OCCUPATIONAL RESPIRATORY DISEASES

Editor
James A. Merchant, M.D., Dr. P.H.

Associate Editors
Brian A. Boehlecke, M.D.
Geoffrey Taylor, M.D.

Technical Editor
Molly Pickett-Harner, M.F.A.

Division of Respiratory Disease Studies
Appalachian Laboratory for Occupational Safety and Health

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Centers for Disease Control
National Institute for Occupational Safety and Health

September 1986

Disclaimer

Mention of company names or products does not constitute endorsement by the National Institute for Occupational Safety and Health.

DHHS (NIOSH) Publication No. 86-102

For sale by the Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20402

ACUTE SILICOSIS

Daniel E. Banks

Acute Silicosis, or silico-proteinosis, is a rare presentation of silica-induced lung disease (Table II-8) (10). This form of silicosis is associated with massive exposures to respirable size particles of high free crystalline silica content over a short period of time. Invariably, this disease is untreatable with a lethal outcome. Betts, in 1900, first described this acute presentation of silicosis in the United States and his description is relevant today (4):

> After coughing has continued for some time there will be. . . loss of appetite, loss of weight and shortness of breath, the respirations running as high as 38 to 42 breaths per minute on the slightest exertion. As the weeks pass, the patient suffers general malaise and soon finds it impossible to get about. . . in (the disease's) later stages, the temperature may rise to 102 or 104. . .

Early reports related acute silicosis to miliary tuberculosis because of the similar rapid downhill course (11). Other early reports described similar presentations in workers who mixed silica and alkali in the production of industrial abrasives (7)(9). Postmortem examination of the lung in these cases revealed (6):

> The presence in every alveolus of large amounts of pink staining fluid, (and) an extreme grade of edema with a very high protein content. Another interesting finding is the presence of epithelium in all the alveoli.

The pathologic and radiographic features of acute silicosis were fully described in 1969. Buechner and Ansari described 4 sandblasters with a mean silica dust exposure of only 4 years, and relentlessly progressive dyspnea, cough, fatigue, weight loss, and pleuritic chest pain (5). Despite the prompt diagnosis and treatment of tuberculosis in 3 cases and appropriate therapy of suspected tuberculosis in the 4th, mean survival time from onset of symptoms was only 7.5 months. All died from respiratory failure.

In all, chest radiographs showed air bronchograms and an alveolar filling pattern. Each man had significant restriction of lung volumes.

Pathologically, the alveolar septae were thickened and infiltrated with mononuclear cells. The lungs were firm and heavy with a pinkish, proteinaceous PAS positive staining alveolar exudate (Figure II-9) identical to that seen in idiopathic alveolar proteinosis and has resulted in the use of the term silico-proteinosis. Typical silicotic nodules were seen in 2 cases, but these were smaller than nodules noted in the chronic form of silicosis.

More recently, Suratt et al reported acute silicosis in 4 tombstone sandblasters (12). These cases were similar to those above in both mean duration of exposure (4 years) and mean survival from onset of symptoms (6 months). Two had pneumothoraxes complicating their clinical course. One developed focal glomerulonephritis and another systemic lupus erythematosis (both had positive anti-nuclear antibodies, a common finding in sandblasters' silicosis (8)). All 4 had a restrictive impairment on spirometry with a significant decrease in diffusing capacity. No chest radiographs revealed the pattern of silico-proteinosis described above. Instead, 2 showed bilateral upper lobe opacities, and 2 showed a reticulonodular pattern.

Despite the absence of a radiograph appearance of silico-proteinosis, postmortem lung examinations in two sandblasters revealed a PAS positive exudate filling the alveoli. Discrete hyalinized and cellular nodules were present in alveolar walls and within the wall of small pulmonary blood vessels. The authors considered these pathologic changes as intermediate between silico-proteinosis and chronic nodular silicosis.

Evidently, then, there is variability in chest radiographs of workers with massive silica dust exposures over a short period. Classically, alveolar infiltrates with air bronchograms are present and correlate with PAS positive proteinaceous

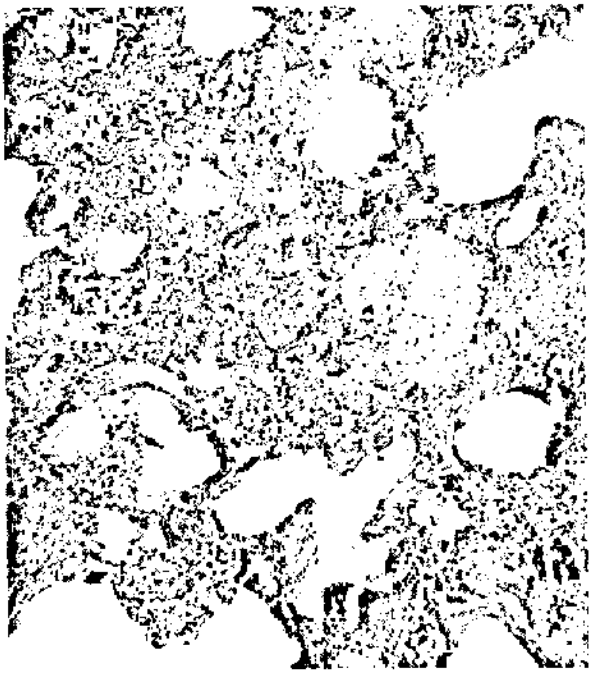

Figure II-9. Photomicrograph (Hematoxylin & Eosin, 50X) from a lung biopsy specimen of a surface coal miner driller who died 26 months after the diagnosis of acute silicosis was made. The photomicrograph shows distorted pulmonary parenchyma, interstitial inflammation and fibrosis and filling of the alveolar spaces with a relatively acellular material with some epithelial cells present. This material gave a positive reaction when stained with Periodic acid-Schiff reagent. (3)

Figure II-10. Chest roentgenogram of a silica flour worker showing diffuse small opacities with a lower lobe predominance, a large opacity in the right mid-lung field, and a right sided air-bronchogram.

alveolar exudate. Alternatively, simple nodular silicosis, which rapidly progresses to progressive massive fibrosis, may be present in those with short-term massive exposures.

Silicotics are particularly prone to mycobacterial infections. Bailey et al found 22 of 83 silicotic sandblasters in New Orleans developed complicating mycobacterial infections, both with typical and atypical (M. kansasii and M. intracellulare) organisms (1). All 18 (diagnosed antemortem) converted positive sputum to negative status under treatment. Control of tuberculosis did not prevent progressive respiratory impairment and 4 of these patients died of respiratory failure. Of the total of 8 deaths in the entire group, 3 occurred in sandblasters with silico-proteinosis.

Recently NIOSH representatives evaluated the health of miners and mill workers at 2 silica flour mills in Southern Illinois (2). Of 61 workers and ex-workers with 1 or more years of exposure to silica, 16 (26%) developed simple silicosis and 7 (11%) had conglomerate silicosis. Four of these 7 had 6 or less years of silica dust exposure. One workman developed the radiographic picture of silico-proteinosis (associated with a mid-lung conglomerate lesion) after only 2-½ years of dust exposure (Figure II-10).

As we enter the decade of the 1980's, it is vexing to acknowledge that silicosis—perhaps the oldest occupationally related disease—exists despite sophisticated control technology. Sandblasters and silica flour mill workers are 2 groups still at high risk of developing acute silicosis. Adequate compliance with current standards and continued surveillance of workers exposed to free silica is essential to prevent severe health effects.

REFERENCES

1. Bailey, W. C., Brown, M., Buechner, H. A., Weill, H., Ichinose, H., and Ziskind, M.: Silico-mycobacterial disease in sandblasters. Am Rev Respir Dis *110*:115- 125, 1974.
2. Banks, D. E., Morring, K. L., Boehlecke, B. A., Althouse, R. B., and Merchant, J. A.: Silicosis in silica flour workers. Am Rev Resp Dis *124*:445-450, 1981.
3. Banks, D. E., Bauer, M. A., Castellan, R. M., and Lapp, N. L.: Silicosis in surface coal mine drillers. Thorax (in press)
4. Betts, W. W.: Chalicosis pulmonum or chronic

Table II-8

IMPORTANT CHARACTERISTICS OF THE DIFFERENT CLINICAL FORMS OF SILICOSIS (8)

Clinical Type	Pathology	Exposure Levels	% Silica in Dust	Usual Exposure Duration	Time from 1st Exposure to Disease Development (X-ray Findings)
Chronic ("Classical") Silicosis	Fibrotic nodules located near respiratory bronchioles	"moderate"	<30%	20-40 yrs.	20 yrs.
Accelerated Silicosis	Fibrotic nodules smaller than those in "classical" silicosis PMF in mid-zones	"moderate-high"	47-84%	5-15 yrs.	4-8 yrs.
"Silico-proteinosis" Acute Silicosis	Diffuse interstitial fibrosis and alveolar lipo-proteinosis	"heavy"	90-100%	3-6 yrs.	1-3 yrs.

interstitial pneumonia induced by stone dust. JAMA *34*:70-74, 1900.

5. Buechner, H. A. and Ansari, A.: Acute silico-proteinosis. Chest *55*:274-284, 1969.
6. Cabot, R. C.: Case records of the Massachusetts General Hospital, Case 20102. New Engl J Med *210*:551-554, 1934.
7. Chapman, E. M.: Acute silicosis. JAMA *98*:1439-1441, 1932.
8. Jones, R. N., Turner-Warwick, M., Ziskind, M., and Weill, H.: High prevalence of antinuclear antibodies in sandblasters' silicosis. Am Rev Respir Dis *113*: 393-395, 1976.
9. MacDonald, G., Piggot, A. P., and Gilder, F. W.: Two cases of acute silicosis. Lancet *2*:846-847, 1930.
10. McKay, W. J., Palmer, W. W., Jr., Boehlecke, B. A., and Solook, J.: Health Hazard and Technical Assistance Report No. 77-101. U.S. Public Health Service.
11. Middleton, E. L.: The present position of silicosis in industry in Britain. Br Med J *2*:485-489, 1929.
12. Suratt, P. M., Winn, W. C., Jr., Brody, A. R., Bolton, W. K., and Giles, R. D.: Acute silicosis in tombstone sandblasters. Am Rev Respir Dis *115*: 521-529, 1977