International Journal of Science and Research (IJSR)
ISSN: 2319-7064
Impact Factor 2024: 7.101

A Rare Autopsy Case Study of Accelerated Pulmonary and Extra Pulmonary Silicosis in Spleen in a Young Male

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Abstract: ***Silicosis is one of the most common occupational lung diseases in the world, caused by inhalation of silica dust (silica dioxide). Taking into account the intensity, time of exposure, onset and development of the disease, we found an autopsy case of accelerated pulmonary and extrapulmonary silicosis in which inorganic particles (presumably silica) were documented inside granulomas and macrophages of the spleen along with tubercular lesions in liver - systemic silicotuberculosis. With present this case to emphasize on the fact that cases of systemic silicosis are often underdiagnosed and to discuss the little understood pathophysiology of disseminated silicosis.***

Keywords: accelerated, extra pulmonary, systemic silicotuberculosis

1. Introduction

Occupations related to silica exposure such as stone cutting, sand blasting, metal casting work and hard rock mining are prone to silicosis. Despite decades of preventive and control measures, silicosis is still prevalent. Inhalation of silica dust is deposited in distal airways and alveoli. This leads to inflammation followed by fibrosis and formation of nodules. [1]

Silicosis is much less prevalent in developed countries due to use of adequate protective measures, but it is still a problem in developing and under developed countries.

2. Case Presentation

32 - year - old deceased male, worked in stone factory for 10 years before he died of silicosis.

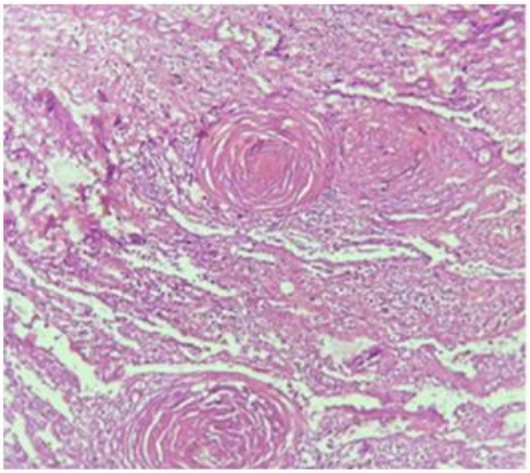
Gross - A piece of Lung weighed 127gm and measured 9x6x3 cm**.** External surface is covered partially with pleura which is thickened at places. On cut surface firm to hard nodules were felt on pressing. Multiple tiny grey brown nodules with diameter ranging from 0.2 to 0.5cm were present. A piece of Liver weighed 70gm and measured 7x6x3.5 cm. The external surface is covered with capsule. Cut surface is grey brown and congested. Piece of Spleen weighed 125gm and measured 8x6x4 cm. External surface is covered with capsule. Cut surface is congested. A piece of Kidney weighed 110gm and measured 7x6x2.5cm. External surface is covered with capsule. On cut surface cortico - medullary differentiation is seen. A piece of heart weighed 49 gm and measured 6x4x2.5cm. The wall thickness is 1.2 cm.

Microscopy - Lung - Multiple sections examined, showed many sclerohyaline nodules, pigment laden macrophages and mild chronic inflammatory cells infiltrate. On applying ZN staining no acid fast tubercular bacilli were seen. On applying MT stain the collagen fibers were stained blue. On polarizing light microscopy silica crystals of various sizes were visualized. Liver - Multiple sections were examined, showed healed tubercular granuloma and pigment laden macrophages. On polarizing light microscopy, silica crystals were not visualized. Spleen - Multiple sections were examined, showed sclerohyaline nodules and congestion. On applying MT stain, collagen fibres were stained blue. On polarizing light microscopy silica crystals of various sizes were visualized.

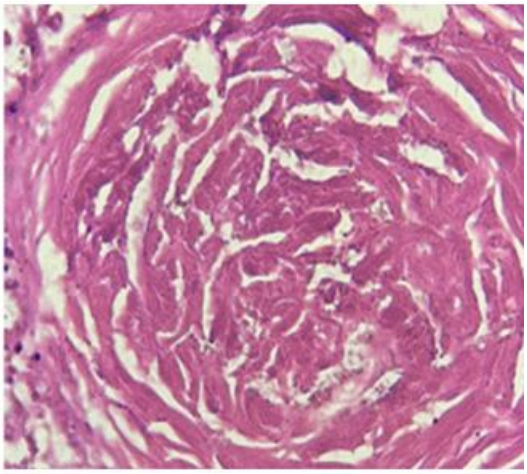
Lung- Gross specimen

Liver- Gross specimen

Spleen- Gross specimen



A.



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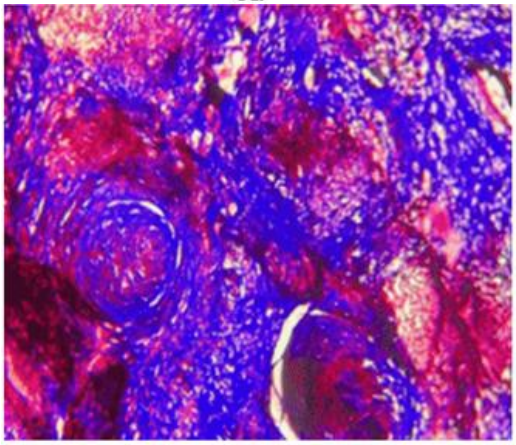
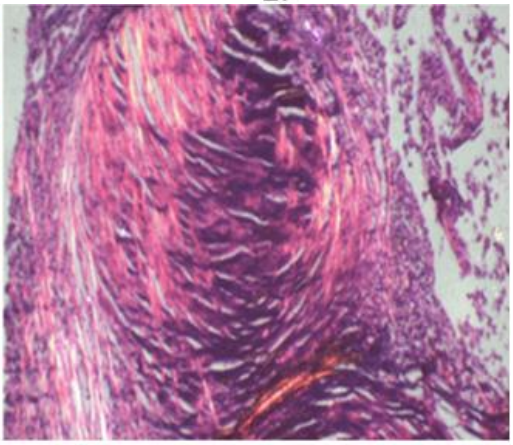
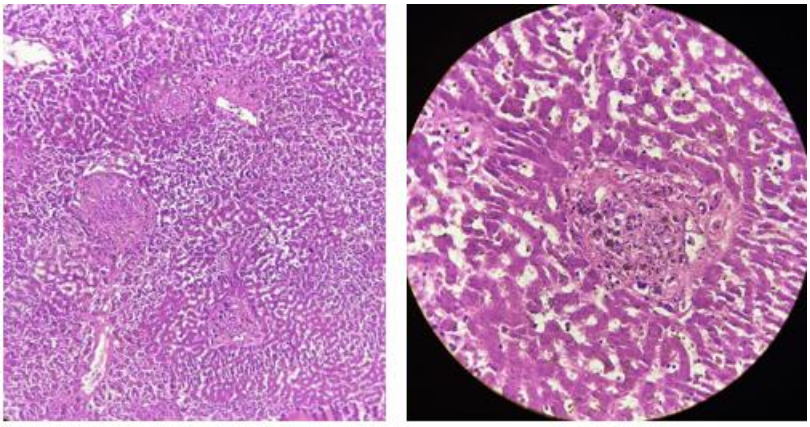


C



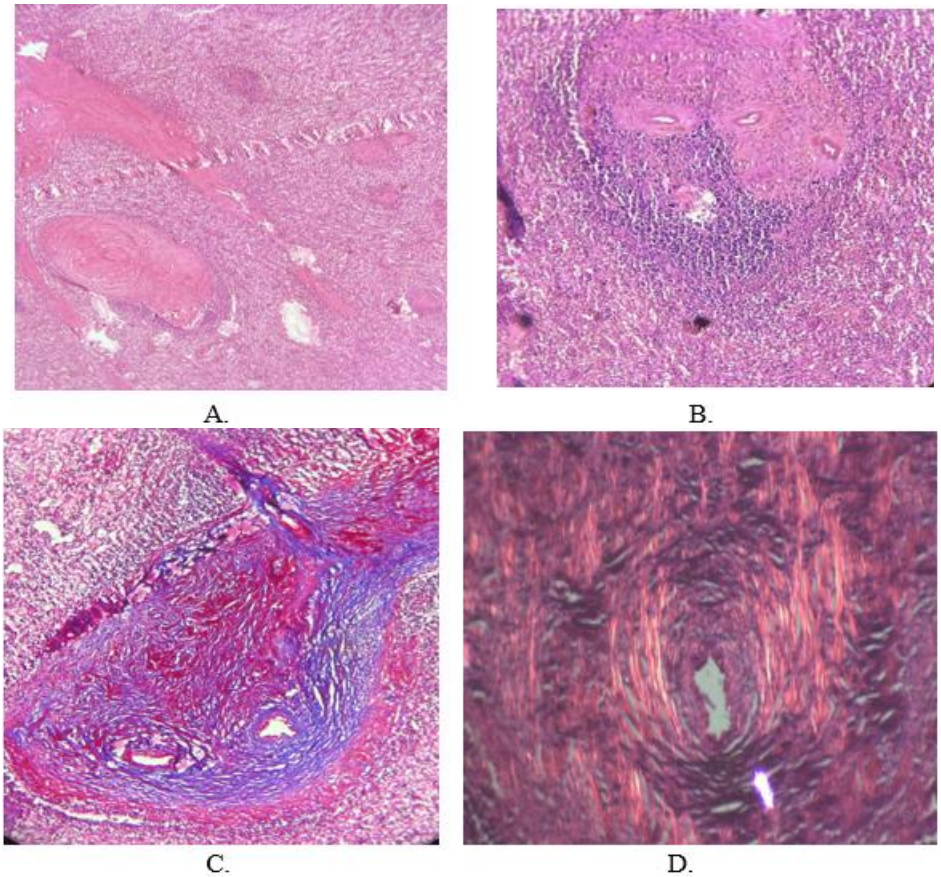
D

A and B - Pulmonary tissue with multiple sclerohyaline nodules, showing whorls of collagen, compatible with silicosis, on HE stain. Magnification 10x and 40x. C - On MT staining, collagen fibres were stained blue. D - on polarising microscope, birefringent silica particles were visualised.



A. B.

A and B - Liver tissue showing multiple granulomas, comprising of epithelioid cells, lymphocytes and few pigment laden macrophages, suggestive of healed tubercular granuloma. Magnification 10x and 40x



A. B.

C. D.

A and B - spleen tissue with multiple sclerohyaline nodules, composed of whorls of collagen. Magnification 4X, 10X. C - On MT staining, collagen fibres were stained blue. D - On polarising microscope, birefringent silica particles were visualised.

3. Discussion

There are three main clinical and pathological varieties of silicosis, [2] depending on the severity of the disease, the pattern of onset and the speed of progression. Its development is determined by the duration and concentration of exposure.3 Chronic silicosis (classic or nodular) is the most common type, resulting from long - term exposure (>10 years) at relatively low concentrations of silica dust. It appears 10–30 years after the first exposure.4 Accelerated silicosis develops 5–10 years after exposure to higher concentrations of silica dust.5 It is similar to chronic silicosis but with an earlier starting, a tendency to progress rapidly and a higher risk of complicated disease (progressive massive fibrosis). Acute silicosis can develop from weeks to 5 years after exposure to very high concentrations of silica dust. It has a rapid and severe onset with diffuse alveolar oedema. The characteristic histopathologic finding is the silicotic nodule mostly located near the respiratory bronchiole. A variety of silicotic lesions derived from thoracic silicosis via lymphohematogenous spread to liver, spleen, bone marrow and extrathoracic lymph nodes are described. In this case silicotic nodules are seen in spleen. The morphologic features of these lesions depend on the extent of macrophage aggregation, the occurrence of fibrogenesis, and the development of necrosis and degradative changes in macrophages and adjacent

extracellular matrix, presumably caused by lysosomal enzymes released from macrophages. The nodule is surrounded by whorled collagen in concentric layers. The sclerohyaline nodule, the characteristic lesion of silicosis, includes all of these features as it evolves through nodular histiocytic and subsequent fibrohistiocytic phases.6 The sclerohyaline nodule occurs infrequently in the spleen and liver, although less typical lesions caused by silica alone or admixed with other dusts seem to occur more commonly in these organs.7 On polarized microscopy, birefringent crystals of silica are seen in both lung and spleen in this case.

In liver, healed tubercular lesions are seen, suggesting tubercular infection. Silica - exposed workers, with or without silicosis, are at increased risk for tuberculosis and nontuberculous mycobacteria - related diseases. The risk of a patient with silicosis developing tuberculosis is higher (2.8 to 39 times higher, depending on the severity of the silicosis) than that found for healthy controls.8 It may be because crystalline silica inhibits the ability of pulmonary macrophages to phagocytosed mycobacteria.9

4. Conclusion

Silicosis should be understood to be a systemic multiorgan disease and further study of extrapulmonary disease is needed, including possible ways of acquisition, routes of dissemination, the role of macrophages and the study of drugs that modify the course of this disease. The independent development of extrapulmonary disease after the treatment of the initial lung disease should be considered in those patients with an unfavourable clinical course.

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