Imaging of Occupational Lung Disease

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KEYWORDS

• Occupational lung disease • Occupational lung diseases • Pneumoconiosis • Pneumoconioses • Imaging • Radiology • Radiograph • Computed tomography

KEY POINTS

- In general, high-resolution computed tomography is more sensitive and specific than radiography for the detection of occupational lung diseases.
- Imaging findings are often nonspecific and must be interpreted with a multidisciplinary approach in the context of occupational history.
- Although often thought of as a static topic, occupational lung diseases are changing with evolving understanding of old diseases and new and emerging diseases.

OVERVIEW

Occupational lung disease refers to a variety of disorders that affect the lungs following inhalation of dusts or chemical antigens in a vocational setting. Despite the safety standards established by health organizations, occupational lung disease represents one of the most common work-related illnesses. Occupational lung diseases are responsible for approximately 70% of all deaths from occupational diseases.[1]

Pneumoconisosis is a subset of occupational lung diseases and is defined as a pulmonary disease caused by inhalation of inorganic mineral dust. Pneumoconioses may be further subdivided clinicopathologically into nonfibrotic and fibrotic subtypes.[2] The nonfibrotic subtype, resulting from inert dusts (iron, tin, barium), involves an accumulation of dust-containing macrophages and may lead to radiographic abnormalities. However, substantial fibrosis and functional impairment do not occur. In contrast, the fibrotic subtype, as seen in silicosis, coal worker's pneumoconiosis (CWP), asbestosis, berylliosis, and talcosis, results in focal or diffuse fibrosis and manifests as substantial radiographic abnormalities as well as functional impairment.

IMAGING IN OCCUPATIONAL LUNG DISEASE

Since 1930, the International Labor Organization (ILO), with support from the National Institute for Occupational Safety and Health (NIOSH), has provided a classification system, which is used worldwide to objectively classify the changes associated with pneumoconiosis based on posteroanterior chest radiographs. This system was revised in 2000 and, following widespread adaptation of digital radiography, extended from analog radiography to include digital radiography in 2011.[3,4]

Chest radiographs are relatively low radiation dose, inexpensive, and widely available. Thus, it is not surprising that radiography remains the first-line imaging examination for both occupational-exposure surveillance programs and the workup of suspected occupational lung diseases. However, high-resolution computed tomography (HRCT) is more sensitive and specific than radiography for the evaluation of occupational lung disease,

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particularly during the early stages.[5–11] HRCT is used to further characterize radiographic abnormalities and for evaluation of symptomatic patients. Given the increased sensitivity and specificity of HRCT, the use of HRCT in screening and surveillance programs has been suggested, and multiple classification schemes for evaluating the HRCT findings of occupational lung diseases similar to the ILO system for radiographs have been developed.[12,13]

No such system has of yet been adopted, mainly because of concerns over radiation exposure and cost. However, the recent adoption of lung cancer screening programs using low-dose computed tomography (CT) as well as studies showing adequate pleural and parenchymal detail in asbestos-exposed patients screened for lung cancer with low-dose HRCT suggests that low-dose HRCT may be a viable future one-stop screening option.[14,15]

The American College of Radiology has developed evidence-based guidelines regarding the imaging of occupational lung diseases, providing direction regarding the 3 classic mineral pneumoconioses: silicosis, CWP, and asbestosis.[16] These guidelines assert the increased sensitivity of CT relative to radiography and note a limited role for MR imaging and PET/CT.

A final note should be made regarding interpretation of imaging findings in occupational lung diseases. With relatively few exceptions, most imaging findings found on radiography and HRCT overlap with other diffuse lung diseases. As such, establishing the diagnosis is often a multidisciplinary endeavor requiring integration of occupational history, physical examination, laboratory testing, pulmonary function testing (PFT), imaging findings, and when the diagnosis is uncertain, pathologic evaluation of bronchoalveolar lavage (BAL) fluid and/or transbronchial or surgical lung biopsy.

Coal Mine Dust Lung Disease

Inhalation of coal mine dust places coal miners at risk for developing a variety of disorders, including chronic obstructive pulmonary disease (COPD), silicosis, CWP, mixed dust pneumoconiosis, and the relatively recently recognized dust-related diffuse fibrosis (DDF). The term coal mine dust lung disease (CMDLD) is now being used to reference this spectrum of diseases.

SILICOSIS

Silicosis is caused by inhalation of inorganic crystalline silicon dioxide (silica) dust. Crystalline silica occurs naturally in rocks and sand. Despite occupational exposure limits in many countries, silicosis remains a common pneumoconiosis worldwide, with recent reports highlighting the risks of new construction materials, quartz conglomerates, and artificial stone products.[17–19] In addition, hydraulic fracturing or "fracking," in which large quantities of pressurized sand, water, and other chemicals are injected into fracture sites during the process of natural gas extraction exposes workers to silica as well as other volatile organic compounds. A recent study conducted by NIOSH researchers found that among 11 sites tested over 5 states, full-shift samples exceeded the permissible exposure limits for silica, in some cases by 10 times the occupational health criteria.[20] Additional potential avenues of exposure include mining, tunneling, drilling, quarrying, stonecutting, concrete manufacturing, polishing, masonry, brick lining, sandblasting, glass manufacturing, foundry work, pottery, ceramic and porcelain manufacturing, boiler scaling, vitreous enameling, and even clothing production (from sandblasting denim).[20–23]

There are multiple identified clinical forms of silicosis, including simple, complicated, accelerated, and acute. Simple silicosis occurs after 10 to 20 years of low- to moderate-level exposure.[24] Following inhalation, macrophages engulf silica, resulting in cytokine release, fibroblast proliferation, and formation of silicotic nodules with concentric layers of collagen and silica-laden macrophages as well as lymphoid cells, which become acellular and hyalinized.[25] Simple silicosis typically causes no symptoms or respiratory impairment.

Radiographic findings include small, well-circumscribed nodules (1–10 mm but typically 2–5 mm) favoring the upper posterior lungs. Nodules can calcify, often diffusely. Pseudoplaques occasionally form because of clustering of subpleural nodules. Mediastinal and hilar lymphadenopathy with or without calcification may be present. Additional findings include pleural thickening and pleural effusions.[26]

HRCT findings include multiple small well-defined nodules, often seen in a centrilobular and perilymphatic distribution (**Fig. 1**). Nodules may be diffuse in distribution or favor the upper lobes. CT can also demonstrate pseudoplaques, pleural thickening, and pleural effusions.[26] Additional CT findings include hilar and/or mediastinal lymphadenopathy, which may calcify.[27] Lymphadenopathy may be the first manifestation following lower levels of silica exposure, preceding the development of parenchymal findings.[23] Subsequent exposure and lymph node damage may interfere with lymph drainage and increase the propensity for parenchymal disease.[28] Eggshell

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