

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.<sup>1,2</sup>

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.<sup>3</sup> 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<sup>☆</sup>



### *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.,<sup>1</sup> 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,<sup>1,2</sup> they also may occur inside consolidations, making their identification more difficult.

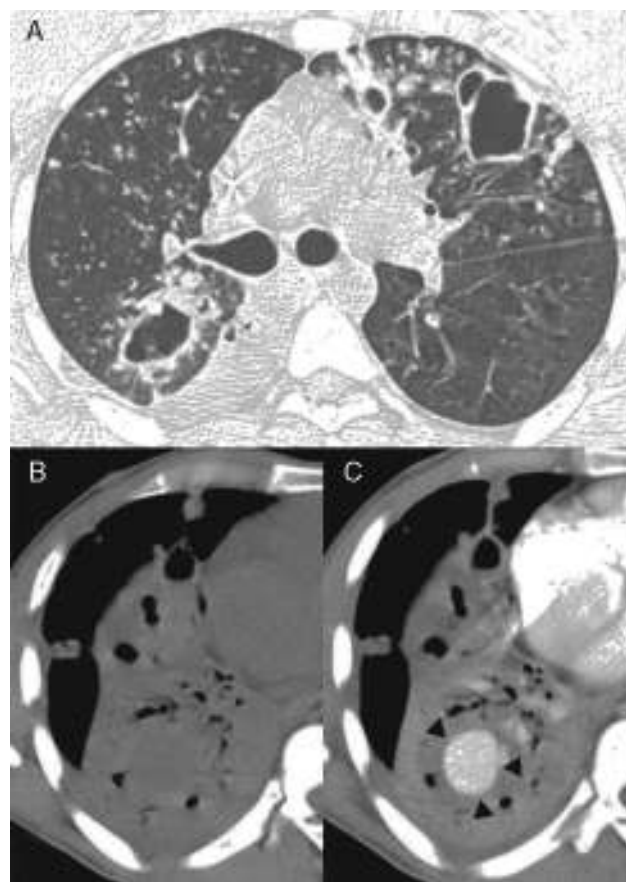
We would like to report a similar case of a 37-year-old man who was admitted to the emergency room with hemoptysis. The patient had a history of pulmonary tuberculosis (TB), treated irregularly for the previous 2 years. Sputum was positive for acid-fast bacilli. Fiberoptic bronchoscopy showed active 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.<sup>3</sup> 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.<sup>1,4,5</sup> Massive hemoptysis associated with chronic cavitory TB usually results from the rupture of a pseudoaneurysm through the cavity wall, and is potentially fatal.<sup>1,4,5</sup>

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.<sup>4</sup> Rasmussen aneurysm can be identified on pre- and post-contrast-enhanced CT images as avidly enhancing nodules located within the walls of tuberculous cavities<sup>2</sup> or consolidations.

The optimal management of massive hemoptysis is currently under debate.<sup>5</sup> 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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pulmonary aneurysm is well described in the literature and is usually successful in treating RA.<sup>1,5</sup> Possible complications of treatment are bleeding recurrence, inadvertent embolization of a spinal artery leading to ischemic myelopathy, and aneurysmal rupture during diagnostic catheter angiography,<sup>1,5</sup> with a high risk of mortality. Surgical excision is recommended only when expert radiological intervention is not available or in the presence of considerable destructive process in the lung.<sup>5</sup>

In conclusion, Rasmussen aneurysm should be included in the differential diagnosis of hemoptysis in patients with TB. Enhanced CT examination plays an important role in the evaluation of these patients.

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## Combined Alpha-1-Antitrypsin Deficiency and Mannose-Binding Lectin Deficiency<sup>☆</sup>



### Déficit combinado de alfa-1-antitripsina y déficit de lectina fijadora de manosa

To the Editor,

Alpha-1-antitrypsin deficiency (AATD) is a hereditary disease. Clinical manifestations include pulmonary emphysema, hepatic cirrhosis, and more rarely, ANCA-positive panniculitis or vasculitis.<sup>1</sup> Few instances of its association with other genetic deficits have been reported in the literature.

We report the case of a 36-year-old man with no toxic habits and a history of atrial fibrillation, treated with oral anticoagulants, referred to the pulmonology department for genetic counselling after the detection of a family member as an AATD index case (father with SZ genotype). Genetic studies were performed, and the patient was diagnosed with type SZ AATD, with serum protein levels of 46.4 mg/dl. Spirometry and high-resolution computed tomography were normal. In subsequent follow-up visits, he began to present repeated bronchial infections, so a full study of primary immunodeficiencies was performed. In this study, very low levels of mannose-binding lectin (MBL) were detected on ELISA (normal range >1000 ng/ml, deficit considered <500 ng/ml and severe deficit <100 ng/ml): the initial result was 7.54 ng/ml, and a subsequent test found 72.807 ng/ml. Other primary immunodeficiency results, including immunoglobulin serum levels, were normal (IgG 1186 mg/dl; IgA 362.80 mg/dl; IgM 75.60 mg/dl).

Alpha-1-antitrypsin (AAT) is a glycoprotein<sup>1</sup> synthesized mainly in the liver. Its principal function is to inhibit neutrophil elastase and other proteases, and its anti-inflammatory, anti-microbial and immunomodulatory properties have been characterized in recent years. AAT deficiency is caused by a mutation on the long arm of chromosome 14. Lectin is a protein synthesized in the liver which is found in different types of biological fluids, and is considered by some authors as an acute-phase reactant.<sup>2</sup> MBL is involved in the activation of the lectin complement system, which has been established as the third activation pathway.<sup>3</sup> MBL deficiency is caused by a mutation on the long arm of chromosome 10. Low serum

levels of MBL are associated with an increased risk of various infections in children. In adults, this deficiency may be associated with concomitant diseases or immunodeficiencies.<sup>4</sup> MBL deficiency has been associated with predisposition to a large number of diseases (autoimmune diseases, in particular), but it generally has more clinical significance when it coexists with another immunodeficiency state.<sup>5</sup> Curiously, the association of MBL deficiency and AATD in the same patient has not been described previously.

The clinical presentations of AATD vary widely, so disease registries and studies are needed to better determine its natural history. Taking into account the prevalence of both deficiencies in the general population, its association in our patient may be a random event. Nevertheless, these immunodeficiencies may need to be ruled out in AATD patients, since they could modify the clinical manifestation and management of the disease, as occurs with other immunodeficiency states.

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