SILICOSIS IN THE MANUFACTURE OF SCOURING POWDER

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SYNOPSIS

Two workers from a factory making abrasive cleaners or scouring powders were found to be suffering from silicosis. One was a fatal case and the other developed pulmonary tuberculosis. Dust measurements of the work process and analysis of the abrasive powder used showed that both workers had been exposed to excessive amounts of free silica. This paper serves as a reminder that a serious silica hazard can exist in the manufacture of scouring powder. It also illustrates the importance of the occupational history in the diagnosis of diffuse pulmonary opacities on Chest X-ray.

INTRODUCTION

Abrasive cleaners are widely used for cleaning of household utensils, sinks, floors etc. They are basically a mixture of detergent (soap powder) and an insoluble abrasive (eg powedered silica or insoluble silicates). In some detergent factories, the raw materials are first processed. In others the prepared constituents are simply mixed and packaged. Various authors have reported a high risk of silicosis in detergent factories. This was particularly so, where control measures were inadequate.

In 1929, Middleton (1) drew attention to the silicosis hazard in the manufacture of scouring powder. Following this, there were a number of case reports of silicosis in this industry (2-6), mainly in the United Kingdom (UK) and the United States (US).

Such reports are now rare (7). In the UK, the use of silica in domestic scouring powders has only recently ceased, and it is still used in non-domestic abrasive soaps (8). Large amounts of silica are routinely used in both domestic and industrial abrasive cleaners manufactured in the US (7, 9). The silica is produced in silica flour mills where a recent report indicated a high risk of silicosis (9). There have however been no recent cases of silicosis among American workers directly involved in the manufacture of scouring powder. This is probably due to effective protective measures employed by the manufacturers (7).

In Singapore, there is at present only one factory involved in the manufacture of scouring powder. There is no silica hazard in this factory as the abrasive used contains less than 1 per cent of free silica. Until mid 1983 there was another factory producing scouring powder using powdered silica as the abrasive agent.

In this report, we describe the case histories of two workers who contracted silicosis as a result of their work in this second factory. They are the first cases of silicosis detected in the local detergent industry. Although there are no workers currently exposed to a silica hazard in this industry, more cases of silicosis among workers who had previously worked in this industry may still be detected for some years to come.

CASE 1

This was a 50 year old woman who worked in the detergent factory since 1973. In 1981, she developed headache, nausea and blurring of vision. Three months later, she was treated for torula meningitis. Cryptococcus neoformans was cultured from her cerebrospinal fluid.

In March 1982, she was again treated for torula meningitis, following a relapse of symptoms. A Chest X-ray done at that time showed a mass in the left mid zone (fig 1) which persisted despite a 6 week course of antibiotics. A clinical diagnosis of torullosis of the lung was made and a left upper lobectomy was done in June 1982.

Her case was referred by the Company to the Ministry of Labour to determine if her condition was work related. In the course of investigations by the Department of Industrial Health (DIH), detailed occupational history and environmental assessments subsequently confirmed that she had been exposed to a significant silica hazard (please see section on "Occupational Exposure").

At the time of investigation by the IHD, she was asymptomatic. There were no crepitations or rhonchi; no cyanosis or clubbing.

Her lung function results in early 1984 indicated a restrictive defect:

	Observed	Predicted
FEV_1 (L)	1.52	2.06
FVC (L)	1.77	2.40
FEV_1/FVC (%)	86%	> 70%

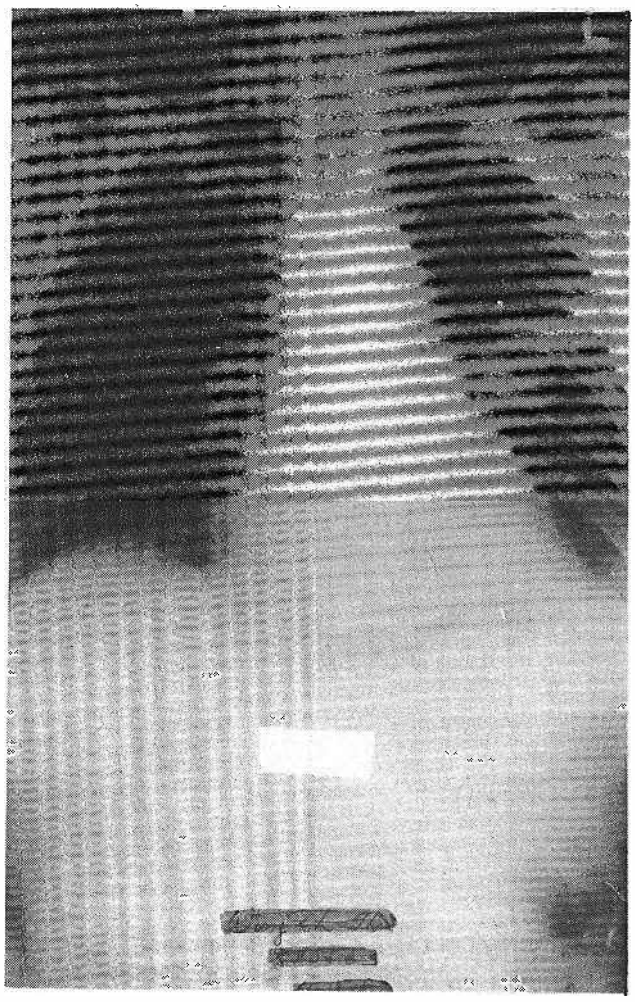

Figure 1
Chest X-rays of Case 1 in Mar 82 showing an opacity in the left mid zone (Prelobectomy).

A review of the prelobectomy Chest X-ray done in June 1982 and the postlobectomy Chest X-ray (fig 2) done in April 1984 showed that both Chest X-rays were suggestive of silicosis classifiable as category 0/1 p under the ILO International Classification of Radiographs of Pneumoconiosis 1980 (10).

Histological examination of the left upper lobectomy specimen taken in June 1982 showed numerous silicotic nodules scattered throughout the lobe, along alveolar septa and adjacent to respiratory bronchioles, pulmonary arteries and in the subpleural region (fig 3). The nodules showed three fairly distinct zones. Innermost was a central hyalinised zone with variable anthracotic pigment and a small amount of birefringent particles of varying shapes, most of them about 2-4 microns in size (fig 4).

Outside this was a zone with many concentrically arranged fibroblasts. The outermost zone or reactive zone was wide and prominent and consisted of cellular fibrosis tissue with macrophages and fairly numerous silica particles and anthracotic pigment (fig 5). Scattered multinucleate giant cells were present here, some with ingested birefringent crystals. A few showed intracytoplasmic laminate calcifications (Schauman bodies).

In several areas, the nodules had become crowded and confluent. These larger nodules and confluent lesions showed stellate outline with extension of fibrosis in the alveolar walls. The earlier lesions consisted of focal collections of macrophages with ingested birefringent crystals in the alveolar septa (fig 6). Later lesions showed central fibrosis and enlargement.

Some of the larger nodules showed central caseous necrosis extending to surrounding hyalinised fibrotic stroma with birefringent particles (fig 7). Acid fast bacilli were demonstrated within the necrotic tissue. The tracheobronchial lymph nodes also showed silicotic nodules, some with central caseous necrosis.

There was no evidence of torullosis in the lung tissue.

A histological diagnosis of silicotuberculosis was made in the light of evidence of industrial exposure (please see section on "Occupational Exposure").

Antituberculous treatment was completed in early 1984. Subsequently her condition was stable clinically as well as radiologically.

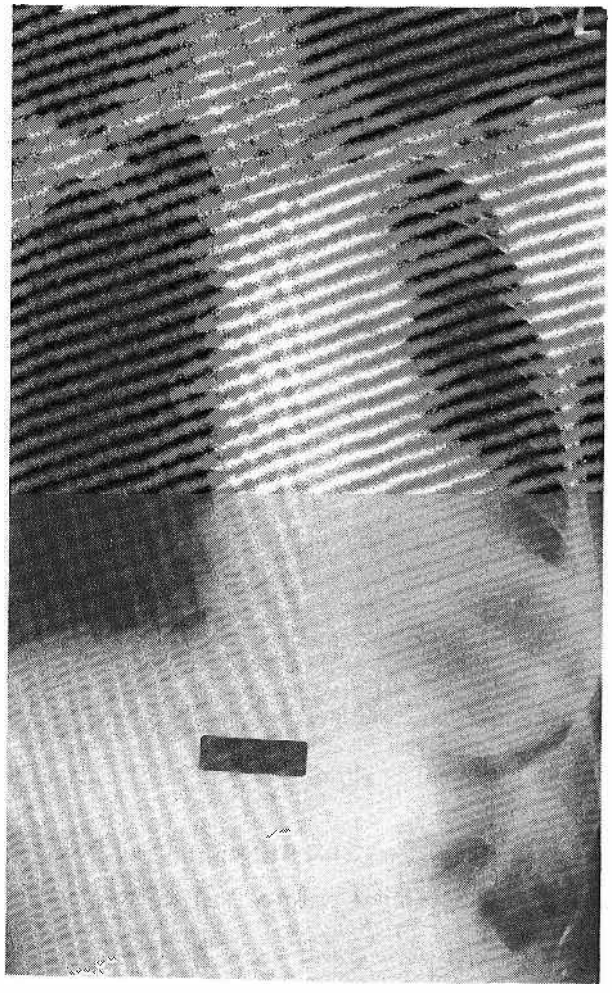

Figure 2
Chest X-rays of Case 1 in Apr 84 showing contraction of the left lung volume after left upper lobectomy. There is mediastinal shift and elevation of the left hemidiaphragm.

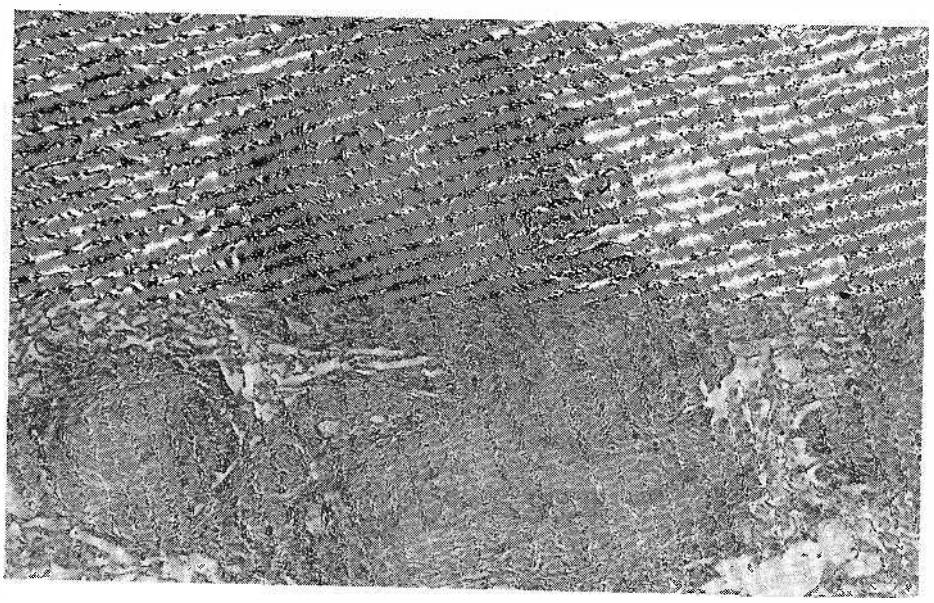

Figure 3
Section of Lung Showing Silicotic Nodules:
Central hyalinised area with surrounding concentric fibrosis and wide reactive periphery in which are multinucleate giant cells. There is a tendency to confluent lesions. Stellate fibrosis with interstitial fibrosis are present.
(H & E × 40 Magnification)

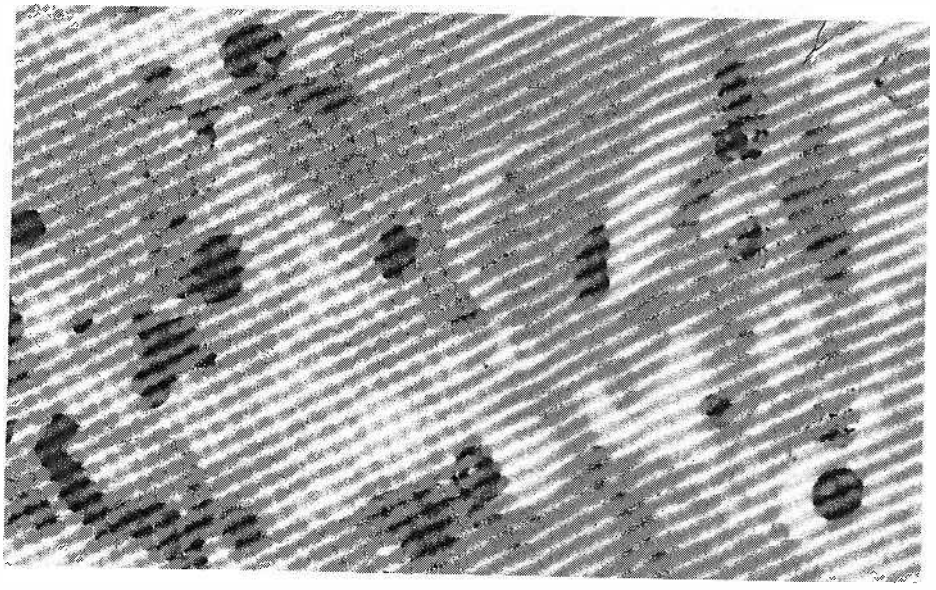

Figure 4
Silicotic Nodule: Central Hyalinised Zone
Central hyalinised collagen with birefringent crystals and anthracotic pigment
(H & E × 1000 Magnification)

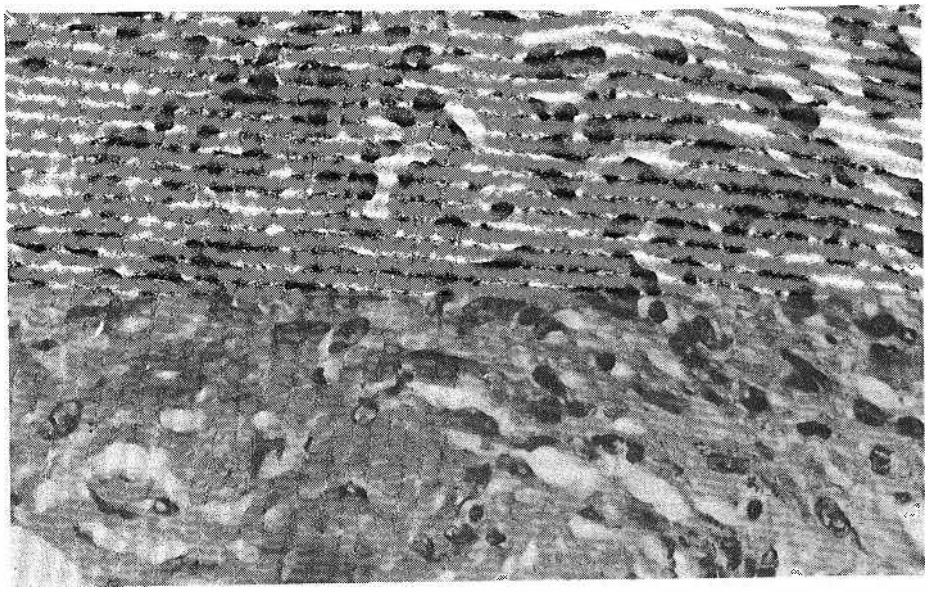

Figure 5
Silicotic Nodule: Peripheral Reactive Zone:
Fibroblasts and macrophages with ingested birefringent particles and anthracotic pigment
(H & E × 400 Magnification)

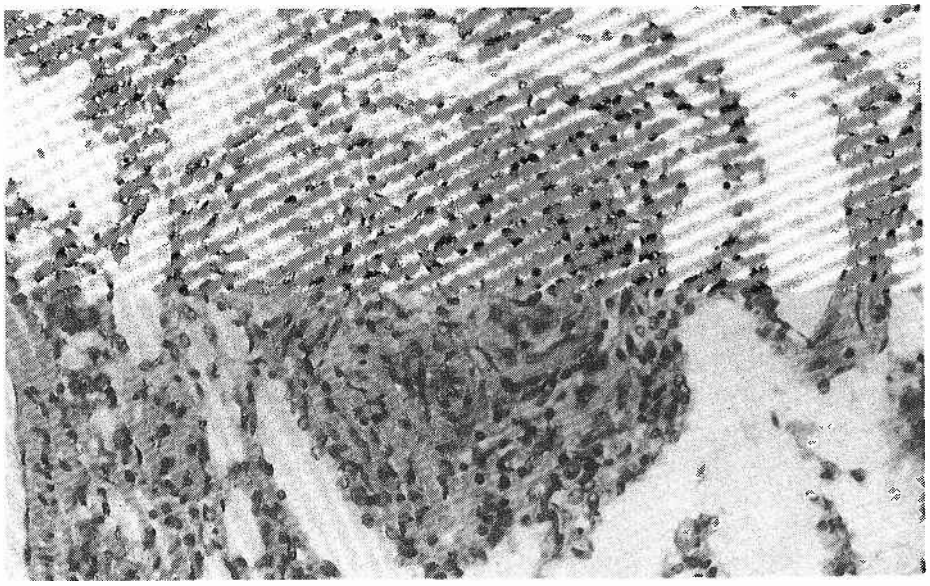

Figure 6
Earlier Lesion:
Alveolar septa with small collections of macrophages containing birefringent particles and fibroblasts
(H & E × 200 Magnification)

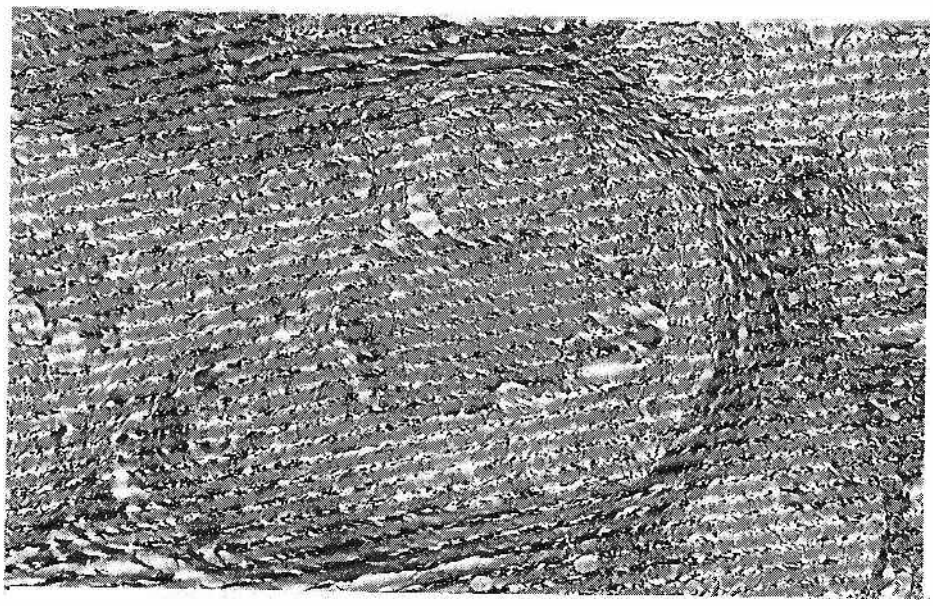

Figure 7
Section of Lung
Large nodules with central caseous necrosis and multinucleate giant cells. Perihery shows small silicotic nodules with stellate fibrosis and fibrosis of alveolar septa.
(H & E × 100 Magnification)

CASE 2

Case 2 worked together with Case 1 in the same factory from July 1971 until January 1981. She died at age of 49 years. The cause of death as recorded in her death certificate was chronic obstructive lung disease and relapsed tuberculosis.

In 1976, her annual miniature Chest X-ray appeared normal (fig 8). In July 1977, opacities were noted in the right upper zone (fig 9). Although sputum smears and

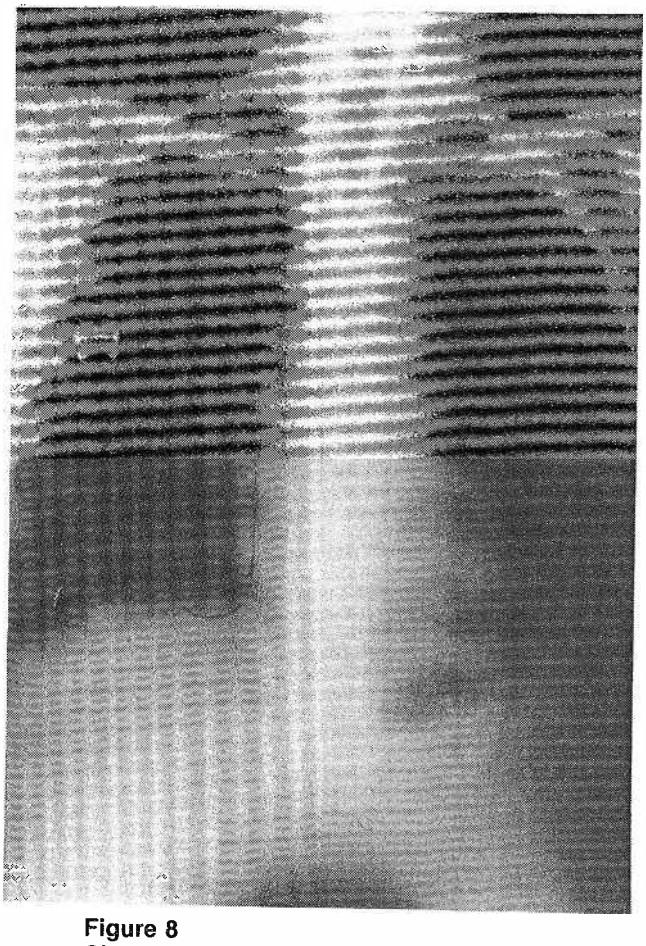

Figure 8
Chest X-ray of Case 2 in 1976 showed no abnormalities.

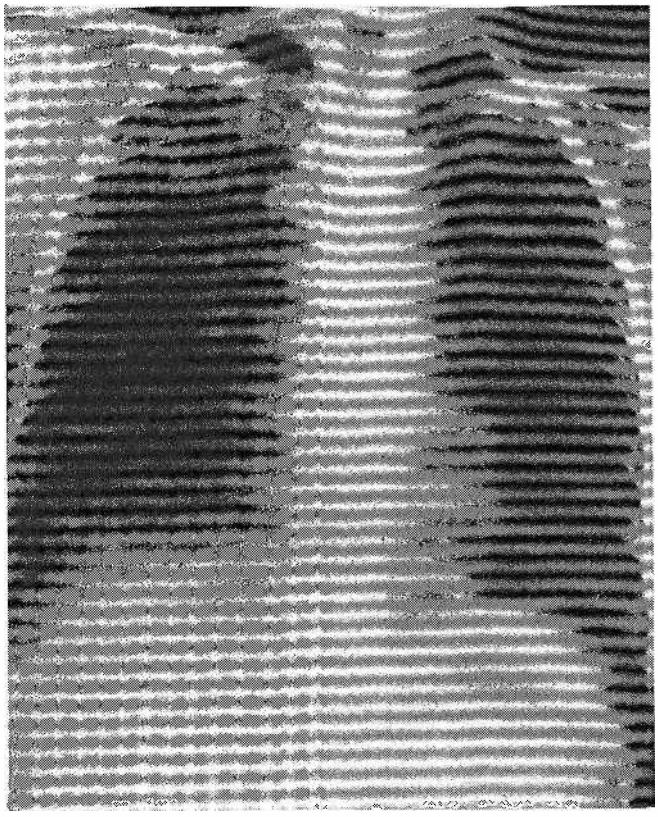

Figure 9
Chest X-ray of Case 2 in Jul 1977 showing opacities in right upper zone.

cultures were negative, she was treated for pulmonary tuberculosis. Two years later, in June 1979 (fig 10), the opacities on Chest X-ray had increased, involving both upper and mid zones bilaterally. Sputum smears and cultures were still negative. She was advised to have a lung biopsy but did not consent to it.

In May 1980, she developed a massive right pneumothorax (fig 11) and was hospitalised for the first time. Following treatment, the lung re-expanded. Bilateral opacities on her Chest X-ray were noted. A diagnosis of relapsed pulmonary tubereculosis was made and second line antituberculous treatment started. She developed further episodes of recurrent pneumothorax in June, July and October 1980 (fig 12). A bronchoscopy done in July 1980 did not reveal any lung tumour.

Despite antituberculous treatment her condition progressively deteriorated. Acid fast bacilli were never isolated from her sputum smears and cultures.

During her last hospital admission in October 1980, her lung function indicated a restrictive pattern. Blood gas estimation showed that she was in respiratory failure at that time:

	Observed	Predicted
Vital capacity (L)	0.53	2.41
Total Lung Capacity (L)	3.67	0.91
FEV_1 (L)	0.48	2.09
FEV_1/FVC (%)	96%	> 70%
DLCO (ml/min/mm Hg)	4.1	12.4
pH	7.423	—
p CO_2 (mm Hg)	33.6	—
p O_2 (mm Hg)	52.1	—

Her Chest X-ray on 20 October 1980 showed confluent shadows in both upper zones consistent with progressive massive fibrosis from advanced silicosis. It was classified as category 3/3 pq A (10) (fig 13).

She defaulted further followup and died at home in April 1981.

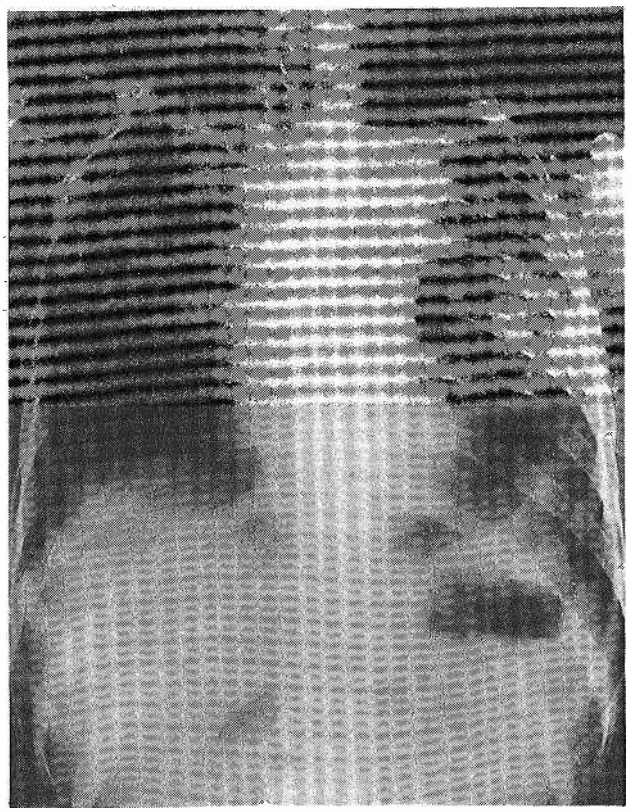

Figure 10
Chest X-ray of Case 2 in Jun 1979 showing opacities in both upper and mid zones.

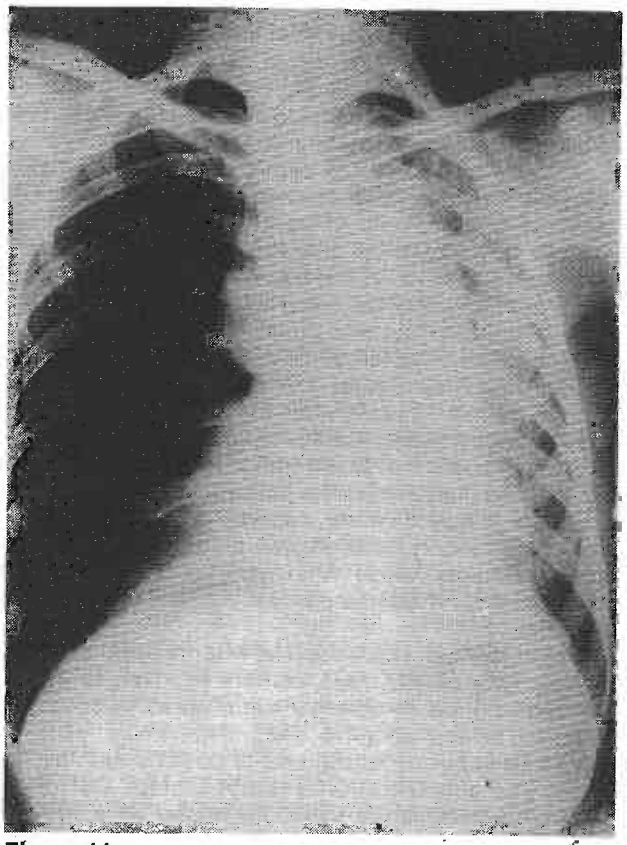

Figure 11
Chest X-ray of Case 2 in May 1980 showing a massive right pneumothorax.

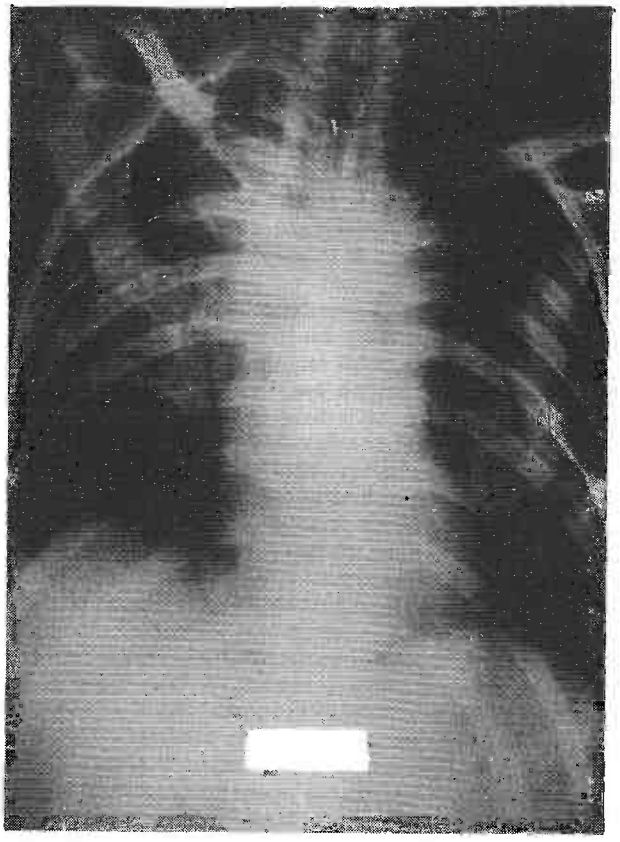

Figure 12
Chest X-ray of Case 2 in Oct 1980 showing bilateral pneumothorax.

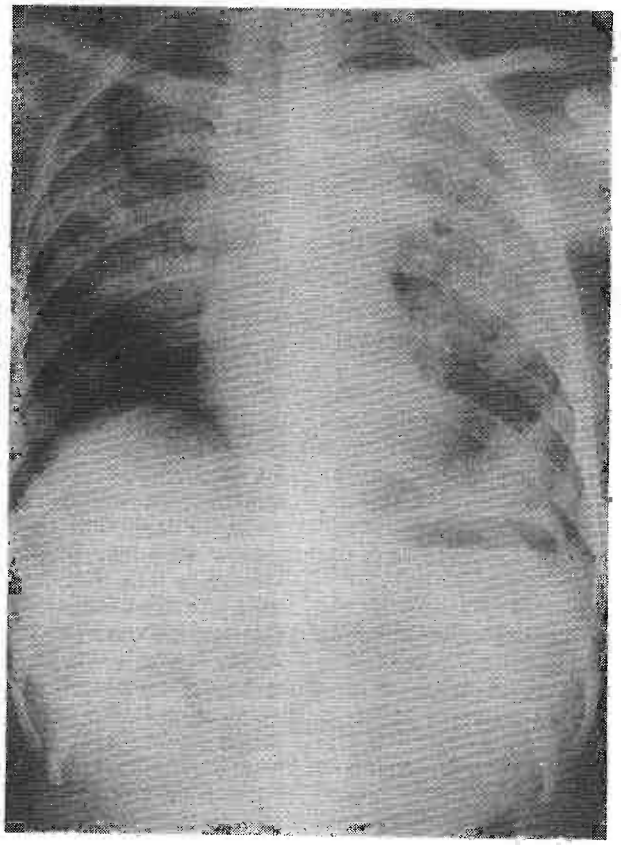

Figure 13
Chest X-ray of Case 2 in Oct 1980 showing progressive massive fibrosis.

OCCUPATIONAL EXPOSURE

Both cases worked in the same detergent factory which manufactured a domestic scouring powder. Their job was to mix soap powder with an abrasive agent (in the form of "fine white sand") by emptying paper bags of these two raw materials into the hopper of a mixing machine. The ratio of the raw material to be mixed was: seven bags of abrasive agent to one bag of soap powder. This work was done in a small mixing room.

Case 1 and Case 2 were the only workers involved in this process. They wore cloth masks, which were ineffective. The process of mixing and pouring was very dusty and there was no local exhaust ventilation or enclosure provided to reduce the dust emission. The room was small and poorly ventilated.

The mixing room was partitioned from a larger general packing and storage area where seven other workers worked. They poured the mixed powder into plastic containers and then capped, labelled and packed them into cartons.

Production of this scouring powder began in 1953 and, until 1978, the process was operating for three days per week. Subsequently, production was reduced to about three days per month. At the time of investigation by the DIH, production of the scouring powder had just ceased, pending transfer of the process overseas.

Analysis of the abrasive agent showed that it contained about 98% of free silica. This was consistent with the manufacturer's description of the abrasive powder which was reported as having 99.5% of free silica (cristobalite) content. Dust measurements were carried out under simulated conditions of the pouring and mixing process. The respirable airborne dust measured at the breathing zone of the worker was 1.1 mg/m^3. This grossly exceeded the threshold limit value of 0.15 mg/m^3 based on 98% cristobalite concentration (11).

Thus both Case 1 and Case 2 had been exposed to very high concentrations of silica dust.

Full-sized Chest X-ray examinations were carried out on the other workers in the general packing area who had also been exposed to the silica hazard, albeit considerably less so than Case 1 and Case 2. Out of the seven workers screened, two had radiological evidence suggestive of silicosis. These two Chest X-rays were graded as category 0/1 p (10). These two workers were among the longest exposed (one for 30 years and the other 16 years).

DISCUSSION

Silicosis may present in different ways depending on exposure and individual variations. Basically there are two forms: chronic and acute silicosis (12). Our two cases would appear to fall under the more common chronic group. Under this group there is a spectrum, ranging from the asymptomatic to the "accelerated" or rapidly progressive type. Case 1 appears to be of the asymptomatic type and Case 2 the accelerated type.

Almost all the previous report of silicosis in the abrasive detergent industry were acute silicosis cases (2-5). These workers presented with progressive dyspnoea, fever, cough, chest pain, malaise and weight loss after a short but heavy exposure to silica. The exposure time varied from a few weeks to a few years (usually in terms of months). The disease was rapidly progressive and uniformly fatal, death resulting from respiratory failure usually within a year of onset of symptoms. There was a high association with pulmonary tuberculosis. Radiologically, there was a diffuse bilateral haze spreading from the hilar region to the lower zones, mimicking pulmonary oedema. The basic histological lesions was an alveolar lipoproteinosis with interstitial fibrosis. Silicotic nodules were few or absent.

Though rarely described in the literature, there have undoubtedly been cases of chronic silicosis among workers in the abrasive detergent industry.

Ahlmark (6) described a case of chronic silicosis in a 43 year old man who developed "stage I" silicosis after 18 years of dusty work in a scouring powder factory. Production of the scouring powder was for two days per week. The silica content of the final product was 90% quartz. The workplace was poorly ventilated and the dust was considerable. No respirators were used. He ceased work when this diagnosis was made. Five years later the disease progressed to "stage II" and 10 years later to "stage III". There was no evidence of tuberculosis. No mention was made of any complication of pneumothorax. It was not stated whether he died of silicosis subsequently.

Unfortunately, in the reports of acute silicosis cases, not much mention was made of the other exposed workers as to whether they also developed silicosis and, if so, the clinical presentation in these workers.

Abdel Salam (13) reported the results of chest radiography of 162 male workers exposed to dust in the manufature of detergent and abrasive soap. The workers in the abrasive soap department were exposed to high concentrations of dust containing free silica (the concentration was not stated). Out of 162 workers screened, 23 were diagnosed as suspected silicosis (increased lung markings). One worker showed linear opacities.

This low prevalence of radiological findings was attributed to the rotation of workers through the different jobs in the factory. The exposure of each individual worker to silica dust was, on the average, equal to the sum of one to two months of exposure each year. Furthermore the turnover of workers was high: 94.4% of the workers examined had been exposed for six years or less and only 5.6% were exposed for longer than six years. Three of the workers of this plant died of advanced silicosis or silicotuberculosis within three years prior to the study. Six other workers were off work at the time of the study. One of them was in hospital with silicotuberculosis.

Why did our two cases not develop acute silicosis? For acute silicosis to develop, certain conditions are thought to be necessary (8). Basically there must have been a short but intense period of exposure to high concentrations of very fine particle size free silica dust.

Animal studies have shown that the fibrogenic activity of free silica administered by the intratracheal route increased significantly with decreasing particle size, and the "optimum fibrogenic size" appeared to be between 1 and 2 microns in diameter (8). Only one of these reports quantified the size of the free silica particles as "mostly" about 1 micron in diameter (4).

In our case, only 17% of the particles in the abrasive agent were less than 5 microns in diameter but only 5% were less than 2 microns. Thus the proportion of fine particles in our case was not very high.

Previous reports have always noted high concentrations of free silica in the scouring powder (ie the end product), ranging from 60 to 90% (3-6). Reported concentrations of free silica in the abrasive agent itself were even higher, usually 99% (6, 9). In our case, the free silica concentration was also high.

In addition to the particle size and concentration of silica dust, the frequency of exposure would also be relevant. Like the case described by Ahlmarl (6), our cases have had less intensive exposure to the silica dust compared to the acute silicosis cases previously reported. Case 1 was exposed for three days a week

from March 1973 to 1978 and thereafter till 1982 for three days a month. Case 2 had a more accelerated form of silicosis than Case 1 possibly because she had been exposed for three days per week for a longer period — from July 1971 to 1978.

Another factor to consider is the type of free silica involved. In our case, cristobalite was used, while in most of the reports of acute silicosis, quartz was used (8). Although lesions of acute silicosis have been produced in animals by experimental inhalation of mixtures of quartz and cristobalite, there do not appear to be any reports of acute silicosis from cristobalite exposure (8).

Observations of occupational exposure to cristobalite have come almost exclusively from workers exposed to calcined diatomite dust or diatomaceous earth. Naturally occurring diatomite contains mainly amorphous silica. When the latter is heated (calcined), a significant proportion is converted to crystalline silica in the form of cristobalite. The cristobalilte content varies from 21-60 percent (8). Cases of silicosis have been described which have certain cliical, radiological and pathological features different from the classical chronic silicosis associated with quartz exposure. Hence the term diatomite dust pneumoconiosis.

Vigliani and Mottura (14) described 13 cases of silicosis from two factories manufacturing filter candles. These workers were exposed to calcined diatomite dust which contained high concentrations of cristobalite. A rather rapid development and severe course of silicosis was noted. Two cases died, one from silicotuberculosis after seven years of exposure, the other from silicosis after eleven years. The other cases had advanced silicosis after being exposed for five years. The majority of workers showed radiological signs of silicosis after at least about four years of exposure. From the above description, the cases do not appear to have the features of acute silicosis. Some of them would seem to come under the category of an "accelerated" type of chronic silicosis.

Case 2 had recurrent spontaneous pneumothorax. This is not a common complication of silicosis in general. However, it is reported that there is an unusual tendency to spontaneous pneumothorax in silicosis cases due to calcined diatomite (cristobalite) exposure (8). In the report by Vigliani and Moturra (14), there were seven cases of advanced silicosis. Of these, three cases showed a spontaneous pneumothorax, one of which was bilateral.

Case 1 had pulmonary tuberculosis. Silicosis is the only penumoconiosis which predisposes to the development of tuberculosis and the latter is still the most common complication (8).

In calcined diatomite (cristobalite) exposure the earliest radiological abnormality consists of linear or round opacities in the upper and mid zones of the lung fields (8). These opacities usually do not exceed about 2 mm in diameter. They have low contrast with the surrounding tissues, rarely possessing the radiodensity of those due to 'nodular silicosis'. The 'typical nodular stage' is lacking, unlike in the classical silicosis due to quartz exposure, although the free silica content of the inhaled dust may be high (4). Thus, Case 1 did not have the typical nodular Chest X-ray picture that would have been expected in a worker with similar exposure to quartz dust.

In the more advanced stages of calcined diatomite (cristobalite) exposure, the opacities coalesce and form well circumscribed homogenous densities in the upper zones, usually bilaterally (8). Case 2's Chest X-ray (fig 13) is consistent with the above description.

Enlargement or egg shell calcification of hilar lymph nodes is not seen in cristobalite exposure (8). Both our cases did not show radiological evidence of such hilar lymph node involvement.

Case 1 showed numerous silicotic nodules with conglomerate lesions, interstitial fibrosis and scar emphysema, besides the features of complicating tuberculosis. The nodules had wide peripheral reactive zones which were consistent with progression of the silicotic process and the presence of tuberculous infection (15). There was an associated stellate fibrosis around the nodules, which is known to occur in tuberculosis.

Unlike Case 1, the few reports of diatomite dust pneumoconiosis (8, 14) have described lung changes consisting of diffuse interstitial fibrosis and irregular focal fibrosis without the typical silicotic nodules. However, King et al (16), in comparing the action of different forms of pure silica on the lungs of rats, showed that pure cristobalite (99%) induced slightly more rapid and severe changes than quartz, and produced a significant number of large stellate, partly cellular nodules which rapidly progressed to acellular confluent fibrosis with some degree of emphysema. This was in contrast to the focal fibrosis induced by amorphous silica.

Therefore, the more widespread diffuse fibrosis and relative absence of typical silicotic nodules seen in exposure to calcined diatomite could be attributed to the presence of significant amounts of amorphous silica. The latter is thought to produce a unique pathological disturbance in the form of diffuse mural thickening and consequent alveolar stasis (15). This in turn inhibits the clearing of fibrogenic silica (cristobalite) from most of the air spaces and prevents its focalisation into nodules.

CONCLUSION

Prognosis wise, life expectancy in silicosis due to cristobalite is rarely shortened and cor pulmonale is probably exceptional (8). Case 1 appeared to be relatively well despite developing pulmonary tuberculosis. In contrast, Case 2 died from cor pulmonale as a result of "accelerated" silicosis and recurrent pneumothorax.

Although the factory has ceased production of the scouring powder, the workers who were previously exposed could develop silicosis in the years to come. Case 1 and the other seven previously exposed workers would be followed up by the DIH with yearly full size Chest X-rays. In addition, an attempt would be made to trace workers who had worked in the mixing room between 1953 and 1973, ie, before the two cases joined the company.

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