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Massive hemoptysis due to Rasmussen aneurysm: detection with helicoidal CT angiography and successful steel coil embolization

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Abstract *Objective:* To present the successful management of two cases of massive hemoptysis related to pulmonary aneurysms in patients with active tuberculosis. *Design and setting:* Retrospective study in the respiratory intensive care unit (ICU) of a university hospital. *Patients:* Between July 1996 and January 2002, 46 cases of hemoptysis related to active tuberculosis needed ICU admission. In two cases, pulmonary aneurysm was the source of bleeding. *Results:* Diagnosis was suspected on enhanced CT scan and confirmed by pulmonary angiograms. Transcatheter occlusion of pulmonary arterial circulation was successful.

Both patients were alive at 1-year follow-up. *Conclusions:* Massive hemoptysis occurring in patients with active tuberculosis could arise from pulmonary aneurysms. In such cases, bronchial artery embolization is ineffective. Before referring those patients for emergency surgery, an alternative strategy using angiographic study and transcatheter occlusion of pulmonary arterial circulation might be of interest.

Keywords Massive hemoptysis · Rasmussen aneurysm · Embolization · Vaso-occlusion of the pulmonary artery

Introduction

In patients with either active or inactive tuberculosis, hemoptysis is a frequent complication. In patients with active tuberculosis, hemoptysis is massive in about 8% of cases, with a mortality of about 5–23% [1, 2]. In the great majority of cases the bleeding source is a bronchial or other systemic artery and its embolization is an effective treatment. In a few cases, the hemoptysis arises from pulmonary aneurysms, needing a specific diagnostic and therapeutic approach.

We report two cases of massive hemoptysis related to Rasmussen aneurysm with short- and long-term favorable outcomes after transcatheter occlusion of the pulmonary arterial circulation.

Patients and methods

From July 1996 to January 2002, 461 patients with massive hemoptysis were referred to the respiratory ICU of Tenon Hospital. Active pulmonary tuberculosis was responsible in 46 patients. In two cases, hemoptysis was related to Rasmussen aneurysms.

Case reports

Case 1

In May 1999 a 42-year-old North African man was admitted with a 2-week history of cough, fever, night sweats and weight loss. On admission, his temperature was 39°C. Clinical examination revealed pneumonia with right lung crackles and right-sided pleural effusion, confirmed on chest X-ray. Initially, metronidazole and cefotaxime were administered for a presumptive diagnosis of bacterial pleuropneumonia.

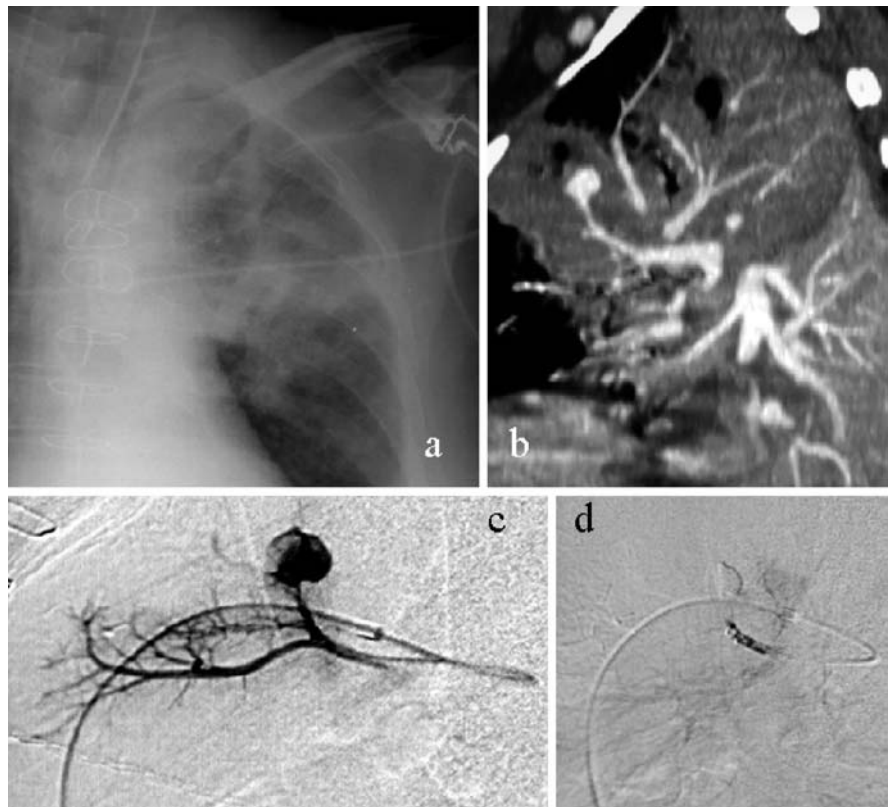
Twenty-four hours later, he experienced a sudden hemoptysis (250 ml red blood) with acute respiratory failure (respiratory rate 32/min, SpO₂ 88% on room air). Intravenous terlipressin was administered and the patient was referred to the ICU. At assessment,



Fig. 1 Enhanced CT. Right lower lobe collapse associated with pleural effusion. (Arrow) Aneurysm of a sub-segmental branch of a Fowler pulmonary artery

the patient had a temperature of 40°C with a pulse rate of 102/min, blood pressure of 160/90 mmHg and SpO₂ was 99% on 91/min of oxygen. Physical examination confirmed right-sided abnormal chest sounds. PaO₂ was 65 mmHg and PaCO₂ 42 mmHg on room air. Chest X-ray and thoracic non-enhanced CT scan showed right lower lobe infiltrates with cavitations and a medium-sized right pleural effusion. At fiberoptic bronchoscopy performed 2 h later,

Fig. 2 a Chest X-ray showing alveolar infiltrates of the left upper lobe with cavitation. **b** Reconstruction of injected CT (Chartres Hospital), which demonstrates the Rasmussen aneurysm very well. **c** Hyper-selective catheterization of a sub-segmental branch of the anterior segmental artery of the left upper lobe. **d** Successful vaso-occlusion with coil



blood clots were found in the entire right bronchial tree with no residual active bleeding. Bronchial arteriography allowed complete embolization of a right basal hypervascularization arising from a common bronchial trunk. Sputum examination revealed acid-fast bacilli (AFB), later identified as multi-sensitive *Mycobacterium tuberculosis*. Antituberculous treatment was begun.

Three days later, recurrence of hemoptysis (30 ml) was noted. Bronchoscopy revealed no active bleeding, but clots in the apical segmental bronchus of the right lower lobe. Thoracic CT scan with intravenous contrast material revealed a contrast-enhanced aneurysm arising from the right segmental pulmonary artery in the apical segment of the right lower lobe (Fig. 1). Pulmonary angiography confirmed the aneurysm and allowed us to perform a vaso-occlusion with two coils. Fifteen days later, the patient was discharged from hospital and after 1 year he was well with no recurrence of hemoptysis. No aneurysm was found at control CT scan.

Case 2

In September 2001, a 62-year-old Indian man was admitted with a 1-month history of weight loss. He had been previously treated for severe heart ischemic disease with left ventricular dysfunction. Chest X-ray revealed a left upper infiltrate with cavitations (Fig. 2a). Sputum smears were positive for AFB, subsequently identified as *Mycobacterium tuberculosis*. Antituberculous treatment was started.

Two weeks later, a 300 ml hemoptysis occurred abruptly, which recurred after a few hours and the patient required mechanical ventilation. A thoracic enhanced CT scan revealed an aneurysm arising from a branch of the left pulmonary artery (Fig. 2b). The patient was transferred to the ICU. On admission, his temperature was 37°C, systolic blood pressure was 70 mmHg and SpO₂ was 60%. Clinical examination showed abolition of the left pulmonary sounds with blood emerging from the tracheal tube. Chest

X-ray revealed an opacity of the whole left lung. On FIO₂ of 100%, PaO₂ was 55 mmHg and PaCO₂ 44 mmHg. Selective intubation failed to improve oxygenation. At fiberoptic bronchoscopy, all clots were removed except those obstructing the left superior bronchus, and saturation subsequently increased. Pulmonary angiography confirmed the presence of an aneurysm (Fig. 2c). Vaso-occlusion with coils was successful (Fig. 2d). The patient was extubated at day 10 and was discharged from hospital 1 month later. At 1-year follow-up, he was well with no recurrence. On control CT scan the aneurysm had disappeared.

Discussion

In patients with massive hemoptysis requiring intensive care, the management usually recommended includes fiberoptic bronchoscopy to assess the site of bleeding and bronchial artery embolization as the first-line treatment. In patients for whom embolization is unfeasible or unsuccessful, emergency surgery is the treatment of choice [3]. However, in the setting of intensive care, postoperative complications were observed in 50% of patients [4] and a fatal outcome occurred in 20% of cases [5], especially when surgery was performed within the first 24 h after hemoptysis.

For the great majority of patients with active tuberculosis and massive hemoptysis, this management is appropriate. Indeed, the bleeding vessel usually arises from the systemic circulation [6]. However, in a few cases Rasmussen aneurysm, which results from the destruction of the media of segmental pulmonary arteries by granulation tissue, is responsible. For those cases, another diagnostic and therapeutic approach might be more appropriate. This approach could take into account the pulmonary artery origin of the hemoptysis and the high risk of mortality. In the series by Auerbach, Rasmussen aneurysm was present in 45 cases and rupture of the aneurysm was the immediate cause of death in 38 of them [7].

Remy-Jardin et al. have shown that angiographic study and transcatheter occlusion of pulmonary arterial branch-

es was a logical, safe and successful approach in cases of pulmonary arteriovenous malformation or hemoptysis of pulmonary artery origin [8]. In our two cases, this approach was successful in the setting of intensive care.

For clinicians, Rasmussen aneurysm may be the cause of hemoptysis in all patients with cavitation due to active or inactive tuberculosis. However, it must be particularly suspected in two situations. In the first situation, as in our first case, a relapse of hemoptysis is observed after a technically good embolization of bronchial arteries. In this situation, the frequency of Rasmussen aneurysm is high, reaching 38% in the series of Sanyika [9]. As recommended by Remy et al. [10], angiographic study (CT scan and/or angiography) must be performed as a first-line investigation in those patients.

In the second situation, as in our second case, massive hemoptysis occurs in a patient with cavitory tuberculosis. In this situation, the frequency of Rasmussen aneurysm is around 4%: two cases out of 59 episodes of massive hemoptysis in the series of Remy et al. [10] and 45 cases out of 1114 consecutive autopsies of patients with chronic pulmonary tuberculosis and cavitation in the series of Auerbach et al. [7]. Currently, angiographic study must be performed as a second-line investigation in all patients with negative bronchial and non-bronchial systemic arteriography [10]. In future, enhanced helicoidal CT scan could be of interest as a first-line investigation.

In cases where angiographic study shows a Rasmussen aneurysm, transcatheter occlusion seems to us a logical conservative treatment option even if clinical experience with critically ill patients is limited and the risk of rupture by increased pressure during the procedure cannot be excluded [10, 11]. In our two cases, this treatment was safe and effective in the long run, as already shown in some patients with hemoptysis due to cavitory tuberculosis, abscess of the lung, necrotic hilar cancer or intracavitary aspergilloma [10, 12].

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