

Silicosis and Pulmonary Sarcoidosis: A Crossroads of Interstitial Lung Diseases

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ABSTRACT

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Introduction

Interstitial lung diseases (ILDs) comprise a heterogeneous spectrum of conditions characterized by inflammation or fibrosis of the pulmonary parenchyma. In this context, silicosis and pulmonary sarcoidosis, traditionally considered separate entities due to their etiology, share more common ground than historically recognized. Growing evidence on the systemic effects of respirable crystalline silica has sparked interest in exploring potential pathophysiological intersections between these two diseases. Silicosis, an occupational lung disease caused by chronic inhalation of crystalline silica, is the most prevalent pneumoconiosis worldwide. Its clinical presentation ranges from chronic forms to accelerated and acute forms, the latter occurring in contexts of intense and prolonged exposure. In recent years, a new scenario has emerged: the epidemic of engineered stone (ES)-related silicosis. This material, widely used in kitchen and bathroom countertops, contains more than 90% silica by weight, compared to granite (40–50%) or marble (<10%). The dry cutting and processing of these materials has triggered epidemic outbreaks in countries such as Australia, Spain, Israel, and the United States. [1] The health impact of this new wave of silicosis is alarming. In Aus-

tralia, an active screening program identified silicosis in 21% of engineered stone workers. [2] In the United States, surveillance studies in California found prevalence rates as high as 12% among evaluated workers, many of whom were uninsured Latin American immigrants.

[1] Despite regulatory efforts, silica exposure in fabrication workshops frequently exceeds permissible limits, even when using wet cutting or localized ventilation. [3,4] In contrast, pulmonary sarcoidosis is a granulomatous disease of unknown etiology, characterized by the formation of non-caseating granulomas primarily affecting the lungs, though it can involve multiple organs. [5] Although traditionally classified as an idiopathic disease, multiple studies have identified associations with environmental, infectious, and genetic factors. Among the suspected environmental factors, exposure to silica dust has gained prominence. A national Danish cohort study involving over 5 million workers evaluated the association between occupational silica exposure and interstitial lung diseases. The authors found a significant increase in the incidence of pulmonary sarcoidosis with higher cumulative exposure to silica, reporting an incidence rate ratio (IRR) of 1.06 for every 50 $\mu\text{g}/\text{m}^3$ -year of exposure. [6] This relationship was also observed with idiopathic interstitial pneumonias (IRR 1.03) and,

as expected, with silicosis (IRR 1.20). [6] These findings support the hypothesis that silica may act as an immunological trigger across a broader spectrum of ILDs.

From a pathophysiological perspective, the mechanisms of both diseases show notable overlaps. Silicosis induces a chronic inflammatory response with persistent activation of macrophages and release of profibrotic cytokines such as TGF- β . In sarcoidosis, although the initial trigger remains unknown, a dysregulated immune response—both innate and adaptive—has been described, with the formation of non-caseating granulomas composed of macrophages, CD4+ lymphocytes, and dendritic cells. [7] Common genetic associations have been identified (e.g., HLA-DRB1, BTNL2, ANXA11), as well as shared immunological pathways, such as NLRP3 inflammasome activation and the production of IL-1 β and IL-17.

The clinical overlap between both conditions can complicate diagnosis. Silicosis and sarcoidosis may share respiratory symptoms, functional abnormalities, and even radiological patterns. In silica-exposed workers who develop pulmonary granulomas, the diagnostic dilemma arises: is it silicosis with an atypical granulomatous reaction or silica-induced sarcoidosis? This gray zone highlights the need to reassess current diagnostic criteria and systematically include occupational context in clinical evaluation.

From a public health perspective, the intersection of silicosis and sarcoidosis compels a reevaluation of prevention, diagnostic, and management strategies.

It is urgent to strengthen epidemiological surveillance in high-risk sectors, incorporate occupational history in medical assess-

ments, and establish more effective screening programs. Additionally, restrictive policies on the use of high-silica materials should be promoted, as Australia has begun doing by banning engineered stone from 2024 onward. [1,7] Silicosis can no longer be considered a relic of the industrial past. Its resurgence in the 21st century in new forms presents challenges that go beyond clinical medicine. Sarcoidosis, for its part, still veiled in idiopathy, begins to reveal patterns pointing to specific environmental factors. The convergence of both diseases in the occupational silica exposure setting is not coincidental, but a call to action for the medical, scientific, and regulatory communities.

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