

Unusual 18F-FDG PET-CT finding of paraneoplasic polymyositis in a patient with lung epidermoïd carcinoma

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[Received 28 III 2020; Accepted 8 VII 2020]

We report the case of a 67 years old male patient, followed for epidermoïd carcinoma of the right lung, and hyper-eosinophilia on peripheral blood exploration. 18F-FDG positron emission tomography-computed tomography (18F-FDG PET-CT) performed

for initial extension assessment showed, in addition to the intense hypermetabolism in the right upper pulmonary lobe related to the primary tumor and the mediastinal lymph node involvement (Fig. 1A), an unusually intense muscular hypermatbolism

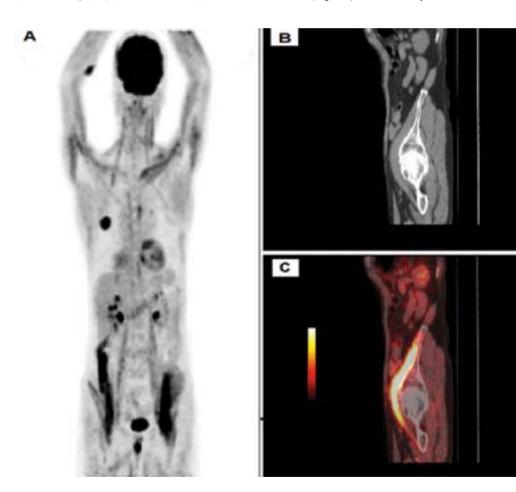


Figure 1. A. Maximum intensity projection PET image revealing intense hypermetabolism in the right upper pulmonary lobe related to the primary tumor associated to mediastinal lymph node involvement with unusual intense hypermatbolism interesting long muscles of the neck, sterno-cleidomastoid muscles, para-vertebral, iliac and ilio-psoas muscles. B. Whole body CT image and (C) 18F-FDG PET-CT fusion image in sagittal sections showing intense and symmetric hypermetabolism in ilio-psoas muscles

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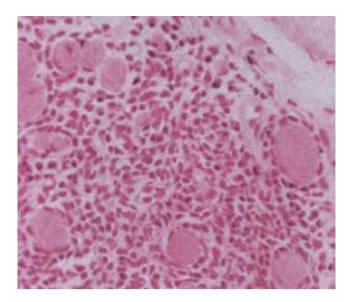


Figure 2: Biopsy of the right ilio-psoas muscle showing myofiber degeneration with predominant inflammatory cell (CD8+ T lymphocyte) compatible with polymyositis

interesting bilaterally and symmetrically long muscles of the neck, sterno-cleido-mastoid muscles, para-vertebral, iliac and ilio-psoas muscles (Fig. 1B and 1C).

Biopsy of the right ilio-psoas muscle reveals evidence of myofiber degeneration and regeneration with lymphocytes invading non-necrotic myofibers, predominant inflammatory cell (CD8+ T lymphocyte) compatible with paraneoplasic polymyositis (Fig. 2). Serum CK (creatine kinase), aldolase and sedimentation rate levels were abnormally very high.

Our patient was put under corticotherapy with good clinical, biological and radiological evolution, even on 18F-FDG PET-CT.

In our knowledge, a link between paraneoplasic polymyositis and cancer has never been clearly defined [1–2], and its clinical expression does not differ from idiopathic polymyositis [3]. Clinicopathologic diagnosis is based on histology, electromyographic data, high-level serum of muscular enzymes (creatine kinase, aldolase), and also an elevated sedimentation rate [4]. This group of patients can be treated by corticotherapy or immunomodulators, and also by physiotherapy [5].

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