A case of silicosis and progressive massive fibrosis with uncertain occupational exposure to silica

**ABSTRACT**

A case of histologically diagnosed silicosis with progressive massive fibrosis (PMF) and low dust exposure is reported. Two experienced occupational medicine practitioners took a detailed exposure history on three occasions. The only reported dust exposure was indirectly from coal-carrying conveyor belts in a power station for 23 years, where he worked as a clerk. The chest radiographs showed features consistent with PMF in 1994, after only 11 years of indirect dust exposure. Two pathologists independently reported PMF. The case demonstrates that even when heavy exposure is thought to be required to produce an occupational disease, the source of exposure may be difficult to ascertain.

**INTRODUCTION**

Progressive massive fibrosis (PMF) describes confluent nodules in the lungs of dust and collagen fibrosis larger than 1 cm in diameter. Although these lesions are termed ‘progressive’, they are not always progressive. PMF is well known to occur with silicosis, but has also been described in association with coal workers’ pneumoconiosis, talcosis and asbestosis. Some of the industries in which the disease has been described are mining, foundries, ceramic factories, refractories and ore and stone crushing.

**Table 1. Occupational exposure history by calendar year, occupation and duration of exposure**

<table>
<thead>
<tr>
<th>Year*</th>
<th>Workplace/Occupation</th>
<th>Exposure</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>1979 – May 1980</td>
<td>Coal mine</td>
<td>Worked in kitchen, cooking. No dust exposure.</td>
<td>1 year, 5 months</td>
</tr>
<tr>
<td>Jun 1980 – Sep 1980</td>
<td>Coal mine</td>
<td>Worked at workshop, issuing spares. No dust exposure.</td>
<td>1 month</td>
</tr>
<tr>
<td>Oct 1980 – Apr 1981</td>
<td>Coal mine</td>
<td>Worked in kitchen, cooking. No dust exposure.</td>
<td>9 months</td>
</tr>
<tr>
<td>May 1981 – Dec 1982</td>
<td>Traffic department</td>
<td>Worked as a traffic officer. No dust exposure.</td>
<td>1 year, 7 months</td>
</tr>
<tr>
<td>Jan 1983 – Oct 1991</td>
<td>Power station</td>
<td>Worked as a filing clerk for 8 years. Duties involved filing office records. Indirect exposure to dust from a coal-carrying conveyor belt passing close to the office. Especially dusty on windy days. Trucks collected coal that had fallen off the conveyor belt and transported it to the coal yard.</td>
<td>9 years</td>
</tr>
<tr>
<td>Jan 1992 – Nov 1993</td>
<td>Power station</td>
<td>Worked as a forklift driver, transporting spares used in the power station. Spent most of the time just outside the stores. Coal dust exposure from coal-carrying conveyor belt.</td>
<td>2 years</td>
</tr>
<tr>
<td>Dec 1994 – Nov 2008</td>
<td>Power station</td>
<td>Worked mainly inside the stores, issuing tools. Worked outside the stores for almost 3 hours in a day. Indirect exposure to dust from coal-carrying conveyor belt passing in front of the stores. Especially dusty on windy days.</td>
<td>12 years</td>
</tr>
</tbody>
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* Patient could not recall exact years in every job.
Cumulative dust exposure is probably the most important factor in the pathogenesis of PMF. Although it usually develops after many years of dust exposure, certain job categories with exposure to high dust levels, such as drilling, shot blasting, moulding and furnace maintenance, are associated with development of advanced dust diseases after a short duration of exposure. PMF has been reported in South African foundries and, although some of these cases had short silica dust exposures, they were of high intensity. Souter et al. showed an increase in the attack rate of PMF in coal miners from 0.8% at a respirable dust concentration of 1.5 mg/m$^3$ to 5% at 6 mg/m$^3$.

We report a case with a histologically confirmed diagnosis of silicosis with PMF after ostensibly low level silica exposure from coal-carrying conveyor belts. Medical practitioners who see cases suggestive of PMF need to consider the diagnosis, even if exposure history does not reveal many years of high dust exposure.

**Case description**

**Background**

In 2007, a 49-year-old man presented to the occupational medicine clinic at the National Institute for Occupational Health with a complaint of progressively increasing dyspnoea for the past four years. The patient denied a history of cough, haemoptysis, night sweats or loss of weight. His medical history included one episode of pulmonary tuberculosis, which was treated in 1986. He had never smoked.

**Exposure history**

Two experienced occupational medicine practitioners elicited an exposure history on three separate occasions. The full service history is provided in Table 1. In summary, the patient reported 23 years of indirect dust exposure from coal-carrying conveyor belts at a power station, from 1983 to 2006. Most of his time was spent working as a filing clerk. It is notable that the radiologic features of large opacities were already present in 1994, after only 11 years of exposure. No historic dust measurements were available for the pertinent years of exposure.

The patient grew up in Limpopo province, an area not known for environmental silica dust exposure.

**Diagnosis**

Nothing remarkable was detected on physical examination. The chest radiographs were read according to the International Classification of Pneumoconioses, devised by the International Labour Organisation. Chest radiographs from 1994 (Figure 1) to 2006 showed bilateral upper zone large opacities and separate, discrete nodular changes. Over the years, the large opacities showed further nodular coalescence and medial migration of left upper zone large opacities. Chest radiographs done in 2007 (Figure 2) showed progression with bilateral upper zone category C large opacities and background small rounded opacities r/r 2/2.

PMF has a differential diagnosis on radiology that includes lung cancer, tuberculosis, sarcoidosis and...
rheumatoid nodules. Repeated sputum tests for tuberculosis were negative and, in view of the absence of high cumulative dust exposure, an open lung biopsy was performed on 5 December 2006.

Two pathologists with experience in occupational lung diseases independently confirmed the presence of PMF composed of confluent silicotic nodules. There was no evidence of malignancy or granulomatous inflammation. A few small foci of encapsulated necrosis were present but there was no evidence of active tuberculosis. The massive fibrosis occupied almost all of the lung biopsy and no separate lesions of coal workers’ pneumoconiosis were seen.

Pulmonary function studies were consistent with a severe airflow limitation: forced vital capacity (FVC) = 3.34 L or 82% of predicted;10 forced expiratory volume in one second (FEV₁) = 1.30 L or 40% of predicted;10 and FEV₁/FVC = 39%. The diffusing capacity was 10.29 mmol/min/kPa or 65% of predicted. No previous lung function data were available for comparison.

**DISCUSSION**

This case of histologically diagnosed silicosis with PMF is very unusual in that the intensity of dust exposure was apparently low and the duration of exposure prior to radiologic changes relatively short. Two questions are relevant in the consideration of this case: the reliability of the exposure history and the diagnosis.

Silicosis and PMF have been reported in Himalayan villagers as a consequence of environmental exposure in the form of frequent dust storms.11 However, no cases from environmental exposure have been reported in South Africa, and the patient recalled no such exposure. The only potential source of silica was from exposure at the power station where dust was generated from coal on conveyor belts, from 1983 to 2006. The patient may have been exposed to dust while working at a coal mine for one month, but he denied significant dust exposure during this time. Silicosis in coal miners is well documented1,12 and has been described in South African coal miners.12 Naidoo et al. reported a prevalence of silicosis and coal workers’ pneumoconiosis of 10.7% and 7.3%, respectively, from an analysis of autopsies conducted from 1975 to 1997 on South African coal miners with exclusive coal mining exposure.12 The mean duration of exposure was 11.0 years. There was a statistically significant association between silicosis and exposure, and an increasing trend in silicosis with increasing duration of exposure.

The diagnosis is convincing as it was histologically confirmed by two pathologists.

**CONCLUSION**

A comprehensive work history revealed only low exposure to dust. Coal dust from South African mines has been shown to contain silica12 and it must be concluded that this was sufficient to cause PMF. This case demonstrates that although heavy exposure is usually required to produce an occupational disease, disease may occur at low levels of exposure on history.

**REFERENCES**