

suggestive of tumor disease (Fig. 1). Thoracentesis was performed and a serofibrinous fluid was drained. This was considered exudate in view of the protein ratio of 0.8, although pH and glucose were normal and the leukocyte count was very low (140 mm^3). Adenosine deaminase levels were normal. Sputum smears and cultures were negative. Cytology revealed inflammation and reactive mesothelial hyperplasia. Fiberoptic bronchoscopy was normal. Positron emission tomography (PET) was negative for malignancy. Magnetic resonance imaging (MRI) identified a poorly delimited, heterogeneous paraspinal lesion, with no vertebral involvement. Possible pleural infiltration with a vascular component and pleural effusion was observed, the origin of which was located in the paraspinal white tissue or parietal pleura (Fig. 1). Rapid pleural filling required frequent drainage by thoracentesis. Finally, video-assisted thoracoscopy was performed and a solid paravertebral and intravertebral tumor was resected. Gross examination revealed an uneven, purplish $4\text{ cm} \times 2\text{ cm} \times 2\text{ cm}$ tumor with hemorrhagic areas, partially enveloped in pleura. It was identified microscopically as a fragment of parietal pleura, with transmural thickening as a result of a poorly delimited, unencapsulated solid tumor, consisting of mature adipocytes intermixed with abundant vessels of varying sizes, and no endothelial cell atypia. The mesothelial sheath showed mild hyperplastic reactive changes (Fig. 1d). Immunohistochemistry showed: CD-34, CD-31 and factor VIII: intense, diffuse positivity in the vascular areas; calretinin, pankeratin, and Ck 5/6: positivity in the mesothelial sheath; Ki57: low proliferative index ($<3\%$). The definitive diagnosis was mesenchymal tumor, consistent with paravertebral chest wall angioliipoma. One year after resection and talc pleurodesis, the patient remains without relapse.

Angioliipomas are benign tumors, generally located under the skin, most often in the trunk or the limbs, although they have very occasionally been described in the thoracic cavity.¹ This is the first report of an intrapleural location with associated effusion. In anatomical pathology terms, these tumors are formed of mature adipocytes and numerous vessels in varying proportions.² They

tend to appear benign on PET imaging.³ CT may reveal heterogeneity with areas of fat attenuation and enhancement in vascular areas, but differences in the ratios of each type of tissue make it difficult to make an accurate diagnosis. Differential diagnoses to consider include infiltrating hemangioma, neuroendocrine tumors, or other mesenchymal tumors. In view of the difficulty of achieving diagnosis before surgery, MRI may be the gold standard imaging test, as it reveals isointense images in T1 (lipomatous component) and hyperintense images in T2 (vascular component).⁴

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Antineutrophil Cytoplasmic Antibodies (ANCA)-Negative Vasculitis in a Patient With Alpha-1-Antitrypsin Deficiency[☆]



Vasculitis con Anticuerpos anticitoplasma de neutrófilos (ANCA) negativos en paciente con déficit de alfa-1 antitripsina

To the Editor

We report the case of a 62-year-old man, former smoker of 20 pack-years, with a history of arterial hypertension, diabetes mellitus and previous ictus with no neurological sequelae. He was referred from the respiratory medicine clinic with dyspnea on moderate effort (mMRC 2). Lung function tests showed FEV₁/FVC 47%; FEV₁ 1.4 l (44%); FVC 3.3 l (82%); VR 4.4 l (182%); TLC 8.0 l (119%); DLCO 49% and KCO 59%. Chest computed tomography showed severe panacinar emphysema, primarily in the lower lobes. Severe alpha-1-antitrypsin (AAT) deficiency (28.4 mg/dl) associated with ZZ phenotype was observed. A diagnosis of COPD with severe airflow obstruction and type ZZ alpha-1-antitrypsin deficiency (AATD) was made, and after abdominal ultrasound confirmed chronic liver disease, the decision was made to administer

replacement AAT. Before initiation of treatment, the patient had an episode of epistaxis associated with purplish lesions in the lower limbs, tending to converge, with no blanchability on diascopy. Pathology study results showed leukocytoclastic vasculitis in small and medium caliber vessels, associated with elevated IgA (754 mg/dl), microhematuria and proteinuria suggestive of nephritis. Together, these signs were consistent with a diagnosis of adult Henoch–Schönlein purpura (HSP). ANCA antibodies (MPO $<0.8\text{ IU/ml}$ and anti-PR3 $<0.4\text{ IU/ml}$) and ENA were negative; ANA were positive with a titer of 1/160 in a fine speckled pattern. On the basis of this diagnosis, treatment began with oral corticosteroids (0.5 mg/kg/day) in a tapering regimen.

One month after beginning this treatment, the patient suffered a fall at home and injured his left arm, with subsequent development of diffuse arthralgia and asthenia. Magnetic resonance imaging revealed cellulitis, arthritis and synovitis in the distal radioulnar and carpometacarpal joints of the upper left limb. Synovial biopsy and fluid culture were performed, confirming synovitis with positive multi-resistant *Pseudomonas aeruginosa* culture. Despite the administration of wide-spectrum antibiotics and systemic corticosteroids, the patient developed multiorgan failure and died in the intensive care unit.

A review of AATD and concomitant necrotizing vasculitis shows that microscopic polyangiitis and Wegener's granulomatosis are the most common forms, while HSP is an unusual manifestation. The mean age for presentation is generally around 48 years, and

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there is no prevalence with regard to sex. All cases present skin involvement, along with kidney or joint disease. In cases of HSP, hepatic cirrhosis is usually observed.^{1,2}

Our patient presented the foregoing conditions, further complicated by severe sepsis due to cellulitis and arthritis of the wrist. Evidence shows that AATD patients can occasionally develop necrotizing panniculitis induced by injury.³ In our case, the histological diagnosis could not be confirmed, since a synovial biopsy, rather than a skin biopsy, was obtained, but the clinical signs and symptoms and progress were consistent with this diagnosis.

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Hemoptysis in Tuberculosis: The Importance of Contrast-Enhanced Computed Tomography[☆]



Hemoptisis en tuberculosis: la importancia de la tomografía computarizada con contraste

To the Editor,

We read with interest the case of a 33-year-old man with hemoptysis reported by Peghini Gavilanes et al.,¹ in which contrast-enhanced computed tomography (CT) of the chest demonstrated a Rasmussen aneurysm secondary to tuberculous infection. The patient died 2 days later due to massive hemoptysis. Although Rasmussen aneurysms frequently present as nodular lesions within or adjacent to cavitory tuberculous lesions,^{1,2} they also may occur inside consolidations, making their identification more difficult.

We would like to report a similar case of a 37-year-old man who was admitted to the emergency room with hemoptysis. The patient had a history of pulmonary tuberculosis (TB), treated irregularly for the previous 2 years. Sputum was positive for acid-fast bacilli. Fiberoptic bronchoscopy showed active bleeding from the right lower-lobe bronchus. Contrast-enhanced chest CT revealed nodules and cavities suggestive of active pulmonary TB, and consolidation in the right lower lobe with a rounded enhancing lesion within the consolidated area (Fig. 1). Transcatheter embolization with coils was performed. The patient also received TB therapy and recovered well, with no recurrence of bleeding during 1 year of follow-up.

With the recent worldwide resurgence of reported cases of *Mycobacterium tuberculosis* infection, recognition of complications and sequelae is important. Hemoptysis – often massive – in the presence of TB can have various etiologies, such as bronchiectasis, aspergilloma, TB reactivation, scar carcinoma, chronic bronchitis, microbial colonization within a cavity, and vascular complications such as pseudoaneurysms.³ Pulmonary artery pseudoaneurysms secondary to pulmonary TB are known as Rasmussen aneurysms and are caused by destruction of the media of segmental pulmonary arteries by granulation tissue from adjacent cavitory TB.^{1,4,5} Massive hemoptysis associated with chronic cavitory TB usually results from the rupture of a pseudoaneurysm through the cavity wall, and is potentially fatal.^{1,4,5}

The diagnosis of Rasmussen aneurysm can be made readily on the basis of characteristic imaging findings. Chest radiographic

findings that may suggest the formation of a pseudoaneurysm include intracavitary protrusion, the replacement of a cavity by a nodule, and a rapidly growing mass.⁴ Rasmussen aneurysm can be identified on pre- and post-contrast-enhanced CT images as avidly enhancing nodules located within the walls of tuberculous cavities² or consolidations.

The optimal management of massive hemoptysis is currently under debate.⁵ Endovascular occlusion of the neck of the

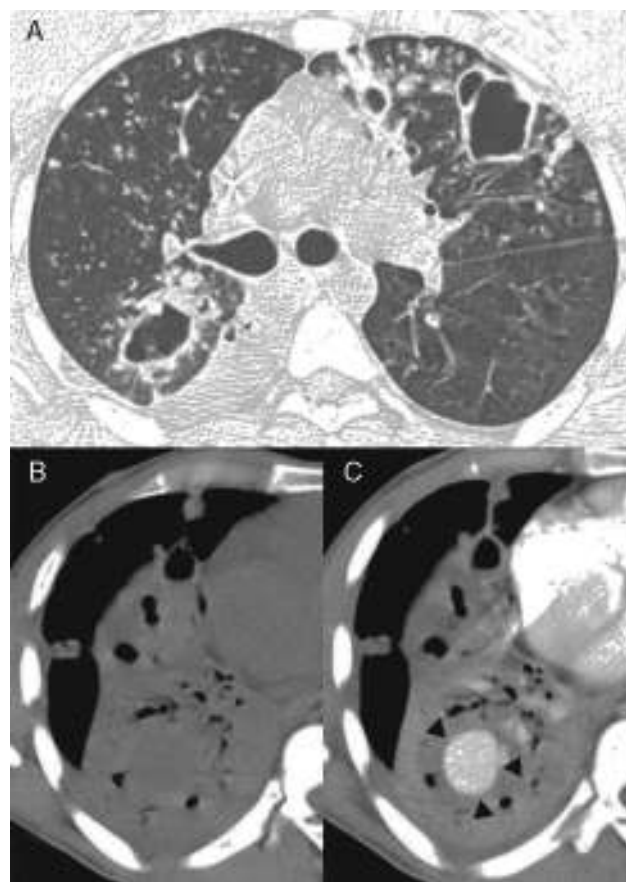


Fig. 1. (A) Axial CT image obtained with the lung window setting at the level of bronchial bifurcation, showing multiple bilateral nodules and cavities in the lung parenchyma. CT images obtained with the mediastinal window setting at the level of the lower lobes before (B) and after (C) contrast administration reveal consolidation in the right lower lobe with a rounded enhancing lesion inside the consolidated area (arrows).

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