

Haemodynamic changes due to systemic arterial shunts in a destroyed lung mimicking pulmonary thromboembolism on CT

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DESCRIPTION

A 51-year-old woman presented to our department with multiple episodes of massive haemoptysis. She had a history of pulmonary tuberculosis. High-resolution CT scan showed a completely destroyed right lung with fibrocavitary changes (figure 1). CT pulmonary and bronchial angiography was performed, which showed non-enhancement of the right pulmonary artery in the pulmonary angiographic phase, leading to suspicion of a pulmonary arterial embolus (figure 2). The bronchial angiographic phase showed brilliant enhancement of the right pulmonary artery and its branches through reversed filling via multiple systemic collaterals arising from the hypertrophied right bronchial, right intercostal and right inferior phrenic arteries (figure 3).

In patients with chronic pulmonary parenchymal inflammation, such as bronchiectasis, pulmonary



Figure 2 Axial CT image in pulmonary angiographic phase showing non-opacification of the right pulmonary artery (black arrow), simulating pulmonary thromboembolism.



Figure 1 Coronal high-resolution CT (HRCT) image showing complete destruction of right lung parenchyma with fibrocavitary and bronchiectatic changes (black arrows).

tuberculosis and chronic obstructive pulmonary disease, pre-existing connections between the bronchial and pulmonary arteries become functional at precapillary and postcapillary levels, often creating a source of haemoptysis.¹

Cases with systemic-pulmonary artery shunts have demonstrated a sharp reduction of pulmonary arterial flow on the affected side with retrograde filling of the pulmonary artery from the systemic circulation. This can be explained by the pressure gradient between systemic and pulmonary circulations that leads to physiological cut-off of the pulmonary arterial flow on the affected side.²

This phenomenon was responsible for non-opacification of the affected pulmonary artery in the pulmonary angiogram phase simulating thromboembolism in our case; however, the brilliant opacification in systemic arterial phase demonstrated patency of the pulmonary artery.

Our case demonstrates the importance of including a systemic arterial phase in cases having severe lung destruction, to prevent the misdiagnosis of pulmonary thromboembolism.



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Figure 3 (A) Axial CT image in bronchial (systemic) angiographic phase showing hyperenhancement of right pulmonary artery (solid white arrow). (B–D) Maximum intensity projection (MIP) images in coronal plane showing hypertrophied and tortuous right bronchial arteries (white arrowheads in (B)), right posterior intercostal arteries (black arrows in (C)) and right inferior phrenic artery arising from right renal artery (dotted white arrow in (D)) forming shunts with the right pulmonary artery.

Learning points

- ▶ Conditions causing extensive pulmonary inflammation and destruction can lead to development of systemic-pulmonary shunting.
- ▶ These systemic-pulmonary shunts can lead to changes in the pressure gradients of the pulmonary circulation and alteration of haemodynamics.
- ▶ Adding a systemic angiographic phase in addition to a pulmonary angiographic phase prevents misdiagnosis of pulmonary thromboembolism in such patients.

Competing interests None declared.

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