cyclophosphamide-topotecan regimen. Tolerance was good and metabolic response was complete, and autologous peripheral blood transplantation was performed. Complete metabolic response persisted in subsequent check-ups.

Ewing's sarcoma is the second most common malignant bone disease in children. Approximately 90% of cases occur in the second decade of life, peaking at the age of 13 years. It is extremely rare in children younger than 5 years, and occurs predominantly in boys, at a ratio of 1.5–2:1. The most common clinical presentation is pain and swelling of the affected region, and a palpable mass may be present.[1] Occasionally (more often in the case of metastatic disease) systemic manifestations occur, such as fever, asthenia, weight loss, leukocytosis, anemia, and raised erythrocyte sedimentation rate, which can lead to an erroneous diagnosis of acute osteomyelitis.[2] The most common disease sites are the diaphysis of the long bones and the pelvis (70%–75% of cases), and it has a predilection for the scapula; it occurs more rarely in the ribs and the vertebrae, but it can affect any bone. About 15%–20% of patients also have pulmonary metastasis on diagnosis, and Ewing's sarcoma can metastasize during the disease course to bone and lymph nodes.[3] Currently, standard chemotherapy for Ewing's sarcoma involves 4–6 cycles of vincristine, doxorubicin (Adriamycin) and cyclophosphamide, alternating with ifosfamide and etoposide (VDC/IE).[4]

Prognosis is determined by various parameters that make up the prognostic index: a poor prognosis is predicted by age >18 years, extraosseous site, size ≥8 cm, distant metastasis (20%), post-therapy histology I, IIA or IIB, EWS-FLI1 type 2 translocation, and raised LDH levels. Our patient presented some features for poor prognosis, such as mediastinal metastasis confirmed on cytology by EBUS and pleuro-pulmonary metastasis shown on PET-CT. Nevertheless, he was able to benefit from the correct treatment offered by an optimal multidisciplinary therapeutic strategy.[5] In the vast majority of cases, the diagnosis of Ewing's sarcoma with pulmonary involvement described in the literature is reached by transthoracic biopsy of accessible pulmonary lesions, so a successful approach to the mediastinal lesions by endobronchial ultrasound is essential. Herein lies the interest in this case: we were able to use a minimally invasive technique with high diagnostic yield to establish a differential diagnosis, ruling out other entities.

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Radiation Pneumonitis Following Hepatic Yttrium-90 Radioembolization☆

Neumonitis por radiación secundaria a radioembolización hepática con itrio-90

To the Editor,

Hepatic radioembolization is a recognized technique that uses yttrium-90 (I-90) in the treatment of hepatocellular carcinoma (HCC) and liver metastases.[1] On rare occasions (less than 1% of cases), this treatment causes radiation pneumonitis.[2,3] We report a case of lung toxicity probably caused by I-90 hepatic radioembolization and explain the underlying pathophysiology.

A 73-year-old man, former smoker with a history of alcoholic cirrhosis and unresectable HCC, consulted due to a 1-month history of progressive dyspnea on exertion. Two months before the consultation, he had undergone hepatic radioembolization with I-90 resin microspheres, with an expected lung dose of 10 Gy. Before treatment, lung scintigraphy after injection of technetium-99m-labeled albumin macroaggregates (MAA-^{99m}Tc) in the hepatic artery had revealed a lung shunt fraction of 10%. On physical examination, the patient presented tachypnea, bilateral crackles on pulmonary auscultation, and peripheral oxygen saturation of 88%, with no signs of heart failure. Chest computed tomography revealed a "crazy paving" pattern, with elevation of the right hemidiaphragm (Fig. 1A). The need for a diagnostic bronchoscopy was discussed, but refused by the patient. Given his history of radioembolization and hepatopulmonary shunt, a presumptive diagnosis of pneumonitis due to radioembolization was established. Treatment began with 20 mg/day prednisone for 2 months, and the patient's clinical condition improved with resolution of changes on tomography (Fig. 1B).

Radiation pneumonitis following hepatic radioembolization with I-90 microspheres has previously been reported. The pathophysiology of this phenomenon involves a shunt between the hepatic arterial circulation and the pulmonary circulation. This shunt is associated with certain liver tumors that lead to the development of abnormal vascular communications. The probability of this complication occurring is proportionate to the magnitude of the hepatopulmonary shunt,[2] so this ratio must be calculated during the treatment planning angiogram by administering a radioactive tracer (MAA-^{99m}Tc) to the hepatic artery and performing a quantitative lung scintigraphy. The shunt fraction is calculated as the ratio between the dose administered and the dose recorded on the lung scintigraphy. If ≥20% or a lung exposure of >30 Gy in a single dose or 50 Gy in accumulated does is predicted, radioembolization is contraindicated.[4,5]

Clinical symptoms include cough, dyspnea and fever. Tomography findings appear 1–2 months after therapy in the form of ground glass infiltrates and septal thickening. The treatment of

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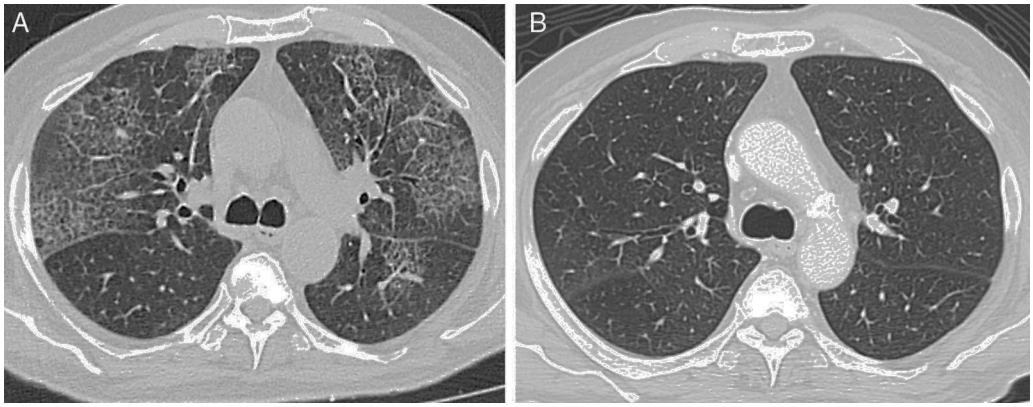

Fig. 1. (A) Chest high-resolution computed tomography showing bilateral ground glass opacities and interlobular septal thickening. (B) Chest high-resolution computed tomography after corticosteroid treatment, showing resolution of the pulmonary lesions.

choice is systemic corticosteroids.[2] Our patient presented clinical and radiological findings consistent with radiation pneumonitis. It is important that this complication is recognized, and it must be taken into account among patients undergoing hepatic radioembolization.

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Dogmas and Medical Beliefs in COPD☆

Dogmas y Creencias Médicas Acerca de la EPOC

To the Editor,

Physicians who spend their lives treating patients have always lived with the social representations of health, disease, and medication. These social representations are construct images of a natural reality and are as old as the disease itself. Based on affective and cognitive predispositions, and often inhabited by fears, beliefs, and magical and supernatural elements, the function of social representations is to make something unusual and unknown familiar to the patient.

Chronic obstructive pulmonary disease (COPD) and inhaled medications do not escape these social representation systems. The theory of social representations was first formulated by social psychologist Serge Moscovici in a paper published in France in 1961.[1] I wonder, however, if COPD is not itself also represented in doctors who usually treat this complex disease in hospitals or in primary health care, and if their representation is haunted by dogmas and medical beliefs.

In COPD, as in many other chronic diseases, non-adherence to medication is a critical issue.[2] Non-adherence to inhaled therapy in COPD has been called a high magnitude problem and a major factor of therapeutic failure, but only a limited number of studies have specifically examined adherence in patients with COPD therapy, and most research was conducted before the widespread availability of inhaled medications taken once or twice daily. In some original articles, many patient characteristics, such as the degree of bronchial obstruction or symptoms like dyspnea, are missing. However, it is well known that dyspnea, fear of dyspnea, and feelings of vulnerability contribute to better adherence to medication. Poor adherence to therapy, therefore, does not seem to make much sense from a clinical point of view, especially in these very symptomatic patients with COPD Gold stage B or D. Poor medication adherence in COPD has become a dogma that may well not correspond to reality, at least in patients with greater severity, and as such remains an open issue that merits further investigation.[3]

Another persistent dogma is the belief that once-daily medication is the best alternative for all COPD patients,[4] because it is easier to use and improves compliance. As effort dyspnea is the main symptom of COPD, the most commonly recommended schedule for bronchodilator therapy administration is early morning. Many patients, however, experience an evening aggravation or at least a fear of a nocturnal aggravation of dyspnea. Patients use inhalers because they feel relief from their dyspnea. As therapy in COPD is to some extent driven by symptoms, a twice-daily bronchodilator regimen may be more suitable in certain patient groups, such as those with exacerbating phenotypes or asthma-COPD overlap syndrome (ACOS).

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