Silicosis and Tuberculosis

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Two subjects had silicosis complicated by tuberculosis. In both patients, there was a relapse of the tuberculosis after chemotherapy was discontinued, in one case after 13 years of therapy with isoniazid and p-aminosalicylic acid. It would appear that the risk of tuberculosis in subjects with silicosis persists for life, and the suggestion is made that chemotherapy should be continued indefinitely.

Complicated silicosis is a serious progressive disease for which there is no specific therapy. Patients with this disease constantly face the threat of pulmonary tuberculosis. The symptoms of tuberculosis are often insidious in onset, and the underlying radiographic abnormalities make the diagnosis even more difficult. When a diagnosis of silicotuberculosis has been established, the duration of adequate chemotherapy remains an unresolved question. The following two case reports illustrate this dilemma.

CASE REPORTS

CASE 1

The first patient was a 58-year-old ex-sandblaster who was admitted to the West Virginia University Hospital, Morgantown, in October 1977 for evaluation of increasing shortness of breath, a productive cough, and low-grade fever. He had a 15-year history of sandblasting when he quit in 1958 because he had developed shortness of breath, hemoptysis, and atelectasis of the left lower lobe, for which he underwent a diagnostic thoracotomy. At that time the diagnosis of conglomerate silicosis was made. The patient was also noted to have a positive tuberculin skin test and was started on therapy with isoniazid (300 mg/day) and p-aminosalicylic acid (5 gm/day). He continued to receive these two drugs until 1971, a total of 13 years. His antituberculosis therapy was discontinued at this time because of gastric irritation.

The patient did well until October 1977, when he developed a cough with yellowish sputum, lethargy, and a catching sensation in his chest. His chest x-ray film revealed the chronic changes previously described, as well as a fluid level in the right middle pulmonary zone (Fig 1). A diagnosis of pulmonary abscess vs cavitary conglomerate silicosis was considered; however, a smear of sputum contained many acid-fast bacilli (later identified as Mycobacterium tuberculosis) that were sensitive to isoniazid, p-aminosalicylic acid, streptomycin, ethambutol, and rifampin.

On physical examination the patient was an obese man with a respiratory rate of 32/min and a temperature of 37.8°C (99.0°F). Examination of the chest revealed dullness and decreased breath sound over both pulmonary bases posteriorly, as well as tenderness and a few crepitations over the right anterior portion of the chest. The patient was started on treatment with ethambutol (800 mg/day), rifampin (600 mg/day), and streptomycin (1 gm three times per week for three months). He is now receiving rifampin and ethambutol and is feeling reasonably well.

CASE 2

The second patient was a 39-year-old ex-sandblaster (1964 to 1969) who was seen in the Chest Clinic on Oct 20, 1976.

FIGURE 1. Posteroanterior chest x-ray film taken in October 1977, showing conglomerate silicosis and tuberculous cavity in right middle pulmonary field.

FIGURE 2. Posteroanterior chest x-ray film taken in October 1978, showing conglomerate silicosis, tuberculosis, and bullae at both bases.

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In 1969, he had been found to have nodular densities on his chest x-ray film, as well as active tuberculosis, and was treated for six months with isoniazid (300 mg/day) and p-amino salicylic acid (10 gm/day), as well as streptomycin (2 gm/week for three months). Administration of isoniazid and p-amino salicylic acid was continued for an additional 18 months.

Six months later, the patient suffered a relapse. Therapy with isoniazid, streptomycin, pyrazinamide, and ethionamide (dosages unknown) was then started and was continued for 12 months. Smears of sputum have been negative since then, and the patient has continued to receive isoniazid (300 mg daily).

On physical examination, the patient was cachectic, with dyspnea and cyanosis, a temperature of 37°C (98.6°F), and a respiratory rate of 32/min. He had scattered rhonchi and decreased breath sounds at both bases and dullness with bronchial breath sounds over the apices (louder on the right than on the left). His chest x-ray film showed evidence of conglomerate silicosis, as well as tuberculosis and bullae at the bases (Fig 2). The forced expiratory volume in one second was 1.04 L (26 percent of predicted), and the arterial oxygen pressure was 54 mm Hg. The patient continues to receive therapy with supplemental low-flow oxygen at night and isoniazid (300 mg/day).

**DISCUSSION**

Published studies of subjects exposed to silica have shown a much higher incidence of tuberculosis than in comparable unexposed groups.1 Before antituberculosis drugs were introduced, tuberculosis was the main cause of death in classic silicosis.2 The response to chemotherapy for tuberculous infections in patients with pneumoconiosis is also less satisfactory than in subjects without the latter.3

Tuberculous infections in experimental animals have been shown to be aggravated by injections or inhalations of silica dust. The potentiation of tuberculous infections in animals exposed to silica is demonstrable in several ways, ie, the establishment of infection with fewer organisms than required to produce lesions in the absence of exposure to silica, higher counts of recovered organisms spread from subcutaneous sites of inoculation, greater propensity for hematogenous and lymphatic spread to the lungs, and fatal outcome to infection only in animals exposed to silica dust.

Studies show that in both experimental animals and human subjects, the growth and spread of tubercle bacilli is increased by concomitant exposure to silica dust.4 There is also experimental evidence to show that the growth of *M tuberculosis* in macrophages is potentiated by the addition of sublethal doses of silica. In the presence of silica, organisms of *M tuberculosis* not only multiply faster but also are released earlier from host cells.5 These factors are evident in vivo and are sufficient to account for the well-documented aggravation of tuberculous infections in both experimental animals and men exposed to silica dust; however, it should be noted that silica does not enhance the growth of *M tuberculosis* in vitro.6 It is known that silicosis has a definite tendency to progress, even in the absence of tuberculous infection, whether or not the subject remains exposed to the dust,7 and that at autopsy, patients with silicosis usually have at least twice as much free silica in their lungs as do normal persons.8 It is likely that silica, which persists in the lungs until autopsy, continues to exert its effects on macrophages throughout life.

The two cases reported herein and the experimental evidence cited would suggest that the coexistence of silicosis and tuberculosis justifies indefinite continuation of isoniazid. It is clear that silica predisposes to tuberculosis, while coal dust does not, although chemotherapy is less than satisfactory in both exposures. No relationship has been found between tuberculosis and the rate of attack of progressive massive fibrosis in coal miners’ pneumoconiosis;9 therefore, the suggestion that chemotherapy should be prolonged in silicosis should not be extended to coal workers’ pneumoconiosis.

**REFERENCES**

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**Percutaneous Use of Fiberoptic Bronchoscope to Investigate Bronchopleurocutaneous Fistula**

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The fiberoptic bronchoscope was used percutaneously to visualize the track of a bronchopleurocutaneous fistula and to obtain tissue and microbiologic specimens for examination. The bronchoscopy turned out to be a very simple procedure and patient discomfort was minimal.

The findings aided in the successful management of this patient.

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