Upper Lobe-Dominant Pulmonary Fibrosis Showing Deposits of Hard Metal Component in the Fibrotic Lesions

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Abstract

We present a 54-year-old man employed in the field of hard metal manufacturing who complained of pro-
gressive dyspnea and weight loss. His chest radiograph showed bilateral fibrosis predominantly distributed in
the upper lobes with bilateral pleural effusions, and a strong reduction in lung volume. Lung histopathology
showed apical cap-like fibrosis but no giant cell interstitial pneumonia. Electron probe microanalysis detected
tungsten deposits in the fibrotic region: we therefore considered this to be a case of hard metal disease. Hard
metal disease should be considered as one possibility in the differential diagnosis of upper lobe-dominant
pulmonary fibrosis.

Key words: electron probe microanalysis, hard metal, pneumoconiosis, pulmonary fibrosis, upper lobe

Introduction

Interstitial lung disorder is thought to be a major disease resulting from hard metal inhalation. A typical pathologic
cchange of hard metal-induced interstitial lung disorder is gi-

ant cell interstitial pneumonia (GIP) (1). Here we present a
case of hard metal disease in which the patient presented
with a unique chest radiograph and unique pathologic find-
ings.

Case Report

A 54-year-old man presented with a 6-month history of
progressive exertional dyspnea and weight loss. He had
never smoked nor been exposed to asbestos. He had worked
as a quality controller of completed materials in a hard
metal manufacturing business for 21 years. He did not wear
a mask at work. On hospital admission, his physical exami-
nation revealed a heart rate of 85 beats/min, blood pressure
of 130/70 mmHg, respiratory rate of 20 breaths/min, and
body temperature of 36.0°C. Auscultation of the lung
showed a decrease in respiratory sounds over both lung
fields, but fine crackles were inaudible. The patient’s fingers
were not clubbed. Laboratory studies revealed only a CRP
level of 0.66 mg/dL (normal <0.20) and a KL-6 level of 648
U/mL (normal <500). A chest radiograph revealed the bilat-
eral opacities to be predominantly distributed in the upper
lobes, with a strong reduction in lung volume (Fig. 1A). It
also revealed the presence of bilateral pleural effusions and
right-sided pneumothorax. A high-resolution CT scan re-
vealed mild pleural thickening and parenchymal reticulation
without honeycombing (Fig. 1B). The results of the pulmo-
nary function tests were as follows: a vital capacity (VC) of
1.12L (30.1% predicted), forced expiratory volume in one
second (FEV₁) of 0.99L (29.8% predicted) and percent
forced expiratory volume in one second (FEV₁%) of 89.2%,
total lung capacity (TLC) of 2.56L (45.1% predicted), and

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carbon monoxide diffusion capacity (DLco) of the lung of 2.07 mL/min/mmHg (9.2 %predicted). The tests showed severe reduction both VC and DLco. The arterial blood gas levels of breathed room air were as follows: pH 7.39, PaO2 88.6 mmHg, and PaCO2 47.4 mmHg. A bronchoalveolar lavage (BAL) from the right S' revealed that alveolar macrophages contained anthracotic materials, despite the patient never having smoked actively or passively. Analysis of the pleural effusion demonstrated the fluid to be exudative: total protein 4.4 g/dL, LDH 233 IU/L, and white blood cell count 4,000/μL. The differential of the white blood cell count measured 67.7% lymphocytes, 15.7% monocytes/macrophages, and 9.3% neutrophils. The Gram stain and culture findings of the BAL fluids and pleural effusion were negative for bacteria, mycobacteria, and fungi. The lung biopsy specimen of the left S1+2 obtained by the video-assisted thoracoscopic surgery (VATS) showed nonspecific scarred fibrosis similar to that seen in the apical cap (Fig. 1C). Pulmonary fibrosis was composed of airspace fibrosis with collapse and an increased elastic framework. The visceral pleura were fibrously thickened. The findings of GIP and usual interstitial pneumonia were not detected in the specimen. Electron probe microanalysis of the lung biopsy specimen detected tungsten deposits (Fig. 2). The patient was given corticosteroid therapy (methylprednisolone, 1 g IV for 3 days). However, the treatment was not effective and oral prednisolone was not administered because the patient did not wish to continue the corticosteroid therapy. Although the pneumothorax improved without thoracic drainage, the patient was hospitalized because of the progressive dyspnea. Three months after admission, he was transferred to a hospital near his home.
Discussion

In this report, we present a case of progressive pulmonary fibrosis occurring in a man who worked in the hard metal manufacturing business. Hard metal is an alloy of tungsten carbide in a matrix of cobalt, to which small quantities of other metals, including titanium, nickel, and chromium, are added. The main occupational respiratory hazard associated with employment in this industry results from the effects of excessive inhalation of airborne dusts and aerosols containing cobalt. Hard metal-induced interstitial pneumonia, which is among the pulmonary complications occurring as a result of hard metal inhalation, is considered to be synonymous with “hard metal disease.” The histopathologic hallmark of this disease is GIP containing multinucleated giant cells (1).

In the present case, a lung specimen obtained by VATS revealed the scarred fibrosis that resembles the finding of “apical cap” described by Yousem (2), but no finding of GIP. Nevertheless, we made a diagnosis of hard metal disease because this case fulfilled the following criteria (3): 1) The patient had a history of working as a hard metal worker. 2) The patient showed symptoms of dyspnea and body weight loss. 3) Pulmonary fibrosis could be detected on the chest radiograph. 4) The histopathology demonstrated a finding of pulmonary fibrosis. 5) Deposits of tungsten were detected in the lung tissue.

In this study, we detected the deposits of hard metal components by using electron probe microanalyzers (EPMAs). EPMAs can detect the distribution of elements, such as tungsten, in the biopsy specimen at the light microscopic level (4). Deposits of cobalt could not be detected in this case, probably owing to its water solubility. In another report of hard metal pneumoconiosis, cobalt was not detected in the lung tissues (5).

Amitani et al reported 13 cases of unique pulmonary fibrosis and proposed “idiopathic pulmonary upper lobe fibrosis” as a distinct clinicopathologic disease entity (6). The characteristic features of these cases include pulmonary opacities predominately distributed in the upper lobes with a high prevalence of pneumothorax. On the other hand, Frankel et al described 5 cases of unique pluroparenchymal lung disease and termed “idiopathic pleuroparenchymal fibroelastosis” as a novel clinicopathologic entity (7). These cases are characterized by a clinical presentation suggestive of a chronic interstitial pneumonia, marked pleural and parenchymal radiographic involvement with an upper lobe predominance. These features resemble those in the present case. The relationship between these diseases and hard metal inhalation should be elucidated.

The patient worked in the office of a manufacturing business so his exposure to hard metal would have been low. No one else in the business showed evidence of hard metal disease, suggesting that the patient possesses a genetic susceptibility to hard metal. Potolicchio et al demonstrated that the susceptibility to hard metal disease is strongly associated with the presence of glutamate 69 in the HLA-DP beta chain (8). Interestingly, the same amino acid variation was detected in the present case.

In summary, we have presented an atypical case of hard metal disease in which the chest radiograph showed upper lobe-dominant pulmonary fibrosis. Treatment with corticosteroids was neither clinically nor radiologically effective. We should consider the possibility of hard metal inhalation in patients showing upper lobe-dominant pulmonary fibrosis so as to avoid the risk of further exposure.

References


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