CASE REPORT

Giant Cell Pneumonitis Induced by Cobalt

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ABSTRACT

Cobalt alone and in combination with tungsten carbide known as hard metal is capable of causing lung damage. This may vary from development of pulmonary oedema to asthma and fibrosing alveolitis. We report a case of giant cell interstitial pneumonitis caused by exposure to cobalt dust which was not identified as the etiological agent initially and hence led to progression of the disease. The patient subsequently improved following cessation of exposure and treatment with oral corticosteriods, thereby stressing the importance of occupational history in all cases of interstitial pulmonary fibrosis.

Key words: *Hard metal disease, Interstitial pneumonitis, Occupational hazard.*

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INTRODUCTION

Respiratory diseases in the hard metal industry have been recognised as a probable occupational hazard since 1940. Hard metal is an alloy of tungsten carbide, cobalt and occasionally other metals like titanium, tantalum, chromium, nickel[1]. Of these, cobalt whether free or in alloy form, is allergenic and cytotoxic and capable of provoking release of a fibrogenic agent from marophages. Three types of disorder are attributed to cobalt exposure: acute (in the form of asthma), subacute (fibrosing alveolitis) and chronic, in the form of diffuse interstitial fibrosis of the giant cell variety[2].

The existence of a link between giant cell interstitial pneumonitis (GIP) and cobalt exposure was published by Abraham and Spragg[3]. There have been only 50 reported cases of cobalt related GIP and probably only three bonafide cases of GIP without hard metal exposure[4]. We report a case of giant cell interstitial pneumonitis caused by exposure to cobalt dust.

CASE REPORT

A 27-year-old male, worked as a press operator and was exposed to cobalt metal powder for a period of three years. He complained of dry cough and progressive exertional dyspnoea since last two years. There was a history of low grade fever for the same duration but no chest pain, haemoptysis or wheezing.

A high resolution computerized tomography (HRCT) scan had been done one year ago and a diagnosis of interstitial lung disease (ILD) was made at another institute. He was treated with low dose oral corticosteroids with some relief in his symptoms. However, the treating physician did not suspect occupational lung disease and hence the patient was allowed to continue work in the same place with recurrence of symptoms after discontinuation of oral corticosteroids.

On physical examination, he had grade 1 clubbing and bilateral fine end inspiratory crackles at the lung bases. His chest radiograph and HRCT

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(Figures 1&2) showed changes of alveolitis: ground glass attenuation of both lung parenchymal peripheries in the lower lobes. The opacities had increased in extent as compared to the previous CT scan. The haematological and biochemical investigations were normal. Rheumatoid factor (RA), and antinuclear antibody (ANA) were negative. Arterial blood gas analysis revealed pH-7.376, PaO_2-75.2 mm Hg, $PaCO_2$-44.2 mm Hg, HCO_3-25.3. Initial pulmonary function tests showed a forced vital capacity (FVC) of 0.95L (Predicted 3.6L, 26% predicted), and forced expiratory volume in one second (FEV_1) of 0.84L (predicted 3.0L, 28% predicted). He was subjected to open lung biopsy which showed a widening of interstitial septae with lymphocytic infiltration and few eosinophils and alveolar lumina filled with desquamated cells along with few giant cells. Some of the alveoli showed edema fluid rich in fibrin. Some foci showed smooth muscle metaplasia. Changes were consistent with giant cell interstitial pneumonitis (Figure 3).

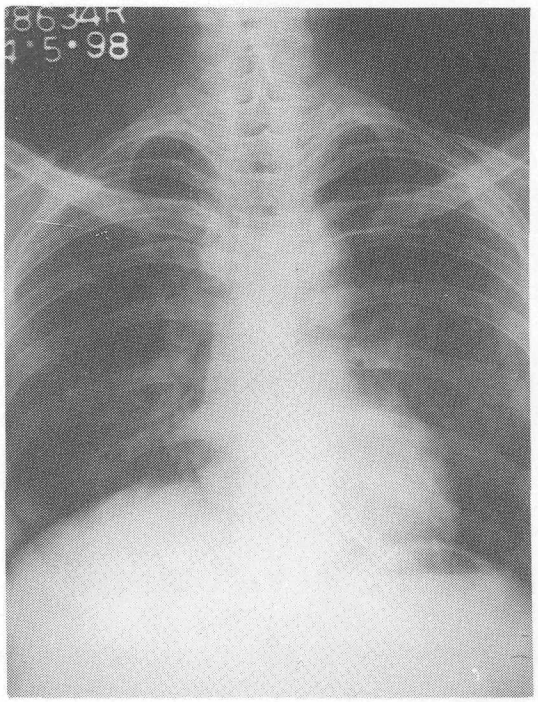

Figure 1. Chest radiograph reveals reticular pattern in the mid and lower zones.

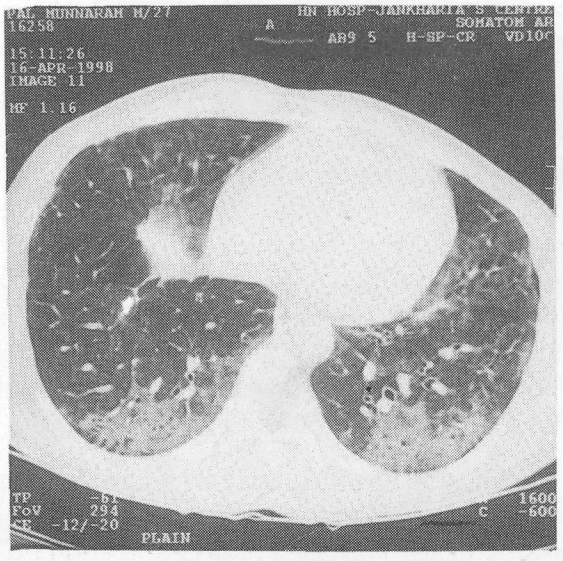

Figure 2. High resolution CT scan shows presence of gorund glass pattern in subpleural region in the lower lobes.

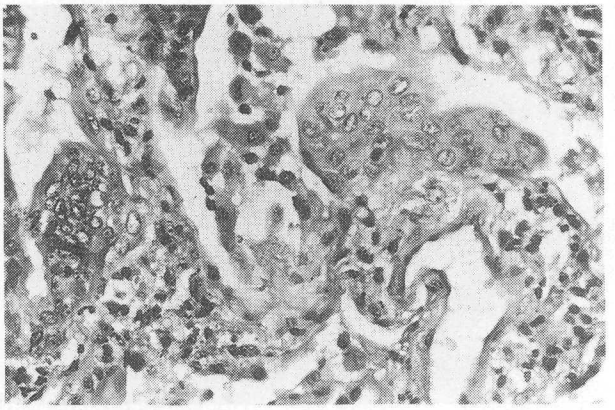

Figure 3. Open lung biopsy reveals multinucleate giant cells lining thickened alveolar septae with mononuclear inflammatory cell infiltrate (H & E X 100).

A diagnosis of cobalt induced giant cell interstitial pneumonitis was made and treatment with oral corticosteroids in a dose of 1 mg/kg initiated. The patient was also asked to change his work and was shifted to other department in the same firm. He showed marked symptomatic improvement and his pulmonary functions after three months of tapering had improved to a FVC of 1.25L (predicted 3.6L, 35% predicted) and FEV_1 of 1.15L (predicted 3.0L, 38% predicted)

DISCUSSION

Cobalt is a silvery blue-white metal with magnetic properties which is obtained when copper is extracted from its ore. Extra fine grade powder is used in the manufacture of hard metal. The

particles are rod shaped with a mean diameter of 1.4 micrometer. In the production of hard metal, tungsten carbide is produced by blending and beating tungsten and carbon, to which cobalt in 3-25% is added. The powdered metal is pressed and fused at approximately 1000°C and the product is finally heated upto 1500°C (sintering). Lung disease appears to be more common in wet (coolant using) areas where cobalt occurs in ionised form but in lower concentrations than in dry areas where it is non-ionised[1,2].

Cobalt is toxic to lung and possibly exerts a synergistic effect when combined with tungsten carbide or titanium. Cobalt is highly soluble in biological fluids and after inhalation there is an initial phase of low level excretion[1]. In ionised form it combines readily with proteins and aminoacids and thus could conceivably act as a hapten capable of promoting immunological reactions[2]. Respiratory effects include development of pulmonary edema following exposure to high concentrations, asthma (occupational) following exposure of 6-18 months, and fibrosing alveolitis. Fibrosing alveolitis is more common among workers in occupations involving metal dust and 10-13% of cases of fibrosing alveolitis may be attributable to metal dust exposure[5].

Subacute fibrosing alveolitis develops within a year or less or after a few years of exposure. Complete resolution occurs in some cases when exposure ceases but is incomplete and may occur on re-exposure. Chronic disease occurs in 2-25 years after exposure, usually in excess of 10 years[1]. In our patient, the disease occurred within one year of exposure to cobalt dust. On histological examination the alveolar walls are thickened with lymphocytes, plasma cells, macrophages, with so called desquamative picture and presence of gaint cell transformation in unusual feature. Hence the term giant cell interstitial pneumonia[6]. Bronchoalveolar lavage reveals presence of multinucleated cells and elemental analysis of lung tissue for cobalt can be done.

A detailed occupational history is essential for identifying the cause of interstitial lung disease, and all hard metal workers should have regular lung functions tests and radiological examinations. The level of respirable dust should be reduced to a minimum by efficient exhaust ventilation of high velocity, low volume type. The alveolitis stage responds to corticosteroids while chronic disease is not amenable to treatment. Further exposure should be prevented once the diagnosis is made.

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