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Positron emission tomography in artificial stone silicosis

Abstract

Artificial stone (AS) silicosis has emerged as a specially aggressive form of silicosis that can lead to pulmonary fibrosis and respiratory failure. The utility of positron emission tomography combined with computed tomography (PET/CT), an essential tool in oncological diseases that shows increased metabolic activity in neoplastic cells, is being evaluated in inflammatory pulmonary diseases. There are few publications about its use in silicosis, almost always referred to isolated cases in the context of studying a possible neoplasm. We present four cases of AS silicosis that underwent PET/CT and discuss its possible usefulness in this entity.

Keywords: silicosis, artificial stone, positron emission tomography

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Introduction

Artificial stone (AS) silicosis has emerged in recent years as a particularly aggressive form of silicosis related to the use of a material in kitchen and bathroom countertops that has high crystalline silica, metal and resin contents.[1,2] Lung tissue is replaced by hyaline micronodules and fibrosis; there is no specific treatment, and in the final stages, the only alternative is transplantation. Clinical, chest X-ray and functional respiratory examinations are used in evaluation and follow-up.[3] High-resolution computed tomography (HRCT) has shown greater sensitivity than chest X-ray in the diagnosis and characterization of progressive massive fibrosis.[4] Positron emission tomography combined with computed tomography (PET/CT) using 18-fluorine-labeled fluordeoxyglucose (^{18}F-FDG) is an essential tool in oncological processes; it detects increased metabolic activity in neoplastic cells, and its utility in inflammatory pulmonary diseases is being evaluated.[5] In pneumoconiosis, increased metabolism has been described in PET/CT studies, but its use has been limited to isolated cases focused on possible pulmonary neoplasms[6]. The following four cases of AS silicosis underwent ^{18}F-FDG PET/CT.

Cases series

Case 1: This case was a 42-year-old patient who was never a smoker and had worked with artificial stone for 15 years. After being referred for consultation for hilar thickening on chest X-ray, he was asymptomatic and had normal spirometry. Chest CT showed hilar lymphadenopathy and a solitary nonspecific pulmonary nodule in the right upper lobe (RUL). Mantoux and sputum cultures were negative. Four years later, he began to experience dyspnea on exertion; pulmonary functional testing and chest X-ray were normal. On control CT, hilar and mediastinal lymphadenopathy were observed; right upper lobe (RUL) showed micronodular infiltrate, and new nodules were observed in the left lower lobe (LLL). A PET/CT was requested and showed a low rate of cell proliferation in both the RUL and LLL lesions (SUVmax 3.1, SUVpeak 2.2, SUVmean 2.45, SUVmin 2.2) and in bilateral hilar mediastinal lymphadenopathy (SUVmax 3.4, SUVpeak 2.4, SUVmean 2, SUVmin 1.4), probably related to inflammation. After detecting nodule progression on a new control CT, surgical lung biopsy was performed, with a pathological diagnosis of silicosis. The patient remains clinically stable.

Case 2: This case was a 63-year-old man, a former smoker (40 packs/year), who worked until 17 years ago cutting quartz conglomerates without adequate protection. He was asymptomatic, but on CT, right hilar thickening, hilar mediastinal lymphadenopathy and bilateral millimeter nodules were observed. Although silicosis was suspected, CT images and smoking history did not rule out a neoplastic origin, so PET/CT was performed and revealed bilateral hilar mediastinal hypermetabolic lymphadenopathy (SUVmax 10, SUVpeak 7.2, SUVmean 5.7, SUVmin 4.1 in the left hilum) and metabolically negative lung micronodules. Bronchoscopy puncture of the subcarinal lymphadenopathy showed no tumor cells. Due to the persistence of lesions in control CT, surgical biopsy was performed, which revealed nodular lesions and granulomas compatible with silicosis.

Case 3: This case was a 45-year-old man, a smoker (25 packs/year) with hypertension and seronegative arthritis, who had worked for 14 years with quartz agglomerates (as a polisher and calibrator) without protection. Chest CT was performed to diagnose a fever of unknown origin with negative microbiological tests, which showed mediastinal lymphadenopathy and a micronodullary pattern in the upper fields. Transbronchial lung biopsy showed fibrous areas with anthracotic pigment, and birefringent crystals were observed with polarized light, leading to a diagnosis of simple chronic silicosis. In control CT, multiple large pulmonary nodules were observed, so PET/CT

was performed, showing hypermetabolic bilateral hilar mediastinal adenopathies (up to SUVmax 3.55, SUVpeak 2.9, SUVmean 1.9 in one right paratracheal), some of them calcified, and nonuptaking subcentimetric pulmonary nodules. These findings suggested an inflammatory origin, so evolutionary follow-up was chosen. The patient is clinically and functionally stable.

Case 4: This case was a 49-year-old man, an active smoker (20 packs/year), who is a manager of a workshop in which he has worked with quartz agglomerate for 21 years. He was diagnosed in 2012 with simple chronic silicosis with dyspnea grade 1 mMRC, occasional rib discomfort, mediastinal lymphadenopathy and multiple millimetric nodules, appearing predominantly in the upper lobes on X-ray and CT, with normal values of spirometry and CO diffusion capacity. Mycobacterial culture and tuberculin test were negative. On control CT 2 years later, a 3.3 cm conglomerate was observed in the RUL, and increased lymphadenopathy with complicated silicosis was diagnosed. With stable clinical evolution, a new CT showed an increased mass size in the RUL. PET/CT (Figure 1) revealed multiple hypermetabolic pulmonary nodules forming condensations-masses (SUVmax 8, SUVpeak 6.7, SUVmean 4.4, SUVmin 3.2), hilar and mediastinal lymphadenopathy (SUVmax 8.4, SUVpeak 6.8, SUVmean 4.9, SUVmin 3.4), and hypermetabolic lymphadenopathy in the hepatic hilum and gastrohepatic ligament (SUVmax 10.4, SUVpeak 7.7, SUVmean 5.6, SUVmin 3.95) that were not detected by CT. These lesions were considered to be related to complicated silicosis. The patient remains clinically, radiologically and functionally stable in follow-up.

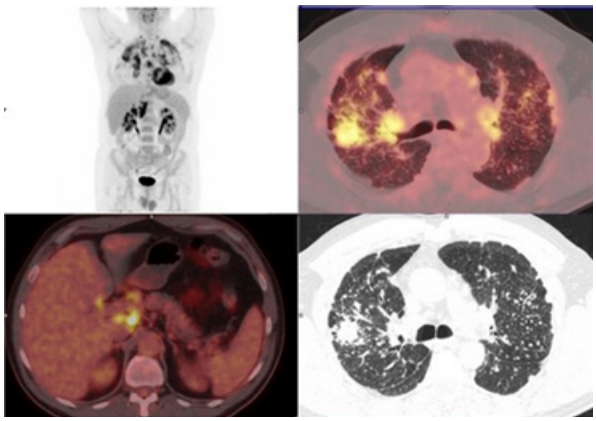

Figure 1 ^{18}F-FDG PET/CT images showing hypermetabolism in lesions imaged by CT (lower right image): hilar mediastinal lymphadenopathies and bilateral pulmonary nodule-masses are shown, and a mass in the right upper lobe is highlighted. Additionally, PET/CT identified hypermetabolic lymphadenopathy in the gastrohepatic ligament that was not detected by CT.

Discussion

Although ^{18}F-FDG PET/CT is mainly used in the evaluation of neoplastic disease, inflammatory cells also show elevated metabolic activity, which can be used to detect and monitor various lung diseases.[5] The role of ^{18}F-FDG PET/CT in AS silicosis has not been studied, and existing publications refer to individual cases in the context of studying a possible neoplasm[7,8] as occurred in the cases we present.

To the best of our knowledge, this is the first series published on the use of PET/CT in this entity. In our cases, the mean values of SUVmax, SUVmean, SUVpeak, and SUVmin were 5.55 (range 3.1-8), 4.45 (2.2-6.7), 3.42 (2.45-4.4), and 2.7 (2.2-3.2) for the lung nodules and 7.15 (3.4-10.4), 5.4 (2.4-7.7), 4.02 (1.9-5.7) and 2.85 (1.4-3.95) for lymphadenopathy, respectively. Currently, specific standardized uptake values (SUVs) have not been established to differentiate inflammation from malignancy, which may be an area for future research.

In 3 of the cases, a biopsy was performed and confirmed the diagnosis of silicosis, and in the other case, the diagnosis was based on the employment history, radiology, and exclusion of other diagnoses.[3]

Experience in inflammatory lung diseases is very limited, but some studies have noted the possible role of ^{18}F-FDG PET/CT in these processes[9,10] and even in fibrosing pathologies.[11,12]

In cases of pneumoconiosis, increased metabolic activity has been identified in areas affected by the inflammatory fibrogenic process,[13] so PET/CT could predict areas where fibrosis and permanent structural changes occur, thus enabling the early detection of evolution to complicated shapes. Likewise, although studies with a greater number of patients are necessary to evaluate the correlation between SUV parameters and inflammation in silicosis, PET/CT can represent a valuable tool to determine the extent of the inflammatory process, predict the evolution of the disease and even monitor the response of the disease to future treatments.[14]

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Conflicts of interest

The authors declare no potential conflicts of interest.

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