Pneumoconiosis Associated with an Esophageal Ulcer and Uptake Revealed in FDG-PET

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Abstract

A 76-year-old man with a past history of pneumoconiosis visited the Department of Gastroenterology in our hospital suffering from dysphagia. Gastroscopy revealed an esophageal ulcer on the top of a torus lesion. Chest computed tomography (CT) revealed it was caused by a swollen lymph node in the mediastinum. Squamous cell carcinoma related antigen (SCC) was elevated to 1.8 ng/ml. To rule out malignancy, we performed fluorine-18 deoxyglucose positron emission tomography (FDG-PET) which revealed a significantly increased uptake in a nodular lesion in the right upper lobe and mediastinal lymph nodes. Biopsy and cytology of the nodular lesion revealed only pneumoconiosis. We must be careful when we interpret the findings of FDG-PET in pneumoconiosis patients.

Key words: pneumoconiosis, silicosis, progressive massive fibrosis, FDG-PET, esophageal ulcer

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Introduction

Pneumoconiosis like silicosis is known to be complicated with lung cancer. The relative risk for pulmonary carcinoma among the workers who have been previously exposed to silica often exceeds 3.0 and is as high as 6.0 (1). The severe fibrosis induced by silicosis makes it difficult to distinguish lung cancer from silicotic lesion roentgenographically as well as clinically (2).

FDG-PET is useful to detect malignancy as a screening test and determine the staging of malignant disease. It is known that some benign lesions reveal an increased uptake in FDG-PET like tuberculosis, sarcoidosis, lung abscesses and so on (3). Reports of FDG-PET findings on pneumoconiosis are limited. Here, we describe a case of pneumoconiosis which was associated with an esophageal ulcer and was suspected of malignancy.

Case Report

A 76-year-old man suffering from dysphagia visited the Department of Gastroenterology in our hospital. He had smoked 20 cigarettes daily for 15 years. He had been involved in the manufacture of artificial abrasives as his occupation for 34 years, and had been diagnosed as having pneumoconiosis.

In our hospital, physical examination revealed fine cracks on auscultation. In a laboratory examination, SCC was elevated to 1.8 ng/ml. Arterial blood gases while breathing room air showed an arterial oxygen tension of 77 mmHg and arterial carbon dioxide tension of 37.2 mmHg. Pulmonary function tests showed decreases in the vital capacity (VC 2.58 L, %VC 75.8%) and the forced expiratory volume in 1 second (FEV1.0 1.64 L, FEV1.0% 68.3%).

Gastroscopy showed an esophageal ulcer on the top of a torus lesion the diameter of which was 2 cm (Fig. 1). The surface of the ulcer was covered with a white coat. The distance from the incisor to the lesion was 28 cm. Chest CT in the mediastinal field showed a swollen subcarinal lymph node (Fig. 2A) which was larger than that shown in the previous CT performed about three months previously (Fig. 2B). The esophageal torus lesion was thought to be caused by the pressure from the lymph node. CT in the lung

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field showed diffuse centrilobular small granular shadows, reticular shadows in the left lower lobe, diffuse ground grass opacity and increased thickness of the interlobular septum (Tables 2C, 2D). A 3 cm mass-like shadow was also shown in the right upper lobe (Fig. 2C). We thought malignancy should be ruled out and performed FDG-PET. It revealed a significantly increased uptake in a nodular lesion in the right upper lobe and mediastinal lymph nodes and a slightly increased uptake in the left lower lung field (Fig. 3). The highest Standardized Uptake Value (SUV) in these lesions was 5.0. Bronchoscopy showed greenish deposits on the mucosa from all the main bronchi to all the lobar bronchi. The diagnosis from the previous 1989 study had revealed that the lesion mainly contained silicon carbide (SiC) and alumina (Al₂O₃) and was due to pneumoconiosis (4). The mucosa of the right second carina was reddish. We performed curettage in the right B1a to obtain cells in the right nodular lesion revealing FDG uptake. Cytology showed macrophages with internalized crystal and coal dust (Fig. 4). A CT guided biopsy to the right nodular lesion showed destruction of normal lung tissue and hyperplasia of the collagen tissue which included crystal and coal dust (Fig. 5). Though we diagnosed the right nodular lesion as progressive massive fibrosis (PMF) and thought malignancy was ruled out, we continued to observe the patient. CT performed about four months later showed no change in the lymph nodes. Gastroscopy performed about 4 months later revealed that the esophageal ulcer had healed.

Figure 1. Gastroscopy showed an esophageal ulcer on the top of a torus lesion the diameter of which was 2 cm. The surface of the ulcer was covered with a white coat.

Figure 2. Chest CT A and B: chest CT in the mediastinal field shows the swollen subcarinal lymph node (A), and it was larger than that shown in the previous CT performed about three months before (B). C and D: CT in the lung field shows diffuse centrilobular small granular shadows, reticular shadows in the left lower lobe, diffuse ground grass opacity and increased thickness of the interlobular septum (C and D). A 3 cm mass-like shadow was also seen in the right upper lobe (C).
Discussion

At first we thought the pressure from the lymph node had caused the esophageal torus lesion and obstruction of the bloodstream had caused the ulcer. However, the ulcer had healed even though the pressure from the lymph node had not changed. We believe mechanical compression of the torus lesion easily occurred with swallowing food, and this caused the ulcer on top of the torous lesion.

The reported data on the PET appearance of pneumoconiosis have been limited. Alavi et al have described that pneumoconiosis with active fibrosis shows variable degrees of FDG uptake (5). They presented four cases of pneumoconiosis (three silicosis and one coal worker’s pneumoconiosis). All of those cases had variable degrees of increased FDG uptake at the nodular lesions and mediastinal lymph nodes. Bandoh et al reported the utility of FDG-PET in the identification of malignancy in the pneumoconiosis setting (6). He presented a case of pneumoconiosis in which CT scan showed each mass-like shadow that was PMF in the bilateral upper lobes. Only one of those mass-like shadows revealed FDG uptake and it was diagnosed as an adenocarcinoma. O’Connell and Kennedy reported a case of silicosis which revealed FDG uptake in nodular lesions, that were PMF and mediastinal lymph nodes (7). In their case, nodular lesions and mediastinal lymph nodes revealed FDG uptake, and FDG-PET was not useful in differentiating malignancy from pneumoconiosis lesions.

SUV is a popular semi-quantitative value to differentiate between malignant and benign lesions in FDG-PET. Generally the diagnosis of cancer can be suspected when lesions have a higher SUV than a cut-off set around 2.5-3.0 (8-10). However, with respect to the assessment of thoracic lesions, Demura et al confirmed that there is considerable overlap between SUV results of malignant and benign lesions, causing difficulty in correctly interpreting FDG-PET data (11). In the present case the highest SUV in the lesion was 5.0. Further accumulation of SUV results in pneumoconiosis with or without malignancy is needed and a cut-off of SUV should be reset in pneumoconiosis patients.

Some benign lesions like tuberculosis, sarcoidosis and lung abscesses have revealed FDG uptake. Some of these uptakes are thought to be related to the presence of inflammatory cells such as macrophages, as well as fibroblasts (5). In this case, the cytology of the nodular lesion revealed that there were macrophages which had internalized crystal and coal dust in the PMF and the biopsy of the nodular lesion.
revealed destruction of normal lung tissue and hyperplasia of collagen tissue which included crystal, coal dust and fibroblasts. These findings are consistent with the hypothesis by Alavi et al (5).

References


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