Positive $^{18}$F-Fluorodeoxyglucose Positron Emission Tomography/Computed Tomography 20 Years after Talc Pleurodesis

Supriya A. Bhupathy, Tung V. Huynh

Department of Medical Education, California University of Science and Medicine, Colton, California, United States

Department of Medical Imaging, Arrowhead Regional Medical Center, Colton, California, United States


Key Messages
Radiologists should consider talcoma in the differential diagnosis of patients who undergo talc pleurodesis, which induces chronic granulomatous inflammation. This is especially relevant in patients with no known risk factors for mesothelioma or pleural metastasis.

Introduction
Talc pleurodesis is often used in cases of recurrent pneumothorax and pleural effusion. Its mechanism is the induction of a chronic granulomatous reaction that induces fibrosis and seals together the visceral and parietal layers of the lung pleura. We present the case of a 63-year-old female who underwent talc pleurodesis and presented with imaging findings concerning for malignancy 20 years later. While cases of positive positron emission tomography/computed tomography (PET/CT) after talc pleurodesis have previously been reported, the lengthy timespan between pleurodesis and positive imaging findings makes this case unique.

Abstract
Talc pleurodesis, a frequently performed procedure for refractory pneumothorax or pleural effusion, induces chronic granulomatous inflammation. It can present years later with pleural thickening and markedly increased uptake on positron emission tomography/computed tomography (PET/CT), mimicking the presentation of malignancies. We present the case of a 63-year-old female with positive $^{18}$F-fluorodeoxyglucose PET/CT 20 years after talc pleurodesis. Malignancy such as mesothelioma could not initially be ruled out. CT-guided biopsy confirmed an extensive foreign-body giant-cell reaction consistent with talc-related inflammatory change. This case highlights the need for the consideration of talcoma in the differential diagnosis of patients who undergo talc pleurodesis, and is unique in the significant timespan of 20 years between pleurodesis and positive imaging findings.

Case History
A 63-year-old female presented with “needle-like” pleuritic right-sided chest pain and sharp substernal and epigastric
chest pain with associated nausea and vomiting. The patient denied shortness of breath, cough, and diaphoresis. Her past medical history was significant for cigarette smoking and for spontaneous pneumothorax 20 years prior treated with talc pleurodesis. The patient also had a history of pneumonia, hypercholesterolemia, and supraventricular tachycardia. She had no known history of prior occupational exposure to asbestos. Physical examination was unremarkable. Initial troponin was negative and EKG (electrocardiogram) showed no ST segment abnormalities.

Chest X-ray showed right-sided linear and band-like pleuroparenchymal densities (Fig. 1). Chest CT showed pleuroparenchymal scarring, emphysematous changes, and nodular pleural thickening with calcifications (Fig. 2). Repeat chest CT without contrast 6 months later showed continued evidence of pleural thickening, which was unchanged from the previous imaging (Fig. 3). Also noted was a nodular density extending through the posterolateral intercostal space into the chest-wall subcutaneous tissue.

Given the patient’s prior history of pleurodesis, the hypermetabolic pleural thickening and chest-wall nodule were suspected to be from the same etiology. Therefore, the posterolateral chest wall was selected for biopsy to minimize risk to the patient. If the results were inconclusive, pleural biopsy would have been subsequently performed. Pathologic evaluation showed an extensive foreign-body giant-cell reaction consistent with talc-related inflammatory changes. Since this pathological diagnosis of talcoma was consistent with the clinical history and imaging findings, pleural biopsy was not performed. Currently, no follow-up imaging is available.

Discussion

PET/CT is sensitive for the detection of neoplasms due to the increased neovascularity and metabolism of cancer cells. This increase in metabolic activity leads to proportionally increased glucose uptake. FDG, which is a radiopharmaceutical glucose analog, is taken up by metabolically active cells in a manner similar to glucose itself. It does not undergo the complete biochemical breakdown of glucose, and therefore becomes trapped in cells where its presence can be quantified. Although FDG uptake can be used to

Fig. 1 Chest X-ray showing right-sided linear and band-like pleuroparenchymal densities (white arrow).

Fig. 2 Initial axial (left) and coronal (right) chest CT with contrast to rule out pulmonary embolism. Pleuroparenchymal scarring, emphysematous changes, and nodular pleural thickening with calcifications are shown (white arrows). CT, computed tomography.

Fig. 3 Repeat axial chest CT without IV contrast taken 6 months after initial CT. Pleural thickening, unchanged from the previous scans, is demonstrated (white arrows). CT, computed tomography; IV, intravenous.
locate tumor cells, the uptake is nonspecific and false positives can occur in benign inflammatory processes.⁴

SUV is a semiquantitative assessment of the radiotracer uptake from a single PET image. Typically, normal tissues have SUVs ranging from 0.5 to 2.5, whereas tissues with increased metabolic activity have SUVs greater than 2.5 to 3.0.¹ The patient’s pleural lesion having an SUV of 17 raised concern for malignancy, which biopsy and pathological evaluation eventually ruled out.

While PET/CT is a useful imaging method for the detection of malignancy, instances of false positives due to talc pleurodesis have been documented.¹,²,⁵,⁶ Talc pleurodesis, which induces intense fibroblast proliferation in the parietal and visceral pleura within 3 days of the procedure, is a clinically effective method for the treatment and prevention of pneumothorax.³ In addition to forming pleural adhesions, pleurodesis causes chronic granulomatous inflammation.¹,² The thickened pleura demonstrates increased FDG uptake within 5 months of pleurodesis and continues until calcification develops.¹ The proposed mechanism for this increased uptake is the activation of white blood cells, which have enhanced levels of glucose uptake transporter (GLUT) genes as well as increased affinity to ¹⁸F-FDG via cytokines and growth factors.⁴ Persistent increased uptake of ¹⁸F-FDG resulting in a false-positive PET/CT indicates that the chronic inflammation and increased metabolic activity induced by talc pleurodesis continue decades after the procedure. Interestingly, the chest-wall component in this patient’s case is likely a talc granuloma along the path of the chest tube placed for the pleurodesis.

Because talc cannot cross into the systemic circulation of alveoli, talc particles have been shown to remain in the pleura for up to 20 years after pleurodesis.⁶ This may cause highly increased FDG uptake which does not indicate malignancy.² The appearance of plaques induced by talc pleurodesis on CT may be mistaken for other conditions such as pleural plaques from asbestososis, mesothelioma, or metastatic disease.⁶ In patients with no history of asbestos exposure and especially those with history of talc pleurodesis, radiological follow-up may be appropriate for initial management.

**Fig. 4** PET 8 months after initial presentation demonstrating significant ¹⁸F-fluorodeoxyglucose uptake with a maximum standardized uptake value of 17. Both the pleural component and chest wall component along the tract of the chest tube are emphasized (white arrows). PET, positron emission tomography.

**Fig. 5** CT and PET CT along chest tube tract. CT, computed tomography; PET, positron emission tomography.
Conflict of Interest
None declared.

References
3 Kapoor V, McCook BM, Torok FS. An introduction to PET-CT imaging. Radiographics 2004;24(02):523–543
4 Bae JM, Lee HY, Choi JY. False-positive uptake on positron emission tomography/computed tomography immediately after lung biopsy: a case report. Medicine (Baltimore) 2015;94(44): e1896