Hard metal pneumoconiosis with spontaneous bilateral pneumothorax*

Pneumoconiose por exposição a metal duro com pneumotórax bilateral espontâneo

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Abstract

Hard metal pneumoconiosis, first described in 1964, is a diffuse disease caused by the inhalation of cobalt particles. The disease can manifest as occupational asthma, interstitial disease or allergic alveolitis. We report the case of a young male, working as a tool sharpener, who presented with dry cough and progressive dyspnea for one year, as well as with spontaneous bilateral pneumothorax at admission. The diagnosis was confirmed by open lung biopsy.

Keywords: Lung diseases, interstitial; Pneumoconiosis; Pneumothorax.

Resumo

A pneumoconiose por metal duro, descrita pela primeira vez em 1964, é uma doença difusa causada por inalação de partículas de cobalto. A doença pode se manifestar de três formas diferentes: asma ocupacional, doença intersticial e alveolite alérgica. Relata-se um caso de um jovem do sexo masculino, afiador de ferramentas, com quadro de tosse seca e dispnéia progressiva há um ano, apresentando-se à admissão com pneumotórax espontâneo bilateral. O diagnóstico foi confirmado através de biópsia pulmonar a céu aberto.

Descritores: Doenças pulmonares intersticiais; Pneumoconiose; Pneumotórax.

Introduction

Hard metal pneumoconiosis is a diffuse disease caused by the inhalation of cobalt particles.[1] Hard metal is an alloy of cobalt and tungsten carbonate, occasionally added with small quantities of other metals, such as titanium, tantalum, nickel and chrome. The remaining metals other than cobalt are considered inert and do not cause lung injury.[2]

Exposure to hard metal can cause occupational asthma and diffuse lung disease.[2] Occupations related to the manufacture and refining of hard metal, as well as those related to the use of cobalt-coated disks to polish diamonds and sharpen tools, are associated with these diseases.[3]

In this report, we present the case of a tool sharpener with diffuse lung disease who secondarily developed spontaneous bilateral pneumothorax.

Case report

A 27-year-old male patient with dry cough and progressive dyspnea for 1 year sought treatment in the Pulmonology Clinic of the Federal University of Goiás *Hospital das Clínicas* in the city of Goiânia, Brazil. He reported worsening dyspnea on minimal exertion in the last two weeks and weight loss (10 kg) in the last six months. The patient denied having a history of

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lung disease. He had a two-year history of occasional smoking and had discontinued the habit 5 years prior. The patient had been a hard metal tool sharpener (saws and knives) for eight years. He worked eight hours a day, did not wear individual protective equipment and used a synthetic diamond grinding wheel in an enclosed space, measuring approximately 5.5 m^2, where he was the only worker.

Physical examination revealed weight loss, dyspnea, an SpO_2 of 86% on room air and diffusely decreased vocal fremitus in both hemithoraces, primarily in the apices, where tympanism could be observed. No other alterations were found.

A chest X-ray performed one month prior to admission revealed bilateral pneumothorax (Figure 1). An HRCT scan showed bilateral pneumothorax, paraseptal emphysema in the apices, some interposed bullae, diffuse ground-glass opacities, perilymphatic nodules, mediastinal lymph nodes and a calcified halo (Figure 2).

The patient was submitted to bilateral closed chest tube drainage with complete re-expansion of the left lung and partial re-expansion of the right lung, where a bronchopleural fistula with medium output persisted. During the same hospital stay, the fistula was sutured and the patient underwent right parietal pleurectomy, left chemical pleurodesis with tetracycline and open lung biopsy (middle lobe).

The anatomopathological findings revealed interstitial pulmonary fibrosis, predominantly peribronchial and periseptal, containing numerous giant cells, alveolar histiocytes and areas of honeycombing (Figure 3). Examination of the pleural tissue showed granulation tissue proliferation, fibrosis, lymphohistioplasmocytic infiltrate, containing some interposed neutrophils, and deposition of fibrin on the surface.

Based on the patient occupational history, as well as on imaging studies and on the anatomopathological examination, a diagnosis of hard metal pneumoconiosis was made.

The patient was started on systemic corticosteroid therapy, initially with methylprednisolone and subsequently with prednisone.

During the follow-up period, the patient had repeated episodes of bilateral pneumothorax and required additional drainage and pleurodesis. He was discharged on prednisone (10 mg/day) and home oxygen therapy.

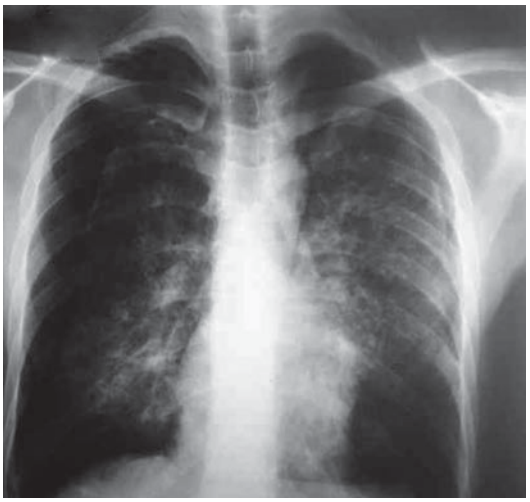

Figure 1 - Chest X-ray revealing diffuse, heterogeneous opacities and bilateral pneumothorax. "BJP" e "080208" por "020808".

A pulmonary function test performed six months after discharge revealed severe restrictive lung disease (VC = 34% of predicted), with negative bronchodilator test, and a moderate reduction in DLCO (60% of predicted).

At this writing, one year after diagnosis, the patient continued on prednisone (10 mg/day), had occasional dry cough, had dyspnea on moderate exertion and presented an SpO_2 of 93% at rest.

Discussion

Hard metal pneumoconiosis was first described in 1969 by Liebow & Carrington, being included among idiopathic interstitial pneumonias. In 2001, the American Thoracic

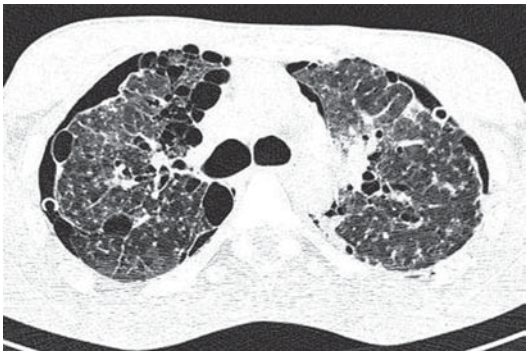

Figure 2 - Chest HRCT slice (without contrast) showing bilateral pneumothorax, bullae, ground-glass opacities and perilymphatic nodules, as well as the presence of bilateral chest tubes.

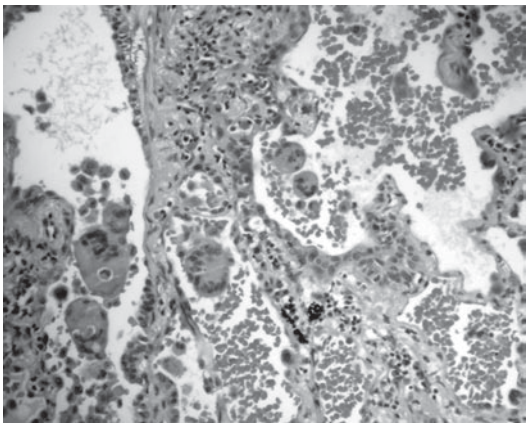

Figure 3 - Histopathological examination revealing multinucleated giant cells as well as peribronchial and periseptal fibrosis.

Society and the European Respiratory Society recognized this entity as pneumoconiosis caused by the inhalation of cobalt or of an alloy of cobalt and other hard metals, and, therefore, it was excluded from the original classification.[4] This entity is also known as hard metal lung, giant cell pneumonitis and cobalt lung.

Hard metal is an alloy of tungsten carbonate, cobalt and small quantities of other metals, such as titanium and tantalum.[3,4] Since it is hard and resistant to high temperatures, it is used for polishing diamonds, galvanizing armaments, drilling oil wells and cutting tools.[5,6]

Although the hard metal alloy has other components, cobalt is the one responsible for inducing lung disease.[5] One group of authors stated that interstitial disease develops only when exposure to cobalt occurs in association with exposure to tungsten carbonate or diamond powder.[1] A small number of exposed workers develop the disease, usually after ten to twelve years of exposure, although the disease can occur early.[4]

Currently, the scientific community recognizes three pathological entities related to the inhalation of hard metal dust: occupational asthma; interstitial lung disease, which occurs in two varieties (non-specific form and giant cell intra-alveolar pneumonitis); and allergic alveolitis or hypersensitivity pneumonitis. Allergic alveolitis or hypersensitivity pneumonitis occurs in the acute phase of exposure, being considered an early (potentially reversible) inflammatory phase of fibrosis, although it can evolve to fibrosis as a result of long-term exposure.[2,4,5,7,8]

The clinical profile of giant cell pneumonia includes dyspnea on exertion, hypoxemia, cough, weight loss, fatigue, wheezing and rales at the end of inhalation.[8] With the progression of fibrosis, there can be digital clubbing, cyanosis, signs of right heart failure, pulmonary hypertension and cor pulmonale.[6,8] Pneumothorax has been reported in one case.[9]

Pulmonary function can show a restrictive pattern, with reduced lung volumes. Occasionally, an obstructive pattern can be found.[4,6] The DLCO tends to decrease in the same proportion as TLC.[7,8]

Chest X-rays can be normal or reveal mild nodular or reticulonodular infiltrates. Being more sensitive in the detection of alterations, HRCT can reveal ground-glass opacities and irregular linear opacities, as well as parenchymal distortion, traction bronchiectasis, bronchiolectasis and reticulation.[4,5] Other isolated findings include large peripheral cysts, which can represent honeycombing, as well as centrilobular nodules and emphysema.[8]

If there is no contraindication, bronchial lavage and transbronchial biopsy should be performed to confirm the diagnosis. If the transbronchial biopsy sample is insufficient or the examination is inconclusive, open lung biopsy should be performed. If there are multinucleated giant cells in the bronchoalveolar lavage fluid accompanied by a clinical and radiological profile consistent with the disease, biopsy in not indicated.[7]

The histopathological pattern comprises mononuclear cell infiltrate, predominantly in regions of the peribronchiolar interstitium, and accumulation of macrophages and multinucleated giant cells within the alveoli,[7,8] with alveolar wall thickening.[7] With the progression of the disease, honeycombing can be observed. Being highly soluble in tissues, cobalt is rarely found in biopsy specimens.[1]

The treatment of hard metal lung disease involves complete removal from exposure and high-dose corticosteroid therapy. When there already is extensive pulmonary fibrosis, there is no significant response to treatment.

A peculiarity of the case reported here was the presence of bilateral recurrent pneumothorax, which required multiple interventions. This is an unusual presentation,[9] which made the management of the case difficult. In addi-

tion, the case was aggravated by the use of corticosteroids, and this might have affected the results of pleurodesis.

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