Nosocomial Fungal Infection During Hospital Renovation

ABSTRACT

Nosocomial fungal pulmonary infections (Zygomycetes, Aspergillus sp.) developed in two premature infants in a special care unit (SCU) adjacent to an area of renovation. Inspection showed that inadequate barriers permitted the passage of airborne particles between the two areas, and cultures confirmed a significantly higher (p≤0.05) density of mold spores in the SCU (0.88 cfu per hour per settling plate) compared to a construction-free comparison area (0.22 cfu per hour per settling plate). The major source of mold was the dust above the hospital's false ceiling. In another construction area, impervious barriers were shown to effectively restrict the dispersal of mold. Our experience adds Rhizopus to Aspergillus as a possible cause of construction-related nosocomial infection. Sporadic episodes will continue to occur until the hazards of renovation are appreciated and effective preventive measures are routinely instituted. [Infect Control 1985; 6(7):278-282.]

INTRODUCTION

Hospital construction is closely controlled by federal, state and local regulations. No guidelines are included for the protection of patients from exposure to the dissemination of fungi as a result of renovations. Although reports have appeared sporadically linking construction with aspergillosis, the risk is still not generally appreciated.

In this article we report fungal infections in two infants probably arising from air contamination by fungi from an adjacent renovation site. We also present some suggestions as to how to prevent such unfortunate occurrences.

DESCRIPTION OF THE OUTBREAK

Our hospital is a tertiary care center for newborn infants. It has a 20-bed Neonatal Special Care Unit (SCU) with facilities for the intensive and routine nursing care of newborns of all birthweights.

During the winter of 1982 to 1983, renovations were underway in an adjacent area for the creation of a separate neonatal intensive care unit. These renovations involved removing the false (drop) ceiling and the demolition of walls which together resulted in the generation of a large amount of dust. The construction area and the SCU were separated by a doorway over which a bed sheet was taped to provide a dust barrier.

While renovations were underway, two premature infants were found to have developed fungal infections. The first was born at another hospital and transferred for management of respiratory distress syndrome. He was treated with assisted ventilation, antibiotics for presumed sepsis, and multiple exchange transfusions for hyperbilirubinemia. The hospital course was complicated by eosinophilia of as much as 42% of 7,600 leukocytes per cu mm. He died on the 45th hospital day, following the development of a desquamating rash, disseminated intravascular coagulation, bradycardia, hypotension and oliguric renal failure. An antemortem skin biopsy showed...
fungal invasion, and *Candida parapsilosis* was recovered on culture. At autopsy, an unsuspected finding was a well developed 0.5 cm abscess in the left lower lobe, which on histologic examination demonstrated the presence of non-candida hyphal elements most of which were characteristic of *Aspergillus*. However, some were also characteristic of *Zygomycetes* (Figure 1). Postmortem cultures of blood, peritoneal and spinal fluids grew *Candida parapsilosis*; however, its significance is uncertain due to the 89-hour interval between death and postmortem examination.

The second infant was transferred to our hospital for ligation of a patent ductus arteriosus. Postoperatively the patient developed oliguric renal failure, pneumothorax, and an exfoliative skin rash. He became hypotensive and anoxic and was treated with assisted ventilation. Antibiotics were administered for presumed sepsis. The infant developed neutropenia. A bone marrow aspiration revealed hypocellular marrow, and he was treated with methylprednisolone (2 mg/kg/day). Skin biopsy revealed fungal elements and *C. parapsilosis* was recovered. Amphotericin B was instituted. Concurrently he developed bilateral hazy infiltrates on chest roentgenogram. The patient died after a postoperative course of 14 days. Postmortem examination 14 hours after death revealed extensive pulmonary infarction, and histologic examination demonstrated widespread evidence of both septate and nonseptate hyphae, with nonseptate hyphae predominating (Figure 2). Culture of the lung at autopsy grew *Rhizopus indicus*. (Speciation was performed by Dr. Irene Weitzman, New York City Department of Health). Cerebrospinal fluid culture was positive for *Cryptococcus laurentii*. The autopsy consent did not include permission to examine the intracranial contents, and no other examination of the cerebrospinal fluid was performed.

**COMMENT**

Both of these sick premature infants had their hospital courses complicated by cutaneous and systemic *C. parapsilosis* infection. *Candida* may have been the cause of death in each. However, autopsy demonstrated that in the first infant there was evidence of an antemortem 0.5 cm non-candidal fungal pulmonary abscess and in the second infant there was an extensive non-candidal fungal pneumonitis. The significance of the isolation of an unusual species of *Cryptococcus* from the spinal fluid in the second case is unknown.

**EPIDEMIOLOGIC INVESTIGATION**

These non-candidal fungal infections were unique in the experience of the Departments of Pediatrics and Pediatric Pathology. Review of autopsies of patients from Bellvue's newborn services from 1948 to the present failed to uncover a single newborn with infection due to *Aspergillus* or *Zygomycetes*. In that 35-year period autopsy records of 16 newborns indicated *Candida* infection while two immunocompromised older children had autopsy evidence of invasive *Aspergillus* infection.

The occurrence of the two unprecedented fungal pulmonary infections during the renovation period suggested the possibility of environmental contamination. Inspection of the construction area indicated that the barrier that separated it from the SCU was ineffective because it was not made of impervious material. In addition it had been repeatedly violated by hospital staff and construction workers. There was visible construction dust in the SCU. The unit was evacuated while a new area was
readied; studies were performed to quantitate the contamination of the SCU air by fungi and bacteria. Thirty or 60-minute settling plates consisting of trypticase soy agar supplemented with 5% sheep blood were placed in the SCU while patients were still present and receiving care. The unit was then evacuated, and additional settling plates were obtained while the unit was being cleaned. A third sampling was taken after the completion of cleaning but before returning the infants to the unit. For comparison, similar plates were obtained from a general medical unit and medical intensive care unit where no renovations were underway. Before cleaning, the settling rate of fungi in the SCU was 0.88 cfu per plate per hour. During cleaning the rate rose to 2.7, but after completion of cleaning the rate fell to 0.16, a figure similar to that of the construction-free control area and significantly lower than that in the SCU before cleaning (Table 1). In addition, 50% of the isolates from the SCU were *Mucor* or *Aspergillus* species (Table 2). Rodac plates (Falcon, Cockeysville, MD) prepared with Sabaroud’s agar were touch imprinted on environmental surfaces and the recovery of molds quantitated. *Mucor, Rhizopus*, and *Aspergillus* were found in cultures of the dust above the false ceiling in the SCU. The density of molds was greater in dust above the ceiling (4.7/sq cm) than in dust on walls, counter tops, and floors (0.46/sq cm).

Upon recognition of the outbreak, patients were moved to an area of the hospital where no reconstruction was underway. Additional dampers were placed in the air ducts of the nursery to permit the isolation of airflow to each of the individual rooms in the unit. Rigid impervious dust barriers were erected to prevent the spread of spores. Areas above the false ceilings were vacuumed as were all ventilation ducts. Air ducts and environmental surfaces were disinfected and the high efficiency particulate air (HEPA) filter that serves the unit was replaced.

The effectiveness of this approach was assessed by prospectively placing settling plates inside and outside barriers demarcating a construction zone from a patient care area in another part of the hospital (Table 3). Air contamination in the SCU and an adjacent control area were reassessed 1 year later and were again found to contain low levels of fungi and bacteria similar to those obtained after disinfecting the SCU (Table 1).

### DISCUSSION

Fungi are ubiquitous in the environment. In particular *Aspergillus* and *Zygomycetes* have been demonstrated in the air in some hospitals in up to 20% of culture attempts but rarely or not at all in others.²⁻⁴ Airborne transmission of *Aspergillus* to hospital patients has been previously reported. Three outbreaks have been related to contamination of incoming air by pigeon droppings. Gage et al described three cases of *Aspergillus* endocarditis following open heart surgery.⁵ Contamination of air in the operating room was attributed to pigeons nesting near the intakes of the hospital's ventilation system. Infections ceased after the roost was moved. Burton et al isolated *Aspergillus fumigatus* from the air ducts of an isolation room in which several renal transplant patients were believed to have acquired aspergillosis.⁶ Pigeon excreta were again implicated, and control measures resulted in a lowered incidence of infection. A malfunctioning air exhaust system, soiled with pigeon droppings, that allowed distribution of contaminated air was implicated in the outbreak reported by Kyriakides et al.⁷ Three other outbreaks have been related to ventilation systems in the absence of external contamination. Rose reported elimination of aspergillosis after moving from an older externally ventilated ward to a new unit which received prefilled unrecirculated air.⁸ Rosen and Steinberg also attributed a decline in the incidence of aspergillosis and zygomycosis to improved hospital ventilation.⁹ Mahoney et al, described 5 cases of nosocomial aspergillosis which occurred within a 2-month period.¹⁰ The first case occurred 8 days after shutdown of the ventilation system for repairs, and the other cases all occurred in rooms supplied by the same ductwork. *Aspergillus* had previously been found on moist air conditioning coils, exhaust systems and filters. After cleaning the system and rooms the incidence of aspergillosis fell to the previous low level.

Aisner et al associated invasive aspergillosis with
environmental contamination from hospital construction.\(^{11}\) Eight cases occurred shortly after moving to a new facility. External contamination seemed unlikely because high efficiency filters were used, air was not recirculated, and spores could not be detected in air ducts. Dust from above false ceilings on pipes and ceiling panels contained multiple species of *Aspergillus*. Moreover, fireproofing that had been installed wet was found to be contaminated with *Aspergillus* and to support its growth.

The careful observations of Arow et al implicated hospital renovations on the floors above a renal transplant unit in the genesis of nosocomial aspergillosis.\(^{12}\) Vibrations caused dust to be dislodged and filter through holes in false ceilings to contaminate the air of corridors and patient rooms. Air quality was poorest in the area of renovation and on the floor immediately below. Fungi were only occasionally isolated from other areas of the hospital.

The environment has long been thought to be a source of *Aspergillus* infection particularly for workers who are occupationally exposed.\(^{13}\) The environment has also been suggested as a source of nosocomial infection with *Zygomycetes*\(^{14,15}\) though to our knowledge no clusters of respiratory infection have been reported.

Doubt has been cast upon the importance of airborne transmission of fungal spores in hospitals by the fact that fungal organisms can be recovered from the nasopharynx or sputum of normal adults, suggesting that unmasking of a latent endogenous infection may be the cause.\(^{16}\) This is unlikely in the cases we observed because the infants were both neonates who were hospitalized throughout their brief lives. It is more likely that these were primary fungal pulmonary infections following inhalation. *Aspergillus* spores with a mean diameter of 3.5 μ, and *Rhizopus* spores with a mean diameter of 6 μ are respirable particles that can reach the terminal airways, impact on and remain in alveoli.\(^{17}\) Our studies suggest that at the time of our measurements, near the end of the construction period, the air in the SCU contained at least a fourfold increase of these respirable fungi compared to a control area distant from construction. Air quality returned to control levels after cleaning and establishing an effective barrier. At least one of the infections we observed occurred with a particularly unusual species of fungus, *R. indicus*, which was also demonstrated in environmental cultures together with *Aspergillus*. Taken together these observations strongly suggest an association between hospital renovation and the infections. In particular the removal of the false ceiling can be seen as particularly likely to have played a role in increasing the number of spores in the air.

The *Candida* infections in our patients were also nosocomial in origin. Infection with this organism was probably not related to air contamination secondary to hospital construction for two reasons: 1) the known ecology and epidemiology of *Candida* and, 2) the failure to recover *Candida* in environmental samples. The isolate of *Cryptococcus laurentii* from the second infant cannot be evaluated, and cryptococi were not identified in environmental cultures.

The validity and reliability of results of volumetric air sampling are believed by many to be superior to those obtained by gravity settling plates. Volumetric air sampling is the more sensitive of the two insofar as it does recover a larger number of fungi per unit of sampling time. By comparison gravity settling plates underestimate airborne fungi.\(^{3}\) However, the proportion of recovered fungi with spore sizes capable of lung penetration is similar by each method, 88% vs. 95% for gravity plates and volumetric sampling respectively.

Our review of the literature failed to uncover any study showing that air contamination as measured by volumetric methods is more predictive of human infection than that measured by settling plates. We agree with LB Hall that "almost any air sampler when its characteristics are recognized and understood will give satisfactory results in the hands of the investigator who uses it day after day."\(^{18}\) Of the eight papers\(^{5-12}\) relating nosocomial aspergillosis outbreaks to the environment only one used volumetric air samples. In that paper,\(^{12}\) where heavy contamination existed, the plates were overgrown and colonies could not be enumerated. Four of the papers\(^{5,7,8,11}\) used gravity settling plates and environmental swab cultures, and three\(^{9,10}\) did not attempt or report cultures. It is difficult to compare the results of one institution to another because of differences in methods of sampling. Furthermore, there are no recognized standards for an acceptable number of fungal spores in the air.

Our data show that even with the lower sensitivity of the settling plate method there was evidence that during the renovations, the environmental air contained larger numbers of spores than were generally present within the institution and that the degree of contamination was reduced when appropriate measures were taken. Moreover, more than 50% of isolates recovered were *Aspergillus* or *Rhizopus*. This is in marked contrast to isolates from control areas as well as to the observations of Sayer et al who studied hospital air using both volumetric and gravity settling methods.\(^{3}\) In that study *Aspergillus* and *Rhizopus* constituted 9.6% of all fungal isolates when measured by volumetric methods and 5.6% when measured by the gravity settling method. Our data are in accord with the data of others showing that unusually high levels of contamination may be associated with the occurrence of invasive disease.\(^{5,7,8,11,12}\) They should not be interpreted

### TABLE 3

<table>
<thead>
<tr>
<th>Location</th>
<th>Plates (n)</th>
<th>Fungi</th>
<th>Bacteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inside Barrier</td>
<td>5</td>
<td>10.8*</td>
<td>29.6</td>
</tr>
<tr>
<td>Outside Barrier</td>
<td>5</td>
<td>1.2</td>
<td>14.4</td>
</tr>
</tbody>
</table>

*cfu/plate hour.
†Chi2=21.04; p<0.001.
as helpful in establishing specific criteria for environmental cleanliness.

Hospital construction is closely regulated by federal, state and local guidelines. However, there are no guidelines for the protection of patients during the process of remodeling. In the absence of additional data on which to base recommendations we would like to offer the following interim suggestions. They are adapted in part from the recommendations of Arnow et al to isolate patients from the hazards and nuisances of remodeling.\(^\text{12}\)

1. Establish impervious barriers between patient and construction areas to prevent dissemination of dust.
2. Vacuum the area above false ceilings, including duct work and pipes prior to construction.
3. Establish traffic control patterns which prevent construction dust from being tracked into patient areas.
4. Ventilate construction areas with negative pressure with respect to adjacent patient care areas. If possible exhaust air from construction areas directly outside the hospital.
5. Carefully clean construction areas before occupancy. Additionally we recommend that when renovation is contemplated infection control practitioners be included at an early stage of planning, prior to contracting of work, to review areas of potential risk and recommend additional control measures.

We hope this report will stimulate further investigation on standards of air quality relating to nonbacterial microorganisms, and their role in nosocomial infection.

REFERENCES


282 Readers' Forum/Krasinski et al

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