Silikosis, Penyakit Akibat Kerja pada Pekerja Industri Konstruksi dan Pertambangan Akibat Paparan Debu Silika

Syazili Mustofa[1]*

[1]Department of Biochemistry, Molecular Biology And Physiology, Faculty Of Medicine, Universitas Lampung, Bandar Lampung

Abstrak

Silikosis merupakan penyakit fibrosis pada paru yang disebabkan oleh inhalasi, retensi, dan reaksi terhadap silika kristalin. Faktor utama yang berperan pada patogenesis silikosis adalah partikel debu dan respons tubuh khususnya saluran napas terhadap partikel debu tersebut. Silika adalah zat yang ditemukan secara alami dalam beberapa jenis batu, pasir, dan tanah liat. Silikosis terutama menyerang pekerja yang terpapar debu silika dalam pekerjaan seperti konstruksi dan pertambangan. Mediator yang paling banyak berperan pada patogenesis silikosis adalah Tumor Necrosis Factor (TNF)-α, Interleukin (IL)- 6, IL-8, platelet derived growth factor dan transforming growth factor (TGF)-β. Kriteria mayor untuk diagnosis silikosis mencakup riwayat pajanan debu silika yang signifikan, kelainan radiologis yang sesuai (seperti nodul paru pada rontgen dada atau CT scan), dan terkadang, gejala klinis seperti batuk dan sesak napas yang progresif. Tatalaksana silikosis bersifat simtomatis, yaitu hanya untuk mengurangi gejala. Sangat penting menghilangkan sumber pemaparan untuk mencegah semakin memburuknya penyakit.

Kata kunci: silikosis, patogenesis, diagnosis, tatalaksana

Silicosis, A Work-Related Disease in Construction Workers and Miners Exposed to Silica Dust

Abstract

Silicosis is a fibrotic lung disease caused by the inhalation, retention, and reaction to crystalline silica. The primary factors contributing to the pathogenesis of silicosis are dust particles and the body's response, particularly the respiratory tract's reaction to these dust particles. Silica is a naturally occurring substance found in some types of rocks, sand, and clay. Silicosis primarily affects workers exposed to silica dust in occupations such as construction and mining. The mediators that play the most significant role in the pathogenesis of silicosis are Tumor Necrosis Factor (TNF)-α, Interleukin (IL)-6, IL-8, the activity of platelet-derived growth factor (PDGF), and transforming growth factor (TGF)-β. The major criteria for diagnosing silicosis include a significant history of silica dust exposure, characteristic radiological abnormalities (such as pulmonary nodules on chest X-rays or CT scans), and sometimes clinical symptoms, including a progressive cough and shortness of breath. The management of silicosis is symptomatic, meaning it is only aimed at reducing symptoms. It is very important to eliminate the source of exposure to prevent the disease from worsening.

Keywords: silicosis, pathogenesis, diagnosis, treatment

Korespondensi: Dr.Si. dr. Syazili Mustofa, S.Ked, M.Biomed., Building C floor 1, Faculty of Medicine, University of Lampung, Jalan Prof. Dr. Sumantri Brojonegoro No. 1, Bandar Lampung 35145, Indonesia, HP 081929345909, e-mail syazilimustofa.dr@gmail.com

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introduction

The construction and mining industries in Indonesia are two significant and interconnected economic sectors. The construction industry plays a vital role in infrastructure development, while mining provides raw materials for various industrial sectors, including the construction sector. Both of these sectors have great growth potential, but they also face challenges, including specific health issues. (1)

Among the many air pollutants in the construction and mining industry work environment, dust is one of the chemical agents that can cause health problems. Dust can also lead to reduced work comfort, visual disturbances, impaired lung function,

pulmonary fibrosis damage, and even general poisoning if inhaled while working. [2]

Pneumoconiosis is a condition that occurs due to the accumulation of dust in the lungs, causing a tissue reaction to that dust. The primary response due to dust exposure in the lungs is fibrosis. Pneumoconiosis refers to various conditions resulting from exposure to inorganic dust, such as silica (silicosis), asbestos (asbestosis), and tin (stannosis), as well as conditions caused by organic dust, including cotton (byssinosis). [3] Silicosis is one of the most common forms of pneumoconiosis caused by the inhalation of crystalline silica dust, characterized by inflammation and scarring in the form of nodular lesions in the upper lobes of the lungs. Silicosis is marked by shortness of breath, fever, and cyanosis. Silicosis is also known by terms such as miner's phthisis, grinder's asthma, and potter's rot. [4]

Etiology

Silicosis is a work-related lung disease caused by inhaling silica dust or silica particles. Workers in sandblasting, drilling, tunneling, silica flour milling, ceramics manufacturing, and others are predisposed to the occurrence of silicosis. [4] Silica is a group of natural minerals divided into two types: non-crystalline (amorphous silica) and crystalline (crystalline silica). [5]

There are three main forms of crystalline silica: quartz, cristobalite, and tridymite. These are the most common and well-known forms, though other, less common forms also exist. **Quartz is** the most abundant form of crystalline silica. **Cristobalite** can form when silica is heated to high temperatures, like in some volcanic rocks. **Tridymite** also forms at high temperatures and is less common than quartz. Other forms mentioned in the search results include coesite **and stishovite, high-pressure** polymorphs of silica. **Moganite is a** silica mineral with a slightly different crystal structure than quartz, **and keatite is another** polymorph of silica. While these other forms exist, the three mentioned above (quartz, cristobalite, and tridymite) are the most important in terms of potential health risks and industrial applications. [6]

Exposure to respirable crystalline silica dust has long been recognized as a health hazard that can cause nodules to form in the lungs. Free silica dust (SiO2), with a diameter of 0.02 to 0.08 µm, primarily contains Si elements inhaled during breathing and accumulated in the lungs. Respirable dust pollution containing silica settles in the bronchioles and alveoli. This dust is fibrogenic and can cause restrictive lung disorders. If fibrosis (lung tissue damage) has occurred, the process of fibrosis will continue, even if exposure to silica dust is no longer present. [7]

Pathogenesis

Silicosis is a fibrosis disease of the lungs caused by inhalation, retention, and reaction to crystalline silica. [6] Inhalation of silica dust is known to cause several abnormalities in the lungs, including acute inflammation, chronic inflammation, pulmonary fibrosis (silicosis), chronic obstructive pulmonary disease, and lung cancer. [8]

The main factors involved in the pathogenesis of silicosis are dust particles and the body's response, particularly the respiratory tract, to these dust particles. The chemical composition, physical properties, dose, and duration of exposure all contribute to the likelihood of developing silicosis. The cytotoxicity of dust particles against alveolar macrophages plays a crucial role in the pathogenesis of pneumoconiosis. Quartz dust is more cytotoxic compared to insoluble types. The chemical properties of dust particle surfaces, specifically the activity of free radicals and iron content, are also of significant importance in the pathogenesis of silicosis. [9]

The pathogenesis of silicosis begins with the response of alveolar macrophages to the dust that enters the respiratory unit of the lungs. Phagocytosis of dust by macrophages occurs, and the subsequent processes depend greatly on the toxic properties of the dust particles. The tissue reaction to dust varies according to the biological activity of the dust. If exposure to inorganic dust is prolonged, an initial inflammatory reaction occurs. The main feature of this inflammation is the accumulation of inflammatory cells in the lower respiratory tract. [10]

Alveolitis can involve bronchioles and even large airways, as it can cause lesions and fibrosis in the alveolar units that are clinically unnoticed. Some dust, such as coal dust, appears relatively inert and accumulates in the lungs in relatively large amounts, with minimal tissue reaction. Inert dust will remain in macrophages until the macrophages die from old age; thereafter, the dust will be released and phagocytosed again by other macrophages. Macrophages with dust inside can migrate to lymphoid tissue or the bronchioles and be expelled through the airways. (11)

In toxicologically active dust, dust particles that are phagocytosed by macrophages can destroy those macrophages, leading to fibrositis. Dust particles stimulate alveolar macrophages to release mediators that initiate an inflammatory response and trigger the process of fibroblast proliferation and collagen deposition. The mediators that play the most significant role in the pathogenesis of silicosis are Tumor Necrosis Factor (TNF)-α, Interleukin (IL)-6, IL-8, platelet-derived growth factor, and transforming growth factor (TGF)-β. Most of these mediators are crucial for the process of fibrogenesis. (12)

The important macrophage mediators responsible for tissue damage, cell accumulation, and stimulation of fibroblast growth are: 1. Oxygen radicals, also known as reactive oxygen species and proteases. 2. Leukotriene LTB4 and IL-8 that are chemotactic for leukocytes. 3. Cytokines IL-1, TNF-α, fibronectin, PDGF, and IGF-1 that play a role in fibrogenesis. (12) Cytokines have been proven to play a role in the pathogenesis of silicosis. Pappas summarizes the cytokines produced by alveolar macrophages in response to inhaled dust particles, which subsequently cause fibrosis in the lung interstitial tissue. These cytokines comprise fibrogenesis factors, such as TNF-α, PDGF, IGF-1, and fibronectin, as well as pro-inflammatory factors, including LBT4, IL-8, IL-6, and MIP-1α. Besides the process of phagocytosis of dust by alveolar macrophages, what is more important is the interstitialization of the dust particles. When dust particles are phagocytosed by macrophages and transferred to the mucociliary system, the process of clearing dust that enters the respiratory tract is considered successful. (13)

The loss of epithelial integrity due to inflammatory mediators released by alveolar macrophages is an early event in the fibrogenesis process in the lung interstitium. Once dust particles have entered the interstitium, their fate is determined by interstitial macrophages, which phagocytize them and then transfer them to the mediastinal lymph nodes, or chronic inflammatory mediators may be secreted in the interstitium. Cytokines released in the interstitium, such as PDGF, TGF, TNF, and IL-1, stimulate fibroblast proliferation, contributing to the development of pneumoconiosis. (14)

The toxicity of dust determines the tissue reaction that occurs in silicosis. Silica dust has a very strong biological effect. The parenchymal reaction can be nodular fibrosis, which is a classic example of silicosis. An irregular and mixed fibrotic picture occurs with mixed dust exposure. Four pathological response patterns are observed in silicosis, namely interstitial fibrosis, nodular fibrosis, a combination of nodular and interstitial fibrosis, as well as focal emphysema and macule formation. (12,14)

Silicosis is a health issue in the construction and mining industries.

Silicosis is a serious health problem in the construction and mining industries. By understanding the risks, implementing appropriate preventive measures, and conducting regular health monitoring, we can protect workers from diseases caused by silica dust. Silicosis is a lung disease caused by exposure to silica dust, particularly affecting workers in the construction and mining industries. Construction workers are exposed to silica dust when working with materials such as concrete, stone, and sand, as well as during demolition work. Jobs involving cutting, grinding, drilling, or blasting materials containing silica can generate inhalable dust and cause silicosis. Mining, whether it involves coal, metals, or other minerals, often releases silica dust into the air. High-risk mine workers are exposed to silica dust, especially during the

drilling, blasting, or processing of materials that contain silica. [15]

This disease cannot be cured, but it can be prevented with appropriate protective measures. The use of personal protective equipment (PPE), such as dust masks, good ventilation, and adherence to safe working practices, is essential to reduce the risk of silicosis among construction workers. In addition to PPE and ventilation, regular health monitoring programs and dust control at the source are crucial in protecting mine workers from silicosis. Silicosis is a chronic disease that cannot be cured, so prevention is the key to protecting workers. Silicosis can cause shortness of breath, chronic cough, chest pain, and even increase the risk of other lung diseases such as tuberculosis and lung cancer. Silicosis can lead to loss of work productivity, high healthcare costs, and even death. [16]

Classification

There are three main types of silicosis: acute, accelerated, and chronic. This classification is based on the duration and intensity of exposure to silica dust. Acute silicosis occurs due to very high silica dust exposure over a short period, often within weeks or months. Symptoms include severe shortness of breath and low blood oxygen levels and can be fatal within months to several years. Accelerated silicosis occurs due to prolonged exposure to high levels of silica dust, typically throughout 5 to 15 years. [17] Symptoms include inflammation, scarring, and rapid progression of symptoms. Chronic silicosis is the most common type, resulting from prolonged exposure to low levels of silica dust (lasting 10 years or more). This disease can cause nodules and scarring in the lungs and lymph nodes and may resemble COPD (Chronic Obstructive Pulmonary Disease). [4]

Diagnosis

The major criteria for diagnosing silicosis include a significant history of silica dust exposure, appropriate radiological abnormalities (such as lung nodules on chest X-rays or CT scans), and sometimes, clinical symptoms like progressive cough and shortness of breath. (18) Here is a more detailed explanation of these criteria:

1. **Significant Silica Dust Exposure:**
   This is the most important criterion. Silicosis is a lung disease caused by inhaling crystalline silica dust. This exposure typically occurs in jobs involving mining, excavation, drilling, tunnel construction, sandblasting, and other work related to silica dust. The level of significant exposure can vary, but it usually involves long-term exposure or high-intensity exposure. [4]
2. **Radiological Abnormalities:**
   Images of the lungs, especially chest X-rays and CT scans, show a characteristic picture of silicosis. This appearance can be in the form of small nodules (silicotic nodules) or fibrosis (scarring tissue) in the lungs. Chest CT scans are considered more sensitive in detecting these abnormalities than chest X-rays. High-resolution computed tomography (CT) of the chest is more sensitive than chest X-rays for detecting simple silicosis. In complicated silicosis, a CT scan provides clearer results (cavities). Although cavities in silicosis can occur without mycobacterial infection, the possibility of this disease needs to be considered. However, despite the higher sensitivity, it is only used in cases where there is clinical or radiological doubt. [8]
3. **Clinical Symptoms:**
   Some individuals may not show symptoms in the early stages of silicosis. However, over time, symptoms such as chronic cough, shortness of breath, chest pain, fatigue, and weight loss may appear. In cases of acute silicosis, symptoms can be more severe and appear more quickly, including fever, severe chest pain, and severe difficulty breathing. [18]

In addition to the major criteria above, lung function tests (such as spirometry) can also aid in the diagnosis of silicosis, especially in assessing the level of lung damage. It is

important to remember that the diagnosis of silicosis must be made by a qualified doctor, taking into account the exposure history, radiological findings, and clinical symptoms of the patient. [4]

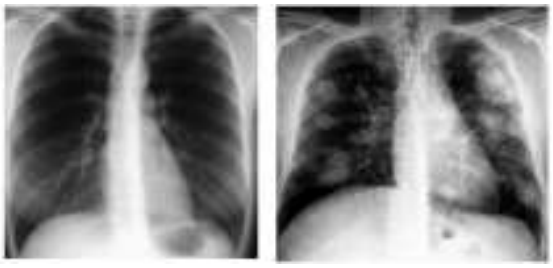

Figure 1. Photo of a plain thorax. (A) Normal thorax photo (B) Thorax photo of a patient with silicosis. Radiographic findings on chest X-ray may include bilateral consolidation and/or ground-glass opacities, which tend to appear in the perihilar region.

Management

The management of silicosis aims to reduce symptoms and prevent complications, as there is currently no cure for this disease. The main treatment is supportive, including the use of bronchodilators to help open the airways, oxygen therapy to address shortness of breath, and vaccinations to prevent lung infections. It is also important to avoid further exposure to silica dust and to maintain a healthy lifestyle [8]. Here are the details of silicosis management:

1. Supportive Treatment:

Bronchodilators: These medications help to widen the airways, making breathing easier. [19]

Oxygen Therapy: Administering supplemental oxygen can help alleviate shortness of breath and fatigue caused by low oxygen levels in the blood.

Vaccination: Individuals with silicosis are more susceptible to lung infections. Vaccinations, such as influenza and pneumococcal vaccines, can help prevent infections. [20]

Lung Rehabilitation: Lung rehabilitation programs can help patients enhance their lung function, strength, and overall quality of life through physical exercise, Education, and behavioral modifications.

2. Prevention of Further Exposure:

Avoid Silica Exposure: The most important step is to avoid further exposure to silica dust. It may involve changing jobs, using personal protective equipment (such as dust masks), and implementing safe work practices in the workplace to prevent exposure. [9]

Workplace Prevention:

It is essential to implement effective dust control measures in the workplace, including utilizing good ventilation, spraying water to reduce dust, and avoiding dry sweeping. [16]

3. Treatment of Complications:

Infections: Lung infections, including tuberculosis (TB), must be treated immediately with appropriate antibiotics. [21]

Lung Transplantation: In severe cases, lung transplantation may be a treatment option. [18]

4. **Routine Check-ups:** Patients with silicosis should undergo regular health check-ups to monitor disease progression and detect complications early. [21]

It is essential to remember that silicosis is an incurable lung disease. Treatment aims to control symptoms and prevent complications. The role of prevention is crucial in reducing the risk of developing silicosis and slowing the progression of the disease.

Conclusion

Silicosis is a work-related lung disease caused by prolonged inhalation of silica crystals, resulting in the formation of fibrotic tissue in the lungs and leading to irreversible damage. This disease typically affects workers in the construction and mining industries. It can be prevented by avoiding exposure to silica dust. The diagnosis of silicosis is made through a combination of a history of silica dust exposure, clinical symptoms, and imaging and lung function tests. The management of silicosis is symptomatic, meaning it is only aimed at reducing symptoms. It is crucial to eliminate the source of exposure to prevent the disease from further deteriorating.

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