



“PNEUMONOLTRAMICROSCOPICSILICOVOLCANOCONIOSIS”

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Abstract: -

PNEUMONOLTRAMICROSCOPICSILICOVOLCANOCONIOSIS the biggest English word, has 45 letters and is used to describe silicosis, a lung condition brought on by breathing in extremely fine silica dust that inflames the lungs. Silicosis is a type of occupational lung illness brought on by breathing in crystalline silica (also known as silicon dioxide, or SiO₂). Workplaces include quartz crushing plants (which grind quartz into flour), ceramic, glass, stone quarries and mines, among other places. This fibrotic and inflammatory process involved silicosis is a result of both humoral and cell-mediated immunological responses. This silica dust can result in fluid retention and scar tissue formation in the lungs, which impairs breathing. Four different forms of silicosis exist: classical, complicated, accelerated and acute. The idea that the pathophysiology of silicosis (black lung disease) involves interactions between silica and pulmonary macrophages. Silica probably affects macrophages via changing how they operate while they are still living rather than just by disrupting them. The release of mediator molecules from macrophages, including interleukin-1 (IL-1), that change other cells behaviour and function is thought to be promoted. In growing silicotic nodules, lymphocytes and macrophages can be seen in close proximity to one another, and bronchoalveolar lavage samples from animals and people who have been exposed to silica dust include higher proportions of lymphocytes than normal. The macrophage has been identified as a key contributor to the accompanying fibrosis to silicosis. Silicosis is a chronic condition that is incurable. Therapies alleviate symptoms and manage infections that silicosis sufferers are susceptible to. Any person with silicosis may experience a number of complications: increased chance of TB, COPD and lung infections. Massive progressive fibrosis, scarring and stiffness of the lung, which makes breathing difficult. Increasing massive fibrosis can manifest as simple or accelerated silicosis, with the accelerated variant with respiratory failure being more typical.

Keywords: - Crystalline silica, Pneumonoultramicroscopicsilicovolcanoconiosis, Silica, Silicosis, Quartz, Black Lung Disease, Interleukin, Lymphocytes, Fibrosis, Macrophages, Silicotic Nodules, COPD, Tuberculosis.

1.Purpose: -

The program's purpose is to educate healthcare professionals on the prevalence of pneumonoultramicroscopicsilicovolcanoconiosis.

2.Objectives: -

- i. Discuss the prevalence of pneumonoultramicroscopicsilicovolcanoconiosis.
- ii. Review the etiology of pneumonoultramicroscopicsilicovolcanoconiosis.
- iii. Provide an explanation of the pathophysiology of pneumonoultramicroscopicsilicovolcanoconiosis.
- iv. List the sign and symptoms of pneumonoultramicroscopicsilicovolcanoconiosis.
- v. Summarize the diagnosis and treatment options for pneumonoultramicroscopicsilicovolcanoconiosis.

3.Introduction: -

Silicosis also known as Grinder's disease, Black lung disease and Potter's rot. Silicosis is a type of pneumoconiosis that commonly affects miners and other workers exposed to free crystalline silica dust for an extended period of time. Worker exposure to crystalline silica damages the lung as silica particles are trapped in tissue, fibrotic nodules and scarring form around them. Silicosis is the medical term for this lung fibrotic disease. When brought on by a specific exposure to the fine silica dust found in volcanoes, this disease's full name is Pneumonoultramicroscopicsilicovolcanoconiosis (1). It is a crystalline type of silicon dioxide (SiO₂) that includes tridymite, quartz, and cristobalite. The most prevalent variety quartz, is a key ingredient in several types of rocks, including granite, slate, and sandstone (2).

4. Background: -

The illness known as pneumonoultramicroscopicsilicovolcanoconiosis (Black Lung Disease) is brought on by cold miners inhaling excessive amounts of coal mine dust, silica dust, or quartz dust. Silicosis, often known as Black Lung Disease, is a lung condition. Black Lung Disease was all but exterminated in the 1990s, but it has suddenly returned with a vengeance (Black Lung Disease Rates Soar, 2014). The Coal Mine Health and Safety Act was passed in 1969, and the prevalence of black lung disease reduced by 90% between 1969 and 1995, according to the CDC (2012). Now, there are more black lung cases than ever before in the United States (3).

According to data from the National Institute for Occupational Safety and Health (NIOSH), the deadliest form of the disease began to manifest itself in 2012 at a rate that had not been observed since the early 1970s. NIOSH found that despite having access to dust suppression technology, mine personnel recent research indicates that dust exposures have not been sufficiently controlled and that a significant fraction of U.S. coal miners continue to develop progressive massive fibrosis (PMF) linked to black lung disease despite OSHA guidelines for best practices for dust control in coal mining (CDC, 2012). Black lung disease is an advanced and fatal form of black lung that has few therapeutic choices and no known cure. According to NIOSH experts, the only cause of fatality for coal miners is excessive coal mine dust inhalation. Annually, black lung in the United States, disease-related pneumoconiosis claims the lives of over 1000 miners (CDC, 2008) (3).

5. Etymology: -

The biggest English word, PNEUMONULTRAMICROSCOPICSILICOVOLCANOCONIOSIS, is a tremendous wonder. NOO-muh-noh-UL-truh-MY-kruh-SKOP-ik-i-koh-vol-KaY-no-KOHnee-O-sis is its phonetic analysis. It denotes that breathing in very fine silica dust, which causes lung illness, an infection of the lungs the acronym PNEUMONO+ULTRAMICROSCOPIC+SILICO+VOLCANO+CONIOSIS stands for the pneumatic system that contains the lungs, while ULTRAMICROSCOPIC denotes ultra-fine matter, SLICO denotes silica, VOLCANO denotes a volcano, and CONIOSIS denotes a sickness brought on by inhaling dust. It is an English term that designates a lung condition also known as silicosis. The Oxford English Dictionary lists it as the longest word in the English language. It is described as “an artificial long word said to mean a lung disease caused by inhaling very fine ash and sand dust”. Everett M. Smith, president of the National Puzzler’s League (N.P.L.), created this word in 1935 during the organization’s annual convention. The phrase was in the headline of an item headlined “Puzzlers Begin 103rd Session” that appeared in the New York Herald Tribune on February 23, 1935. Through understanding the 45-Letter Word. The National Puzzler’s League recognised pneumonoultramicroscopicsilicovolcanoconiosis as the longest word in the English language at the opening session of its 103rd semi-annual meeting, which was held yesterday at the Hostel New Yorker. Electrophotomicrographically had previously held that distinction. The answerers went on to explain that the 45-letter word is the name of a particular type of pneumoconiosis brought on by incredibly tiny particles of silica volcanic dust (4).

6. Clinical Forms: -

Table 1: -Schematic comparison of the clinical forms of silicosis (Source- Calderon et.al, 2018)

Type	Lesion index	Appearance	Initial symptoms	Tests respiratory function	Type opacity	Complication
Classical	Socavón: 8 to 10 years Tajo: 15 years	Slow	Asymptomatic or minimal dyspnea	Controversial: minor restriction or obstruction	Small: 0.5 to 5 mm from small to medium size	20 to 30% drift to complicated silicosis
				Slight decrease in CO diffusion		
Complicated	5 to 10 years	Slow	Productive cough or minimal dyspnea	Marked restriction and decrease in O saturation and diffusion	Big	Bacterial infection, mycobacteria or N. asteroid
					≥10 mm	Pneumothorax It ends in respiratory failure or cor pulmonale
Accelerated	4 to 6 years	Brusca	Intense dyspnea	Rapid deterioration towards the restriction	Small 0.5 to 5 mm Abundant amount	About infection or association with autoimmune disease: scleroderma or RA in the Caplan Collinette syndrome
Acute	6 m to 2 years	Violent	Dyspnoea	Marked restriction	Small from 1 to 5 mm, but very abundant	Alveolar proteinosis correlated to the clinical picture
						Mycobacteria
						Extra-pulmonary 'silicosis' nodules

7.Etiology: -

Exposure to crystalline silica dust can cause a range of severe illnesses, such as silicosis, which causes the lungs to become scarred and cause severe, irreversible shortness of breath. In certain cases, lung transplants are required or the patient would die (15). Inhaling quartz, silica, or coal mine dust can result in black lung disease. Several kinds of dust enter the bloodstream when they are inhaled by a person and pass through the lungs' alveoli. The effects of the dust's introduction into the bloodstream include the white blood cells to release cytokines, which are peptides, proteins, and glycoproteins. In an immune response that stimulates the movement of cells towards sites of inflammation, trauma, and infection, cytokines are cell signalling molecules that help in cell-to-cell communication, according to Mandel (2013). This stimulation of fibroblast occurs as a result of the immune response. Fibrosis results from fibroblast stimulation. Fibrosis causes fibrotic disease, which causes the lungs to scar excessively as a result of extracellular matrix synthesis, deposition, and contraction (Leask, and Abraham, 2004). According to Gurvan et al. (2010), extracellular matrix is a group of chemicals that cells produce outside of their cells and which give the cells around them structural support. The walls of the lungs' air sacs become inflamed due to fibrosis. The tissue between the air sacs then scars, making the lungs rigid. The American Lung Association (2015) states that it may take years for persons who breathe coal dust to exhibit any symptoms. Yet as time passes and the coal dust becomes deeply ingrained in the lung, it finally hardens the lung. Breathing becomes more challenging and gets worse with time as the lung hardens (3).

7.Pathophysiology: -

The particular mechanism that triggers the development of pneumoultramicroscopic silicovolcanoconiosis also known as silicosis is still unclear (1). What is known is that, Particles smaller than ten microns are the most dangerous to humans (6). The tiny alveolar sacs and tubes of the lungs, which are where oxygen and carbon dioxide gases are exchanged, can get deeply encrusted with these microscopic silica dust particles if they are breathed. Consequently, the lungs are unable to clear the air of the dust by coughing up phlegm. Leukotriene B4, interleukin-1, and other cytokines are released by ingested macrophages in response to the inhalation of fine silica dust particles, which leads to an inflammatory reaction. Leads to fibrosis and the development of nodular lesions by promoting fibroblast proliferation and collagen production around the silica particles (2). Crystalline silica appears to have inflammatory effects that are mediated by Nalp3 in the inflammasome (7). In nodular silicosis, the characteristic lung tissue pathology consists of fibrotic nodules with concentric collagen fibre arrangements that resemble a "onion skinned" a central hyalinization zone, and a cellular peripheral zone, as well as light birefringent particles that can be seen under polarised light. Microscopic pathology reveals a periodic acid-Schiff pattern in acute silicosis, a cellular infiltration of the alveolar walls and positive alveolar exudate (alveolar lipoproteinosis) (2-8).

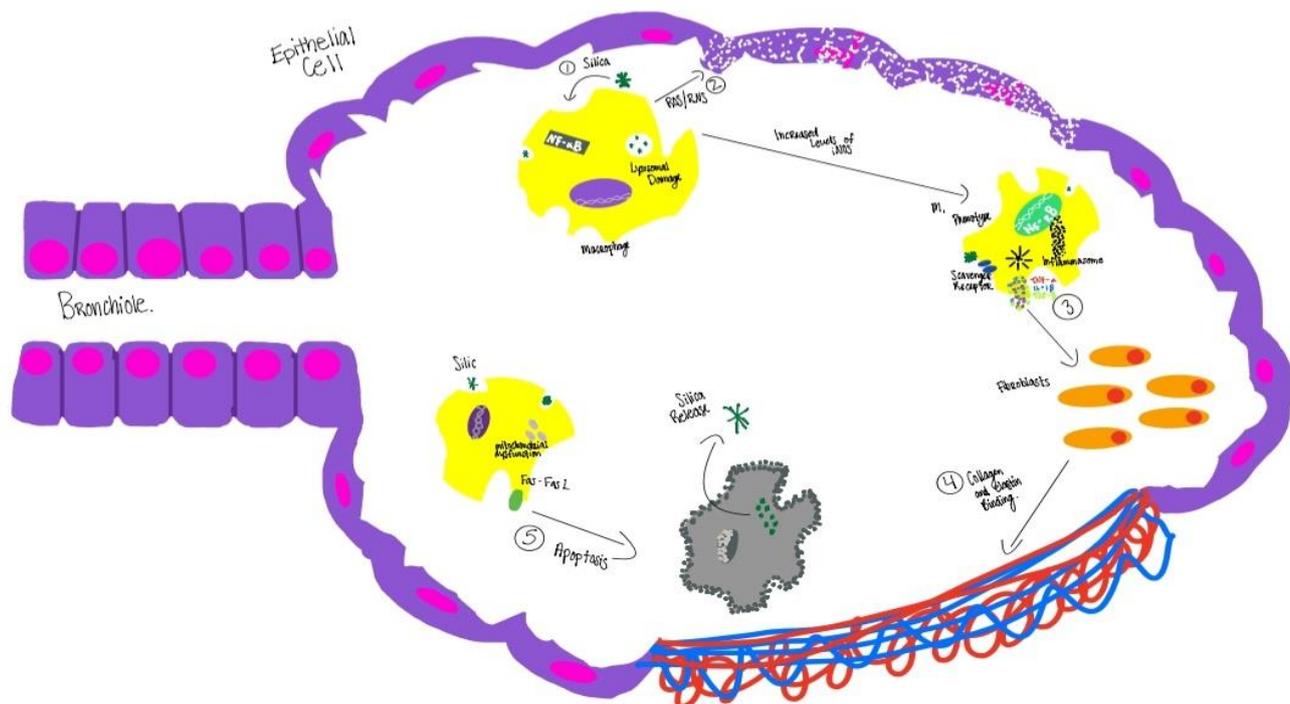


Fig 1:- The five phases of lung damage.

(Source- Kowalewski, 2022)

The Five Phases of Lung Damage:-

- i. Direct cytotoxicity
- ii. Generation of reactive species
- iii. Production of inflammatory mediators
- iv. Fibrosis
- v. Cell death occurs by apoptosis

- **Direct cytotoxicity:** - Long-term inhalation of potentially hazardous substances, such as silica, causes silicosis, an inflammatory lung condition that has been linked to cytotoxicity (6).
- **Generation of reactive species:-** Reactive oxygen species (ROS) and reactive nitrogen species (RNS) are examples of highly reactive compounds. ROS include peroxides, superoxide and hydroxyl radicals. Examples include nitric oxide, enzymes, and proteins of RNS. The biological defence system against microbial diseases essentially gave rise to both species, although they can also unintentionally target other harmful substances like particles. Silica particles and alveolar macrophages work together to cause respiratory bursts. Increased oxygen consumption and the production of ROS, which impairs the survival of lung cells. Nitric oxide is a crucial component in this procedure due to its significance in the pathophysiology of silicosis. The nitric oxide synthase (NOS) enzyme, which creates nitric oxide, transforms L-arginine into L-citrulline. This results in a response from Peroxynitrite, a product of superoxide, harms DNA and mitochondria (9-6).
- **Production of inflammatory mediators:** - Macrophages are the first cells to engage with silica particles, and this interaction can activate a number of extracellular signals that lead to the polarisation of these cells. M1 macrophages are responsible for the inflammatory reactions (6).
- **Fibrosis:** - After exposure to silica, inflammation worsens when the macrophage receptors are blocked. Fibroblasts must be delivered to the wounded area because of this. Silica particles greater ability to cause tissue injury, when combined with a variety of additional measures, is in charge of the remodelling of the lung and granuloma function, both of which impact lung function (6).
- **Apoptosis:** - Moreover, after apoptosis, cells release chemotactic molecules that attract other inflammatory cells, so escalating the inflammatory response. Silica particles are released back into the lungs when macrophages go through apoptosis, continuing the ongoing process of tissue destruction (6).

8. Sign and Symptoms: -

Pneumonoultramicroscopicsilicovolcanoconiosis is slow to develop; signs and symptoms may not appear until years after exposure. Signs and symptoms include the following: -

- Dyspnea (Shortness of breath)
- Cough (persistent and sometimes severe)
- Fatigue
- Tachypnea (Rapid breathing)
- Shallow breathing
- Fever
- Cracked nails (Gradual dark shallow rifts in nails eventually leading to cracks as protein fibers within nail beds are destroyed.)
- Anorexia
- Hoarseness
- Malaise (feeling of discomfort or lack of illness)
- Sleep problems
- Increased susceptibility to pulmonary tuberculosis

In advanced cases, the following may also occurs:

- Cyanosis (bluish skin)
- Cor pulmonale (right ventricle heart disease)
- Lung edema
- Inflamed lungs
- Scarred lungs
- Swollen lymph nodes
- Pleural Effusion
- Respiratory failure
- Lung cancer
- COPD (3)

9. Illness Associated with Silicosis: -

There are numerous diseases that have been linked to various silicosis forms, including the following-

- a) **Cancer-** Crystalline silica has been classified as a human Group-1 carcinogen by the International Agency for Research on Cancer (IARC). During a lifetime of exposure at the current OSHA standard of 0.1 mg/m³, the probability of developing silicosis (ILO category 1/1 or more) is anticipated to rise by 30% or even more. The link between exposure to crystalline silica and stomach or esophageal cancer is not well established. In a case-control study, individuals with a history of occupational exposure to crystalline silica were much more likely to develop pathologically confirmed stomach cancer than the controls. There are several noteworthy links between crystalline silica exposure and esophageal cancer, digestive system malignancies, and intestinal or peritoneal cancer. (9).

- b) **Autoimmune Disorders-** Many autoimmune disorders and silica exposure have long been linked in case studies (systemic sclerosis, rheumatoid arthritis, lupus, chronic renal disease). Over the past ten years, larger epidemiologic investigations have found more and more evidence of this connection (1).
- c) **Tuberculosis-** Types of silicosis may be complicated by tuberculosis, but those with acute and rapid disease may be most at risk. Even without silicosis, exposure to silica alone may predispose someone to this infection. The most common mycobacteria are atypical mycobacteria and *M. tuberculosis* (15).
- d) **Fungal Infection-** Several fungi can cause fungal infections in silicotic lungs (silico-mycosis). A 52-year-old male with a history of prolonged silica exposure was previously shown to have chronic necrotizing pulmonary aspergillosis with aspergilloma. Although this fungus-related co-infection may be lethal, it hasn't been identified as a typical co-morbidity (9).
- e) **Chronic Obstructive Pulmonary Disease (COPD)-** Chronic bronchitis and emphysema are two illnesses that fall under the umbrella of COPD. As a result of COPD, the lungs airways become damaged, constricted, and more difficult to breathe into and out of. When compared to obstructive lung disease brought on by exposure to tobacco smoking, chronic obstructive pulmonary disease (COPD), including emphysema and chronic bronchitis, may be the result of exposure to silica and coal mine dusts. In sensitive people, this may seriously impair respiratory function (1).
- f) **Renal Diseases-** An auto immunological mechanism has been hypothesised as the cause of the association between silica exposure and kidney illness (chronic renal disease and glomerulonephritis). Over and above the 2% baseline risk of end-stage renal disease, the excess risk of end-stage renal disease attributable to a lifetime of occupational exposure is estimated to be 14% (1).

9.Potential Biomarkers of Silicosis: -

- a) **Cytokines-** By releasing a variety of mediators, including cytokines and chemokines, alveolar macrophages play a significant role in the progression of silicosis. These mediators are part of a complex network of interactions that lead to the onset of lung damage, inflammation, and possibly fibrosis. $TNF\alpha$, $TNF\beta$, $IL\alpha$ and $IL\beta$ are among the various cytokines that have been investigated as silicosis biomarkers (1).
- b) **Angiotensin converting enzyme-**Angiotensin 1 converting enzyme (ACE, peptidyl dipeptide hydrolases) is a glycoprotein that is membrane-bound and responsible for the conversion of Angiotensin 1 to Angiotensin 2 as well as for the destruction of Bradykinin. Because ACE is mostly found in the extensive capillary bed of the lungs, the serum activity of ACE in pulmonary disorders is of interest (1).
- c) **Serum copper-** It is documented in the literature that Cu has a fibrogenic characteristic and as the key pathologic alterations in silicosis involve fibrosis and the proliferation of collagen tissue in the lungs there could be probable relation with elevated levels of blood Cu (1,16).
- d) **FAS ligand (FasL)-** Silicosis is characterised by immunological anomalies, including the development of autoantibodies and autoimmune disease consequences (1).

10.Diagnosis:-

The NIOSH Coal Workers Health Surveillance Program can screen for Pneumonoultramicroscopic silicovolcanoconiosis (black lung disease) in coal miners (3). The simultaneous occurrence of the following factors serves as the basis for silicosis diagnosis-

- 1) Occupational history of crystalline silica exposure.
- 2) Characteristics radiologic findings as follows: simple chest X rays.
- 3) Ruling out other possible diseases.

➤ Occupational History: -

Because silicosis is an occupational disease, the following details must be considered:

- A detailed occupational case history must be gathered with a view to estimate accumulated exposure to crystalline silica. This is difficult job for the clinicians particularly in cases where there is frequent job change by the affected person. Nevertheless, past and present job's activities details and duration of exposure dust must be recorded. To estimate accumulated exposure to crystalline silica, a detailed occupational case history must be gathered. This is a difficult job for clinicians, especially when the affected person changes jobs frequently. Nonetheless, the details of previous and current job activities, as well as the duration of dust exposure, must be documented.
- Job description in detail.
- The usage of masks is one example of a protective measure. Other institutional practises include wet drilling, water jet cutting, providing ventilation, and removing dust, among others (10,11).

➤ Standard Chest X-ray: -

On the basis of high-quality chest X-rays, experienced and competent clinicians can diagnose silicosis. The International Labor Organization (ILO) has developed a classification system for coding radiological changes in order to standardise the diagnosis process (11).

The classification system is made up of five parts;

- i. Radiograph technical quality: -1: good, 2: acceptable, 3: poor, and 4: unacceptable.
- ii. Parenchymal changes: size, profusion, shape, and location.
- iii. Opacities-small: - They are classified as profusion (3mm to 10mm in size).
- iv. Opacity-high: - Large opacities are those with a dimension greater than 10 mm. They are further classified as A, B, and C based on their combined dimensions and location in the lungs.
- v. Pleural abnormalities: - They are classified according to their type (pleural plaques, diffused pleural thickening), location (chest wall, diaphragm, or other), presence of calcification, and extent (length of involvement of the sternum).
- vi. Symbols: - Their use is relevant because they provide useful information on dust exposure and etiological aspects of the disease, but they are not required.

In 2011, the ILO introduced the use of digitised images, and it now provides 22 standardised images. It also provides technical information on how to read radiographs using monitors. The ILO specifies the width (54 cm) of the monitors used in diagnostic radiology, luminance ratio (250 candelas/m²), and pixel size (maximum 210 microns) (10,11).

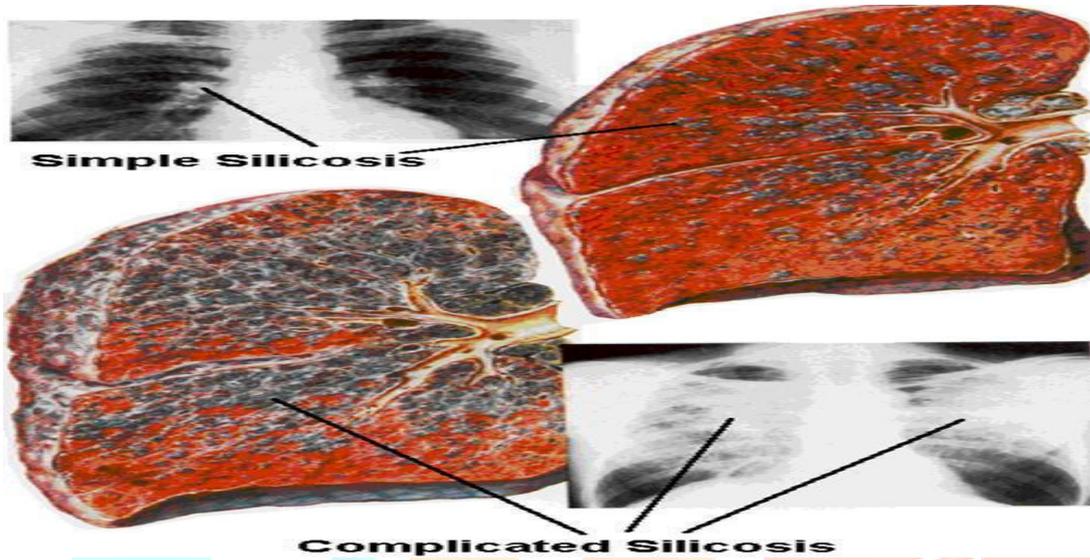


Fig 2: - Chest x-rays of the silicosis patients.

(Source- Shamim et.al, Vol.5 (Iss.7): July, 2017)

➤ **Ruling out other Diseases: -**

Several other differential diagnostic tests are performed by the clinician on a case-by-case basis to rule out other diseases. Some of these additional tests are as follows: -

- i. **Lung Function Tests: -** These tests assess your lung's ability to breathe properly and deliver oxygen to your bloodstream. Two separate tests are used to make these measurements: spirometry and diffusion capacity. They are also used to assess the extent of damage to your lungs (12).
- ii. **High Resolution Computed Tomography (HRCT): -**
The HRCT technique is helpful in identifying alterations in vascular anatomy and lung architecture, such as calcification of nodules in some lung regions. According to reports, HRCTs are more accurate than X-rays in identifying silicosis. Yet, according to various studies, the approach has more drawbacks than benefits. Standards for reading HRCTs are not sufficiently clear, which raises the possibility of diagnoses being made in circumstances when they are not. A generalised application of this approach would result in the observation of lung nodules with unknown significance, which would cloud the diagnosing process. Additionally, the employer could consider the employee unfit for the position based on these results. Hence, it is not advised to utilise it in the diagnosis of silicosis (ILO,2011) (11,19).
- iii. **Progressive Massive Fibrosis: -**
Manifests as bilateral upper lobe masses that are made up of nodules that have coalesced. Cavitation could be noticed. Fibrosis may cause these masses to withdraw towards the hilum, giving the lower lung fields the appearance of being inflated. There could be calcifications in the hilar (2).
- iv. **Histology Findings: -**
One of the most useful diagnostic techniques for identifying silica-induced lung injury is histopathological analysis of the lungs. Comprehensive pulmonary pathology assessment using when a patient is exposed to pure silica, separate nodules that are exceedingly hard and vary in colour (grey, blue, or green) have been seen in simple silicosis; nevertheless, those nodules can also be black (coal miners' silicosis) or red (silicosis in a hematite miner). Aggregates of dust-filled macrophages that are nodular to stellate in shape and gathered around a collagenous core area can be used to identify these early lesions. A decreased relative number of inflammatory

cells around the periphery over time causes the central collagen to become clearly whorled. When silica and another dust, such as iron oxide, are combined to cause mixed dust pneumoconioses, the nodules typically retain their stellate structure, although the core collagen region exhibits comparatively less of the spiral pattern that is typically seen in typical silicosis (9).

v. **A Tuberculin Skin Test Using Purified Protein Derivative(PPD):-**

Treatment for a latent or current infection is advised if the test result is positive (i.e., there is 10 mm or more of induration at the site). Sputum samples from people who received a positive skin test result should be examined under a microscope for acid-fast bacilli (AFB) and cultured for AFB to detect active illness (2).

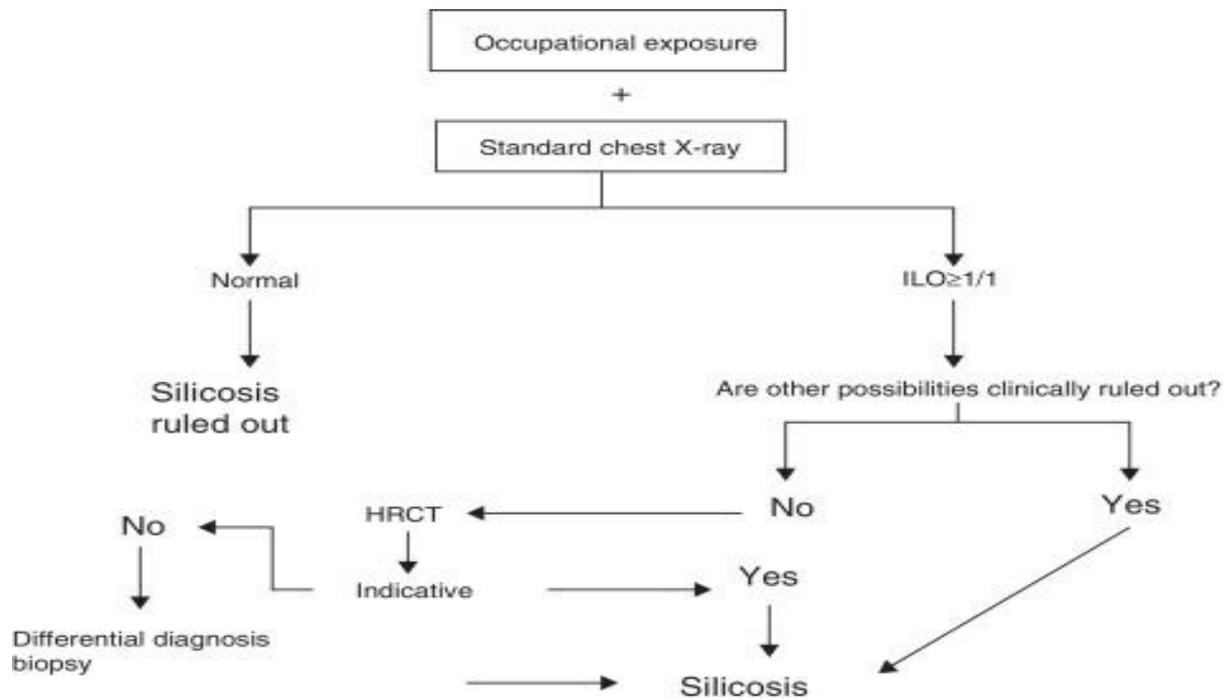
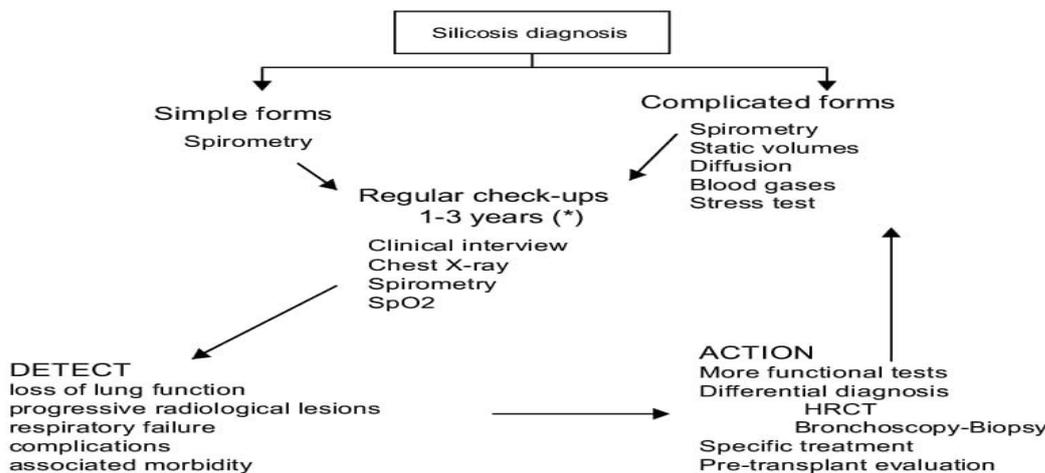


Fig 3:- Algorithm for diagnosis of silicosis.

(Source- Álvarez et.al, 2015)



(*) Order ITC 2585/2007 for the protection of workers from dust, with respect to silicosis

Fig 4: - Algorithm for monitoring silicosis patients.

(Source-Álvarez et.al, 2015)

11.Prevention: -Table 2: - Silicosis prevention
(Source- Kowalewski, 2022)

1. Primary prevention	<ul style="list-style-type: none"> ➤ Monitor levels of respirable dust ➤ Recommend personal protection measures
2. Secondary prevention	<ul style="list-style-type: none"> ➤ Monitor exposed workers ➤ Smoking cessation ➤ Monitoring for tuberculosis infection
3. Tertiary prevention	<ul style="list-style-type: none"> ➤ Avoid exposure to dust inhalation ➤ Report cases, recommend occupational disease assessment ➤ Monitoring for tuberculosis infection ➤ Treatment for airflow limitation and Respiratory failure

12.Treatment: -

The condition silicosis is chronic, progressive, and incurable. Depending on how severe it is, it can result in morbidity, disability, and even death. There is still no cure that works to reverse lesions or decrease their progression (10). Treatment options are aimed at prevention (3). The present focus of treatment is on symptom relief and avoiding complications (4). The conditions of each person's work during coal dust exposure determine the severity of their disease (The American Lung Association, 2015). However, available therapies are intended to lessen the disease's symptoms and complications. There are several treatments that are comparable to those given to people with chronic lung disorders, for example: -

1. Titrated oxygen therapy (monitor patient for decompensation)
2. Invasive mechanical ventilation
3. Inhaled short-acting bronchodilators include beta agonists for example:
 - a. Albuterol
 - b. Levalbuterol (Xopenex)
 - c. Anti-cholinergics
 - d. Ipratropium (Atrovent)

Short-acting bronchodilators alleviate dyspnea and increase activity tolerance. Systemic corticosteroids may be used as an additional treatment option to help improve hypoxemia and forced expiratory volume in one second (3). When necessary, respiratory infections are treated with antibiotics. Limiting exposure to irritants and stopping smoking are further treatments. Individuals who have silicosis are highly susceptible to getting TB. It is thought that silica prevents the body's immune system from effectively combating the TB-causing bacteria. Regular skin tests should be performed to screen for exposure to TB. Skin tests that are positive should receive anti-TB medication. Any modification to the chest x-appearance rays could indicate TB. Severe silicosis patients may require a lung transplant (14). A few experimental therapies are the inhalation of d-penicillamine, polyvinyl pyridine-N-oxide, and powdered aluminum therapy with corticosteroids. The natural tetrandine extract may slow rapid progression of silicosis (4). Nonetheless, according to the American Lung Association, 2015 demonstrates that medical management is aimed at treating the symptoms and complications of these conditions in order to reduce breathing difficulties (3).

13.Conclusion: -

As pneumonoultramicroscopic silicovolcanoconiosis (silicosis) cannot be cured, prevention is the key to controlling the condition. Silicosis remains a serious occupational health risk for workers involved mostly in mining, foundry, and construction activity. It is a significant cause of respiratory morbidity and pulmonary function decline. A frequent co-morbidity that exacerbates the silicosis disease is TB. Other significant problems include lung cancer and disorders of the connective tissue. Silicosis cannot be cured, but if you take appropriate precautions to safeguard your family and yourself, you can avoid getting it. So, it is essential to reduce exposure through a variety of preventative strategies and routine screening for early diagnosis in order to lessen the burden of silicosis globally.

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