

There are differences in the latency for developing mesothelioma after exposure to radiotherapy as well as in the age of presentation compared with asbestos-related mesothelioma. After a review of the published literature,<sup>2-4</sup> we have identified 6 cases reporting malignant pleural mesothelioma induced by radiotherapy used for the treatment of breast cancer. The time interval between the radiation and the appearance of the mesothelioma ranged between 10 and 30 years, and the mean age at presentation of the mesothelioma was 55. In asbestos-related mesotheliomas, there is a greater time interval between the exposure and the appearance of the tumor (usually 30-40 years) and patients also present older ages.

The diagnosis is difficult, especially in cases with no history of exposure to asbestos and due to the need for a large tissue sample for the anatomic pathology study. Although pleural cytology or cytological puncture can guide the diagnosis, a technique is required to provide a biopsy fragment of optimal size. Thus, thoracoscopy is the diagnostic technique of choice.<sup>5</sup> PET/CT can direct the area for biopsy as it identifies tumor areas thanks to the greater greediness of neoplastic cells for glucose. In the case described, its contribution is demonstrated. Given our patient's characteristics and history and despite the fact that the initial diagnostic suspicion was pleural metastasis of breast carcinoma, the stepwise diagnostic study based on recommendations<sup>6</sup> essentially would not have varied even if mesothelioma was considered among the main suspected diagnoses.

We present a case of pleural mesothelioma secondary to radiotherapy used as a treatment for breast cancer. In spite of the limited frequency of pleural mesothelioma associated with radiotherapy due to breast carcinoma, we should consider this diagnostic pos-

sibility in patients with pleural thickening indicative of tumor affection and a history of thoracic radiotherapy. The application of the diagnostic protocol based on guidelines has been shown to be useful.

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## Spontaneous Pneumomediastinum: Is It a Sign of Severity, or Does It Depend on the Underlying Respiratory Process?\*

### El neumomediastino espontáneo: ¿indica «gravedad» o esta depende del proceso respiratorio subyacente?

Dear Editor,

With regard to the question we pose in the title, we would like to comment on spontaneous pneumomediastinum (SPM).

We have read with great interest the recently published Letter to the Editor, "A Child With Severe Pneumomediastinum and ABCA3 Gene Mutation: A Puzzling Connection".<sup>1</sup> We are interested in the relationship described between mutations of the ABCA3 gene and certain pulmonary pathologies associated with alterations in the surfactant. However, we disagree with the transcendental role that the authors attribute to SPM and its status of severity, which we believe to be inaccurate. Furthermore, we believe that what should be explored is the relationship between these genetic alterations and the pulmonary pathology that leads to the SPM, as the latter is just a consequence (presence of air in the mediastinum with no known cause).

SPM is a rare pathology in children and adults.<sup>2,3</sup> It is observed as a consequence of an increase in intra-alveolar pressure, alveolar rupture and migration of the air dissecting the peribronchial and perivascular sheaths of the pulmonary hilum, extending to the mediastinum.<sup>2-5</sup> This, at the same time, can propagate towards

the subcutaneous, endothoracic and peritoneal tissue, and even to the spinal canal. This mechanism is also known as the "Macklin effect", as Macklin was able to demonstrate this experimentally in 1937 by inflating the bronchi of cats. A few years later, Hamman made the first clinical report.<sup>4</sup>

Predisposing conditions that have been described include asthma, interstitial pulmonary diseases, COPD, bronchiectasis, lung cysts and lung cancer, among others. An increase in intra-alveolar pressure produced, for example, during vomiting, the inhalation of toxins, intense cough, physical exercise or childbirth, together with bronchopulmonary infections or the ingestion of a foreign body, may trigger an SPM.<sup>5</sup> The patient who was reported had presented acute respiratory infection associated with cough.<sup>1</sup>

SPM is considered a process with little clinical impact and a good prognosis.<sup>2-5</sup> It requires no more than oxygen therapy, analgesia, follow-up and treatment of the underlying cause, after which it completely resolves in a matter of a few days. Poor patient evolution is usually caused by the associated underlying lung disease. Tension pneumothorax and pneumopericardium are the exceptions: these should be considered severe and required specific treatment (emergency drainage).<sup>3,6</sup>

In our setting, pneumomediastinum and later subcutaneous emphysema are often a cause for panic in patients, family members and even some health-care staff, which may sometimes lead to inappropriate behavior and treatment. We therefore believe that it is important to clarify the true meaning of SPM.

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## Peripheral Polyneuropathy in a Patient With Severe Chronic Obstructive Pulmonary Disease<sup>☆</sup>

### Polineuropatía periférica en un paciente con enfermedad pulmonar obstructiva crónica grave

Dear Editor,

Peripheral neuropathy (PN) is a disease that affects the peripheral nerves due to any number of causes (hereditary, infectious, toxic, etc.). It may be classified according to the number of nerves that are affected, as mono or polyneuropathy, and according to

**Table 1**  
Electromyographic Characteristics of Peripheral Neuropathies (PN).

	Motor fibers			Sensory fibers		
	Amplitude	Duration	Conduction velocity, %	Amplitude	Duration	Conduction velocity, %
Axonal PN	Low	Normal	>70	Low	Normal	>70
Demyelinating PN	Low	Increased	<50	Low	Increased	<50

Modified from Gupta and Agarwal.<sup>5</sup>

the affection of the nerve structure, as demyelinating, axonal or mixed.<sup>1</sup>

We present an 80-year-old patient, who has been an ex-smoker for the last 30 years, with an accumulated tobacco consumption of 70 pack-years. The subject had been diagnosed with chronic obstructive pulmonary disease (COPD) with severe airflow obstruction (post-bronchodilator forced expiratory volume in 1 s [FEV<sub>1</sub>] of 40%) and had been treated with long-term home oxygen therapy at 2 l/min, 24 h/day (PaO<sub>2</sub> in stable phase 64 mmHg with oxygen and 46 mmHg without) for more than 10 years. The last COPD exacerbation was in 2010.

The patient came to our pulmonology consultation for a scheduled visit in May 2011. During this visit, he reported having a burning chest pain which made it difficult to breathe. The pain was daily and continuous, although it improved when lying down, and became more intense in the evening. The patient also complained of a tickling sensation in both feet and in the right thigh, with numbness of the internal dorsum of the left foot. Given the clinical suspicion for PN, an EMG was ordered, which revealed normal nerve conduction velocity with decreased nerve amplitude and with no positive sharp waves in either leg. The patient was sent to the neurology consultation for a complete study.

During follow-up, the patient presented neuropathic pain in both legs, and treatment was initiated with Hidroxil B1,B6,B12 and pregabalin at an increasing dosage until reaching 300 mg/day, with poor pain control. This therapy was therefore substituted with

carbamazepine, which provided better control of the symptoms, although the patient currently continues to have paresthesia in the legs and occasional pain.

The actual prevalence of PN in COPD is currently unknown, although there are several studies that indicate a frequent affection of the peripheral nervous system, which on most occasions is subclinical.<sup>2</sup>

In the physiopathology of PN in COPD, several factors have been implicated. Although for the moment we do not precisely understand its contributing role, hypoxemia could be the main factor in the development of this type of PN.<sup>3</sup> The neurophysiological characteristics are compatible with an axonal polyneuropathy with predominantly motor involvement<sup>4</sup> (Table 1) and greater affection of the legs, although it may involve any region.<sup>3</sup>

In conclusion, PN seems to be a relatively frequent affection in patients with COPD, especially in more hypoxemic patients who are treated with home oxygen, as in the case of our patient. Although the affection is usually subclinical, we should be alert to the appearance of compatible symptoms in order to reach a correct diagnosis and initiate early treatment of symptoms.

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