Thorax 1983;**38**:275–278

Silicosis in surface coalmine drillers

DANIEL E BANKS, MICHAEL A BAUER, ROBERT M CASTELLAN, N LEROY LAPP

From the Division of Respiratory Disease Studies, National Institute for Occupational Safety and Health, and the Pulmonary Diseases Section, West Virginia University School of Medicine, Morgantown, West Virginia, USA

ABSTRACT Surface coalminers are generally thought to be at minimal risk of developing pneumoconiosis. Biopsy-proved silicoproteinosis was found in a 34-year-old surface coalmine driller, and two of nine other drill crew members who worked for the same company had chest radiographic findings compatible with simple silicosis. Reanalysis of data from a previous United States Public Health Service survey of surface coalminers, after exclusion of those with underground mining experience, showed that 38% of the cases of pneumoconiosis occurred in drill crew members, a group comprising only 11% of the study population. On the basis of these data surface coalmine drillers appear to have an increased risk of developing occupational lung disease.

The development of silicosis among underground coalminers has been well documented, particularly among roof bolters and transportation workers. We report a case of acute silicosis in a surface coalmine drill operator. An important feature is the occurrence of rapidly fatal silicosis in a miner with no underground experience who had not worked in enclosed spaces.

The recognition of acute silicosis in the index case prompted a medical survey of nine other current and former drill crew workers who worked for the same company. Two of these nine had simple silicosis. In addition, we reanalysed data from a previous survey of the respiratory health of surface coalminers,[1] which concluded, "current surface mining techniques are not likely to lead to the development of pneumoconiosis." We found that drill crew workers have a disproportionately high risk of developing pneumoconiosis.

Case report

A 34-year-old white man was admitted to hospital in April 1979 with a six-month history of progressive dyspnoea, dry cough, weight loss of 13 kg, and Raynaud's phenomenon. He had operated a rotary drill using a dry drilling technique at a surface coal mine for the preceding five years. Previously he had been in good health and a routine examination and chest radiograph in February 1977 both gave normal results.

Physical examination showed an afebrile, slender man, breathing 30 times per minute at rest. His blood pressure was 110/84 mm Hg and his pulse rate was 88 beats per minute and regular. There were coarse crackles at both lung bases, healed ulceration on several fingertips, and sclerodactyly.

The haemoglobin concentration, packed cell volume, white blood cell count and differential, and serum electrolyte concentrations were normal. The blood urea concentration was 7·7 mmol/l (20 mg/100 ml) and the serum creatinine concentration 130 μmol/l (1·5 mg/100 ml). Urine analysis showed microscopic haematuria, grade 2+ proteinuria, and occasional hyaline casts. The serum albumin concentration was 21 g/l, and the C3 component of complement was 1·66 g/l (normal 0·88–1·77 g/l). Cryoglobulins, antinuclear antibodies, and rheumatoid factor were not detected but circulating immune complexes[2] were present. An intermediate tuberculin (PPD) skin test produced no reaction, but a mumps skin test gave a positive response. The urinary protein excretion was 0·75 g/24 h and creatinine clearance was 62 ml/min.

The forced vital capacity (FVC) was 3·47 l (63% of predicted[3]). The single-breath transfer factor was 6·32 ml/min/mm Hg (2·11 mmol min^{-1} kPa^{-1}: 18% of predicted[4]). Analysis of arterial blood gases

Address for reprint requests: Dr Daniel E Banks, Department of Medicine, Section of Pulmonary Diseases, Tulane University School of Medicine, 1430 Tulane Avenue, New Orleans, Louisiana 70112.

Accepted 7 December 1982

obtained at rest in room air showed that the pH was 7·44, arterial carbon dioxide tension ($PaCO_2$) 32 mm Hg (4·3 kPa) and arterial oxygen tension (PaO_2) 63 mm Hg (8·4 kPa). A chest radiograph showed a hazy, bilateral basal alveolar filling pattern (fig 1).

An open lung biopsy showed grossly distorted architecture with extensive interstitial fibrosis and scattered foci of small non-caseating granulomas. Polarised light microscopic examination showed many angular, weakly birefringent particles in the areas of fibrosis. Scanning electron microscopy in conjunction with back-scattered electron imaging and energy dispersive x-ray analysis showed the particles to be quartz. Some of the granulomas were necrotic and packed with this crystalline material. Many alveolar spaces were filled with periodic acid-Schiff (PAS) staining material, compatible with silicoproteinosis[5] (fig 2).

No treatment is recognised as effective in silicoproteinosis, but because circulating immune complexes were present the patient was treated with daily oral cyclophosphamide (2 mg/kg). Despite eight months of treatment he became more dyspnoeic and lost an additional 12 kg in weight. A second radiograph, taken two months after treatment had been discontinued, showed progressive massive fibrosis (fig 3). His FVC was 1·77 l (33% of predicted) and he was unable to perform a single-breath diffusing capacity manoeuvre owing to breathlessness. Antinuclear antibodies were observed in a homogeneous and peripheral pattern at a titre of 1/640 after cyclophosphamide had been discontinued. The patient died 26 months after his initial presentation. No necropsy was permitted.

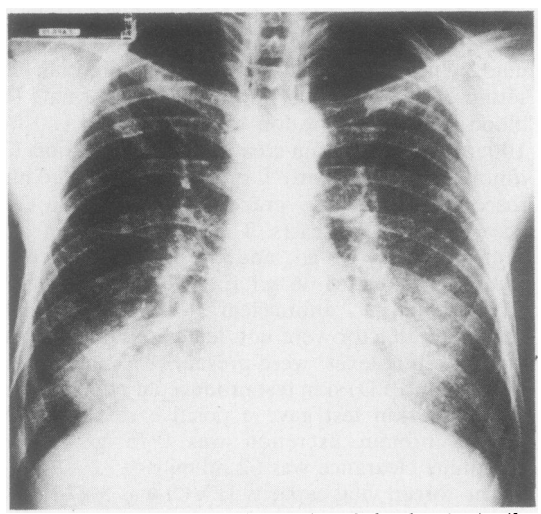

Fig 1 *Posterioranterior chest radiograph taken in April 1979 showing an alveolar filling pattern in both lower lung zones.*

Findings in other drill crew workers

Because acute silicosis implies exposure to very high concentrations of respirable free silica, we examined nine of the 10 other current and former drill crew workers who had worked for the same company at the same mine site for one or more years during 1972–80. Chest radiographs were independently evaluated by three "B" readers proficient in the 1971 ILO U/C classification for pneumoconiosis.[6] When at least two of the readers interpreted the chest radiograph as showing category 1/0 or greater profusion for small rounded opacities, it was considered positive. Two additional cases of silicosis were found in this group of workers. Two men, aged 28 and 31 years, who had worked as drill operators for

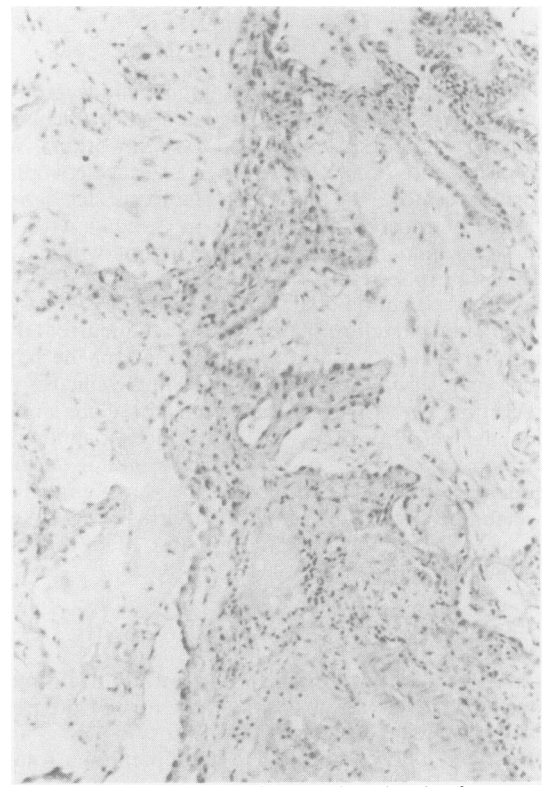

Fig 2 *Photomicrograph showing disordered pulmonary parenchyma, interstitial thickening with inflammatory cell infiltration, and filling of the alveolar spaces with relatively acellular eosinophilic material, which gave a positive reaction when stained with periodic acid-Schiff reagent. (Haematoxylin and eosin, × 50.)*

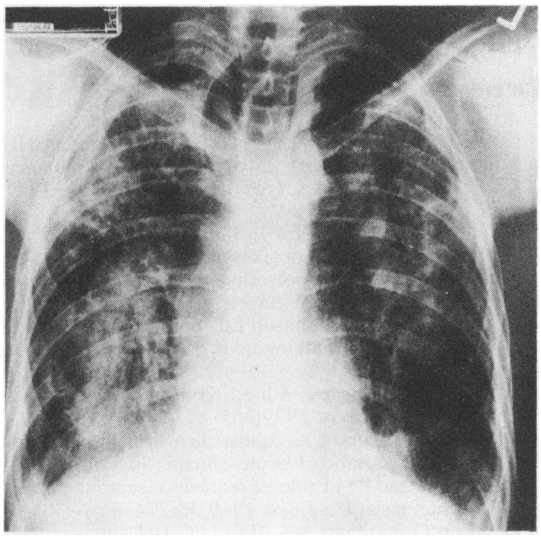

Fig 3 *Posterioranterior chest radiograph taken in June 1980 showing conglomerate mass lesions in both lungs and loss of volume in the right upper lobe with tracheal distortion, blunting of both costovertebral angles, and bilateral pleural thickening.*

four and six years, had chest radiographs showing simple silicosis (both category 1/2 q) with normal lung function.

We reviewed all personal dust measurements collected for the years 1972–80 in accordance with Mine Safety and Health Administration (MSHA) regulations. Of 20 dust samples taken for the three men with silicosis, only one was above the allowable respirable coal dust limit of 2·0 mg/m³. Three samples were collected for the driller with acute silicosis, the highest being 0·4 mg/m³. Silica content was not measured in any of the dust samples collected. No core samples were available for review, but much of the overburden rock in the area was sandstone, which is likely to have a high free silica content.

Review of previous survey

The occurrence of two further cases of pneumoconiosis among the nine other crew members we examined led us to question whether drill crew workers are at risk of developing silicosis throughout the surface coalmine industry. We therefore re-evaluated data collected during a 1972–3 United States Public Health Service respiratory health survey of surface coalminers at eight surface coal mines, located in Pennsylvania, Ohio, Indiana, Illinois, Kentucky, and Alabama.[1] Radiographs had been interpreted on the basis of the UICC/Cincinnati classification.[7] Fifty-nine of 1438 (4%)

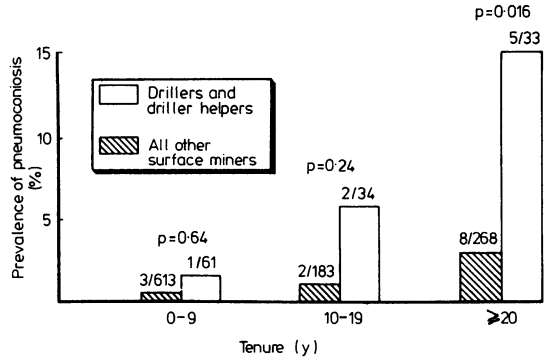

Fig 4 *Prevalence of pneumoconiosis by job and tenure as determined by the 1972–3 US Public Health Service survey of surface coalminers[1] (p values determined by Fisher's exact test, two tail).*

surface miners had radiographic evidence of pneumoconiosis. Of these 59, 38 had previous underground mining experience. On the basis of these results the authors concluded that "employment in surface mining was not likely to cause the development" of coalworkers' pneumoconiosis.

For our reanalysis we excluded data from 246 miners with underground mining exposure. Of 1192 surface miners with no underground mining experience, 21 had simple silicosis (category 1/0 or greater profusion of rounded opacities). None had progressive massive fibrosis. Six per cent (eight of 128) of the drill crew members had pneumoconiosis compared with 1% (13 of 1064) of all other surface coalminers ($p < 0·001$ according to the χ^2 text) and the increase was greatest in those exposed for the longest periods (fig 4). These figures may underestimate the true prevalence because only current workers were surveyed and those with occupationally induced respiratory disease may have left the mining industry for health reasons.

Discussion

To uncover coal seams for surface mining overlying rock strata have to be removed. The first stage consists of drilling into the earth's surface with large mobile drilling rigs, the drill operator sitting in a cab in close proximity to the drill. After holes have been drilled, explosive charges are inserted and detonated. Removal of the overburden is then accomplished by earth-moving heavy equipment. At the mine site the drill crew (including drill operators and driller helpers) often work well ahead of and apart from workers responsible for removing the overburden and coalmining. Drill crew members are at little risk of developing coalworkers' pneumoconiosis, but because of their exposure to dust generated by

drilling through silicaceous rock they are at risk of developing silicosis.

During the years 1972–80 personal respirable dust measurements over a workshift were required for each surface miner. If the measurement exceeded half of the respirable coal dust limit further sampling was necessary; otherwise, yearly sampling was considered adequate. In surface coal-mining operations the silica content of the dust samples was often not measured. If the overburden were entirely quartz the respirable dust limit would be 0·1 mg/m^3, and dust levels of 2·0 mg/m^3 (the allowable limit for mixed coal dust) would be grossly excessive.[8] The development of pneumoconiosis in these subjects suggests that the annual personal dust sample failed to reflect true exposure to silica. To estimate exposure more accurately the Mine Safety and Health Administration has recently replaced personal dust sampling once a year with work-site dust sampling every two months and determination of the quartz content of the dust (Code of Federal Regulation, vol 30, part 71).

In summary, the belief that only coalminers who work underground have an appreciable risk of developing pneumoconiosis needs to be modified. Our findings indicate that, just as roof bolters and transportation workers in underground coal mines are at risk of developing silicosis, so too are drill crew workers in surface coal mines.

We are grateful for the assistance of FHY Green, V Vallayathan, and J Tucker.

References

[1] Fairman RP, O'Brien RJ, Swecker S, Amandus HE, Shoub EP. Respiratory status of surface coal miners in the United States. *Arch Environ Health* 1977; **32**:211–5.

[2] Digeon M, Laver M, Riza J, Back JF. Detection of circulating immune complexes in human sera by simplified assays with polyethylene-glycol. *J Immunol Methods* 1977;**16**:165–83.

[3] Morris JF, Koski A, Johnson LC. Spirometric standards for healthy non-smoking adults. *Am Rev Respir Dis* 1971;**103**:57–67.

[4] Cotes J. *Lung function*. 4th ed. Oxford: Blackwell Scientific Publications, 1979;365–87.

[5] Buechner HA, Ansari A. Acute silico-proteinosis, a new pathologic variant of acute silicosis in sandblasters, characterized by histologic features resembling alveolar proteinosis. *Dis Chest* 1969;**55**:274–84.

[6] Jacobson G, Lainhart WS. ILO U/C 1971 international classification of radiographs of pneumoconiosis. *Med Radiogr Photogr* 1972;**48(3)**:65–110.

[7] UICC Committee. UICC/Cincinnati classification of the radiographic appearances of pneumoconiosis. *Chest* 1970;**58**:57–67.

[8] American Conference of Governmental Industrial Hygienists. *Threshold limit values for chemical substances and physical agents in the workroom environment with intended changes for 1972*. Cincinnati: American Conference of Governmental Industrial Hygienists, 1972.