

Asbestos-Related X-Ray Changes in Foundry Workers

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Michigan has a statewide mandatory occupational disease reporting system. As part of that system, reports are received from hospitals, physicians, death certificates, the workers' compensation bureau, and company medical departments. Based on this reporting, the State of Michigan has a special emphasis program for the surveillance of silicosis, a known disease outcome among foundry workers.

From 1985-1996, 115 cases reported to the State Surveillance System as silicosis, pneumoconiosis not specified, or pulmonary fibrosis were reclassified as having asbestos related x-ray changes after a B-reader interpretation of each case's chest x-ray. During this same period there were an additional 697 reports confirmed as silicosis and 6,724 cases reported to the surveillance system as asbestosis.

Among the 115 reports reclassified as having asbestos-related x-ray changes without evidence of silicosis-related x-ray changes, 54 had worked in foundries. Only 7 (14.8%) of these individuals had their primary work in maintenance in the foundry; 40 (85.1%) had their primary foundry work in a production job; and for 10 individuals the occupation was not known. Asbestos has been used in foundries on pipe laggings, boiler coverings, as insulation in fan housings, in gloves, aprons and curtains, as insulation in cupolas, and in ladles and insulation in sand molds.

Clinicians caring for foundry workers need to be aware that asbestos-related x-ray changes are not uncommon in this population and asbestos exposure should be considered as one of the carcinogens contributing to the known increased risk of lung cancer among foundry workers. Am J. Ind. Med. 34:197-201, 1998. © 1998 Wiley-Liss, Inc.

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INTRODUCTION

Foundry workers are known to be exposed to silica and to be at increased risk for developing silicosis [Finkelstein, 1994; Karava et al., 1976; Landrigan et al., 1986; Oudiz et al., 1983; Renes et al., 1950]. As part of a statewide surveillance system for silicosis, a number of foundry workers initially suspected to have silicosis but ultimately

documented with asbestos-related x-ray changes have been identified. In this article, we report a series of patients with asbestos-related x-ray changes whose asbestos exposure occurred from their work in a foundry. Sources for asbestos exposure in foundries are also reviewed.

METHODS

In 1987, the State of Michigan in cooperation with Michigan State University received financial assistance from the National Institute for Occupational Safety and Health (NIOSH) to institute a statewide surveillance program for silicosis. Silicosis reporting was encouraged as part of a broader effort to encourage the reporting of all occupational illnesses. Sources used to identify persons with occupational disease were: 1) reports from hospitals begin-

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ning in 1987; 2) reports from physicians beginning in 1989 including physicians certified by NIOSH to interpret x-rays for pneumoconiosis (B-readers); 3) death certificates beginning in 1985; 4) workers' compensation claims beginning in 1985; and 5) reports from company medical departments beginning in 1989.

Reporting of known or suspected occupational illnesses by employers, clinics, health professionals, and hospitals was required by law (Part 56 of P.A. of 1978). When a report was received of suspected silicosis, pneumoconiosis not specified, or pulmonary fibrosis, the patient was interviewed and a recent chest x-ray was obtained. The interview included a cigarette smoking history, work history, and medical history for the diagnosis of respiratory conditions. The chest x-ray was interpreted by a NIOSH certified B-reader (KDR). If a chest x-ray was unavailable, then the x-ray report from the radiologist was reviewed. Some of the patients initially suspected to have silicosis who were interviewed and for whom medical records including a chest x-ray were available were determined to have asbestos-related lung damage, not silica-related. When a lung biopsy had been performed, the pathology report was also reviewed. The criteria for asbestos-related x-ray changes were an x-ray interpretation involving at least the lower lobes, showing a minimal level of profusion of linear opacities s, t, or u and 1/0 or greater profusion per the International Labour Office's [ILO, 1980] classification system for pneumoconiosis and/or pleural changes (unilateral or bilateral). Patients classified as having asbestos-related lung disease also must have had the absence of rounded opacities on the chest x-ray and the absence of silicotic nodules on the pathology report. The x-ray was not considered to have positive pleural changes if the only x-ray finding was a blunted costophrenic angle.

The protocol for this activity was approved by the Michigan State University Human Subjects Review Board.

RESULTS

From 1985–1996, 115 patients were reclassified as having asbestos-related x-ray changes who had initially been reported as having either silicosis, pneumoconiosis not specified, or pulmonary fibrosis. During this same period, there were an additional 6,724 reports of people initially reported as having asbestos-related changes. Radiographs from these individuals were not reviewed and we do not know where they worked. Also during this period, there were 697 patients who were confirmed to have silicosis. Among these 697, 81 (11.6%) had evidence of mixed dust disease (s/t/u profusion changes in lower zones in addition to p/q/r profusion opacities in upper zones), 36 (5.2%) had mixed dust disease and pleural changes, and 49 (7.0%) had pleural changes in addition to p/q/r profusion opacities in the

upper zones. Among these 166 individuals with x-ray evidence of both silica and asbestos exposure, 121 (72.9%) had their primary work exposure in 36 different foundries. For 14 individuals, their primary job was in maintenance.

Fifty-four of the 115 patients reclassified as having asbestos-related x-ray changes but no silicosis had their primary exposure to asbestos in 17 different foundries. Three additional patients worked in an unknown foundry. Only 9 of the 49 (18.4%) individuals whose primary exposure to asbestos was in a foundry and for whom we had a complete work history had worked at other jobs with possible asbestos exposure, such as being in the Navy, or working in shipyards or steel mills. The following results are on the 57 individuals who had worked in foundries.

Twenty-eight individuals (49.1%) were African-American, 28 (49.1%) were Caucasian, and 1 (1.8%) was Hispanic. The average date of birth for these foundry workers was 1924. The distribution of the decades they had begun work in a foundry is shown in Figure 1. Eighty percent began their work in a foundry in the 1940s–1960s. The distribution of years worked is shown in Figure 2. Sixty-three percent of these individuals worked at a foundry for 20 or more years.

Table I lists the primary foundry job of the 57 patients. For approximately 15%, the primary job was maintenance, while the other 85% had worked in production. Three of the production workers had also had a maintenance job in the foundry, but it was not their longest-held job at the foundry. Five of the production workers and four of the maintenance workers had possible asbestos exposure at other jobs besides a foundry. They had worked at other jobs or been in the Navy, with possible asbestos exposure for 2–12 years (average 5.3 years).

Twenty-six individuals had parenchymal disease without pleural disease (nine, 1/0; five, 1/1; six, 1/2; one, 2/1; four, 2/2; and one, 2/3), 17 had pleural disease without parenchymal disease, and 14 had both parenchymal and pleural disease. Twenty-one of the 31 individuals with pleural disease had bilateral changes and 4 of 31 had calcification in their pleura.

CASE REPORTS

Patient 1

A 69-year-old African-American male had a chronic productive cough for six years and was bothered by dyspnea on exertion. At the age of 19 he went to work at a foundry pouring iron. He worked at three other foundries as a laborer, a molder, and a core maker until he retired when he was 67. He first began to work in the 1930s. He had been hospitalized approximately 15 times for his lung disease. He

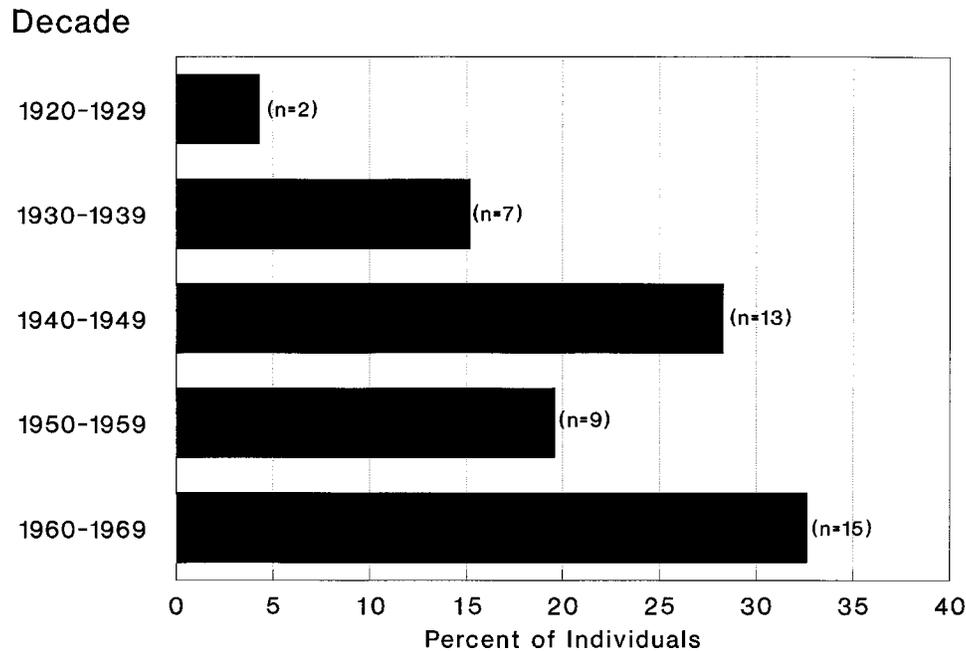


FIGURE 1. Distribution by decade when individuals with asbestosis began to work in a foundry, 1985-1996. Total number of individuals: 46. Decade was unknown for 11.

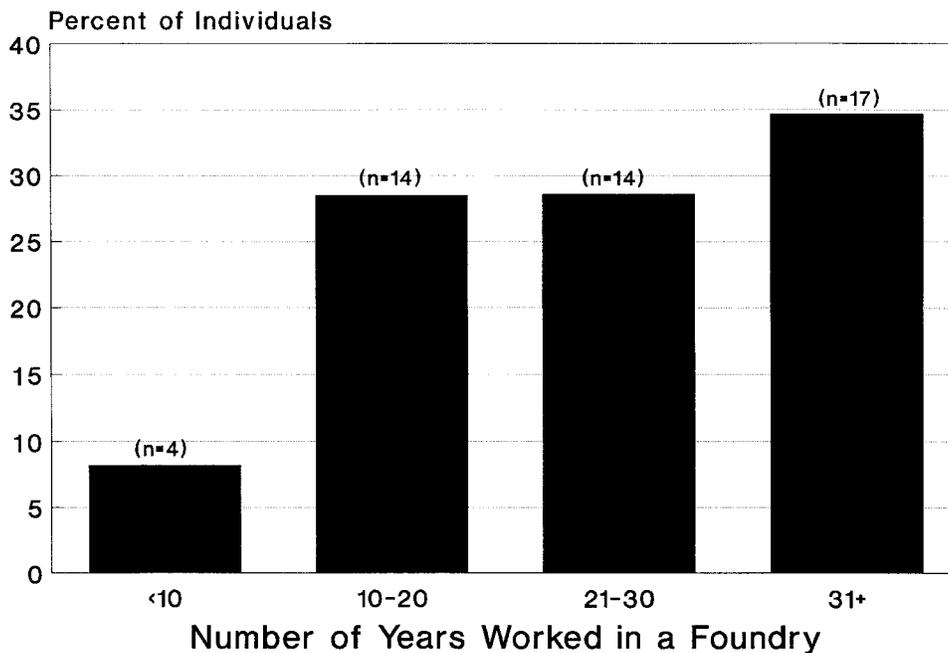


FIGURE 2. Distribution of years worked in a foundry for individuals with asbestosis, 1985-1996. Total number of individuals: 49. Number of years was unknown for eight.

had smoked a pack of cigarettes a day from his teens until he was 65. He never applied for workers' compensation. His x-ray showed linear opacities in the lower lobes which were classified s/t type, with 1/1 profusion, per the ILO criteria. His FVC was 3.01 liters (84% of predicted), FEV₁ was 1.14

liters (47% of predicted), and the FEV₁/FVC ratio was 38%. His RV/TLC was 34%, with a predicted of 38%. His diffusing capacity corrected for hemoglobin was 12.5 ml/min/mmHg, which was 53% of predicted. He had no improvement with bronchodilators.

TABLE I. Primary Foundry Occupation of Individuals With Asbestosis: 1985–1996*

Skilled trades (maintenance)	7	(14.8%)
Molder	11	(23.4%)
Laborer	10	(21.3%)
Chipper/grinder	5	(10.6%)
Casting	3	(6.4%)
Other production	11	(23.4)

*Total number of individuals = 47. Occupation was unknown for 10.

Patient 2

A 77-year-old white man had dyspnea when hurrying on level ground or walking up a slight hill. He did not have a chronic cough. Beginning in the 1940s, he worked for 26 years as a millwright, repairing machinery at two different foundries. Previous to that, he had been in the Army and then worked for 3 years in outdoor carpentry work. He had been hospitalized approximately ten times for his lung disease. He had smoked up to four packs of cigarettes a day from his teens until he was 30. He had been awarded workers' compensation. His x-ray showed linear opacities in the lower lobes which were classified s/t type, with 1/2 profusion, per the ILO criteria. His FVC was 1.89 liters (55% of predicted), FEV₁ was 1.27 liters (56% of predicted), and the FEV₁/FVC ratio was 86%.

Patient 3

A 48-year-old white male had dyspnea on exertion. Beginning in the mid 1960s, he began to work at a foundry in the sand mixing and sand core production area. He worked there for 14 years. Previous to that, he had worked in a plastic factory and done maintenance work in a trailer park for 5 years. After leaving the foundry, he had worked for 9 years on an assembly line loading windshields. He had applied for workers' compensation. He had smoked a quarter of a pack of cigarettes a day until he quit at the age of 36. His x-ray showed no parenchymal changes but he did have bilateral pleural thickening, classified as noncalcified, circumscribed, B2 width and extent, per the ILO criteria. His FVC was 2.98 liters (62% of predicted), FEV₁ 2.53 liters (71% of predicted), and the FEV₁/FVC ratio was 85%.

DISCUSSION

Workers in foundries are known to be at increased risk of silicosis. Similar to the findings in other industrial settings [Lilis et al., 1980; Rosenman, 1991], it is not surprising that foundry workers are also at increased risk of asbestos-related lung disease. As part of a statewide surveillance system for silicosis, we identified 57 individuals initially

reported to us with either silicosis, pneumoconiosis not specified, or pulmonary fibrosis who had an x-ray characteristic of asbestosis and/or asbestos-related pleural disease. These individuals had worked in 17 different foundries; the exposures to asbestos therefore have not been limited to a particular foundry. Among the 697 confirmed individuals with silicosis reported during the same time period, there were 166 (27.8%) individuals with x-ray changes of silicosis (round opacities in upper lobes) who also had either pleural or parenchymal asbestos-related x-ray changes. One hundred twenty-one of these 166 (72.9%) individuals had their primary job in a foundry. They had worked in 36 different foundries. The 178 individuals with any evidence of asbestos-related changes on their x-ray (57 with only asbestos and 121 with both silica and asbestos changes) had worked in a total of 43 different foundries.

In a previous study at a single grey iron foundry in another state, we found the prevalence of asbestos-related parenchymal disease was .8%, and asbestos-related pleural disease without parenchymal changes was 2.5% [Rosenman et al., 1996]. In that same foundry, the prevalence of silicosis was 2.5%. We used the same criteria as in that report to classify x-rays as either asbestos- or silica-related. The prevalence of x-ray changes consistent with silicosis was statistically related to estimates of mean and cumulative silica exposure. Neither the parenchymal nor pleural changes consistent with asbestos exposure were associated with silica exposure estimates that were calculated for each worker [Rosenman et al., 1996], therefore strengthening the argument for asbestos exposures in the foundry as a cause of disease among these workers.

We also received death certificates of 13 individuals who died with mesothelioma who had worked at a single foundry in Michigan.

Typically, asbestos-related lung disease is found among the maintenance workers in production facilities. In this group of 57 individuals, x-ray changes were found in greater numbers of non-maintenance workers. Among the 47 individuals whose job title in the foundry was known, seven had a primary job in maintenance and another three had worked in maintenance at some time. Relatively few (five production workers and four maintenance workers) had been exposed to asbestos at other jobs besides foundries. When they had been exposed to asbestos at other jobs, it was for a relatively short part of their working career.

Asbestos has been used in foundries in different ways: on pipe lagging; on boiler coverings; as insulation in fan housings; in gloves, aprons, and curtains containing asbestos; and as insulation in cupolas and ladles. It has also been suggested that talc used as a detaching agent in sand molds may cause asbestos-related x-ray changes [Scancarello et al., 1996]. However, this conclusion has been challenged as being inadequately documented [Langer and Nolan, 1997].

Other possible uses of asbestos include its use as insulation in sand molds.

We were unable to identify any published reports of air measurements for asbestos in foundries. We did identify two other health studies indicating asbestos exposure in foundries. The prevalence of pleural changes was 11% overall and 21% among workers 50 years old or older in a Canadian copper refinery and foundry [Ostiguy et al., 1995]. No air measurements for asbestos were reported. The pleural changes were attributed to the use of asbestos-containing insulation on pipes and furnaces. In the second study, five workers who had been employed for 20–31 years in foundries and iron reclamation plants in Texas had analysis of the ferruginous bodies obtained on bronchoalveolar lavage [Dodson et al., 1993]; 12–60% of the ferruginous bodies were consistent with asbestos exposure. Amosite asbestos was reported on transmission electron microscopy.

This review examined only a small portion of workers with asbestos-related x-ray changes. Since the focus of the surveillance system under which these asbestos-exposed foundry workers were identified was silicosis, we did not attempt to document the complete work histories and other relevant medical findings of the other 6,274 individuals reported during this time period, who were reported specifically with asbestosis or pleural plaques. A detailed look at this larger group would provide an estimate of the prevalence of asbestos-related x-ray changes among foundry workers.

The recognition that asbestos exposure has occurred in foundries has two important implications. Clinicians who are taking care of foundry workers need to be aware that their patients may have been exposed to asbestos as well as silica, and that workers with asbestos-related x-ray changes are not uncommon in this population. Previous mortality studies of foundry workers have shown an increased risk from lung cancer [IARC, 1987]. Exposure to asbestos, along with silica and the polycyclic aromatic hydrocarbons from the metal fumes, all need to be considered as potential causative agents for the increased risk of lung cancer found among foundry workers.

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