A Nosocomial Outbreak of *Mycobacterium tuberculosis*

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Background. The national incidence of tuberculosis (TB) is increasing, and hospitals are a site of transmission. We investigated a nosocomial outbreak of TB at a 160-bed community hospital in South Carolina that highlights the central role that primary care physicians must play to control this epidemic.

Methods. We reviewed medical records to identify potential source cases. We retrospectively evaluated exposures to suspected source patients and the subsequent tuberculin reactivity of the 38 hospital employees who had a previous negative tuberculin skin test and were assigned to the ward where the outbreak began. We also evaluated the out-of-hospital contacts of TB cases.

Results. A review of medical records identified one patient who had died of prostate cancer and chronic cavitary pneumonia but was never in isolation nor evaluated for TB. Ward employees who worked while this patient was hospitalized had an increased risk for skin-test conversion (43% [12 of 28] vs 0% [0 of 9]; relative risk undefined; $P=.02$). Among employees who worked with this patient, skin-test converters worked more shifts with (median, 10.5 vs 7), dispensed more medication to (median 7 doses vs 1), and wrote more notes on (median 18 vs 5) the index patient than did nonconverters. Five of 12 of the patient's close out-of-hospital contacts had newly recognized positive tuberculin skin tests. Among 20 casual contacts, there were no new skin-test conversions.

Conclusions. A high index of suspicion, prompt isolation and diagnostic testing of potentially infectious hospitalized patients, and a thorough investigation of contacts of patients with TB are needed to prevent TB transmission.

Key words. Cross infection; tuberculosis; *Mycobacterium tuberculosis*; disease outbreaks. (*J Fam Pract 1994; 39:21-25*)

After three decades of steady decline, the incidence of tuberculosis (TB) in the United States increased 11% between 1985 and 1990.[1] Most observers believe that much of this increase is related to the human immunodeficiency virus/acquired immune deficiency syndrome (HIV/AIDS) epidemic.[2-4] Previous outbreaks have demonstrated that hospitalized patients are an important source of transmission.[5-8] With an increasing prevalence of HIV-infected persons[9] and an increased incidence of TB, we can expect more opportunities for significant nosocomial outbreaks. We report a nosocomial outbreak of TB that did not involve HIV-infected persons but underscores the importance of physicians considering the diagnosis of pulmonary TB.

In August 1991, the South Carolina Department of Health and Environmental Control learned that a hospital nurse, Nurse X, had a diagnosis of active pulmonary TB with many acid-fast bacilli in her sputum. Nurse X worked on the general medical ward of a 160-bed community hospital in South Carolina (Hospital A). When questioned, the employee health nurse at this institution reported that during their annual employee tuberculin skin-testing program in December 1990, an unusually large number of employees, most of whom worked on Ward W, had tuberculin skin-test conversions. Although tuberculosis is a reportable disease in South Carolina, skin-test reactivity to tuberculin antigen is not, so Hospi-

Submitted, revised, March 24, 1994.

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ISSN 0094-3509

tal A had not previously contacted the health department. We initiated an investigation to determine the source of the outbreak and to develop recommendations for control measures.

Methods

We reviewed the records of tuberculin skin-test results from all Hospital A employees. We defined a skin-test converter as a person who had at least one previous unreactive Mantoux tuberculin skin test (no measurable induration 48 to 72 hours after administration) that, on retesting at a later date, was reactive (induration ≥10 mm, 48 to 72 hours after administration), as documented by the hospital's employee health nurse.

We searched hospital billing records and identified all patients who were admitted to Hospital A between October 1, 1989, and December 1, 1990, and had a discharge diagnosis of TB. We also reviewed county health department records of patients with TB and compared them with hospital logs. As the focus of our investigation narrowed, we reviewed the medical records of all patients who had been admitted to Ward W between June 15, 1990, and October 1, 1990, had a diagnosis of pneumonia, and subsequently died.

We reviewed the work schedules of Ward W employees and counted the number of shifts they worked with each patient known or suspected to have TB.

The county health department performed tuberculin skin testing of the out-of-hospital contacts of suspect cases. Persons sharing a household or frequently sharing the same room or car with the patient were classified as close contacts. Persons with some, but less regular and frequent exposure were classified as casual contacts. We classified persons with induration ≥5 mm on tuberculin skin testing as reactors. We reviewed the records of contact investigations of patients with a diagnosis of TB.

Using Epi Info[10] software, we compared proportions between groups using chi-square or, where appropriate, the two-tailed Fisher's exact test.

Results

There was a marked increase in the number and the rate of tuberculin skin-test conversions among Hospital A employees in 1990 (Figure). Eleven of the 17 converters in 1990 worked on Ward W, the 30-bed general medical ward where Nurse X, the index case, worked. Ward W employees were significantly more likely to have converted their skin test than were non–Ward W employees (11 of 39 [28%] vs 4 of 337 [1%]; $P<.001$). In the 5 years

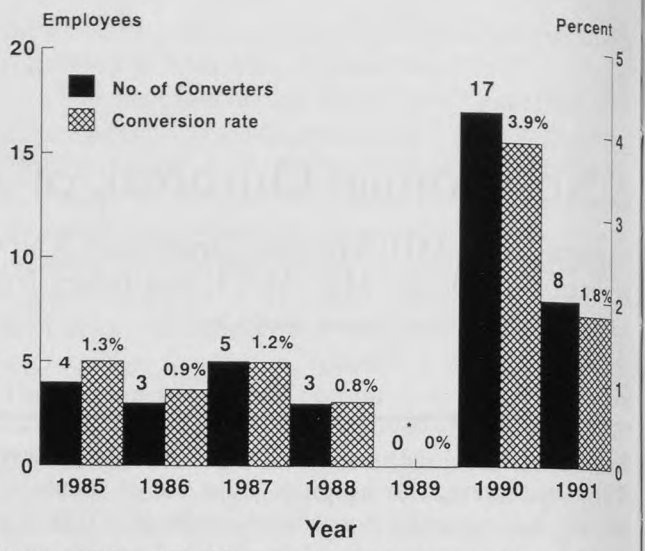

Figure. Tuberculin skin test conversions among Hospital A employees, South Carolina, 1985–1991. *0 of 358 employees tested in 1989 were converters.

before 1990, there had not been a single skin-test conversion among Ward W employees. One Ward W employee was found to have converted her skin test in August 1991, but because she had missed her 1990 appointment for tuberculin skin-testing, she was not included in the 1990 count. For analysis, she was included as part of the Ward W converters.

All Ward W employees who converted their skin test were directly involved in patient care. Seven were patient care nurses, two were nursing assistants, two were orderlies, and one nurse coordinated discharge services for patients. The four non–Ward W employees who converted their skin tests were not as directly involved in patient care. One each worked as a housekeeper, a dietary assistant, a security guard, and a laundry washer. Three of the Ward W converters had chest radiographs consistent with pulmonary TB. Nurse X had a normal chest radiograph in December 1990, but in July 1991, she had an infiltrate on chest radiograph as well as a sputum specimen that grew 4+ *Mycobacterium tuberculosis*. The other two employees had small pulmonary infiltrates consistent with TB in December 1990, but neither of them had a cough or sputum cultures that grew *M tuberculosis*, and none of their out-of-hospital contacts were tuberculin reactors.

The number of Hospital A patients with a diagnosis of TB at discharge was only slightly higher in 1990 (n=17) than in previous years (eg, n=11 in 1989; Table). Record review identified 13 patients who had been hospitalized on Ward W between October 1, 1989, and December 1, 1990, and discharged with a diagnosis of tuberculosis. No patients with a diagnosis of TB were

Table. Patients with Discharge Diagnosis of TB at Hospital A, by Year

Year	Number of Patients
1986	9
1987	5
1988	14
1989	11
1990	17

hospitalized during a time when more than 9 of the 12 Ward W converters worked at least one shift.

We next identified seven Ward W patients who had a diagnosis of pneumonia and died between June 15, 1990, and October 1, 1990. We chose this time frame because it was the only one during which all 12 Ward W converters shared working hours. One patient's presentation was particularly consistent with TB. Patient A was a 79-year-old man with metastatic prostatic carcinoma who was being treated with radiation therapy, doxorubicin hydrochloride, and prednisone. A chest radiograph on May 22 revealed "a shadow" in the left lower lobe. On June 22, a chest radiograph showed a larger infiltrate in the left lower lung field. On July 10, the patient was admitted to Ward W for mental status changes and fever. His cough, which produced copious sputum, was mentioned 25 times in the hospital chart. A chest radiograph on July 13 was noted to have a persistent dense consolidating alveolar left mid- and lower-lung infiltrate with "questionable areas of central cavitation."

Patient A's only diagnostic sputum specimen was collected on July 14, 1990, 4 days after beginning intravenous ceftriaxone sodium. The Gram stain showed numerous epithelial cells and few polymorphonuclear leukocytes. The culture grew *Enterobacter cloacae* and *Candida albicans*. No sputum was evaluated by acid-fast stain or cultured for mycobacteria. Patient A was not tuberculin skin-tested. He died of progressive respiratory insufficiency on July 25, 1990.

Among the 38 Ward W employees who worked between June 1 and October 1, 1990, those who were working while Patient A was hospitalized had an increased risk for skin-test conversion (43% [12 of 28] vs 0% [0 of 9]; relative risk undefined; $P=.02$). All 12 Ward W converters worked at least six shifts on Ward W during Patient A's hospitalization. Converters worked a median of 10.5 shifts during Patient A's hospitalization compared with a median of 7 shifts among nonconverters. The seven patient care nurses who were converters and had worked on Ward W during Patient A's hospitalization had administered a median of 7 medications and written a median of 18 notes in his chart. The nine patient care nurses who were not converters but had shared shifts with Patient A had administered a median of one medication and written a median of five notes in his chart.

County health department investigators identified 15 of Patient A's close out-of-hospital contacts. Three of these had a history of reactive tuberculin skin tests. Of the 12 without a history of a positive tuberculin test, five (42%) had reactive skin tests. There were no reactors among 20 of Patient A's casual contacts.

Patient A's contact investigation was further notable because his next door neighbor, designated as Case C, was his nephew. Case C had experienced several months of cough, fatigue, weight loss, and fever before dying of untreated pulmonary TB in December 1989 at 41 years of age. Of his 48 contacts who were tuberculin skin-tested by the health department after his death, 14 (29%) had reactive skin tests and three had developed pulmonary TB. Before his death, Case C spent several hours each week visiting and playing cards with Patient A. During the investigation of Case C's contacts, Patient A was not named and therefore was not skin-tested.

Nurse X, the index case, was identified as a converter in December 1990. In January 1991, she began isoniazid preventive therapy through her private physician, but she discontinued therapy after a few weeks because "it made me feel bad." Of the 11 Ward W converters, only two had completed the recommended course of isoniazid at the time of the August 1991 investigation. Of the remaining nine, two never followed up with the health department or their physician, one refused isoniazid, and six started but never completed a course of isoniazid.

Ward W had two respiratory isolation rooms with double doors and independent ventilation that was 100% exhausted. The isolation rooms had negative airflow when the doors were properly shut, although we noted them to be propped open with trash cans on several occasions. In all other patient rooms and ward work areas, air was filtered and 80% recirculated as part of the hospital-wide ventilation system. There had been no major changes in the physical layout of the ward or in the ventilation system since the hospital was constructed 10 years earlier. Patients were placed in the isolation room only if their attending physician specifically ordered it. Patient A was never placed in an isolation room.

Discussion

Patient A, who never had a diagnosis of TB, was the likely source case for most or all of this outbreak. While he was immunosuppressed, Patient A had regular exposure to a person with active pulmonary TB who infected his close contacts. Patient A's illness was clinically compatible with TB. Employees who had more contact with this patient

were more likely to be converters. Persons with close contact to Patient A outside the hospital were more likely to be newly recognized reactors than were those with only casual contact.

It is possible but unlikely that several source patients spreading TB to the staff could have caused this cluster. The number of patients with TB treated at the hospital in 1990 was not significantly more than that of previous years. Most patients with TB were hospitalized on Ward W, yet the annual employee skin-test program had detected no skin-test conversions on Ward W during the 5 years preceding the outbreak. Considering that there had been no transmissions to staff in the several years during which patients with TB had been cared for on Ward W and that there had been no changes in the physical layout, ventilation system, or isolation procedures since the hospital opened, it is unlikely that the transmissions were from multiple patients during the year of the outbreak.

Nurse X was not the source of the outbreak. She was among the 11 Ward W converters identified in December 1990. At that time, she did not have a cough and her chest radiograph was normal, and therefore she was not capable of transmitting TB in the weeks or months before December 1990. Similarly, the other two employees who had chest radiograph changes consistent with pulmonary TB in December 1990 were not clinically infectious and did not infect their out-of-hospital contacts.

This outbreak illustrates the risk of not considering the diagnosis of TB. Unrecognized cases of TB can spread disease more readily than suspected cases because unrecognized patients are not placed in respiratory isolation or treated with medication that will make them less infectious.[11] Older autopsy series suggest that one half of active cases of TB are not diagnosed during the patient's life.[12–14] More recently, researchers at a Los Angeles general hospital noted that 52% of hospitalized HIV-infected patients in whom TB was ultimately diagnosed did not have such a diagnosis before death or in their first 3 weeks of hospitalization.[15] If tuberculosis had been considered in Patient A, it could have been diagnosed and the outbreak prevented.

With the national increase in TB incidence, all physicians, and especially those working in primary care, need to recognize that TB is a diagnostic possibility, particularly in patients who have persistent cough, weight loss, anorexia, fever, and abnormal chest radiographs.[16] The basic tests required for diagnosis (tuberculin skin-testing and acid-fast smears and mycobacterial cultures) are inexpensive and widely available. Patients in whom TB is suspected should be placed in acid-fast bacilli (AFB) isolation until three sputum examinations are negative for AFB, or until they are placed on antituberculous chemotherapy and are responding clinically, and the concentration of organisms in their sputum is decreasing.[16]

If all skin-test reactors had followed standard guidelines for isoniazid prophylaxis, Nurse X's case of TB and the secondary spread of infection to family members could have been prevented.[17] Primary care physicians can encourage infected patients to complete a course of isoniazid while monitoring them for drug side effects. The Centers for Disease Control and Prevention (CDC) recommends that health-care facility personnel who do not complete a full course of preventive therapy should be considered for reassignment.[16]

Other measures to interrupt transmission of TB include contact investigation and surveillance. Although Patient A was a contact of Case C, a patient known to have TB, Patient A was not named as a contact during the contact investigation. Effective contact tracing requires sufficient resources for local health departments to fully interview and skin-test all contacts. This contact investigation was difficult because the case patient had died, and therefore, the investigators were forced to rely on the recall of family and friends.

The CDC recommends that skin-test conversions among patients and health care facility personnel be analyzed regularly to estimate the risk of TB transmission and the effectiveness of infection control and screening practices.[16] Hospital A kept meticulous records on individual employees, but the focus was on the history and clinical management of each employee. In small hospitals, maintaining a separate log book of all employees who are tuberculin converters (listed by age, race, sex, job description, and job location) would provide an aggregate view and make geographic clustering more apparent. Also, skin-test programs that are staggered throughout the year (eg, on each employee's anniversary of employment) are more likely to result in early identification of an outbreak or cluster, thereby allowing more rapid widespread screening and effective secondary prevention.

This nosocomial TB outbreak was unusual in that its likely source was identifiable. The pattern of transmission identified in the investigation suggests that the prevention of nosocomial transmission depends not on new technologies, but rather on a high index of suspicion for TB among clinicians, rigorous adherence to current guidelines for isolation and isoniazid preventive therapy, and improved public health infrastructure for TB contact investigations.

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