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Clinical Note

Hard Metal Interstitial Lung Disease

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ABSTRACT

Hard metal lung disease is an unusual disease which can occur in individuals exposed to hard metals. Clinically, the condition resembles hypersensitivity pneumonitis depending mainly on individual susceptibility, which eventually progresses to pulmonary fibrosis. We present two patients with pulmonary fibrosis, who were actually diagnosed after an exhaustive anamnesis and examination of the tissue by scanning microscope to discard hard metals. The evaluation of wedge biopsies by scanning electronic microscope can be very helpful in those cases without a specific diagnosis.

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Enfermedad pulmonar intersticial por metales duros

Palabras clave:
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RESUMEN

La neumonitis por metales duros es una enfermedad infrecuente que aqueja a personas expuestas a polvo de metales duros. La presentación clínica es la de una neumonitis por hipersensibilidad y los pacientes pueden evolucionar a una fibrosis pulmonar, dependiendo probablemente de su susceptibilidad. Presentamos 2 casos correspondientes a 2 mujeres con fibrosis pulmonar. La anamnesis exhaustiva y el estudio del tejido de la biopsia para descartar la presencia de metales duros permitieron el diagnóstico de esta enfermedad. Se discute la utilidad de la biopsia para su valoración histológica y su estudio posterior con el microscopio electrónico de barrido.

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Introduction

Hard metal lung disease is an unusual occupational disease which can occur in workers from industries that manufacture or manipulate tools composed of hard metals. Hard metal is a generic term used to describe a group of artificial materials composed, above all, of tungsten carbide and small amounts of cobalt, with the addition of other metals such as titanium, molybdenum, tantalum, vanadium or chrome. They are known as hard metals due to their high heat resistance and hardness.[1,2] The prevalence of the disease among workers in contact with these metals varies from 0.7 to 13%.[2-4] Its clinical presentation is similar to hypersensitivity pneumonitis, in its subacute form with later development into pulmonary fibrosis. The exact pathogenesis is not clear, it is different to conventional pneumoconiosis, given that the disease depends on the susceptibility of the individual more than the accumulative effect of exposure.[1,5,6,8]

Confirmation of cobalt exposure must be assessed in the patient's workplace context. Contact can be determined through the measurement of cobalt concentrations in the urine, although, due to its fast elimination, only high levels are detected in individuals that have been in contact on the days prior to the analysis. Furthermore, there is no direct relationship between the degree of exposure to cobalt and the appearance of the disease.[1] Given that the radiological and functional alterations are not specific, they could be mistakenly labelled as an idiopathic disease unless bronchoalveolar lavage (BAL) or surgical biopsy to observe giant cannibalistic or emperipolesis

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cells[1,9] are performed or the lung tissue studied with scanning electron microscope (SEM) to detect particles, such as was carried out in the two cases presented.

Clinical Observation

The first case corresponds with a 37 year old female, smoker of 20 packets/year, with no noteworthy pathological background. She worked as a clerk and, for the last 7 years as a jewellery polisher for one year in the company and then in her home, activity which she left two years before consultation. She presented pain in the back and lower side of mechanical nature on applying finger pressure, no fever, cough without expectoration. Auscultation showed bibasal "velcro" crackles and in the exploration, 6 year acropachies. The functional respiratory tests showed a restrictive pattern with decrease in carbon monoxide diffusion (D_LCO), 53% forced vital capacity (FVC); 63% maximum expiration volume in the first second (FEV_1); 85% FEV_1% and 37% D_LCO. The high resolution thorax tomography showed a reticular pattern in bases, with extensive areas of honeycomb lung and bronchiectasis by traction (fig. 1). The bronchoalveolar lavage displayed 42% macrophages, 7% lymphocytes, 39% polymorphonuclears and 12% eosinophils, without evidence of giant cells with emperipolesis. The surgical biopsy showed a pattern of fibrosing nonspecific interstitial pneumonia, abundant multinucleated giant cells with emperipolesis in the airspace (fig. 2). Faced with the difficulty of finding a nexus between the patient's disease and the pneumonitis by hard metals, a paraffin block was studied with SEM. Detected in the tissue were, among other metals, gold chrome plating and niobium, although, due to the chronicity of the lesion and the known solubility of the cobalt that favours its disappearance from the tissue, the latter was not detected. Treatment with corticoids was established without improvement.

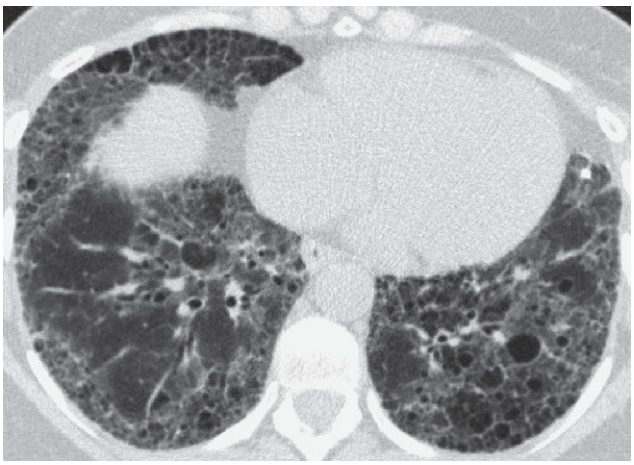

Figure 1. High resolution computerised tomography (HRCT) of patient 1 thorax that shows a peripheral micro cystic pattern that predominates in the bases, with honeycomb and cystic images as well as traction bronchiectasis.

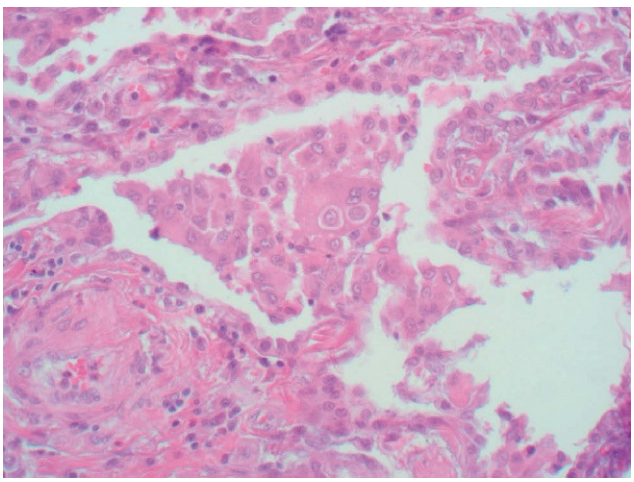

Figure 2. Pulmonary biopsy with presence of intra-alveolar multinucleated giant cells with phenomenon of emperipolesis (macrophages in the cytoplasm) (hematoxylin and eosin stain, ×400).

The second case refers to a 56 year old woman, without harmful habits or allergies, a tungsten carbide lathe operator for bicycle axle, among other tasks. Presents coughing and long evolution expectoration and in the high resolution tomography of the thorax, a reticular pattern with extensive areas of frosted glass was observed. The lung function tests showed 77% FVC; 80% FEV_1; 37% FEV_1% and 63% D_LCO; that is, slight restriction with moderate alteration of the CO diffusion. The BAL displayed the following formula: 80% macrophages, 10% lymphocytes, 8% polymorphonuclears and 2% eosinophils. No giant cells with emperipolesis were identified. The biopsy presented the usual pattern of interstitial pneumonia with centrilobular affectation and abundant intra-alveolar macrophages with abundant polarisable intracytoplasmic material, with no evidence of giant cells with emperipolesis. In this case, due to the suspicion of hard metal disease as a consequence of working with tungsten carbide, the SEM study was performed and abundant minerals were detected: carbon, silica, lead, zinc and aluminium, as well as fibreglass and brass. Treatment with corticoids was established at a dose of 30 mg/day without functional improvement.

Discussion

Hard metal interstitial disease was first described in Germany in 1940.[7] The clinical presentation is variable, in the form of subacute alveolitis or interstitial fibrosis. Retrospective studies, such as the one carried out by Posgay et al[5] on hard metal industry workers followed up for 30 years, showed no correlation between intensity, duration of the exposure, the stage and progression of the fibrosis. In 45% of the cases there was a progression of the disease after ceasing work. A fundamental aspect is that the disease can occur after a short exposure, which indicates that individual susceptibility, more than dust accumulation, plays an important role in the disease.[6]

Given the absence of specific alterations in the clinic, the radiology and laboratory analysis, the BAL study can be of aid if multinucleated giant cells with cannibalism phenomenon are observed. These cells should be considered pathognomonic to the disease, always within the appropriate clinical context; however, their absence does not exclude the disease.[1] Within this hypothesis, the study of the biopsy with the corresponding diagnosis of giant cell pneumonitis is sufficient. In the absence of these cells, the SEM study of the particles in the paraffin embedded tissue allows for detection of the presence of increased amounts of tungsten or other hard metals.

Both women arrived at the consultation referred from other centres for study. Both showed highly advanced clinical, radiological and anatomopathological conditions and the diagnostic protocols had not reached a definitive diagnosis. The age of appearance of the disease in both patients was too early to consider an idiopathic pulmonary fibrosis. The absence of giant multinucleated cell with cannibalism phenomenon in the BAL aided the surgical biopsy to evaluate the fibrosis and perform the ultrastructural study of the paraffin embedded tissue. In both cases, the suspicion of pneumonitis by hard metals was elevated given the use of a lathe without a face mask. In the first case, the presence of giant cells with emperipolesis presupposed the finding of hard metals such as niobium; on the other hand, in the second, no hard metals were detected, although there were abundant metals in high concentrations.

The origin of hard metals of the first patient was associated with the use of the lathe to polish jewels, although the probable incidence in the fibrosis of smoking was not ignored. In the second case, the use of utensils with diamonds to polish is well known. These materials are not hard metals since they do not contain tungsten carbide, but are included in the same category. Diamonds are a particular form of carbon and the proportion of cobalt as an adhesive that diamond utensils contain is much higher than the content in hard metals.[6] The absence of cobalt in the biopsy could be due to the elevated solubility of metal. As far as the rest of the metals are concerned, aluminium and silica have been associated to interstitial fibrosis,[2] in return, the probable participation in the symptoms of lead and zinc is unknown.

In summary, faced with a suspected case of hard metal disease, the particle analysis through SEM of the biopsy tissue can be fundamental to establishing diagnosis in patients with a labour background.

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