Current Trends Silicosis: Cluster in Sandblasters -- Texas, and Occupational Surveillance for Silicosis

In November 1988, a physician in west Texas reported three cases of silicosis in sandblasters to the Ector County Health Department (ECHD). One of the workers, a 34-year-old man, subsequently died with acute silicotic alveolar proteinosis. All three workers had been employed at a facility that sandblasted oil-field drilling pipes. Following the physician's report, in January 1989, the ECHD and the Environmental Epidemiology Program, Epidemiology Division, Texas Department of Health (TDH), contacted local physicians and identified seven additional sandblasters who had onset of silicosis since 1985.

An investigation by ECHD and TDH included a review of personal and occupational histories obtained from each worker. Local radiologists evaluated chest radiographs; for four cases, a "B" reader* also reviewed the most recent chest radiograph for each patient for evidence of pneumoconiosis using the 1980 International Labour Office (ILO) guidelines (1). TDH staff reviewed lung tissue pathology reports and conducted an environmental survey of the plant.

Each of the 10 workers had histories of occupational exposure to silica and a chest radiograph consistent with pneumoconiosis; eight had a lung tissue pathology report of silicotic nodules or silicotic alveolar proteinosis (2). All were Hispanic males; their mean age at diagnosis was 30.5 years (range: 24-50 years), and seven were less than 30 years of age. All had chest radiographic abnormalities consistent with silicosis, most commonly reported as severe, diffuse interstitial disease with multiple small rounded opacities in most lung fields. The four radiographs evaluated by the "B" reader confirmed these findings, with ILO profusion category ranging from 1/1 (small opacities definitely present on comparison with standard radiographs) to 3/3 (the highest level of small opacity profusion as defined by standard radiographs). Six patients underwent bronchoscopic lung examination with transbronchial lung biopsy, and three underwent thoracotomy. Lung tissue pathology reports described silicotic nodules in eight of the surviving patients; in the ninth, the specimens did not support a definitive diagnosis. Silicotic alveolar proteinosis was identified in the fatal case. Tuberculosis was considered in all the reported patients, three of whom had reactive tuberculin skin tests. However, all sputum and tissue samples obtained from all patients were negative for Mycobacterium tuberculosis.
All 10 workers had used sandblasting machinery. Nine of them had worked at one facility, which employed approximately 60 persons. Duration of exposure to sandblasting ranged from 18 months to 8 years (mean: 4.5 years). Nine workers (eight at the one facility) reported no previous silica exposure; the remaining worker had sandblasted oil-field drilling equipment elsewhere for 3 years before working at the one facility for 5 years.

The sandblasting operation required that a rod containing a 1:1 mixture of flint and garnet (20.5% free silica) be passed under high pressure through the drilling pipe to remove contaminants and to prepare the interior surface for the application of a protective plastic coating. Although blast cabinets connected to exhaust systems enclosed the sandblasting operation, the cabinets were in poor repair and permitted the release of clouds of dust throughout the work area. Protective booths, constructed in an attempt to reduce worker exposure to silica, drew air from areas with substantial silica contamination. Workers manually shoveled used sandblasting material back into the machinery for re-use.

Personal breathing-zone air samples, obtained in November 1988, documented respirable free silica exposures ranging from 400 to 700 ug/m³ for workers in the sandblasting area and were consistent with results reported by the Occupational Safety and Health Administration (OSHA) during a similar environmental inspection. These exposures substantially exceeded 100 ug/m³, the current OSHA permissible exposure limit (PEL) for respirable silica (in effect since September 1989) (3).** Supplied-air respirators had not been used during sandblasting. Workers reported wearing only disposable respirators, and none of the affected workers had been fit-tested for a respirator.

Silicosis is a reportable condition in Texas; the TDH is intensifying case detection efforts to determine whether the cluster reported here reflects an industry-wide problem. Reported by: D Fleming, D Maynard, B McKinney, Ector County Health Dept; DM Perrotta, PhD, L Schulze, J Pichette, MS, Epidemiology Div, Texas Dept of Health. Div of Respiratory Disease Studies, National Institute for Occupational Safety and Health, CDC.

Editorial Note

Editorial Note: This report underscores the potential health hazards associated with abrasive blasting with silica sand--hazardous work through which even young workers can contract silicosis, a serious and potentially fatal respiratory disease. The investigation illustrates conditions characteristically present at sandblasting worksites associated with cases of silicosis: failure to substitute less toxic abrasive blasting materials; presence of massive clouds of dust, resulting in dangerous exposures to silica for unprotected sandblasters and co-workers; and lack of an effective respiratory protection program for workers. In addition to producing intense dust exposures, the process of abrasive blasting typically fractures sand into finer particles. Freshly fractured silica appears to be more biologically active than aged silica (5). This factor may contribute to the development of acute accelerated forms of silicosis, as described in this report.

Abrasive blasting is used in many industries and settings (e.g., to clean sand from foundry castings, to prepare ship hulls and metal bridges for painting, and to clean surfaces of stone buildings), and silicosis associated with sandblasting has been described among shipyard workers (6), tombstone sandblasters (7), and others (6). Silicosis specifically associated with the process of cleaning oil-field drilling pipe has not been previously reported; however, an independent clinical report has described diagnostic procedures used for one of the 10 cases reported here (8).
Materials less hazardous than silica sand are available for abrasive blasting (9,10). Because of the severe risk for silicosis associated with abrasive blasting with silica sand and the difficulty in controlling these hazards, NIOSH recommended in 1974 that silica sand (or other substances containing greater than 1% free silica) be prohibited as abrasive blasting material (4).

Surveillance of silicosis among sandblasters is problematic for at least three reasons (6). First, there is no single union or central registry of sandblasters. Second, sandblasting may be only an intermittent part of a worker's job responsibilities, and certain occupations (e.g., painter) may involve intermittent sandblasting. Third, even when a sandblaster dies with silicosis, this diagnosis may not be listed on the death certificate (6).

Each case of silicosis should be considered a sentinel health event indicating the potential presence of ongoing hazardous worksite exposures that need evaluation; physician reporting is a useful adjunct to this process. The TDH participates in the Sentinel Event Notification System for Occupational Risks (SENSOR) program (11), a collaborative effort involving NIOSH and 10 state health departments.*** The program is intended to improve occupational disease surveillance at the state and local levels through targeted, provider-based reporting of selected work-related conditions or exposures and worksite follow-up of reported cases. In addition to Texas, five other states (Michigan, New Jersey, New York, Ohio, and Wisconsin) in this program list silicosis or silica exposure as a SENSOR condition for targeted surveillance.

To facilitate provider-based surveillance of work-related conditions and to enhance uniformity of reporting in the states, NIOSH periodically disseminates recommended surveillance case definitions for selected occupational diseases and injuries. Because these definitions are intended for surveillance-related functions, they may differ from those used for other purposes, such as determining workers' compensation or level of disability. The surveillance reporting guidelines and case definition for silicosis**** (see box) are recommended for surveillance of work-related silicosis by state health departments receiving reports of cases from physicians and other health-care providers.

### References


5. Vallyathan V, Shi X, Dalal NS, Irr W, Castranova V. Generation of free radicals from freshly...


State health departments should encourage physicians, including radiologists and pathologists, as well as other health-care providers, to report all diagnosed or suspected cases of silicosis. These reports should include persons with:

A. A physician's provisional or working diagnosis of silicosis OR

B. A chest radiograph interpreted as consistent with silicosis OR

C. Pathologic findings consistent with silicosis. State health departments should collect appropriate clinical, epidemiologic, and workplace information on reported
persons with silicosis as needed to set priorities for workplace investigations. Surveillance Case Definition

A.

1. History of occupational exposure to airborne silica dust* AND

2. Chest radiograph or other imaging technique interpreted as consistent with silicosis**

OR B. Pathologic findings characteristic of silicosis.*** *Exposure settings associated with silicosis are well characterized and have been summarized in several reviews (12,13). The induction period between initial silica exposure and development of radiographically detectable nodular silicosis is usually greater than 10 years. Shorter induction periods are associated with heavy exposures, and acute silicosis may develop within 6 months to 2 years following massive silica exposure. **Cases can be classified as simple or complicated. Simple silicosis is present if the largest opacity is less than 1 cm in diameter. Complicated silicosis (also known as progressive massive fibrosis (PMF)) is present if the largest opacity is greater than or equal to 1 cm in diameter. Common radiographic findings of nodular silicosis include multiple, bilateral, and rounded opacities in the upper lung zones; other patterns have been described. Since patients may have had mixed dust exposure, irregular opacities may be present or even predominant. Radiographs interpreted by NIOSH-certified "B" readers should have profusion categories of 1/0 or greater by the International Labour Organization classification system (1). A bilateral alveolar filling pattern is characteristic of acute silicosis and may be followed by rapid development of bilateral small or large opacities. ***Characteristic lung tissue pathology (2) in nodular silicosis consists of fibrotic nodules with concentric "onion-skinned" arrangement of collagen fibers, central hyalinization, and a cellular peripheral zone, with lightly birefringent particles seen under polarized light. In acute silicosis, microscopic pathology shows a periodic acid-Schiff positive alveolar exudate (alveolar lipoproteinosis) and a cellular infiltrate of the alveolar walls.

Disclaimer All MMWR HTML documents published before January 1993 are electronic conversions from ASCII text into HTML. This conversion may have resulted in character translation or format errors in the HTML version. Users should not rely on this HTML document, but are referred to the original MMWR paper copy for the official text, figures, and tables. An original paper copy of this issue can be obtained from the Superintendent of Documents, U.S. Government Printing Office (GPO), Washington, DC 20402-9371; telephone: (202) 512-1800. Contact GPO for current prices.

**Questions or messages regarding errors in formatting should be addressed to mmwrq@cdc.gov.