Pneumoconiosis — Symptoms and Treatment

Pneumoconiosis is an occupational lung disease that is related to coal-dust exposure. Macrophages and fibroblasts become activated and focal lung fibrosis occurs. If untreated, the disease can progress to massive lung fibrosis, which could be fatal. History taking and chest X-rays are the mainstay diagnostic tests to confirm and stage pneumoconiosis. Symptomatic treatment is the primary approach. Lung transplantation might be an option for patients with advanced pneumoconiosis.

Overview of Pneumoconiosis

Pneumoconiosis can be classified into different types depending on the type of dust causing the disease. Examples are berylliosis, coal worker’s pneumoconiosis (CWP), silicosis, and asbestosis.

Pneumoconiosis is defined as the chronic accumulation of fine dust in the lung parenchyma and the inflammatory reaction in response to coal, dust, or minerals. The inflammatory or fibrotic responses and the actual type of disease that develops depend on the type and amount of dust inhaled.

The causative factors and presentations of pneumoconiosis are tabulated
Simple and progressive CWP are the two distinct variants of this condition. As the name implies, patients with simple CWP have limited lung disease that is not progressive, while patients with progressive CWP have an ongoing disease process that is associated with extensive lung fibrosis.

**Epidemiology of Pneumoconiosis**

Pneumoconiosis is an occupational lung disease, which is more common among people within certain professions such as coal mining. Therefore, most patients in the United States are from Pennsylvania, Maryland, Virginia, and Kentucky, where coal mining is common. Pneumoconiosis has an estimated prevalence of 30%, while that of progressive CWP can be as high as 2.5% in people exposed to coal dust.

Additionally, pneumoconiosis-related mortality has significantly decreased in the past decades due to several reasons. While the most important cause for the reduction of pneumoconiosis-related mortality can be traced to a decline in the coal-mining workforce, early diagnosis and prompt treatment have also been linked to a decrease in disease progression.

Pneumoconiosis is a chronic disease that develops over the years. Therefore, the majority of patients are usually > 50 years of age. Pneumoconiosis has been identified in both genders and no racial differences have been noted.

**Pathophysiology of Pneumoconiosis**

Different stages of pneumoconiosis can be identified with each having its own characteristic pathogenic features. The earliest stage of coal-dust accumulation in the lungs is known as anthracosis. While anthracosis is common among coal miners, it has also been encountered in tobacco smokers, who have no history of exposure to coal dust.

When an individual inhales coal dust either while smoking or during coal mining, carbon dust reaches the terminal bronchioles. Next, the alveolar macrophages engulf these carbon particles. While this phenomenon is usually adequate to clear out coal dust, the system can sometimes become overwhelmed, as in the case of coal miners. Eventually, the coal dust-filled macrophages start accumulating in the alveoli and trigger an inflammatory response.
The first step in the pathogenesis of pneumoconiosis is believed to be the activation of fibroblasts, which secrete reticulin. Reticulin is responsible for the entrapment of macrophages in the alveoli. An important constituent of coal dust is silica, which is responsible for the accelerated lysis of macrophages and the recruitment of additional fibroblasts. Recruited fibroblasts secrete collagen and commence the process of fibrosis.

At first, the distinct focal areas of macrophages, which are filled with coal dust and activated fibroblasts form coal macules. These are pathognomonic histologic patterns of CWP. The next step in the pathogenesis of CWP involves the aggregation of multiple coal macules, deposition of additional collagen, and the development of interstitial lung fibrosis. The consequence of this pathology is the formation of focal emphysema, which can initially appear asymptomatic.

CWP can become progressive and extensive lung fibrosis may ensue as coal macules start recruiting more activated immune cells. It is believed that rheumatoid arthritis and the rheumatoid factor are important factors for the progression of CWP. Pneumoconiosis, when associated with rheumatoid arthritis, is known to cause Caplan syndrome, which is characterized by progressive massive lung fibrosis.

Clinical Presentation of Pneumoconiosis

Pneumoconiosis, like other forms of interstitial lung disease, can be diagnosed based on detailed history-taking combined with advanced imaging modalities. Social history taking, including occupational history, is essential in the evaluation of patients with suspected pneumoconiosis. Those working in the coal-mining industries are at an increased risk of pneumoconiosis. Additionally, the determination of the silica content of coal that the patient was exposed to, is essential to assess the risk of progressive CWP.
Smoking history constitutes an important factor because coal miners who smoke are more likely to exhibit symptoms of CWP compared to non-smokers. Unfortunately, the majority of patients with pneumoconiosis do not consider changing their career as a viable option.

Simple CWP can be asymptomatic or may present with non-specific symptoms such as a productive cough. Once the disease progresses, patients can develop dyspnea that is typically progressive. If left untreated, right-sided heart failure may develop.

Pneumoconiosis can also be associated with the occlusion of small bronchioles, which predisposes the patient to infectious complications. Those with an infectious disease can develop fever, night sweats, and chest pain.

Diagnostic Workup for Pneumoconiosis

Laboratory investigations are not helpful in the confirmation of the diagnosis; however, they can exclude infectious complications. Therefore, patients with pneumoconiosis who present with a fever should be indicated a complete blood count and sputum culture to exclude possible infectious diseases.

Chest X-ray provides valuable information in the diagnosis and staging of pneumoconiosis. Patients with simple CWP can present with lung nodules < 1 cm in diameter. These nodules are usually found in the upper lobes of the lungs.
Patients who present with lung nodules > 1 cm in diameter are considered to have **progressive massive fibrosis**. These patients usually have **multiple small nodules and a large nodule** in addition to simple CWP.

**Pulmonary function tests** indicate normal results in patients with simple CWP. Those with complicated or progressive CWP can have **decreased ventilatory capacity**, which can be detected using these tests.

**Treatment of Pneumoconiosis**

Unfortunately, there is **no definitive treatment** for pneumoconiosis regardless of the stage of the disease. Current treatment approaches are mainly **symptomatic**. One of the main problems with pneumoconiosis is **hypoxia**; therefore, **oxygen therapy** is indicated in patients with moderate to severe hypoxemia.

Additionally, recent evidence has shown that **avoiding exposure to coal dust** will not stop disease progression once the patient has developed progressive CWP. Therefore, a change in the line of work as a means to reverse pneumoconiosis is questionable. On the other hand, it is possible to halt disease progression in patients with simple CWP if they transfer to a job that has less exposure to coal dust.

Patients with **Caplan syndrome** can develop hypoxemia. **Oxygen therapy** combined with **bronchodilators** is indicated in such patients. Since Caplan syndrome is related to rheumatoid arthritis, **disease-modifying antirheumatic drugs** are usually indicated, per current guidelines for the treatment of rheumatoid arthritis.

Patients with pneumoconiosis are at a significant risk of **influenza-related mortality**; therefore, immunization against influenza is recommended. Patients with unexplained weight loss, fever, night sweats, and chronic cough may have a **superimposed mycobacterial infection**; thus, specific treatment for mycobacterial infection should be commenced.

Patients with progressive massive lung fibrosis or complicated CWP might be possible candidates for bilateral or single **lung transplantation**. A study reports that patients...
respond well after lung transplantation. These patients, however, had not reached the stage of cor pulmonale or right-sided heart failure. Mortality is high in patients with cor pulmonale due to massive lung fibrosis.

References

Silicosis and coal workers’ pneumoconiosis via nih.gov
Coal Worker’s Pneumoconiosis via medscape.com

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