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Involvement of Mediastinal Lymph Nodes by Ewing's Sarcoma☆

Afectación adenopática mediastínica por sarcoma de Ewing

To the Editor,

We report the case of 20-year-old man with a history of Ewing's sarcoma in the left humerus diagnosed at the age of 15, who has been in medical oncological follow-up since then. After the diagnosis, he received 5 cycles of neoadjuvant VAC/ifosfamide-VP-16, followed by radical surgery, resulting in pathological complete response according to pathology reports. The patient completed 12 adjuvant cycles of the same regimen and proceeded to regular monitoring. After 2 years free of disease, he received a diagnosis of probable pleuro-pulmonary relapse on a PET-CT, which showed hypermetabolic foci in the oblique fissure of the right lung and in the right subcarinal and hilar lymph nodes, although the latter were described as probably of a non-specific inflammatory nature, and close monitoring with imaging techniques was recommended. No known drug allergies. No toxic habits or occupational exposure. Student; no significant family history. He was referred by the medical oncology department to the respiratory medicine outpatient clinic due to the presence of mediastinal lymph nodes showing suspected tumor relapse on PET-CT. Clinical examination showed some degree of asthenia, but no other symptoms. Physical examination was normal. Clinical laboratory tests revealed completely normal serum biochemistry, complete blood count, and coagulation parameters. A computed tomography of the chest, abdomen and pelvis requested by the medical oncologist revealed a conglomerate lymph node mass in the subcarinal space, with bilateral hilar lymphadenopathies, and no other findings. Given these results, a PET-CT was requested, revealing the presence of hypermetabolic foci on the right lung and right subcarinal and pulmonary hilar lymphadenopathies, with higher metabolic intensity than the previous routine PET-CT (Fig. 1). Endobronchial ultrasound under anesthetist-controlled deep sedation was performed, revealing a rounded subcarinal lymph node mass measuring 12 mm with well-limited borders, homogeneous echostructure, and absence of central hilar structure or signs of necrosis. The lesion was aspirated 3 times for *in situ* cytopathology in the examination room, and enough material was obtained for subsequent studies. The pathological diagnosis was metastatic Ewing's sarcoma. After diagnosis, the patient received four chemotherapy cycles with the

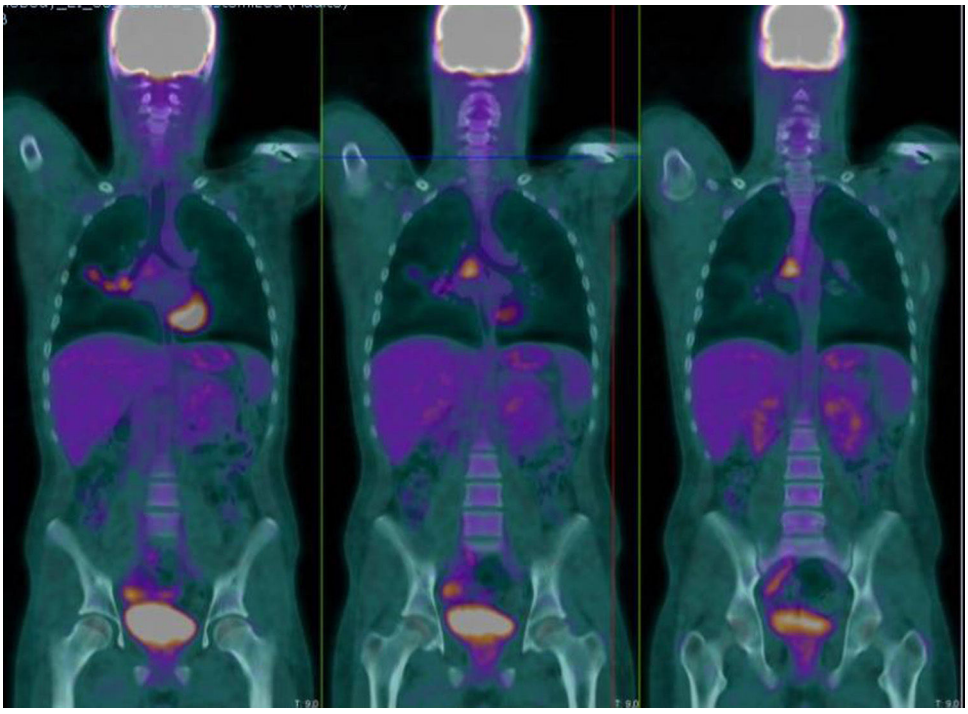

Fig. 1. Hypermetabolic foci in subcarinal and right pulmonary hilar lymphadenopathies.

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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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