British Journal of Industrial Medicine 1983;**40**:22–27

Mica pneumoconiosis

D DAVIES AND R COTTON

From City Hospital, Nottingham NG5 1PB, UK

ABSTRACT Two men developed pneumoconiosis after grinding and packing powdered mica. The disease was characterised by progressive dyspnoea, a restrictive impairment of ventilation, a reduced transfer factor, and hypoxaemia. Radiographs showed widespread fine nodular and linear shadows. Progression occurred after cessation of exposure, but this was much more pronounced in the man who died from coronary artery disease. Postmortem examination showed widespread fine fibrosis and nodules measuring up to 1·5 cm in diameter, all related to the deposition of doubly refractile crystals. Mineral formed over 9% of dry tissue weight, and electron microscopy and *x*-ray analysis showed it to be muscovite. Other minerals were not found.

Mica is the name given to a group of rock-forming minerals that are complex silicates of aluminium with either alkaline metals or with iron and magnesium. They are distinguished by the perfection with which they split into thin sheets and by a vitreous pearly lustre. Muscovite is a clear and colourless potassium mica that was formerly used for glazing windows and is still used in lamp chimneys and doors of stoves. In powdered form it is used in manufacturing paint and wallpaper and as a lubricant. It is used also in the rubber industry in the same way as talc to prevent sticking.

Doubt has been expressed as to whether mica produces pneumoconiosis.[1] This is because mica often occurs in association with granite and other rocks with a high quartz content, and when the crude material is crushed and milled there may be considerable exposure to silica. Some of the reported cases had also been exposed to other dusts. Seven cases of pneumoconiosis, however, were reported among 79 millers of mica which was considered not to contain free silica,[2] and a muscovite grinder developed a disease similar to asbestosis.[3] Fibrotic disease was found in a man where *x*-ray diffraction showed only biotite, another form of mica.[4] Pimentel and Menezes[5] reported a mica grinder and packer with progressive disease and extensive alveolar wall thickening. Plate-like crystals were found in the lung, and these were shown by histochemistry and *x*-ray diffraction to be muscovite. This patient also had liver granulomas containing mica.

We report two further cases of mica pneumoconiosis in men grinding muscovite.

Received 8 February 1982
Accepted 23 February 1982

Process

Mica for the factory, in the form of muscovite, was imported from southern Africa and some from India. It arrived in sacks as a mixture of lumps and powder and was tipped into a hopper and fed into a crusher. It then passed through grinding machines and ended up as a very fine dry powder—99·5% of the particles were below 20 μ and 10% below 2·5 μ in diameter. Up to about 1967 the only extractor fan was over the crusher. Later, others were put over the grinding machines but did not appear to be very effective. Until about 1969 simple gauze face masks were available but dust respirators were available thereafter.

Case 1

This man, born in 1910, did various non-dusty jobs until 1957 when he started to work as a grinder. After about six years he became a foreman and usually worked for 12 hours on six days a week. Serial chest radiographs were taken and they were normal up to 1960. By 1963 minimal fine nodulation was visible in the right lower and left mid-zones. It increased and was easily visible by 1967 when he was told about it. Application for certification was rejected by a pneumoconiosis medical panel, and he continued with the same work.

When first seen in 1973 he complained of gradually increasing shortness of breath for four years so that he was unable to keep up with others of his age when walking on the level. Some cough, non-purulent sputum, and slight wheezing had been present for three years. He had smoked 10 cigarettes a day from the age of 16 until 1950.

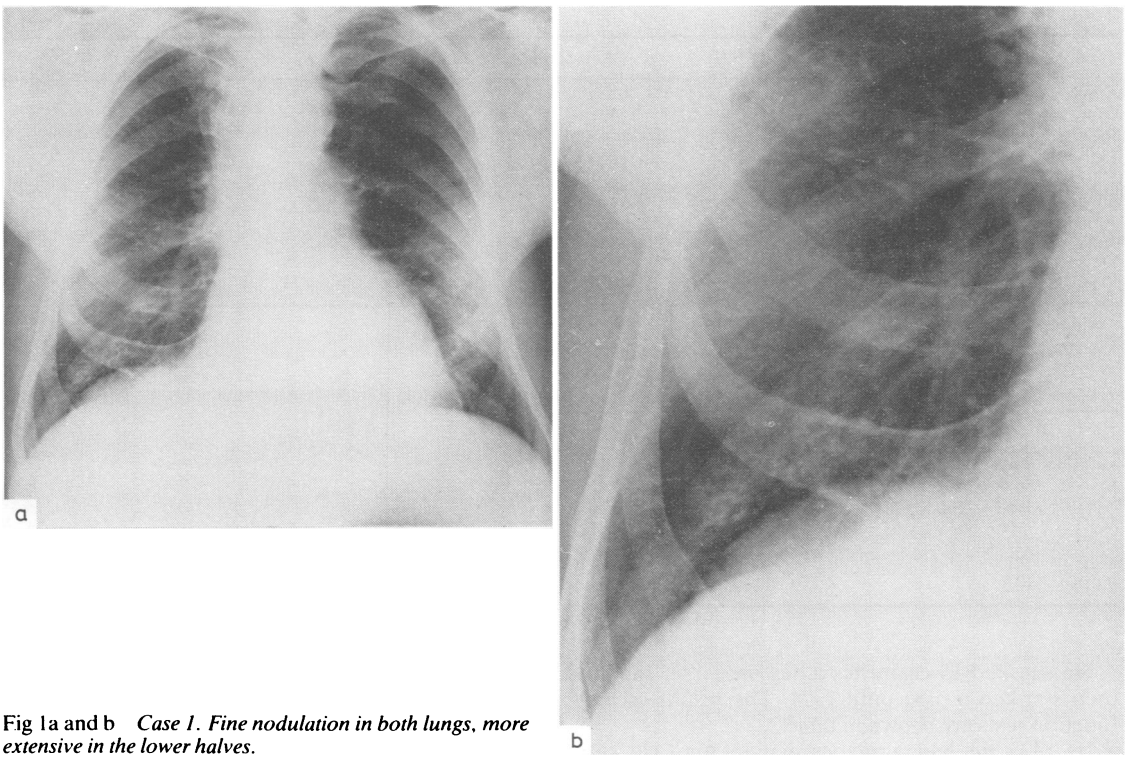

Fig 1a and b *Case 1. Fine nodulation in both lungs, more extensive in the lower halves.*

On examination his general condition was good. There was no clubbing of the fingers. He became breathless quite easily, and there were a moderate number of crackles at the lung bases. He reapplied for certification, and a disability assessment of 20% was made.

He was made redundant in January 1974 and has not worked since. Dyspnoea has increased, and he now has to stop after walking 100 metres. Some cough, sputum, and mild wheezing persist, and since 1977 he has occasionally coughed up blood. Clubbing has not developed but the crackles have become somewhat more extensive. Fine nodulation, most obvious in the lower halves of the lungs, extended up to 1978 (fig 1), but there has been no detectable change since. Disability assessment was increased to 40% in 1977 and so remains.

In 1967 he was found to be hypertensive, and he has been treated with diuretics and, at various times, with methyldopa and debrisoquine.

LABORATORY FINDINGS

Between 1967 and 1978 the ESR was persistently raised between 19 and 32 mm in one hour (Westergren); in 1980 it was 11 mm. The only autoantibody found in his blood has been to gastric parietal cells on two occasions. Mineral crystals have not been seen in his sputum. The lung function tests are shown in table 1. He also has moderate arterial hypoxaemia that increases on exercise.

Case 2

This man, born in 1917, was not exposed to dust until 1951 when he began to work on a mica grinding machine. He did this until 1959 when he moved to non-dusty work. In 1962 he returned to the mica factory and spent the next two years packing powdered mica from a hopper into bags. He was not exposed to dust thereafter. A routine chest radiograph in 1958 strongly suggested the presence of fine generalised nodulation. A further film in 1962 showed no change but in 1964 the nodules were more profuse. An application to a pneumoconiosis medical panel for certification was rejected. At the time he had no significant symptoms except for one small haemoptysis. By 1972 he was complaining of mild shortness of breath and he had a slight dry cough. He was certified in that year, and a 20% disability assessment was made.

Table 1 *Lung function tests in case 1*

Date		*FVC (l)*	*FEV_1 (l)*	*PEFR (l/min)*	*TLC (l)*	*RV (l)*	*DCO* (%)*
May	1958	3·8					
June	1962	3·9		440			
February	1967	3·95	3·1				
August	1969	3·7	2·8				
June	1973	3·6	2·6	470			
July	1976	3·2	2·6	410			58
September	1977	3·3	2·5	415	5·56	2·26	57
June	1978	3·45	2·5	435	5·65	2·20	56
June	1980	3·1	2·25	425	5·63	2·53	49
December	1981	2·7	2·0	380	4·98	2·28	55
Predicted	1981	4·05	2·8	510	6·85	2·54	100

*Transfer factor, single breath.

Table 2 *Ventilatory tests in case 2*

Date		*FVC (l)*	*FEV_1 (l)*	*PEFR (l/min)*
May	1957	4·35		
May	1958	4·05		
April	1962			550
May	1966	4·4	3·45	
February	1975	3·7	2·75	
May	1975	3·2	2·5	410
April	1976	2·5	2·15	380

He smoked 15 cigarettes a day until 1965, and he then smoked a pipe until 1974. The results of lung function tests are shown in table 2.

In 1973 he had a myocardial infarct. He subsequently had several more episodes of severe chest pain, attributed to further infarcts, and he began to show evidence of heart failure. His eyesight deteriorated because of retinitis pigmentosa. Clubbing of the fingers and persistent basal lung crackles were first noted in 1975 and nodular and linear shadows continued to increase (fig 2). He took diuretics continuously from 1973, and he died from heart failure in January 1977.

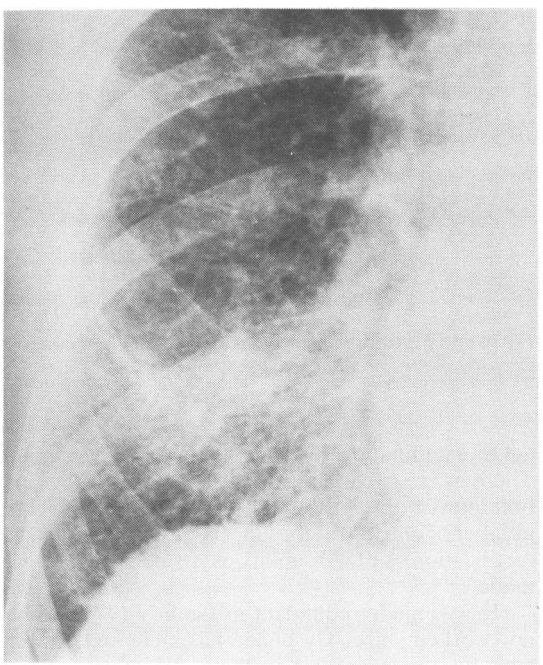

Fig 2 *Case 2. In 1976 there are extensive fine nodular and linear shadows, especially in the lower half. Larger nodules are just visible in the lower zones.*

POSTMORTEM EXAMINATION

Heart

Death was largely due to severe ischaemic heart disease with extensive fibrous replacement of the myocardium in the posterior and anterior walls of the left ventricle. There was severe coronary artery atheroma with old occlusions of the right coronary artery 2 cm from its origin and of the anterior descending branch of the left coronary artery, also 2 cm from its origin. There was no evidence of recent infarction. The heart weighed 380 g and slight right ventricular hypertrophy was apparent.

Lungs

Loculated bilateral pleural effusions each of 200 ml were present in addition to dense bilateral fibrous adhesions. The lungs felt nodular and were inflated with formalin and fixed before incision. After fixation, dissection showed the presence of pulmonary emboli and infarcts related to subsegmental arterial branches and associated with deep vein thrombosis in the legs but no atrial thrombus was found. Before inflation the lungs weighed 1040 g (right) and 840 g (left).

Section of the lungs showed similar parenchymal changes in both with ill-defined fibrous nodules up to 1·5 cm in diameter, more pronounced in the lower lobes and widespread fine fibrosis in all lobes but dominantly basal (fig 3). There was considerable focal

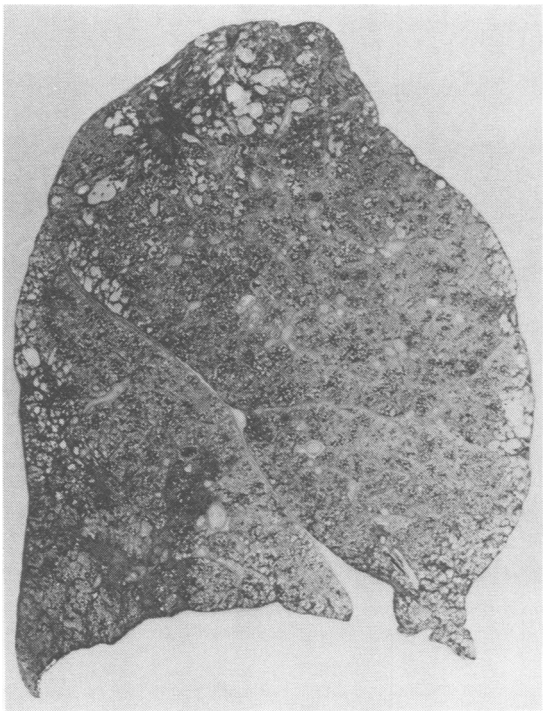

Fig 3 *Case 2. Whole lung paper mounted section showing nodules and foci of finer fibrosis.*

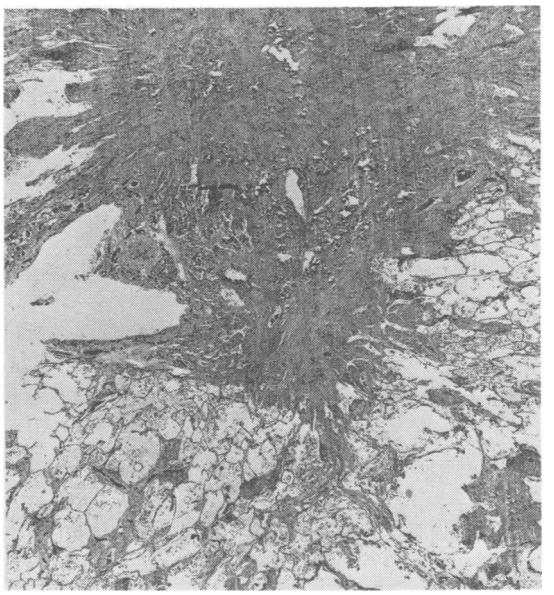

Fig 4 *Case 2. A large fibrous nodule with irregular radiating margin where the lung parenchyma shows alveolar dilatation (H & E × 14·7.)*

emphysema directly related to the fibrosis, but the major bronchi showed no significant abnormality and, in particular, no evidence of chronic bronchitis. Carbon pigmentation was relatively slight. Figure 4 shows a larger nodule and fig 5 the widespread fine fibrosis.

Histological examination of the fibrotic foci, whether nodules or more diffuse, showed collagen intimately related to clusters of doubly refractile crystalline material (fig 6). In places where the changes were less severe the fibrosis could be identified as starting in relationship to heavy concentrations of the crystalline material in the interstitial tissue of the alveolar septa. Some histiocytes and foreign body giant cells were seen but there were no granulomas. There appeared to be a direct correlation between the concentration of crystalline material and the extent of the fibrosis.

Other organs

The only other abnormalities were of generalised arterial atherosclerosis and changes due to congestive cardiac failure.

MINERALOLOGY

Samples of upper lobe and lower lobe were examined at the department of mineral exploitation, University College, Cardiff. Dust contained in fractions of the tissue was extracted using a procedure developed for the recovery of inorganic particular matter from lung tissue. Wet tissue was dried to a constant weight before preparation and the dust from the two samples examined by a combination of *x*-ray diffraction and electron microscopical techniques.

The amount of dry tissue from the two samples was 104 mg and 103 mg respectively with dust residues of 11·6 and 7·0 mg—that is, very large quantities of mineral matter, averaging 9·3% of the dry tissue weight. Electron microscopy showed a mineral consisting of thin flat plates varying in size from more than 50 μm to less than 1 μm (fig 7).

Individual particle analysis was carried out by an energy dispersive *x*-ray analysis device fitted to the electron microscope, and this showed material of one composition only, containing potassium, aluminium, and silicon, together with a small amount of titanium in the dust. Comparison with published results for the mineral muscovite shows similar findings (table 3). This indicated the dust to be practically pure muscovite and confirmation of this was forthcoming by examining the dust residues by *x*-ray diffraction using a diffractometer and comparing the results with those obtained from a pure sample of muscovite. Identical *x*-ray reflection occurred in all three samples at 9°, 18°, and 27°.

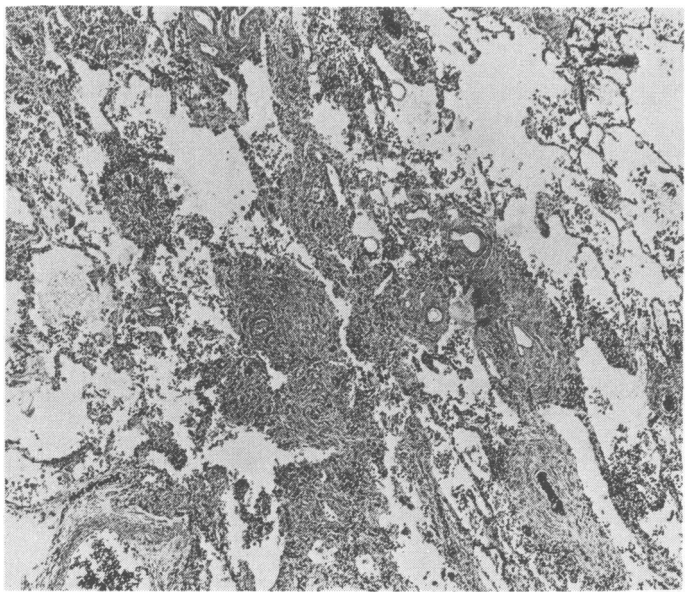

Fig 5 *Case 2. Multiple small fibrotic lesions forming a cluster, each centred around a vessel. (H & E × 50.)*

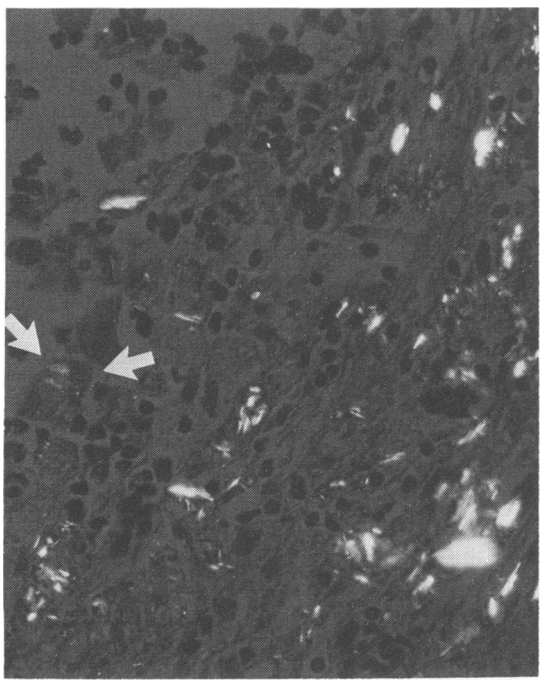

Fig 6 *Case 2. When viewed under polarised light large masses of crystalline material are shown in spaces between collagen fibres and also within histiocytes (arrows). (H & E (polarised) × 490.)*

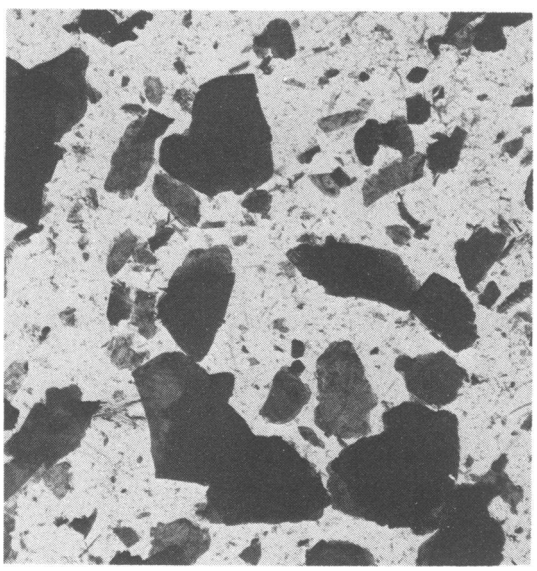

Fig 7 *Case 2. Electron micrograph on nuclepore filter showing thin flat plates of mineral. (H & E × 2600.)*

Table 3 *Analysis of mineral residue from two lung samples and the published results for muscovite (oxide weight percentage)*

	SiO_2	Na_2O	MgO	Al_2O_3	K_2O	TiO_2	FeO
Sample 1	45·8	0·5	1·4	32·6	10·5	0·3	4·1
Sample 2	46·9	0·6	1·0	32·8	10·1	0·3	3·2
Muscovite	45·2	0·6	0·1	36·9	10·1	0·01	0·1

Discussion

The first patient showed radiographic evidence of pneumoconiosis after six years and exposure continued for a further 10 years. He has not developed clubbing, but crackles may be heard over the lungs. He has a moderate restrictive impairment of ventilation, a low total lung capacity, a fairly severe reduction in transfer factor, and he develops increasing hypoxaemia on exertion. His disability is quite severe and increasing. Radiographically the changes fall into category 2 simple pneumoconiosis, and there has been only a little progression since dust exposure ceased. There is no evidence that hypertension or the drugs used in its treatment have contributed to his disability.

The second patient was initially exposed for eight years, and the first mild radiographic changes were detectable after seven years but did not progress over the next four years. He then had two more years of exposure and progression was clearly evident by its end. Deterioration continued after dust exposure ceased, reaching category 3 simple pneumoconiosis, and this was matched by the development of fairly severe restrictive impairment of ventilation. Towards the end coronary artery disease and heart failure contributed to his disability.

At necropsy his lungs were found to contain quantities of almost pure mica but no other mineral substances. There was fine interstitial fibrosis related to the deposits of mica but also larger fibrous nodules measuring up to 1·5 cm in diameter. He did not have rheumatoid arthritis but tests for rheumatoid factor were not done.

Mica therefore produces a disease characterised by a restrictive impairment of ventilation, a reduced transfer factor, and hypoxaemia at rest or on exercise. Clubbing of the fingers may develop and crackles are present over the lungs. Radiographs show fine nodular and linear shadows. Clinical and radiographic deterioration usually continues after dust exposure has ceased. The disease has many features in common with asbestosis but case 2 also developed larger fibrotic nodules that can, because of their size (larger than 1 cm), be classified as progressive massive fibrosis. This is seldom seen in asbestosis.

We are very grateful to Dr F D Pooley of University College, Cardiff, for carrying out the mineral analysis.

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