Retrograde systemic to pulmonary shunt simulating a pulmonary embolism

A. Lacout\textsuperscript{a,*}, M. El Hajjam\textsuperscript{b}, A. Khalil\textsuperscript{c}, P. Lacombe\textsuperscript{b}, P.-Y. Marcy\textsuperscript{d}

\textsuperscript{a} Medical Imaging Centre, 47, boulevard du Pont-Rouge, 15000 Aurillac, France
\textsuperscript{b} Radiology Department, Hôpital Ambroise-Paré (AP—HP), 9, avenue Charles-de-Gaulle, 92100 Boulogne-Billancourt, France
\textsuperscript{c} Radiology Department, Hôpital Tenon, AP—HP, 4, rue de la Chine, 75020 Paris, France
\textsuperscript{d} Head and Neck and Interventional Radiology Department, Antoine Lacassagne Cancer Research Institute, 33, avenue Valombrose, 06189 Nice cedex 1, France

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The existence of a systemic to pulmonary shunt should be taken into consideration in patients with suspected pulmonary embolism, as it can be a source of false positives on the angiography scan. This concerns false pulmonary arterial defects caused by the blood flow coming from the bronchial systemic arteries and contaminating the pulmonary arterial circulation. The knowledge of thoracic vascular duality, of its physiological and pathological connections as well as the performance of a thoracic angiography scan during the aortic phase make it possible to get rid of these artefacts and avoid inappropriate anticoagulation.

**Case report**

We report the case of a 30-year-old female patient of African origin admitted to the emergency room for dyspnoea and haemoptysis, which was suggestive of the diagnosis of pulmonary embolism. Her medical history was marked by pulmonary tuberculosis treated 10 years earlier. Upon admission, the patient was apyretic with an SaO\textsubscript{2} of 96\% in surrounding air. An HIV serology was positive with CD4 levels of more than 500/mm\textsuperscript{3}. Chest radiography showed sequellae retractile lesions of the upper left lobe (Fig. 1). The angiography scan was carried out on a 16 slice multidetector computed tomographic scanner (CT-scan) at a pulmonary arterial time (injection of 60 ml of lobitridol 300 mg I/ml (Xenetix, Guerbet — France) at a flow rate of 3.5 ml/s, with automatic start of acquisition after detection of a bolus by a region of interest (ROI) placed in the trunk of the pulmonary artery). The examination showed a filling defect in the trunk of the pulmonary artery and the absence of opacification of the left pulmonary artery, suggesting a massive pulmonary embolism (Fig. 2). The parenchymatous analysis showed paracatricial bronchiectasis, a large excavation of the upper right lobe filled in with dependent material and alveolar bleeding in the lower left lobe (Fig. 3).

\textsuperscript{*} Corresponding author.
E-mail address: lacout.alexis@wanadoo.fr (A. Lacout).

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Due to the positivity of the aspergillus serology and the negativity of the direct BKs, the diagnosis of chronic cavitary aspergillosis in addition to pulmonary sequela was retained, explaining the haemoptysis. The problem of a proximal "embolism" of the pulmonary arterial trunk was thus raised as well as the place of anticoagulant treatment in the context of haemoptysis.

The infectious context and the radio-clinical discrepancy between a pulmonary embolism that appeared massive and the absence of clinical signs made it possible to question this diagnosis and to retain the diagnosis of a retrograde systemic to pulmonary shunt.

In order to confirm this diagnosis, and before administering any treatment, a control CT-scan was carried out twelve hours after the first one, this time during the aortic phase (injection of 60 ml of lobitrodil 300 mg/l/ml [Xenetix, Guerbet - France] at a flow rate of 3.5 ml/s, with automatic start of acquisition after detection of a bolus by an ROI placed at the aortal arch).

This examination made it possible to definitively eliminate the diagnosis of embolism due to the disappearance of the filling defect in the pulmonary artery (Fig. 4). It can therefore be deduced that on the initial CT-scan carried out during the pure pulmonary arterial phase, the defect was a non-opacified aortic flow through the systemic arteries and breaking through to the pulmonary arterial trunk.

The final diagnosis was that of haemoptysis secondary to chronic cavitary aspergillosis in addition to sequelae of tuberculosis. The left lung was of the systemic type, as it was vascularized exclusively by the systemic arteries via a retrograde systemic to pulmonary shunt. This shunt was caused by the bronchial systemic hypervascularization secondary to the destruction of the pulmonary functional unit by the left upper lobe sequelae of tuberculosis and the aspergillosis infection (Fig. 5).

**Discussion**

The multi-detector CT-scan is the reference examination for the diagnosis of pulmonary embolism [1]. The multiplicity of examinations in this indication raises the possibility of false positives for pulmonary embolism [2]. In patients with extensive infectious or inflammatory pulmonary disease, the low pressure functional pulmonary vascularization is gradually destroyed. It is automatically replaced by a nourishing circulation of high pressure aortal origin in the form of systemic neovascularization of bronchial and nonbronchial origin. The pathological pulmonary parenchyma...
recruits the bronchial arteries. It also recruits the systemic arteries of the thoracic wall as soon as a pleural symphysis is created. This is a major risk factor for haemoptysis. At the same time, the anastomoses present under normal conditions between the bronchial systemic arteries and the pulmonary arteries become hypertrophied, through which the aortal blood flow (at high pressure, at a mean of 120 mmHg) erupts into the pulmonary arteries (at low pressure, at a mean of 20 mmHg). These anastomoses create a systemic to pulmonary shunt that can be anterograde or retrograde depending on the level of obstruction of the pulmonary artery bed (Fig. 6) [3,4]. At the pulmonary arterial phase on the angiography CT-scan, the arrival of a bronchial systemic blood flow of aortic origin, not yet opacified, into the pulmonary artery via the shunt creates a flux effect and can be confused with a pulmonary embolism [5]. To avoid this diagnostic pitfall that could lead to inappropriate anticoagulation, particularly in a context of haemoptysis, the acquisition should be carried out at a phase of vascular equilibrium that allows for homogeneous opacification of all the mediastinal vascular structures (pulmonary arteries, aorta and its branches, heart, coronary arteries, etc.) for example, during a ”triple rule out angiography CT-scan” [6].

**Figure 3.** Initial thoracic angiography CT-scan, carried out at the pulmonary arterial phase (pulmonary window). Axial cut (a) and coronal reconstruction (b): excavated cavity of the upper right lobe, with heterogeneous dependent material (arrow) related to aspergillosis. Ground glass aspect in the left lower lobe secondary to haemoptysis.

**Figure 4.** Control thoracic angiography CT-scan carried out at the aortal phase (mediastinal window). Axial cut (a) and oblique sagittal reconstruction (b): same cut level as images 2a and 2b for perfect understanding: disappearance of the defect of the pulmonary arterial trunk.

Figure 5. Control thoracic angiography CT-scan carried out at the aortal phase (mediastinal window). Axial MIP (a) and coronal MIP (b): neovascularization coming from the lateral and medial thoracic arteries and the intercostal arteries entering directly into the trunk of the pulmonary artery (arrows). The blood circulates from these arteries (at high pressure) towards the left pulmonary artery (at low pressure) creating a retrograde systemic to pulmonary shunt.
Conclusion

Bronchial systemic hypervascularization following a chronic pulmonary inflammatory or infectious process could cause a systemic to pulmonary shunt, which can be a cause of a wrong diagnosis of pulmonary embolism. The knowledge of the intricacies of the pulmonary nourishing and functional circulation is absolutely necessary to the understanding of the balance between the two systems and any abnormalities that are observed.

It is classic to say that an angiography CT-scan to look for pulmonary embolism is technically optimal when only the pulmonary arteries and the right cavities are opacified. However, this pure pulmonary arterial phase can be a source of diagnostic pitfalls. Therefore, the angiography scan carried out at the aortic phase seems to us to be an excellent compromise that allows for an optimal analysis of all of the thoracic vascular structures.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

References

