Occupational and Environmental Lung Disease

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KEYWORDS

• Silica • Coal • Asbestos • Hard metal • Beryllium • Occupational asthma • Hypersensitivity pneumonitis • Biomass

KEY POINTS

- Occupational and environmental lung disease is a major cause of respiratory impairment worldwide.
- Despite regulations, exposures to the "classic" dusts, such as silica, coal, and asbestos, continue to be a worldwide cause of disease.
- New etiologies for occupational lung disease continue to emerge, and known causes are emerging in new industries.
- Nonoccupational environmental lung disease contributes to major respiratory disease, asthma, and chronic obstructive pulmonary disease.
- Knowledge of the imaging patterns of occupational and environmental lung disease is critical in managing patients with suspected or occult exposures.

INTRODUCTION

The association of occupational lung disease (OLD) with mining has been recognized since the 1500s when silica exposure and tuberculosis were described by Agricola.[1] Occupational and environmental lung diseases still remain major causes of pulmonary impairment worldwide. Globally, Driscoll and colleagues[2] report an estimated 386,000 respiratory deaths and almost 6.6 million disability adjusted life years (DALYS) attributable to occupational airborne particulates.

The "classic" mineral dust pneumoconioses, asbestos, silica, and coal, remain a worldwide problem. The Comparative Risk Assessment Study by the World Health Organization estimates that these pneumoconioses resulted in 30,000 deaths and 1,288,000 DALYS; it has been suggested that the methodology in this study grossly underestimated the number of deaths due to strict data collection requirements.[3]

In the United States, industrial regulations have decreased the prevalence of pneumoconioses. Since the introduction of the Coal Miners Health and Safety Act in 1969, the percentage of underground miners with a 20-year to 24-year tenure with radiographic findings of coal worker's pneumoconiosis (CWP) declined from 32.8% in 1970 to 1.9% in 2009. Despite these strict regulations, higher rates of CWP and progressive massive

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fibrosis (PMF) are reported in mines with fewer than 50 employees compared with larger mines.[4]

Immunologic factors contribute to OLD. Hypersensitivity pneumonitis (HP) occurs across professions. Hard metal and beryllium result in immune-mediated toxicity. Recent literature proposes the concept of mixed-dust exposure resulting in an immune-mediated chronic interstitial pneumonia pattern, although the impact of smoking and environmental risk factors remain unclear.[5]

Airway-related respiratory disease is the largest category of OLD, with estimates that occupational exposure accounts for 11% of asthma morbidity and mortality, translating globally to 38,000 deaths and 1.6 million DALYS.[6] Occupational diacetyl exposure and nylon flock are recognized causes of airways disease.

Finally, nonoccupational environmental exposures significantly contribute to major respiratory disease, asthma, and chronic obstructive pulmonary disease (COPD). Four major drivers of global lung disease are tobacco, indoor air pollution (biomass fuel burning), external air pollution, and occupational exposure.[6] A thorough evaluation of these factors is beyond the scope of this article. The contribution of tobacco and age in the development of lung fibrosis is briefly discussed.[7,8]

Identifying occupational exposures as a cause of lung injury requires a thorough knowledge of the patient's history, physical examination, laboratory testing, and medical imaging. The low cost and limited radiation exposure of chest radiography make it the most widely used medical imaging examination for workforce screening. High-resolution computed tomography (HRCT) has become the test of choice for characterizing diffuse lung disease, even supplanting the need for surgical lung biopsy in many patients with idiopathic pulmonary fibrosis.[9] The sensitivity and specificity of HRCT exceed those of chest radiography in the setting of pneumoconiosis.[10] This article reviews the findings of the classic mineral dust pneumoconioses, immune-mediated, and airway centric OLDs, and describes several more common environmental lung diseases. Given the often long latency of these diseases and occasionally occult nature of exposures, physicians must anticipate these diseases in their practice and recognize characteristic imaging features.

IMAGING FEATURES OF OCCUPATIONAL LUNG DISEASE

Dusts

Pathophysiology of particulate clearance

Pneumoconiosis, meaning "dusty lungs," is most often due to coal dust, silica dust, and asbestos fiber inhalation and is among the most common causes of OLD. When dust is inhaled, the larger particulates deposit on the mucosa of the nose and large airways and are cleared by mucociliary transport in approximately 8 hours. Smaller particles reach the alveoli and are phagocytized by alveolar macrophages. Macrophages migrate to the lymphatics where they are eliminated. Lymphatic drainage is driven by pulmonary artery pressure (lower in the apices and on the right) and chest wall excursion (lowest in the upper posterior chest wall). The slowest lymphatic clearance and thus greatest retention of particles is in the upper posterior lung, right worse than left. A high particulate burden rapidly overwhelms these mechanisms. Macrophage aggregates may be engulfed by type I pneumocytes and incorporated into the interstitium. Dust particles can cause direct epithelial damage, resulting in bronchitis and impaired ciliary clearance. Alveolar macrophages release inflammatory mediators that produce extracellular matrix components, such as collagen, and stimulate fibroblasts leading to fibrosis. Recruitment of peripheral blood monocytes and neutrophils causes alveolar inflammation and damage to the alveolar epithelial cells. Damage to the airspaces can result in emphysema. Mineral dust exposure is a complex interaction of reactive oxygen species, antioxidants, cytokines, growth factors, eicosanoids, proteases, and antiproteases, leading to lung dysfunction and pathology.[11]

Deposition of fibers depends on the ratio of fiber length and width (aspect ratio), which determines aerodynamic properties. Deposition in larger airways occurs as the ends of inhaled fibers impact the mucosa, particularly at sites of directional change, such as bifurcations. Most fibers deposited in the mucosa are removed via mucociliary clearance, migrating fibers to the larynx to be swallowed and removed by the gastrointestinal tract. In the periphery, airflow is slower, and fibers settle by gravity. Thin fibers tend to penetrate deep into the lung, aligning with laminar flow. Fibers deposited in the nonciliated airways beyond the terminal bronchioles may migrate through the epithelium into the interstitium, ultimately leading to pulmonary fibrosis.[12] Fibers cleared via lymphatic drainage to the pleura accumulate at lymphatic stoma inciting inflammation, ultimately leading to pleural plaque formation and mesothelioma.[13] Longer fibers that cannot be broken down or engulfed by alveolar macrophages remain in the lung as asbestos bodies.

Silica

Silica or silicon dioxide is most commonly encountered in its crystalline form (quartz or cristobalite).