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EC PULMONOLOGY AND RESPIRATORY MEDICINE

Case Report

Aorto-Pulmonary Shunt as a Complicating of Post-Tuberculosis Bronchiectasis and Simulating a Pulmonary Embolism in an African Pneumological Practice

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Received: August 06, 2020; **Published:** August 26, 2020

Abstract

Bronchiectasis is a common sequelae of pulmonary tuberculosis. It is associated with a parenchymal neovascularization and enlargement of bronchial-systemic anastomoses which can create a systemic-pulmonary shunt. Systemic blood flow into the pulmonary arteries can mimic pulmonary embolism on CT angiography. We report a case of aorto-pulmonary shunt complicating post-tuberculous bronchiectasis simulating pulmonary embolism.

Keywords: *Bronchiectasis; Sequelae of Tuberculosis; Systemic-Pulmonary Shunt; Pulmonary Embolism*

Abbreviation

PTB: Pulmonary Tuberculosis; PE: pulmonary embolism

Introduction

Pulmonary tuberculosis (PTB) remains a major public health problem in developing countries [1]. The outcome of the disease with or without treatment can lead to parenchymal sequelaes including bronchiectasis. Post-inflammatory or post-infectious bronchiectasis is followed by a progressive destruction of the functional pulmonary vascularization. This phenomenon is accompanied by systemic neovascularization and an enlargement of bronchial-systemic anastomoses which can create a systemic-pulmonary shunt [2, 3]. Systemic blood flow into the pulmonary arteries can mimic pulmonary embolism on CT angiography [3]. We report a case of an aorto-pulmonary shunt as a complication of post-tuberculosis bronchiectasis simulating pulmonary embolism. The relevance of this case report lies in the rarity of the case in the literature as well as in the input of the thoracic aortic CT angiography in differentiating pulmonary embolism in patients with sequelae of tuberculosis in an African pulmonology practice.

Case Report

A 49-year-old patient with a known medical history of diffuse, bilateral post-tuberculosis bronchiectasis leading to chronic respiratory failure was admitted to the emergency department for acute chest pain, dyspnea and abundant hemoptysis. On her admission, the physi-

cal examination noted a fever at 38.2 ° C, a polypnea at 28 cycles/ min with oxygen saturation at 88%. Biological testing noted a D-dimers level at 2,500 ng/l. The thoracic CT angiography showed a lacunar image in the left lower lobar artery suggesting a pulmonary embolism (Fig.1). Parenchymal analysis showed foci of multiple bilateral diffuse predominantly cystic bronchiectasis, located in the right upper lobe and left lower lobe with signs of bronchial infection. It also showed parenchymal systematized condensation in the lower left lobe (Fig.2). Cytobacteriological examination of the sputum identified a multi-resistant alpha hemolytic streptococcus, sensitive to macrolides and aminoglycosides. The acid- fast bacilli test, the mycobacterial culture and the GeneXpert in sputum were all negative as well as the aspergillus serology. The diagnosis then was a bacterial infection of bronchiectasis due to alpha hemolytic streptococcus complicated by a pulmonary embolism. Antibiotic therapy based on azithromycin and gentamicin was administered, combined with anti-coagulant treatment based on enoxaparin at a curative dose followed by acenocoumarol. The problem that arose then was the persistence of hemoptysis two weeks after treatment started and thus, a bronchial arterial embolization was indicated. Two weeks after the first thoracic CT angiography, a control one was performed before the arterial embolization and did not show a pulmonary embolism (Fig.3C). This examination ruled out the diagnosis of embolism due to the absence of the lacunar image of the left lower lobar artery. It was then deduced that the gap in the left lower lobar artery seen in the initial thoracic CT angiography corresponded to an unopacified aortic blood flow passing through the systemic arteries, complicating an aorto-pulmonary shunt. It also showed the presence of an anastomosis between the left inferior lobar artery and the descending thoracic aorta with passage of systemic blood flow into the pulmonary circulation(Figure 3D).

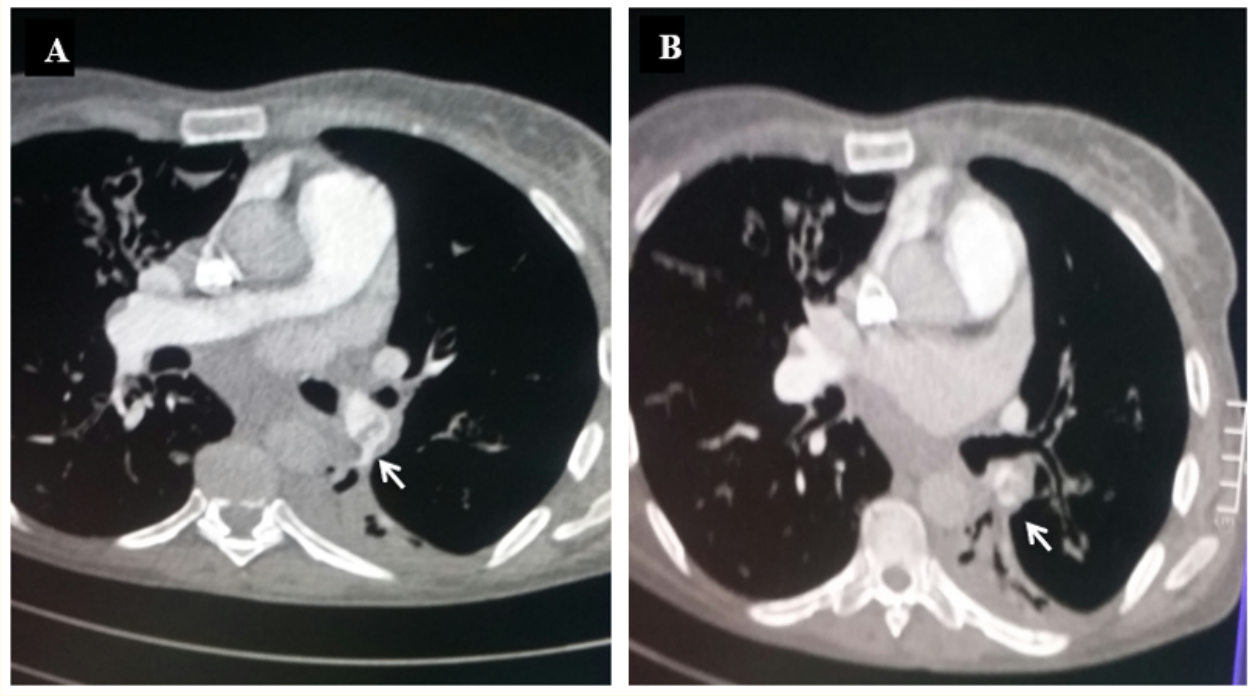

Figure 1: *Initial thoracic CT angiography, performed in the pulmonary artery phase (mediastinal window), axial slice: hypodense gap in the left lower lobe artery (arrow) suggesting pulmonary embolism.*

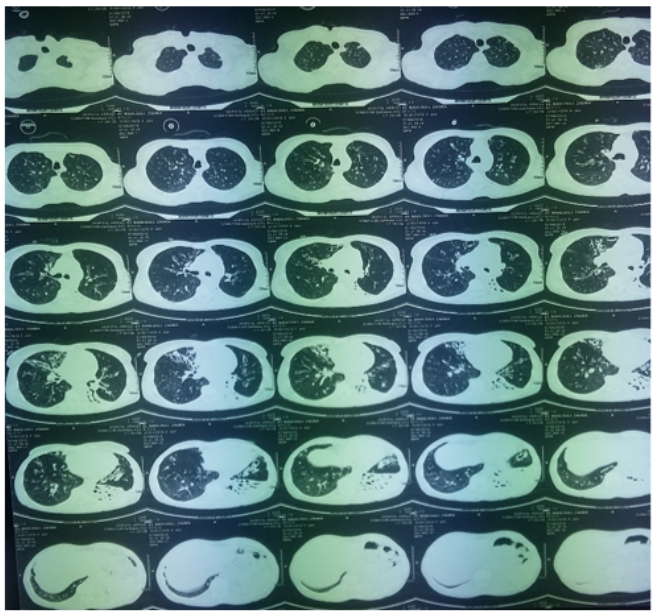

Figure 2: *Initial thoracic CT angiography (axial window), foci of multiple bilateral diffuse cystic and moniliform bronchiectasis, predominantly cystic, predominant in the right upper lobe and left lower lobe with signs of bronchial superinfection, focus of systematized lower lobar parenchymal condensation left.*

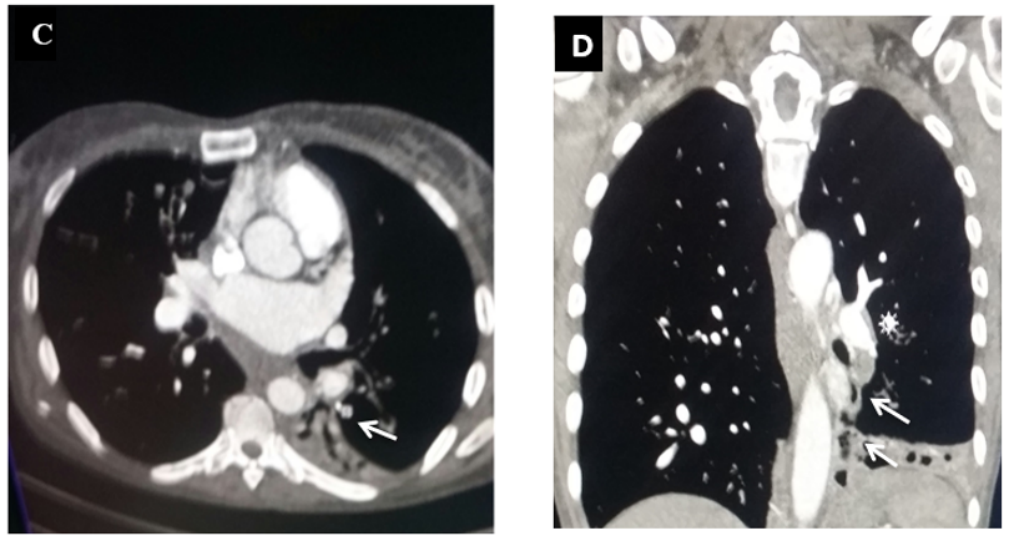

Figure 3: *Control thoracic CT angiography, performed in the aortic phase and the pulmonary artery phase (mediastinal window), axial slice (C), same section plane as figure 1B for a perfect comparison: disappearance of the gap in the left lower lobe artery (arrow). Coronal slice (D): aorto-pulmonary anastomosis with passage of systemic blood flow into the pulmonary circulation (arrow).*

Discussion

PTB contributes significantly to the global health burden particularly in developing countries. This is not only due to the morbidity and mortality associated with active disease, but also the morbidity associated with sequelae [1, 4]. Post-tuberculosis bronchiectasis is a frequent complication of pulmonary tuberculosis. Deshpande SS and al. reported a 77% frequency of bronchiectasis in former tuberculosis patients with a predominance of cystic forms followed by tubular forms [2]. Hatipoglu A and al found that bronchiectasis was observed in 71% of patients with sequelae of tuberculosis. [5].

During post-tuberculosis bronchiectasis bronchial arteries may be enlarged. Deshpande SS and al. reported dilated bronchial arteries in 3% of patients with sequelae of pulmonary tuberculosis [2]. In patients with extensive infectious or inflammatory pulmonary disease, the functional pulmonary vasculature at low pressure is gradually destroyed. It is automatically supplanted by a high pressure nourishing circulation of aortic origin in the form of systemic neovascularization of bronchial and non-bronchial origin [2, 3]. The enlargement of the bronchial arteries leads to the development of bronchopulmonary and arteriovenous anastomoses, causing a systemic-pulmonary shunt [2, 3]. During the pulmonary artery phase of a CT angiography, a not yet opacified original broncho-systemic blood flow going into the pulmonary artery via the shunt, can lead to confusion with a pulmonary embolism [3, 6]. To avoid the risk of an erroneous diagnosis of pulmonary embolism, which may expose the patient to unwarranted anticoagulant treatment and to the risk of hemorrhage, CT angiography should be performed in a vascular equilibrium phase allowing homogeneous opacification of all mediastinal vascular structures (pulmonary arteries, aortas and its branches) [3.7].

Conclusion

During post-tuberculous sequelae (bronchiectasis or extensive parenchymal destruction), bronchopulmonary and arteriovenous anastomoses inherent in the bronchial neovascularization can be the cause of a systemic-pulmonary shunt. The flow of systemic blood into the pulmonary circulation can lead to the erroneous diagnosis of pulmonary embolism. Therefore, in case of a clinical presentation of pulmonary embolism in patients with sequelae of tuberculosis, the CT angiography should be performed in the aortic phase in order to avoid false image traps of pulmonary embolism.

Conflict of Interest

The authors declare that they have no conflicts of interest in relation to this article.

Author's Contribution

P.P. Koumeka: abcd, S. Ait Batahar : bcd, M. Bougadoum: bc, G.S. Moussounda: bc, M. Bougadoum : bc, S. Ait Batahar: bcd, L. Amro: bcd. a: Design and development of the study; b: Data analysis and interpretation; c: Writing of the article, or critical analysis leading to significant modifications to the intellectual content; d: Final approval of the version submitted after critical review.

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Volume 9 Issue 9 September 2020