bronchogenic cyst and branchial cleft, thyroglossal, thymic, thyroid and enteric duplication cysts, lymphangiomas, dermoid cysts, teratomas and neuromas with the same topography.[1] Once diagnosis has been confirmed and any possible inflammation or sepsis have been treated, the treatment of choice is surgery. Choice of the surgical approach is wholly dependent on the topography and size of the cyst, and it is essential to remove the entire cyst in order to confirm the diagnosis, rule out neoplastic proliferation,[4] and ultimately control the disease.

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Rasmussen's Pseudoaneurysm in a Patient With a History of Pulmonary Tuberculosis☆

Seudoaneurisma de Rasmussen en un paciente con antecedente de tuberculosis pulmonar

To the Editor:

A 33-year old Romanian man was seen in the emergency room of our hospital for cough with hemoptysis. He reported fever lasting 48 h and night sweats lasting 2 weeks. He mentioned a history of pulmonary tuberculosis (TB) treated 5 years previously. The patient also showed signs of respiratory distress. Based on this information, we performed a chest X-ray, which showed bilateral interstitial and alveolar opacities. Subsequent contrast-enhanced multidetector computed tomography (MDCT) of the chest showed several consolidations (some cavitary), extensive bronchiectasis and a well-defined round lesion measuring 3 cm in the apical segment of the right lower lobe (RLL), with contrast uptake in the arterial phase and washout in the venous phase (Fig. 1). These
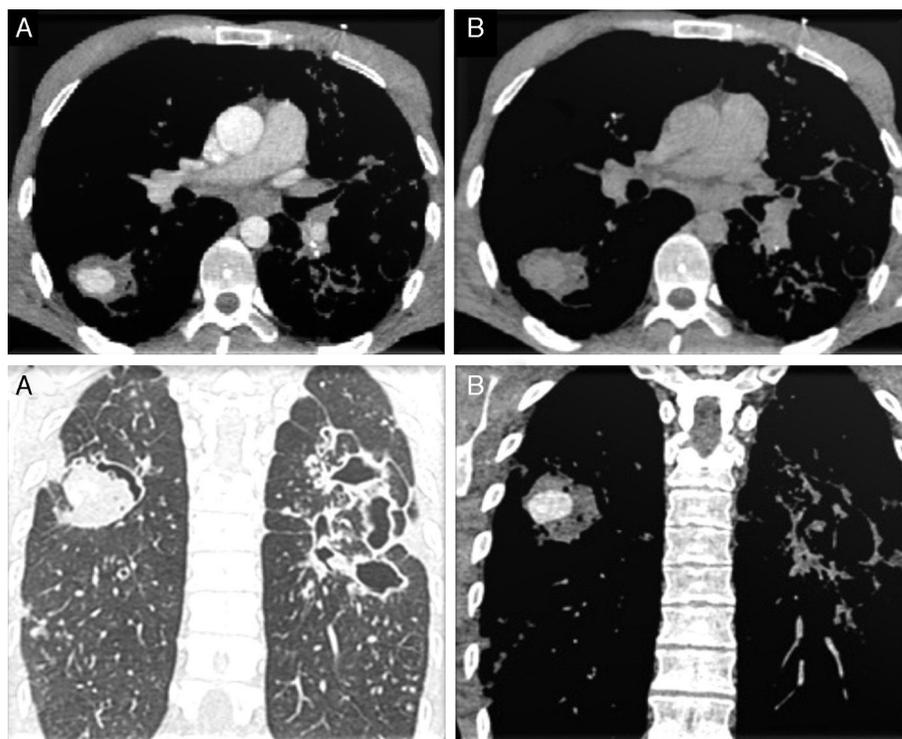

Fig. 1. Mediastinal window CT scan axial slices: lesion in the RLL, with well-defined borders, showing contrast uptake in the arterial phase (A top) and washout in the venous phase (B top). Parenchymal window (A bottom) and mediastinal window (B bottom) CT scan coronal slices in the arterial phase: contrast-enhanced nodular lesion in the RLL and images of bronchiectasis and extensive cavitation in the left hemithorax.

findings were suggestive of Rasmussen's pseudoaneurysm secondary to tuberculous infection.

The patient was admitted to the intensive care unit and given tuberculostatic therapy after the initial diagnosis was confirmed by Ziehl-Neelsen staining. Because of his hemodynamic instability, embolization as a secondary prevention measure was ruled out, as was resection of the lesion. Two days after admission, the patient presented massive hemoptysis, probably due to rupture of the lesion, and died.

Up to one third of patients with active TB will present massive hemoptysis over the course of the disease, with asphyxia, not the hemorrhage *per se*, as the principal cause of death.[1] In TB, the arterial damage is caused by replacement of the adventitia with granulation tissue, which is then replaced with fibrin, resulting in dilatation of the arterial wall. However, most hemoptyses will be caused by vascular erosion, without the formation of pseudoaneurysms.

These pseudoaneurysms, which were first described in 1868 by Fritz Valdemar Rasmussen, can originate in the bronchial vasculature (most frequently, in up to 90% of cases)[2] non-bronchial systemic arteries, or pulmonary artery branches. Hemoptysis, when secondary to TB, should alert clinicians to this diagnosis, which is best confirmed with a CT scan.

Hemoptysis appears in the pulmonary parenchyma as areas of ground-glass attenuation and areas of obstructive atelectasis due to blood in the bronchi, although these signs are non-specific.[3] The identification of a nodular image with intense contrast uptake during the arterial phase followed by washout in the venous phase is indicative of this type of vascular lesion.

A multidisciplinary[4] therapeutic approach is needed, aimed at maintaining airway permeability, optimizing oxygenation, and achieving hemodynamic stability.[4] Due to the considerable risk of complications, the final treatment of choice is percutaneous embolization (which can also be preventive) of the systemic arteries feeding the lesion, or even lobectomy in cases of serious, refractory disease.[5] Our protocol includes MDCT in order to locate the source of bleeding. This is followed by selective embolization of bronchial or pulmonary systemic arteries guided by the vascular map obtained with MDCT. If embolization is not effective, lobectomy can be considered.

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Severe Community Acquired Pneumonia Due to *Legionella maceachernii* Infection☆

Neumonía grave adquirida en la comunidad debida a infección por Legionella maceachernii

A 39-year-old man (heavy smoker, hypertensive and moderately obese) presented at the Internal Care Unit in October 2012 suffering from a 3-day history of dyspnea, paroxysmal productive cough and retrosternal pain, with no fever. Scarce end-expiratory crackles in middle and lower lung fields bilaterally with associated mildly prolonged expiratory phase of respiration and mild leucocytosis were recorded. The patient refused hospitalization, and two days later he returned due to worsened, intense dyspnea at rest, heart rate of 130/min and fever (38.5 °C). A new chest X-ray revealed more intense alveolar infiltrates, diffuse and expanded throughout the whole left lung and the right middle lung field (Fig. 1). Routine blood tests showed leukocytosis, elevated neutrophiles and monocytes, relatively increased CRP (7.5 mg/dl), ESR (73 mm/h), ALT (83 U/L) and LDH (484 U/L).

The patient was administered levofloxacin, piperacillin/tazobactam, supplementary oxygen, inhaled bronchodilators and oseltamivir (for 5 days) because influenza pneumonia could not be excluded; however, his condition deteriorated and non-invasive bi-level positive pressure ventilation was applied with full-face mask for 24 h. Ten days later he was discharged completely recovered.

During hospitalization, 3 whole blood and serum samples (on second admission, at 21 days and one month later) were collected together with sputum, pleural fluid and urine samples. Sera were tested by IFA for IgM and IgG antibodies against *L. pneumophila* sg1, *L. pneumophila* sgs 2–14 and Legionella species (*L. rubrilucens*, *L. anisa*, *L. brunensis*, *L. quinlivanii*, *L. maceachernii*, *L. oakridgensis*, *L. taurinensis* and *L. londiniensis*) using a homemade slide. Sera tested positive for *L. maceachernii* IgG antibodies in all samples (first: 1/960, second: 1/3840, third: 1/3840); titers for remaining species and/or serogroups ranged from 0–1/480. IgM antibodies tested positive (1/50) only for the first sample and only for *L. maceachernii*. All sera tested negative for Hepatitis Viruses, HIV, *Coxiella burnetii*, *Mycoplasma pneumoniae* and *Chlamydia pneumoniae*.

DNA was extracted (Qiamp DNA blood mini kit, Qiagen, Hilden, Germany) from whole blood and sputum samples and was tested by multiplex Real-time PCR for *L. pneumophila* and Legionella species,[1] which was positive, at low copy numbers, for Legionella species on the pleural fluid only.

An isolate, following culture (buffered charcoal yeast extract medium supplemented with α-ketoglutarate (BCYE-α) and BCYE with polymyxin B, anisomycin, and vancomycin petri dishes at 36 °C/2.5% CO_2) of whole blood pleural fluid and sputum samples was tested by MALDI-TOF MS (Bruker Daltonics) and matched Legionella species at a score of 1.99.

Legionella maceachernii infection is mostly expressed as pneumonia,[2] although recently a case of soft-tissue infection by the pathogen was reported.[3] All 6 patients described so