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**Editorial**

# Artificial Stone Silicosis: Understanding an Emerging Occupational Epidemic

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Documented as early as ancient Egypt and formally defined by the first international scientific consensus at the 1930 Johannesburg Conference on Silicosis, silicosis is among the oldest recognized occupational diseases. For much of its documented history, it was predominantly associated with well-known traditional industries and generally diagnosed in older workers after prolonged exposure. This epidemiological profile began to change in the early 2000s with the outbreak of denim sandblasting-associated silicosis, which was first brought to the attention of the international scientific community through reports from Türkiye. That outbreak—characterized by exceptionally intense exposure, short latency, rapidly progressive disease, and a strikingly young affected population— now finds a troubling parallel in the artificial or engineered stone industry, a relatively new but highly hazardous sector producing similarly aggressive disease among young workers exposed to extraordinarily high concentrations of respirable crystalline silica (RCS). In this issue of *Thoracic Research and Practice*, the review entitled "Artificial Stone Silicosis: New Face of an Old Disease" provides a timely and clinically oriented synthesis of the occupational context, pathogenesis, clinical manifestations, diagnostic challenges, management, and prevention of this emerging disease.[1]

Since its introduction in Italy in the early 1970s, artificial stone (AS) has evolved into a global industry, increasingly displacing natural materials such as marble and granite in kitchen and bathroom surfaces, interior finishes, and decorative applications, while production and demand continue to expand despite the considerable occupational health burden associated with its fabrication.[2] Parallel to the expansion of the industry, the number of reported patients has increased steadily, with case series, cross-sectional studies, and epidemiological reports now emerging from a growing number of countries. In the most recent American Thoracic Society workshop report summarizing the available evidence, at least 6,351 cases of AS silicosis had been identified worldwide.[3] However, this figure almost certainly represents only the tip of the iceberg, as the true global burden remains unknown and is likely to be substantially underestimated. Much of the literature, from a limited number of countries, consists of case reports, case series, registry analyses, selected screening cohorts, and retrospective studies. Differences in case definitions, exposure characterization, imaging protocols, follow-up periods, and reporting systems complicate comparisons across studies. Underestimation is likely to be greatest in countries where fabrication occurs in small workshops with limited regulatory oversight.

Understanding why AS silicosis has become an increasingly important occupational respiratory health problem requires more than merely documenting new cases. Although its pathogenesis shares the core mechanisms of conventional silicosis, including macrophage activation, oxidative injury, inflammasome signaling, and progressive fibrosis, AS dust is a complex mixture rather than pure crystalline silica.[4] Polymeric resins, pigments, transition metals, and other additives may modify particle toxicity and intensify inflammatory and fibrogenic responses. Experimental studies increasingly suggest that the distinctive toxicity of AS dust is not solely determined by its high crystalline silica content (over 90%), but may also reflect the contribution of metals and other non-silica constituents that amplify inflammatory, cytotoxic, and oxidative responses and disrupt epithelial barrier integrity.[5,6] The current epidemic is simultaneously a consequence of hazardous material

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composition, uncontrolled high-intensity exposure, fragmented occupational surveillance, delayed clinical recognition, and inadequate regulatory action. The review emphasizes that exposure is not limited to workers directly operating cutting tools; cleaners, installers, drivers, and other workers in poorly controlled environments may also experience substantial secondary exposure.

Histopathological observations of mixed patterns, including silicotic fibrosis alongside silicoproteinosis, further support the concept that AS-related disease may extend beyond the conventional categories of acute, accelerated, and chronic silicosis. This mixed phenotype may also contribute to the rapid clinical progression observed in some workers. Another intriguing feature is the occurrence of radiological patterns that may mimic sarcoidosis, particularly in patients with chronic silicosis; such diagnostic overlap appears to be encountered more frequently in AS silicosis than in more traditional forms of the disease.[7]

The limitations of conventional chest radiography also deserve emphasis. Although chest radiographs and the International Labour Organization classification remain important in occupational health practice, they may fail to detect early disease associated with AS. High-resolution computed tomography is more sensitive in detecting early nodules, ground-glass opacities, conglomerate fibrosis, emphysema, and mixed radiological patterns. However, the optimal role and frequency of computed tomography in worker surveillance remain unresolved because of radiation exposure, cost, accessibility, and the absence of universally accepted screening protocols. The review therefore highlights an important gap: improved sensitivity must be balanced against feasibility and potential harm, particularly when screening young workers repeatedly over many years.

In our clinical experience, although many patients demonstrate the conventional radiological features of silicosis—including upper-lobe-predominant centrilobular or perilymphatic nodules, coalescent opacities, progressive massive fibrosis, and calcified or non-calcified mediastinal and hilar lymphadenopathy—some cases appear to show a less typical distribution. In particular, confluent mass-like opacities and consolidations may predominate in the middle lung zones, sometimes extending to the upper zones and abutting the pleural surface. Whether this pattern represents part of the broader radiological spectrum of AS silicosis remains uncertain. Nevertheless, it raises the possibility that the complex composition of AS dust—including finely and freshly fractured crystalline silica, resin-derived emissions, adhesives, additives, pigments, and metals—may influence not only disease severity but also its radiological phenotype. This hypothesis requires confirmation through larger, systematically characterized cohorts with detailed exposure, radiological, pathological, and mineralogical assessments.

Therapeutic uncertainty further strengthens the case for prevention. Supportive treatment, management of complications, whole-lung lavage in selected cases of silicoproteinosis, and experimental antifibrotic or immunomodulatory approaches may provide benefit in specific situations, but none currently offers a reliable means of reversing established fibrosis. Lung transplantation remains the only definitive option for advanced disease, yet access is limited, and it cannot compensate for a preventable exposure that continues to cause new cases. Therefore, the most important message of this review is not merely that clinicians should recognize AS silicosis earlier. The hazard posed by AS is already well established, and the priority must be to prevent disease before it develops through rigorous application of the hierarchy of controls. The most effective approach is control at the source, through elimination or substitution of high-silica materials with lower-silica or silica-free alternatives, complemented by rigorously implemented engineering controls such as wet cutting, effective local exhaust ventilation, and appropriate dust containment. Regulatory action is increasingly reflecting this principle. Australia has taken the most decisive step by prohibiting the manufacture, supply, processing, and installation of AS benchtops, panels, and slabs since July 2024.[3] California has also moved in this direction: following the adoption of strengthened silica standards, the state is now advancing rulemaking aimed at prohibiting the fabrication and installation of AS containing more than 1% crystalline silica, a potentially major step forward in primary prevention.[3,8]

Recognition must also trigger action. Each diagnosis should be considered a sentinel event requiring removal from exposure, notification to the relevant authorities, evaluation of coworkers, workplace investigation, and reassessment of exposure controls. Medical surveillance should complement, not replace, primary prevention. Respiratory protective equipment alone is insufficient when elimination, substitution, enclosure, automation, wet methods, and effective local exhaust ventilation have not been implemented.

Available screening studies suggest that the burden of AS silicosis is substantial, with reported prevalence ranging from approximately 12% in a small United States cohort to 23–28% in Australian screening programmes.[1,4] These figures likely still underestimate the true burden, particularly in countries without systematic surveillance.

In parallel, systematic characterization of the clinical and radiological spectrum of AS silicosis—including both the similarities to and the differences from conventional silicosis—may help improve recognition, phenotyping, and early diagnosis. Prevention must remain central to this agenda, with particular emphasis on primary prevention and effective engineering controls rather than reliance on personal protective equipment alone. Finally, future research should focus on quantifying the true burden of AS silicosis, clarifying how AS exposure differs from more traditional sources of RCS, and developing validated approaches for early detection, including biomarkers and low-dose computed tomography-based screening strategies.

**Conflict of Interest:** Ozlem Kar Kurt, MD, serves as the Editor of the journal. She was not involved in the peer review or editorial decision-making process for this manuscript and had no access to any information regarding its review.

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