bronchogenic cyst and branchial cleft, thyroglossal, thyritic, thyroid and enteric duplication cysts, lymphangiomas, dermoid cysts, teratomas and neuromas with the same topography. 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, and ultimately control the disease.

References


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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

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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.

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findings were suggestive of Rasmussen’s pseudoaneurysm sec-
to 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 instabil-
ity, 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-
ive 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 multidisciplinary4 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. Scare 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 infil-
trates, 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, 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/tazo-
bactam, 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 recov-
ered.

During hospitalization, 3 whole blood and serum samples (on
second admission, at 21 days and one month later) were collected
along with sputum, pleural fluid and urine samples. Sera were
-tested by IFA for IgM and IgG antibodies against L. pneumophila
sg1, L. pneumophila sg2–14 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 pneu-
moniae.

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,5
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% CO2) of whole blood pleural fluid and sputum sam-
ple 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

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