STUDY OF SILICOSIS IN THE INDUSTRIAL DISEASE HOSPITAL (ESI HOSPITAL) MADRAS

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ABSTRACTS

14 cases of silicosis who were working in Madras Stone Industry is reported. Only four cases were suffering from silicosis and 10 from solice tuberculosis. They were all working in the industry for a period of 10 to 20 years. 85.7% were in the 40–49 age group. X-ray mottling in 85.7% of cases. Sputum AFB positive in 71% of cases.

With the positive history of occupation in the stone industry with quartz cystals, feldspar and dried dust of finely powdered material, the cases were investigated. Chest X-ray, sputum analysis and lung biopsy were done to confirm the diagnosis. Literature of silicosis is reviewed.

MATERIALS AND METHODS

Patients who are referred for symptoms of chest disease are examined after getting detailed history of their stone-dust exposure. Blood examination, sputum examination and radiological examination were done in all the cases. Radiological, findings with a positive occupational exposure history were taken as confirmatary of the diagnosis. Lung biopsy, though done in one case, is not indicated in all the cases.

CASE REPORT

Case I

Mr. M, 38 years old working in a factory at Porur, Madras, came to the hospital first on 29/10/85 with the complaints of cough with expectoration and chest pain of one month duration. O.E. Afebrile, few scattered rales heard at the apices. Sputum-AFB negative; TC-8600/cells. P 65 to 20 E15 ESR-12/30 for half and one hour. X-ray chest PA view-mottling over both lung fields, more dense at the apices. Diagnosis of miliary tuberculosis was made and treatment started with INH/SM and Eifa with Prednisolone. He was on treatment regularly; On 29/9/86 he came with cough and chest pain of one month duration. O.E. Afebile, R.S. few rales heared TC-8800/cm DC-P62 L30 E8 ESR/32/72 for one and one-half hour sputum AFB-negative: X-ray chest PA view increased mottling with fibrosis of both lung fields with dense hilum; occupational history—worked as rolling shift worker for more than twenty years. Operator for mixing of sand and stones into fine powder; white stones (quartz), red stones (feldspar) and sands are mixed and made into powder form. Lung biopsy was done by Menghini's aspiration needle. Occupational history with clinical features, the diagnosis of Silico-tuberculosis was made.

Case II

Mr. S, 36 years—working in the above company as Grinder Operator. C/O cough with minimal sputum of one month duration R.S. Rales over high infraclavicular area present. TC-9400/10mm DC-P50 L 34-E-16 ESR 3/7mm for half and one hour sputum for AFB negative. Culture negative, X-ray chest PA view shows right special hazziness with rounded densities all over lung fields suggestive of Silico-tuberculosis.

Case III

Mr. P, 30 years—working in the above company; sand stones are pressed into moulds, trimming is also done under pressure in the heat room. Mantoux-negative, sputum AFB-negative TC 8400 DC: P60 L30 E10 ESR 7/14, half an hour C.S. No growth in culture, X-ray chest PA view patch of opacity right base.

Tables I, II, and III indicate that 85.7% were in the 40–49 years of age group and X-ray evidence also 85.7%. Sputum possibility of AFB is 71%.

Table I
Age Incidence

Age	No. of Cases	%
20–29	0	—
30–39	2	14.2%
40–49	12	85.7%

Table II

Chest X-ray	No.
Mottling—both lungs	12.85%
Patchy Opacity—Pneumonia	2–14%

Table III
Investigations

Sputum—AFB	10	71.4%
ESR	7	50.0%

Case IV

Mr. K, 36 years—working in the above industry for twelve years. Had treatment for tuberculosis with history of cough and expectoration for one year. Now on examination he is afebrile R.S. Rales over infraclavicular area present. Mantoux negative, sputum AFB negative. Culture—No organism grown; X-ray chest PA view shows fine reticular pattern over both lung fields. With a positive history of occupation in the above industry with exposure for more than twelve years diagnosed as silico-tuberculosis and treatment started.

DISCUSSION

Silica may exist in the combined forms called SILICATES which are themselves fibrogenic. It is respirable free silica incrystalline forms that causes SILICOSIS. Quartz, crystals (white stones), Feldspar (red stones) and dried dusts of finely powdered materials may become airborne before wetting or after drying.

PATHOGENESIS

When silica particles are installed and deposited in the lung periphery, they are ingested by macrophages. They enter these cells surrounded by an envelope of all membrane, the phagozome. Enzymes are secreted into this envelope; Silica particle destablizes the membrane, which ruptures and release these enzymes into the cytoplasm, killing the cell. Cell rupture releases the silica particles into the environment where they are encountered by successive waves of macrophages that meet the same fate. The process continues until macrophages can no longer reach silica particles. This can result, if silica is localized and resulting tissue reaction forms an effective barrier. What properties of silica accounts for cyto-toxicity? Angularity and sharpness of the crystals and silicic acid may cause rupture of macrophages—Fibrosis may be due to fibrogenic factor released from dusted macrophages. Silica by altering the immune system provide supply of macrophages to an area containing silica. Silicotic lessions contain plasma cells and immune globulin.

PATHOLOGY

In the chronic form of silicosis, the characteristic lession is the silicotic nodule. These are more numerous in the upper lobes. Lesions consists of concentric whorls of hyalinised, relatively acellular material.

Outside this zone of cellular connective tissue infiltrated with lymphocytes. Central hyalinised zone may contain some silica and most of the particles are found in the periphery.

IN SIMPLE SILICOSIS

The disease does not progress beyond the state of isolated silicotic nodules. Silicotic nodules become conglomerate forming masses of organizing fibrous tissue. This process called progressive Massive Fibrosis (PMF) attended by Massive obliteration of underlying lung tissue.

SILICO-TUBERCULOSIS

Mycobacterial infections may complicate silicosis because of the central role of macrophages in defense of the lungs against those infecting organisms.

Accelerated silicosis: seen in circumstances of more intense exposure—Progression is faster.

Acute Silicosis is due to heavy exposure to respirable free silica.

Silicotic lymphonodes may contain silicotic nodules.

CLINICAL FEATURES

1. Breathlessness on exertion.
2. Cough
3. Little sputum.

X-ray (1) rounded small opacity –1.5 mm; (2) Enlargement of hilar lymphnodes-nodes have peripheral calcification egg-shell pattern; (3) PMF—Coalescence of rounded small opacities to form larger aggregates bilateral and upper zones. They lead to large pneumoconiotic opacities; (4) These masses begin to contract, leaving clear spaces between their lateral margins and pleural surface (Angel Wings) appearance produced by sub-pleural emphysema and contracting large opacities; (5) Later leads to shrunken lungs and kinking of trachea; (6) Calcification rarely in silicotic nodules enhancing their radiographic visibility; (7) Acute silicosis produces lung consolidations in middle and lower zones.

PHYSIOLOGY

Obstruction, mixed obstruction and restriction, and restriction or pure restriction lead to abnormal spirometric tests.

COMPLICATIONS

1. Infections include tuberculosis and nocardia commonly.
2. High risk of developing collagen disease.
3. FMF leads to pneumothorax bronchopleural fistula, chronic cor pulmonale and respiratory failure.

PROGNOSIS

Is guarded because of progression of fibers is despite cessation of silica exposure. A patient with silicosis and positive tuberculin reaction has high risk of developing active P.T. and PMF.

TREATMENT

There is no satisfactory treatment for established fibrosis due to silica. Ssurveillance and treatment for complicating infections are indicated. Further exposure in fibrogenic dust is to be stopped. Jobs and habits (e.g.) sucking, that increase the risk of developing obstructive lung disease should be avoided.

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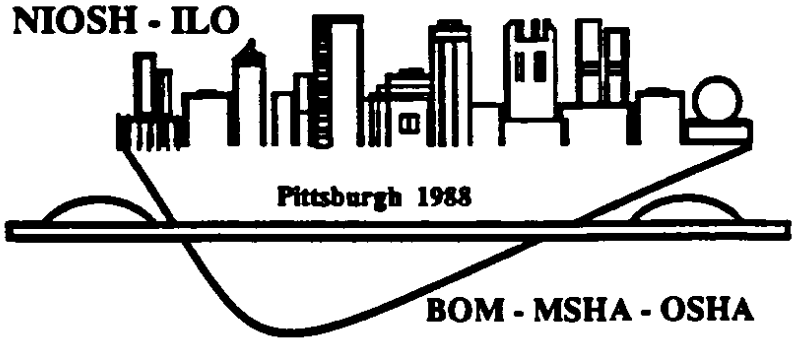

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