findings were suggestive of Rasmussen's pseudoaneurysm sec-
ondary 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 mas-
usive 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 pseudo-
aneurysms.

These pseudoaneurysms, which were first described in 1868
by Fritz Valdemar Rasmussen, can originate in the bronchial vas-
culature (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 dur-
ing 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.\(^5\) Due to the considerable risk
of complications, the final treatment of choice is percutaneous
embolization (which can also be preventive) of the systemic arter-
ies feeding the lesion, or even lobectomy in cases of serious,
refractory disease.\(^6\) 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, lobec-
tomy can be considered.

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

*Neumonia grave adquirida en la comunidad debida a infección por Legionella maceachernii*

A 39-year-old man (heavy smoker, hypertensive and mod-
erately 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 respira-
tion and mild leucocytosis were recorded. The patient refused
hospitalization, and two days later he returned due to wors-
ened, 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 neutrophils and monocytes, rela-
tively 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

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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 dyspnea 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 laboratory 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,2 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.

References


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