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

☆ Please cite this article as: Pasparaki E, Chochlakis D, Damianaki A, Psaroulaki A. Neumonía grave adquirida en la comunidad debida a infección por *Legionella maceachernii*. Arch Bronconeumol. 2015;51:97–98.

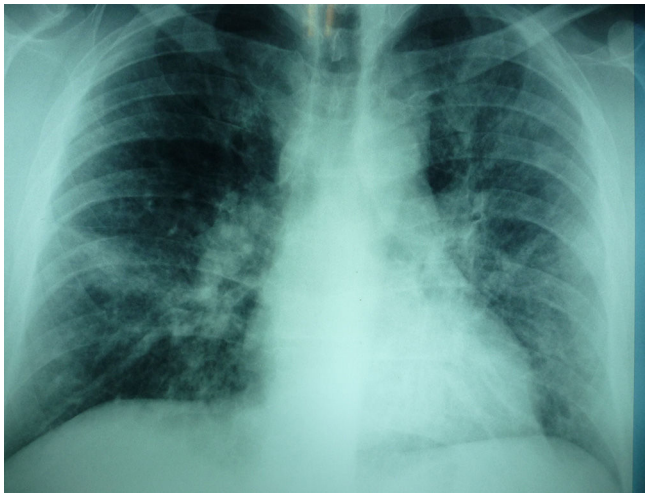

Fig. 1. Chest X-ray of the patient at hospital admission showing alveolar infiltrates, diffuse and expanded throughout the whole left lung and the right middle lung field.

far had underlying disorders, such as HIV infection, multiple myeloma, pulmonary fibrosis, systemic lupus erythematosus, and autoimmune hemolytic anemia, or had undergone liver transplantation. All but one patient died. Since the clearance of *L. maceachernii* from the lungs of infected mice has shown to be relatively quick compared to other species,[4] the increased mortality might be due to the concomitant presence of an underlying disease.

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Pulmonary Capillary Hemangiomatosis: A Diagnostic Challenge☆

Hemangiomatosis capilar pulmonar: un reto diagnóstico

Pulmonary capillary hemangiomatosis (PCH) is a low-grade pulmonary malignancy due to interstitial proliferation of capillary-like vessels occurring in patients of any age or sex. Prognosis is poor, with an estimated mean survival of 36 months.[1]

A 53-year old man, former smoker (39 pack-years), presented with worsening dyspnoea upon exertion and fatigue lasting 4 years. On admission, the patient was markedly tachypneic (respiratory rate 35 breaths/min) due to severe hypoxemia (40.5 mmHg). Electrocardiographic examination showed a PR interval of 140 milliseconds, with pulmonary P waves, right bundle branch block, and a heart rate of 94 bpm. Findings of llaboratory tests were unremarkable. Complete pulmonary function tests were not performed, as the patient was not compliant. Spirometry showed a mild obstructive ventilatory defect not reversible upon bronchodilation. Standard chest X-ray showed non-specific hilar congestion (not shown). Echocardiography revealed severe hypokinesia of the right ventricle along with a marked dilation of the right atrium and an estimated systolic pulmonary artery pressure of 70 mmHg. Right heart catheterization was refused. Thromboembolic pulmonary disease was ruled out by contrast medium computed tomography (CT) (Fig. 1a). Main imaging findings are shown in Fig. 1b-c. The patient was started on oral therapy with carvedilol (12.5 mg/day) and furosemide (125 mg/day) along with supportive care, and discharged home. He was re-admitted after 3 months due to clinical worsening and further studies were carried out showing that more than 90% of cells from broncho-alveolar lavage stained positive for iron using Perl's, suggesting iron deposition, while lung biopsy was highly suggestive of PCH (Fig. 1d-e-f). The patient was referred to a lung transplantation center. Now, over 50 months after diagnosis, his clinical condition is still seriously compromised, though stable.

We reported a case of PCH with an atypically long clinical course (6 years from clinical onset) along with a non-specific radiologic pattern. PCH may clinically masquerade as idiopathic pulmonary arterial hypertension (IPAH), or pulmonary veno-occlusive disease (PVOD). In PAH, differential diagnosis is crucial because pulmonary vasodilators may cause massive pulmonary edema in patients with PCH or PVOD.[2] Radiological characterization with chest HRCT is useful, but lung biopsy is mandatory for confirmation.[3] In our patient, chest HRCT was not typical for PCH due to the absence of centrilobular lung nodules,[4] and final diagnosis was based on pathology data. PCH is characterized by alveolar wall thickening due to capillary proliferation. Infiltration and compression of pulmonary veins by new capillaries can result in secondary PVOD. PCH can be differentiated from IPAH or PVOD on the basis of the diameter of new pulmonary capillaries (larger in PVOD > PCH > IPAH) and the size of centrilobular nodules (wider in PCH>PVOD; absent in IPAH).[5] Interstitial fibrosis, hemosiderosis and changes due to pulmonary arterial hypertension may also be found.

Clinicians and radiologists should bear PCH in mind, as early identification may improve patient management. PCH behaves like a low-grade non-metastatic vascular neoplasm, with a slow progressive clinical course. Prognosis is poor and lung transplantation is the best option.

☆ Please cite this article as: Rea G, Valente T, de Rosa N, Muto M, Berritto D, Bocchino M. Hemangiomatosis capilar pulmonar: un reto diagnóstico. Arch Bronconeumol. 2015;51:98–99.