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"PATTERN OF ASBESTOS WORKERS IN BOMBAY" *

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SUMMARY :

A study of 28 workers exposed to asbestos, and reporting to this unit is presented. They were thoroughly investigated with radiographs, lung function and arterial blood gases at rest and after exercise to detect established cases of asbestosis. Basal rales were heard in 15 (46%) workers). In 24 subjects there were obvious abnormalities on radiographs suggestive of asbestosis. Radiologically the small reticulonodular opacities in lower and middle zones were commonly found. In 5 workers there was evidence of obstruction but in most cases restrictive functional defect was seen. Arterial oxygen desaturation on exercise occured in 8 (35%) workers.

INTRODUCTION :

Alongwith wider use in industries, world consumption of asbestos is escalating; so also the awareness of health hazards due to asbestos exposure has come significant in past 30 years. Though a typical advanced asbestosis is diagnosed without difficulty, on the basis of clinical findings, radiological abnormalities and restrictive charges in pulmonary function, but the industrial physician should be concerned with diagnosing, an early asbestosis. Leathart [1] has suggested that the earliest finding in asbestosis may be the presence of basal crepitations. While other workers [2-4] have found that abnormalities in lung function particularly reduced VC and diffusing capacity may precede changes in the chest roentogenogram or are seen with equivocal radiological changes.

An over-all diagnosis of asbestosis is based upon: physical signs of pulmonary fibrosis, radiological abnormalities and restrictive changes in lung functions. With slight modification of standad criteria, [5-10] we used the following evidence for the diagnosis of asbestosis :

(1) Adequate history of exposure to asbestos fibre at work.

(2) Clinical presentation of breathlessness, cough, clubbing and fine rales on ausculation.

(3) Radiological evidence of pleural plaques, calcification or reticulonodular shadows suggestive of interstitial fibrosis.

(4) Restrictive functional disability and arterial blood gases alteration at rest or on exercise testing.

We could not use D_{co} as a test due to technical reasons as also lung biopsy [11] as unnecessary for confirming the diagnosis of asbestosis. Demonstration of asbestos bodies in sputum was also not included in criteria as it does not usually prove asbestosis. [10,12]

MATERIAL & METHODS :

From suspected exposed workers, 28 were studied fully to evaluate for

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abnormalities associated with asbestosis. They had been working in different factories in the city. The investigations consisted of full radiograph, clinical examination, lung function and blood gas studies. Chest examination, particularly included looking for fine basal rales and presence of finger clubbing. Spirometry was done on Stead-Well's spirometer before and after a bronchodilator aerosol. All lung functions have been expressed as percentage of predicted values as per Indian norms.[13] As per standard criteria normal functions were within ± 20% of predicted values, mild disability between 60-80% moderate between 40-50% and severe disability less than 40%. In all subjects blood gas studies before and after exercise (as an alternative to Dco studies) was done on the same day.

RESULTS :

All 28 workers were males with a mean age of 36 years (Table No. 1).

Table 1 : Age Distribution of asbestos exposed workers.

Age in Years	
21-30	4 (14%)
31-40	17 (63%)
41-50	6 (20%)
51-60	1 (3%)

Mean Age : 46 Years.

Of these 20 (71%) were smokers, including 3 heavy (20+/day) smokers. These belonged to different departments like winding, spinning. polishing etc. with a mean exposure to asbestos fibre of 12.9 years. Of these 20 (72%) had been exposed for over 10 years. While mean latent period before any clinical symptoms was 11.9 years but 6 (20%) manifested after an exposure of over 15 years. (Table 2).

Table 2: Duration of exposure to asbestos and time lag before development of symptoms.

Yrs. of exposure	No. of patients	Period	No. of patients
1-5	1 (3%)	1-5	5 (18%)
6-10	7 (25%)	6-10	8 (29%)
11-15	14 (47%)	11-15	9 (33%)
16-20	7 (25%)	16-20	6 (20%)
Mean - 12.9 years		Mean - 11.9 Years	

Breathlessness on exertion was a prominant symptom in 26 workers (93%). Dull aching chest pain and feeling of tightness in chest was commoner in workers with pleural or advanced pulmonary involvement. Unusually 22 (79%) workers also complained of anorexia, weakness and weight loss (Table No. 3).

Table 3 : Clinical symptoms in 28 asbestos workers.

1. Breathlessness	26(93%)
2. Anorexia, Wt. Loss Weakness etc.	22(79%)
3. Chest pain	16(57%)
4. Cough	15(46%)

Mean duration of symptoms 2.73 Yrs.

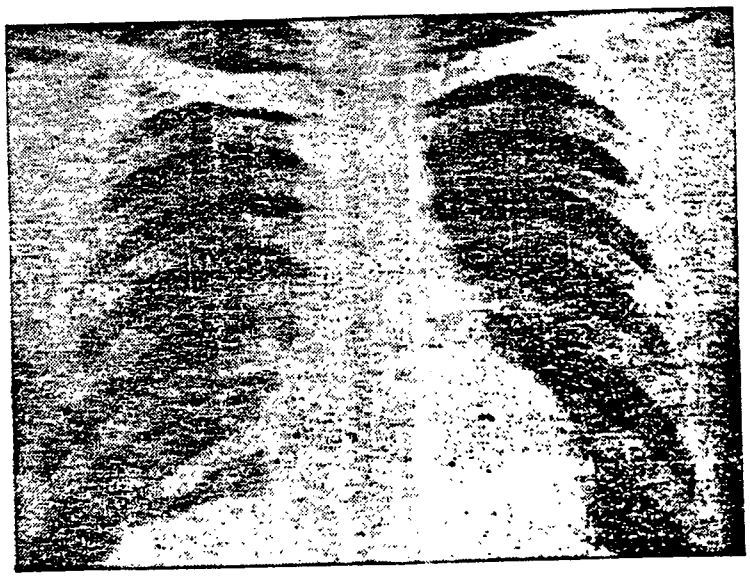
Finger clubbing (gr. I-II) was seen in 7 workers (25%). The presence of fine rales was common in subjects present where'in the radiograph showed small reticulo-nodular deposits or honey combing.

Radiological evidence suggesting asbestosis was seen in 24 patients (Table No. 4). Mainly there were bilateral fine reticulo-nodular deposits in lower and mid zones (79%) (Fig. 1 & 2). Pleural involvement was seen in 15 (54%). It was more common on the lateral parietal layer than on diaphragmatic layer (Fig. 3). One worker presented with circular mass on left lateral wall in upper zone with pleural involvement on right side thought to be malignant. However this had not changed over 4 years as compared with earlier X-rays : later we aspirated pus by direct puncture from this mass (Fig. 4).

Table 4 : Radiological patterns in asbestos workers

Pleural involvement only	2 (7%)
Parenchymal involvement only	9 (32%)
Pleural & Parenchymal	" 13 (47%)
Normal	2 (7%)
Nonspecific	2 (7%)

Low FEV^1 was seen in 5 workers (17%); while VC% of predicted was abnormal in 15(53%) workers, suggesting that restrictive ventilatory defect is more easily judged by FVC



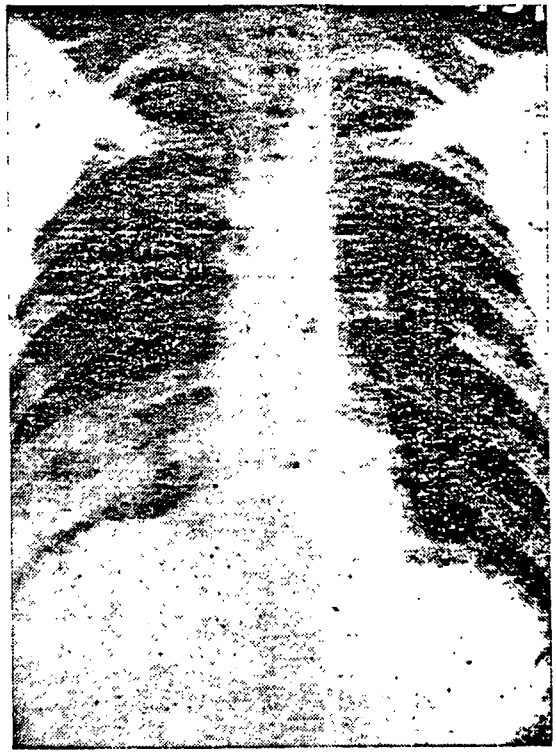
Small 'bead-like' and fine linear opacities in early asbestosis.



Smalt nodular and linear opacities, mare in both lower zones than mid zone. Note the shaggy heart borders.



Bilateral extensive pleural thickening with calcification.



Cicular rounded opacity on left lateral wall.

(Table No. 5). This was probably commensurate to the degree of pleural involvement (Table No. 6) 5 workers refused arterial blood gas study. Mean pO_2 at rest was 86 mm of Hg while mean $paCO_2$ was 36.8 mm of Hg suggesting mild hyperventilation at rest. Mean SaO_2 was 96% at rest while it decreased to 95% on exercise (Table No. 6). Significant arterial oxygen desaturation on exercise occured in 8(35%) workers.

DISCUSSION :

The study narrates our experience and may not reflect on the relative incidence of asbestosis in Bombay. The hazards due to exposure of asbestosis were first recognised in Europe [14] at the beginning of the 20th century. Murray reported the first case of pulmonary asbestos is in 1907. In 1930, asbestosis was recognised as an industrial disease and preventive measures were taken.

It is reported [15] that usually a delay of several decades occurs between first exposure and onset of symptoms and in no study in man, it has been possible to measure the dose. In our study mean lag period was 11.9 years.

Though we have not assessed the role of smoking in our study, this has been established by others. [16] Asbestos inhalation also produces pulmonary fibrosis. [17-18] While a high prevalence of fibrosis is seen in smoking asbestos workers but it is uncertain whether the two have an additive effect. [18]

Table 5 : Spirometric indices in asbestos workers

%	$\frac{FEV_1}{FVC}$ %	$\frac{VC\ Obs.}{VC\ Pred.}$ %	Mean
∠40	—	4(14%)	—
∠60 ⊿40	1(3%)	4(14%)	$\frac{FEV_1}{FVC}$ %- 86.27%
∠80 ⊿60	4(14%)	7(25%)	$\frac{VCObs}{VC\ Pred.}$% 76.82%
Normal	23(83%)	13(47%)	

Table 6 : Summary of radiographic and functional changes.

Sr. No.	Radiology	PFT O	PFT R	PaO_2 Rest	in mm of Hg	Exer.	SaO_2 On Exer.
1.	ABN (1)		ABN (Mod)	76	—	81	NC
2.	ABN (1)	N	ABN (Mod)	91	—	93	↓
3.	ABN (2)	N	N	106	—	101	↓
4.	ABN (3)	N	ABN (Mild)	78	—	94	↓
5.	ABN (2)	N	N	102	—	97	NC
6.	ABN (2)	N	ABN (Mild)	100	—	106	NC
7.	ABN(NS)	N	N	84	—	112	↑
8.	ABN (2)	N	ABN (Mod)		—		—
9.	N	N	N	112	—	111	NC
10.	ABN (2)	N	ABN (Mild)	92	—	95	NC
11.	ABN (3)	N	ABN (S)	70	—	51.5	
12.	ABN (3)	N	ABN (Mild)	81.5	—	87	↑
13.	ABN (3)	N	ABN (Severe)	56	—	60	↓
14.	ABN (3)	N	ABN (Severe)	102	—	62	↓
15.	ABN (3)	N	N	83	—	88	↓
16.	ABN (3)	N	ABN (Severe)	66	—	43.5	
17.	ABN (2)	N	N		—		—
18.	ABN (2)	N	N	99	—	100	NC
19.	ABN (3)	ABN (Mild)	N		—		—
20.	ABN (3)	N	N	100	—	97	NC
21.	ABN (2)	ABN (Mod)	ABN (Mod)	64	—	65	NC
22.	ABN (2)	ABN (Mild)	N		—		—
23.	ABN (3)	ABN (Mild)	N	76	—	73	↓
24.	N	N	N		—		—
25.	ABN (3)	N	ABN (Mild)	71	—	83	↕
26.	ABN (3)	N	ABN (Mild)	90	—	81	
27.	ABN (3)	ABN (Mod)	ABN (Mild)	100	—	112	NC
28.	ABN (NS)	N	N	88	—	98	↑

O-Obstructive R-Restrictive NC-No change ABN Abnormal

1-Pleural involvement
2-Parenchymal involvement
3-Pleural + Parenchymal involvement
N-Normal NS-Non-specific

↑ SaO_2 — ∠ + 0.5%
↓ SaO_2 — ∠ — 0.5%
NC ± 0.5%

There is high risk of developing lung cancer in asbestos workers who are smokers. In a study by Selikoff [19] the combination of asbestos exposure and cigarettes increased the risk 90 times as compared with control group. Relative contribution of cigarette smoking to malignancy is not assessed in our study as the exposure period is short.

Clinical features of asbestosis are not pathognomonic. Breathlessness on exertion was the predominant symptom in our study. Breathlessness is seen first after heavy work and then with smaller exertion as the disease progresses. As reported by Parkes [10], the degree of breathlessness does not correlate well with abnormal physical signs in the lung and radiographic appearances. Other surprising feature was presence of generalised symptoms like anorexia, weight loss and weakness in 22 workers (79%). This perhaps related to chronic tiredness and mild dyspnoea. In majority of cases only a small quantity of sputum was complained. Chest tightness was common in patient with advanced disease probably due to stiffness of lung while chest pain was present with involvement of pleura.

Fine crepitations are the earliest and most constant physical sign as reported earlier. [7, 20] They were first detected either at the bases after a deep inspiration. As the fibrosis progress they become more numerous and widespread. An important feature of this is that they are persistent and not altered by coughing, as is often with other lung diseases.

The difficulties in the diagnosis of early radiological changes of asbestosis has been emphasized repeatedly by Pendergras since 1938, so it is not surprising to find a difference of opinion in radiological diagnosis. Radiographic abnormalities like bilateral small irregular linear opacities predominant in the lower lung fields, are suggestive of interestitial fibrosis [22, 23]

The early bilateral changes are first seen in costophrenic angles in different forms of increased basal vascular markings; these may be thickened particularly at branchings with small rounded bead like opacities along the path of finer markings and a horizontal pattern resembling kerley's B lines. [23,24]

In more advanced cases a honey combing pattern is common either due to fibrosis or superimposition effect of different radiodensities. When fibrosis is severe, the outlines of the diaphragm and heart shadow may become blurred and shaggy (Fig. 2). Rounded opacities similar to silicosis are occasionally seen probably due to free silica present in asbestos cement dust. [25] But larger opacities resembling those of Caplan's syndrome and progressive massive fibrosis are unusual, with asbestosis. According to Yazicoglu et al, [26] the first obvious sign sign is the development of geographic calcified plaques in the pleura, radiographic changes of pulmonary fibrosis develop later.

As suggested by Gibbs. [27] the changes in pulmonary mechanics were considered to be early because they occured in workers who had no radiological evidence of disease or definite changes in other measurements of lung function. The characteristic abnormalities reported include progressive reduction in VC, TLC and in diffusing capacity, increased static recoil pressure, decreased lung compliance and decreased VC% predicted, without airways obstruction. The restrictive lung lesion in asbestosis with decreased inspiratory capacity and decreased lung volume is probably caused both by pleural adhesion and plaques and by shrinkage due to pulmonary fibrosis. [28]

Airways obstruction was not considered a feature of asbestosis.[4] Though recent reports[29] express the view that obstruction especially of small airways is not uncommon. It is widely known[4,14] that the main functional defect in asbestosis is reduced diffusing capacity of the lungs, this was inferred from occurence of arterial oxygen desaturation especially after exercises as in our study too.

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