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Radiological Manifestation of Progressive Massive Fibrosis as a Complication of Silicosis-Case Report

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ABSTRACT

Progressive massive fibrosis (PMF) is the outcome of complicated silicosis and falls in the category of occupational lung diseases. The underlying etiological factors responsible for this are fine particles of silica, inhaled by workers in certain specific occupation. We present a 42-year-old male patient with chief complaint of breathlessness and had occupational background in relation to sandblasting. HRCT chest had shown confluent fibrotic densities in bilateral upper lobes with loco regional bronchietatic changes and adjacent pleural thickening. The patient was diagnosed of having PMF on the history and classical HRCT findings.

Keywords: *Humans; Occupations; Silicon Dioxide; Silicosis*

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.[1] Pneumoconiosis may be clinico-pathologically classified as fibrotic or non-fibrotic. 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.[2] Imaging plays an indispensable role in the evaluation of occupational lung disease. The radiograph of chest and HRCT

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chest are the 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.[3]

CASE REPORT

A 42-year-old male patient presented with chief complaint of breathlessness for the past 2-3 years which got aggravates for 10 days. No past history of tuberculosis, diabetes mellitus or hypertension noted. Patient had an occupational history of sandblaster for the past 15 years. On general physical examination no significant abnormality seen except tachypnea of 32/min. Oxygen saturation was 92% in room air. The baseline laboratory investigations were within normal limits. The patient was referred to radiology department for further radiological workup. X-ray chest posteroanterior view was done which revealed multiple calcific nodules in bilateral lung fields predominantly in upper and mid zones associated with adjacent fibroreticular opacities (Figure1). There were few rounds to oval shaped calcified opacities seen in bilateral hilar region suggestive of calcified lymph nodes. There were also few inhomogeneous plaque-like opacities present in bilateral upper, mid zone and in perihilar locations.

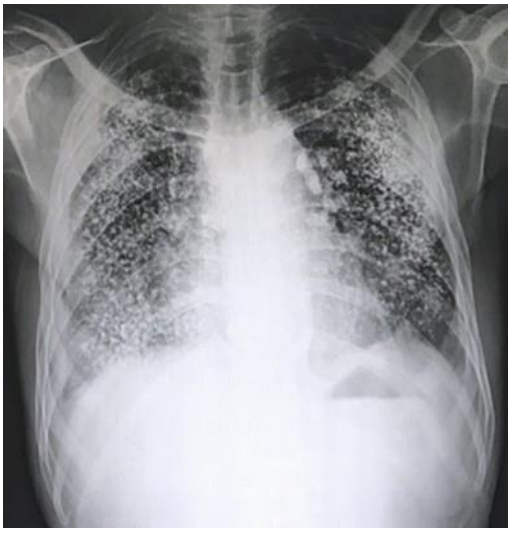
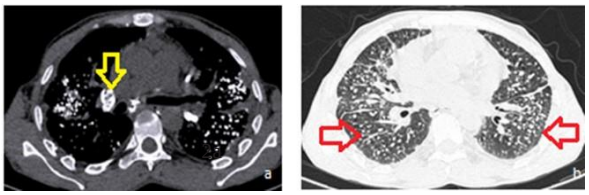


Figure 1. *Chest X-ray postero-anterior (PA) view shows multiple bilateral calcified opacities in upper and mid zones with few oval shaped radio-opacities in bilateral hilar region.*

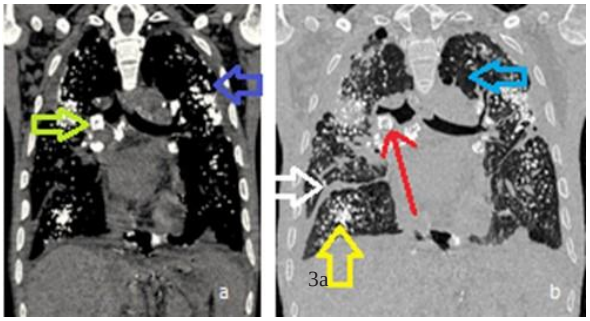
The patient was further advised for HRCT chest which had revealed multiple calcified random nodules in bilateral lung fields with loco-regional and sub-pleural fibro-atelectatic changes. Confluent fibrotic densities were seen in bilateral upper lobes in perihilar location with loco-regional brochiectatic changes and adjacent pleural thickening (Figures 2a and 2b).



3b

Figure 2*. HRCT chest.* ***2a.*** *Axial section in mediastinal window shows multiple tiny calcified nodules in bilateral upper lung fields. Multiple calcified mediastinal and hilar lymph nodes are also seen (yellow arrow) .Loco-regional and sub-pleural fibro atelectatic changes are seen.* ***2b.*** *axial section in lung window shows multiple bilateral small focal lung parenchymal densities (red arrows) with atelectatic changes.*

Multiple egg shell calcifications were seen in bilateral hilar and mediastinal regions (Figures 3a and b).



3a

Figure 3. *Coronal 3D reformatted HRCT images.* ***3a.*** *mediastinal window shows multiple tiny calcified nodule in the bilateral upper lung fields (blue arrow) with calcified mediastinal and hilar lymph nodes (green arrow).* ***3b.*** *Lung window shows multiple tiny calcified nodules in the bilateral lung fields*

(yellow arrow) with multiple "egg shell" calcification in mediastinal and bilateral hilar regions (red arrow). Local fibro atelectatic changes (white arrow) are present associated with para septal emphysematous changes (blue arrow).

On the basis of history, clinical presentation and imaging manifestation we made a diagnosis of complicated silicosis with progressive massive fibrosis. The patient was prescribed for symptomatic treatment with domiciliary oxygen along with inhaled short acting beta2 agonist. There is slight improvement as the patient is on regular follow up.

DISCUSSION

Silicosis is a fibrotic pneumoconiosis caused by inhalation of fine particles of crystalline silicon dioxide (silica) associated with mining, quarrying and tunneling occupation. Silicosis is also called as "potters rot". The aerodynamic particle size of 0.5 to 5 microns are deposited in the secondary pulmonary lobule. The entity leads to fatal and irreversible changes. Complicated silicosis, also known as progressive massive fibrosis, develops through confluence of individual silicate nodules. PMF is the result of two mechanisms by immunological and mechanical routes. The clinical, imaging and pathological manifestations of occupational lung diseases overlap in different causative factors as lung parenchymal response is similar. The radiograph of chest is the most important and primary diagnostic tool for evaluation. Plain chest radiograph with the background of clinical history, as in our present case, sometimes is sufficient for the confirmation of the diagnosis. Plain radiograph can detect calcified parenchymal opacities, fibrotic changes and calcified lymphadenopathy.[4,5] High-resolution CT (HRCT), is superior to chest radiography in the detection of fine parenchymal abnormalities.[6,7] HRCT chest is more accurate in providing differential diagnosis and is free from Interobserver variation in its interpretation. The application of CT to the occupational lung diseases helps in depicting morphological feature of respiratory manifestation more adjacent to pathology. HRCT is not cost effective and involves radiation exposure are the reason for excluding in the screening examination of such cases.[8] The CT appearance shows bilateral perihilar plaque like soft tissue densities showing multiple calcified nodules, commonly involving apical and posterior segments of the upper lobes associated with para septal emphysematous changes with the time, these large opacities migrate towards hila, accompanied by development of para cicatricial emphysema.[9] Progressive massive fibrosis occurs less frequently than in silicosis, seen as large masses of more than 1 cm in diameter. These masses develop in mid zones or the periphery of upper lung and migrate toward hila, leaving emphysematous spaces between them and pleura. Para cicatricial emphysema develops with growth of large opacities, which may cavitate, with or without infection. As HRCT detects pulmonary involvement of the occupational lung diseases earlier than conventional radiographs, early treatment and intervention to exposed individuals in initial stages would show better result. The findings have to be differentiated from other similar entities like pneumoconiosis, pulmonary alveolar microlithiasis, varicella pneumonia, sarcoidosis and fungal infections like blastomycosis, coccidiomycosis. Magnetic resonance imaging evaluation helps in differentiating PMF from lung cancer lesions where it is T2WI dark in the former and T2WI bright in the later. PMF is FDG-avid on PET-CT.[10] The management includes practicing good occupational hygiene. The norm of symptomatic treatment remains the call for the management.[11]

CONCLUSION

HRCT plays a pivot role in diagnosing the cases of pneumoconiosis with the delineation of findings to the level of basic lung unit of

secondary pulmonary lobule.

CONFLICT OF INTEREST

None

SOURCES OF FUNDING

None

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