Pulmonary fibrosis in a tool sharpener

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INTRODUCTION

This article presents and discusses a case of 36-year-old tool grinder who presented with cough and dyspnoea. A chest radiograph showed nodular shadows in the lung fields. An open lung biopsy confirmed fibrosis and its cause.

DISCUSSION

Tungsten carbide has been manufactured and widely used in industry since the 1920s. The manufacture of hard metals involves mixing metal ingredients and binders in a powder form. Hard metal alloys, typically tungsten carbide, titanium carbide and cobalt, are formed under high pressure, heated in furnaces and shaped into the final products. These are typically cutting or drilling tools as these have a hardness similar to that of diamond (Scherrer and Maillard, 1982). The products undergo precision grinding and honing for final shaping. It is thought that the principal diseases associated with exposure in this industry result from the toxic effects of cobalt (Van der Eekhout et al, 1989).

The histopathological hallmarks of this disease are giant cell interstitial pneumonia and the presence of multinucleated giant cells (Austenfeld and Colby, 1989). There are two different types of pulmonary reactions to tungsten carbide (Coates and Watson, 1971). A milder form presents like bronchospasm or bronchitis, induced by exposure and relieved by removal of the subject from the source. In the severe form, patients develop varying degree pulmonary fibrosis. Radiographic changes range from nodular, reticular and linear densities to very dense coalescent areas. In a large survey, some patients were noted to develop obstructive lung defects, even non-smokers. Lung function tests showed an association of worsening with duration of exposure (Sprince et al, 1984).

A study of 12 tungsten carbide workers with diffuse interstitial lung disease found a mean duration of exposure of 12.6 years before symptoms appeared (Coates and Watson, 1971). This patient has had small amounts of exposure for many years, resulting in interstitial fibrosis. HM

CASE REPORT

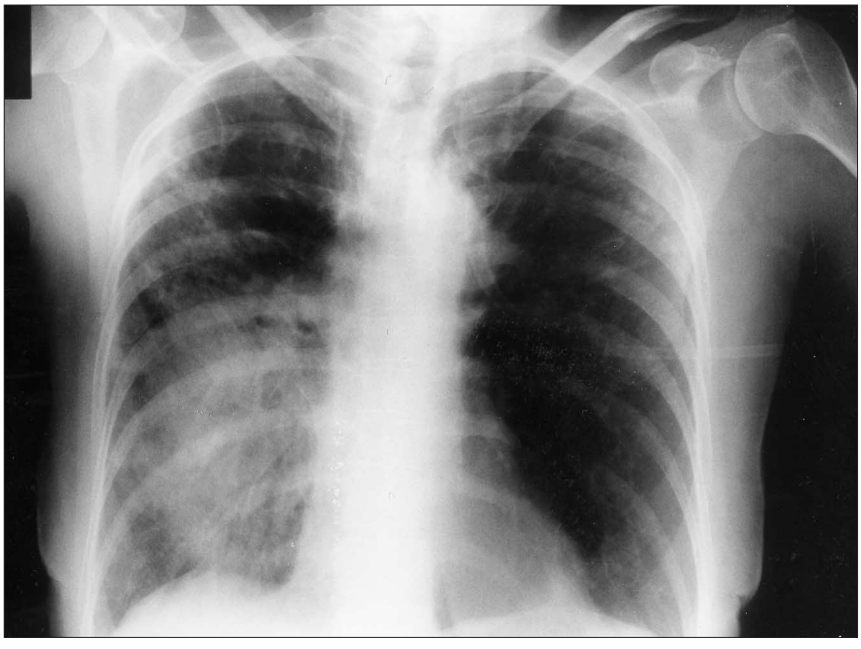
A 36-year-old man who had been employed as a tool grinder and sharpener for the last 18 years presented with dyspnoea, dry cough and left-sided pleuritic chest pain of 6 weeks duration. He recalled an episode of a very small amount of haemoptosis. He described his work environment as dusty and under ventilated. His work involved sharpening tungsten carbide parts.

He had no previous history of tuberculosis, asthma or chronic bronchitis. He had no pets. He never worked in coal mines and was a chronic smoker. Physical examination showed no evidence of clubbing, cyanosis or lymphadenopathy.

Auscultation of chest revealed bilateral widespread fine end inspiratory crackles. Bilateral diffuse micronodular shadows were seen on the chest X-ray (*Figure 1*). Lung function tests showed a mild restrictive pattern (forced expiratory volume in 1 second = 3.8 litres, forced vital capacity = 4.94 litres). He underwent open lung biopsy. Histology confirmed that there were multiple foci of interstitial fibrosis associated with inflammatory infiltrate, small collection of macrophages with black particles and occasional giant cells. Mineral analysis of lung tissue revealed high levels of tungsten. These findings supported the diagnosis of heavy metal pneumoconiosis.

The patient was started on intravenous steroids, resulting in remarkable improvement in the general condition and exercise tolerance. He was discharged on oral steroids and he reported good performance at the local gymnasium, which he was unable to do before admission.

Figure 1. Chest X-ray demonstrating bilateral micronodular shadowing.



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