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Giant-cell Interstitial Pneumonia in a Gas Station Worker

Giant-cell interstitial Pneumonia (GIP) is a very uncommon respiratory disease. The majority of cases of GIP are caused by exposure to cobalt, tungsten and other hard metals. In this report, we describe GIP in a patient who worked in gas station and dealt in propane gas vessels. He presented with clinical features of chronic interstitial lung disease and underwent an open lung biopsy that showed DIP-like reaction with large numbers of intra-alveolar macrophages and numerous large, multinucleated histiocytes which were admixed with the macrophages. Analysis of lung tissue for hard metals was done. Cobalt was the main component of detected hard metals. Corticosteroid therapy was started and he recovered fully.

Key Words : *Lung disease, interstitial; Cobalt; Giant cells*

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INTRODUCTION

In 1968, Liebow classified chronic interstitial pneumonias that included usual interstitial pneumonia (UIP), desquamative interstitial pneumonia (DIP), lymphoid interstitial pneumonia (LIP), bronchiolitis obliterans interstitial pneumonia (BIP), and giant cell interstitial pneumonia (GIP) (1).

Giant cell interstitial pneumonia is a very uncommon disease. In GIP, the interstitium and alveolar walls are thickened and infiltrated by mononuclear cells, and the diagnostic feature is the presence of large numbers of giant cells filling the air space (2). Recent studies suggest that the majority of cases of GIP are caused by exposure to cobalt, tungsten carbide as components of hard metals (3). It is, therefore, considered as a form of pneumoconiosis and currently the term of hard metal lung disease or hard-metal pneumoconiosis is preferred (2, 3). We report a case of giant cell interstitial pneumonia diagnosed by open lung biopsy and mineralogical analysis method.

CASE REPORT

A 51-year-old man presented with a 6-month history of gradually aggravated dyspnea. He felt dyspnea after going up 3 flights of stairs at his first visit to hospital. He had smoked 20 cigarettes daily for 40 years. He worked in gas station as an owner and dealt in propane gas vessels. As a hobby, he made plates for stones by sandpapering wood. Physical examination revealed bilateral fine inspiratory crackles. Finger clubbing was not observed. Pulmonary function test showed moderate obstructive impairment. The forced vital capacity (FVC) was 3.25 L (72% of predicted), the forced expiratory volume 1 second (FEV1) was 1.7 L (53% of predicted) and the FEV1/FVC was 58%. Posteroanterior chest radiography on admission revealed bilateral patchy subpleural opacities in both middle and lower lung zones (Fig. 1). Initial high resolution computed tomography (HRCT) showed patchy areas of subpleural consolidation and ground glass attenuation, subpleural linear opacities, interlobular and intralobular septal thickenings (Fig. 2).

The results of hematologic, serum chemical profile, and urine test were normal. All serologic tests (even for collagen disease) were negative. Thoracoscopy-guided lung biopsy was performed at the right middle lobe. The tissue was examined histologically and mineralogically. The main finding is a DIP-like reaction with large numbers of intra-alveolar macrophages. Additionally, numerous large, multinucleated histiocytes that had ingested inflammatory cells were admixed with the macrophages (Fig. 3). There were no evidence of inclusion bodies of viral disease or granulomatous lesions. Analysis for inorganic particles was carried out. The analysis method was

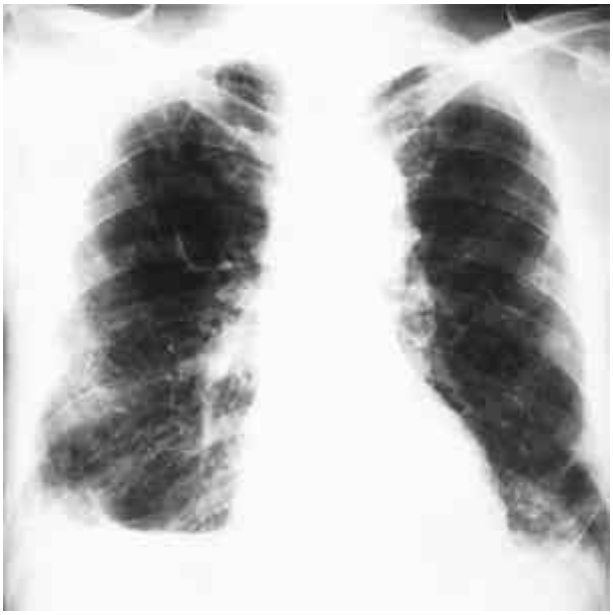

Fig. 1. Initial posteroanterior chest radiography on admission revealed bilateral patchy subpleural opacities in both middle and lower lung zones.

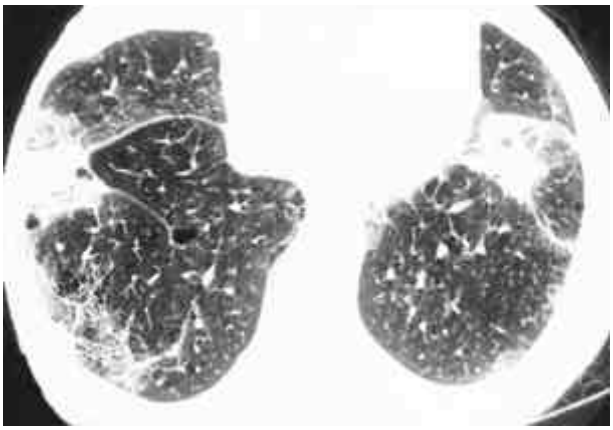

Fig. 2. Initial high resolution computed tomography (HRCT) showed patchy areas of subpleural consolidation and ground glass attenuation, subpleural linear opacities, interlobular and intralobular septal thickenings.

atomic absorption spectrophotometry (Varian Zeeman method) (4). 1.14 ± 0.04 $\mu g/g$ of cobalt and small amount of Pb, Cr, Ni were detected. The patient was started on 60 mg of prednisolone a day and remained separated from work with symptomatic improvement. After two month, follow-up chest radiography and HRCT showed that parenchymal lesions decreased in their extent.

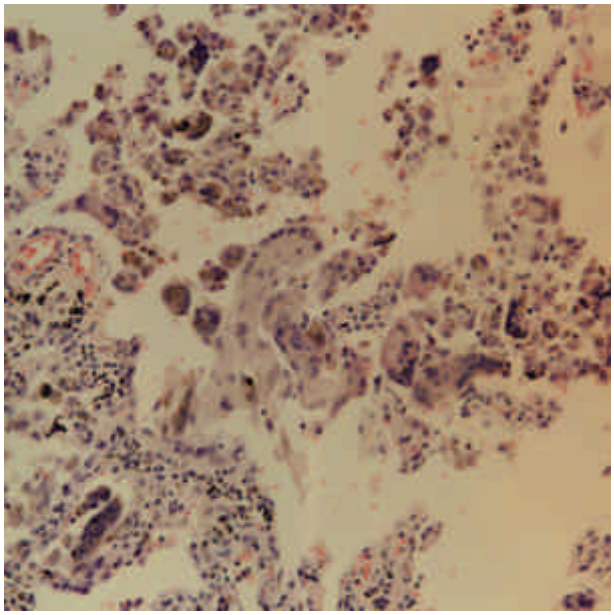

Fig. 3. The main finding is a DIP-like reaction with large numbers of intra-alveolar macrophages. Numerous large, multinucleated histiocytes that have ingested inflammatory cells are admixed with the macrophages (H&E, ×200).

DISCUSSION

GIP is a very rare chronic interstitial pneumonia described originally by Liebow (1). It is almost exclusively in workers exposed to hard metal, an alloy of tungsten carbide and cobalt (3). It therefore should be considered a form of pneumoconiosis. Hard metal has 95% of the hardness of diamond, used for cutting, drilling, and grinding (5). Metallic particles less than 2.0 μm in diameter are readily inhaled (6). The diagnostic criteria of hard metal lung disease are a history of exposure to hard-metal dust, characteristic clinical features, radiologic findings of interstitial lung disease, histologic findings of interstitial fibrosis, DIP-like pattern and identification of the constituents of hard metal in the lung tissue (3). In this case, there was no definite history of exposure to hard metal such as grinding or shaping. The patient had only dealt with barrels of propane gas and had ground wood with sandpaper for making plates for stones as his hobby. The cause-and-effect relationship is not certain. There have been some reported cases of GIP in housewives who had no history of exposure to hard metal (7,8).

The similar histologic feature is found in giant cell pneumonia secondary to infection (9-11). The giant cell pneumonia secondary to infection are characterized by inclusion body, positive serology for infection, positive viral culture, immunohistochemical findings, and necrotizing granulomatous response (3, 9-11). However, in

GIP necrotizing granulomatous response is not found.

The clinical features of GIP are nonspecific. Patients complain of shortness of breath, cough and dyspnea on exertion (3, 12-14). Radiologic features are micronodular, reticulonodular, or diffuse interstitial infiltrates. Pulmonary physiologic studies usually show restrictive impairment. Generally, if exposure is avoided, patients improve in symptoms and physiologic study, although residual lesions tend to persist (3).

Chest radiographic findings of GIP range from normal to bilateral reticular infiltrates with end-stage interstitial fibrosis. Usual radiographic findings consist of a diffuse micronodular and reticular pattern, sometimes with lymph node enlargement.

The useful methods for detection of hard metals in tissue are microprobe analysis and X-ray diffraction method (3, 12, 15). Cobalt was found in a small portion of the cases because of its solubility, but tungsten carbide stays longer in tissue, and serve as a marker of exposure (15).

Hard metal related lung disease other than GIP found in the hard-metal industry are – occupational asthma, hypersensitivity lung disease and the other forms of chronic interstitial lung disease (14, 16, 17).

As a conclusion, physicians should pay attention to and find out the causative factors in the patients with respiratory symptoms, in workers exposed to hard metals.

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