Abscess and Empyema Caused by *Legionella micdadei*

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*Legionella micdadei* is the second most common species implicated in the occurrence of *Legionella pneumonia* (D. J. Bremer, Semin. Respir. Infect. 4:190–205, 1987). Although there has been a reported lung abscess caused by dual infection (*L. micdadei* and *L. pneumophila*), there are no known cases of *L. micdadei* as the only causative organism. We report a case of a patient with a lung abscess from which *L. micdadei* was the sole organism isolated.

*Legionella micdadei* is the second most common species implicated in the occurrence of *Legionella pneumonia* (1). Although there is a report (2) of a lung abscess caused by a mixed infection of *L. micdadei* and *L. pneumophila*, no cases in which *L. micdadei* was the only pathogenic organism have been described. We report a case of a patient with a lung abscess in which *L. micdadei* was the sole etiological agent.

A 57-year-old white female was admitted to the hospital with the chief complaint of fever (maximum temperature, 40.0°C), a nonproductive cough, and edema of her lower extremities. She denied any chills or night sweats. Her past medical history was significant because of a fever of unknown etiology in 1983 which resolved untreated after 3 months. One year prior to admission, hepatosplenomegaly was noted. Physical examination and laboratory testing were unrevealing.

Physical examination on admission was significant for a clear chest examination, a 3/6 holosystolic murmur at the left upper sternal border, a palpable liver edge (14-cm span), a large spleen, and markedly edematous lower extremities.

Laboratory results showed a leukocyte count of 2,800/mm³, a hemoglobin level of 7.8 g/dl, and 92,000 platelets per mm³. Liver function tests showed a total bilirubin level of 1.3 mg/dl, an alkaline phosphatase level of 171 U/liters, a serum aspartate aminotransferase level of 90 U/liter, a serum alanine aminotransferase level of 35 U/liter, and a lactate dehydrogenase level of 234 U/liter. Prothrombin, partial thromboplastin, and bleeding times were normal. A chest X-ray obtained on admission showed no abnormalities.

An echocardiogram showed mild mitral valve regurgitation with left atrial enlargement, a liver biopsy revealed localized chronic inflammation and fibrosis with piecemeal necrosis, and a bone marrow biopsy showed noncaseating granulomas. She was started on steroids empirically and was discharged and given a prescription of 30 mg of prednisone twice a day. After discharge she did well at home for approximately 10 days, at which time she developed fever to 40.5°C, chills, back pain, and a dry, nonproductive cough. Ciprofloxacin, 500 mg orally twice a day, was started, but there was no clinical response after 2 days. A follow-up chest X-ray showed a left pleural effusion, and she returned to the emergency room.

At that time, physical examination was unchanged from that at the previous admission except for diminished breath sounds at the left base. Laboratory results were significant for abnormal liver function tests, a leukocyte count of 21,400/mm³, hemoglobin of 12.1 g/dl, and platelets of 143,000/mm³.

A chest tube was placed, with drainage of a purulent exudate showing a pH of 6.78, a leukocyte count of 141,000/mm³ (neutrophils, 74%; lymphocytes, 5%; monocytes, 9%; macrophages, 11%; mesothelial cells, 1%), a glucose level of 47 mg/dl, a lactate dehydrogenase level of 6,780 U/liter, and a total protein level of 4.5 g/dl. Cultures for bacteria, mycobacteria, yeasts, and fungi were performed. In addition, specimens were sent for virus isolation. Smears of the fluid were appropriately stained and examined for microorganisms. The procedure was complicated by laceration of the spleen with hemorrhage. She was transferred to the surgical intensive care unit, where, after numerous transfusions, her hemodynamic condition stabilized. She was started on gentamicin, 100 mg intravenously piggyback every 8 h, and clindamycin, 600 mg intravenously piggyback every 6 h. Because of the granulomas seen in the previous bone marrow biopsy specimens, ethambutol, 800 mg orally every day, and isoniazid, 300 mg orally every day, were started empirically. The patient was taken off steroids in a tapered manner.

A repeat computed tomography scan showed a new air-fluid cavity consistent with a lung abscess in the posterior segment of the left upper lobe; direct fluorescent-antibody results from the original pleural fluid specimen were positive for *L. micdadei* (negative for *L. pneumophila*). She was started on intravenous erythromycin, 1 g every 6 h. Medications for tuberculosis were continued, but ethambutol was changed to rifampin so that the medications were also directed at *Legionella* spp. Bacterial and tuberculous cultures were never positive; however, *L. micdadei* was cultured on buffered yeast charcoal extract medium and was confirmed serologically and by the lack of gelatin liquefaction, β-lactamase production, and autofluorescence. The patient was discharged after a 3-week course of erythromycin, and after 1 year of follow-up she is doing well.

Since the 1976 description of *L. pneumophila*, more than 30 species have been identified as belonging to the family Legionellaceae (1, 3, 4). Eighty-five percent of *Legionella* infections are due to *L. pneumophila*, and the remainder are due to other species. Of the remaining 15%, the most common species isolated has been *L. micdadei* (1).

The organism was first described in 1979 in renal trans-
plant recipients (6). The organism was shown to be identical to the previously identified agent reported by Tatlock (7) after the isolation of the bacterium from a presumably healthy patient with a mild febrile illness.

*L. micdadei* pneumonia most commonly occurs as a pneumonic process indistinguishable from that in patients with *L. pneumophila* infections. Patients frequently present with pleuritic chest pain, dyspnea, a nonproductive cough, and high temperatures. In a review, Muder et al. (5) described characteristic radiographic manifestations. They found that nodular infiltrates are infrequent and that multilobular involvement is uncommon, with little clinical correlation related to radiographic severity. Radiographic findings were more severe when there was simultaneous infection with *L. pneumophila* (5).

There has been a reported case of *L. micdadei* implicated in the formation of a lung abscess, but this was in concert with *L. pneumophila* (2). The case described here demonstrates that *L. micdadei* can be the sole pathogenic agent in a lung abscess.

REFERENCES