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PERSONAL EXPERIENCES IN DIAGNOSIS OF ASBESTOSIS *

by Dr. S. R. KAMAT **

During my medical training it was considered common knowledge that people working in stone quarries get tuberculosis. However the concept of mechanisms in silicosis became clearer to me only during the period of training under British teachers. Over 25 years, I have seen only few cases of active tuberculosis with silicosis. The common silicosis that is seen in Bombay city is among workers working in foundry, dye casting, sand blasting, stone-grinding and quarries. It shows a picture of punctate multiple deposits.

In our unit we have seen progressive massive fibrosis in persons exposed to cement, sand and grain storage. The acute progressive silicosis[1] as seen in slate-pencil workers which shows chronic large consolidation ocupying both lung fields is uncommon in this city. It is common in Madhya Pradesh. These are young patients with breathlessness, cough, haemoptysis and black sputum. There is widespread prevalence of silicosis in Andhra Pradesh, Madhya Pradesh and Gujarat. This is vividly shown by a recent case of 24 years old female. She had worked for only 5 years in an open quartz mine. However she virtually lived in such mines alongwith her parents and later her husband for 19 years. There is reportedly so much dust in the vicinity of such open mines anywhere in India that the whole community inhales silica dust. The type of silicosis these people get is very disconcerting. This female patient died of respiratory and cardiac failure despite intensive management in our ward. Despite absence of infection her major complaint was cough with scanty expectoration, occasional blood-streaking and slight edema of feet. We covered her heart failure adequately but could not combat lung failure. She was malnourished, continued to have marked tachycardia due to pulmonary hypertension, anoxia and died with exhaustion.

The other common pneumoconiosis seen in Bombay is byssinosis, in cotton mill workers, particularly in presence of cough. In latter years these patients do not have work relationship but still improve after staying away from work for several weeks. They do show greater decline in lung function over a work day, and have increased IgG and IgE levels in comparison to cases with chronic bronchitis [2]. They also show an increased rate of annual decline in lung function as compared to control subjects. [3] We have also found few cases of byssinosis in workers working in not so dusty areas, cotton testing, ginning workers and those who have worked a few. years.

In contrast, other pneuomoconiosis are less frequent in India. We have seen cases with coal and gold pneumoconiosis, Berylliosis[4] and Arc welder's lung[5] In the latter case there was signficant increase in fibrosis with round cell infiltration in lung. It was then pointed out that at his Naval

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workspot he may have been incidentally exposed to asbestos material. However we did not find any physiologic or histopathologic changes consistent with asbestosis in this case. Even in Britain where asbestosis is diagnosed alongwith mesothelioma often, several cases have been found in dock workers and casual labourers[5].

First asbestosis case I saw in India was in Madras. A 35 year old non-smoking male complained of breathlessness on short exertion and cough with expectoration. He had worked as sprayer of railway coaches for 10 years and stopped due to chest symptoms. The radiograph suggested areas with collapse and fibrosis in both lower zones and an associated hyperluccnce in upper zones. Pulmonary function test showed slight obstruction and anoxia on exercise. The patient earlier had been submitted to bronchography which confirmed loss of lung volume and crowding of bronchi without dilatation. Our Physicians are still looking for asbestos bodies in sputum for confirmation of diagnosis of asbestosis This patient or several of our later patients never showed any, on routine sputum examination. Their presence only indicates an exposure to asbestos in a community. There is poor correlation between asbestos bodies in sputum and lung tissue[7].

In 1970 in Bombay we saw a male patient aged 37 years who had worked as asbestos sprayer for 12 years. Later, the factory employed him as clerk as he was breathless for 6 years. However for past 2 years he was unable to do even that job. He smoked 15 cigarettes a day and had stopped smoking due to chest symptoms. He never had any haemoptysis or large quantity of sputum but was treated as tuberculosis for 2 months. He had lost atleast 4 kgs. in weight over one year and had gr. IV dyspnoea (on slight exertion). He also developed swelling of feet and signs of cardiac failure over 6 months. Clinical examination revealed accentuated pulmonary second sound, rales at both bases and right mid zone, dullness in lower zones and trachea was deviated to right sde. An ECG revealed RBBB and RVH. The radiographss done over 2 years showed increased heart size, linear fibrosis, loss of lung volume and basal congestion. After treatment of heart failure the signs of failure reduced. Several admissions revealed a variable degree of anoxia. Pulmonary function studies revealed reduction of vital capacity (24% of predicted) FEV_1 /FVC%-86% FEV_1 -0.92 Lit. FVC-1.07 lit. MBC-47.6 Lit. mild reduction in expiratory flow rates (PEF 210 Lit). The oxygen uptake at rest was normal (228 ml). There was mild drop in oxygen saturation on exercise (from 89.1 to 87.0%) alongwith reduction in pCO^2 (29 mm). His capacity to do exercise was very limited (280 kgm/min). Patient died during fourth admission with exhaustion. cardiac and respiratory failure.

In 1974, we saw an industrialist aged 45 years, who had signs of bilateral basal fibrosis and had undergone several investigations here and abroad including open lung biopsy. He also gave a history of tobacco smoking (60/day) and pethidine addiction; from both he was salvaged. During his young apprentice days he was exposed to asbestos ore as it was brought to his godown by railway wagons in Bombay. He used to supervise breaking of asbestos ore and its storage by his workforce for 4 years but never handled it himself. He was also exposed to talc clay and iron ores for few years. He started feeling tired at the end of this period and was getting cough with slight breathlessness on heavy exertion.

He was married at the age of 28 years but found difficult to enjoy marital life; therefore he took to addiction but was salvaged. Over previous 10 years he continued to do his executive job with some disability. When we saw him at the age of 45 he had a few rales. The radiograph had remained unchanged over 12 years. It showed small areas of fibrosis at both bases. Lung biopsy has revealed nonspecific lung and some pleural fibrosis with silica dust; several lung functions had shown reduction in diffusion capacity and a drop in pO_2 on exercise (Table 1). Our lung functions showed a reduction in VC (60%) with some obstruction which improved after aerosol. His diagnosis was almost certainly asbestosis gr. I.

A typical mild case can be described as follows. A male aged 45 years (Engineer) has been working in asbestos yarn making for 22 years and also smoked 30 cigarettes per day for 35 years. He had a history of smoker's cough for several years which was worse for 3 months with small quantity of sputum. He was complaining of tiredness and fatigue on exertion particularly on climbing for several months (gr. II dyspnoea). After giving some bronchodilators he had no lung signs. A chest radiograph revealed increased vasculature, calcified scars in right paracardiac region and grade I linear fibrosis suggestive of asbestosis. The lung functions were normal VC-3.08 Lit (98%) of predicted, FEV_1 -VC%-86%, FEV_1 -2.66 lit. PEF-300 to 330 lit. after bronchodilator aerosol. Minute ventilation and oxygen uptake were normal at rest and on exercise. However his capacity to do exercise was limited to 400-500 Kgm/min. Arterial pO_2 at rest was 85 mm and declined to 75 mm after exercise, pCO_2 at rest was 34 and rose to 36 mm on exercise. He developed respiratory acidosis; pH was 7.44 at rest and changed to 7.34 on exercise, pO_2 after oxygen was 500 mm indicating a mild diffusion block. We have seen several patients who are exposed regularly to asbestos-like insulation material, work in engineering or electrical industries getting symptoms similar to this patient.

A factory dealing with asbestos cement sheets requested us to read their workers' radiographs. In this factory it was learnt that several older workers were complaining of respiratory problems and had taken retirement. The regular workers (789) presently in employment were studied for symptoms and radiographs were read by two trained readers independently and in consultation. We found 28% as having gr. 1|1 asbestos related fibrosis. Another 8.4% radiographs were suggestive of gr. 1|2 asbestosis. There were changes suggestive of nonspecific scars in 16.3% and tuberculous scars in 2.4%. We do think quite a few of these scars may be also related to work exposure. There were calcified pleural scars in 2.8% subjects. We also later studied a similar number of radiographs from another factory in India and found a large prevalence of abnormalities attributable to asbestos exposure. Due to administrative considerations, these findings were explained away.

With intelligent cooperation of workers and some doctors we were privileged to see 28 workers working in asbestos industry over last 2 years[9]: 20(71%) of these were smokers. They had a mean work exposure of 12.9 years. One of these developed symptoms before 5 years of exposure and 7 after an exposure of 6-10 years. Most had exertional dyspnoea as the prominent symptom. Chest pain (usually dull) came with feeling of tightness. In 57% there was weight loss; weakness was complained by 79% and cough was present in 46%. Finger

clubbing was seen in 25%. On clinical examination only a few had evidence of rhonchi or bronchospasm; 8 had diminished breath sounds and rales were present in 46%.

Of 28 cases 24 subjects were considered as having asbestosis. Thus 79% showed linear and nodular fibrosis in lower and mid zones. Pleural scars were seen in 15 subjects. One worker with circular masses in both lungs was suspected to have malignancy but on direct aspiration this proved to be a collection of pus. We also have seen a case with bilateral pleural transudates in a station master who was working at a station where asbestos-loaded wagons were unloaded. One of our patients with gr. II asbestosis developed cancer and died soon after discharge. We had another patient who had advanced cardiac failure with asbestosis. Routine spirometry was normal in 48% and showed restriction in 53%; low FEV_1 was seen in 17%. In 82% workers there was no evidence of obstruction. Of 23 patients who had blood gas studies done at rest and exercise, 11 had above 90 mm pO^2 at rest, 8 subjects had resting pO_2 levels below 90 mm. After exercise testing there was no adequate rise in pO_2 in 13 patients; in 9 patents there was significant drop in pO^2 on exercise.

We have seen patients with pleural plaques in nearly asymptomatic workers but large shadows seem uncommon. Intermittently we see exposed workers who complain of symptoms which may be related to work in asbestos industries. Several times we have heard that the management or medical consultants or union officials do not encourage obtaining a confirmation of diagnosis. We have noticed that radiographs done are regularly read as bronchiectasis or nonspecific fibrosis. As in our first case from Madras, several workers as they complained of cough or haemoptysis are treated as tuberculosis and several workers are persuaded to retire with questionable benefits. Mild attempts at getting some of our patients compensated have met with covert resistance and interpersonal difficulties.

A typical case from factory may present to his doctors after 8-15 years of exposure to asbestos. He notices some dyspnoea or tiredness which later progresses to chest pain, anorexia and loss of weight. Clinical examination shows few rales but there may be no clubbing. Radiographs may show micro-nodular and linear deposits particularly at right base and sometimes a ground glass appearance elsewhere. Pulmonary function may show mild restriction, lowered O_2 uptake on exercise and a drop in pO_2 on exercise. This may be due to gr.I asbestosis with pleural thickening and moderate functional disability.

We suspect that several experts in India are trying to minimise the occurrence of asbestos related diseases with following defensive arguments (a) We do not have the same intensity of industrial exposure; (b) Our workers are not very productive and do not get heavily exposed; (c) Our workers smoke heavily, imbibe alcohol and often get complicating diseases like infections, bronchiectasis or tuberculosis (d) Indians are perhaps more resistant to diseases prevalent in developed countries. (e) By text book criteria unless asbestos bodies in sputum are found or lung biopsy shows asbestos bodies, or radiographs show advanced changes similar to silicosis, we should not diagnose asbestosis. Some of these arguments according to our group are not strictly based scientific evidence.

As symptoms with asbestosis are not

pathognomonic the diseases in pleura, lung should be investigated properly .We have seen cases of mesothelioma or lung cancer diagnosed on autopsy in our institute and mesotheliomas have been reported in farmers of Maharashtra perhaps with exposure to asbestos containing earth. We believe that asbestosis and related diseases may be common in cities of India and it needs greater vigilance, suspicion and proper investigation to diagnose them more often. With rapid industrialisation taking place in our country the medical experts should practice this approach to encourage sustained productivity, labour welfare and to prevent premature loss of young workers.

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