Silicosis not an Uncommon Problem - Case Report

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Introduction :

Silicosis refers to lung disease attributable to the inhalation of silicon dioxide, or silica in crystalline form usually as quartz and less commonly in other forms such as cristobalite and tridymite. This disease though becoming rare in western world is not uncommon in Bangladesh. It is caused by the inhalation of the free crystalline silicon dioxide (silica) dust or quartz particles.

Hippocrates reported that metal diggers or miners breathed with difficulty. Dutch pathologists in the 1700s noted that the lungs of stone cutters cut like a mass of sand[1]. In the early decades of this century, up until world war-II, silicosis remained the major cause of morbidity and mortality in men exposed to silica dust in a variety of occupations, many if not most of which remain risk occupations today[2].

Three clinicopathologic types of silicosis have been described, a chronic or classic form that typically follows exposure, measured in decades rather than years, an accelerated form following shorter, heavier exposure and acute form following intense exposure to the fine dust of high silica content for periods measured in months rather than years.

Tuberculosis may complicate the disease at any stage, but particularly at risk are cases of acute and accelerated disease[3,4]. Although Mycobacterium tuberculosis is the usual organism, atypical organisms are seen in certain exposures situations for instances acute silicosis[5,6] and probably reflect the profile of infection in the population from which the particular work force is drawn[3,7,8,9].

Extrathoracic manifestations of silicosis includes silicotic lesions in the lymph nodes in the neck and abdominal chains and occasionally in organs such as liver, spleen[10,1,5] and bonemarrow[11]. There is also an association between exposure to silica and certain connective tissue diseases other than rheumatoid arthritis, such as progressive systemic sclerosis[10,1,5]. Systemic lupus erythematosus and possibly a nephropathy involving glomerular capsular loops and basement membranes.

More recently associations between exposure to silica and/or the presence of silicosis with lung cancer have also been described[12].

Silica particles are ingested by alveolar macrophages and incorporated to phagosomes, they result in major damage to lysosomal membranes. This is followed by cell injury and death.

The presence of silicosis in an individual or a work force means that there has been exposure to quartz dust. The risk of developing disease is determined by the lung dust burden, which in turn is related primarily to the intensity, nature and duration of exposure.

The natural history of silicosis, even in the chronic form varies greatly. A report of a case control study based on the swedish silicosis register indicated that progression of disease was more likely to occur if exposure continued for 02 years or more after the earliest radiologic change was detected than if exposure after detection did not exceed two years[13].

The main symptom is breathlessness, first noted under the stress of effort and later at rest as the large working reserve of the lung is diminished[9]. In the absence of complications there may be no physical signs, when complications occurs, they may be reflected in chest examinations clubbing is not a feature of silicosis[14]. In the acute form particularly, breathlessness may become disabling with months, followed by impaired gas exchange and organ failure.

Uncomplicated silicosis is characterised by the presence of small rounded opacities on the chest x-ray. Silicotic nodules are usually though not invariably symmetrically distributed and tend to occur first in the upper zones, later though not invariably, involving other zones[1,5] occasionally they are calcified, resembling microlithiasis[10,15]. enlargement of hilar nodes may precede the development of the parenchymal lesions. Egg shell calcification when present, is strongly suggestive though not pathognomonic of silocosis[10,15].

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Complicated silicosis is characterised by the co-absence of small rounded opacities to form larger lesions. Sometimes CXR picture compatible with tuberculosis. Occasionally there is a single large (coin) lesion or silicoma, to be differentiated from lung cancer.

Lung functions shows reductions in lung volumes (vc, TLC, but often not FRC & RV) and in compliance, with impaired mixing; in addition, ventilation an effort may be increased and gas exchange impaired[1,16]. Diffusing capacity may or may not be affected[1,16]. In the accelerated and acute forms, functional changes are more marked and progression is more rapid.

Management and Control :

Once established, the fibreoptic process of chronic silicosis is thought to be irreversible. Management of the individual case is thus directed towards preventing progression and the development of complicated disease, which is usually accompanied by impairment and eventual disability.

Advice on leaving or changing a current job should be given in the light of knowledge of the environmental conditions past and present[19,20].

Use of cytotoxic and anti-inflammatory agents, including steroids not at present considered promising.

Acute silicosis is managed with bronchoalveolar lavage, steroids, or both when the disease manifests as alveolar proteinosis[21].

Case Report (4 cases) :

Four patients who are stone cutter by profession presented to us with occasional fever for last 2 years, occasional cough and dyspnoea on exertion. During this period there was no haemoptoysis and there was no loss of weight. Their age was between 30-60 years. All of them was in that profession for last 8-12 years. Among the 4 patients, 2 patients received antikochs for 6 months but without any improvement. Complete blood picture revealed no abnormalities. All of them were non diabetic but all of them have a history of smoking for 10-15 years. Sputum AFB (three sample) revealed negative for Tubercle Bacilli. tuberculin test showed an induration of 8-10 mm. CxR revealed mottled opacities involving all the zones of both lung fields. Pulmonary function test revealed restrictive pattern. Fibreoptic bronchoscopy revealed no abnormalities and bronchoalveolar lavage reavealed no AFB.

From the occupational history and radiographic abnormalities a diagnosis of silicosis was made.

As there is no effective treatment for silicosis palliative nonspecific symptomatic measures were taken. Patients improved with low flow oxygen, antibiotics, patients were discharged with advice to change the profession and to come for follow up.

Discussion :

Silicosis is not an uncommon problem in our country but many of them are treated with antikochs because radiologically CxR is consistent with tuberculosis. It is not possible to know how many workers have silicosis since notification procedures differ in different countries and many workers at risk are never surveyed by radiography.

On a worldwide basis many individuals are exposed to silica dust at work. For instance it has been estimated that over one million Americans are so exposed; furthermore, in from 30% to 50% of workplaces in different industrial sectors inspected between 1979 and 1982, at least one airborne dust sample contained over 100 $\mu m/m^3$ of crystaline silica. From this data, the US department of labor estimates that 59,000 cases of silicosis may be expected[22]. Numbers at work in mines and quarries and at risk in 14 countries in Europe have been estimated at 839,000; in 8 countries in Australia at 189,000 and in 6 countries in Africa at 394,000[23,7]. So there is chance of silicosis in a good number of people in different part of world, unfortunately in Bangladesh there is no such data by which we can assume how many people are at risk of developing silicosis.

The occupational history and radiographic findings almost always suffice to make the correct diagnosis of silicosis[10,15]. Problems arise when the history of exposure is remote, forgotten or missed (hence the importance of a careful and comprehensive occupational history) or when the radiologic features are unusual. Only in cases of mixed dust exposures might lung biopsy or other invasive studies be required. If coexisting infection with mycobacterium tuberculosis is suspected, bronchoscopy may be employed to collect cultures of organisms not found in sputum samples.

In the presence of a history of exposure and the characteristics radiography changes, the diagnosis

of silicosis should present no difficulty[10,15]. Problems arise when the history of exposure is remotes forgotten or missed (hence the importances of a careful and comprehensive occupational history) or when the radiologic features are unusual for example when the radiologic lesions are solitary or few or are confirmed to fibrotic changes in a single lobe, as in some cases of rapidly developing silicosis[5]. Dietection of silica in bronchoalveolar lavage material may raise suspicion. Lung biopsy (transbronchial, transthoracic or open) may be necessary to clarify the nature of the usual and/or conglomerate lesions, in which progressive maassive fibrosis, lung cancer, and tuberculosis are all possible diagnosis. Biopsy material should always be submitted to microanalysis for dust including silica, because for the most part the decision to perform a biosy indicates an element of doubt in the diagnosis[5].

Infection by mycobacteria or fungi, cor-pulmonale, spontaneous pneumothorax, broncholithiasis and tracheobronchial obstruction from polypoid granuloma developing in the neighbourhood of egg shell calcification in hilar nodes[10,5,3].

Diagnosis of active tuberculosis in the silicotic subject may be more difficult than in the nonsilicotic subject[10,4,17]. The presence of cough, haemoptysis, and systemic symptoms (weight loss, fever) should always raise the suspicion and prompt an active search for organisms and histologic evidence inbiologic material from any of the following sources, sputum bronchoalveolar lavage or lung biopsy material, pleural fluid or biopsy specimens and urines. It is invariably the chest roentgenogram, not the other features, that gives the key to the diagnosis[18].

As there is no effective treatment for silicosis, management of the individual case is thus directed toward preventing progression and the development of complicated disease. Advice on leaving or changing a current job. Lung - transplantation is offered to patients with end stage silicosis.

Preventive measures are based on control through minimizing generation of respirable dust and providing adequate ventilation at the work site. Respirator masks may be used.

Workers exposed to a risk of silicosis should be offered chest radiography at regular intervals, probably at least every four years, in order that the earliest signs of disease may be detected and the worker protected from further exposures.

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