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Review

Artificial Stone Silicosis: New Face of an Old Disease

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ABSTRACT

Artificial stone (AS) silicosis has emerged as a major global occupational health concern, challenging the perception of silicosis as a disease of the past. Driven by the fabrication of AS products containing high concentrations of crystalline silica (>90%), this rapidly progressive form disproportionately affects younger workers and develops after a much shorter exposure duration (median 7–19 years) than in classic silicosis. As cases continue to emerge worldwide, clinicians and occupational health practitioners are increasingly likely to encounter affected workers in clinical practice. This review provides a clinically focused synthesis of the current evidence on the epidemiology, pathophysiology, clinical manifestations, diagnosis, and management of AS silicosis. Compared with traditional silicosis, the unique physicochemical properties of AS dust may amplify lung toxicity and promote a self-perpetuating inflammatory response. This frequently results in rapid development of progressive massive fibrosis, with disease progression often continuing after exposure cessation. Diagnosis is often delayed because of overlapping clinical features and the limited sensitivity of conventional screening tools, highlighting the importance of early clinical recognition, detailed occupational history-taking, and the use of high-resolution computed tomography where appropriate. Lung transplantation remains the only definitive treatment for advanced disease, underscoring the importance of prevention. Effective prevention requires strong workplace controls, systematic medical surveillance, and appropriate regulatory measures. Clinicians play a key role in early detection, patient education, and mandatory disease reporting. Coordinated action across clinical, occupational, and policy domains is essential to improve recognition, strengthen preventive measures, and avert further cases of this highly preventable occupational disease.

KEYWORDS: Silicosis, dust, occupational diseases, silicon dioxide

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INTRODUCTION

Silicosis is caused by the inhalation of respirable crystalline silica (RCS), which refers to the fraction of silica dust small enough to penetrate beyond the upper airways and deposit in the lower respiratory tract. It is one of the oldest recognised occupational fibrotic lung diseases.[1]

Historically, silicosis was closely associated with industries that dominated early industrial economies such as mining, quarrying, tunnelling, and sandblasting.[2] Occupational silicosis became a major concern globally in the 1900s, exemplified by the Hawk's Nest Tunnel disaster in the United States (US) during the 1930s, in which over 700 tunnel workers died from exposure to large amounts of silica dust.[2] However, improved industrial hygiene practices, medical surveillance, and regulatory controls in many high-income countries over the past decades have led to a substantial reduction in the incidence of silicosis.[1] This led to a widespread perception of silicosis as a "disease of the past" in modern industrialised societies.

Three main clinical forms of silicosis have been described, largely corresponding to the intensity and duration of RCS exposure. Chronic (or classic) silicosis is the most common form and arises after long-term exposure to relatively low concentrations, whereas accelerated silicosis develops over a shorter latency following higher-intensity exposure.[1] Acute

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silicosis (also known as silicoproteinosis), the least common but most severe form, results from intense exposure to RCS during a brief period and may manifest within weeks to years.[1,2]

Emergence of Artificial Stone in the Built Environment

Unlike naturally occurring silica-containing materials, historically encountered in the industries described earlier, artificial stone (AS) is a relatively new, manufactured composite introduced commercially in the 1980s.[2,3] AS, also known as engineered stone, reconstituted stone, quartz conglomerate or agglomerated stone, is manufactured by combining finely crushed stone with polymer resins, pigments, and other additives, which are then moulded and cured to produce a dense, non-porous surface.[2,4] A defining feature of AS is its exceptionally high crystalline silica content, often exceeding 90% by weight, which is markedly higher than the silica content of natural stones such as marble (which contains minimal silica) or granite.[4,5] This distinctive composition differentiates AS from traditional silica-containing materials and has important implications for RCS generation during fabrication and finishing.

A New Face of a Preventable Disease

AS has become increasingly used for kitchen and bathroom countertop fabrication because of its durability, appearance, and cost-effectiveness.[4,6] Correspondingly, there has been a re-emergence of silicosis in a more aggressive and rapidly progressive form.[2,7] Since the 2010s, silicosis linked to AS fabrication has been reported in multiple regions, including Israel, Europe, the United Kingdom, the US, China, Australia, and Türkiye.[3,8-11] These cases have highlighted a paradox in which a well-characterised, preventable historical occupational disease has resurfaced in modern economies because of contemporary materials and work practices. This marks a shift in the epidemiology and clinical presentation of silicosis, and has prompted renewed scrutiny of existing exposure controls, surveillance systems, and regulatory frameworks. The extent of recognition of AS silicosis as an emerging disease of concern is also reflected in the rapidly growing body of clinical research and literature, with publications increasing substantially since 2014 and accelerating markedly over the past decade (Figure 1).

This review aims to synthesise the current evidence on AS silicosis, with a particular focus on lessons for clinicians, occupational health practitioners, and policymakers.

Main Points

- Artificial stone (AS) silicosis is a rapidly progressive form of silicosis, characterised by a shorter latency period and more aggressive clinical course than traditional silicosis.
- The high crystalline silica content of AS, together with the generation of fine respirable dust during fabrication, are likely the principal drivers of disease development and severity. Disease progression may continue despite cessation of AS dust exposure.
- Clinicians, particularly those in occupational health, play a critical role in the early detection of AS silicosis through comprehensive occupational history-taking, timely diagnosis, and targeted health surveillance of exposed workers.
- Prevention relies on eliminating or minimising respirable crystalline silica exposure through effective controls such as avoidance of dry processing, regular health surveillance, and appropriate respiratory protection.

Occupational Context of AS Silicosis

Manufacturing and Fabrication Processes of AS

The currently widely used AS manufacturing system was first developed in the 1970s,[2] and involves vibro-compression under vacuum, whereby finely crushed stone, predominantly crystalline silica, is combined with polymeric resin binders, pigments, and additives to form dense, uniform slabs.[2,12]

High-risk exposures occur during fabrication and installation. Fabrication processes include cutting, grinding, drilling, polishing, and edge finishing to customise slabs for kitchen and bathroom benchtops.[2,7] Compared with natural stone fabrication, AS processing generates substantially higher RCS exposures because AS has a higher silica content. Dust generated during dry-cutting of AS typically contains more than 80% crystalline silica, whereas dust from natural stone contains substantially lower proportions of crystalline silica, averaging 1–4% in marble and 30–50% in granite.[8,13,14]

Exposure assessments have shown that dry cutting can generate RCS concentrations exceeding 40 mg/m^3, exceeding various legislative occupational limits of 0.05 mg/m^3.[2,4] Wet cutting substantially reduces exposure, but does not eliminate the risk. Furthermore, brief dry tasks frequently occur during finishing and on-site installation, where water suppression may be unavailable.[4]

Occupational Context and High-risk Work Settings

AS fabrication is commonly undertaken in smaller enterprises with fewer than 10 workers.[10,15] AS workers tend to use small, handheld power tools without integrated water-delivery systems, such as angle grinders, because of their lower cost and greater flexibility.[2,4] Additional workplace risk factors that exacerbate exposure to RCS for both AS workers and bystanders include inadequate local exhaust ventilation, poor general ventilation and limited segregation of dusty tasks,[4,7,15,16] and non-compliance or incorrect use of respiratory protective equipment (RPE). This, coupled with productivity pressures, a lack of comprehensive occupational safety programmes and weak regulatory oversight,[15,17,18] substantially increases RCS exposure risk.

Processing of AS could also release volatile organic compounds (VOCs), such as styrene and other aromatic hydrocarbons, which may contribute to inflammatory and obstructive airway disease observed in exposed workers.[2,4]

Epidemiology

Global Burden of AS Silicosis

Estimates from the Global Burden of Disease Study 2021 indicated that the age-standardised incidence, mortality,

and disability-adjusted life years attributable to silicosis have declined since 1990,[19] with a greater burden observed in medium and medium-high sociodemographic indices regions.[19] However, these estimates do not distinguish between occupational exposure sources and cases attributable to AS. The American Thoracic Society Workshop Report, released in 2026, estimated that at least 6,351 cases of AS silicosis had been identified worldwide as of December 2025, based on available data from 12 countries across the Americas, Europe, the Middle East, Asia, and the Pacific region.[20] However, as acknowledged in the report, this figure was likely underestimated because many countries lack systematic medical screening, disease surveillance, and universal access to healthcare, leading to undetected or unreported cases.[20] As a result, the true global burden of AS silicosis remains unknown.

Clinical Epidemiology of AS Silicosis

AS silicosis represents a distinct epidemiological pattern compared with classic silicosis. Affected AS workers are typically relatively young, commonly diagnosed in the mid-40s, and often present after a markedly shorter exposure duration (median 7–19 years), with rapid disease progression.[2,10,21] These features contrast sharply with classic silicosis, which typically manifests after decades of low-level exposure and in older individuals.[1,7,16,21] Progression to progressive massive fibrosis (PMF) may continue even after cessation of exposure.[10]

Pathophysiology of AS Silicosis

Mechanism of Lung Injury

The pathogenesis of AS silicosis shares core pathways with traditional silicosis (Figure 2), but appears to be amplified by the distinctive physico-chemical properties of AS dust that may contribute to AS's toxicity and accelerate disease progression.

In addition to its high crystalline silica content, AS typically contains 8–20% polymeric resin used to bind crushed stones.[13] This resin coating on silica particles may stabilise surface radicals produced during cutting, influence their reactive pathways, and thereby amplify oxidative stress and injury.[13,23]

AS dust also contains transition metals (e.g., cobalt, copper, aluminium, titanium) derived from pigments and resins[13,24] which have been associated with increased macrophage cytotoxicity and inflammatory responses independent of silica concentration.[25] This further promotes reactive oxygen species generation and activation of pro-fibrotic inflammatory pathways, thereby acting as an adjuvant that intensifies the fibrogenic response to silica exposure.[13,25] The cumulative oxidative stress and cytokine release lead to macrophage death, releasing the indigestible RCS particles back into the alveolar space. They are again re-phagocytosed by newly recruited macrophages, establishing a self-perpetuating "vicious cycle" of inflammation that continues even after workplace exposure has ceased.[1,26] This persistent inflammatory cycle drives fibrogenesis through growth factors from alveolar macrophages and epithelial cells [e.g., transforming growth factor-β (TGF-β) and platelet-derived growth factor], resulting in progressive parenchymal fibrosis.[11,26]

Individual genetic susceptibility appears to affect disease risk and severity. Polymorphisms in cytokines and inflammasome genes such as TNF-α, interleukin-1 (IL-1), TGF-β-1, and FASLG have been associated with susceptibility to silicosis and disease severity, albeit inconsistently across different populations.[4]

Histopathological Patterns and Unique Features of AS Silicosis

AS silicosis frequently presents as a complex "mixed" phenotype, instead of neatly fitting into the distinct classical forms.

In early disease, lung biopsies demonstrate inflammatory nodules composed predominantly of histiocytic cells and fibroblasts, without central hyalinisation.[5] Birefringent dust particles consistent with silica may be identified by polarised

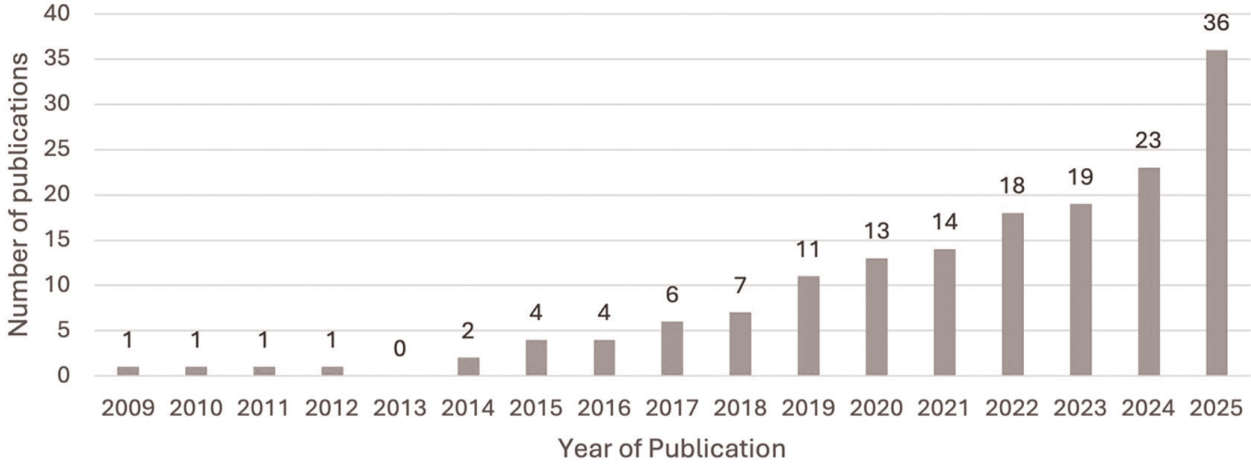


Figure 1. Trend of the publication on artificial stone (AS) silicosis found in the PubMed Database from 2009 to 2025. A simple search strategy was used on the PubMed database to identify English-language publications on AS silicosis; the search employed the keywords (silicosis[All Fields] OR "silicosis" [MeSH Terms] OR "silica dust" [All Fields]) AND ("artificial stone" OR "engineered stone" OR "quartz conglomerate*" OR "agglomerated stone" OR "reconstituted stone" OR "artificial silica"), covered the period 1 Jan 2009 to 31 Dec 2025, and its results were retrieved on 25 June 2026. A detailed list of the retrieved publications can be found in Supplementary Material 1

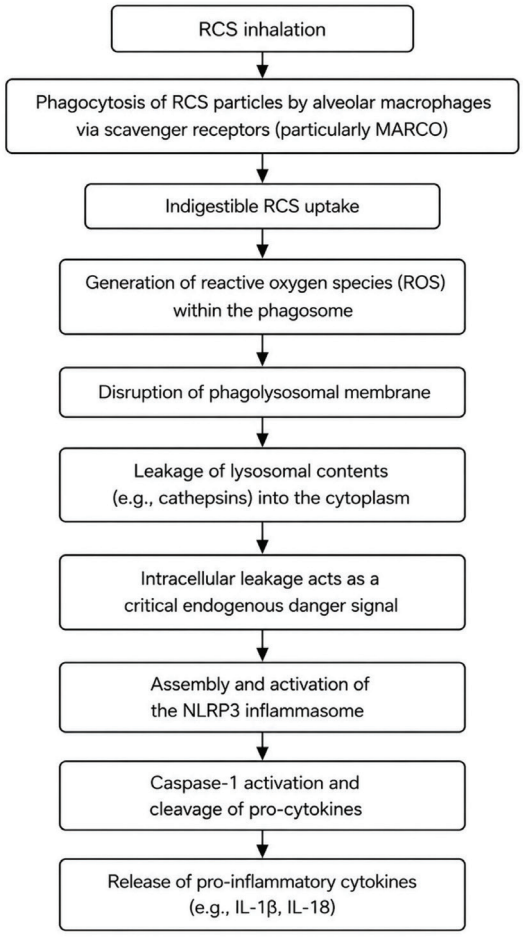


Figure 2. Simplified pathogenesis of silicosis.[5,11,22] Inhaled respirable crystalline silica (RCS) particles are phagocytosed by alveolar macrophages via scavenger receptors, particularly the macrophage receptor with collagenous structure (MARCO). Because RCS is indigestible, its uptake generates ROS and disrupts phagolysosomal membranes, resulting in leakage of lysosomal contents into the cytoplasm. This intracellular danger signal triggers assembly and activation of the NLRP3 inflammasome, leading to caspase-1-mediated cleavage of pro-cytokines and release of pro-inflammatory cytokines, such as interleukin (IL)-1β and IL-18

light microscopy, which provides an important diagnostic clue.[27] In advanced disease, coalescence of silicotic nodules into large fibrotic masses with marked distortion of lung parenchyma[27] results in PMF. A pathologically distinctive feature of AS silicosis is the coexistence of dense hyalinised fibrosis (chronic silicosis) and proteinaceous material within alveolar spaces (silicoproteinosis/acute silicosis;[27] see Figure 3), reflecting the extremely high intensity of RCS exposure in AS fabrication.[2]

Elemental analysis of AS-exposed lung tissue has further demonstrated high concentrations of aluminium and silicon at the centre of silicotic nodules, a mineralogical pattern that differentiates AS silicosis from natural stone silicosis.[5,25]

Without particulate identification under polarised light microscopy, the cellular aggregations seen in early silicosis mimic the non-caseating granulomas commonly seen in classical sarcoidosis, which is usually idiopathic.[20,28] Hence, early-stage AS silicosis is frequently misdiagnosed as sarcoidosis due to their histopathological and clinical overlap, making histopathological characterisation of the condition challenging.[23] In addition, transition metals present in AS dust, including aluminium, titanium, and zirconium, may also induce sarcoid-like granulomatosis, making the diagnosis more challenging.[29]

To reflect the expanding histopathological spectrum of AS silicosis, evolving terminology with descriptive indicators such as "silica-induced sarcoidosis", "sarcoid-like granulomatosis induced by silica", "sarcoid-like granulomatous lung disease" has been introduced in the scientific literature.[30] To unify these similarly overlap conditions that appear to exist along a continuum, silicosarcoidosis is an increasingly commonly used term to refer to a recognised entity characterised by the concurrent histological presence of silicotic nodules and epithelioid granulomas.[31] However, consensus on these entities remains unclear. Histopathological differentiation of these entities from AS silicosis remains clinically and radiologically challenging and requires integration of occupational history and clinical trajectory.

Underdiagnosis and Surveillance Gaps

Despite increasing recognition, AS silicosis remains underdiagnosed in many countries. Surveillance systems for occupational lung disease are often fragmented, passive, or absent, especially in small-scale enterprises that dominate the AS industry.[32] Diagnostic delays are frequent, with many cases initially misdiagnosed as infection, sarcoidosis, or autoimmune disease,[8,16,21] especially in the absence of a detailed occupational history.[7,8] Evidence from jurisdictions that have implemented systematic screening suggests that a substantial proportion of AS workers would remain undetected without active surveillance. Screening programmes in Australia identified silicosis in approximately 23–28% of screened workers,[32] with many AS workers asymptomatic at the time of diagnosis, underscoring the limitations of symptom-based case finding.[32]

These gaps underscore the need for improved occupational health surveillance, clinician awareness, and regulatory oversight in industries involving AS fabrication.

Regulatory Landscape of AS

The rise in AS cases has prompted regulatory responses in several jurisdictions (Table 1).

Clinical Manifestations

Symptomology

AS silicosis predominantly manifests as accelerated disease with mixed features, though acute and chronic patterns may also occur depending on exposure intensity and duration. Typically, symptoms in AS workers often appear after a much shorter latency period (ranging 4–10 years) due to the high intensity of exposure,[2,7] unlike traditional chronic silicosis (Table 2).

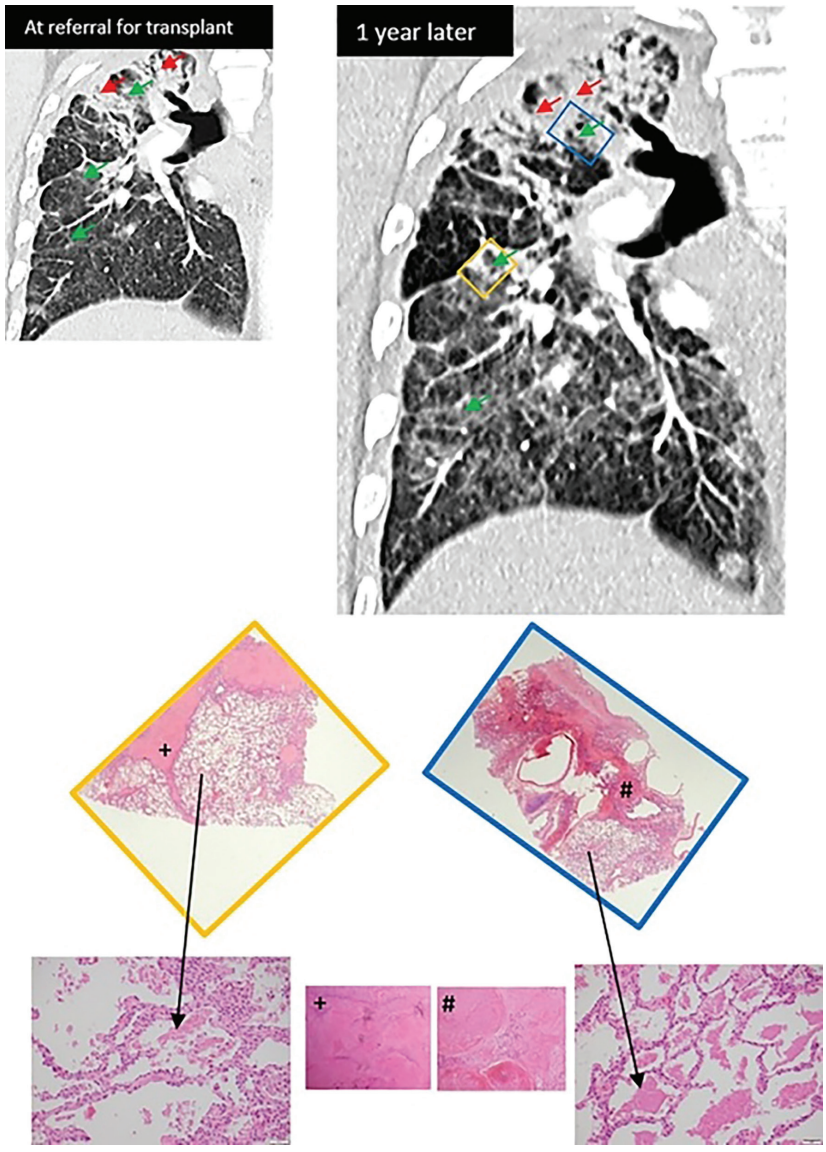


Figure 3. Radiological and histopathologic findings of an artificial stone worker with silicosis at referral for transplant and 1 year later. Conspicuous areas of alveolar proteinosis (black arrows) corresponding with areas of ground glass changes on computed tomography chest imaging (green arrows). Adjacent parenchyma shows dense fibrosis, silicotic nodules, and progressive massive fibrosis (PMF) (indicated by '+' and '#'). Red arrows indicate upper lobe confluent opacities, traction bronchiectasis, hilar retraction, and volume loss, consistent with PMF. Reproduced from Levin et al.,[27] with permission from John Wiley and Sons

The clinical presentation of AS silicosis can range from asymptomatic disease in early stages, which may not correlate with radiological severity, to severe and debilitating respiratory failure with complications, including pulmonary hypertension and right heart failure.[2,3,21] Patients may present with non-specific respiratory and constitutional symptoms, contributing to diagnostic confusion with infectious or inflammatory diseases.

Other Associated Conditions

Beyond classic silicosis, RCS exposure has been associated with a broader spectrum of interstitial lung diseases (ILDs). Notably, it is an independent risk factor for idiopathic pulmonary fibrosis (IPF), with an estimated 4% of IPF cases attributable to RCS exposure.[2] Although 9–10% of workers with known occupational silica exposure have been reported to exhibit radiological features of usual interstitial pneumonia (UIP), including lower-zone predominant abnormalities and honeycombing, without findings of classic silicosis, a UIP pattern has not yet been described in published case series involving AS workers.[2]

Other pulmonary conditions commonly associated with AS silicosis include work-related asthma; chronic obstructive pulmonary disease (COPD), including emphysema, arising from polymer resins and VOCs; and lung cancer, given that RCS is classified as a Group 1 carcinogen by the International Agency for Research on Cancer.[2,13] Silica-induced impairment of alveolar macrophage function also confers a markedly increased susceptibility to mycobacterial infections, including silicotuberculosis and non-tuberculous mycobacterial disease, particularly *Mycobacterium kansasii*.[2]

Table 1. Key regulatory responses in the US, Australia, and the UK

Jurisdiction	Key response
US	US federal regulation of AS nationally remains anchored to the Occupational Safety and Health Administration respirable crystalline silica standard. However, across the different states, varying regulatory standards specific to AS have been imposed. Arising from the California silicosis epidemic in AS workers in 2025, California adopted a permanent silica standard to enhance worker protection during high-exposure trigger tasks involving AS containing more than 0.1% crystalline silica or natural stone containing more than 10% crystalline silica.[33] As of June 2025, silicosis also became a reportable disease in California.[33] In May 2026, the California Occupational Safety and Health Standards Board approved a petition to commence the process for prohibiting the fabrication and installation of AS products containing more than 1% crystalline silica.[34]
Australia	Since July 2024, the use, supply, and manufacture of AS benchtops, panels and slabs are banned across Australia while the importation of these products has been banned since January 2025.[35] Australia remains the first and only country thus far to implement a nationwide AS ban as of May 2026. Under the model Work Health and Safety Regulations, AS is defined as "an artificial product that: (1) contains at least 1% crystalline silica as a weight/weight concentration, and (2) is created by combining natural stone materials with other chemical constituents (such as water, resins, or pigments), and (3) becomes hardened".[35]
UK	In May 2026, the Health and Safety Executive published the first Control of Substances Hazardous to Health guidance sheet for AS workers, following concerns arising from silicosis deaths among young workers. The guidance outlines the controls that should be in place when working with the material, which effectively rules out dry-cutting, unless businesses can demonstrate an equally effective or better control is in place.[36]

AS: artificial stone, US: United States, UK: United Kingdom

Table 2. Disease spectrum and relevance in AS silicosis[2,27,32,37]

Disease spectrum	Characteristics	Relevance in AS silicosis
Acute silicosis	May manifest within weeks to a few years (typically less than 5 years) of first exposure. Rapidly progressive respiratory and constitutional symptoms, often resulting in respiratory failure and high mortality. Radiological descriptions may include diffuse centrilobular nodules, ground-glass opacities, and patchy bilateral consolidation on HRCT imaging.	Less common as a standalone entity but its features have been reported following exceptionally high-intensity RCS exposures. Both radiological and histopathological findings consistent with silicoproteinosis have been identified in a subset of AS workers.
Accelerated silicosis	Typically develops within 2 to 10 years of initial exposure. Rapid clinical, radiological, and functional progression, with overlapping features of both acute and chronic disease. Early disease may be radiographically occult on plain chest radiography, with abnormalities detectable only on HRCT, underscoring the limitations of conventional screening modalities. As disease progresses, imaging findings may include silicotic nodules, interstitial fibrosis, ground-glass opacities, and early conglomeration.	Predominant disease pattern observed in AS silicosis, reflecting the high silica content of AS and the frequent occurrence of high-intensity exposure during fabrication tasks. Carries a high-risk of progression to PMF in AS workers, often over a relatively short time frame and despite cessation of exposure.
Chronic silicosis	Classified into simple silicosis and complicated silicosis or PMF. Simple silicosis is characterised by small (less than 1 cm), upper lobe-predominant nodules and may be asymptomatic, often detected through surveillance imaging. These nodules may enlarge and coalesce into conglomerate masses consistent with PMF, accompanied by parenchymal distortion, lymphadenopathy, and emphysematous change.	Less frequently observed on its own in AS workers compared with traditional silica-exposed industries but remains relevant, particularly among individuals with longer employment duration and lower-intensity exposure.

AS: artificial stone, HRCT: high resolution computed tomography, PMF: progressive massive fibrosis, RCS: respirable crystalline silica

High-intensity exposure to RCS in AS is strongly associated with autoimmune diseases such as systemic sclerosis, rheumatoid arthritis, and systemic lupus erythematosus.[9,38] This occurs alongside recognised syndromes, including Erasmus syndrome (systemic sclerosis with silica exposure) and Caplan's syndrome (rheumatoid arthritis with lung nodules).[39] Silica has also been associated with immune dysregulation, manifested by elevated autoantibody levels (e.g., anti-nuclear antibodies), circulating immune complexes, and increased immunoglobulin production, even in the absence of autoimmune disease.[39]

Silica exposure has also been linked to renal diseases such as chronic kidney disease and glomerulonephritis, potentially through both direct nephrotoxicity from silica particle deposition and indirect immune-mediated mechanisms.[2,11]

Disease Progression After Exposure Cessation

A clinically striking feature of AS silicosis is the potential for continued rapid radiological and functional deterioration even after exposure has ceased.[10] Studies have shown that up to 56% of AS workers exhibited radiological progression of two or more International Labour Organization (ILO) subcategories, and 37% developed PMF (Figure 4) following a mean follow-up of only four years.[10] The decline in lung function, with mean annual reductions in forced vital capacity (FVC) and forced expiratory volume in one second (FEV_1) of approximately 87 mL and 83 mL, respectively, was observed, with 25% of patients experiencing even greater losses, exceeding 150 mL per year in FVC and 133 mL per year in FEV_1.[10]

This distinguishes it from chronic silicosis, which tends to stabilise or progress slowly after cessation of exposure.[3,10] This is consistent with the self-perpetuating inflammatory cycle described earlier.

Effect of Smoking

Tobacco smoking acts synergistically with RCS exposure to exacerbate pulmonary toxicity.[40] Critically, smoking substantially amplifies the risk of comorbid conditions in RCS-exposed workers, including lung cancer, COPD, tuberculosis, and autoimmune diseases, underscoring its contribution to overall morbidity and mortality in this population.[11,38,41]

A greater loss of lung function, with reductions in diffusing capacity of the lungs for carbon monoxide (DLCO), FVC, and FEV_1, was observed in AS workers who were current or former smokers compared with non-smokers.[42] However, current evidence suggests that smoking may not be the primary driver of radiological disease progression to PMF in AS workers.[10,42]

Diagnostic Approach for AS Silicosis

History Taking and Physical Examination

Occupational history is key to diagnosing AS silicosis, but it requires specific, targeted questioning, as workers may not recognise the term "engineered/artificial stone", may misclassify the material as "marble" or "granite", or may face language barriers, thereby concealing the true nature of the exposure.[2,8,15] Therefore, clinicians must explicitly ask about the fabrication of AS, including the duration of work, specific tasks performed (e.g., cutting vs. installing), and the use of dry cutting or grinding methods.[2] Given the shorter latency observed in AS silicosis, even relatively brief exposure histories are clinically relevant.[2,13]

Physical examination findings are frequently unremarkable with limited sensitivity and are generally unreliable for screening or diagnosis of AS silicosis.

Clinical Investigations

Pulmonary function testing (PFT) provides complementary information, but it has limited sensitivity in early-stage disease.[1] Restrictive, obstructive or mixed patterns with reduced DLCO have been observed in AS silicosis,[1,7] but can be normal despite radiologically confirmed silicosis.[2] Therefore, normal spirometry and gas transfer do not exclude the diagnosis and should not delay further evaluation in AS-exposed individuals.

Radiological classification of silicosis with plain chest radiographs is conventionally based on the ILO International Classification of Radiographs of Pneumoconioses,[43] but has limited sensitivity for AS silicosis, with up to 39.6% false negative rates.[32] High-resolution computed tomography (HRCT) with the International Classification of HRCT for Occupational and Environmental Respiratory Diseases (ICOERD)[44] is therefore increasingly preferred, albeit not a universally accepted CT-based classification system. In some areas, such as Western Australia, low-dose HRCT with modified ICOERD is mandated for screening and diagnosis.[2,45] HRCT commonly demonstrates mixed features, including bilateral upper-lobe fibrosis, small centrilobular or perilymphatic nodules, ground-glass opacities, and PMF.[7]

Invasive investigations, including bronchoscopy or lung biopsy, are not routinely required when the exposure history and

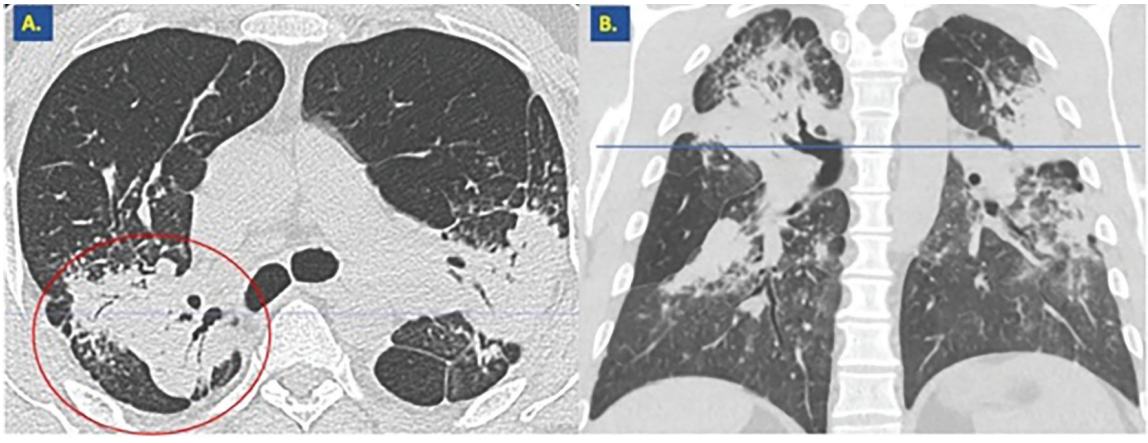


Figure 4. Complicated silicosis/progressive massive fibrosis. A 44-year-old man who worked in the engineered-stone countertop manufacturing industry presented with dyspnoea. Chest computed tomography (A, axial; B, coronal) revealed large bilateral masses predominantly in the posterior upper lungs with partially sharp margins, hilar and pulmonary distortion, bronchiectasis, and mild para-cicatricial emphysema (red circle), consistent with PMF. The blue horizontal line in the coronal view (B) corresponds to the level of the axial CT image (A).Reproduced from Fazio et al.,[2] with permission from Springer Nature

imaging findings are compatible. They may be considered when radiological features are atypical or when infection, malignancy, or sarcoidosis cannot be excluded.[2,5] When a biopsy is performed, histopathology typically demonstrates silicotic nodules, often with features of alveolar proteinosis in acute or accelerated forms of the disease.[7] Mineralogical analysis to identify metals may, in selected cases, assist by supporting attribution of metals to AS exposure.[5]

Laboratory investigations are non-specific and primarily serve to identify comorbidities and exclude alternative diagnoses. Elevated serum angiotensin-converting enzyme levels may be observed, but they are non-specific and occur in conditions including sarcoidosis, contributing to diagnostic confusion.[2,46]

Overall, AS silicosis is diagnosed by comprehensive occupational history-taking, radiological evaluation, and exclusion of alternative diagnoses.

Clinical Management and Treatment

Clinical management is primarily supportive and focuses on symptom control, the prevention and treatment of complications, and the optimisation of functional status.[2] Pharmacological therapies are limited and largely directed at associated conditions or secondary complications.[1,2]

Lung transplantation remains the only definitive treatment for AS silicosis.[2,7] However, access to transplantation may be constrained by socioeconomic barriers such as a lack of necessary medical insurance or financial means.[3] Among those who underwent transplantation, short- to medium-term survival appeared comparable to that for other indications, such as IPF, despite recipients being significantly younger at the time of transplantation.[7] Nevertheless, long-term prognosis remains guarded, as median post-transplant survival is limited (reported to be approximately seven years), thereby exposing younger recipients to the risk of premature mortality.[11]

However, specific interventions are being explored for AS silicosis. For patients presenting with silicoproteinosis, whole-lung lavage (WLL) has been used to clear proteinaceous material and dust burden from the alveoli.[47] While some studies have shown radiological improvement and symptomatic relief following WLL, evidence regarding improvement in lung function (spirometry and DLCO) has been mixed or statistically non-significant, and long-term survival benefits remain unclear.[47] Additionally, antifibrotic agents (e.g., nintedanib, pirfenidone, tetrandrine) are currently being evaluated for silicosis.[48,49]

Occupational Management

Prevention and Control Measures

Occupational management should be initiated promptly following diagnosis. The primary recommendation following diagnosis is removal from further exposure to RCS. However, removal does not guarantee disease stabilisation.[10]

If complete withdrawal from the industry is not immediately feasible, strict work restrictions and redeployment to a dust-free environment should be implemented.[1] Return to a silica-related workplace should only be considered after a formal medical assessment confirms fitness for work and after verification that effective workplace controls have been established.[50] Given that AS silicosis frequently affects relatively young workers with substantial functional impairment, disability assessment and workers' compensation form an important part of management.[1] In practice, navigating compensation systems can be challenging. Clinicians can facilitate access to appropriate medical, occupational, and compensation pathways.

Workplace Management

The prevention of AS silicosis relies heavily on the Hierarchy of Controls, summarised in Table 3.

Workplace Hygiene Monitoring

Regular workplace environmental monitoring is essential to ensure that workers' exposure to RCS remains below stipulated exposure limits and to verify that control measures are functioning effectively.

Personal exposure monitoring is typically conducted by placing respirable cyclones in the worker's breathing zone during specific tasks or for a full shift to collect dust for gravimetric analysis.[15,17,52] Workplace monitoring programmes should also encompass support staff who are exposed to RCS as bystanders.

Training and Education

Administrative controls and personal protective equipment (PPE) rely heavily on worker compliance, underscoring the importance of adequate training and education.[4] Employers must ensure that workers understand the serious health hazards associated with RCS exposure and can identify high-exposure tasks. Training should cover the correct implementation of controls, good housekeeping practices, and the proper use, fit, and maintenance of RPE.[33,51]

Medical Surveillance

Medical surveillance is an important secondary prevention strategy aimed at detecting silicosis in its early stages. Surveillance programmes should begin with a baseline medical assessment before, or shortly after, the commencement of silica-exposed work, followed by periodic evaluations.

Challenges and Gaps

Incomplete Understanding of AS Silicosis

The exact mechanism driving the accelerated and severe nature of AS silicosis remains poorly understood, although it has been postulated to result from the unique physicochemical properties of AS dust.[13] While the exceptionally high silica content is a primary factor, researchers hypothesise that other components of AS, specifically transition metal ions and polymer resins, significantly modify and amplify silica toxicity.[5,7,13,25]

Table 3. Hierarchy of controls in AS silicosis at the workplace	
Elimination	The most effective strategy to protect workers is to eliminate the hazard from the workplace entirely. A prime example of elimination is Australia's world-first regulatory action to completely ban the use, supply, and manufacture of engineered stone containing respirable crystalline silica, which took effect in July 2024.[35]
Substitution	Where complete elimination is not practical, substitution involves replacing high-silica engineered stone with lower-silica or silica-free alternatives.[4,6] While substitution reduces silica exposure, ongoing evaluation is required to ensure these new materials do not introduce alternative respiratory hazards.[4]
Engineering controls	Automated Machinery: The use of computer numerical control machines, robotic arm saws, and automated edge-shaping machines isolates the worker from the dust source, significantly reducing their exposure compared to using hand-held tools. [2,4,50] Wet Cutting: Processing AS with integrated water delivery systems (water-fed tools or sheet-flow wetting) should be used to suppress dust generation.[2] However, wet methods alone are often insufficient to reduce RCS below permissible exposure limits.[4] Dust Extraction: To further reduce RCS levels, wet cutting can be combined with LEV or on-tool dust extraction systems, which capture the dust directly at the point of generation before it enters the worker's breathing zone.[4] Further dust reduction can be achieved using extraction wall booths, suction benches, or waterfall curtain ventilation systems that use both air circulation and water to pull dust out of the environment.[2,17]
Administrative controls	Workshop Layout and Job Rotation: Workspaces should be designed to physically isolate dust-generating tasks, protecting support employee or nearby workers from bystander exposure. Employers can also implement shift or job rotations to minimise the total hours an individual worker spends performing high-exposure activities. Routine Housekeeping: Meticulous housekeeping prevents secondary exposure from settled dust becoming airborne again. Workplaces should prohibit dry sweeping or the use of compressed air for cleaning. Instead, they should utilise wet mopping, floor drainage systems, or industrial vacuums fitted with the appropriate high-efficiency particulate air filters.[2,17] Hygiene and Laundering: To prevent contamination outside the dusty work area, employers should provide separate designated changing and eating areas, and an industrial laundering service to clean dusty workwear.[51] Equipment Maintenance: There should be regular inspection, cleaning, storage, and maintenance of all machinery, ventilation systems, and personal protective equipment.
PPE	RPE should only be used as a supplementary measure in conjunction with robust engineering and administrative controls. AS workers must be provided with respirators appropriate to the level of RCS exposure and the tasks performed. International guidance generally recommends selecting respirators with an APF sufficient to reduce worker exposure below applicable occupational exposure limits.[52,53] In practice, filtering facepiece respirators (such as N95 or P2 masks) and elastomeric half-mask respirators typically provide an APF of 10 while full-face respirators provide an APF of 50, and PAPRs offer varying levels of protection depending on their configuration (e.g., loose-fitting hood PAPRs provide an APF of 25, half-face PAPRs an APF of 50, and tight-fitting full-face or helmet/hood PAPRs provide an APF of 1000).[33,52] However, N95 and P2 filtering facepiece respirators do not protect against VOCs emitted during AS fabrication. This should therefore be considered when selecting appropriate RPE, particularly where combined exposures to dust and chemical vapours may occur, and respirators with suitable chemical filter cartridges may be required.[2] In addition, workers who are not directly involved in fabrication tasks, such as forklift drivers, cleaners, and quality assurance staff, may still experience exposure to RCS due to ambient dust and poor housekeeping practices. These workers should therefore also be included within workplace respiratory protection programmes.[53]

AS: artificial stone, LEV: local exhaust ventilation, RCS: respirable crystalline silica, RPE: respiratory protective equipment, VOC: volatile organic compound, APF: assigned protection factor, PAPR: powered air-purifying respirator, PPE: personal protective equipment

Diagnostic Uncertainty and Inaccuracy

Diagnosing AS silicosis can be challenging because its radiological and clinical features overlap with those of other diseases such as sarcoidosis, respiratory bronchiolitis, hypersensitivity pneumonitis, and lung cancer, which can lead to misdiagnosis and delayed management.[23] This diagnostic uncertainty is compounded by a lack of validated, specific biomarkers for early diagnosis and prognostication, as no biomarker has yet demonstrated sufficient specificity or diagnostic accuracy for routine clinical use in silicosis.[54]

Limited Treatment Modalities

Currently, there is no curative therapy for silicosis; lung transplantation is the only definitive treatment for end-stage disease.[2] While drugs approved for IPF have shown promise in reducing silica-induced lung inflammation, collagen deposition, and fibrotic progression in mice, robust human clinical trials are still needed.[26,48]

There is emerging research on the use of mesenchymal stem cell therapy to reduce inflammation, and nanotechnology is being explored to create nanocarriers that enhance targeted

drug delivery and retention in the lungs while minimising systemic toxicity,[22,26] but they continue to remain experimental.

There is a growing interest in immunomodulatory approaches targeting early inflammatory pathways in silicosis. For example, inhalation of anti-NLRP3 molecules targeting the NLRP3 pathway or IL-1 pathway antagonists such as anakinra may provide more targeted delivery of the drug and limit systemic toxicity.[38]

Suboptimal Medical Surveillance Strategy and Implementation

Current surveillance strategies rely heavily on chest radiographs and PFT, but there is a growing consensus that these tools are inadequate. Although HRCT is more sensitive in detecting early pathological changes, it is often associated with higher costs and radiation exposure, which complicate its use for frequent, lifelong screening.[46]

Artificial intelligence (AI) is increasingly being explored as a potential tool to address limitations in screening, diagnosis, and staging of pneumoconiosis, including AS silicosis. Recent advances in AI, including deep learning and convolutional neural networks, show promise for improving screening sensitivity and staging accuracy, with reported diagnostic accuracies exceeding 90% in preliminary studies.[55,56] However, differentiation from similar ILDs remains challenging, and clinical integration requires further validation and interpretability tools.[23,55,56]

Even with an ideal medical surveillance and screening tool, the implementation of surveillance for silicosis remains inconsistent despite legal requirements, particularly in small fabrication workshops.[6,16,18]

Lack of Low-silica and Silica-free Alternatives

Australia's world-first ban on AS has accelerated the adoption of next-generation low-silica or silica-free alternative materials, but their long-term health effects remain uncertain. The toxicological profile of replacement materials is not yet fully characterised.

Early evidence raised concerns about the composition and toxicity of dust generated during the processing of low-silica and zero-silica products. They are highly heterogeneous and commonly incorporate recycled glass, aluminosilicates such as feldspars, aluminium trihydrate, and polymeric resins.[4] During machining, these materials can generate respirable dust containing alternative metals and VOCs, which may result in health effects similar to those of AS.[4]

Continued longitudinal evaluation is required to ensure that these alternatives do not introduce unintended health risks.

Role of the Clinician and Call to Action

Maintain High Index of Suspicion

AS silicosis shifted the demographic of occupational lung disease. A high index of suspicion among medical professionals is necessary when evaluating relatively young workers presenting with symptoms of ILD and a short latency period.[9] Occupational history remains key, with a low threshold for further workup in exposed workers.

Provide Patient Education

For diagnosed or suspected cases, clinicians must clearly communicate the severe risks of continued exposure to RCS, including accelerated decline in lung function, and strongly advise immediate removal from the exposure source to reduce disease severity.[6] There is also a need for transparent discussions about the natural progression and prognosis of silicosis, potentially leading to PMF, respiratory failure, and the need for lung transplantation, even after workplace exposure has ceased.[10]

Clinicians must educate patients on the warning signs of disease complications and other associated conditions, and consider implementing routine screening for complications.

For workers who are starting work or continuing to work in the industry, education and training regarding workplace safety measures are vital. Clinicians should emphasise the importance of the hierarchy of controls, explaining to workers and employers that they should not rely solely on PPE; these must be used in conjunction with other control measures. As the AS industry often employs migrant workers, educational programmes should account for language and demographic factors.[15] Hazard communication through translated materials, audiovisual resources, and safety signage is important to ensure comprehension and to reinforce safe work practices.

Reporting and Surveillance

The medical professional's role extends beyond individual patient care. Clinicians must also comply with mandatory reporting requirements for silicosis, which vary by jurisdiction but generally require notification of occupational lung diseases to public health authorities or occupational safety regulators.

These public health notifications are critical because they serve as sentinel health events, signalling hazardous occupational exposures and triggering workplace investigations by regulatory agencies, including worksite inspections, enforcement of silica exposure standards, and medical screening of similarly exposed, potentially asymptomatic co-workers.[6,16]

Notification also contributes to disease registries, facilitating the collection of critical epidemiological data, improving the understanding of the natural history of silicosis, identifying emerging sources of silica exposure, and informing prevention strategies and therapeutic research.[9]

CONCLUSION

AS silicosis represents a preventable resurgence of severe silica-related lung disease and an emerging public health crisis. Unlike classic silicosis, it affects younger workers exposed to high-intensity RCS and leads to a more aggressive and rapidly progressive disease. This reflects not only the potentially hazardous properties of materials but also gaps in workplace controls, surveillance, and early detection.

Effective prevention requires a combined approach. Regulations must be paired with strong controls and systematic medical surveillance. Clinician awareness and timely reporting are equally critical to enable early detection and interventions.

Global responses remain uneven, with many jurisdictions relying on conventional silica standards that may be inadequate for AS exposures. There is a clear need for greater international harmonisation of regulations, exposure limits, and surveillance systems, alongside targeted protection for vulnerable workers, particularly those in small enterprises and among migrant populations.

AS silicosis is a sentinel warning of the consequences of delayed implementation of occupational health safeguards. Coordinated action across clinical, occupational, and policy domains is essential to halt the resurgence of this preventable disease in modern workplaces.

Footnotes

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Concept - Design - Data Collection or Processing - Analysis or Interpretation - Literature Search - Writing: All authors contributed equally to all sections.

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Declaration Regarding the Use of AI and AI-assisted Technologies: During the preparation of this work, the authors utilised Claude v 4.6 Sonnet solely to assist with language editing to improve linguistic clarity, and OpenAI's ChatGPT v 5.6 Luna, based on scientific content created by the author(s), for the visual formatting and conceptual layout of "Figure 2. Simplified Pathogenesis of Silicosis.". The final schematic was verified against established pathophysiological pathways to ensure scientific accuracy. After carefully reviewing and editing the content as necessary, the authors take full responsibility for the publication's content. This incorporation of AI tools primarily affected the visual formatting and conceptual design of Figure 2.

Supplementary Material Link: https://d2v96fxpocvxx.cloudfront.net/25021570-dd5c-48cc-b052-771af43e75cc/content-images/929bef3f-12a6-4394-8a59-2fa4fc594bfd.pdf

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