Article Review

Silicosis: Mechanisms, Clinical Aspects, and Impacts due to Silica Exposure

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Abstract

Silicosis, an occupational lung disease, has significant mortality rates in Indonesia, as reported by Global Health Grove in 2013. The death rate for silicosis stands at 69.3%, with the typical age at death ranging from 40 to 44 years for men and approximately 80 years for women. The pathogenesis of silicosis begins when respirable crystalline silica (RCS) particles enter the airways. These RCS particles bypass the mucociliary defense mechanisms of the respiratory tract and reach the alveoli. Workers frequently exposed to silica are at high risk of developing silicosis, which significantly impacts morbidity and mortality. The diagnosis of silicosis can follow the seven-step principle for determining occupational diseases. Although silicosis is linked to serious conditions such as tuberculosis, autoimmune diseases, and lung cancer, no effective therapy exists. Treatment remains symptomatic, adjuvant, and supportive. To prevent occupational lung diseases, it is essential to involve the government in policy-making for industrial management and workers.

Keywords: occupational lung disease, pneumoconiosis, silicosis

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INTRODUCTION

Pneumoconiosis is among the most challenging lung diseases to diagnose and assess due to its prolonged progression.[1] According to the International Labour Organization (ILO), pneumoconiosis is an illness caused on by accumulation of dust in the lungs that triggers a tissue reaction to the dust. Pneumoconiosis is part of Occupational Lung Disease (OLD).[2] Silicosis is classified as a subtype of OLD and is caused by the inhalation, retention, and pulmonary reaction to particles of Silicon dioxide (SiO2) ≤10 microns.[3] Approximately 59% of the earth's formations are composed of silica, which is also referred to as silicon dioxide (SiO2).[4]

Silicosis was classified as "Miners' Phthisis" by Benardo Ramazzini. In 1870, the term "Silicosis" was first implemented by Peacock and Greenhow.[5] Inhalable silica is called Respirable Cristaline Silica (RCS).[6] Silicasis accounted for 39% of all pneumoconiosis cases worldwide in 2017, and an estimated 2 to 3 million workers are exposed to silica annually.[7] An estimated 1.7 million construction and maritime workers are exposed to silica in the United States. Approximately 6.4% of the mining and construction the workers in Australia has exposure to silica, of which 3.3% are exposed to high levels. A mining sector employs 1.7 million individuals in India, of which three million are at high risk of silica exposure. A work environment in China contaminated over 23 million employees with silica.[8,9]

The prevalence of silicosis in Indonesia is not well-documented. Numerous small-scale investigations across various industries have provided data on silicosis. A study conducted in the ceramics industry in 2000 found that 1.5% of workers were affected by silicosis., while a study conducted in a cement facility located in West Java from 1990 to 2003 revealed a silicosis incidence of 2.06%.[10]

According to a 2013 report by Global Health Grove, the silicosis mortality rate in Indonesia was 69.3%, with the average age of death being between

40 to 44 years for men and 80 years for women.[11] The aim of this article review is to examine the clinical aspects, disease progression mechanism, and physiological effects of silicosis exposure.

DEFINITION AND CLASSIFICATION

Silicosis is an occupational disease caused by the inhalation, retention, and pulmonary reaction to silicon dioxide (SiO2) particles ≤10 microns. Inhalation of silica particles may result in fibrosis and inflammation of the pulmonary tissues. Occupational silicosis can result from silica exposure, which is contingent upon the worker's dose, duration, and intensity of exposure. In 1996, the World Health Organization (WHO) established that hazardous dust particles is ≤10 microns.[12,13]

According to the Indonesian Ministry of Health, hazardous dust particles differ in size from 0.1 to 10 microns. The approximate range of RCS is 10 microns. Silica can be classified as either crystalline or amorphous (non-crystalline) according to its morphology. Quartz (found in sand and rocks), cristobalite, trydimite, coesite, and shistovite (found in magma) comprise the crystalline form. The alternative form is amorphous and can be separated into vitreous silica (which has negligible fibrogenic properties) and diatomite (which is derived from marine organisms).[14,15]

Silicosis can be classified as chronic/classic, accelerated, or acute, according to the duration, intensity, and dosage of exposure. The main manifestation is chronic or classic silicosis.[1] An exposure lasting between 10 to 15 years is required to induce symptoms. Classical or chronic silicosis is classified into two distinct varieties, Progressive Masive Fibrosis (PMF) and simple silicosis, according to the progression of the disease.[9] Simple silicosis is a form of chronic/classic silicosis with pulmonary nodules on radiology but without clinical symptoms. PMF is the union of small nodules in simple silicosis that form large conglomerate nodules with a diameter of more than 10 mm.[16,17]

Intense exposure causes accelerated silicosis, which develops over 5 to 10 years, compared to the longer duration of chronic or classic silicosis. Inflammation, scar tissue formation, and symptoms appear more rapidly.[1,8] The most lethal variety of silicosis is acute silicosis, also known as silicosproteinosis. This progressive condition is induced by exposure to extremely high concentrations of silica particles and develops over a few months to five years. Acute silicosis is potentially fatal, with a very poor prognosis.[1,18]

PATHOGENESIS

Silicosis pathogenesis initiates with the entry of RCS into the respiratory tract. RCS enters the airway directly into the alveoli, circumventing the mucociliary defense mechanism of the respiratory tract.[8,15] Alveolar macrophages induce phagocytosis of silica particles that infiltrate the alveoli. By through interacting with Macrophage Receptors with a Collagen Structure (MARCO), which is present on the surface of alveoler macrophages, the particles are phagocytosed. Silica particle clearance from the airway by alveoler macrophages, which have phagocytosed such particles, can be accomplished or not.[4,15] The pathway of pathogenesis is shown in Figure 1.

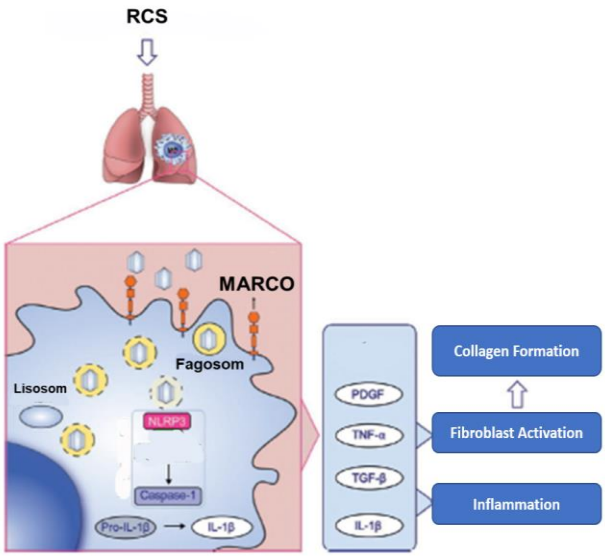

Figure 1. Pathogenesis of Silicosis[15]

Alveolar macrophages are successful if they can discharge the phagocytized silica particles through lymphatic channels to nearby lymphoid glands or clear them by mucociliary movement to be expectorated or swallowed. The potential reason

behind the inability of alveolar macrophages to eradicate silica particles is their toxicity. Particles of silica possess piezoelectric characteristics, specifically the capacity to generate electrical polarity.[8,15]

This property induces the production of Reactive Oxygen Species (ROS), which induces inflammation and fibrosis, and exerts a direct cytotoxic impact on bodily cells. Phagosomes disintegrate due to the toxic effect of silica, which results in the return of unattached silica particles to the cytoplasm. As the consequence of lysosomal enzymes liberated during phagosome disintegration, macrophages suffer apoptosis.[8,15]

Alveolar macrophage damage activates NOD-LRR and pyrin domain-containing protein-3 (NLRP3) inflamosomes and forms ROS. Subsequently, this protein induces the rolling motion of the Caspase-1 enzyme. This enzyme serves as a precursor to the synthesis of inflammatory mediators, Interleukin-1 (IL-1) in particular. Pro-Interleukin-1β is transformed into Interleukin-1β (IL-1β) by caspase-1.[1,15]

Additionally, this conversion stimulates the release of additional inflammatory mediators. Platelet Derived Growth Factor (PDGF), Transforming Growth Factor-β (TGF-β), as well as Tumor Necrosis Factor-α (TNF-α). Inflammatory mediators induce the activation of fibroblasts, forming collagen, and tissue inflammation.[4,8,15] Silica particles are released by destroyed alveolar macrophages and eventually re-phagocytosed by other macrophages, thus initiating a continuous process of tissue damage.[1,15]

Wijaya et al investigated the correlation between worker serum Transforming Growth Factor-β1 (TGF-β1) and silica exposure. The study findings indicated that laborers who were exposed to silica experienced an important elevation in serum TGF-β1, with a correlation that was statistically significant (P<0.05). The ability of TGF-β1 to promote fibroblast cell proliferation and enhance the transcription of genes associated with collagen and fibronectin synthesis significantly contributes to the development of pulmonary fibrosis. Isomerization-related to fibrosis is most prevalent among the forms of TGF-β1.[9]

RISK FACTORS

Naturally occurring minerals also contain silica in diverse concentrations, such as granite (25–40% silica) and sandstone (67% silica).[8] Frequent silica exposure among workers is one of the risk factors for silicosis, a condition that can result in severe illness and death. Sandblasters, cement, ceramics, construction of ships, mining, stone grinding, stone processing, construction, glass manufacturing, and sandblasting pose a significant silica exposure hazard, according to a number of studies conducted in both developed and developing nations.[5,17] According to research conducted in South Dakota, United States, a 45-years exposure to silicosis at a dose of 0.09 mg/m3 is associated with a 47% increased risk of mortality.[19]

DIAGNOSIS

The seven-step process for identifying occupational diseases, applicable to the diagnosis of silicosis, is outlined in Regulation No. 56 of 2016 by the Minister of Health of the Republic of Indonesia. This regulation specifies the stages for implementing occupational disease services: establishing a clinical diagnosis, identifying workplace exposure, establishing the relationship between exposure and clinical diagnosis, determining the magnitude of exposure, identifying individual factors contributing to the disease, assessing exposure outside the workplace, and confirming the diagnosis of occupational disease.[20]

Silicosis is diagnosed through medical history, radiological examination, and exclusion of other disorders. Occupational history is crucial in supporting the diagnosis of silicosis, while social history is also extremely useful, particularly details regarding the nature, duration, intensity, and dose of exposure in the work environment. Clinical manifestations may vary according to the disease's stage. Chronic silicosis is typically asymptomatic, with symptoms manifesting after prolonged exposure. The predominant symptom of simple silicosis-type chronic silicosis is a productive cough with mucus, caused by occupational dust exposure.[1,10]

Shortness of breath and cough with sputum are common symptoms of progressive massive fibrosis (PMF). These symptoms typically appear years after the initial exposure.[10] The symptoms of accelerated silicosis are comparable to those of simple silicosis; the only distinctions are in the duration of exposure and radiological characteristics. The more severe symptoms of acute silicosis are a result of the high exposure dose. Possible symptoms include respiratory failure, coughing, fever, and pleural pain; mortality is the most severe consequence. Particularly severe symptoms are observed in cases of silicosis that are concomitant with other diseases affecting the ballast.[1,10] According to a certain study, 35.6% of patients with silicosis reported breathing symptoms of three or more.[4,10,18]

Especially thoracic photographs, as shown in Figure 2, radiologic examination are necessary for diagnosing silicosis. The thoracic image of simple silicosis reveals various diffuse nodules ranging in diameter from 2 to 5 mm, which are mainly found in the upper airways. Silicosis is characterized by the eggshell calcification of the hilum lymph nodes that circle the nodes. On PMF radiographs, large opacities manifest as nodules exceeding 1 cm in diameter.[5,6,21] Accelerated silicosis is characterized by the appearance of opacities more rapidly than simple silicosis.[1,5]

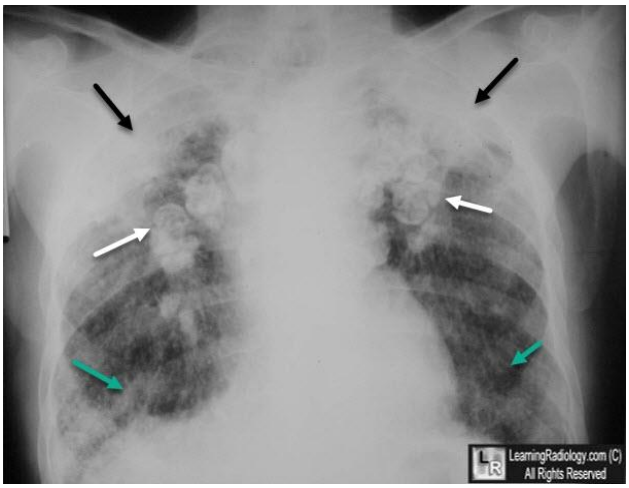

Figure 2: X-Ray of Silicosis: Progressive Masive Fibrosis with Egg Shell Features[5]

Table 1 presents the radiological characteristics of silicosis as classified by the ILO. These characteristics include size, shape, and profusion category (density/homogeneity). Good photo quality is required prior to interpretation. Size and shape are subsequently used to categorize minor opacities. The diameters of small opacities are classified as p (1.5 mm or less), q (1.5-3 mm), or r (3-10 mm). Small irregular opacities were categorized as s, t, or u using the same criteria as small round opacities.[1]

Table 1. Radiological Classification of Silicosis According to the International Labor Organization[1]

Category	Scale
SMALL OPACITY (<1cm)	
Profusion Major Scale	
0	0/-, 0/0, 0/1
1	1/0, 1/1, 1/2,
2	2/1, 2/2, 2/3
3	3/2, 2/3, 3/+
Size and Round Shape	
p	≤1–5 mm
q	1.5–3 mm
r	3–10 mm
Size and Irregular Shape	
s	≤1–5 mm
t	1.5–3 mm
u	3–10 mm
LARGE OPACITY (>1cm)	
A	One consolidation measuring 1–5 cm in diameter/multiple consolidation, each exceeding 1 cm in diameter.
B	One or more consolidation of a greater diameter/ exceedingly numerous in comparison to category A, with ridge area not surpassing the right upper lung field area.
C	One or more consolidation that surpass the area of the right upper lung field or the right third of the field in terms of total area.

On the basis of a 4 point major category scale (0–3), numerous minor opacities were categorized into 3 subcategories per major category, for a total of 12 points: 0/- to 3/+. The significant opacities were classified as A, B, and C. On the basis of their location, width, extension, and degree of calcification, pleural abnormalities were categorized.[1]

Opacities in centrilobular nodules, bilateral ground-glass opacities, and regions of inhomogeneous consolidation or homogeneous ground-glass opacities resulting from intralobular septal thickening are visible on High-Resolution Computed Tomography (HRCT) images. This modality may be employed in situations characterized by uncertainty. Research indicates that HRCT exhibits greater sensitivity in detecting specific conditions, including nodular changes in the lung parenchyma, PMF, bullae, emphysema, pleural, mediastinal, and hilus changes in silicosis, compared to conventional radiology.[2,5]

Magnetic Resonance Imaging (MRI) and other pulmonary imaging modalities aid in distinguishing between PMF and lung malignancy. Positron Emission Tomography (PET) modalities assist in distinguishing chronic lung cancer from active inflammation.[8] Patients with silicosis are depicted in HRCT images in Figure 3.

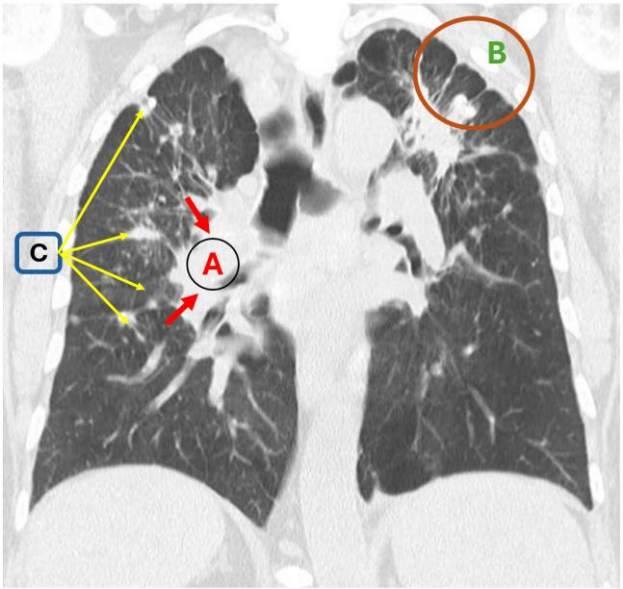

Figure 3. High-Resolution Computed Tomography of Silicosis: Progressive Masive Fibrosis[8]
(A) Confluent Reticulation; (B) Mosaic Pattern of Attenuation; (C) Ground glass Opacities

A histopathological analysis of the lung affected by silicosis reveals nodules measuring 3 to 6 mm in diameter, which change color to black or red upon exposure to purified silica. The initial lesion may have a noduled appearance due to macrophage dust aggregates enveloping collagen. Silicotic nodules are composed of collagen filaments at their core, enveloped by macrophage dust. Necrosis, granulomas, and giant cells are all indicative of tuberculosis. Periodic acid-schiff staining indicates thickened intralobular septa, and lesions are visible radiologically when nodules are predominately hyaline.[8,22] The histopathological characteristics of silicosis are shown in Figure 4.

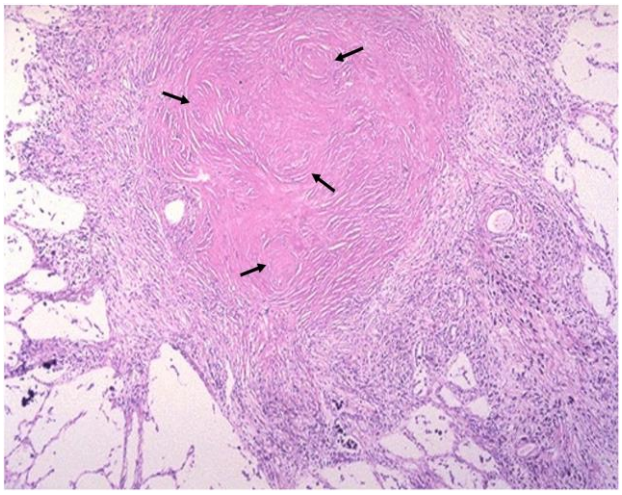

Figure 4. Histopathology of Silicosis: Coalescent Fused Silicotic Nodule have a Whorled and Hyalinized Appearance[22]

The clinical assessment of patients with silicosis is supported by pulmonary function tests, including spirometry and diffusion capacity. For it to evaluate or identify the health consequences of occupational dust exposure, spirometry is vital. Spirometry may yield normal tests, obstruction, retriction, or a combination of the two may indicate aberrant results. The prevalent abnormality is obstruction.[8,17]

In 2015, 17 individuals (71.4%) with more than five years of work experience had impaired lung function, while 6 individuals (28.6%) had normal lung function, according to research conducted by Sulfikar. Among those with less than five years of work experience, 18 individuals (94.7%) had impaired lung function and 1 individual (5.3%) had normal lung function. A correlation between working hours and the

occurrence of impaired pulmonary function has been established in accordance with the findings of this study.[7,23]

The study by Marini et al involving employees in the ceramic industry revealed a correlation between the duration of work and the prevalence of pulmonary restriction. In comparison to those who had worked for less than 5 years, the likelihood of developing pulmonary restriction was 19 times higher among subjects with more than 5 years of work experience. Furthermore, individuals with over five years of work experience exhibited reduced values for Forced Vital Capacity (FVP) and Forced Expiratory Volume in the one second (FEV1). Fibrosis and silica nodules, which impede lung development and decrease, are responsible for this.[8]

MANAGEMENT AND PREVENTION

At this time, there is no known effective treatment for silicosis. The treatment remains supportive, adjuvant, and symptomatic. Supportive treatment includes bronchodilators, oxygen supplementation, and infection prevention. Pharmacological treatments include aluminum citrate and corticosteroids. Steroids can reduce the pulmonary inflammatory response and improve pulmonary function. In theory, inhalation of aluminum citrate powder reduces the solubility of silica particles in the airways by coating them.[18,24]

Protecting the silica surface from tissue injury, Polyvinyl pyridine n-oxide (PVNO) functions as a hydrogen acceptor. Free radicals are decrease and DNA damage is prevented by PVNO. The kidneys and liver, nevertheless, are profoundly harmed by PVNO.[16,24] Pirfenidone is currently in clinical trials as an in silicosis anti-fibrosis medication. Pirfenidone and Nintedanib showed efficacy not only in patients with IPF but also in a broad spectrum of pulmonary fibrosis conditions.[8] No one of the therapies mentioned above target silica particles directly.[4]

OLD's management consists of both occupational and medical management. Standard operating procedures, service standards, and professional standards are adhered to when administering medical care. The treatment for OLD is not well-defined. Occupational management comprises community-based and individual-based occupational management, encompassing activities such as early detection and prevention of occupational diseases, assessment of work eligibility, determination of return to work, and evaluation of lung disability.[20]

Upregulation of antioxidant production can protect against ROS induction by silica, according to the study. According to Sato et al., the potent antioxidant heme oxygenase ((HO)-1) can inhibit the activation of ROS. Murine et al performed to evaluate the use of bone marrow mononuclear or mesenchymal cells for silicosis therapy showed reduced inflammation and enhanced lung function. Morales et al. described how the administration of mononuclear and mesenchymal cells derived from bone marrow improved patient outcomes and tolerance.[18]

The information available to OLD patients regarding pulmonary rehabilitation is limited. The study by Ochmann et al. lasted 4 weeks and involved 263 patients with OLD who were subsequently followed for 3 to 12 months. A comprehensive assessment was conducted on the patients, including an examination of their pulmonary function, maximal exercise capacity, skeletal muscle strength, respiratory symptoms, exacerbations, quality of life, melancholy, and medical consultation.[25,26]

Rehabilitation resulted in enhancements to both the 6-minute walk test and skeletal muscle strength. Treadmill, ergometer, skeletal muscle training, Nordic Walk, breathing exercises, rest techniques, and nutritional education were among the exercises provided. Four evaluations were conducted: on 1st and 2nd of training, on the final day of training, at 3rd month follow-up, and at 12th month follow-up. Patients with silicosis exhibited notable enhancements in pulmonary function, as evidenced by improvements in FEV1 and FVC values.[25,26]

Although silicosis is an incurable illness, its occurrence can be circumvented. To mitigate the progression of the illness, it is critical to eradicate the origin of the exposure. Efficient reduction of dust

levels in the workplace is achieved through the implementation of water-based material processing techniques, which effectively mitigate dispersed dust. Workers must utilize protective equipment if particulate levels continue to be elevated. Working environment surveillance can aid in the prevention of silicosis. In industries where dust cannot be controlled, such as sandblasting, employees must don hoods or air filter equipment.[17]

Treatment is less significant than prevention. The prevention of OLD requires the collaboration of industry management, the government, and employees. Tertiary prevention, secondary prevention, and primary prevention are the three phases of prevention initiatives. Primary prevention represents the most efficacious approach to prevention. Preventing workplace exposure to sensitizing agents is the objective of primary prevention.[3]

Personal Protective Equipment (PPE), regulation, screening of potential workers, substitution, elimination, ventilation, and modification are the fundamental principles of primary prevention. Aiming to prevent the advent of disease, primary prevention entails the reduction and elimination of exposure to hazardous substances. This is accomplished through the use of PPE, the avoidance of causative substances, and the enhancement of workers' capabilities to mitigate risks prior to sensitization.[3] Figure 5 shows the concept of OLD prevention.

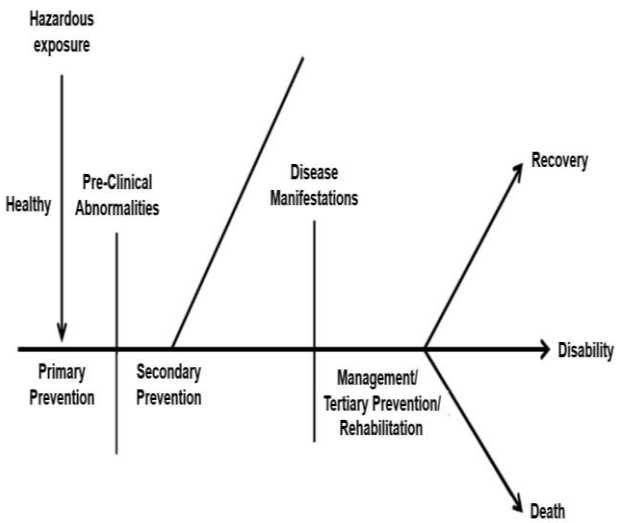

Figure 5. Concept of Occupational Lung Disease Prevention[3]

The objective of secondary prevention is to evaluate the efficacy of interventions and detect diseases at an early stage through the detection of preclinical changes. Secondary prevention includes routine assessments, which comprise spirometry, questionnaires, physical examinations (especially pulmonary examinations), and radiological examinations.[3]

Regular checks are performed at specified time intervals, differing in frequency and scope according to the characteristics and magnitude of the potential hazards. Tertiary prevention endeavors to reduce complications, prevent disability, and enhance quality of life so that individuals can lead typical lives and obtain social acceptance. As in the case of asbestosis, the principle of tertiary prevention is to limit the physical and social impairment caused by the occupation that results in symptoms, mutation, rehabilitation, and compensation for permanent disability.[3]

Workers exposed to silica should be routinely photographed of the thoracic region. Sandblasting employees every 6 months and other employees every 2 to 5 years for the purpose to detect the disease early. X-ray of the chest that reveal silicosis are advised to be taken to prevent silica exposure.[17] Certain nations implement regulations regarding RCS limits, referred to as Time-Weight Average (TWA), which computes the mean of the particulate concentrations in the air permitted for the duration of 5 working days. Globally, the RCS limit is approximately 0.1 mg/m^3 to 0.05 mg/m^3. According to NIOSH, laborers continue to be at risk for developing chronic silicosis beyond these limits.[8]

Limiting exposure to silica and ceasing smoking are important factors to consider. Since individuals with silicosis are at an increased risk of developing pulmonary tuberculosis (PTB), annual screening is recommended.[17] Transplantation of the lungs is one treatment option for severe silicosis. Patients with silicosis who underwent lung transplantation had a maximum 1-year survival rate of 88% and a maximum 3-year survival rate of 76%.[27] The extent of the lesions, the tissue response to silica,

the extent of fibrosis lesions, the progression of the disease, and organ failure all influence the prognosis. Using proper education, employers and employees can prevent the development of silicosis.[3]

THE IMPACT OF SILICOSIS ON THE BODY

Silicosis and PTB often occur together. Radiologic examination and sputum examination are done to check for PTB involvement. Patients with silicosis have a 3 to 10 times greater risk of developing PTB. The mechanism that increases the susceptibility of silicosis patients to PTB is recurrent injury to alveoler macrophages due to the toxic effects of silica. This mechanism causes alveoler macrophages to have no defense capability when assaulted by Mycobacterium tuberculosis (MTb).[8,28]

Active PTB among employees may surpass 20% in cases where the community has a high prevalence of PTB.[20] Due to the high incidence of PTB among silicosis laborers, chemoprophylaxis has become necessary to prevent PTB. Isoniazid administration guidelines for PTB prophylaxis recommend PTB screening for silica-exposed laborers and chemoprophylaxis for latent PTB.[8] The South African Guidelines for Isoniazid Preventive Therapy in Patients with Silicosis propose an isoniazid dosage of 5 mg/kg bw. Daily administration of 25 mg of pyridoxine is concurrent with the use of isoniazid.[29]

Sarcoidosis has been observed to be associated with silica exposure, according to epidemiologic investigations. According to a study of 2,187 silica-exposed laborers, 3.94 percent had sarcoidosis. The initial phases of silicosis and sarcoidosis exhibit comparable pathological and radiologic features, leading to potential confusion.[3] There appears to be a correlation between silica exposure and autoimmune diseases, specifically Rheumatoid Arthritis (RA) and Systemic Sclerosis (SS), according to epidemiologic reports that have incorporated meta-analyses.[3,30]

Exposure to silicosis triples the relative risk of developing RA compared to those who are not exposed. The incidence of SS is elevated in those who are exposed to silica. An increase in Antineutrophil Cytoplasmic Antibody-Associated Vasculitides (ANCA-AAV), an autoimmune disease, has been documented in silicosis accompanying RA and SS.[3,30]

The International Agency for Research on Cancer (IARC) has categorized RCS as carcinogenic, and silica was categorized as a group 1 carcinogen in 1997. Survivors of silicosis have an elevated risk of developing lung cancer.[3] In a study involving 1,681 cases of lung cancer and 2,053 controls, Kachuri et al. discovered that patients who had been exposed to silica had an increased risk of developing lung cancer. A silica exposure duration exceeding 30 years was found to be correlated with an elevated risk of developing Lung Cancer (LC), whereas individuals with minimal silica exposure for over 30 years did not exhibit any risk factors for developing LC.[18,31]

According to the findings of Kachuri et al., the combination of silica exposure and smoking among employees increases the risk of developing LC. It is advised by the National Comprehensive Cancer Network (NCCN) that smokers who have been exposed to carcinogens in the workplace undergo HRCT screening as early as 50 years of age. Recurrent inflammation causes LC in silicosis, which raises the danger of genotoxic damage. Neoplasmic alterations are facilitated by recurrent inflammation, which operates via the replication of oncogenes, inhibition of cell growth inhibitors, and reduction in tumor cell extravasation, metastasis, and angiogenesis.[18]

As a result of scarring and lung injury, PMF obstructs air exchange and can lead to complications, most notably Pulmonary Hypertension (PH) and right heart defects. A correlation was identified in a meta-analysis between silicosis and cardiac failure. Individuals who have silicosis are more susceptible to developing Right Heart Failure (RHF). The preliminary hypothesis posits that the presence of silica induces inflammation and compromised vascular circulation. The development of ulcers and significant fibrosis in the lungs may lead to the occurrence of pneumothorax as a complication.[3,32,33]

CONCLUSION

The most prevalent occupational lung disease caused by silica particle inhalation is silicosis. Silicosis has three subtypes based on severity, duration, and exposure dosage: chronic or simple silicosis, accelerated silicosis, and acute silicosis. It is caused by a malfunction in the defense mechanism of alveolar macrophages. Workers such as sandblasters, construction workers, stonesetters, and employees in glass, cement, and ceramics factories are at a heightened risk for developing silicosis. Diagnosis involves a combination of clinical history, physical examination, and radiological assessment. Currently, there is no effective treatment for silicosis. Additionally, silicosis can lead to pulmonary tuberculosis, autoimmune disorders, and lung cancer. Improving prevention efforts at the primary, secondary, and tertiary levels require collaboration among industrial management, the government, and workers. Key measures include limiting silica exposure, ceasing smoking, and undergoing routine PTB screening.

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