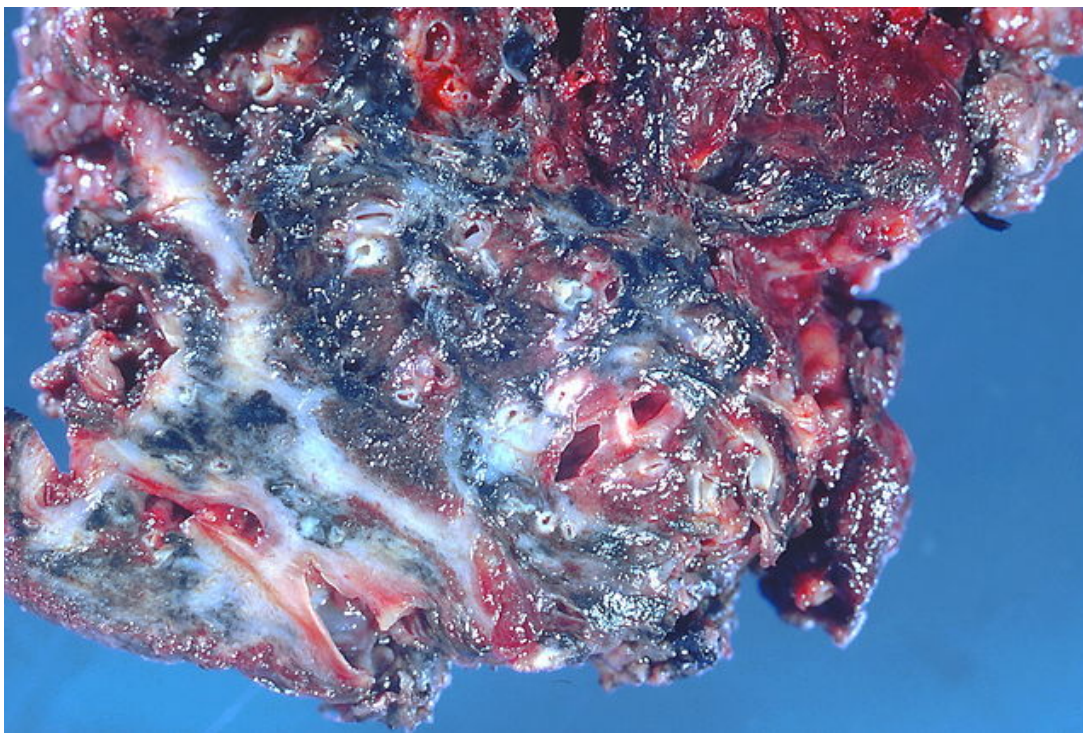


Asbestosis and Silicosis — Causes and Treatments

[See online here](#)

Occupational lung disease covers a specific area of knowledge in respiratory medicine. In these cases, information on the patients' history plays an important role in the detection of these disorders, guiding investigations and management. Awareness of this topic is essential.



General Information

Occupational lung disease is a broad term that refers to a **group of disorders in which injury to the respiratory tract has been caused by exposure to various substances in the environment or workplace**. Occupational exposure can lead to **obstructive lung disease**, as in the case of occupational lung disease, or it can lead to **restrictive lung disease**. Some cases, notably with asbestos exposure, can result in malignancy.

While there are a number of different occupational lung diseases, we will focus on two of the most common types: asbestosis and silicosis. Both of these lung diseases fall under the term **pneumoconiosis**, which refers to the accumulation of non-biologic dust in the **lungs**, triggering parenchymal fibrosis. Silica and asbestos particles are both **non-biologic fibrogenic dusts**.

Asbestosis

Definition of asbestosis

An asbestosis is a form of **interstitial pulmonary fibrosis** that can develop following the **inhalation of asbestos fibers**.

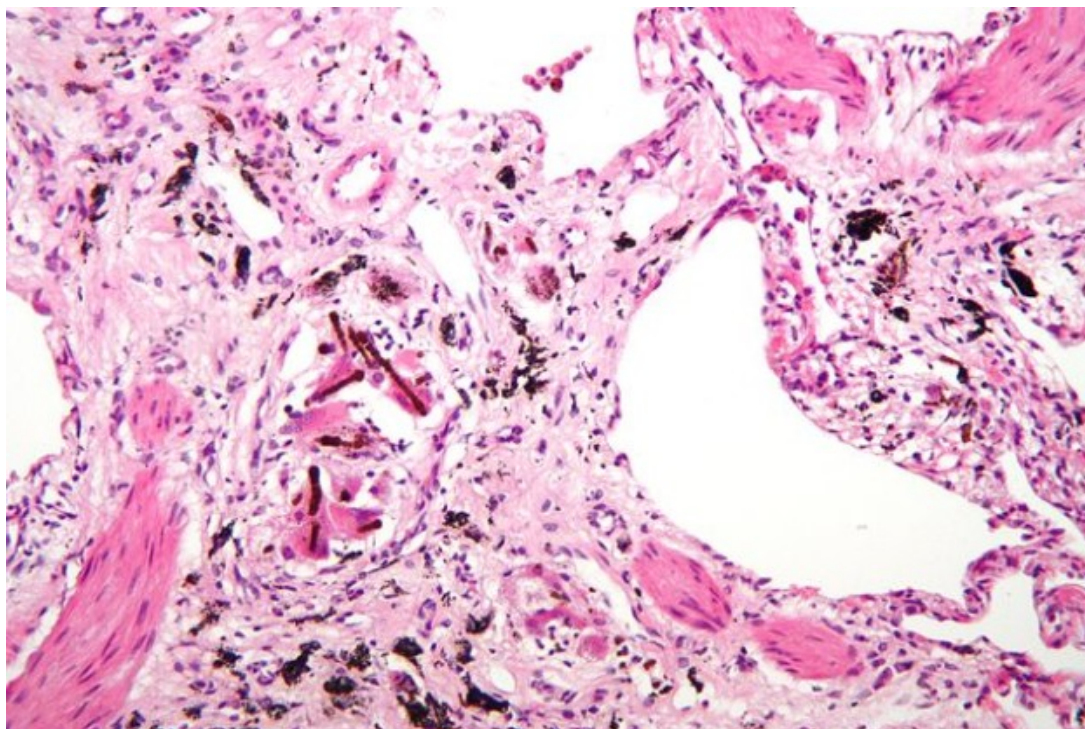


Image: "High magnification micrograph of asbestosis of the lung, with characteristic ferruginous bodies and interstitial fibrosis. Lung biopsy. H&E stain" by Nephron. License: [CC BY-SA 3.0](https://creativecommons.org/licenses/by-sa/3.0/)

Epidemiology of asbestosis

Cases of asbestosis are generally associated with **exposure to asbestos before the 1980s**—which was before regulations were introduced to curb exposure—with patients generally being **over the age of 50**. **Rates of morbidity and mortality related to asbestosis have increased in the past 30 years**. Each year, there are **about 1500 deaths** from the disease. The use of asbestos has been noticeably reduced in the United States and Europe, and it has been banned in many countries as well.

Etiology of asbestosis

Asbestosis develops due to **inhalation of asbestos fibers < 10 microns**. Some key settings where people were exposed to asbestos included **asbestos mining, asbestos milling, asbestos cement manufacture, ship or building insulation manufacture or installation, asbestos textile manufacture and working with spray products containing asbestos**. People working in these fields can also **expose family members to asbestos** fibers through contaminated work clothes and boots.

Pathology and pathophysiology of asbestosis

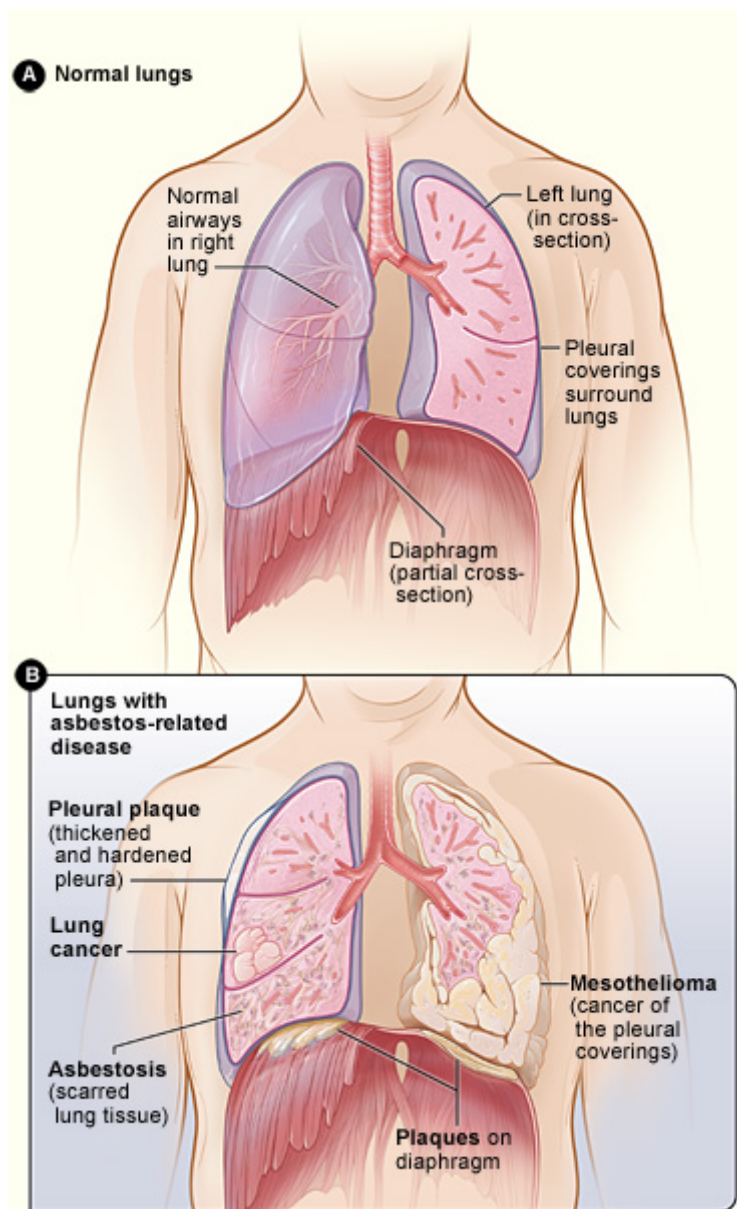


Image: "Figure A shows the location of the lungs, airways, pleura, and diaphragm in the body. Figure B shows lungs with asbestos-related diseases, including pleural plaque, lung cancer, asbestosis, plaque on the diaphragm and mesothelioma." by National Heart Lung and Blood Institute. License: Public Domain

The development of asbestosis begins with the inhalation of asbestos fibers following occupational exposure. However, the development of this disease is **influenced by a range of factors**, such as the **size and type of the asbestosis fibers, duration of exposure, the intensity of exposure**, along with individual factors relating to **genetic susceptibility and cigarette smoking**. Generally, it is said that **the higher the levels of asbestos exposure, the greater the risk of developing asbestosis**, which reflects a **dose-response relationship**. When asbestosis develops, the disease may continue to progress even when the affected individual is no longer exposed to asbestos fibers.

Asbestos fibers are **fibrogenic** and **cannot be metabolized in the body**. Following inhalation of asbestos particles, these particles enter the lungs and deposit in the alveolar

ducts. This results in a process of inflammation known as **alveolar macrophage alveolitis**. Activated macrophages release various cytokines, including tumor necrosis factor (TNF) and interleukin-1beta (IL-1), leading to the **development of fibrosis**. Fibrosis generally occurs in the lungs' lower lobes. It can become extensive, and peribronchial fibrosis may cause narrowing of the airway.

It has also been noted that smoking increases the risk of developing asbestosis. This is probably related to the impaired clearing of asbestos fibers from the lung due to the effects of smoking.

Symptoms of asbestosis

In general, a **latency period** has been described, between the development of asbestosis and the manifestation of clinical symptoms of the disease. This clinical latency period is often at least 20 years.

Patients may be asymptomatic or they may develop symptoms and signs of asbestosis. Clinical features may include:

- Progressive shortness of breath
- Cough
- Inspiratory crackles
- Cyanosis
- Clubbing

Complications of asbestos exposure

Lung cancer can result from asbestos exposure and is the most common malignancy associated with it. Usually, at least 15 to 19 years pass after exposure before there is an increased risk of lung cancer development. An increased amount of exposure is associated with a higher risk for the development of lung cancer. As mentioned before, if a person who is already exposed to asbestos also smokes, this will have an additive effect and further increase their risk of lung cancer.

Another type of malignancy that can occur is **mesothelioma**, which can affect the pleura and peritoneum. More than 80 % of mesotheliomas may be related to asbestos exposure, which is why patients with occupational exposure may be able to claim compensation. The development of mesothelioma can occur in as little as 1 to 2 years after asbestos exposure or later, up to 40 years after exposure.

Diagnosis of asbestosis

Pulmonary function tests and asbestosis

[Pulmonary function tests](#) can be used to demonstrate a pattern of **restrictive lung disease** with reduced functional vital capacity (FVC) and a normal FEV1/FVC ratio. They will show a **reduction in lung volume and diffuse capacity**, and there may be some **mild airflow obstruction**. In some cases of asbestos exposure and smoking, an obstructive pattern may be discovered (reduced FEV1 and reduced FEV1/FVC ratio).

Radiology



Image: "Chest X-ray in asbestosis shows plaques above diaphragm" by Clinical Cases. License: [CC BY-SA 2.5](#)

A chest x-ray is the preferred initial investigation method for asbestosis. On a chest x-ray, the **hallmark for asbestos exposure is the discovery of pleural plaques**. Pleural plaques are thickenings of the parietal pleura, which are especially found near the lower lung fields, cardiac border, and diaphragm. However, the presence of pleural plaques does not indicate impairment of the lung function.

In cases of asbestosis, a chest X-ray may reveal linear streaking at the lower zones of the lungs, representing interstitial fibrosis, and in advanced cases, honeycomb changes may be detected. The presence of pleural plaques, being indicative of asbestos exposure, helps with the diagnosis.

The **best imaging test** to diagnose asbestosis is by using a **high-resolution CT scan** of the chest. This test is more sensitive compared to a chest X-ray for asbestosis diagnosis. The scan should show interstitial fibrosis in the lower lung zones, with the involvement of the rest of the lungs to varying extents, and may also show pleural thickening.

Differential diagnosis of asbestosis

- Idiopathic pulmonary fibrosis (absence of the history of asbestos exposure)
- Hypersensitivity pneumonitis (generally acute exposure to inorganic material such as mold in silage, hay or bacteria)
- Silicosis (history of exposure to silica)
- Connective tissue disease (history of rheumatoid arthritis, scleroderma or SLE)
- [Sarcoidosis](#) (multi-organ involvement)

Therapy of asbestosis



Image: "This fibrous plaque on the diaphragmatic pleura has a verrucous appearance" by Yale Rosen.
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There is **no definitive treatment** for asbestosis. It is managed like other cases of diffuse interstitial lung fibrosis—with **supportive care**. If there is evidence of obstructive disease, **bronchodilator therapy** can be administered where appropriate. Lung infections, indicated by sputum changes, increased shortness of breath and fever, should be managed using antibiotics.

It is also important to **advise and assist patients to quit smoking**. Asbestos exposure further increases the risk of lung cancer in smokers.

Patients with asbestosis can be offered **pulmonary rehabilitation**, which has proven short-term benefits. This can include education, exercise training, nutrition advice, and psychosocial support. Rehab can help improve a patient's exercise tolerance, shortness of breath and quality of life. Also, when required, oxygen therapy can be given to help with exercise tolerance. Providing oxygen therapy can also help prevent pulmonary hypertension and cor pulmonale.

In very severe cases, with end-stage respiratory failure refractory to oxygen therapy, patients may become eligible for a **lung transplant**.

Progression and prognosis of asbestosis

The prognosis of asbestosis is **associated with the degree of exposure** to asbestos in the past and the severity of fibrosis that is found.

Silicosis

Definition of silicosis

Silicosis refers to pulmonary fibrosis caused by **inhalation of silica particles** following occupational exposure.

Epidemiology of silicosis

In the United States, there are about 160 deaths from silicosis each year. In most cases, silicosis is found in people who were **exposed to silica before the 1980s, and who are now over the age of 50.**

Etiology of silicosis

Silicosis is caused by occupational exposure to free silica or crystalline quartz. Silica is found as a key element in sand and rock. Occupations in which exposure can occur include **mining, sandblasting, stone-cutting, glass and cement manufacturing, silica flour production and quarrying of granite.** In Europe, the use of silica as an abrasive material is banned, however, it is not in the United States.

Pathology and pathophysiology of silicosis

Silica is fibrogenic, specifically crystalline silica (quartz). After exposure and inhalation of fine dust containing silica, **silica particles that are 5 microns or less enter into the alveoli.** Within the alveoli of the lung, **macrophages engulf the silica particles, resulting in their lysis.** At those sites in the lung where silica is deposited, an **inflammatory response** begins. Apart from the direct cytotoxic effect, the main effects of silica are to activate macrophages, leading to **a cascade of events that promote fibrosis.** Activated macrophages, in turn, activate fibrogenic proteins and growth factors that result in the stimulation of **fibroblasts** and production of **collagen.**

Most patients who develop silicosis are reported to have various immunologically related findings, such as rheumatoid factor, antinuclear antibodies, polyclonal hypergammaglobulinemia, and immune complexes. However, the role of these factors is not understood.

Similar to asbestosis, the development of silicosis occurs with a **dose-response pattern.**

Symptoms of silicosis

General symptoms

Silica exposure can result in **three types of disease manifestations: acute, subacute and chronic silicosis.** In most cases, the symptoms of silicosis appear after a period of at least 10 years post exposure. However, there are acute cases where silicosis develops within a short time frame of at least 10 months.

The different types of silicosis vary in their rate of development and severity but cause similar respiratory symptoms:

- Progressive shortness of breath
- Cough
- Wheezing

Acute symptoms

Acute silicosis is a more aggressive and rare form of the disease. It can develop after intense exposure to fine silica particles over a period of several months. This type of exposure occurs when there is inadequate respiratory protection. These are the same symptoms that develop in the more common chronic silicosis but at a quicker rate. In

most cases, acute silicosis results in death.

Subacute symptoms

Subacute silicosis is also an accelerated form of the disease, which develops as a result of heavy exposure to silica. In these cases, the complication of progressive massive fibrosis is more common.

Chronic symptoms

Most symptoms of silicosis occur chronically, generally after at least 10 years since exposure to silica. Simple chronic silicosis has few symptoms and signs. In complicated chronic silicosis, however, the patient may develop progressive shortness of breath. **Progressive massive fibrosis is a final complication of complicated chronic silicosis.**

Pulmonary tuberculosis and other lung infections can occur as a complication of silica exposure, called silico-tuberculosis. This is because **silica is cytotoxic to alveolar macrophages, the main defense against these lung infections.** Another complication that can possibly occur is the development of an autoimmune connective tissue disorder such as scleroderma and [rheumatoid arthritis](#). Silica is also listed as a **possible lung carcinogen** by the International Agency for Research on Cancer.



Image: "Miner's lung with silicosis and tuberculosis" by Museomed. License: [CC BY-SA 3.0](#)

Diagnosis of silicosis

Radiology

A chest x-ray is an initial test performed to screen for silicosis. In chronic silicosis, a chest x-ray may show **tiny round opacities (nodules)**, generally in the upper zones of both

lungs. Such radiographic findings are found generally 15 to 20 years following exposure. There also may be the appearance of “eggshell-like” calcification of the hilar lymph nodes. In rare cases of acute silicosis, significant miliary infiltration may be observed.

A high-resolution CT chest scan is more sensitive than a chest x-ray to detect and diagnose interstitial fibrosis due to silica exposure. It will also show the extent of fibrosis, from simple silicosis to massive fibrosis. In acute silicosis, the characteristic picture is called “**crazy paving**”.



Image: “Complicated silicosis” by Gumersindorego. License: [CC BY-SA 3.0](https://creativecommons.org/licenses/by-sa/3.0/)

Pulmonary function testing

Pulmonary function tests are usually normal in simple silicosis. In some cases, it may reveal a **restrictive pattern** with reduced forced vital capacity, a normal FEV1 to FVC ratio, reduced vital capacity and reduced diffusing capacity. Usually, patients with silicosis also have **obstructive changes due to the effects of exposure to non-specific dusts or smoking**.

Differential diagnosis of silicosis

- Asbestosis (history of asbestos exposure)
- Idiopathic pulmonary fibrosis (absence of a history of exposure to metals or mineral dusts)
- Sarcoidosis (multisystem disease of unknown etiology, possibly autoimmune)
- COPD
- Drug or radiation-induced fibrosis

Therapy of silicosis

Management of silicosis is largely supportive and focuses on the prevention of further progression and complications. Evidently, preventing the patient from further exposure is essential. As individuals with silicosis are at risk of developing tuberculosis, constant surveillance is necessary. If patients are found to be tuberculin positive, then they have a 30-fold increased risk of developing tuberculosis, and, therefore, treatment of latent TB should be instituted.

In cases of acute silicosis, **whole-lung lavage** may be used, which is intended to physically remove silica particles from the alveoli of the lung.

Progression and prognosis of silicosis

The **prognosis of chronic silicosis is generally good**. However, if silicosis leads to **progressive massive fibrosis, mortality is high**, and this complication is more common in sub-acute disease patterns. Acute silicosis is the rarest and aggressive type, commonly resulting in death.

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