

Original research article

High resolution C.T. in occupational lung diseases-a prospective study

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ABSTRACT:

Introduction: Occupational lung diseases represent the most frequently diagnosed work-related conditions. Although it can be detected by chest radiography, high-resolution computed tomography (CT) has been shown to be superior to chest radiography in depicting parenchymal, airway, and pleural abnormalities.

Aim: To study the HRCT features of occupational lung diseases in industrial workers.

Materials and Methods: This prospective study was conducted among 217 patients attended medicine and respiratory medicine out patient departments in one tertiary care medical college hospitals in Tamilnadu and Kerala between Jan 2019 to June 2019 were taken for study .

Results: Prevalence of occupational lung diseases was higher among males compared to females. Prevalence was significantly higher among quarry workers. then road workers, concrete workers, waste cotton mill and cotton mill , reeling ,weaving mill, ginning mill workers.

Conclusion: HRCT is very useful in detecting occupational lung diseases Some occupational lung diseases have characteristic radiologic features suggesting the correct diagnosis. In other such diseases, definite diagnosis cannot be made on the basis of imaging features alone, but a combination of clinical features with related occupational history and radiologic findings can significantly improve diagnostic accuracy

Keywords: occupational lung diseases, high resolution C.T.,HRCT

INTRODUCTION

Occupational lung disease comprises a wide variety of disorders caused by the inhalation or ingestion of dust particles or noxious chemicals. These disorders include pneumoconiosis, asbestos-related pleural and parenchymal disease, chemical pneumonitis, occupational infection, hypersensitivity pneumonitis, and organic dust toxic syndrome. Most of these disorders produce diffuse lung disease. Some occupational lung diseases have characteristic radiologic features suggesting the correct diagnosis, whereas in others, a combination of clinical features, related occupational history, radiologic findings, and literature supporting an association between the exposure and the disease process is required for diagnosis. With advances in chest radiology, including high-resolution CT, radiologists play a key role in the clinical evaluation of occupational lung diseases. Thousands of environmental toxins and commercial chemicals are in use today. These agents may become aerosolized or airborne in the form of fibers, fumes, mists, or dust. Next to injuries, occupational lung

disease represents the most frequently diagnosed work-related condition Perhaps the greatest increase in pulmonary hazards over this period has been in occupational allergic disorders, asthma, and hypersensitivity pneumonitis.

Individuals living in major metropolitan areas may inhale more than 2 mg of dust each day, and workers in dusty occupations may inhale up to 100 times that amount. The development of occupational lung disease in an individual worker is dependent on the toxic effects of the inhaled substance, the intensity and duration of the exposure, and the physiologic and biologic susceptibility of the host. The physical state of the inhaled substance (eg, solid, fume, or mixture), its solubility, and its aerodynamic dimensions determine the initial location of disease activity. Depending on the solubility and reactivity of the inhaled substance, acute or chronic reactions occur as particles are deposited in the lower respiratory tract. Acute reactions with associated inflammation and edema, or more chronic reactions characterized by fibrosis or granuloma formation, have been demonstrated following inhalation of many environmental agents

This is a tissue reaction to the presence of an accumulation of dust in the lungs. One clinic pathologic form of this reaction is fibrosis, which can be focal and nodular (as in silicosis) or diffuse (as in asbestosis). It often results in radiographic abnormalities and, if extensive enough, may lead to significant functional impairment. The other form consists of aggregates of particle-laden macrophages with minimal or no accompanying fibrosis, a reaction that is typically seen with inert dusts such as iron, tin, and barium.

MATERIALS AND METHODS:

This prospective study was conducted among 217 patients attended medicine and respiratory medicine out patient departments in one tertiary care medical college hospitals in Tamilnadu and Kerala between Jan 2019 to June 2019 were taken for study .

RESULTS:

TOTAL 217 PATIENTS. MALE 116 FEMALE 101

Characteristics	Category	Frequency%
Age (years)	<20	19(9%)
	20--39	89(41%)
	40--59	83(38%)
	≥ 60	26(12%)
DURATION OF EXPOSURE	Below 5 yrs	30(14%)
	5-15 yrs	143 (66%)
	Above 15 hrs	44(20%)
Using personal protective equipments/mask	Yes	48(22%)
	NO	169 (78%)
Body Mass Index (kg/m²)	19-25	138(64%)
	>25	79(36%)
Duration of SIGNS and symptoms	Less than 1 yrs	30 (14%)
	1-3yrs	78(36%)
	3-5yrs	50(23%)

Characteristics	Category	Frequency%
	More than 5yrs	59 (27%)
Daily Exercise, Walking,outdoor games,	Yes	46(21%)
	No	171(79 %)
Family history of lung disease	NO	172(79%)
	YES	45(21%)
Family Income in rupees per month	≤ 15,000	106(49%)
	>15,000	111(51%)
OCCUPATION	SAND MINING	30(14%)
	ROAD	63(29%)
	COTTON MILL	69(32%)
SMOKING	WELDING	20(9%)
	CONCRETE	35(16%)
	YES	89 (41%)
	NO	128 (59%)
XRAY FINDINGS ALONE	46(21%)	
X RAY AND HR CT FINDINGS	93(43%)	
X RAY ,HRCT AND MRI FINDINGS	78(36%)	

The principal sources of industrial exposure to silica are free silica in mining, quarrying, and tunneling; stonecutting, polishing, and cleaning monumental masonry; sandblasting and glass manufacturing; and, in foundry work, pottery and porcelain manufacturing, brick lining, boiler scaling, and vitreous enameling. Coal miners are exposed to dusts that contain a mixture of coal, mica, kaolin, and silica in varying proportions. Silicosis and coal worker pneumoconiosis (CWP) are distinct diseases, with differing histologic features resulting from the inhalation of different inorganic dusts. However, the radiographic and high-resolution CT appearances of silicosis and CWP are quite similar, so that the two disease entities cannot be easily or reliably distinguished in individual cases.

The characteristic radiologic abnormality seen in patients with simple silicosis or CWP consists of small, well-circumscribed nodules that are usually 2–5 mm in diameter but range from 1 to 10 mm, mainly involving the upper and posterior lung zones. Although there is a tendency for the nodules in silicosis to be better defined

than those in CWP, this is not always the case. These small nodules indicate the presence of simple or uncomplicated silicosis or CWP. The appearance of large opacities or hyperattenuating areas over 1 cm in diameter (progressive massive fibrosis) indicates the presence of complicated silicosis or complicated CWP. These masses tend to develop in the mid zone or periphery of the upper lung and migrate toward the hila, leaving overinflated emphysematous spaces between the conglomerate mass and the pleura. They are often bilateral, symmetric, and calcified and can demonstrate cavitation. Egg-shell calcifications in hilar and mediastinal lymph nodes are occasionally seen.

Acute silicosis, also known as silico-proteinosis, is a rare condition related to heavy exposure to respirable free silica in enclosed spaces in which there is minimal or no protection from the silica. Exposure times are frequently as short as 6–8 months. The disease is often rapidly progressive, with death caused by respiratory failure. Proliferation of type II pneumocytes and profuse surfactant production characterize the process histologically. The radiologic and pathologic appearances of acute silicosis are quite different from those of classic silicosis and are similar to those of pulmonary alveolar proteinosis. Chest radiographs demonstrate a pattern of diffuse airspace or ground-glass disease in a perihilar distribution with air bronchograms. Tuberculosis and infection with atypical mycobacteria are frequent complications.

Siderosis The majority of cases of siderosis are seen in electric arc or oxyacetylene torch workers, who are exposed to iron oxide in fumes during the welding process. The radiographic pattern in pure siderosis consists of diffuse fine reticulonodular opacities. Nodular opacities are less dense and less profuse than those in silicosis. In contrast to the majority of cases of pneumoconiosis, the radiographic abnormalities can partially or completely disappear when patients are removed from dust exposure. High-resolution CT findings, which have been described in arc welder pneumoconiosis, include widespread, poorly defined centrilobular micronodules and branching linear structures or extensive ground-glass attenuation without zonal predominance and fibrosis.

Hard Metal Pneumoconiosis Radiographic findings consist of a diffuse micronodular and reticular pattern, sometimes associated with lymph node enlargement. The reticulation may be coarse and in advanced disease may be accompanied by small cystic spaces. High-resolution CT findings consist of bilateral areas of ground-glass attenuation, areas of consolidation, and extensive reticular hyperattenuating areas and traction bronchiectasis, findings that are indicative of fibrosis.

Asbestos-related Pleural and Parenchymal Disease: There are two major sources of exposure to asbestos dust: (a) the primary occupations of asbestos mining and processing, and (b) secondary occupations such as insulation manufacturing, textile manufacturing, construction, shipbuilding, and the manufacture and repair of gaskets and brake linings. The inhaled asbestos fiber is long (up to 100 µm in length), penetrates deeply into the lung and pleura, and has a fibrogenic effect on respiratory bronchioles, alveoli, and pleura. Clinical manifestations typically do not appear until 20 years after initial exposure. Asbestos-related diseases include benign pleural diseases (plaques, diffuse pleural thickening, effusion, calcification), parenchymal diseases (asbestosis [parenchymal fibrosis caused by asbestos inhalation], rounded atelectasis, benign fibrotic masses, transpulmonary bands), and malignancy (malignant mesothelioma, bronchogenic carcinoma).

Asbestos-related Benign Pleural Disease: Pleural plaques are the most common manifestation of asbestos exposure. They serve as a marker for exposure and are not usually associated with symptoms or functional impairment. Generally, plaques cannot be detected at standard chest radiography until at least 20 years after initial exposure. Plaques that arise from the visceral pleura are very rare and are usually found in the lower aspects of the

interlobar fissures; they may calcify and are usually associated with extensive parietal pleura plaque formation. Asbestosis rarely occurs in the absence of pleural plaques .

Radiologic changes consist of small, irregular opacities or hyperattenuating areas in a linear pattern. The fine reticulation eventually progresses to a coarse linear pattern with honeycombing. These abnormalities are usually most severe in the lower lungs, the posterior lungs, and in a subpleural location. These findings are similar to those in idiopathic pulmonary fibrosis. The presence of pleural plaques lends support to the radiologic diagnosis of asbestosis. However, plaques are not invariably present in patients with asbestosis. High-resolution CT including prone scans is a sensitive, reliable means of detecting thoracic abnormalities in individuals exposed to asbestos. Prone scans allow basal structural abnormalities to be reliably distinguished from gravity-related physiologic phenomena .

Major CT findings in early asbestosis include thickened intralobular lines that have been shown at histologic analysis to be due to peribronchiolar fibrosis, thickened interlobular lines, subpleural curvilinear lines, pleura-based nodular irregularities, patchy areas of ground-glass attenuation, small cystic spaces, and small areas of hypoattenuation. Thickened interlobular lines have been shown to be due mainly to interlobular fibrotic or edematous thickening. Areas of ground-glass attenuation are the result of alveolar wall thickening due to fibrosis or edema

Rounded AtelectasisThe most common of the benign masses caused by asbestosis exposure is rounded atelectasis, a form of peripheral lobar collapse that develops in patients with pleural disease. It usually occurs in the subpleural, posterior, or basal region of the lower lobes

Bronchogenic Carcinoma Bronchogenic carcinoma is estimated to develop in 20%–25% of workers who are heavily exposed to asbestos. The risk of bronchogenic carcinoma in asbestos workers who smoke may be as much as 80–100 times that in the nonsmoking, nonexposed population. Asbestos-related tumors frequently occur in the periphery of the lungs.

Malignant MesotheliomaDiffuse malignant mesothelioma is an uncommon and fatal neoplasm of the serosal lining of the pleural cavity, peritoneum, or both. The risk of mesothelioma in an asbestos worker is approximately 10% over his or her lifetime. At CT, the combination of mediastinal pleural involvement and thick (>1 cm), nodular, circumferential pleural thickening is highly suggestive of diffuse mesothelioma rather than benign pleural disease. Mesothelioma may also manifest as a single, discrete pleural mass. The tumor may extend into the interlobar fissures and interlobular septa, with superficial invasion of the underlying lung. Findings in patients with advanced tumor consist of invasion of the chest wall, pericardium, diaphragm, and abdomen

Chemical PneumonitisThe inhalation of noxious chemical substances is a comparatively uncommon but significant cause of occupational lung disease. The mechanism of pulmonary toxicity caused by different agents varies considerably. Noxious chemicals include organic (eg, organophosphates, paraquat, polyvinyl chloride, polymer fumes, smoke), nonorganic (eg, ammonia, hydrogen sulfide, nitrogen oxide, sulfur dioxide), and metal (eg, cadmium, mercury, nickel, vanadium) compounds.

Occupational InfectionOccupation-related infections may develop in pet shop owners, butchers, farmers, tannery workers, archaeologists, or health care workers. Bacterial infections include anthrax, brucellosis, or tularemia. Fungal infections include blastomycosis, coccidioidomycosis, and histoplasmosis. In addition, mycobacterial infections may occur in health care workers. Leptospirosis is a disease produced by any of the

group of spirochetes of the *Leptospira* genus. It occurs in agricultural workers in endemic areas. The main sites of involvement are the liver, central nervous system, kidneys, skeletal muscle, and lungs. Radiographic findings include small nodular opacities, confluent areas of consolidation, and diffuse bilateral ground-glass areas of increased opacity. These findings are compatible with the pathologic features of petechial and multifocal pulmonary hemorrhage. Pulmonary abnormalities resolve within 2 weeks in most patients

Hypersensitivity Pneumonitis : High-resolution CT findings in acute hypersensitivity pneumonitis typically consist of diffuse airspace consolidation, whereas those in subacute hypersensitivity pneumonitis consist of patchy or diffuse areas of ground-glass attenuation and small, centri lobular nodular areas of hyper attenuation. Chronic hypersensitivity pneumonitis is characterized by the presence of fibrosis, although findings of active disease are often present. Findings in fibrosis include intra lobular interstitial thickening, irregular interfaces, irregular interlobular septal thickening, honeycombing, and traction bronchiectasis. The fibrosis can be diffuse but usually involves mainly the middle or lower lung zones. Relative sparing of the lung bases usually allows distinction of this entity from idiopathic pulmonary fibrosis, in which the fibrosis usually predominates in the lung bases



Isocyanates are used for the large-scale production of polyurethane polymers, which have an almost endless variety of applications in the manufacture of flexible and rigid foams, elastomers, adhesives, and surface coatings. Acute or chronic exposure to high concentrations of isocyanates can result in respiratory health hazards through a direct irritant effect. In some exposed workers, isocyanates can cause occupational asthma or, less often, hypersensitivity pneumonitis through an apparently sensitizing mechanism. Because of their wide industrial use, isocyanates are the principal cause of occupational asthma, which is now the most common respiratory disease linked to the work environment. Although isocyanates are not organic dusts, the hypersensitivity pneumonitis they cause is clinically, radiologically, and pathologically identical to organic dust-related hypersensitivity pneumonitis.

Organic Dust Toxic Syndrome: Organic dust toxic syndrome can be defined as a febrile illness following exposure to organic dust in individuals who do not have evidence of hypersensitivity pneumonitis. However, some investigators believe that the two abnormalities represent a spectrum of findings in the same disease rather than two separate entities. Typically, fever and influenza-like symptoms occur 4–8 hours after dust exposure; symptoms of dry cough, chest tightness, mild dyspnea, and wheezing may also be present. Organic dust toxic syndrome is thought to be much more common than farmer lung. It has been estimated that the disorder may be 50 times more common than hypersensitivity pneumonitis in farmers. Organic dust toxic syndrome encompasses diseases previously described by a variety of terms, including *humidifier fever* (in office and hospital workers), *pulmonary mycotoxicosis*, *grain fever*, *pig fever*, *cotton fever* (byssinosis), and *wood-chip*

fever. The common pathogenetic link between these disorders may be the presence of a bacterial endotoxin in the dust or aerosol .



Byssinosis occurs in textile workers exposed to the dust of cotton, flax, hemp, and jute. In its early stages, byssinosis is characterized by acute dyspnea, cough, and wheeze on Monday morning following a weekend away from the workplace; symptoms decrease during the work week, despite continued exposure. The prevalence and severity of symptoms and functional impairment are proportional to the duration and intensity of exposure. Although the pathogenesis of byssinosis is unclear, it may be related to the presence of a bacterial endotoxin rather than the textile itself. The disorder shares some features with hypersensitivity pneumonitis: Both frequently occur after exposure to dust, and affected patients improve rapidly without therapy .

To our knowledge, information on radiologic findings in byssinosis is not currently available in the radiology literature. In a survey performed by Lu et al ,chest radiographs revealed small opacities with profusion greater than 1/0 according to the International Labor Organization classification scheme in 8.7% of 140 cotton workers and 4.3% of nonsmoking control subjects, although the additive effect of smoking on radiographic findings in the workers could not be ignored.

CONCLUSIONS:

Some occupational lung diseases have characteristic radiologic features suggesting the correct diagnosis. In other such diseases, definite diagnosis cannot be made on the basis of imaging features alone, but a combination of clinical features with related occupational history and radiologic findings can significantly improve diagnostic accuracy. Radiologists should continue their involvement in the evaluation of occupational lung diseases : The study shows higher prevalence of occupational lung diseases compared to other parts of India possibly attributable to rapid industrialization, cotton and waste cotton mills, dust, smoke, air pollution and climate of Coimbatore and Kerala Awareness and Preventive interventions need to be taken to reduce disease burden at community level

REFERENCES

- 1.Kun-II Kim, Chang Won Kim, Min Ki Lee, Kyung Soo Lee, Choong-Ki Park, Seok Jin Choi, Jong Gi Kim Imaging of Occupational Lung Disease
- 2 .Schwartz DA, Peterson MW. *Occupational lung disease. Adv Intern Med* 1997; 42:269-312.
- 3 .Cullen M, Cherniack M, Rosenstock L. *Medical progress: occupational medicine. N Engl J Med* 1990; 322:594-601
- 4 Seaton A. *The clinical approach*. In: Morgan WKC, Seaton A, eds. **Occupational lung diseases**. 2nd ed. Philadelphia, Pa: Saunders, 1984; 9-17.

- 5 .Fraser RS, Muller NL, Colman N, Pare PD. **Diagnosis of diseases of the chest** 4th ed. Philadelphia, Pa: Saunders, 1999
6. Stark P, Jacobson F, Shaffer K. *Standard imaging in silicosis and coal worker's pneumoconiosis*. **Radiol Clin North Am** 1992; 30:1147-1154. Medline, Google Scholar
- 7 .Webb WR, Muller NL, Naidich DP. **High-resolution CT of the lung** 2nd ed. Philadelphia, Pa: Lippincott-Raven, 1996; 149-191. Google Scholar
- 8 .Remy-Jardin M, Remy J, Farre I, Marquette CH. *Computed tomographic evaluation of silicosis and coal worker's pneumoconiosis*. **Radiol Clin North Am** 1992; 30:1155-1176. Medline, Google Scholar
- 9 .Goodman GB, Kaplan PD, Stachura I, et al. *Acute silicosis responding to corticosteroid therapy*. **Chest** 1992; 101:366-370. Crossref, Medline, Google Scholar
- 10 .Dumontet C, Biron F, Vitrey D, et al. *Acute silicosis due to inhalation of a domestic product*. **Am Rev Respir Dis** 1991; 143:880-882. Crossref, Medline, Google Scholar
- 11 .Dee P, Suratt P, Winn W. *The radiographic findings in acute silicosis*. **Radiology** 1978; 126:359-363. Link, Google Scholar
- 12 .Kim K-I, Choi SJ, Sohn HS, et al. *High-resolution CT findings of welders' pneumoconiosis*. **J Korean Radiol Soc** 1996; 34:367-371. Google Scholar
- 12 .Han D, Goo JM, Im J-G, Lee KS, Paek DM, Park SH. *Thin-section CT findings of arc-welders' pneumoconiosis*. **Korean J Radiol** 2000; 1:79-83. Crossref, Medline, Google Scholar
- 13 .Akira M. *Uncommon pneumoconioses: CT and pathologic findings*. **Radiology** 1995; 197:403-409. Link, Google Scholar
- 14 .Gardiner K, Trethowan NW, Harrington JM, Rossiter CE, Calvert IA. *Respiratory health effects of carbon black: a survey of European carbon black workers*. **Br J Ind Med** 1993; 50:1082-1096. Medline, Google Scholar