

Letters to the Editor

Intrathoracic Vagal Nerve Schwannoma***Schwannoma del nervio vago intratorácico**

Dear Editor:

Neurogenic tumors represent approximately 20% of all mediastinal neoplasms in adults.¹ Anatomically, they can derive from the nerve sheath, the ganglion cells or from the paraganglion system. The majority are benign tumors and are located in the posterior mediastinum at the level of the paravertebral gutter, coming from the root of either an intercostal or sympathetic nerve.² Only between 4% and 6% originate in the visceral compartment from the nerve sheaths of the vagus or phrenic nerves.³

We present the case of a 53-year-old male with a personal history of HTN and right peripheral facial paralysis for the last 2 years. In the study for a possible myopathy, simple chest x-ray revealed a posterior mediastinal mass, and the patient was referred to our department. The study of the patient was completed with thoracic CT that confirmed a mass measuring 63 × 57 mm that was hypodense, walled and localized in the posterior mediastinum (Fig. 1). It was causing compression and a displacement of the lumen of the lower third of the esophagus and of the posterior

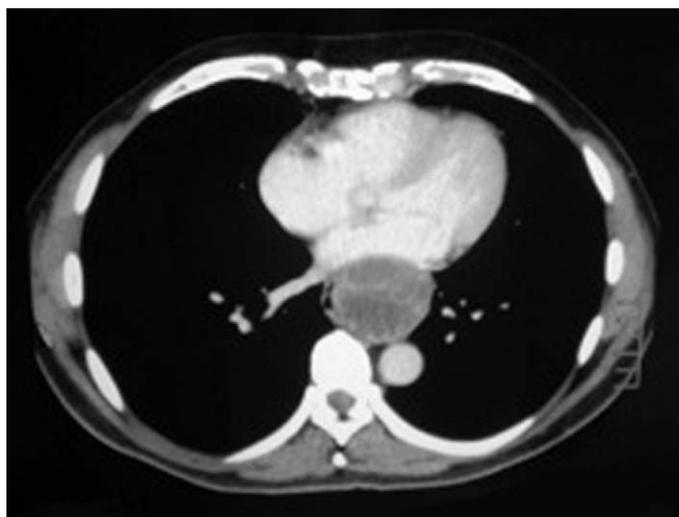


Fig. 1. CT axial cut showing a posterior mediastinal mass compressing the esophageal lumen and the left auricle.

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wall of the left auricle, compatible with a possible esophageal leiomyoma. Left posterior thoracotomy was carried out through the 7th intercostal space, finding a cystic, encapsulated tumor located in the pathway of the left vagus nerve. The lesion was extra-esophageal and was covered by the paraesophageal mediastinal pleura. It was completely resected after ligating the vascular pedicles and liberating the adhesions over the esophagus. The histopathological report revealed a cystic tumor measuring 65 × 58 mm that was unilocular and presented a classic biphasic pattern, alternating with areas of Antoni type A and type B of the benign schwannomas. Post-op presented no complications, and the patient was discharged on the third day after the intervention.

The patient currently continues to be asymptomatic and disease-free 26 months later.

Schwannomas are tumors of the nerve sheath originating in the nerve roots or the peripheral nerves.⁴ More often than not, they are located in the head, neck and extremities.^{1,2} They may also present within the thorax, originating in the sympathetic chain, spinal nerve roots, intercostal nerves, brachial plexus, phrenic nerve or vagus nerve.^{1,2} Schwannomas of the vagus nerve are located above all in on the left side,⁵ as in our case, as the nerve trunk is longer and wider.

They usually present no symptoms⁴ except for the compression of neighboring structures, frequently appearing as incidental radiological findings.^{3,4} The treatment of choice is surgical exeresis,² which also allows for the diagnosis to be made. The approach may be either by means of thoracotomy or video-assisted thoracoscopy.² The latter may be contraindicated in tumors larger than 6 cm.⁵ In our case, we decided to perform an approach through a posterior thoracotomy in the 7th intercostal space due to the size of the lesion and the differential diagnosis with an esophageal leiomyoma-type tumor or gastrointestinal stromal tumor (GIST).

Complete exeresis of benign schwannomas is considered curative as recurrence is infrequent.

Although they are very rare, schwannomas of the vagus nerve should be included in the differential diagnosis of posterior mediastinal masses in close association with the esophagus, together with other mediastinal neurogenic tumors, lymphomas, mediastinal cystic lesions and esophageal tumors.

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New Challenges for the Pulmonologist in the Diagnosis of Latent Tuberculosis Infection: Biological Treatment and Pulmonary Tuberculosis[☆]

Nuevos retos para el neumólogo en el diagnóstico de la infección latente tuberculosa: tratamientos biológicos y tuberculosis pulmonar

Dear Editor:

There is more and more accumulated evidence that questions the safety of treatment with tumor necrosis factor (TNF) antagonists regarding the development of tuberculosis (TB). Although we have more data available on the risk for TB in patients treated with etanercept and infliximab, there is less known about the risk regarding adalimumab. We present the case of a patient with psoriasis treated with adalimumab who developed pulmonary tuberculosis associated with pleural effusion.

The patient is a 75-year-old male professional enologist with a medical history of diabetes mellitus, spondyloarthritis and current treatment with adalimumab. Prior to the start of the treatment, he presented normal chest radiography, negative tuberculin test, and since he presented no TB risk contacts, treatment for latent tuberculosis infection (LTI) was not indicated. One month after the start of the treatment, he consulted for a symptoms evolving over 3 weeks of continuous thoracic pain in the right hemithorax, accompanied by low fever, asthenia and anorexia, with no additional cough or expectoration. He had been receiving treatment with ciprofloxacin for 1 week, with no improvement. On chest radiograph, pleural effusion was observed up until the half of the right hemithorax, with segmental alveolar infiltrates in the upper left lobe. The study of the pleural liquid revealed: uncomplicated, predominantly lymphocytic exudate (65%); ADA 82 U/l; negative smear, Ziehl–Neelsen stain and PCR for mycobacteria; negative bacteria culture and Gram stain. Mycobacterial culture confirmed the presence of *M. tuberculosis* complex, sensitive to all drugs. The patient was treated with anti-tuberculosis drugs, and adalimumab was withdrawn with no immediate complications. During follow-up, the patient presented an episode of interstitial nephritis secondary to adalimumab, which was resolved after the withdrawal of the drug.

Anti-TNF drugs, such as etanercept, infliximab and adalimumab, are fundamental in the treatment of inflammatory diseases, but the relative risk for TB increases between 1.6 and 25 times with this treatment, depending on the clinical context, the drug used and the country of origin.^{1,2} The rate of TB in patients treated

with anti-TNF is 3–4 times greater in those who receive infliximab and adalimumab than in those receiving etanercept.³ LTI treatment with isoniazid for 9 months is adequate in patients with a positive tuberculin test, a chest radiograph suggesting disease or if there has been close contact with a case of tuberculosis.⁴ These guidelines have been shown to diminish in 80% the incidence of TB in our country.¹ Our patient developed tuberculosis with pleural affection, despite following the recommendations for TB presentation by the Spanish Society of Rheumatology, therefore it is of interest to provide more evidence in addition to what has been recently reported about the complications of treatment with adalimumab.⁵ Although the time elapsed from the introduction of adalimumab until the onset of the symptoms could make us contemplate reactivation, a negative Mantoux at the start of the treatment would rule this out. The introduction of the diagnostic method based on the *in vitro* production of interferon gamma in response to *M. tuberculosis* antigens (*interferon-gamma release assay* [IGRA]) would be especially useful in patients in whom negative tuberculin test should not rule out a latent infection, distinguishing true positives from the false positives caused by anti-tuberculosis vaccination.

We pulmonologists have a new challenge in controlling TB: patients who use anti-TNF drugs. The implementation of the new LTI diagnostic techniques would be of great use for this purpose.

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