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COAL PNEUMOCONIOSIS

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ABSTRACT

La neumoconiosis del carbón es una patología muy poco investigada, en la actualidad no se ha logrado establecer su fisiopatología específica, pues se relaciona con otras enfermedades causadas por aspiracion de minerales. Las organizaciones para la salud laboral alrededor del mundo se han esforzado por establecer su incidencia en los mineros de regiones específicas y el impacto de la legislación local sobre la misma. En este documento se revisa su historia, conceptos generales, epidemiología y fisiopatología .

INTRODUCTION

Occupational diseases have been part of the history of humanity since its inception. Pneumoconiosis was described by Hippocrates in the 4th century BC, however, no interventions were reported to protect coal workers until 500 years later, when Gaius Pliny Secondary who in his work "Natural History" describes the use of bladders or bags on the face, in order to protect themselves from particulate matter. In 1866 Zenker coined the term "Pneumonokoniosis" for the first time.

Coal pneumoconiosis is part of diffuse interstitial lung diseases, and like them, it is caused by inhalation of inorganic dusts, generally during occupational exposures. The fourth International Conference on Pneumoconiosis in Bucharest 1971 defines it as "Accumulation of dust in the lungs and the tissue reaction to its presence" but, at present it is clarified that its reactions are not carcinogenic.

This pathology has multiple forms of presentation depending on the substance associated with the exposure, so it is important to mention silicosis and asbestosis when talking about coal pneumoconiosis, since they are routinely confused. For these diseases there is a mortality rate of 14.2 per million and 6.9 for asbestosis that presented an increase in frequency of 10 times, representing 52% of deaths related to pneumoconiosis in the USA, during the period from 1990 to 199 30,000 were reported deaths and 1240,000 years of healthy life lost. On the global scene, the WHO attributes 1.6 out of every 100 deaths to particulate matter.

There is a broad consensus regarding the global underreporting of pneumoconiosis incidence, morbidity and mortality. In the 2019 study by Leonard et al., This disease affected 60,000 miners across the United States, with a higher occurrence in those who worked in the industry long-term.

In Colombia there are few studies aimed at studying CP (coal pneumoconiosis). Garrote et al in a descriptive cross-sectional study, carried out in the Boyacá mines in 2013 in which 170 male workers were analyzed, reported a prevalence of pneumoconiosis of 8.8%. 15.9% of the workers studied presented radiological patterns compatible with pneumoconiosis. Furthermore, a 2018 study reported a prevalence of 33.8% in coal mine workers in the departments of Boyacá, Cundinamarca and Norte de Santander.

Coal is a material of non-uniform composition, which is why it is usually contaminated by silica, asbestos and multiple minerals that vary according to its extraction site. Aspirated particles are smaller than 10nm and their damaging effects are triggered by excessive or prolonged inhalation of inorganic powders. Pulmonary deposition is mediated by factors related to particulate matter, worker, geographic characteristics, and respiratory rate. (See table 1)

Table 1. Risk factors associated with the pulmonary deposition of particles. (RF: Respiratory rate)

FACTORES DEL POLVO	FACTORES DEL TRABAJADOR	FRECUENCIA RESPIRATORIA	FACTORES GEOGRÁFICOS
- Concentración y composición de las partículas - Tamaño de la partícula - Forma de la partícula: los anfiboles se depositan con mayor facilidad.	- Tiempo de exposición - Factores de riesgo individuales	- Actividad física genera aumento de FR - altura del sitio de trabajo aumenta la FR, sobretodo en personal en proceso de adaptación.	- En lugares secos, aumenta la cantidad de polvo.

Smoking has been shown to be an individual risk factor that predisposes to the presentation of the disease in a higher percentage.

Regarding the pathophysiological mechanism, it is important to mention that to date it is undetermined, however, the first theories regarding DILD, mention that a specific or indeterminate causative agent produces damage to the alveolar epithelium, which activates inflammatory cells (macrophages, lymphocytes, mast cells, eosinophils, neutrophils) and parenchyma such as fibroblasts and epithelial cells, resulting in secretion of inflammatory mediators, alveolitis and pulmonary fibrosis. However, the most recent publications consider inflammation a secondary actor, placing the lesion of the alveolar epithelium as the protagonist, which stimulates fibroblastic proliferation and therefore the process of pulmonary fibrosis. In the bronchoalveolar lavage of these patients, increased amounts of different cytokines such as IL-6, TNf-□ or insulin-like growth factor type 1 (IGF-1) have been found that also contribute to the fibrosis process.

Progressive massive fibrosis begins with the formation of macules and later nodules, whose agglomerations have a diameter equal to or greater than 20mm and are typically located in the apical lobes. These lesions in turn allow a classification of the disease as evidenced in Table 2.

Table 2. Classification of coal pneumoconiosis according to

Simple	- **Lesiones < 20 mm** - **Paciente asintomático** - **Historia de exposición ocupacional**
Complicada	- Lesiones >20 mm con fibrosis circundante - Desestructuración del parénquima - Enfisema - Disnea de esfuerzo - Alteración de la función pulmonar

The clinical presentation is variable and the diagnosis can even be reached by an incidental radiographic finding in totally asymptomatic patients. The symptoms generally presented are dyspnea on exertion and a dry cough, but we can also find a cough with expectoration and melanoptysis. On auscultation, a decrease in vesicular murmur is evident. Eventually, patients with a chronic presentation present with increased dyspnea, airflow obstruction, emphysema, respiratory failure or cor pulmonale.

The diagnosis of CP is made based on a history of exposure, compatible radiological findings and histological studies when the symptoms are atypical or there is no clear exposure. The chest radiograph shows a bilateral nodular pattern (> 2mm) of apical predominance that can involve the entire lung, in advanced cases there are masses and the hilum is enlarged and calcified, to make a better interpretation the criteria are used of classification of ILO (International labor office) 2011 where it is clarified that no radiographic pattern is pathognomonic of dust exposure, however, when it is suspected according to the clinical history, it allows a complete and systematic analysis. (see image 1)

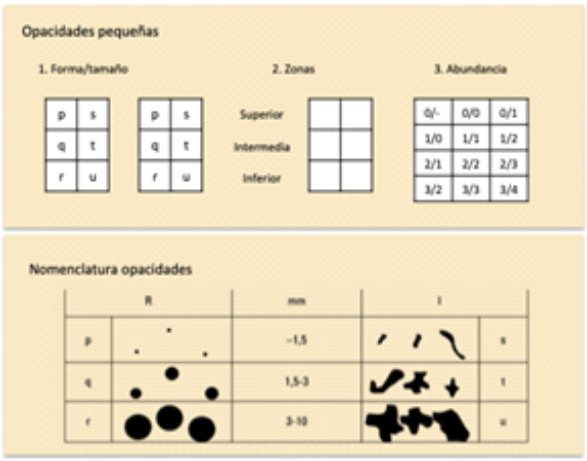

Image 1.

a) Reference should be made to the shape and size of the lesions by means of two letters and the profusion of the lesion by means of two numbers.

b) Nomenclature of the opacities found radiologically according to ILO.
Taken from: Martínez González C, et al.

Spirometry should be performed at the time of diagnosis and during follow-up, regardless of the findings that can be very varied depending on the progress of the disease and the predominance of the affectation, the alterations can have a restrictive and / or obstructive pattern with or without capacity diffusion conserved. Thrombocytosis appears as a relevant finding in the blood picture. Furthermore, according to Uygur et al, high mean platelet volume and platelet distribution width can be considered markers of CP and an important indicator of disease progression.

Table 3. Radiological and spirometric findings according to the degree of the disease.

Clasificación	Hallazgos radiológicos	Espirometría
Neumoconiosis simple	- Nódulos <20mm - Parénquima conservado	- Alteraciones obstructivas - Capacidad de difusion alterada o preservada
Neumoconiosis complicada	- Nódulos >20 mm en aglomeraciones bilaterales - Fibrosis circundante - Masas apicales - Hilio agrandado y calcificado	- Alteraciones obstructivas y/o restrictivas - Capacidad de difusión alterada o preservada según afectación

Currently, pharmacological treatment is used to reduce the severity of symptoms by means of bronchodilators, however, depending on the progression of the disease, some patients may benefit from oxygen therapy and pulmonary rehabilitation. Regarding surgical treatment, in the United States there is an increase in the frequency of lung transplants in patients with terminal lung disease, caused by coal pneumoconiosis, however, it is preserved as a last resort, which improves the survival of these patients after 6.6 years, compared with a mean of 2.8 to 3.7 years in patients with conservative treatment.

CONCLUSIONS

The complications of CP are varied and poorly documented, 10% of lung cancer cases are attributed to occupational exposure, however, this neoplasm does not present characteristics different from that caused by tobacco. An increased probability of developing cancer and COPD has been established in workers with tobacco habit who are exposed to asbestos, radon, arsenic, coal dust, diesel fuel fumes and silica. Asbestos is related to pleural mesothelioma in 75 to 85% of cases due to its action as a cocarcinogen and the risk is proportional to the time and intensity of exposure.

Pneumoconiosis predisposes to infection by tuberculosis and atypical mycobacteria, the mining population has a 3 times greater risk of presenting the disease and this relationship increases with the exposure time, if there is co-infection with HIV, infections by opportunistic pathogens can occur more easily. In coal workers with circulating rheumatoid factor, Caplan syndrome may occur with the appearance of circumscribed rheumatoid nodules of 0.5 to 5 cm in diameter around the bronchioles, cases of rheumatoid arthritis have been reported, however, there is no evidence of predisposition to the latter.

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