

suggestive of aspergillosis associated with microbiologic identification in lavage fluid, or a positive galactomannan serum assay.¹ A background inquiry revealed that the patient had moved to a new house and received a large quantity of flowers before the onset of her symptoms. Because the patient was not immunocompromised, a diagnosis of fungal tracheobronchitis was not initially considered; this delayed the diagnosis by 6 months, which is in agreement with the literature.³ In immunocompetent patients, we consider *Aspergillus* tracheobronchitis in the presence of numerous gelatinous whitish plaques in the trachea and right and left bronchi. In this case, the earliest indication of an *Aspergillus* infection was mucoid impaction along the bronchus. Although hyphae were seen in the biopsy, fungal cultures of sputum and respiratory secretions were negative, and no galactomannan positivity was identified. Mucoid impaction and bronchocentric granulomatosis are seen in half of ABPA cases.⁴ After failing to identify an alternative cause, we interpreted bronchocentric granulomatosis in this patient as a histopathologic manifestation of fungal hypersensitivity.⁴

Cases of bronchocentric granulomatosis are classified according to the clinical presentation. The first subtype includes asthma and atopy, while the second subtype is idiopathic; some case reports suggest an association with other underlying diseases such as bronchogenic carcinoma, post-radiation pulmonary fibrosis, rheumatoid arthritis, and granulomatosis with polyangiitis.⁴ Patients with the first subtype tend to be younger (20–40 years), while non-asthmatic patients tend to be older (30–70 years). In our case, atopy was the only co-morbidity.⁴ Corticosteroids are effective mainly in patients with asthma and atopy.^{5,6} Corticosteroid therapy was effective in our patient, and resolved all symptoms.

In conclusion, bronchocentric granulomatosis caused by *Aspergillus* tracheobronchitis should be considered in immunocompetent patients with tracheobronchial infiltrates on CT scan who do not respond to inhaled steroids or broad-spectrum antibiotics.

Our patient yielded negative cultures and normal galactomannan values, but tracheal involvement was suggestive of *Aspergillus* tracheobronchitis, and hyphae without tissue or vascular invasion were identified in a biopsy. Therefore, recovery of fungal hyphae in a biopsy specimen from an immunocompetent patient with atypical respiratory symptoms and CT findings should not be dismissed as contamination.

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Pulmonary Thromboembolism as a First Manifestation of Atrial Myxoma[☆]



Tromboembolismo pulmonar como primera manifestación de un mixoma auricular

To the Editor,

Atrial myxomas are the most common benign primary cardiac tumors. They occur preferentially in the left atrium and clinical manifestations can be varied, with asthenia, anorexia, and weight loss due to intracavitary flow obstruction in the mitral valve and systemic embolisms; however, only exceptionally do they cause pulmonary thrombosis.^{1–3}

A 47-year-old woman, smoker of 20 pack-years, with no other history of interest, was admitted to the respiratory medicine department with a 4-month history of dyspnea on moderate exertion, general malaise, asthenia, right pleuritic pain, and polyarthralgia. She had also had fever and mild hemoptysis in the previous 4 days. Physical examination showed basal oxygen saturation 94%, heart rate 103 bpm, BP 107/73 mmHg, with signs of pleural effusion on auscultation. Of note on clinical laboratory tests were elevated CRP (95.7 mg/l) and 18 500 leukocytes/mm³ with neutrophilia. Chest X-ray showed consolidation in the left

lower lobe with associated pleural effusion. Blood cultures, sputum microbiology, and antigen detection in urine were negative for pneumococcus and *Legionella*. Diagnostic thoracentesis was performed and was compatible with uncomplicated neutrophilic exudate. Cytology for malignant cells and microbiological cultures were negative. Despite starting empirical wide-spectrum antibiotic treatment, the patient's clinical, hemodynamic, and radiological situation worsened, with increased work of breathing, tendency to hypotension, and mildly altered level of consciousness. An angio-CT was performed, which revealed bilateral pulmonary thromboembolism (PTE), a filling defect in the left atrium, and multiple bilateral opacities consistent with pulmonary infarcts. Eco-Doppler of the lower limbs ruled out deep vein thrombosis (DVT).

On echocardiography, a large mass was observed in the left atrium (6.3×3.2 cm) that protruded in diastole toward the left ventricle, obstructing the outflow. Signs of severe pulmonary hypertension were also seen, with pulmonary artery systolic pressure 85 mmHg, and estimated left ventricular ejection fraction 60% (Fig. 1).

In view of the severity of the clinical picture and hemodynamic instability, the patient was transferred to the Intensive Care Unit. She subsequently worsened significantly, and the cardiovascular surgery department was contacted for removal of the intracardiac tumor. The pathology report of the surgical specimen confirmed a diagnosis of atrial myxoma. After surgery and anticoagulant treatment, clinical progress was very satisfactory; in a follow-up echocardiography, the pulmonary hypertension had resolved and the pulmonary angio-CT showed resolution of the intravascular

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Fig. 1. Echocardiography showing a large mass in the left atrium (6.3×3.2 cm) in diastole protruding into the left ventricle.

clots. A thrombophilia study was also requested, and the patient was found to be a heterozygous carrier of the factor V Leiden mutation.

A myxoma is a benign cardiac tumor; however, its benign nature is relative, since it sometimes produces distant metastases and paraneoplastic syndromes caused by cytokines and growth factors produced by the tumor.^{4,5} Clinical presentation depends on size and location, and it can cause intracardiac flow obstruction, stenosis, and mitral regurgitation in the left cavities.⁵ Echocardiography is the diagnostic test of choice, and the curative treatment is surgical resection. Being a carrier of factor V Leiden mutation is not in itself an independent risk factor for the development of PTE, but rather an additional factor along with others that confer moderate or high risk.

The presence of systemic embolisms has been frequently noted in reviews of atrial myxoma. Our case is exceptional in that the myxoma manifested as PTE without associated DVT. We believe that this may be the consequence of various concomitant risk factors: hypercoagulability derived from the myxoma, thrombophilia, concomitant infection, and obstruction of the blood flow by the tumor which may promote the presence of “in situ” thrombi in the pulmonary circulation.

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Pneumothorax due to Radiofrequency Ablation for Post-Herpetic Neuralgia[☆]



Neumotórax secundario a ablación por radiofrecuencia de neuralgia postherpética

To the Editor,

Postherpetic neuralgia is the most common late complication of herpes zoster, and occurs in 9%–19% of patients.¹ It is caused by nerve damage in the area affected by the virus and is defined as persistent pain longer than 3 months after resolution of the characteristic vesicular rash.^{2–4} The main clinical problem is persistent pain that interferes with the patient's daily activities. The chest region (dermatomes T1 to T12) is the area most commonly affected by postherpetic neuralgia, with an incidence of up to 50%.⁵ Various treatments have been proposed,^{6–10} including drugs, botulinum toxin injections, nerve blocks, peripheral nerve stimulation, surgical intervention, pulsed radiofrequency treatment, and radiofrequency ablation.¹¹

Given the scarcity of published scientific evidence for iatrogenic pneumothorax following the use of conventional radiofrequency, we present the case of a 62-year-old woman, with no toxic habits, diagnosed with fibromyalgia and osteoporosis, who developed pos-

therpetic neuralgia as a late complication of thoracic herpes zoster infection in 2011. She had been monitored by the pain unit since 2012, and had received several treatments without success, including nerve block with local anesthetics and steroids in 2015. In April 2016, she began treatment with conventional intercostal radiofrequency, involving stimulation of the painful area above the right 9th intercostal space at 80° for 90 s, without complications. A second incident-free radiofrequency session was conducted 12 weeks later, followed in December by a third conventional radiofrequency session at the 5th intercostal space. During this last session, the patient developed dyspnea, tachycardia 110 bpm, and hypotension, so an emergency chest X-ray was performed, showing right pneumothorax (Fig. 1). A chest tube was placed, after which complete reexpansion of the right lung was observed. The patient was discharged from the pulmonology department 48 h after the procedure with no complications.

Postherpetic neuralgia generally responds to drug therapy, and this should be used before any other intervention is attempted. Refractory cases can be treated with minimally invasive, though not entirely risk-free procedures such as nerve block, after which 0.09% of patients develop pneumothorax, a figure that rises to 0.42% if all patients are routinely given a chest X-ray.¹² However, pneumothorax is not a common complication of radiofrequency techniques: no events were reported in larger series of up to 96 patients undergoing this treatment for postherpetic neuralgia.⁹

Radiofrequency is a minimally invasive intervention for the management of chronic pain, and its use in chronic pain units has been increasing in recent years.

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