Research & Reviews: Journal of Clinical and Medical Case Studies

Synoptic Overview of Black Lung Disease in Coal Workers

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Commentary

Received: 27-May-2022, Manuscript No. JCMCS-22-68623; **Editor assigned:** 01-Jun-2022, Pre QC No. JCMCS-22- 68623 (PQ); **Reviewed:** 14-Jun-2022, QC No. JCMCS-22- 68623; **Revised:** 21-Jun-2022, Manuscript No. JCMCS-22- 68623 (R); **Published:** 28-Jun-2022, DOI: 10.4172/J Clin Med Case Stud.7.5.003.

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DESCRIPTION

Long-term exposure to coal dust results in Coal Workers Pneumoconiosis (CWP) is also known as black lung disease or black lung. Coal miners and others who work with coal frequently experience this phenomenon. Both silicosis from breathing in silica dust and asbestosis from breathing in asbestos dust are similar to it. Inhaled coal dust gradually builds up in the lungs causing fibrosis, necrosis and in severe cases and inflammation.

Following the initial, milder stage of the illness known as anthracosis, coal workers pneumoconiosis develops which is a severe state. Due to air pollution, this is frequently asymptomatic and is present in all urban dwellers to some degree. Simple coal workers pneumoconiosis and complicated coal workers pneumoconiosis are two more severe forms of the disease that can develop from prolonged exposure to large amounts of coal dust (Progressive Massive Fibrosis or PMF).

Industrial bronchitis which is clinically described as chronic bronchitis (i.e., a productive cough for three months every year for at least two years) linked to workplace dust exposure is more frequently experienced by workers exposed to coal dust. Age, employment, exposure and smoking all affect the likelihood of developing industrial bronchitis. Studies of coal miners have revealed a 16%–18% incidence of industrial bronchitis which is lower than that of smokers (who are more likely to develop bronchitis). Globally, CWP caused 25,000 deaths in 2013 compared to 29,000 deaths in 1990. The National Institute of Occupational Safety and Health later conducted a study in 2018 that revealed a resurgence of the incurable respiratory illness with the highest rate seen in roughly 20 years.

Compared to silica dust, coal dust is not as fibrogenic. The body is unable to either destroy or remove coal dust that has entered the lungs. The particles remain in the lungs residing in the connective tissue or pulmonary lymph nodes after being engulfed by local alveolar or interstitial macrophages. The macrophage is sufficiently stimulated by coal dust to release a variety of substances including enzymes, cytokines, oxygen radicals and fibroblast growth factors, all of which play a crucial role in the fibrosis and inflammation of CWP. Under a microscope, granular, black areas represent collections of macrophages that have accumulated carbon. The lung may grossly appear black in severe cases. These aggregations have the potential to develop nodular lesions, fibrosis and inflammation within the lungs. The following three factors are necessary to diagnose CWP:

- Chest radiography that supports CWP
- A sufficient amount and latency of prior exposure history to coal dust (usually from underground coal mining)
- Eliminating alternate diagnosis (CWP)

Although they are not a component of the diagnostic criteria, symptoms and pulmonary function testing are related to the level of respiratory impairment. As previously mentioned, silicosis and CWP can seem almost identical on chest X-rays. High-Resolution Scanning (HRCT) and chest CT are more sensitive than ordinary X-ray for detecting the tiny spherical opacities.

There are no known causes, remedies or cures for pneumoconiosis. Some patients get oxygen to help them breathe and they get advice to stop smoking to prevent further lung function loss. In the worst-case scenarios, a lung transplant could be done to help the patient live longer. Large cavities within the lung may result from the centers of dense lesions becoming necrotic as a result of ischemia. Avoiding coal dust inhalation is the main preventative measure for coal workers' pneumoconiosis. Avoiding smoking, protecting from potentially harmful airborne particles, getting regular pulmonary exams, and learning about the risks of lung diseases in your workplace are a few ways to prevent this disease.