Case Reports

Hard metal lung disease and pneumothorax

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This case report describes a case of spontaneous pneumothorax in a metal grinder that failed to resolve with medical management. A lung biopsy taken during the subsequent thoracotomy revealed changes that were characteristic of giant cell interstitial pneumonia, as seen in hard metal disease. Pneumothorax has been described in many forms of interstitial lung disease but not in this type of occupational disease. Awareness of this possible association can result in: (1) the removal of an affected worker from his adverse work environment before advanced disease develops; and (2) the safe control of the work environment, to the advantage of other similarly exposed workers.

Introduction

Hard metal is an uncommon cause of interstitial lung disease. Some types of interstitial lung disease are characteristically associated with an increased risk of spontaneous pneumothorax. The present case report describes a case of hard metal lung disease that presented with an acute pneumothorax.

Case Report

A 26-year-old man who had never smoked presented in early 1993 with dyspnoea of sudden onset. He denied previous cough, breathlessness, weight loss or ill health. He had no finger clubbing, but inspiratory crackles were audible at the bases of his lungs. His chest radiograph showed widespread group glass shadowing in both lungs, predominantly in the upper and middle zones, with a large left pneumothorax.

He had an intercostal tube inserted into his left pleural space with underwater drainage. Due to a persistent air leak and failure of the pneumothorax to resolve, he had a thoracotomy 2 weeks later. His lungs appeared abnormal; a biopsy was taken from the lingula, and a parietal pleurectomy was undertaken. Post-operatively, his lung was slow to expand, and the intercostal tube was removed 2 weeks later.

The patient had been employed for 8 yr in a small workshop, grinding tungsten carbide tips with diamonds. He undertook both dry and wet (using a soluble oil and water mix) grinding, but wore no mask. All the grinding machines had local exhaust ventilation. Investigations at the work place demonstrated that these exhaust ventilation systems were ineffective. Urine cobalt levels of surveyed workers were normal, but background cobalt levels in the work room were high, and the personal exposure measurement of one worker (0.16 mg m⁻²) exceeded the current recommended maximum exposure limit (0.1 mg m⁻²).

No other processes took place in this workshop, where 12 machines were in operation.

With hindsight, the present patient appreciated that he had been breathless for some time, which he had attributed to a general lack of fitness.

Lung biopsy (Plate 1) showed chronic interstitial pneumonitis, with marked ‘desquamative’ features, lymphocytic and plasma cell infiltration,
PLATE 1. Lung biopsy, × 260. An alveolus lined by plump (and occasional multi-nucleate) epithelial cells and containing multi-nucleate intra-alveolar macrophages. The adjacent interstitium contains a dense infiltrate of lymphocytes and plasma cells. Many of the alveolar macrophages had multi-nucleate giant cells and there were also multi-nucleate alveolar cells. The changes observed were characteristic of giant cell interstitial pneumonia (GIP), seen in hard metal disease.

Lung function tests showed a restrictive ventilatory defect with lung volumes and transfer factor reduced to about one-third of the patient's predicted values. A computed tomographic scan of the thorax showed widespread reticulo-nodular shadowing, most pronounced in the mid zones, with linear shadowing in the upper zones, and several cystic air spaces at the extreme apices. The patient has been treated with oral corticosteroids for 18 months. He is now free of symptoms and abnormal physical signs. The abnormalities on his chest radiograph have largely resolved. His lung function tests have shown a small improvement in lung volumes, but no improvement (Table 1) in gas transfer ($K\text{CO}$).

Investigation of the patient's place of work identified no disease in the other six employees. The ventilation systems were improved, and regular health surveillance was introduced.

**Discussion**

Hard metal is an alloy of tungsten carbide in a matrix of cobalt, to which small quantities of titanium, nickel, chromium, vanadium and molybdenum may be added. It is widely used in industry where extreme hardness and stability at high temperatures is required, as in drill bits or cutting tools, artillery shells and armour plating.

Respiratory disease in hard metal workers was first described in 1940 (1). It has been reported in those inhaling hard metal dust during its manufacture, and in those grinding hard metal tools. Hard metal can cause both interstitial lung disease (hard metal disease) and asthma (2,3). Asthma in hard metal workers is provoked by inhalation of cobalt but not tungsten carbide, suggesting it develops as the outcome of a hypersensitivity response to cobalt (4).

Interstitial lung disease in hard metal workers is characterized by the presence of giant cell transformation of alveolar macrophages and Type 2 epithelial cells (4), the characteristic features of giant cell interstitial pneumonia (GIP) (5). Following the description of GIP

<table>
<thead>
<tr>
<th>Months after presentation</th>
<th>FVC ($l$)</th>
<th>TLC ($l$)</th>
<th>$T_l\text{CO}$ (m mol min$^{-1}$ kPa$^{-1}$)</th>
<th>$K\text{CO}$ (m mol min$^{-1}$ kPa$^{-1}$ l$^{-1}$)</th>
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</thead>
<tbody>
<tr>
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<td>1:87</td>
<td>2:8</td>
<td>3:63</td>
<td>1:60</td>
</tr>
<tr>
<td>8*</td>
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<td>3:0</td>
<td>4:00</td>
<td>1:54</td>
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<td>4:01</td>
<td>3:87</td>
<td>1:34</td>
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<td>2:77</td>
<td>4:33</td>
<td>5:69</td>
<td>1:52</td>
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<tr>
<td>Predicted value</td>
<td>5:23</td>
<td>7:13</td>
<td>12:0</td>
<td>2:14</td>
</tr>
</tbody>
</table>

*Measurements at Royal Brompton Hospital, remainder at Castle Hill Hospital. FVC, forced vital capacity; TLC, total lung capacity; $T_l\text{CO}$, carbon monoxide transfer factor; $K\text{CO}$, carbon monoxide transfer coefficient.
in hard metal workers, a similar histological pattern of response was identified in diamond polishers using diamond grinding wheels with cobalt as a binder (6), suggesting cobalt as the cause of the GIP as well as asthma in hard metal workers.

Acute GIP can resolve with avoidance of exposure and can also be improved with oral steroids (4,7). Chronic GIP resembles cryptogenic fibrosing alveolitis clinically, functionally and radiographically, and is less improved by avoidance of exposure and treatment with oral steroids.

Several uncommon types of interstitial lung disease, such as eosinophilic granuloma, lymphangioleiomyomatosis and tuberous sclerosis, are characteristically associated with a increased risk of spontaneous pneumothorax. It can also be a rare complication of more common types of interstitial lung disease, such as sarcoidosis, and rheumatoid arthritis. The present case report describes a case of hard metal disease which presented with a spontaneous pneumothorax. No previous similar cases have been found in a literature search.

The possibility of underlying respiratory disease should be considered in all cases of apparently spontaneous primary pneumothorax. This can lead to the early diagnosis of interstitial lung disease before lung fibrosis has become established, and at a stage of the disease which may be amenable to corticosteroid treatment, or in pneumoconiosis by moving the patient away from exposure to the cause of disease.

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References