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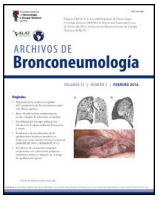

Scientific Letters

Skeletal Muscle Metastasis: An Uncommon Finding in Lung Cancer☆

Metástasis musculoesqueléticas: hallazgo infrecuente asociado al cáncer de pulmón

To the Editor,

Lung cancer (LC) is the fifth leading cause of death worldwide.[1] Unfortunately, at the time of diagnosis almost half of patients have distant metastases (most frequently in the brain, bone, liver, and adrenal glands),[2] and it is estimated that 60% of patients with early stage disease may present micrometastases.[3]

The metastatic spread of CP in the skeletal muscle is an uncommon finding (<1%), associated with poor prognosis and an average life expectancy of 6 months. Three theories have emerged to explain the low affinity of tumor cells for muscle tissue: the immunological theory (the role of humoral and cellular immunity); the metabolic theory (possible involvement of oxygen fluctuations, variable pH, and lactic acid production); and the mechanical theory (possible protective effect of muscle contractions due to high pressure and variable blood flow).[3]

The clinical presentation of musculoskeletal metastases (MSM) varies widely, from lesions that can be asymptomatic or painful and/or palpable, or can cause functional limitation in the affected area, to incidental findings in complementary imaging test.[2–4] The initial diagnostic approach in patients with suspected MSM usually begins with a computed tomography (CT) scan of the chest. Surov et al. proposed 5 radiologic patterns[4] for the characterization of MSM: type I: intramuscular mass; type II: abscess-like lesion; type III: diffuse muscle tissue infiltration; type IV: lesion with multiple calcifications, and type V: intramuscular bleeding pattern.[3] The use of additional techniques, such as magnetic resonance imaging (very useful for differentiating between MSM and primary malignant muscle lesions), and positron emission tomography with ^{18}F-fluorodeoxyglucose (greater proven sensitivity for detecting MSM and skin lesions) should also be considered.[3] However, a definitive diagnosis requires histological analysis of the lesion.

Below, we discuss 3 clinical cases of MSM in patients with LC and the different imaging diagnostic tests that were performed. The first patient was a 57-year-old woman who reported constitutional symptoms, dyspnea, and a non-painful deep adherent mass in the right flank. Chest CT revealed a right hilar lesion with multilevel mediastinal involvement, and a tumor on the left abdominal oblique muscle, classified according to its CT radiological pattern[4] as type I (Fig. 1A). The second case was an 83-year-old man with a painful tumor (necrotic cystic mass measuring 35 × 26 × 46 mm on the flexor digitorum superficialis muscle of the hand) and a 4-month history of lack of function in the right forearm. The extension study revealed 2 pulmonary masses, consistent with pulmonary adenocarcinoma (tumor classification

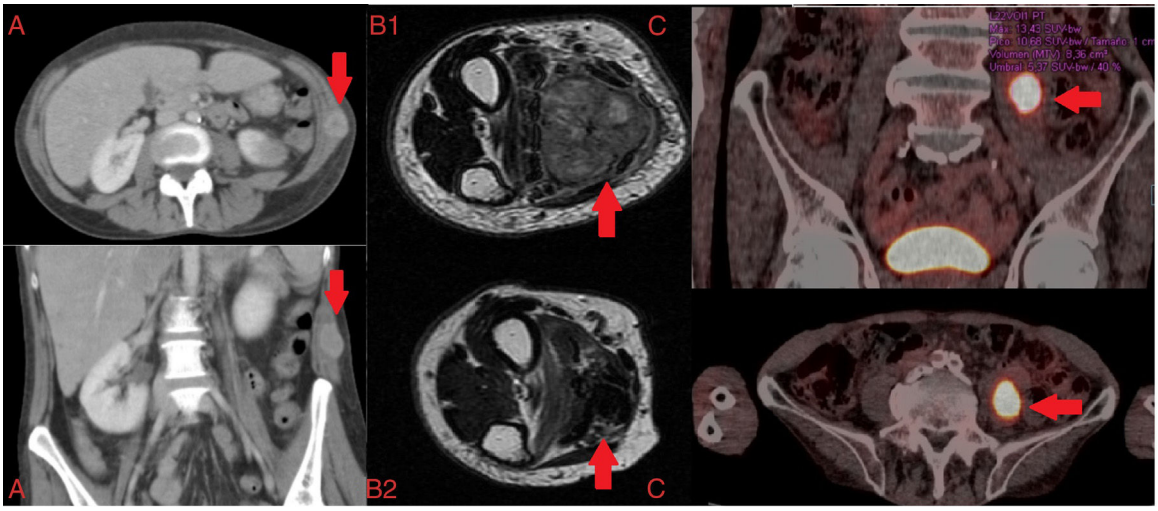

Fig. 1. (A) Axial and coronal computed tomography: musculoskeletal metastasis on the left abdominal oblique muscle. (B) Axial T2 magnetic resonance image: musculoskeletal metastasis on flexor digitorum superficialis muscle of the hand; (B1) pre-treatment; (B2) post-treatment. (C) Chest PET-CT: musculoskeletal metastasis on the left iliopsoas muscle.

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cT4NxM1b[1]). The patient received local palliative radiation therapy and chemotherapy with platinum/pemetrexed (Fig. 1B1 and B2). The third case was a 73-year-old man receiving active treatment guided by sensitivity testing results for documented *Mycobacterium xenopi* infection. Positron emission tomography showed a lesion measuring 40 × 25 mm with central cavitation in the left upper lobe (SUVmax 28.38) (Fig. 1C) and a hypermetabolic focus located in the left iliopsoas muscle with SUVmax 13.43, suggestive of MSM.

In all 3 cases, histological specimens were obtained for characterization, and the results were consistent with high grade undifferentiated tumor, striated muscle infiltrated with adenocarcinoma, and squamous carcinoma, respectively, all originating in the lung. The clinical progress of the patients differed: death 2 weeks after diagnosis, pain control, and reduced tumor size (Fig. 1B2) after targeted oncological treatment; clinical stabilization was achieved in the last 2 cases described.

Given the low prevalence of MSM, a detailed differential diagnosis that includes the more common malignant and benign entities (sarcomas, primary muscle lymphomas, and myxomas/hemangiomas) must be made. Although no clinical guidelines are available for the specific management of MSM, treatment is based on general oncological principles guided by clinical picture, site, and life expectancy, and approaches include observation, surgical excision (persistent solitary lesions after a period of remission), chemotherapy and radiation therapy (useful for pain control and for the reduction of tumor size).[2–4]

The correct identification of MSM in LC patients is essential for clinical management and prognosis. For this reason, the possible neoplastic etiology of any muscle lesion, whether symptomatic or not, detected in LC patients must be evaluated with combined radiological procedures and histological confirmation of the lesion.

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Hyponatremia in COPD: A Little Known Complication☆

La hiponatremia en la EPOC, una complicación poco conocida

To the Editor,

Chronic obstructive pulmonary disease (COPD) is a chronic inflammatory disease, and in some patients, extrapulmonary manifestations can worsen prognosis.[1]

COPD patients often have other concomitant diseases, particularly cardiovascular conditions, and it has been postulated that COPD plays a significant role in the pathogenesis of these processes.[2]

Hyponatremia developing during hospitalization for a COPD exacerbation is relatively common, and is associated with a poorer clinical course.[3] Low sodium in blood may be a sign of water retention due to other comorbidities, such as heart or kidney failure, drug treatments, adrenal insufficiency after withdrawal of corticosteroids, or syndrome of inappropriate ADH secretion (SIADH). Diseases that may present with SIADH include lung infections (pneumonia, pulmonary abscess, tuberculosis, aspergillosis), asthma, COPD, lung tumors, cystic fibrosis, and acute respiratory failure.[4] Hypoxia is associated with ADH secretion,[5] but hypercapnia is more commonly associated with this phenomenon.

Both in stable COPD and during exacerbations, hyponatremia, due to its prevalence, its impact on prognosis, and varying etiologies (which can coexist), is a challenge for the clinician and requires appropriate follow-up and treatment. Although the relationship between COPD and SIADH is often mentioned in the literature, we did not find any references to hyponatremia caused by SIADH in COPD (Medline and Pubmed searches, keywords: SIADH and COPD).

For this reason, we believe that our report of a patient with SIADH due to COPD is of interest and provides a good illustration of certain aspects of the differential diagnosis and treatment.

Our patient was an 84-year-old man, active smoker, with a history of benign prostate hypertrophy and exacerbator phenotype COPD with emphysema, and very severe obstruction (FEV1 27%), receiving treatment with siladosin, omeprazole, and glycopyrronium/indacaterol. He presented with intense dyspnea and cough with greenish expectoration. The only finding of note on physical examination was the presence of disperse rhonchi in both hemithoraxes and the absence of edema or signs of fluid overload. O_2 saturation was 89% with home oxygen therapy at 2 l per minute (lpm). Clinical laboratory tests showed microcytic anemia (hemoglobin 10 g/dl), plasma sodium 111 mEq/l (normal value [NV]: 135–155 mEq/l), plasma osmolarity 229 mOsm/l (NV: 280–300), PCR 64 mg/l (NV: 0–5), normal creatinine levels, urinary sodium 76 mEq/l (NV: 54–150), and urine osmolality 273 mOsml/l

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