

Spectrum of high-resolution computed tomography imaging in occupational lung disease

Bhawna Satija, Sanyal Kumar, Umesh Chandra Ojha², Dipti Gothi¹

Department of Radiodiagnosis, ¹Pulmonary Medicine, ²Institute of Occupational Health, Environment and Research, Employee's State Insurance Hospital and Post Graduate Institute of Medical Science and Research, Basaidarapur, New Delhi, India

Correspondence: Dr. Sanyal Kumar, B-1/57, First Floor, Paschim Vihar, New Delhi - 110 063, India. E-mail: satijabhawna@yahoo.com

Abstract

Damage to the lungs caused by dusts or fumes or noxious substances inhaled by workers in certain specific occupation is known as occupational lung disease. Recognition of occupational lung disease is especially important not only for the primary worker, but also because of the implications with regard to primary and secondary disease prevention in the exposed co-workers. Although many of the disorders can be detected on chest radiography, high-resolution computed tomography (HRCT) is superior in delineating the lung architecture and depicting pathology. The characteristic radiological features suggest the correct diagnosis in some, whereas a combination of clinical features, occupational history, and radiological findings is essential in establishing the diagnosis in others. In the presence of a history of exposure and consistent clinical features, the diagnosis of even an uncommon occupational lung disease can be suggested by the characteristic described HRCT findings. In this article, we briefly review the HRCT appearance of a wide spectrum of occupational lung diseases.

Key words: High-resolution computed tomography; occupational lung disease; pneumoconiosis

Introduction

Occupational lung disease represents the most frequently diagnosed work-related condition after injuries. These comprise of various disorders secondary to the inhalation or ingestion of dust particles or noxious chemicals, and include pneumoconiosis, asbestos-related pleural and parenchymal disease, chemical pneumonitis, infection, hypersensitivity pneumonitis, and organic dust toxic syndrome.

Pneumoconiosis may be clinico-pathologically classified as fibrotic or non-fibrotic. Recent decades have seen a marked increase in concern about the adverse health effects of harmful exposures in the workplace. Recognition of

occupational lung disease is especially important not only for the primary worker, but also because of the implications with regard to primary and secondary disease prevention in the exposed co-workers.

The clinical, radiologic and pathologic manifestations of occupational lung disease may be identical to nonoccupational variants because of the lung's limited capacity of response to injury. A high degree of suspicion directing a thorough occupational history to search for potential exposures is the key to accurate diagnosis. Diagnosis of an occupational lung disease requires definite history of exposure to an agent known to cause interstitial lung disease (ILD), an appropriate latency period, a consistent clinical presentation, physiologic and radiologic pattern and exclusion of other known causes of ILD. When these conditions are fulfilled, the need for lung biopsy can be obviated. A biopsy needs to be performed for atypical presentations, both clinical and radiological, or when the exposure is to a new or poorly characterized agent.

Imaging plays an indispensable role in the evaluation of occupational lung disease. The radiograph of chest is the

Access this article online

Quick Response Code:



Website:
www.ijri.org

DOI:
10.4103/0971-3026.125564

most important diagnostic tool for evaluation. It can be unique or highly suggestive of an occupational disorder and may be sufficient, along with an appropriate exposure history, to establish a diagnosis. Despite the well-established role of chest radiography in accurately and inexpensively displaying a wide range of pulmonary pathology, equally well established limitations have been documented. The findings can be nonspecific and the sensitivity low, missing as many as 10 to 15 percent of cases with pathologically documented disease. It has been proved beyond doubt that CT, particularly high-resolution CT (HRCT), is superior to chest radiography in the detection of parenchymal abnormalities, is more accurate in providing differential diagnosis, and is free from considerable interobserver variation in its interpretation. The application of CT to the occupational lung diseases attempts to describe morphological feature of respiratory manifestation more adjacent to pathology. It is indicated as a thorough investigation for positive cases screened by chest radiograph. The utility of CT as a screening modality is still a question of debate. Cost and availability of the test as well as radiation issues are the reasons of excluding CT from screening tests. As HRCT detects pulmonary involvement of the occupational lung diseases earlier than conventional radiographs, intervention to exposed individuals in earlier stages would show better outcome. Also a well-spread awareness of certain dusts like asbestos as a definite carcinogen among workers has lead to demand of more sensitive screening for dust-related respiratory diseases. To overcome the concern of radiation exposure, a low dose technique and acquisition of limited number of slices can be introduced while using CT for screening purposes. Magnetic resonance imaging (MRI) has a limited role in evaluation of patients with occupational lung disease. It is helpful for distinguishing between progressive massive fibrosis (PMF) and lung cancer. PMF will show hypointense signal on both

T1W and T2W sequence whereas a lung cancer would be hyperintense on T2W sequence.

In this article, we briefly review the high-resolution computed tomography (HRCT) appearance of a wide spectrum of occupational lung diseases. The HRCT findings were evaluated in the background of occupational history, and histopathologic diagnosis was obtained through transbronchial or CT-guided biopsy, where the imaging findings were not specific.

Silicosis

Silicosis is caused by the inhalation of fine particles of crystalline silicon dioxide. Occupations such as mining, quarrying, drilling, foundry working, ceramics manufacturing, sandblasting, construction, roadwork, glass manufacture and tunnelling are associated with silicosis.

It occurs in two clinical forms: Acute silicosis and classic silicosis. Classic silicosis is further classified as simple or complicated. The complications include tuberculosis and carcinoma.

Acute Silicosis

Acute silicosis occurs after a very large, acute exposure to silica dust, primarily among sandblasters.

HRCT findings [Figure 1] include multiple bilateral centrilobular opacities, multifocal patchy ground-glass opacities, and consolidation with occasional crazy paving.^[1,2]

Classic Silicosis

Simple form

Simple silicosis is characterized by the presence of multiple

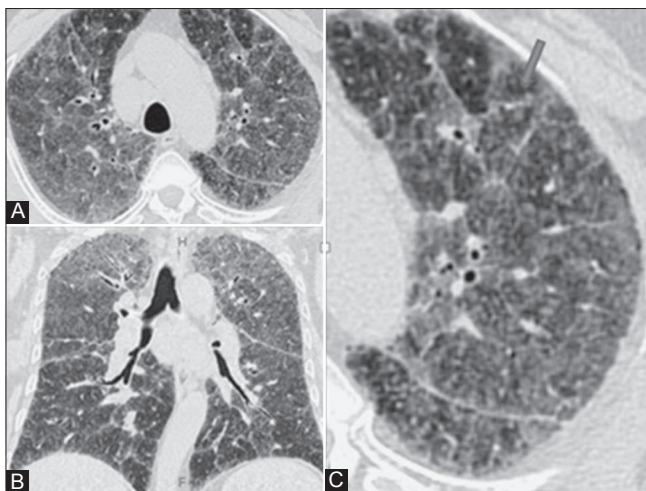


Figure 1 (A-C): A 38-year-old mason with silicoproteinosis. (A, C) Axial HRCT image (C:-600, W:1600) shows a geographic distribution of ground-glass opacity, interlobular septal thickening (arrow in C), and small nodules with crazy paving appearance. (B) Coronal reformation shows extensive but asymmetric involvement of both lungs

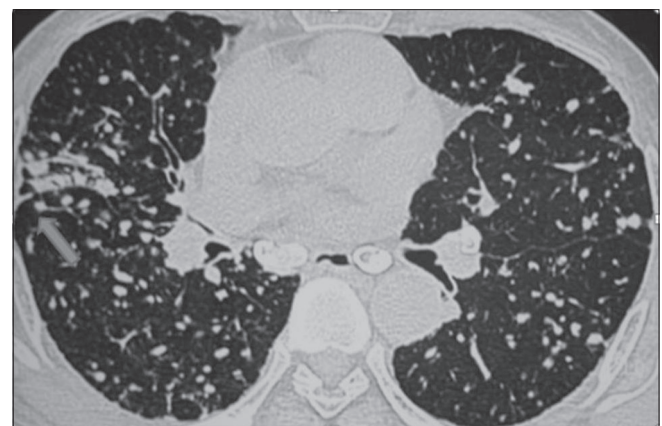


Figure 2: A 62-year-old man, a stone crusher with silicosis. Axial HRCT image (C:-600, W: 1600) shows small, well-defined nodules in the upper lobes. Coalescence of subpleural nodules forms pseudoplaques (arrows)

small nodules, 2-5 mm in diameter [Figure 2], accompanied by calcifications.^[3] The distribution may be diffuse, though upper lobe with posterior zone predominance is characteristic. HRCT shows perilymphatic distribution with nodules being observed in centrilobular, paraseptal, and subpleural regions. Subpleural nodules have rounded or triangular configuration resembling pleural plaques on confluence [Figure 2]. Hilar and mediastinal lymphadenopathy may precede the parenchymal lesions. Eggshell pattern of calcification of lymph nodes [Figure 3] is common.^[4,5]

The differential diagnosis include sarcoidosis and pulmonary lymphangitis carcinomatosa (PLC). These can be differentiated on the basis of history and careful evaluation of computed tomography. Central clustering of nodules in peribronchovascular distribution and presence of focal or multifocal abnormalities intermixed with near normal areas of lung suggest the diagnosis of sarcoidosis. In silicosis and coal workers pneumoconiosis, the nodules appear bilaterally symmetrical and show uniform distribution. Presence of reticular opacities and beaded septa is more characteristic of sarcoidosis and PLC.

Complicated form

Complicated silicosis, also known as progressive massive fibrosis, develops through confluence of individual silicotic nodules. The CT appearance includes focal soft-tissue masses, typically measuring more than 1 cm in diameter, with irregular margins, calcification, and commonly involving apical and posterior segments of the upper lobes, surrounded by areas of emphysematous change^[6,7] [Figure 3]. With progressive fibrosis, these large opacities migrate towards hila, accompanied by development of paracatricial emphysema.

Silicotuberculosis

Radiologic features include asymmetric nodules or consolidation, cavitation, and rapid disease progression [Figure 4].

Coal Worker's Pneumoconiosis

Coal worker's pneumoconiosis (CWP) results from exposure to washed coal or mixed dust consisting of coal, kaolin, mica, and silica.

Simple Form

The CT features include diffuse, small, 1-5 mm sized nodules, most numerous in the upper lung zone. They typically show a perilymphatic distribution, but sometimes centrilobular predominance may be observed [Figure 5]. These nodules have less distinct margins with granular appearance than those of silicosis and are smaller.^[8,9] The subpleural nodules may aggregate to form pseudoplaques. Lymph node calcification occurs less frequently. On CT,

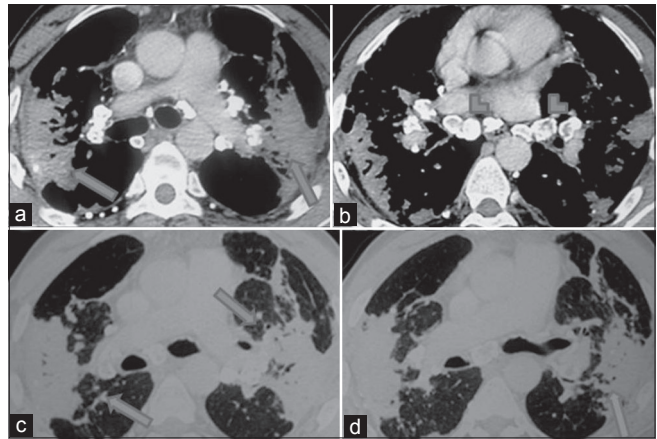


Figure 3 (A-D): A 49-year-old man working in glass manufacturing industry, with complicated silicosis and progressive massive fibrosis. Contrast-enhanced CT images in mediastinal (A, B) (C:50, W:350) and lung windows (C:-600, W: 1600) (C, D) show large, partially calcified fibrotic masses in the upper lobes (arrows) and peripherally calcified (eggshell) mediastinal and hilar lymph nodes (arrowheads)

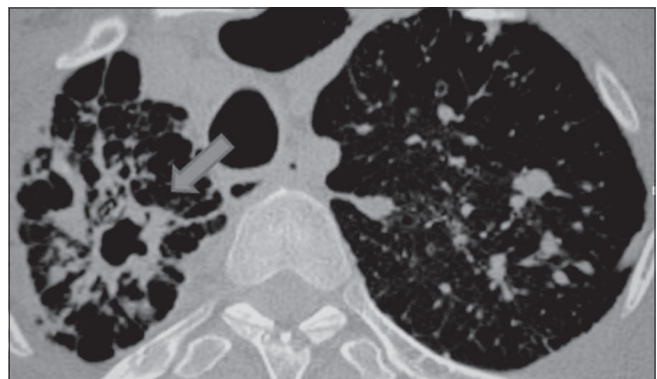


Figure 4: Silicotuberculosis in a 52-year-old stoneworker. Axial HRCT scan (C:-600, W: 1600) shows an irregular thick-walled cavitary lesion (arrow) in the upper lobe of right lung, suggestive of pulmonary tuberculosis. Multiple ill-defined nodular lesions seen in both lungs, with areas of paracatricial emphysema

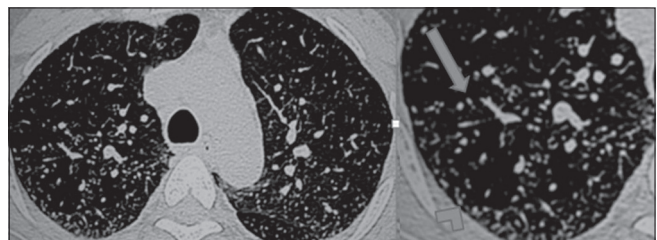


Figure 5: Coal worker pneumoconiosis in a 62-year-old man who worked for 20 years in a coal mine. Axial HRCT (C:-600, W: 1600) obtained at the level of the aortic arch shows numerous small centrilobular (arrows) and subpleural (arrowheads) nodules in both lungs

calcification is observed within the nodules in 30% of patients and develops as a central nodular dot.

Complicated Form

Progressive massive fibrosis occurs less frequently than in silicosis, seen as large masses of more than 1 cm in diameter.

These masses^[10] develop in mid zones or the periphery of upper lung and migrate toward hila, leaving emphysematous spaces between them and pleura [Figure 6]. Paracatricial emphysema develops with growth of large opacities, which may cavitate, with or without infection. Interstitial lung fibrosis (pattern similar to usual or nonspecific interstitial pneumonia) develops in less than 20% of coal workers and there is associated increased incidence of lung carcinoma.^[11]

Asbestos-Related Lung Disease

Asbestos exposure is seen in construction trades, building maintenance, mining, milling, industries manufacturing brake linings and pads, tiles, bricks, insulation material, and linings of furnaces and ovens, shipbuilding and repair, and automobile and railroad work.

Asbestos-Related Pleural Disease

Although quite uncommon, pleural effusion is the earliest manifestation.^[12] These are usually exudative and may be unilateral or bilateral. As the effusion regresses, diffuse thickening of the affected visceral pleura develops in more than 50% of patients.^[13]

Pleural plaques are the most common manifestation.^[14] These most commonly develop along the postero-lateral chest wall between the sixth and tenth ribs and along the central diaphragm with relative sparing of apices and costophrenic angles. These are seen as discrete, focal irregular areas of pleural thickening, commonly affecting the parietal pleura [Figure 7].

Parietal pleural thickening or pleural effusion associated with lung disease can be seen in rheumatoid arthritis, lymphangiomyomatosis, coal worker's pneumoconioses, tuberculosis, nontuberculous mycobacteria, and lymphangitic spread of carcinoma. Normal extrapleural fat, transverses thoracic and subcostalis muscles, and segments of intercostals veins can sometimes mimic pleural thickening. Visceral pleural thickening can be seen in diseases producing pulmonary fibrosis.

Rounded Atelectasis

This is also known as asbestos pseudotumor or Blesolvsky's sign. HRCT shows a peripheral mass abutting the pleura, round or oval in shape, with or without lung distortion and with a curving tail of bronchovascular structures spiraling into the mass (comet tail sign) [Figure 8]. There is associated ipsilateral pleural abnormality, either effusion or thickening.^[15]

Asbestosis

This refers to interstitial fibrosis secondary to asbestos exposure. HRCT findings [Figure 9] include subpleural

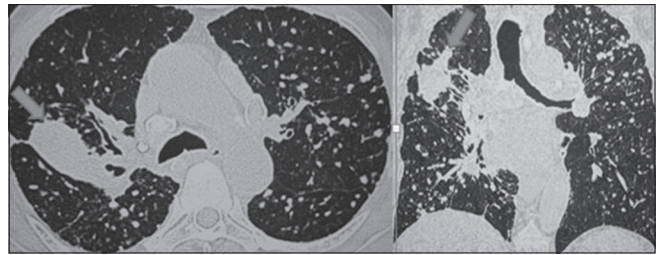


Figure 6: A 57-year-old male coal miner with complicated progressive massive fibrosis. Axial and coronal HRCT image (C:-600, W: 1600) shows large fibrotic mass in the right upper lobes (arrow) surrounded by numerous smaller nodules

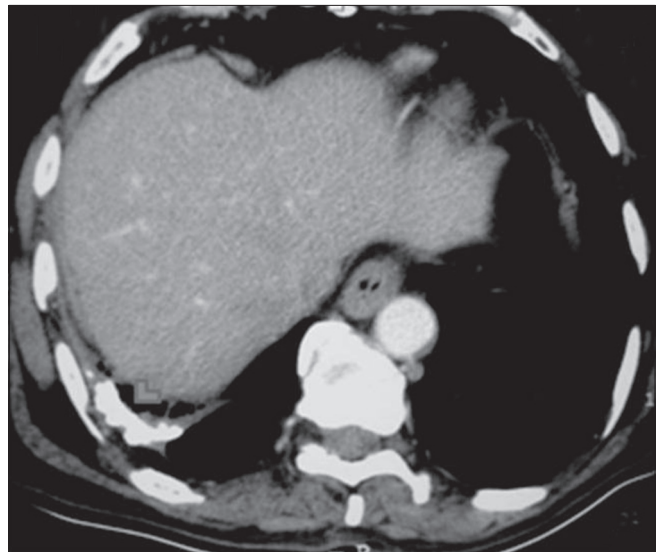


Figure 7: A 72-year-old man working in clutch plate manufacturing unit, with asbestos-related pleural disease. Axial CT image in mediastinal window (C:-600, W: 1600) shows calcified pleural plaque (arrowhead)

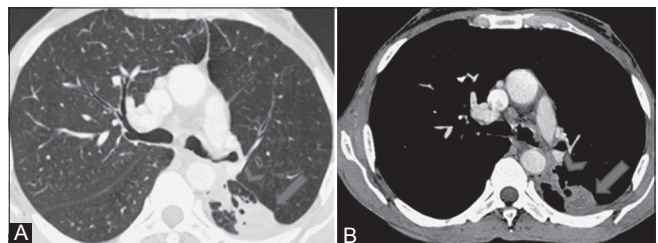


Figure 8 (A, B): A 54-year-old man with asbestos-related pleural disease and rounded atelectasis. CT image in lung (A) (C:-600, W: 1600) and mediastinal (B) (C: 50, W: 350) windows shows a round, subpleural focus of consolidation (arrow). This is intimately related to the adjacent pleural plaque and thickening with a curving tail of bronchovascular structures spiraling into the mass, "comet tail sign" (arrowheads)

curvilinear opacities, ground-glass opacity, subpleural poorly defined centrilobular nodules, thickening of interlobular septa, parenchymal bands, traction bronchiectasis, and occasionally honeycombing.^[13,14] The presence of pleural disease and poorly defined centrilobular nodules in the subpleural regions is helpful in differentiating asbestosis from other causes of pulmonary fibrosis.

Mesothelioma

Unilateral pleural effusion is the most frequent manifestation. A combination of mediastinal pleural involvement and thick (>1 cm), nodular, circumferential pleural thickening is highly suggestive [Figure 10].

Calcosis

Calcosis is caused by inhaling limestone dust. Limestone consists predominantly of calcium carbonate but may also contain magnesium oxide, silica dioxide and aluminium oxide. Pure limestone itself does not cause pneumoconiosis.

Calcosis is caused by inhaling limestone dust. HRCT findings [Figure 11] are not well established. Small nodules have been described in calcosis.^[16,17]

Talcosis

Talc is hydrated magnesium silicate used in the leather, ceramic, paper, plastics, rubber, building, paint, and cosmetic industries.^[18] Talc exposure may occur as a result of inhalation or by intravenous administration,^[1] which occurs most often during recreational drug use.

HRCT findings [Figure 12] include small centrilobular and subpleural nodules and heterogeneous conglomerate masses with internal foci of high attenuation that correspond to talc deposition.^[19]

Berylliosis

Berylliosis is a chronic granulomatous hypersensitivity reaction occurring in both acute and chronic forms. Beryllium exposure occurs in industries such as nuclear power, aerospace, ceramics, metal manufacturing, and dentistry. The acute cases have practically been eliminated through observation of strict workplace protection rules.

HRCT findings of chronic berylliosis [Figure 13] are similar to those of sarcoidosis and include small nodules with peribronchovascular distribution, smooth or nodular interlobular septal thickening, ground-glass opacity, and bronchial wall thickening.^[20] Mediastinal and hilar lymphadenopathy is seen in about 25% of patients.

The histology and radiological appearance of chronic beryllium disease is indistinguishable from sarcoidosis. A diagnosis of chronic beryllium disease requires a lung biopsy proving granulomatous inflammation and evidence of sensitivity to beryllium shown at blood testing or in bronchoalveolar lavage fluid [beryllium lymphocyte proliferation test (BeLPT)]. BeLPT has become a standard tool in the clinical screening of

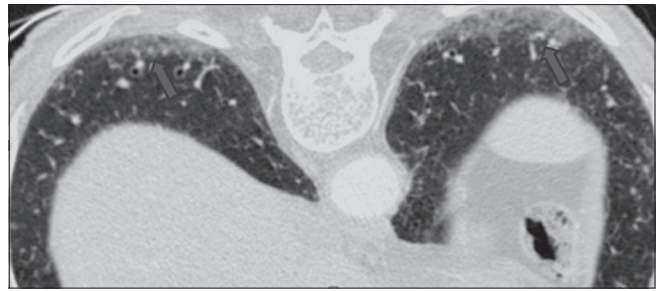


Figure 9: Asbestosis in a 53-year-old man who worked for 30 years in an automobile factory. Axial HRCT scan (C:-600, W: 1600) obtained at the level of the liver dome with the patient prone shows patchy ground-glass opacities and interlobular septal thickening (arrows), suggestive of early-stage asbestosis

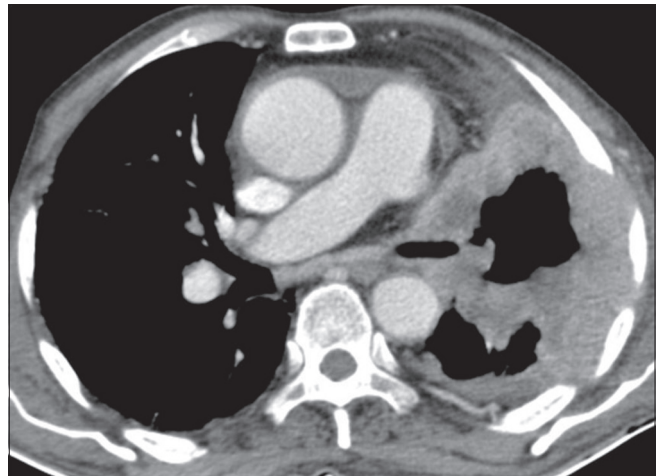


Figure 10: A 69-year-old man with mesothelioma. Contrast-enhanced CT in mediastinal window (C: 50, W: 350) image shows nodular enhancing pleural thickening, involving mediastinal pleura and encasing the left lung

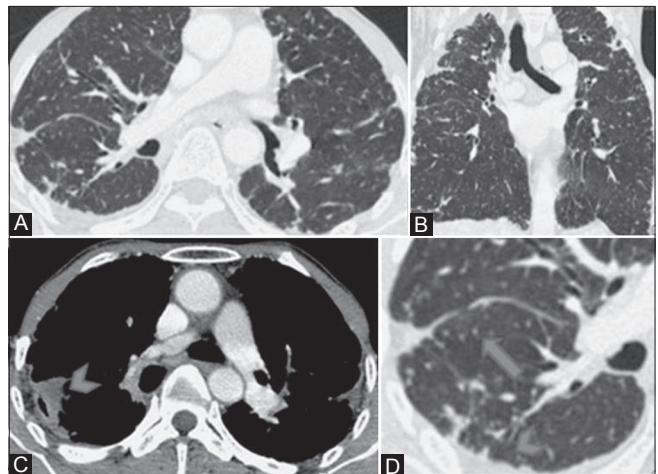


Figure 11 (A-D): Calcosis in a 52-year-old marble worker. (A, B, D) Axial, coronal, and magnified HRCT images (C:-600, W: 1600) show small diffuse nodules (arrow D) and some subpleural compounding pseudoplaques (arrowheads). Mediastinal window (C) (C: 50, W: 350) shows pseudoplaque (arrowhead)

suspected cases, e.g., “sarcoidosis” patients exposed to metals.

Hard Metal Pneumoconiosis

Hard metal pneumoconiosis, formerly classified as giant cell interstitial pneumonia, results from exposure to tungsten carbide, cobalt and diamond dust produced in hard-metal industry. It is a spectrum of diseases

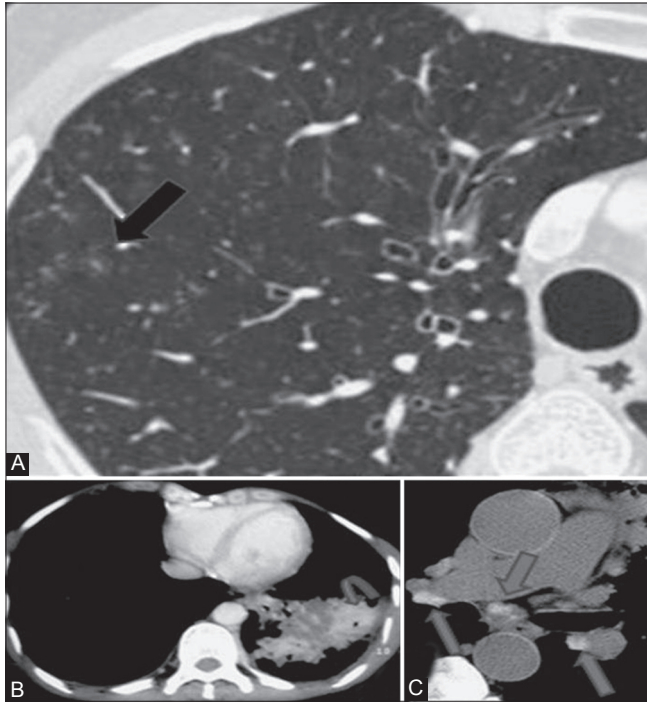


Figure 12 (A-C): Talcosis in a 63-year-old woman working in rubber industry. Axial HRCT images (A) (C:-600, W: 1600) show small centrilobular nodules (black arrows) in upper lobes. In the mediastinal windows (B, C) (C: 50, W: 350), conglomerate mass is seen to contain high-attenuation material (curved arrow); it is also seen in mediastinal lymph nodes (arrows, C)

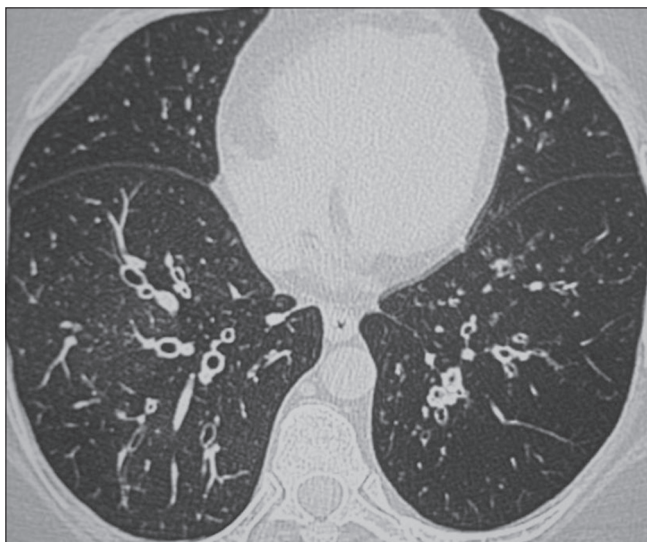


Figure 14: Hard metal (nickel) pneumoconiosis in a 52-year-old lathe machine worker. Axial HRCT image (C:-600, W: 1600) shows minimal bilateral central cylindrical bronchiectasis, fine nodules, and mosaic attenuation, suggestive of obliterative bronchiolitis

comprising occupational asthma and obliterative bronchiolitis [Figure 14], the earliest manifestations, and giant cell interstitial pneumonia and interstitial fibrosis, a late feature.^[21]

HRCT findings [Figure 15] consist of bilateral ground-glass opacities, tiny nodules, reticular opacities, traction bronchiectasis and consolidation. Lower lobe predominance has been described.

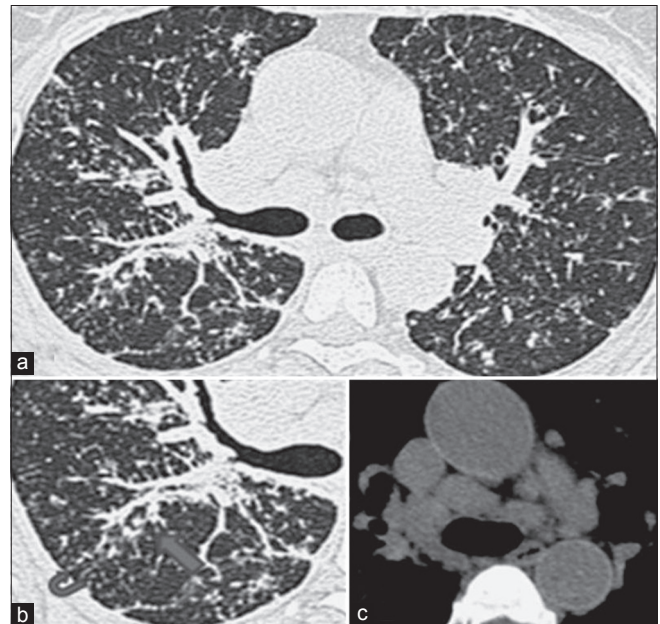


Figure 13 (A-C): A 45-year-old woman working in dental prostheses manufacturing unit, showing chronic berylliosis. (A, B) Axial HRCT images (C:-600, W: 1600) show interlobular septal thickening, small perilymphatic nodules [subpleural (curved arrow), peribronchial (straight arrow)], and a mosaic pattern of attenuation. (C) Mediastinal window (C: 50, W: 350) shows mediastinal and bilateral hilar lymphadenopathy



Figure 15: A 38-year-old machinist with hard metal pneumoconiosis. Axial HRCT image (C:-600, W: 1600) shows numerous poorly defined centrilobular nodules in the upper lobes

Siderosis

The majority cases of siderosis are seen in electric-arc and oxyacetylene welders. Other occupations at risk include mining and processing of iron ores, iron and steel rolling mills, foundry workers and silver polishers.

HRCT [Figure 16] shows widespread ill-defined small centrilobular nodules and, less commonly, patchy areas of ground-glass attenuation without zonal predominance.^[18] Emphysema is often seen. Though siderosis is not usually associated with fibrosis or functional impairment,^[22] symptomatic disease with interstitial fibrosis has been described in arc welders.^[23] Findings of interstitial fibrosis include septal thickening with or without honeycombing [Figure 17].

Aluminum Dust Pneumoconiosis

Exposure to aluminium occurs in production of aluminium, aluminium arc welding, grinding or polishing of aluminium products or in the manufacture of aluminium based abrasive grinding tools.

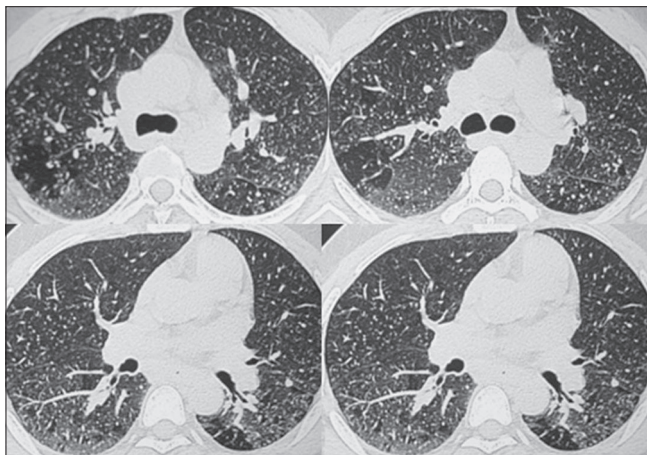


Figure 16: Siderosis in a 39-year-old arc welder who presented with complaint of cough. Axial HRCT images (C:-600, W: 1600) show multiple small and poorly defined centrilobular nodules in the upper lobe of both lungs, with areas of ground-glass attenuation.

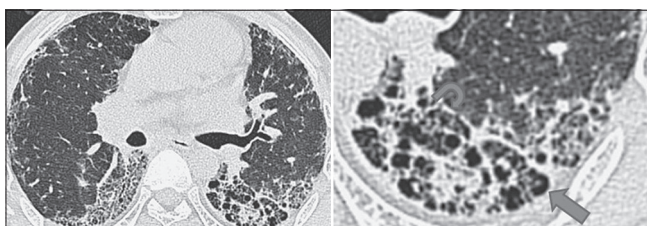


Figure 18: A 62-year-old man with history of exposure to aluminum for 18 years. Axial HRCT image (C:-600, W: 1600) shows intralobular interstitial thickening (curved arrow), ground-glass attenuation, and areas of low attenuation, suggestive of honeycombing (straight arrow). There is diffuse asymmetric involvement of bilateral lungs, with upper lobe predominance

It is associated with pulmonary fibrosis, granuloma formation, desquamative interstitial pneumonia (DIP), and alveolar proteinosis. HRCT findings include subpleural or diffuse honeycombing resembling idiopathic pulmonary fibrosis (IPF), centrilobular nodules resembling silicosis, or irregular reticulation [Figure 18], with upper lobe predominance.^[24,25]

Hypersensitivity Pneumonitis

Hypersensitivity pneumonitis (HP), also known as extrinsic allergic alveolitis, develops as a result of repeated inhalation of antigenic organic and low molecular weight inorganic particles. Common industrial antigens causing HP include isocyanates (paint sprays), plastics (packing plants), *Mycobacterium avium* complex (metal working fluids), *Aspergillus* (agriculture), and thermophilic actinomyces (agriculture).

It is traditionally grouped both clinically and radiologically into acute, subacute, and chronic forms.^[26]

HRCT findings in the acute phase [Figure 19] consist of diffuse ground-glass opacity, reticular opacities, and small poorly defined nodules predominately in the lower lung zones.^[27]

In subacute HP [Figure 20], the HRCT findings include patchy or diffuse ground-glass opacity, small (<5 mm) and poorly defined centrilobular nodules, and patchy lobular air trapping. Sparse thin-walled cysts occur in about 10% of patients and mild mediastinal lymphadenopathy develops

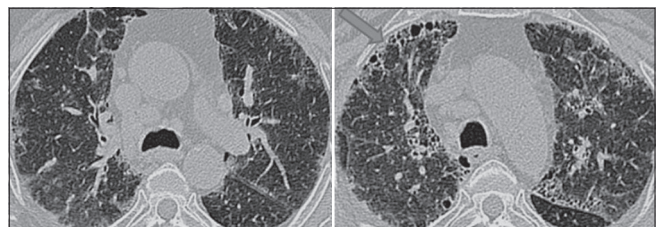


Figure 17: Siderosis with interstitial fibrosis in a lathe machine worker. Axial HRCT images (C:-600, W: 1600) show interlobular and intralobular septal thickening with honeycombing (arrow) in bilateral upper lobes

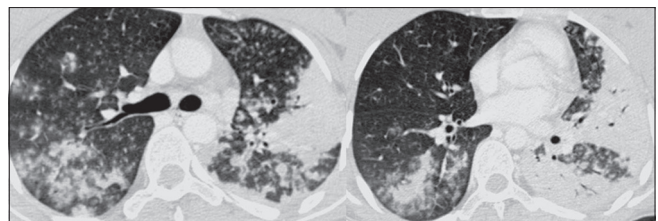


Figure 19: Isocyanate-induced acute hypersensitivity pneumonitis in a 36-year-old man. Axial HRCT scan (C:-600, W: 1600) shows patchy areas of consolidation and ground-glass attenuation in both lungs and less profuse small nodular hyperattenuating areas

in about 50%. The HRCT findings of chronic HP [Figure 21] are extremely variable. The characteristic features include presence of reticulation, architectural distortion, traction bronchiectasis, bronchiolectasis and honeycombing. These findings reflect fibrosis, and show middle and lower lung zone predominance, with relative sparing of the lung bases. There may be superimposition of findings of subacute HP, including ground-glass opacities, ill-defined centrilobular nodules, and mosaic attenuation with air trapping on expiratory scans.

Flavor Worker's Lung

Flavor worker's lung is the development of obliterative bronchiolitis after exposure to diacetyl (2,3-butanedione) used in butter flavoring of microwave popcorn.

HRCT findings include mosaic attenuation with air trapping on expiratory imaging [Figure 22].^[28] Bronchial wall thickening and bronchiectasis may also be present.

Chemical Pneumonitis

The inhalation of noxious chemical substances, though not common, is a significant cause of occupational lung disease. These chemicals include organic (organophosphates,

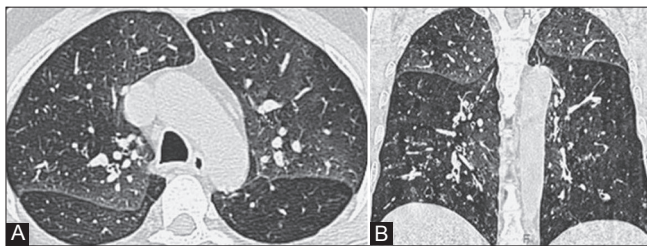


Figure 20 (A, B): A 52-year-old farmer with subacute hypersensitivity pneumonitis. (A, B) Transverse and coronal HRCT images (C:-600, W: 1600) show patchy ground-glass opacity and lobular hyperlucency, suggestive of air trapping

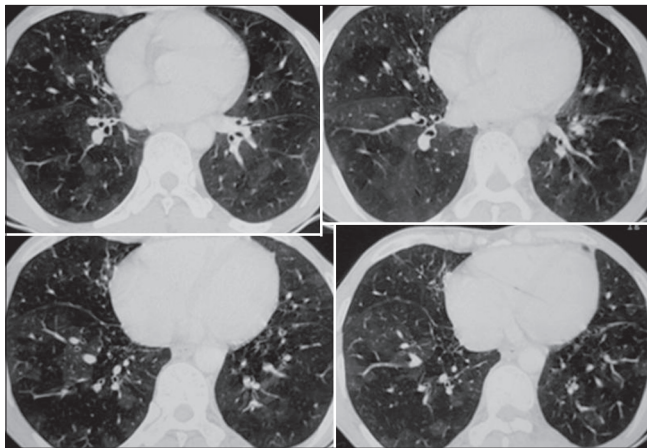


Figure 22: Popcorn plant worker with obliterative bronchiolitis. Axial HRCT images (C:-600, W: 1600) show mosaic attenuation with areas of hypoattenuation, suggestive of air trapping

paraquat, polyvinyl chloride, polymer fumes, smoke), inorganic (ammonia, hydrogen sulfide, nitrogen oxide, sulphur dioxide), and metal (cadmium, mercury, nickel, vanadium).

HRCT in acute exposure [Figure 23] may show centrilobular or patchy areas of ground-glass opacity, presumably due to pulmonary edema. Bronchiolitis obliterans [Figure 24] may develop weeks to months after the exposure, with findings of bronchiectasis, bronchiolectasis, mosaic perfusion, and air trapping.^[29]

Organic Dust Toxic Syndrome

It refers to a febrile illness following exposure to organic dust without evidence of HP. It comprises humidifier fever (office

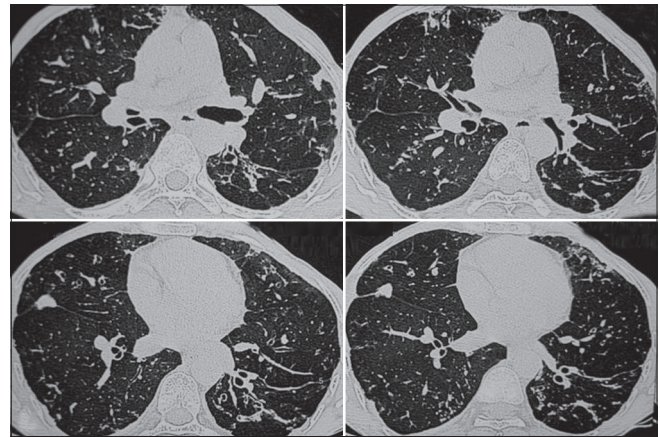


Figure 21: Chronic hypersensitivity pneumonitis in a 55-year-old plastic industry worker. Axial HRCT images (C:-600, W: 1600) show findings of fibrosis predominantly in the upper lobes with traction bronchiectasis (arrow)

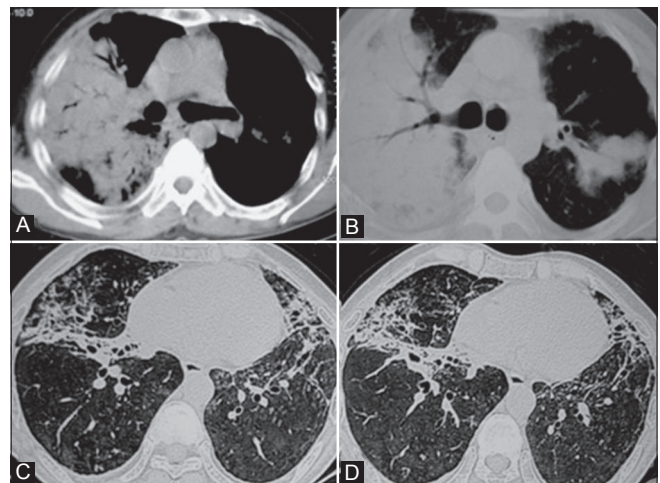


Figure 23 (A-D): Paraquat poisoning in a 19-year-old man. Axial HRCT at day 2 of admission (A, B) (C:-600, W: 1600) shows areas of bilateral consolidation, suggestive of pulmonary edema. (C, D) Axial HRCT obtained after 8 weeks shows reticular opacities and traction bronchiectasis corresponding to the affected areas in A and B, indicating progression to interstitial fibrosis

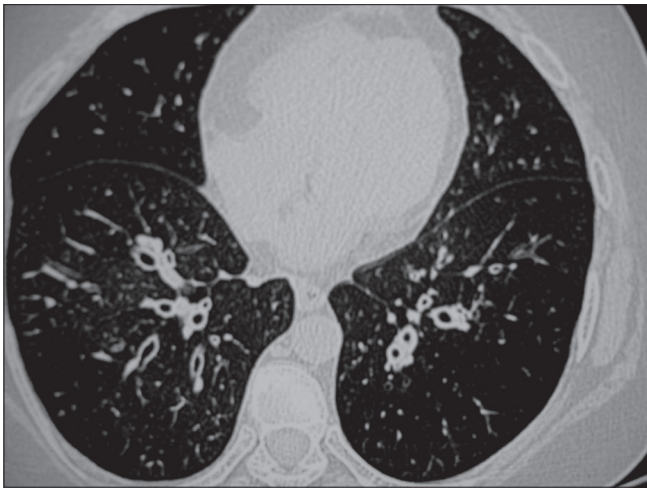


Figure 24: Bronchiolitis obliterans due to chlorine inhalation from bleaching agent in a 56-year-old woman laundry worker. Axial HRCT scan (C:-600, W: 1600) shows bronchiectasis and areas of hypoattenuation throughout both lungs

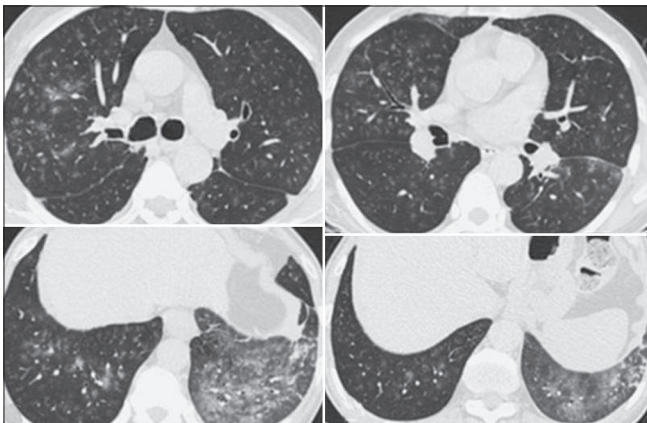


Figure 25: Byssinosis in a 56-year-old woman working in a cotton mill. Axial HRCT scan(C:-600, W: 1600) shows numerous ill-defined small nodules with ground-glass attenuation in both lungs with basal predominance

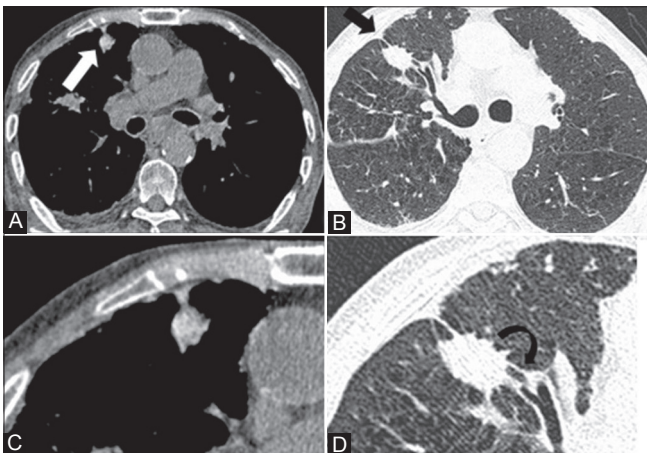


Figure 26 (A-D): A 35-year-old man with asbestos-related pleural disease and rounded atelectasis. CT image in mediastinal (A) (C:50, W: 350) and lung (B) windows (C:-600, W: 1600) shows a round, subpleural focus of consolidation (arrow). This is intimately related to the adjacent pleural plaque and thickening with a curving tail of bronchovascular structures spiraling into the mass, "comet tail sign" (curved arrow)

and hospital workers), pulmonary mycotoxicosis, grain fever, pig fever, cotton fever (byssinosis), and wood-chip fever.

The imaging findings [Figures 25 and 26] of byssinosis in cotton workers have been sparsely described; however, basal predominant ground-glass opacities with associated centrilobular nodules have been reported on HRCT.^[30]

Conclusion

HRCT has assumed an increasingly important role in evaluation of patients with diffuse lung disease including occupational lung disease. It is indicated in symptomatic patients or patients with abnormal pulmonary function findings, with a normal or questionable chest radiograph. Even when the chest radiograph is abnormal, HRCT is useful to make a specific diagnosis or limit the differential diagnosis. It plays a critical role in assessing disease activity. The presence of ground glass opacity and nodules suggest active disease which may be reversible on cessation of exposure, whereas presence of fibrosis is a marker of disease irreversibility. It also plays an important guide in determining need, optimal site and type of lung biopsy.

Occupational lung disease is a diverse group of preventable pulmonary diseases. The characteristic radiological features suggest the correct diagnosis in some, whereas a combination of clinical features, occupational history, and radiological findings is essential in establishing the diagnosis in others. In the presence of a history of exposure and consistent clinical features, the diagnosis of even an uncommon occupational lung disease can be suggested by characteristic HRCT findings.

References

1. Dee P, Suratt P, Winn W. The radiographic findings in acute silicosis. *Radiology* 1978;126:359-63.
2. Marchiori E, Ferreira A, Müller NL. Silicoproteinosis: High-resolution CT and histologic findings. *J Thorac Imaging* 2001;16:127-9.
3. Oikonomou A, Muller NL. Imaging of pneumoconiosis. *Imaging* 2003;15:11-22.
4. Antao VC, Pinheiro GA, Terra-Filho M, Kavakama J, Müller NL. High-resolution CT in silicosis: Correlation with radiographic findings and functional impairment. *J Comput Assist Tomogr* 2005;29:350-6.
5. Chong S, Lee KS, Chung MJ, Han J, Kwon OJ, Kim TS. Pneumoconiosis: Comparison of imaging and pathologic findings. *Radiographics* 2006;26:59-77.
6. Begin R, Bergeron D, Samson L, Boctor M, Cantin A. CT assessment of silicosis in exposed workers. *AJR Am J Roentgenol* 1987;148:509-14.
7. Begin R, Ostiguy G, Fillion R, Colman N. Computed tomography scan in the early detection of silicosis. *Am Rev Respir Dis* 1991;144:697-705.
8. Young RC Jr, Rachal RE, Carr PG, Press HC. Patterns of coal workers' pneumoconiosis in Appalachian former coal miners. *J Natl Med Assoc* 1992;84:41-8.

9. Remy-Jardin M, Degreef JM, Beuscart R, Voisin C, Remy J. Coal worker's pneumoconiosis: CT assessment in exposed workers and correlation with radiographic findings. *Radiology* 1990;177:363-71.
10. Williams JL, Moller GA. Solitary mass in the lungs of coal miners. *Am J Roentgenol Radium Ther Nucl Med* 1973;117:765-70.
11. Katabami M, Dosaka-Akita H, Honma K, Saitoh Y, Kimura K, Uchida Y, *et al.* Pneumoconiosis-related lung cancers: Preferential occurrence from diffuse interstitial fibrosis-type pneumoconiosis. *Am J Respir Crit Care Med* 2000;162:295-300.
12. Roach HD, Davies GJ, Attanoos R, Crane M, Adams H, Phillips S. Asbestos: When the dust settles an imaging review of asbestos-related disease. *Radiographics* 2002;22 Spec No: S167-84.
13. McLoud TC, Woods BO, Carrington CB, Epler GR, Gaensler EA. Diffuse pleural thickening in an asbestos-exposed population: Prevalence and causes. *AJR Am J Roentgenol* 1985;144:9-18.
14. Akira M. High-resolution CT in the evaluation of occupational and environmental disease. *Radiol Clin North Am.* 2002;40:43-59.
15. Lynch DA, Gamsu G, Ray CS, Aberle DR. Asbestos-related focal lung masses: Manifestations on conventional and high-resolution CT scans. *Radiology* 1988;169:603-7.
16. Crummy F, Carl I, Cameron CH, Heaney LG. A possible case of pneumoconiosis in a limestone quarry worker. *Occup Med (Lond)* 2004;54:497-9.
17. Doig AT. Disabling pneumoconiosis from limestone dust. *Br J Ind Med* 1955;12:206-16.
18. Kim KI, Kim CW, Lee MK, Lee KS, Park CK, Choi SJ, *et al.* Imaging of occupational lung disease. *Radiographics* 2001;21:1371-91.
19. Marchiori E, Souza Júnior AS, Müller NL. Inhalation pulmonary talcosis: High-resolution CT findings in 3 patients. *J Thorac Imaging* 2004;19:41-4.
20. Newman LS, Buschman DL, Newell JD Jr, Lynch DA. Beryllium disease: Assessment with CT. *Radiology* 1994;190:835-40.
21. Kelleher P, Pacheco K, Newman LS. Inorganic dust pneumonias: The metal-related parenchymal disorders. *Environ Health Perspect* 2000;108(Suppl 4):685-96.
22. Han D, Goo JM, Im JG, Lee KS, Paek DM, Park SH. Findings of arcwelders' pneumoconiosis. *Korean J Radiol* 2000;1:79-83.
23. McCormick LM, Goddard M, Mahadeva R. Pulmonary fibrosis secondary to siderosis causing symptomatic respiratory disease: A case report. *J Med Case Rep* 2008;2:257.
24. Kraus T, Schaller KH, Angerer J, Hilgers RD, Letzel S. Aluminosis-detection of an almost forgotten disease with HRCT. *J Occup Med Toxicol* 2006;1:4.
25. Akira M. Uncommon pneumoconioses: CT and pathologic findings. *Radiology* 1995;197:403-9.
26. Silva CI, Churg A, Muller NL. Hypersensitivity pneumonitis: Spectrum of high-resolution CT and pathologic findings. *AJR Am J Roentgenol* 2007;188:334-44.
27. Silver SF, Müller NL, Miller RR, Lefcoe MS. Hypersensitivity pneumonitis: Evaluation with CT. *Radiology* 1989;173:441-5.
28. Newman LS, Buschman DL, Newell JD Jr, Lynch DA. Beryllium disease: Assessment with CT. *Radiology* 1994;190:835-40.
29. Wright JL. Inhalational lung injury causing bronchiolitis. *Clin Chest Med* 1993;14:635-44.
30. Kim KI, Kim CW, Lee MK, Lee KS, Park CK, Choi SJ, *et al.* Imaging of Occupational Lung Disease. *Radiographics* 2001;21:1371-91.

Cite this article as: Satija B, Kumar S, Ojha UC, Gothi D. Spectrum of high-resolution computed tomography imaging in occupational lung disease. *Indian J Radiol Imaging* 2013;23:287-96.

Source of Support: Nil, **Conflict of Interest:** None declared.