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Seminars in
ROENTGENOLOGY

Occupational Lung Disease

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Introduction

Occupational lung diseases comprise a wide group of disorders that can be acute or chronic and can affect the airways, lung parenchyma, or pleura. These disorders result from inhalation of a variety of dust particles and chemicals and continue to be a leading cause of work-related illnesses in the United States.[1] Development of occupational lung disease depends on toxic effects of inhaled substances in addition to both duration and intensity of exposure. Establishing a diagnosis of occupational lung disease can be challenging, as a single agent, such as asbestos, can have a variety of manifestations; a single disease, such as hypersensitivity pneumonitis (HP), can result from a wide variety of inhaled dusts or antigens; and the latency between the exposure and clinical disease may extend over years or even decades.

Work-related asthma is the most common form of occupational lung disease. Recognition of occupational causes of HP continues to increase. Although many occupational lung diseases have characteristic imaging findings, establishing an accurate diagnosis still requires integration of a thorough occupational exposure history, an understanding of clinical presentation, and results of appropriate diagnostic testing including pulmonary function testing and diagnostic imaging.

In this review, we discuss a variety of occupational lung diseases including pneumoconiosis, HP, and work-related asthma. The radiographic and computed tomography (CT) findings are described and illustrated.

The "classic" pneumoconiosis is characterized by mineral dust deposition in the lung and the accompanying host immunologic response and is characterized by the mineral responsible. A wide variety of inhaled minerals are associated with pulmonary manifestations, although the most common are coal dust, crystalline silica, and asbestos. Other inert dusts may result in radiographic abnormalities, including iron (siderosis), tin (stannosis), and barium (baritosis). The diagnosis of pneumoconiosis requires sufficient mineral dust exposure and duration, characteristic findings on chest radiograph, and absence of another illness that may have a similar appearance such as granulomatous infection or sarcoidosis. This review will first describe the characteristic findings of the 3 major pneumoconioses: silicosis, coal workers' pneumoconiosis (CWP), and asbestosis.

Silicosis

Silicosis results from inhalation of crystalline silicone dioxide (silica) and its accumulation in the lungs. Overall, 4 clinical forms of silicosis are recognized: simple silicosis, complicated silicosis, accelerated silicosis, and acute silicosis (silicoproteinosis). Occupations associated with silicosis include mining, quarrying, tunneling, foundry work, sandblasting, drilling, polishing, and ceramics manufacturing.[2] Lung injury occurs when inhaled silica particles are ingested by alveolar macrophages, which subsequently release cytokines as a consequence of the cytotoxic effects of silica. Fibroblasts are recruited to the lungs, where they form silicotic nodules consisting of mature collagen centrally and silica-laden macrophages peripherally.

Simple silicosis is defined as the presence of silicotic nodules in the lungs and typically develops 10-20 years after exposure. When crystalline silica in the periphery of these nodules induces a cascading fibrotic response, complicated silicosis or progressive massive fibrosis (PMF) ensues.[3] The primary chest radiographic finding of simple silicosis is multiple small nodules ranging from 1-10 mm, typically between 2 and 5 mm (Table 1). These nodules have a predilection for the apical and posterior segments of the upper lobes with centrilobular and perilymphatic distributions apparent on high-resolution CT (HRCT) (Fig. 1). In a perilymphatic distribution, nodules are seen along the bronchovascular bundles and in the subpleural interstitium. Subpleural nodules can coalesce to form pseudoplaques (Fig. 2). Ill-defined or branching centrilobular opacities have also been described as an early or atypical manifestation of silicosis. Hilar and mediastinal lymphadenopathy occurs in up to three-quarters of patients, and up to two-thirds of patients may have dystrophic calcification in lymph nodes, most commonly punctate and occasionally peripheral (eggshell)[4-7] (Fig. 3). With lower levels of silica exposure, lymphadenopathy may

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Table 1 Radiologic Findings of Silicosis

Simple: 2- to 5-mm nodules, upper and posterior lung, perilymphatic distribution, lymphadenopathy with or without calcification (eggshell), and pseudoplaques
Complicated: >1-cm, unilateral or bilateral, symmetric poorly marginated opacities (PMF) in the periphery of midlung, retracting toward hilum. May calcify or cavitate. If cavitary, suspect tuberculosis. Paracicatricial emphysema
Accelerated: Radiographically same as complicated silicosis, develop more rapidly owing to heavy exposure over short period
Acute (silicoproteinosis): GGO, crazy-paving, consolidation, and CLN

CLN, centrilobular nodules; GGO, ground-glass opacity.

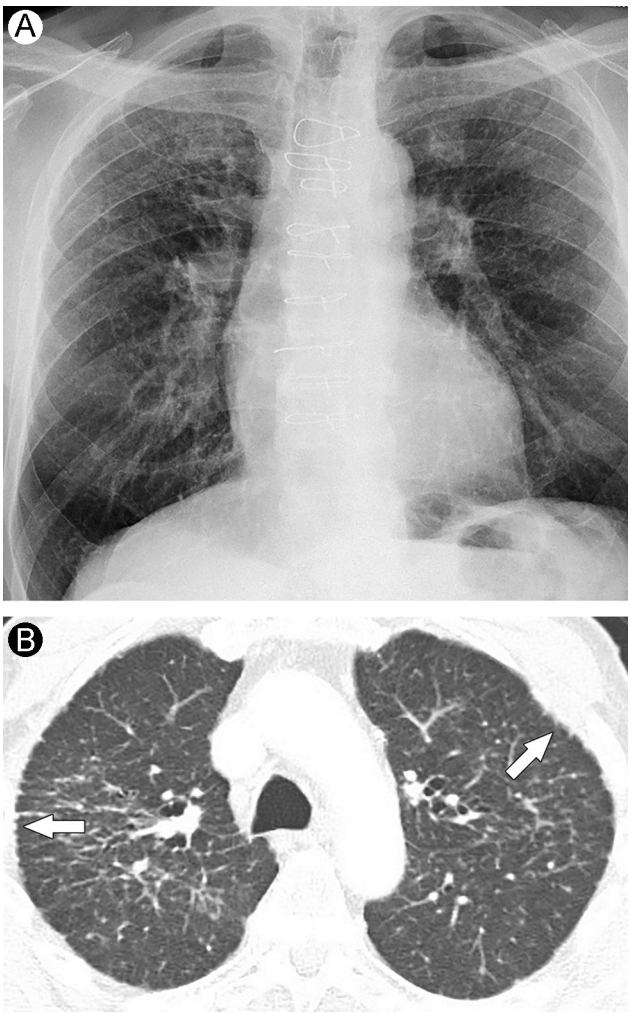

Figure 1 Male sandblaster with silicosis. (A) Posteroanterior radiograph shows small, poorly defined nodules in the upper lungs. (B) HRCT image shows a perilymphatic distribution of micronodules including subpleural (arrows) and peribronchovascular locations.

precede parenchymal findings. Subsequent lymph node damage may impair silica clearance and increase the risk of parenchymal disease.[8] Most patients with simple silicosis have little or no signs or symptoms of lower respiratory tract disease.

Complicated silicosis or PMF develops from coalescence of silicotic nodules and is defined by the presence of large (>1 cm) bilateral, typically symmetric opacities. These conglomerate masses usually develop in the periphery of the midlung zone, retracting toward the hilum. The lateral margins of large pneumoconiotic opacities typically are smooth and parallel the curvature of the chest wall. Additionally,

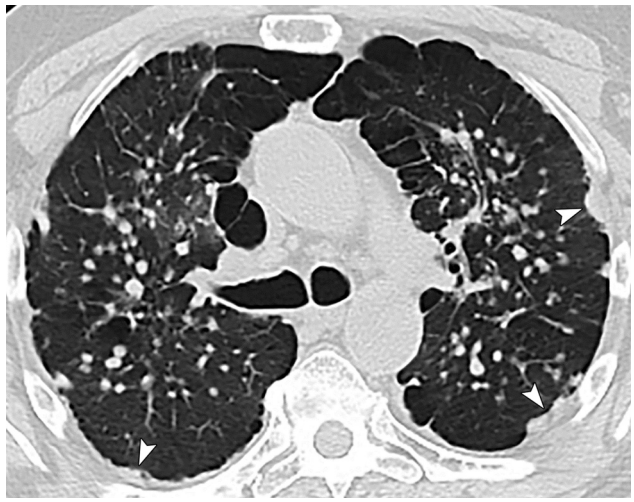

Figure 2 Man with simple silicosis. Unenhanced CT shows well-defined micronodules in the upper lobes along with paraseptal emphysema. Coalescent subpleural nodules form pseudoplaques (arrowheads).

paracicatricial emphysema frequently develops between the pleura and large opacities. HRCT better shows the coalescent nodular margins of large opacities, associated dystrophic calcification, as well as the background of small pneumoconiotic nodules (Fig. 3). As the number and size of the large opacities increase, the profusion of small pneumoconiotic nodules decreases, reflecting the coalescence of the small nodules. Cavitation of large opacities occurs because of ischemic necrosis or in association with tuberculosis.[5,7,9,10] Patients with PMF are usually symptomatic and present with varying degrees of respiratory dysfunction.

Exposure to high levels of silica dust over a short period of time (typically 4-10 years), can lead to accelerated silicosis. The clinical presentation of affected patients is similar to those with complicated silicosis. Rarely, acute silicosis (silicoproteinosis) can develop following acute exposure to a very high concentration of silica dust in an enclosed environment, primarily occurring in sandblasters.[11] Proliferation of type II pneumocytes results from the large silica exposure, leading to exuberant surfactant production and subsequent overfilling of air spaces. Histopathologic and imaging findings are similar to pulmonary alveolar proteinosis. Silicoproteinosis manifests on HRCT as patchy ground-glass opacity, consolidation, and poorly defined centrilobular nodules. A "crazy-paving" pattern consisting of ground-glass opacity with superimposed septal thickening has also been reported. Silicoproteinosis is usually progressive and leads to death from respiratory failure.[12,13]