CME

Silicosis: No longer exclusively a chronic disease

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ABSTRACT

Silicosis typically has been classified as a chronic disease that develops after at least 10 years of exposure to silica dust, and often is associated with miners and stone workers. As industries have changed over time, other types of workers (including those in artificial stonework, jewelry polishing, and denim production) have become exposed to high levels of silica, leading to the development of acute and accelerated silicosis. Acute silicosis can develop in as little as a few months, and accelerated silicosis can develop in as little as 2 years. No cure exists for any form of silicosis, and lung transplantation is the only lifesaving treatment. Primary care clinicians must understand when patients are at risk for developing silicosis and not assume that a short time of exposure precludes the development of silicosis.

Keywords: silica, exposure, silicosis, worker, health, stone

Learning objectives

- Identify all forms of silicosis and in what industries patients are at higher risk for developing silicosis.
- Describe why clinicians must elicit a complete and accurate occupational history to assess patient risks and avoid misdiagnosis.
- Explain why patient education is crucial to help patients avoid developing acute and accelerated silicosis.

Silicon dioxide (SiO_2), also known as silica, is a chemical compound found in the Earth's crust; its crystalline form is the principal component in more than 95% of rocks.[1] When mortar, concrete, sand, and stone are ground, cut, sawed, drilled, polished, or crushed, crystalline silica particles are reduced to sizes less than 10 micrometers in diameter, becoming what is defined as respirable.[2] Because of their small size, respirable particles can bypass the body's natural defenses such as cilia and mucus in the nasal cavity and upper airways, travel deep inside the respiratory system, and deposit directly into the lungs.[1] Inhaling respirable crystalline silica particles can lead to silicosis, a progressive fibrotic lung disease caused by the prolonged or massive inhalation of crystalline silica. Alveolar macrophages are thought to interact with and ingest inhaled silica particles, beginning an inflammatory response, and the inflammation subsequently leads to fibrosis and formation of collagen-containing silicotic nodular lesions in upper pulmonary lobes. People who work in stone cutting, mining, construction, sandblasting, and surface drilling are at highest risk for developing silicosis.[2] According to the federal Occupational Safety and Health Administration (OSHA), about 2.3 million patients in the United States are exposed to silica at work.[2]

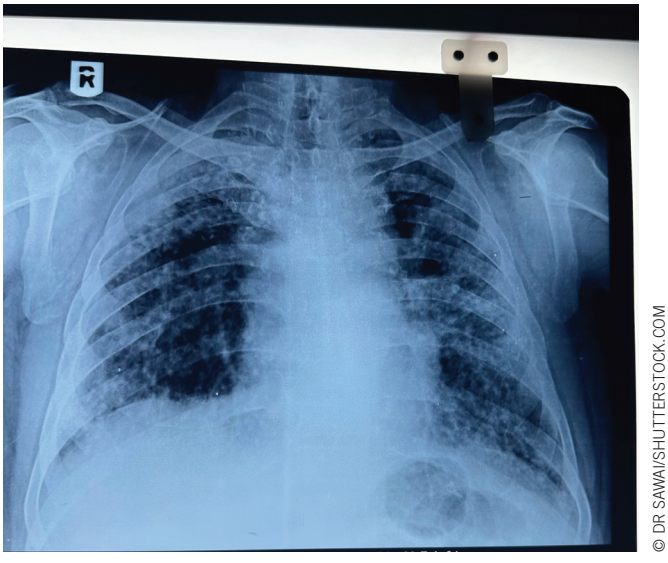

FIGURE 1. Chest radiograph of a patient with silicosis

HISTORY

Silica dust exposure is one of the earliest recognized causes of lung disease. Hippocrates first recognized silica's effect on respiratory function in 430 B.C.[1] In the 1550s, Georgius Agricola wrote about men dying of pulmonary disease after working in the mining industry in Central Europe. In 1770, Italian physician Bernardino Ramazzini identified silicotic nodules in the lungs of stone cutters. In the mid to late 1800s, mechanization in the mining industry resulted in rapidly increasing levels of silica exposure and cases of silicosis.[1]

Silicosis was first recognized as a chronic disease that occurred primarily in miners and stone workers after at least 10 years of exposure to respirable crystalline silica

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Key points

- Manipulation of artificial stone materials exposes workers to extremely high amounts of silica that can cause accelerated and acute forms of silicosis.
- PAs might be the first clinicians to assess patients with severe forms of silicosis that are becoming prevalent.
- Complete occupational histories and knowledge of which industries present increased risk for developing silicosis can aid in early diagnosis of the deadly disease.
- To reduce patient risk for developing silicosis, educate patients about risks and ways to reduce exposure to crystalline silica.

dust. The patterns of exposures related to onset of the condition have changed over time as the related industries have changed. The dust created in modern industries such as fracking, jewelry polishing, benchtop fabrication, and denim production can contain high levels of respirable silica. These exposure levels, often at orders of magnitude higher than those found in the mining industry, put workers in danger of developing silicosis in shorter time frames. Various types of silicosis have been described, based on time of exposure, including chronic with more than 10 years of exposure (further described as simple, complicated, or interstitial pulmonary fibrosis), accelerated with 5 to 10 years, and acute with fewer than 5 years.

The acute and accelerated forms of silicosis can occur in as little as a few months because of the high percentage of silica. Clinicians, including those in primary care settings, need to understand when patients are at risk for developing silicosis and that a short exposure time does not preclude development of silicosis. This also underscores the importance of adequate occupational histories to identify risk in the context of exposure type and time. Clinicians also need to be able to properly educate patients on the risks in certain industries and how to avoid developing silicosis, because the only long-term treatment option is lung transplantation.

CLASSIFICATION

The three classifications of silicosis are based on the concentration of respirable particles inhaled and their effects on the lungs.[3,4] Chronic silicosis and accelerated silicosis can be similar in presentation but differ in length of exposure and speed of progression; accelerated silicosis has overlapping characteristics of both chronic and acute silicosis (**Table 1**).

Chronic silicosis This form is the classic clinical case of silicosis that is typical in miners and quarry workers, presenting many years after first exposure. Chronic silicosis develops from low levels of silica dust exposure over a long period of time, typically 20 years or more. Symptoms can include cough, dyspnea, and fatigue, with the most common symptom being chronic productive cough.[5]

Chronic silicosis can be simple or complicated (**Table 1**). Simple chronic silicosis classically presents with silicotic nodules in the upper lobes of the lung. Complicated chronic silicosis occurs when the silicotic nodules enlarge and coalesce, forming large areas of fibrosis, a condition called progressive massive fibrosis (PMF). Patients with chronic silicosis have variable lung function; some patients have

TABLE 1. Forms of silicosis

Form	Duration of exposure (years)	Radiologic findings	Clinical findings
Simple chronic	>10	• Round or irregular nodules <1 cm ± calcification of nodules • Upper lobe predominant	• Asymptomatic • Chronic cough • Exertional dyspnea • Obstructive, restrictive, or mixed defects
Complicated chronic/ conglomerate/PMF	>10	• Conglomerate masses >1 cm migrating to the hilum ± calcification of nodules: uniform (53%) or speckled (26%) pattern; rarely with an eggshell pattern (5%) ± central cavitation • Diffuse reticulonodular fibrosis • Surrounding emphysema • Pleural thickening	• Asymptomatic • Chronic cough • Exertional dyspnea • Weight loss • Respiratory failure
Acute (silicoproteinosis)	<5	• Bilateral perihilar consolidation • Centrilobular nodules • Ground-glass changes • Crazy paving	• Dyspnea • Cough • Weight loss • Respiratory failure
Accelerated	2 to 10	• Rapidly progressive nodules and masses • Features of PMF	• Dyspnea • Cough • Respiratory failure

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normal lung capacity and others have mixed restrictive and obstructive abnormalities depending on the disease stage. Patients with advanced disease, especially those with PMF, typically exhibit restrictive defects.[6]

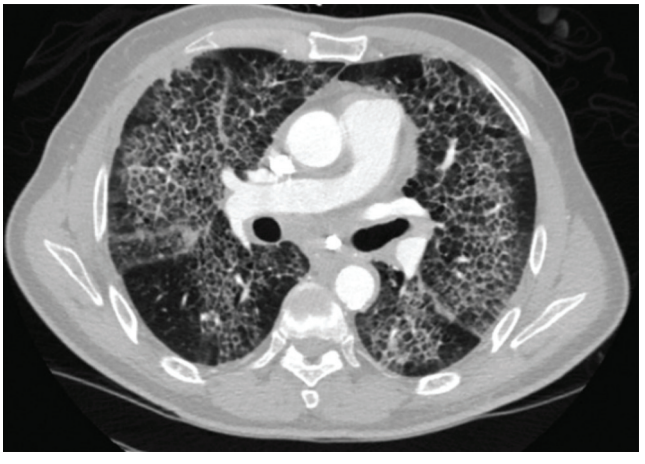
Acute and accelerated silicosis Current evidence on acute and accelerated silicosis is limited to case reports and small series.[1,7] Both types of silicosis are associated with higher concentrations of inhaled respirable silica and shorter exposure periods compared with chronic silicosis. Acute and accelerated silicosis can cause respiratory and systemic symptoms (**Table 1**). Clinicians should be aware of these forms of silicosis because they can develop and progress much more quickly than chronic silicosis.

Acute silicosis, also known as silicoproteinosis, can cause symptoms in a few weeks to 5 years.[6] The reason for the broad range in symptom development is not well defined in the literature. Acute silicosis can cause fever, headache, unintended weight loss, shortness of breath, productive or nonproductive cough, chest tightness, and pleuritic chest pain. Because the symptoms are nonspecific, acute silicosis can be misdiagnosed as a lung infection, especially in young patients, if clinicians do not recognize or understand the exposure risks of respirable crystalline silica.[8,9] Presenting dyspnea can progress rapidly to respiratory failure and death, hence the need for prompt diagnosis.[1,6]

Accelerated silicosis, also known as subacute silicosis, typically develops 2 to 10 years after the first exposure to high concentrations of respirable crystalline silica.[10] A patient with accelerated silicosis, like one with chronic silicosis, presents with silicotic nodules and interstitial fibrosis, but the radiographic and clinical progression of accelerated silicosis is much more rapid. Accelerated silicosis also shares features with acute silicosis, specifically the protein buildup in the alveoli caused by silica (silicoproteinosis). Patients with accelerated or acute silicosis also may have extrapulmonary involvement, affecting the liver and kidneys, because of systemic particle migration from the lungs.[1,6,11,12] The mortality of chronic silicosis associated with older industries such as the mining industry is 6 per 1,000 workers, or less than 1%. A literature review of published case reports and longitudinal cohort studies performed by Barnes and colleagues suggests that the mortality of modern at-risk occupations (including stone crushing, sandblasting, jewelry polishing, and artificial stone benchtop manufacturing) is 10% to 100%.[1]

FIGURE 2. CT scan showing crazy paving—polygonal honeycomb-like interlobular septal thickening

Reproduced with permission from Verma R, Abdoh M. Crazy paving pattern. *Clin Case Rep.* 2017;5(4):533-534.



TESTING

A chest radiograph is the primary imaging modality for detecting pulmonary abnormalities and is used in screening and diagnosis of silicosis. However, because acute and accelerated silicosis can present with more diffuse and irregular fibrosis or none at all, a plain chest radiograph is not reliable for screening in these cases.[1] High-resolution CT is superior to chest radiograph in detecting the initial phase of silicosis but has limited utility in screening for silicosis because of its cost, lack of clear standards, and risk of false positives. Pulmonary function tests may be useful to evaluate severity of lung dysfunction at diagnosis and during follow-up. Pulse oximetry and arterial blood gas analysis might be useful for detecting hypoxemia (at rest and with exercise in patients with silicosis and in patients with respiratory failure). Lung biopsy generally is not needed for diagnosis but may be useful to exclude other potentially treatable diseases and to assess disease severity for consideration of lung transplant. Clinicians also should consider assessing for latent or active tuberculosis (TB), which often occurs concurrently with silicosis.

RADIOLOGY FINDINGS

Radiographically, a patient with simple chronic silicosis has bilateral reticulonodular opacities, typically in the upper lung fields, as well as pleural thickening (**Figure 1**). Pleural effusion may be present, although it is less common. Patients with complicated chronic silicosis display conglomerate opacities, usually with calcification and thickening, on imaging.[13]

In patients with acute silicosis, radiologic findings can include ground-glass opacities, bilateral patchy centrilobular nodularity, interlobular septal thickening, bilateral perihilar consolidation, and crazy paving (ground-glass opacities with superimposed interlobular septal thickening, **Figure 2**).

Imaging in patients with accelerated silicosis shows similar findings as in those with chronic silicosis, but fibrosis may be more irregular and/or diffuse (**Figure 3**), or it also might not be apparent at all.[1,6,13]

AT-RISK OCCUPATIONS

Artificial stone kitchen and bathroom countertops, also referred to as agglomerated quartz and manufactured, engineered, or reconstituted stone, are made from finely

crushed rocks and a polymer resin that binds the particles. The rock particles and resin are molded and heat-cured to form the finished product, which contains 85% to 93% crystalline silica, compared with less than 30% in natural stone.[1,14] Cutting, grinding, and polishing these countertops creates respirable particles with extremely high crystalline silica content.

In the United States, the exposure limit for respirable crystalline silica is 0.05 mg/m^3 as an 8-hour time-weighted average (TWA); employers are required to assess workers' exposure only if the action level of 0.025 mg/m^3 TWA is suspected to be exceeded.[2] These exposure limits, however, often are based on the lowest limit of detection of current sampling analytical methods and not on health effects. Although there are no limits for short-term (typically 15-minute) exposures, the recommendation is that workers should not be exposed to triple the 8-hour limit in a 30-minute period and should never be exposed to five times the 8-hour limit.[15] Dry-cutting manufactured stone creates almost 300 times the recommended limit; manufactured stone cut with water dust suppression still creates 30 times the recommended limit.[1] Because of their high exposure levels, workers involved with fabrication, polishing, installation, and assembly of artificial stone kitchen and bathroom countertops are at risk for developing acute and accelerated forms of silicosis.

In the denim industry, sandblasting uses silica-containing sand to give jeans a worn-out look; this exposes workers to extremely high levels of respirable silica that can cause accelerated, acute, and chronic silicosis. Akgun and colleagues performed a 4-year follow-up study of former denim sandblasters and found a silicosis prevalence rate of 96.4% after only a mean occupational exposure of 3.5 years or less.[16] Jewelry polishing is another profession with high silica dust exposures. Panchadhyayee and colleagues found that jewelry polishing workers developed acute silicosis after a mean exposure period of 3.4 years.[17]

Cases of acute silicosis also have been reported in quartz millers, brick masons, tombstone sandblasters, and workers doing both wet and dry grinding of cement containers.[18] Recent outbreaks of accelerated silicosis have occurred among workers in the artificial stone industry and those who sandblast denim.[6,18]

DIAGNOSIS

Clinicians should suspect silicosis in patients with a history of crystalline silica exposure and symptoms such as chronic dyspnea and/or cough. A diagnosis of silicosis is confirmed by presence of characteristic radiologic findings and the exclusion of other conditions. Chest radiographs, high-resolution CT, pulmonary function tests, and health and exposure questionnaires are the main methods used in surveilling workers exposed to respirable crystalline silica. These methods, however, cannot detect disease until it has significantly progressed. Some studies have reported the limitation of chest radiography in diagnosing occupational lung disease, but the International Labour Organization International Classification of Radiographs of Pneumoconiosis still recommends it.[19] High-resolution CT is more sensitive and specific than radiography but is not recommended because of its high cost, low accessibility, and radiation exposure.[19-22]

FIGURE 3. Chest radiographs showing transition in a patient with accelerated silicosis

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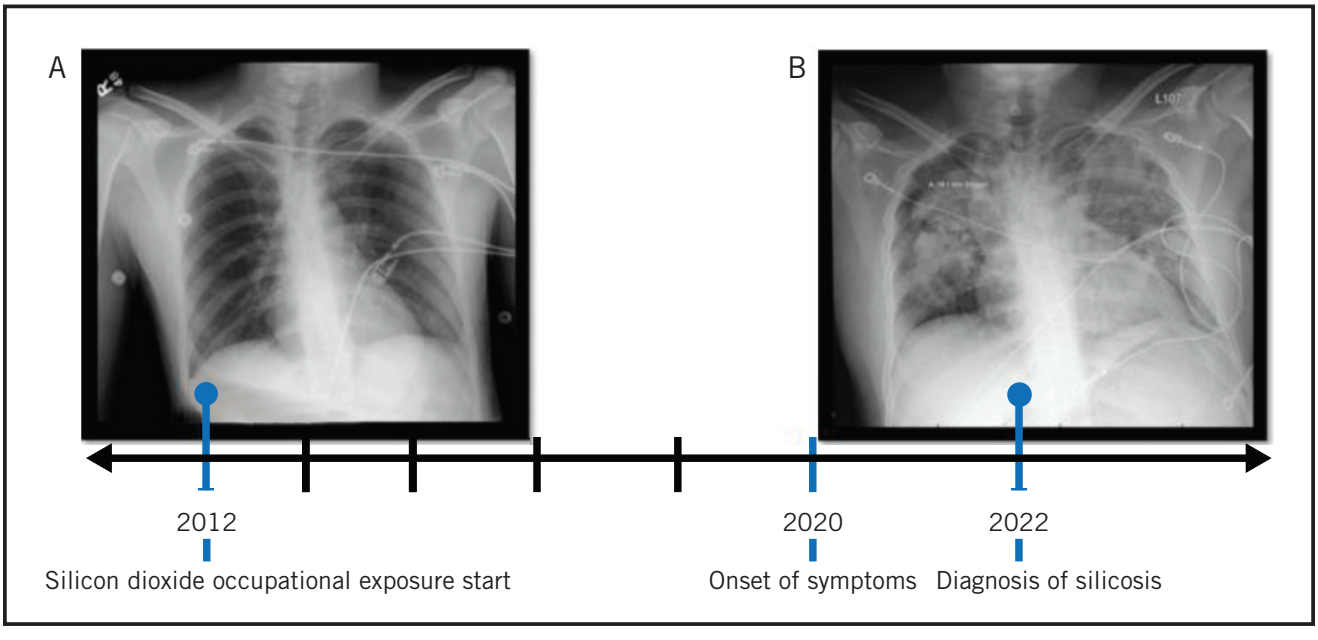

Lung biopsy is the only way to definitively diagnose silicosis in patients who do not have a history of occupational exposure (or if the exposure history is not understood by the clinician), when discrepancy exists between chest radiography and CT results, and when atypical presentations cause clinicians to consider other diagnoses.[23] However, the safety of lung biopsy is questionable—Akgun and colleagues reported suspicion of an association between invasive procedures and mortality following case studies of patients with acute or accelerated silicosis who died after surgical biopsy.[24] History of exposure to respirable crystalline silica is key in diagnosis. Additionally, smoking or preexisting respiratory disease, such as chronic obstructive pulmonary disease (COPD), affects the defense and clearance systems and may predispose patients to silicosis.

The novelty of acute and accelerated silicosis and lack of clinical understanding have caused misdiagnoses of some patients. Guarnieri and colleagues described two cases of accelerated silicosis in artificial-stone workers who were misdiagnosed as having sarcoidosis.[12] The misdiagnosis reportedly was the result of overlooking the occupational exposure of both patients to silica dust and not recognizing the atypical presentation of silicosis: occurrence in young workers, localization to the lung lymph nodes, and extrapulmonary involvement.[12] According to Shukla and colleagues, acute silicosis in particular often is misdiagnosed as pneumonia, pulmonary edema, or TB.[25]

Several diseases are associated with silicosis, which also can complicate the diagnosis. Studies have shown that silica dust exposure is associated with emphysema in smokers and nonsmokers alike.[26-29] Thus, silicosis should not be ruled out in patients with obstructive pulmonary symptoms.

Silica exposure also is associated with an increased risk for TB.[30] Silica and silicosis are risk factors for developing mycobacterial lung infections. According to Shukla and colleagues, patients with silicosis have a 10- to 30-fold increased incidence of TB.[25] Fundamental research suggests that pulmonary macrophages damaged by silica may be unable to fight off the mycobacteria.[25,31-34] Clinicians should closely investigate mycobacterial lung infections in workers with high silica exposures, to not miss the possible diagnosis of silicosis.

Silicosis is associated with an increased risk of developing autoimmune diseases and lung cancer.[35] Specific autoantibodies have not been identified, but studies have demonstrated an association between silica exposure and systemic sclerosis, rheumatoid arthritis, antineutrophil cytoplasmic antibody-related vasculitis, and systemic lupus erythematosus.[36] Silica dust is believed to promote or accelerate disease development, break immune tolerance, initiate autoimmunity, or magnify autoimmune vulnerability.[35]

Respirable crystalline silica was recognized as early as 1997 by the International Agency for Research on Cancer as a human carcinogen, and its carcinogenicity was reconfirmed in a 2009 report.[37]

THE IMPORTANCE OF HISTORY-TAKING

As in any diagnosis, the first step is gathering the pertinent history from the patient. A diagnosis of silicosis requires a history of exposure to silica dust (even if minimal) along with positive radiographic and histopathologic findings.[38] A complete occupational history, including specific exposure details of materials and length of time, is essential to be able to rule silicosis in or out. Primary care clinicians need to be aware of the acute and accelerated forms of silicosis so as not to exclude silicosis as part of the differential when confronted with a young patient with cough, dyspnea, and fatigue.

TREATMENT

No cure exists for silicosis. The primary approach to management involves prevention of further crystalline silica inhalation; lung transplant may be considered in select patients. Sidney-Filho and colleagues studied the effects of lung transplantation on pulmonary function and life expectancy in patients with end-stage silicosis and found that lung transplantation significantly improved pulmonary function and survival.[39] Without transplantation, treatment is supportive, focused on alleviating symptoms and preventing complications. Therapeutic measures include bronchodilators for symptoms of airflow obstruction, smoking cessation, immunization (for example, influenza and pneumococcal vaccines), cough suppressants, mucolytics, antimicrobial therapy for lung infections, long-term oxygen therapy for patients with severe hypoxemia caused by COPD, corticosteroids, and pulmonary rehabilitation.[6,40,41] Other supportive treatments that may be used in practice include chest physiotherapy, whole-lung lavage, and medications to prevent TB (such as isoniazid, rifampin, or pyrazinamide).[25]

PREVENTION AND EDUCATION

Because the only long-term treatment for silicosis is lung transplantation, prevention is key. In addition to avoiding at-risk occupations, the only way to prevent silicosis is to remove exposure by engineering and administrative controls such as local exhaust ventilation systems, air filtering, wet cutting methods, and housekeeping techniques such as removal of dust from surfaces.

Use of adequate respiratory personal protective equipment (PPE) is another important way to control exposure. Field studies have shown that workers tend to remove PPE when they are not actively performing dust-generating tasks, but exposure still occurs from particles in the air and particles settling on surfaces.[42] Additionally, PPE alone is not a sufficient form of exposure prevention because it is only effective if workers are fit-tested, are wearing it correctly and continuously for the duration of the work shift, have appropriate filters, and if the equipment is regularly maintained.

Although exposure control experts focus on eliminating exposure, clinicians can focus on education. Patients should

be educated on the severity of silicosis and lack of treatment options so they understand the importance of continuously wearing appropriate and adequate PPE such as respirators. Carrieri and colleagues state that silica dust created from grinding and cutting artificial stone can be resuspended when people and materials are moved, so removing masks while not actively cutting is dangerous.[14] Patients also need to be educated on the importance of using the engineering controls put in place by their employer, which might include dust suppression, water jet sprays, dust hoods on grinders, down-draft tables, and floor drainage systems.[42]

Another important piece of patient education is secondary prevention to stop continued silica exposure in patients with silicosis. The CDC and OSHA provide Safe Work Practices for exposure prevention.[2,43] Nevertheless, workers need to understand that even when exposure stops, silicosis continues to progress.[23] At-risk patients should be encouraged to stop smoking because smoking has been shown to increase the risk for death in silica-exposed patients.[44]

CONCLUSION

Clinicians need to understand that silicosis can be more than a chronic disease that presents after many years of exposure to silica. Cases of acute and accelerated silicosis are increasing and need to be recognized as potential diagnoses in young workers with pulmonary complaints.

In primary care settings, physician associates/assistants (PAs) might be the first clinicians to assess patients with the severe forms of silicosis that are becoming more prevalent, especially in workers involved in denim sandblasting, artificial stonework, and jewelry polishing. When clinicians focus on gathering complete occupational histories and understand which industries present increased risk for workers to develop silicosis, they can help in early diagnosis of a deadly disease. Better engineering controls must be developed in industries where employees are exposed to high levels of respirable crystalline silica. By educating patients about risks and ways to reduce or eliminate exposure to crystalline silica, clinicians can help their patients avoid developing silicosis. **JAAPA**

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