Bioaerosol Lung Damage in a Worker with Repeated Exposure to Fungi in a Water-Damaged Building

Douglas Trout, Jonathan Bernstein, Kenneth Martinez, Raymond Biagi, and Kenneth Wellingford

Division of Surveillance, Hazard Evaluation and Field Studies, National Institute for Occupational Safety and Health, Centers for Disease Control and Prevention, Cincinnati, Ohio, USA; 2Division of Immunology, Allergy Section, University of Cincinnati College of Medicine and Technology, National Institute for Occupational Safety and Health, Centers for Disease Control and Prevention, Cincinnati, Ohio, USA

There has been increased concern over health effects related to potential exposure of building occupants to bioaerosols. We report the case of a worker with a respiratory illness related to bioaerosol exposure in a water-damaged building with extensive fungal contamination. We performed environmental tests to evaluate potential exposure to fungi, and we used mucociliary-specific antibodies to ascertain the extent to which environmental factors may affect respiratory health. The building was a four-story, concrete-frame structure with a steel-reinforced concrete floor. The building had a history of water damage and was being used as a hotel.

Case Presentation

Clinical evaluation. A 36-year-old white man, with a past history of pneumonia disease or tuberculosis, presented to his primary care physician in June 1998 with a 3-month history of dry cough and a 1-week history of fever and rhinorrhea. A chest X-ray revealed scattered rhinorrhea and rales on the right side, an initial scan X-ray was unremarkable. Over a period of several months, the patient was treated with several courses of antibiotics for a presumptive diagnosis of pneumonia. Pulmonary function testing (PFTs) revealed a reduced forced expiratory volume in the first second of expiration (FEV1), and total lung capacity (TLC). The diffusing capacity corrected for alveolar volume ([DLco/VA]) was normal.

Occupational history revealed that the patient had been a hotel manager at one hotel for the previous 14 years. Two months before the presentation, he had a co-worker begin management of the extent of water damage and fungal growth in the hotel room located where the patient had complained of water leakage and odors (Figure 1). These conditions included trapping water and making holes in the walls. Respiratory protection was worn. Shortly thereafter, the patient reported the onset of a nonproductive cough. The patient performed these tasks for 5 days until the hotel was closed in October 1998. 5 months after the initial assessment for water damage. However, in the primary supervising judge of the damaged areas, the patient continued to enter and all parts of the building. The patient had no prior history of exposure to farmers or other environments where it would have been exposed to excessive amounts of fungi.

Seven months after his initial presentation, the patient was referred for further evaluation. Dyspnea and cough were noted to be temporarily related to his presence in the building. His white blood cell counts and eosinophils were not normal. Histopathological examination was normal. Histoelectrophoretic and antibody responses to Histoplasma capsulatum were not elevated. A repeat chest X-ray was unremarkable. An initial high-resolution computed tomography (HRCT) of the chest with a combination challenge test was negative.

Received 18 December 2000; accepted 1 February 2001.
was performed and was successful (the cumulative dose of methotrexate [milligrams] that cause a 20% fall in FVC [25 mg/mL methotrexate]). A repeat FBC of the chest performed several weeks later with approximately equal adverse and excrecy was suggestive of an interstitial muscular pattern consistent with bronchiolitis. Symptom was minor for a moderate restrictive defect (Table 1). Based on the patient's history and laboratory findings, he was advised to completely avoid all exposure to his workplace (10 months after the initial investigation for work exposure).

The patient was subsequently referred for bronchoscopy. Transbronchial biopsy was unremarkable. Bronchoalveolar lavage (BAL) was not performed. The patient was given a provisional diagnosis of restrictive lung disease consistent with a chronic form of interstitial pneumonitis, and treatment was begun with high-dose prednisone (120 mg/day). Repeat bronchoscopy (6 months after the initial bronchoscopy) revealed 12% lymphocytes in the BAL fluid (normal ± 5%) and a CD4/CD8 ratio of 0.8 (normal reference ≥ 1). On the basis of continuing symptoms, evidence of advancing restrictive lung disease (partial relief removal from the workplace) and concerns over the side effects of prednisone, treatment with methotrexate was initiated at 10 mg/week and was subsequently increased to 7.5 mg twice weekly at the end of the year, which gradually decreased. Repeat FBC of the chest was normal. The patient's symptoms progressively improved, and his lung function remained stable (Table 1).

Environmental evaluation. In November 1996, we conducted environmental surveys including both air and surface sampling in a total of 19 rooms of the 10-story hotel. Results that had experienced water damage.

Table 1. Pulmonary function testing of lung function remained stable.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>FVC (L)</td>
<td>3.60 (60%)</td>
<td>3.48 (60%)</td>
<td>3.37 (60%)</td>
<td>3.10 (60%)</td>
<td>3.07 (60%)</td>
</tr>
<tr>
<td>FEV1 (L)</td>
<td>2.90 (60%)</td>
<td>2.78 (60%)</td>
<td>2.69 (60%)</td>
<td>2.66 (60%)</td>
<td>2.68 (60%)</td>
</tr>
<tr>
<td>FEV1/FVC</td>
<td>0.81 (100%)</td>
<td>0.81 (100%)</td>
<td>0.81 (100%)</td>
<td>0.81 (100%)</td>
<td>0.81 (100%)</td>
</tr>
<tr>
<td>DCO (L/min/kPa)</td>
<td>5.65 (71%)</td>
<td>5.65 (71%)</td>
<td>5.65 (71%)</td>
<td>5.65 (71%)</td>
<td>5.65 (71%)</td>
</tr>
<tr>
<td>TLC (L)</td>
<td>8.90 (60%)</td>
<td>8.90 (60%)</td>
<td>8.90 (60%)</td>
<td>8.90 (60%)</td>
<td>8.90 (60%)</td>
</tr>
<tr>
<td>N1, N2, N3</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
</tr>
</tbody>
</table>

From the table it is evident that the respiratory system was not affected by the exposure to the hotel environment.

Table 2. Results of air and surface lung function testing.

| Table 2. Results of air and surface lung function testing (n = 12) |
|---------------------|-----------------|-----------------|-----------------|
| Asbestos fibers | Median indoor | Lower level | Upper level | Median outdoor |
| Cigarette smoke | 1.00 (95% CI 2.30) | 1.00 (95% CI 2.30) | 1.00 (95% CI 2.30) | 1.00 (95% CI 2.30) |

The results of air and surface lung function testing are presented in Table 2. The samples were taken from representative groups of the hotel's employees. The results of the analysis of pollutants in the hotel environment were consistent with the results obtained in the outdoor air. The percentage of asbestos fibers in the indoor air was similar to the percentage of asbestos fibers in the outdoor air. The results of the analysis of cigarette smoke in the indoor air were consistent with the results obtained in the outdoor air. The results of the analysis of cigarette smoke in the outdoor air were consistent with the results obtained in the indoor air. The results of the analysis of cigarette smoke in the outdoor air were consistent with the results obtained in the indoor air. The results of the analysis of cigarette smoke in the outdoor air were consistent with the results obtained in the indoor air. The results of the analysis of cigarette smoke in the outdoor air were consistent with the results obtained in the indoor air.
Investigative. Mycotoxins produced by Stachybotrys chartarum for Minutary Oncological neutrophils were identified in 8 of 18 samples. The complex trichothecenes, satratoxin and violacein, produced by Stachybotrys chartarum, mixed spores, and violacein, produced by M. catenulatum, were also identified.

Results of the cultured air samples are presented in Table 2. Two of the samples were overloaded with fungi; growth in mushrooms could not be counted; results in Table 2 reflect the 12 samples that could be counted. In water-saturated areas, Penicillium sp. and Aspergillus sp. were the predominant species detected. Stachybotrys was identified from 5 of the 12 sampling locations. Results of the nonculturable techniques were consistent with those obtained by cultured sampling, with Stachybotrys identified from 13 of 14 sample locations.

Laboratory evaluation. Because there are currently no usable biological techniques to assess exposure to mycotoxins, we performed a pilot serologic evaluation using a test developed under theft by specific IgG and IgM antibodies to keratin is mediated by several different fungi including Stachybotrys. The detection does not appear to occur at an increased rate of occurrence.

In a study performed by the NIOSH Human Subjects Research Board, personnel from former hotel employees (n = 50) were evaluated for mycotoxin exposure by measuring IgG and IgM antibodies in their sera. There were no significant differences between groups, but the trend was toward a higher level of antibody in the exposed group.

Discussion

Fungi are ubiquitous in the environment, and most individuals are exposed to them on a daily basis. Many fungi are capable of producing mycotoxins, and some studies suggest that mycotoxins may have some degree of occupational exposure to mycotoxins. An important aspect of the mycotoxin exposure is the determination of whether the exposure is relevant to the mycotoxin. The exposure to mycotoxins is a complex issue, and factors such as exposure level, duration, and the presence of other substances may influence the outcome.

The mycotoxins produced by fungi are of concern because they can cause various health problems. The mycotoxins produced by fungi can cause respiratory, gastrointestinal, and neurological effects. The mycotoxins produced by fungi can also cause cancer and other chronic diseases.

The mycotoxins produced by fungi are of concern because they can cause various health problems. The mycotoxins produced by fungi can cause respiratory, gastrointestinal, and neurological effects. The mycotoxins produced by fungi can also cause cancer and other chronic diseases.

The mycotoxins produced by fungi are of concern because they can cause various health problems. The mycotoxins produced by fungi can cause respiratory, gastrointestinal, and neurological effects. The mycotoxins produced by fungi can also cause cancer and other chronic diseases.

The mycotoxins produced by fungi are of concern because they can cause various health problems. The mycotoxins produced by fungi can cause respiratory, gastrointestinal, and neurological effects. The mycotoxins produced by fungi can also cause cancer and other chronic diseases.

The mycotoxins produced by fungi are of concern because they can cause various health problems. The mycotoxins produced by fungi can cause respiratory, gastrointestinal, and neurological effects. The mycotoxins produced by fungi can also cause cancer and other chronic diseases.

The mycotoxins produced by fungi are of concern because they can cause various health problems. The mycotoxins produced by fungi can cause respiratory, gastrointestinal, and neurological effects. The mycotoxins produced by fungi can also cause cancer and other chronic diseases.

Figure 1. Water damage and fungal growth in a room with walls sheared from walls.

Figure 2. A pilot study of the mycotoxin exposure associated with the mycotoxin exposure in a group of people who were exposed to the mycotoxin exposure. The mycotoxins produced by fungi are of concern because they can cause various health problems. The mycotoxins produced by fungi can cause respiratory, gastrointestinal, and neurological effects. The mycotoxins produced by fungi can also cause cancer and other chronic diseases.

Although the mycotoxins produced by fungi are of concern because they can cause various health problems. The mycotoxins produced by fungi can cause respiratory, gastrointestinal, and neurological effects. The mycotoxins produced by fungi can also cause cancer and other chronic diseases.

Although the mycotoxins produced by fungi are of concern because they can cause various health problems. The mycotoxins produced by fungi can cause respiratory, gastrointestinal, and neurological effects. The mycotoxins produced by fungi can also cause cancer and other chronic diseases.

Although the mycotoxins produced by fungi are of concern because they can cause various health problems. The mycotoxins produced by fungi can cause respiratory, gastrointestinal, and neurological effects. The mycotoxins produced by fungi can also cause cancer and other chronic diseases.

Although the mycotoxins produced by fungi are of concern because they can cause various health problems. The mycotoxins produced by fungi can cause respiratory, gastrointestinal, and neurological effects. The mycotoxins produced by fungi can also cause cancer and other chronic diseases.

Although the mycotoxins produced by fungi are of concern because they can cause various health problems. The mycotoxins produced by fungi can cause respiratory, gastrointestinal, and neurological effects. The mycotoxins produced by fungi can also cause cancer and other chronic diseases.

Although the mycotoxins produced by fungi are of concern because they can cause various health problems. The mycotoxins produced by fungi can cause respiratory, gastrointestinal, and neurological effects. The mycotoxins produced by fungi can also cause cancer and other chronic diseases.
Figure 2. Approach to patients with suspected building-related disease from biochemical exposure: component clinical evaluation when history and physical examination is suggestive of respiratory illness.

Assessment of Acute Exposure

1. Aerosol exposure (inhaled irritant). Local irritation is a consistent feature of exposure. A history of exposure should also be elicited.

2. Blood count, respiratory function tests.


6. Immunologic test: skin test, lung function tests, measurement of serum markers.


Supportive Treatment

1. Antibiotics for primary or secondary infections.

2. Immunomodulators.

3. Immunosuppressants.

4. Supportive therapy for acute exacerbation of underlying disease.

Supplementary References

1. ASHRAE. Indoor and outdoor air quality. 1998. American Conference of Governmental Industrial Hygienists, Cincinnati, OH.


3. Dean J, Dean J, Dean J, Dean J, Dean J. The role of the lung in the development of acute respiratory symptoms associated with exposure to indoor pollutants. Inhalation Toxicology and Air Pollution 1997; 9:189-203.

4. ASHRAE. Indoor and outdoor air quality. 1998. American Conference of Governmental Industrial Hygienists, Cincinnati, OH.


6. Dean J, Dean J, Dean J, Dean J, Dean J. The role of the lung in the development of acute respiratory symptoms associated with exposure to indoor pollutants. Inhalation Toxicology and Air Pollution 1997; 9:189-203.

7. ASHRAE. Indoor and outdoor air quality. 1998. American Conference of Governmental Industrial Hygienists, Cincinnati, OH.


9. Dean J, Dean J, Dean J, Dean J, Dean J. The role of the lung in the development of acute respiratory symptoms associated with exposure to indoor pollutants. Inhalation Toxicology and Air Pollution 1997; 9:189-203.

10. ASHRAE. Indoor and outdoor air quality. 1998. American Conference of Governmental Industrial Hygienists, Cincinnati, OH.


12. Dean J, Dean J, Dean J, Dean J, Dean J. The role of the lung in the development of acute respiratory symptoms associated with exposure to indoor pollutants. Inhalation Toxicology and Air Pollution 1997; 9:189-203.

13. ASHRAE. Indoor and outdoor air quality. 1998. American Conference of Governmental Industrial Hygienists, Cincinnati, OH.


15. Dean J, Dean J, Dean J, Dean J, Dean J. The role of the lung in the development of acute respiratory symptoms associated with exposure to indoor pollutants. Inhalation Toxicology and Air Pollution 1997; 9:189-203.

16. ASHRAE. Indoor and outdoor air quality. 1998. American Conference of Governmental Industrial Hygienists, Cincinnati, OH.


18. Dean J, Dean J, Dean J, Dean J, Dean J. The role of the lung in the development of acute respiratory symptoms associated with exposure to indoor pollutants. Inhalation Toxicology and Air Pollution 1997; 9:189-203.

19. ASHRAE. Indoor and outdoor air quality. 1998. American Conference of Governmental Industrial Hygienists, Cincinnati, OH.


21. Dean J, Dean J, Dean J, Dean J, Dean J. The role of the lung in the development of acute respiratory symptoms associated with exposure to indoor pollutants. Inhalation Toxicology and Air Pollution 1997; 9:189-203.

22. ASHRAE. Indoor and outdoor air quality. 1998. American Conference of Governmental Industrial Hygienists, Cincinnati, OH.


24. Dean J, Dean J, Dean J, Dean J, Dean J. The role of the lung in the development of acute respiratory symptoms associated with exposure to indoor pollutants. Inhalation Toxicology and Air Pollution 1997; 9:189-203.

25. ASHRAE. Indoor and outdoor air quality. 1998. American Conference of Governmental Industrial Hygienists, Cincinnati, OH.


27. Dean J, Dean J, Dean J, Dean J, Dean J. The role of the lung in the development of acute respiratory symptoms associated with exposure to indoor pollutants. Inhalation Toxicology and Air Pollution 1997; 9:189-203.

28. ASHRAE. Indoor and outdoor air quality. 1998. American Conference of Governmental Industrial Hygienists, Cincinnati, OH.


30. Dean J, Dean J, Dean J, Dean J, Dean J. The role of the lung in the development of acute respiratory symptoms associated with exposure to indoor pollutants. Inhalation Toxicology and Air Pollution 1997; 9:189-203.