Letters to the Editor

Tuberculin Skin Test Conversion Among Healthcare Workers After Exposure to a Patient With Pleural Effusion and Parenchymal Disease Unapparent on Chest X-Ray

To the Editor:

Tuberculosis (TB) remains an important health concern throughout the world, especially in developing nations. Recent projections suggest that approximately 15 million individuals in the United States are currently infected with *Mycobacterium tuberculosis*.[1] Rapid institution of airborne precautions has proven effective in decreasing nosocomial spread from patients with pulmonary TB,[2] but patients with pleural TB are perceived not to be at risk of transmitting TB.[2] We describe two healthcare workers who had tuberculin skin test (TST) conversions after being exposed to a patient with pleural TB and unapparent parenchymal disease.

A 75-year-old male nursing home resident was admitted to our university medical center after he pointed to his chest and appeared dyspneic to nursing home staff. He was also hypoxic but no cough was observed. Anteroposterior chest radiograph on admission revealed a large left-sided pleural effusion. A pulmonary parenchymal infiltrate could not be ruled out. Initial thoracentesis revealed 510 white blood cells/μL and 1,350 red blood cells/μL. The white blood cell differential was 54% polymorphonuclear leukocytes and 37% lymphocytes. Lactate dehydrogenase was 1,382 U/L and protein was 4.7 g/dL. Serum lactate dehydrogenase was 814 U/L and total protein was 5.6 g/dL. Pleural fluid was negative on acid-fast bacillus smear. Subsequent taps were not sent for acid-fast bacilli. A TST performed 10 months earlier in the nursing home was reported as nonreactive.

The patient was not placed on airborne precautions. Repeat chest roentgenograms continued to display a left lower lobe opacity. Computed tomography of the chest performed 4 days after admission confirmed a large pleural effusion and also showed consolidation in the left lower lobe and multiple, small, subcentimeter, poorly defined, ground glass nodular opacities throughout both lungs. A left-sided calcified hilar lymph node and an axillary node were also present. Seven days after admission, the patient died as a result of respiratory failure and shock. Two weeks after admission, the pleural fluid culture was positive for *M. tuberculosis*. At autopsy, caseating granulomas were noted in the lungs. Rhodamine stain was positive for acid-fast bacilli. A diagnosis of TB had been entertained, but the patient had never been treated with antimycobacterial therapy.

Approximately 7,000 healthcare workers are administered TSTs each year at our medical center. Only 6 inpatients with TB were cared for that year, resulting in 3 conversions, 2 of which followed exposure to this unisolated patient.

Forty-one individuals were exposed to the patient during his hospitalization. Of these, one house officer who assisted with a thoracentesis developed an 18-mm reaction to purified protein derivative on tuberculin skin testing performed 6 weeks after the exposure. The results of her chest roentgenogram were negative and she had no signs or symptoms of TB. She had had a negative TST 7 months prior to this exposure. In addition, a primary care nurse had a TST with a 22-mm induration at 3 to 4 weeks after exposure. She also had no symptoms or signs of TB and her chest radiograph had negative results. A TST had been negative 5 months before the exposure. This patient was the only known exposure to TB for these two healthcare workers. Prior to conversion, both employees denied being near homeless shelters, nursing homes, jails, prisons, hospice wards, and human immunodeficiency virus wards. Both individuals were offered and administered isoniazid.

Tuberculous pleural effusion is believed to be the result of delayed hypersensitivity to *M. tuberculosis* antigens from organisms and microbial products that have been released into the pleural space from caseous subpleural foci.[3,4] Therefore, isolated tuberculous pleural effusion is not thought to pose a risk of transmission. In a recent study, patients with tuberculous effusions had a concurrent infiltrate in 70% of the cases.[1] When there is a coexisting infiltrate, it may involve any lobe and upper lobe lesions may be cavitary.[3]

As demonstrated by this case, patients with tuberculous pleural effusions may have pulmonary parenchymal involvement that is not apparent on a chest roentgenogram. Airborne precautions should be instituted until the diagnosis of parenchymal pulmonary TB is excluded. This is especially true if the patient is coughing or has had a change in voice. Chest computed tomography can be helpful, as in this case, in revealing parenchymal lesions not obvious on chest x-ray.

REFERENCES

1. American Thoracic Society. Diagnostic standards and classification of tuberculosis in adults and children. *Am J Respir Crit Care Med* 2000;161:1376-1395.
2. Cookson ST, Jarvis WR. Prevention of nosocomial transmission of *Mycobacterium tuberculosis*. *Infect Dis Clin North Am* 1997;11:385-409.
3. Seibert AE, Haynes J Jr, Middleton R, Bass JB Jr. Tuberculous pleural effusion: twenty-year experience. *Chest* 1991;4:883-886.
4. Valdes L, Alvarez D, San Jose E, et al. Tuberculous pleurisy: a study of 254 patients. *Arch Intern Med* 1998;158:2017-2021.

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