Hard metal lung disease in an oil industry worker*

Doença pulmonar por metal duro em trabalhador da indústria petrolífera

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Abstract

Hard metal lung disease, which manifests as giant cell interstitial pneumonia, is caused by exposure to hard metal dust. We report the case of an oil industry worker diagnosed with hard metal lung disease. The diagnosis was based on the clinical, radiological and anatomopathological analysis, as well as on pulmonary function testing.

Keywords: Alloys/adverse effects; Cobalt; Tungsten; Occupational exposure; Lung diseases, interstitial.

Resumo

A doença pulmonar por metal duro é uma pneumonia intersticial por células gigantes relacionada com a exposição à poeira composta por metais duros. Neste artigo é relatado o caso de um profissional da indústria petrolífera, diagnosticado com doença pulmonar por metal duro com base na documentação clínica, radiológica, funcional pulmonar e anatomopatológica.

Descritores: Ligas/efeitos adversos; Cobalto; Tungstênio; Exposição ocupacional; Doenças pulmonares intersticiais.

Introduction

Lung diseases secondary to dust inhalation and presenting an immune response mechanism involving hypersensitivity, as is the case of hard metal lung disease, tend to be designated based on histopathological aspects or pathophysiological mechanisms, despite meeting some of the criteria of pneumoconioses. Inhalation of hard metal dust is a type of occupational exposure that is rarely investigated. There is an inflammatory process that can evolve progressively, causing significant impairment of respiratory function and gas exchange, as well as considerable morbidity and mortality. We report a case of lung disease due to exposure to hard metals. The diagnosis was based on the investigation of the occupational exposure, together with the review of the clinical, radiological and histopathological findings.

Case report

A 50-year-old male patient sought treatment at our facility. He had worked as an industrial plumber on an oil platform for 9 years and had been admitted to another hospital in 1996 for the investigation of dyspnea on exertion, mild productive cough, evening fever and arthralgia (all of which had started 5 years before that admission and had worsened 3 months prior). The patient reported having been treated for pneumonia on four occasions, with temporary improvement, had a history of systemic arterial hypertension and was a former smoker (20 pack-years) who had not smoked for 5 years. A chest X-ray showed bilateral interstitial infiltrate (Figure 1a); spirometry revealed signs of restrictive lung disease (FEV_1 = 1.56 L [46.9% of predicted]; FVC = 2.01 L [51.2% of predicted]; FEV_1/FVC = 77.9% [91.6% of predicted]); during

* Study carried out at the Interstitial Diseases Outpatient Clinic of the Walter Cantídio University Hospital, Federal University of Ceará, Fortaleza, Brazil.

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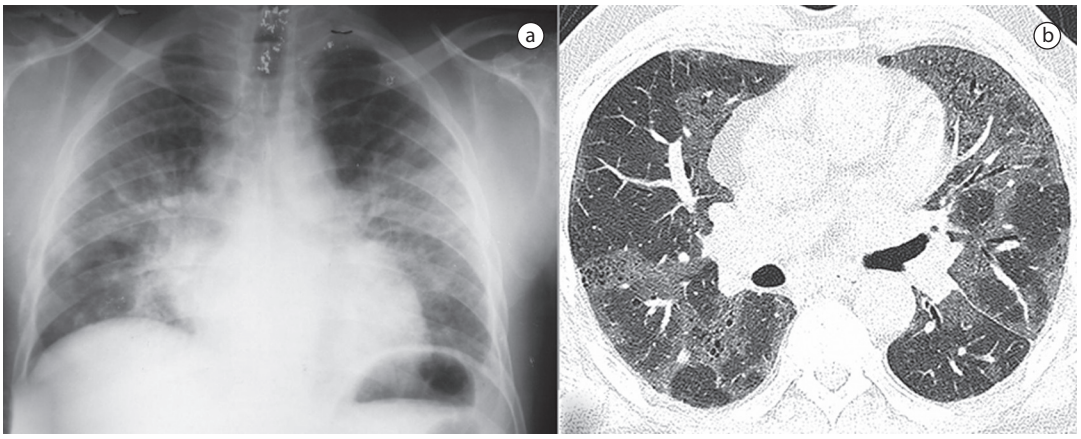

Figure 1 - In a), chest X-ray revealing bilateral interstitial infiltrate (1996); in b), HRCT showing areas of ground-glass attenuation, traction bronchiectasis, and interposed cystic images (2006).

the six-minute walk test, the patient presented a significant reduction in SpO_2, which decreased from 96% to 87%; serology for HIV, hepatitis B and hepatitis C, as well as venereal disease research laboratory test serology, all yielded negative results; FAN = 1:160; and testing for anti-extractable nuclear antigen was negative. The patient was submitted to an open lung biopsy, which revealed desquamative interstitial pneumonia (Figure 2a), and started treatment for idiopathic interstitial pneumonia with prednisone at a dose of 60 mg/day for three months, the dose being tapered. The patient discontinued exposure after the onset of symptoms. He had no medical evaluations between 1998 and 2005, when he sought treatment at the interstitial disease outpatient clinic of our hospital. He was not using any medication, and his pulmonary function had worsened. Physical examination revealed fine crackles in the lower and posterior thirds of both hemithoraces. An HRCT scan showed ground-glass attenuation and weak interposed honeycombing (Figure 1b). The requested review of the slide of the lung biopsy detected the presence of giant cells (Figure 2b). When asked about occupational exposure, the patient revealed having worked as an oil industry plumber, during which time he often assembled tubing using tools (grinders, sanders) containing disks made of hard metal. He reported that those disks became blunt with use and needed to be sharpened, and that his

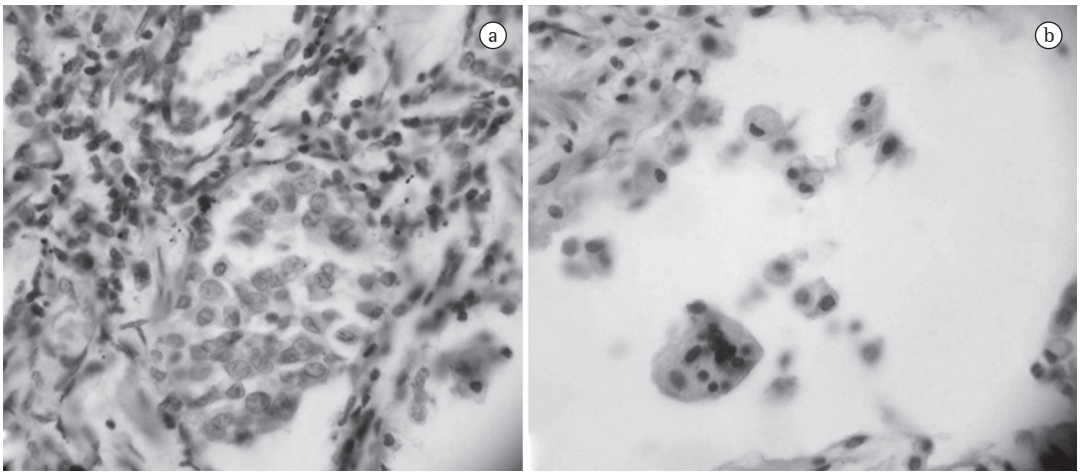

Figure 2 - In a), anatomopathological examination showing macrophages in the alveolar lumen, consistent with desquamative pneumonia (H&E; magnification, ×400); in b), anatomopathological examination revealing the presence of giant cells (H&E; magnification, ×400).

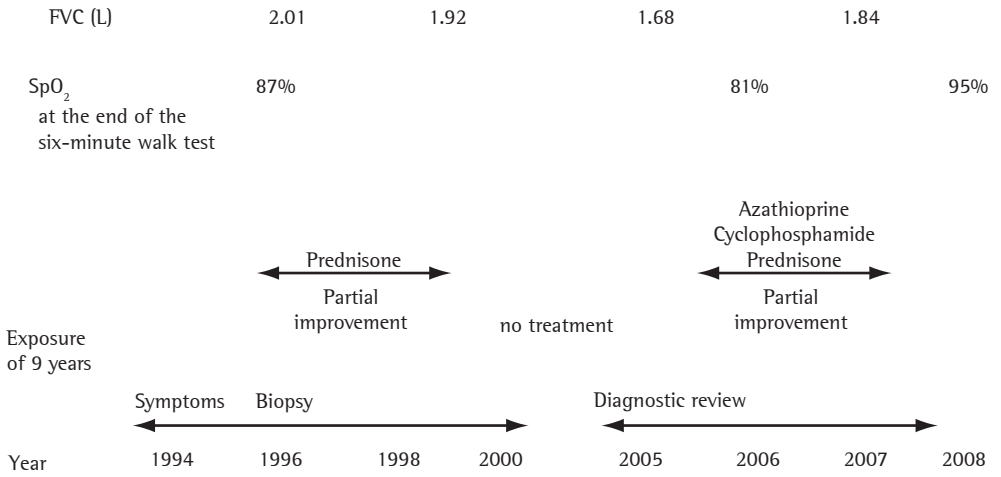

Figure 3 - Clinical and functional evolution of the patient in terms of diagnostic and therapeutic procedures.

only protection was a visor. He performed this activity, almost continuously, at least five days a week from 1982 to 1991, when the symptoms began. At that time, a chest X-ray requested by the company physician revealed pulmonary infiltrate. The patient was instructed to take a leave from work and seek specialized help. Based on these data, the patient was diagnosed with hard metal lung disease. Treatment with immunosuppressants (azathioprine and pulses of cyclophosphamide) and prednisone was initiated, resulting in clinical improvement as well as in radiological and functional stability. Figure 3 summarizes the clinical and functional evolution of the patient, correlating it with the diagnostic and therapeutic procedures to which the patient was submitted over the course of his evolution.

Discussion

In 1968, Liebow developed an original histological classification for chronic interstitial pneumonias, classifying them as usual interstitial pneumonia, desquamative interstitial pneumonia, lymphoid interstitial pneumonia, bronchiolitis obliterans with interstitial pneumonia, and giant cell interstitial pneumonia.[1] Studies suggest that most cases of giant cell pneumonia are caused by exposure to components of hard metals, and giant cell pneumonia was excluded from the classification of idiopathic interstitial pneumonia proposed by the American Thoracic Society/European Respiratory Society, according to which "hard metal lung disease" is the term currently preferred.[1-3]

Hard metals consist primarily of cobalt, tungsten and a lower percentage of other metals. Since hard metals are 95% as hard as diamond,[1] are highly resistant to compression, as well as to bluntness due to oxidation, and maintain good thermal stability, they are used in the manufacture of industrial cutting, grinding and drilling tools, such as the saws, pliers, drills and grindstones used in the aeronautical, automobile, electrical, oil and mining industries.[1,2,4] The manufacture and use of these tools result in occupational exposure to hard metal dust.[4] Other professionals who are at high risk include dental technicians and diamond cutters.[4,5] The occurrence of giant cell pneumonia in diamond cutters who use abrasives containing only cobalt corroborates the participation of cobalt in causing hard metal lung disease.[5,6] Conversely, one group of authors have considered the possibility that there is a synergy between cobalt and tungsten.[2]

The process by which giant cells are formed and the exact role played by cobalt remain unknown.[6] Giant cell pneumonia does not meet the classical definition of pneumoconiosis, which is typically a deposition disease with a dose-response relationship. Due to its similar clinical presentation, hypersensitivity pneumonia should be included in the differential diagnosis, although only hard metal lung disease includes the presence of giant cells in its the pathology[6] and, unlike hypersensitivity pneumonia, has an inorganic cause. Currently, the scientific community recognizes at least four distinct pathological

entities related to the inhalation of hard metal dust: typical giant cell pneumonia; desquamative interstitial pneumonia without giant cells; acute allergic alveolitis with the possibility of chronicity; and asthma.[5]

The symptoms of hard metal lung disease are nonspecific: dry cough; progressive dyspnea on exertion; digital clubbing/cyanosis; chest pain; fatigue; and weight loss.[7,8] Chest X-rays reveal a diffuse reticulomicronodular pattern, occasionally accompanied by lymph node enlargement, and, in the advanced stage of the disease, can show small cystic formations.[9] An HRCT scan can show areas of bilateral ground-glass opacity, areas of consolidation, extensive areas of reticular hyperattenuation, and traction bronchiectasis—findings that indicate fibrosis.[9,10] Other findings include honeycombing, centrilobular nodules, and emphysema.[7] Pulmonary function testing reveals restrictive lung disease.[8] The histopathological pattern can show desquamative interstitial pneumonia and giant cell interstitial pneumonia, with or without bronchiolitis obliterans, and a variable degree of interstitial fibrosis.[11]

The treatment depends on the stage of the disease. In the acute and subacute phases, in which patients have symptoms similar to those of asthma and hypersensitivity pneumonia, the management includes avoiding future exposure, by means of leaves from work, together with the use of bronchodilators and inhaled corticosteroids. As the fibrotic process begins, systemic corticosteroids can be used.[2] The complete resolution of the pathological process can occur after the end of exposure and the early initiation of corticosteroid therapy.[12] In the case reported here, systemic corticosteroids and immunosuppressants were used, resulting in clinical improvement. This is the second case reported in Brazil. In 1992, the case of a 29-year-old patient exposed to hard metal dust in a metallurgical plant in Brazil was presented at the International Conference on Occupational Lung Diseases.[12]

In summary, the diagnosis of hard metal lung disease requires a high degree of clinical suspicion, in addition to radiological and histopathological findings consistent with the disease. Systematically asking individuals about occupational exposure to hard metal dust constitutes a fundamental element of the etiologic investigation. Careful anamnesis makes an idiopathic cause less likely, with significant clinical repercussions, and occupational leaves of absence are an important therapeutic tool, being associated with a better prognosis.

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