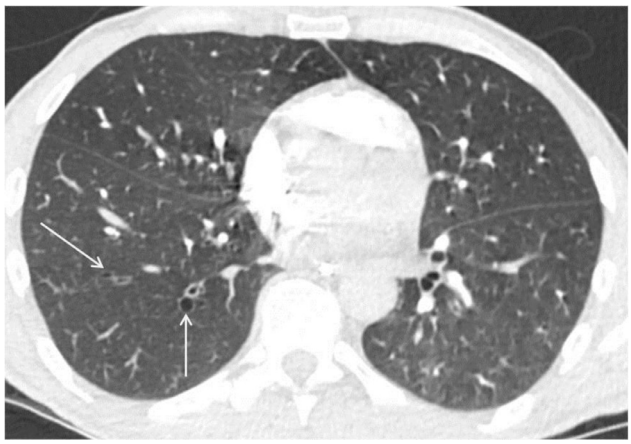

Fig. 2 – CT pulmonary angiogram in lung window showing the "double bronchus sign" due to the gas in the segmental and subsegmental pulmonary branches (arrows) adjacent to air-filled bronchi.

arteries of the right lower lobe, giving rise to the so-called "double bronchus" sign [1] (Fig. 2).

The patient was started on anticoagulation therapy, and to our knowledge had no further symptoms related to pulmonary embolism.

Discussion

PAE is a rare complication of central venous catheterization, associated with estimated mortality rate of about 20% [2,3]. The extent of morbidity resulting from PAE depends on the amount and propagation speed of gas introduced into the venous circulation, underlying host cardio-pulmonary reserve and cranio-caudal position of the right ventricular outflow tract at the time of air embolization [4–6]. Although, there is no consensus about the amount of intravascular air considered fatal, it has been proposed that a rate of air entry of 100 mL/s for 2 seconds, or more than 200 to 300 mL of intravascular air could result in death [7,8].

Air embolism may occur when there exists a connection between vascular system and ambient air, such as the transient tract created by removal of a central venous line. Additionally, a pressure gradient is needed to drive ambient air into the vascular system. Such a gradient occurs naturally, as the central venous pressure is lower than the atmospheric pressure in 40% of patients [9]. The gradient between external atmospheric pressure and intravascular venous pressure can be further increased by hypovolemia or by inspiration, the latter as a result of negative intrathoracic pressure. Thus, hypovolemia and inspiration during central venous catheter removal are two factors that increase the risk of air embolism [3].

Clinical manifestations of PAE may include acute tachypnea, hypotension, hypoxia, wheezing, characteristic precordial sucking sound on osculation, cardiac arrest, and death [10]. In the presence of a pulmonary-systemic shunt, PAE may result in neurological symptoms [11].

Venous air embolism may lead to right ventricle outflow tract obstruction, blocking blood flow to the pulmonary arteries which, in turn may lead to vasoconstriction due to hypoxia and acute pulmonary hypertension.

If intravascular air passes into the distal pulmonary circulation, it may lead to vascular obstruction [4], which increases the risk of thrombus formation due to blood flow stasis. However, to our knowledge, this complication has not been previously described in English language medical literature.

Conventional chest radiographic findings of PAE may include intra-cardiac air, air in the main pulmonary artery, focal oligemia, enlarged pulmonary arteries, hepatic venous air, or pulmonary edema [11]. Oppendeimer et al found that the majority of PAE direct radiographic manifestations disappear within 15 minutes of acute event [12].

On computed tomography, the most frequent finding of PAE is intraluminal air in the pulmonary arterial trunk and/or

right cardiac chambers. Other findings include dilatation of the pulmonary arteries and pulmonary edema.

To our knowledge, there is only one published report of intraluminal gas in the peripheral pulmonary arteries, described as "double bronchus sign" by Moser et al [1].

Ours is the first report of PAE with associated bland thrombus. We believe that our case gives credence to the idea that blood flow stasis secondary to PAE can lead to pulmonary arterial thrombosis. Therefore, we suggest that patients with acute thoracic symptoms following manipulation of central venous lines be assessed with CT pulmonary angiography.

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