Community-Acquired Lung Abscess Caused by Legionella micdadei in a Myeloma Patient Receiving Thalidomide Treatment

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Legionella infection causes 2 to 14% of community-acquired pneumonia (CAP). Legionella micdadei constitutes <1% of these infections. We describe a case of cavitary L. micdadei CAP in a myeloma patient receiving thalidomide treatment. The importance of considering pneumonia and problems in diagnosing pneumonia caused by L. micdadei in this patient population are reviewed.

CASE REPORT

A 51-year-old male presented with fever with 2 weeks of coughing and increasing shortness of breath. On the night prior to admission, the patient described rigors and night sweats. There were no other symptoms and no recent history of travel or animal exposure. Coincident with the onset of the patient’s illness, family members also developed fever with mild upper-respiratory-tract symptoms and malaise. The patient had stage IIIA myeloma diagnosed in 2001 and was initially treated with autologous bone marrow transplantation. Disease recurrence in 2002 was managed with bortezomib (Velcade). Due to disease progression, therapy was changed to thalidomide, 200 mg/kg body weight daily, and prednisone, 10 mg daily. The myeloma was described as being stable over the preceding 3 months, and there was no history of hospital admission for over 6 months. The patient had a documented severe allergy to fluoroquinolones.

On admission, the patient had a temperature of 38.6°C, a heart rate of 48 beats/min, a respiratory rate of 22 breaths/min, and an oxygen saturation of 88% on room air. The chest examination revealed bronchial breath sounds and crackles to the left upper lobe. The remainder of the examination was normal, with the exception of the presence of oral thrush. The patient was admitted for investigations and started receiving vancomycin and ceftriaxone therapy. Blood tests were normal, with the exception of a mild anemia (hemoglobin, 119 g/liter) and lymphopenia (lymphocytes, 0.2 × 10⁹/liter). The patient was not neutropenic. A chest X ray revealed a large opacification consistent with lung consolidation or abscess (Fig. 1A). Computerized axial tomography of the chest confirmed a large left-upper-lobe cavitating lesion (Fig. 1B). Bronchoalveolar lavage (BAL) fluid culture was negative on Gram’s stain, acid-fast stain (done at a reference laboratory for mycobacteriology), and immunofluorescent stain for Pneumocystis carinii.

Since the initial blood cultures and respiratory samples were culture negative, the infectious disease service was consulted. Vancomycin therapy was discontinued, and azithromycin therapy was started. Test results for legionella urine antigen were negative. Transthoracic needle aspiration of the lesion and chest tube drainage of the effusion were performed. The effusion was exudative, with a lactate dehydrogenase level of 2,450 IU/liter. Modified acid-fast staining (7) tests of BAL fluid and lung aspirate were requested by the infectious diseases service. Both samples were positive for small coccobacilli with this stain (Fig. 2), and cultures subsequently grew Legionella micdadei (confirmed with partial 16S rRNA gene sequencing).

The patient improved within 6 to 7 days after the initiation of azithromycin therapy and made a complete recovery. Due to the family syndrome, which was similar to Pontiac fever, further questioning identified the use of a portable humidifier that was rarely cleaned. Samples of legionella culture taken from the humidifier were sent to a reference water testing laboratory, but results were reported as unreliable due to an overgrowth of multiple bacteria in the sample. Testing of the home water system was not performed.

Legionella infections cause 2 to 14% of community-acquired pneumonia (CAP) (4, 28, 30). In a recent study, Legionella micdadei was present in less than 1% of cases of community-acquired pneumonia (32). L. micdadei is more commonly identified as the cause of nosocomial pneumonia (18, 26). Cavitary pneumonia is reported in transplant patients (9, 12, 21) or human immunodeficiency virus (HIV)-infected individuals (15, 24). In a case series of renal transplant recipients with L. micdadei pneumonia, 18.5% (5 of 27) of the patients developed a lung abscess (29). There is one previous case of L. micdadei infection described in a patient with multiple myeloma (19) and one case of nosocomial L. micdadei pulmonary abscess and empyema formation reported in a nontransplant patient (13).

This case is unique in that we describe the first case of community-acquired cavitary L. micdadei pneumonia in a patient whose only known risks were underlying myeloma and the use of low-dose prednisone. More importantly, there are no other cases in the literature that describe L. micdadei infection
in the setting of thalidomide therapy for multiple myeloma. Thalidomide and its immunomodulatory analogues are being used increasingly to treat malignancies (2) and for the treatment of other diseases (11). Thalidomide has been associated previously with lung toxicity (5), which may in itself predispose patients to pulmonary infections. Whether the use of this drug alone or in combination with steroids increases the risk of opportunistic infections remains to be proven. However, *Nocardia farcinica* infection has been reported in patients receiving thalidomide during treatment for leprosy (1), and *Pneumocystis* pneumonia has been reported in patients receiving thalidomide in combination with temozolomide (20).

This case also demonstrates some of the pitfalls in diagnosing legionellosis. It is important to note that the urinary antigen test only reliably detects *Legionella pneumophila* serogroup 1, and many commercial direct-fluorescent antigen kits detect only *Legionella pneumophila* species. Thus, the use of culture or nucleic acid detection methods is required to definitively diagnose infection with legionella strains other than *L. pneumophila*. In this case, the findings of coccobacilli in the modi-

![FIG. 1. A chest X ray (A) and computed tomography scan (B) obtained at the patient's admission showing a large left-upper-lobe infiltrate.](image1)

![FIG. 2. Modified acid-fast staining of pleural fluid showing short acid-fast rods subsequently identified as *L. micdadei*.](image2)
fied acid-fast stain allowed a rapid presumptive diagnosis of infection with *L. micdadei*. The use of carbol fuchsin, instead of safranin, as the counterstain in the Gram’s stain test would also have resulted in the detection of these bacteria.

On presentation, the patient was bradycardic. Bradycardia is an unusual presentation. Int. J. Lepr. Other Mycobact. Dis. 69:1–8.

The authors declare they have no competing interests.

REFERENCES


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