Pulmonary Mycobacterium gordonae Infection in a Two-Year-Old Child: Case Report

Infection and neoplasia should be considered in the differential diagnosis when extensive pulmonary disease with bilateral pleural effusions and hilar adenopathy is present in a patient with fever and dyspnea. Although pulmonary disease is more often attributable to a single etiology, it is not unusual for infection to accompany a neoplasm. Mycobacterium gordonae is usually considered a non-pathogenic commensal, but it has been recognized as a pathogen in immunosuppressed patients [1]. We describe what is, to our knowledge, the first case of pulmonary disease due to M. gordonae in a child.

A 2-year-old girl was admitted to the hospital with fever and dyspnea. She had been well until 3 weeks before admission when she began to have intermittent fevers and constipation. Her fever became persistent (temperature to 40°C) and was associated with worsening dyspnea, lethargy, and anorexia during the week before admission. There was no history of cough, emesis, rhinorrhea, or weight loss, and immunizations were current. An aunt had cared for her since she was 5 months old, but she had frequent contact with her mother who was a multi substance abuser.

On admission, the patient was in mild respiratory distress. Vital signs were as follows: temperature, 39.8°C; respirations, 52; pulse, 182; and oxygen saturation, as determined by pulse oximetry, 92% while she was breathing room air. Mild subcostal retractions and weight loss, and immunizations were current. An aunt had cared for her since she was 5 months old, but she had frequent contact with her mother who was a multistuff substance abuser.

She remained febrile and dyspneic, and a left-sided thoracentesis was performed 48 hours later. Ten milliliters of bloody fluid were obtained, and analysis of this fluid revealed the following values: WBCs, 814/mm³ with 62% lymphocytes, 31% mesothelial cells, 3% histiocytes, 2% neutrophils, and 2% monocytes; RBCs, 750,000/mm³; leukocyte count was 10,000/mm³ with 50% neutrophils, 38% lymphocytes, 9% monocytes, and 3% band forms; the hemoglobin level was 9.7 g/dL, and the platelet count was 383,000/mm³. A chest radiograph revealed bilateral pleural effusions (larger on the right side than on the left), hilar adenopathy, and an opacity at the right lung base. Supplemental oxygen and intravenous cefuroxime (150 mg [kg · d] divided q8h) were administered, and a tuberculin test with PPD and a mumps control was administered. She remained febrile and dyspneic, and a left-sided thoracentesis was performed 48 hours later. Ten milliliters of bloody fluid were obtained, and analysis of this fluid revealed the following values: WBCs, 814/mm³ with 62% lymphocytes, 31% mesothelial cells, 3% histiocytes, 2% neutrophils, and 2% monocytes; RBCs, 750,000/mm³; glucose, 90 mg/dL; lactate dehydrogenase, 970 U/mL; protein, 3.7 g/dL; and pH, 8.5. Gram staining did not reveal any organisms. A CT scan of the chest revealed extensive mediastinal and hilar adenopathy, multiple round masses within the pulmonary parenchyma and pleura, and bilateral pleural effusions. The mumps and PPD intradermal tests were nonreactive. A CT scan of the abdomen and pelvis revealed a round, noncalcified presacral mass (6.5 cm in diameter) displacing the rectum and bladder anteriorly; associated destruction of the inferior sacrum and marked adenopathy surrounding the superior mesenteric vessels were observed. Smears of gastric aspirates, leukems, and pleural fluid showed many acid-fast bacilli. Therapy with isoniazid (10 mg/[kg · d] po), rifampin (20 mg/[kg · d] po), pyrazinamide (20 mg/[kg · d] im), and streptomycin was begun for presumed pulmonary tuberculosis.

Because it was unlikely that the abdominal mass was tuberculous in origin, the patient underwent thoracoscopy and biopsy of the left pleura, which revealed a malignant endodermal sinus tumor involving the pleura and the left apical parenchyma; granulomatous disease was not seen. The serum level of α-fetoprotein was >350,000 ng/mL. Treatment with etoposide, carboplatin, and bleomycin was begun on the 13th hospital day. She remained febrile until hospital day 24 and was discharged 9 days later at which time ethambutol (15 mg/[kg · d] po) was substituted for streptomycin in the regimen.

Six weeks after thoracentesis was performed, her pleural isolate was determined to be M. gordonae by DNA probe analysis (Gen-Probe, San Diego). Ethambutol therapy was discontinued after 12 weeks. The remaining portion of her sacrococcyx was removed 2 weeks later; evidence of residual tumor was not found. On completion of mycobacterial susceptibility testing, her regimen was changed to rifampin and clarithromycin (15 mg/[kg · d] po). A CT scan of the chest showed resolution of adenopathy, masses, and nodules with only minimal residual effusion and subsegmental atelectasis. Subsequent smears and cultures of gastric aspirates were negative for M. gordonae. Therapy with antimycobacterial agents was discontinued after 5 months, and chemotherapy was completed 8 weeks later. Seventeen months after initial diagnosis findings on all imaging studies and her α-fetoprotein level were normal.

When smears from gastric aspirates, leukems, and pleural fluid were found to be positive for acid-fast bacilli, our suspicion of mycobacterial disease was confirmed. However, when the organism was identified as M. gordonae, a review of the literature was required to convince us of its role in this case. M. gordonae is a scotochromogen (Runyon Group II) that is commonly isolated from soil and water. It is usually considered an environmental contaminant when isolated from clinical specimens, since it has rarely been shown to cause significant disease. As a result, it was historically considered to be one of the least pathogenic mycobacteria. In a review of nontuberculous disease among patients with AIDS [2], M. gordonae was one of several mycobacteria that caused the cases (3%) not attributable to Mycobacterium avium complex.

Although isolation of this organism in clinical specimens does not immediately indicate disease, M. gordonae has been convincingly shown to cause a variety of clinical diseases such as arthritis, tenosynovitis, soft-tissue infection, osteomyelitis, and pneumonitis [1, 3]. M. gordonae has also been shown to disseminate to the CSF, and to peritoneal and pleural fluids and bone marrow as well as to hepatic, renal, and cardiac tissue [1, 3]. In a review by Weinberger et al. [1], several criteria were proposed to establish pathogenicity among organisms of low virulence. Supportive evidence in this case includes large numbers of organisms seen on direct smears; positive smears from three different sites including a sterile body site; illness consistent with mycobacterial disease; clinical response to antimycobacterial therapy; and smears and cultures that were negative for M. gordonae after treatment was completed.

The agents that have most consistently been active in vitro against M. gordonae have been ethambutol, rifabutin, clarithromycin, cycloserine, and the fluoroquinolones [1, 4, 5]. Clofazi-
mine, rifampin, ethionamide, kanamycin, and streptomycin have been active against more than half of the isolates tested, but only rare isolates are susceptible to isoniazid or pyrazinamide [1, 5]. The isolate from our patient was resistant to isoniazid and pyrazinamide. The long-term prognosis for patients with *M. gordonae* infection appears to be related to underlying disease and its resolution. Because our patient responded to treatment within weeks and had no evidence of residual disease, we assume the prognosis in her case will be determined by her response to cancer chemotherapy. 

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Peripartum Bacteremia with CDC Group HB-5 (*Pasteurella bettyae*)

The gram-negative lactose-nonfermenting bacillus CDC group HB-5, recently named *Pasteurella bettyae* [1, 2], is typically indole-positive, reduces nitrate, and produces acid from glucose and fructose. Growth occurs on a triple-sugar-iron slab and variably on MacConkey agar [3]. *P. bettyae* has been isolated from genitourinary sources [4–10] and blood [3, 9]. We report what we believe to be the first established case of *P. bettyae* bacteremia associated with peripartum fever.

A 25-year-old Haitian woman presented 1 day prior to hospital admission, at full term of her pregnancy, because of frequent contractions and no fetal movement. Her temperature was 37.3°C; heart rate, 104; respiratory rate, 20; and blood pressure, 120/74 mm Hg. Fetal heart tones were present at 142/min. On examination, membranes were intact. After brief observation, she was discharged.

The patient’s membranes ruptured spontaneously on the day of admission. The following day, the patient became febrile (temperatures to 39.4°C). Meconium-stained fluid was noted and labor was induced. Two sets of blood cultures (Septi-Chek, Roche Diagnostic Systems, Montclair, NJ; in Columbia broth and brain-heart infusion broth) were performed, and intravenous administration of ampicillin and gentamicin was begun. On the day following admission, she had a vaginal delivery of a 3,147-g boy with Apgar scores of 4 (at 1 minute) and 8 (at 5 minutes). Manual removal of the placenta was performed.

Both sets of blood cultures (including both aerobic bottles and one anaerobic bottle) yielded a gram-negative rod that was subsequently confirmed by the Massachusetts Department of Public Health State Laboratory Institute as *P. bettyae*. The anaerobic bottle also yielded *Veillonella parvula*.

Kirby-Bauer disk-diffusion testing against ampicillin, pipercillin, cephalothin, cefoxitin, ceftriaxone, imipenem, gentamicin, tobramycin, amikacin, ciprofloxacin, and trimethoprim-sulfamethoxazole yielded zone sizes that are established as susceptible for other aerobic gram-negative rods. The patient defervesced on the third hospital day. Antibiotic therapy was changed to administration of oral amoxicillin, and she was discharged on day 5.

Previous reports have documented recovery of *P. bettyae* from genitourinary sites, including genital ulcer exudates [5], Bartholin gland abscess [4, 9], penile discharge [4], cervix [3, 4], vagina [3, 4, 9], scrotal abscesses [7], and urine [6]. In addition, isolates have been recovered from amniotic fluid [3, 8] and placenta [3].

The isolation of *P. bettyae* from the blood has been rarely reported; only 5 of 59 isolates sent to the CDC [3] and 1 of 3 isolates submitted to the National Institute of Health (Tokyo) from 1970 to 1983 were from blood [9]. The growth of *P. bettyae* in two different sets of blood cultures makes it highly unlikely that this represents a contaminant. In view of the presence of this organism in the genitourinary tract in several other studies, it is not surprising that bacteremia in the setting of a peripartum infection could be caused by *P. bettyae*.

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