Concurrent Silicosis and Pulmonary Mycosis at Death

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To examine risk for mycosis among persons with silicosis, we examined US mortality data for 1979–2004. Persons with silicosis were more likely to die with pulmonary mycosis than were those without pneumoconiosis or those with more common pneumoconioses. Health professionals should consider enhanced risk for mycosis for silica-exposed patients.

The pneumoconioses are a group of irreversible but preventable interstitial lung diseases, most commonly associated with inhalation of asbestos fibers, coal mine dust, or crystalline silica dust. In response to inquiries from silica-exposed workers concerned about diagnoses of coccidioidomycosis or cryptococcal meningitis for their co-workers, we examined whether excess risk for mycosis exists among persons with silicosis.

The Study

We examined mortality data from the National Center for Health Statistics of the Centers for Disease Control and Prevention, which are coded for causes of death reported on death certificates for all US residents according to the International Classification of Diseases (ICD) (www.cdc.gov/nchs/products/elec_prods/subject/mortmcd.htm). Because silicosis usually has a latency of ≥20 years, we restricted our analyses to persons ≥45 years of age at death. We separately evaluated 3 groups of persons who had died among persons with silicosis; 79% asbestosis, and 53% CWP (Table 1). Persons with silicosis were 4.5× (95% CI 3.4–6.0×) more likely to have any mycosis at death than were those without pneumoconiosis. Persons with silicosis were 2.9× (95% CI 1.9–4.4×) more likely than those with asbestosis and 6.7× (95% CI 4.3–10.5×) more likely than those with CWP to have pulmonary mycosis at death.

We computed prevalence rate ratios and 95% confidence intervals (CIs) to separately compare pulmonary mycosis prevalence at death among persons with silicosis, asbestosis, and CWP with that for persons in the referent group and to compare pulmonary mycosis prevalence at death among persons with silicosis with that for persons in the 2 pneumoconiosis comparison groups. Each ratio was computed by dividing the proportion of mycosis deaths in 1 group by the corresponding measure in the comparison group.

Decedents with pneumoconiosis and mycosis were rare, and most mycoses were pulmonary: 77% in persons with silicosis; 79% asbestosis, and 53% CWP (Table 1). Persons with silicosis were 4.5× (95% CI 3.4–6.0×) more likely to have any mycosis at death and 9.5× (95% CI 6.9–13.1×) more likely to have pulmonary mycosis at death than were those without pneumoconiosis. Persons with silicosis were 2.9× (95% CI 1.9–4.4×) more likely than those with asbestosis and 6.7× (95% CI 4.3–10.5×) more likely than those with CWP to have pulmonary mycosis at death.

Among persons who died with pneumoconiosis, aspergillosis was the most common pulmonary mycosis. Those with silicosis were more likely than those without any pneumoconiosis to have aspergillosis, coccidioidomycosis, or cryptococcosis at death (Table 2). Among the 48 decedents who had silicosis and mycosis, 9 also had tuberculosis, 4 had diabetes (2 with tuberculosis), 2 had lung malignancy (1 with tuberculosis), and none had received organ transplants. From 1987 (the first year of ICD codes for HIV) to 2004, 1 decedent with silicosis had HIV but not mycosis. Of 8 decedents with aspergillosis and HIV, 2 had mycoses; no decedents with CWP had HIV. When we lim-
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Table 1. Cause of death with any death certificate mention of selected pneumoconioses and mycoses (underlying or contributing cause), US residents ≥45 y of age, 1979–2004*

| Cause           | No. deaths | Any mention | Mycoses, no. (%) | Pulmonary Prevalence rate ratio† (95% CI) |
|-----------------|------------|-------------|------------------|-----------------------------------------|
|                 |            | All  | Pulmonary | All  | Pulmonary | All  | Pulmonary |
| Silicosis       | 6,723§     | 48 (0.71) | 37 (0.55) | 25 (52) | 21 (57) | 4.5 (3.4–6.0) | 9.5 (6.9–13.1) |
| Asbestosis      | 23,899§    | 58 (0.24) | 46 (0.19) | 22 (38) | 18 (39) | 1.5 (1.2–2.0) | 3.3 (2.5–4.4) |
| CWP             | 46,088§    | 72 (0.16) | 38 (0.08) | 29 (40) | 16 (42) | 1.0 (0.8–1.2) | 1.4 (1.0–2.0) |
| All other deaths¶ | 51,677,216 | 81,699 (0.16) | 29,914 (0.06) | 33,941 (42) | 12,982 (43) | NA | NA |

*Data from National Center for Health Statistics (www.cdc.gov/nchs/products/elec_prods/mortmcd.htm). CI, confidence interval; CWP, coal worker pneumoconiosis; NA, not applicable.
†Proportion of decedents with each selected pneumoconiosis who had mycosis coded as the underlying cause of death as or as a contributing cause of death on the entity axis compared with the analogous proportion of pulmonary mycosis decedents without any mention of a selected pneumoconiosis as underlying cause of death or a contributing cause of death on the entity axis (all other deaths).
‡When International Classification of Diseases (ICD) 9th or 10th revision codes for opportunistic mycoses and ICD-10 codes for unspecified types of mycoses were excluded, the prevalence rate ratio and 95% CI for deaths with any mention of silicosis became 10.4 (7.5–14.4); asbestosis 3.3 (2.4–4.4); and CWP 1.5 (1.1–2.1).
§No. persons who died for which the selected pneumoconiosis was coded as the underlying cause of death or as a contributing cause of death on the entity axis, excluding decedents with multiple pneumoconioses.
¶Excludes deaths with any mention (underlying or contributing cause) of silicosis, asbestosis, and/or CWP.

We found that persons who die with silicosis are more likely to die with pulmonary mycoses than are those who die without pneumoconiosis or who die with the more common pneumoconioses. Insofar as silica dust impairs cellular defense, silica-exposed workers (without silicosis) may be at increased risk for fungal infections, as they are for mycobacterial infections (/).

Aspergillosis was the most common mycosis among persons with pneumoconiosis. Aspergillosis is a known complication in patients with underlying pulmonary disease, such as pulmonary tuberculosis and pneumoconiosis (2), in which silica-impaired macrophages are incapable of targeting inhaled conidia (3). The rarity of candidiasis in persons with silicosis may reflect the fact that healthy workers are less likely to have concurrent diabetes or HIV infection.

Concurrent mycosis was specific to decedents with silicosis compared with those with asbestosis or CWP, possibly because coal mine dust and asbestos fibers are less toxic to macrophages than are crystalline silica. Persons with asbestosis and CWP are also less commonly affected by autoimmune diseases and systemic immunologic complications than are those with silicosis (4,5).

Direct impairment of macrophage function by crystalline silica and poor drug penetration into silicotic lung nodules have resulted in high (>20%) treatment failure and relapse rates for patients with silicosis who are receiving chemotherapy for tuberculosis (6). This finding has prompted prolonged and more aggressive treatment of tuberculosis for such persons (7). Similarly, treatment of mycosis in...
patients with silicosis or substantial past exposure to silica dust may require prolonged treatment and possibly chronic suppressive antifungal therapy, as is used for patients with immunocompromised conditions (9).

Lacking population-based surveillance data for silicosis and mycosis illness and silica exposures, we relied on death certificate data, which have limitations; e.g., only \( \approx 1 \) of 6 persons who had silicosis had silicosis recorded as a cause of death on the death certificate (9). Also, many persons with substantial exposure to silica dust never receive a diagnosis of silicosis. We were unable to address the question of possible increased risk for mycosis among silica-exposed persons, and our analysis may underrepresent the actual extent of concurrent silicosis and mycosis. Another limitation was use of ICD-9 and ICD-10 coding for fungal infections and for fungal infection causing death. Sensitivity of this method can vary at different institutions and over time, especially if fungal infections are underdiagnosed. In addition, the ICD-9 classification codes for many mycoses do not differentiate pulmonary from other types of mycoses.

Health professionals should consider enhanced risk for mycosis with regard to preventive interventions, differential diagnosis, and mycosis treatment of silica-exposed workers. Measures to protect silica-exposed workers with coexposure to fungi include reducing silica exposure; wetting soil and bird droppings to suppress fungal-contaminated dust; maintaining good personal hygiene; and, in areas with endemic inhaled fungi, using enclosed operator cabs with high-efficiency particulate air filtration or personal respiratory protection for particulates (10).

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