**Bacillus cereus** Necrotizing Pneumonia in a Patient with Nephrotic Syndrome

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**Abstract**

*Bacillus cereus* (*B. cereus*) is a Gram-positive rod that is widely distributed in the environment and can be a cause of food poisoning. We herein present a case of *B. cereus* necrotizing pneumonia in a patient with nephrotic syndrome under corticosteroid treatment after developing transient gastroenteritis symptoms. *B. cereus* was isolated from bronchial lavage fluid and transbronchial biopsy specimens. A multiplex polymerase chain reaction analysis of the toxin genes revealed a strain possessing enterotoxicity. The patient recovered after one week of intravenous meropenem followed by a combination of oral moxifloxacin and clindamycin. *B. cereus* is a pathogen that causes necrotizing pneumonia in immunocompromised hosts.

**Key words:** Bacillus cereus, necrotizing pneumonia, nephrotic syndrome, corticosteroid, hypogammaglobulinemia

(DOI: 10.2169/internalmedicine.52.7282)

**Introduction**

*Bacillus cereus* (*B. cereus*) is a Gram-positive, aerobic-to-facultative, spore-forming rod that is widely distributed in the environment (1). Although it is well known as a cause of food poisoning (2), the clinical relevance of *B. cereus* is often disregarded. When isolated from clinical specimens, it is usually considered a culture contaminant. However, severe hematogenous infections caused by *B. cereus* have been reported, especially in drug addicts, premature neonates and patients with severe underlying diseases or compromised immunity (3). *B. cereus* rarely causes lower respiratory tract infections, although most reported cases of *B. cereus* pneumonia involve fatal outcomes despite intensive antibiotic therapy.

We herein describe a case of *B. cereus* necrotizing pneumonia in an immunocompromised patient with nephrotic syndrome under treatment with high-dose corticosteroids.

The definitive diagnosis of *B. cereus* pneumonia was made based on cultures of bronchial lavage fluid and transbronchial lung biopsy specimens.

**Case Report**

A 43-year-old man presented with a 3-month history of progressive edema in both lower extremities and the face. On examination, his urinary protein level was 6.0 g/day and his serum albumin level was 1.1 g/dL. His renal function was mildly impaired (estimated glomerular filtration rate: 61.1 mL/min/1.73 m²) and hypogammaglobulinemia (immunoglobulin G (IgG): 224 mg/dL, immunoglobulin A (IgA): 322 mg/dL and immunoglobulin M (IgM): 58 mg/dL) was present. After obtaining the results of a renal biopsy, the patient was diagnosed with minimal change nephrotic syndrome. Following three days of pulsed steroid therapy with 500 mg/day of methylprednisolone, oral prednisolone (PSL) was administered at a dose of 50 mg/day. Diarrhea and...
vomiting were noted on day 2 of oral steroid therapy; however, there were no symptoms suggestive of sepsis. As the patient’s proteinuria and hypoalbuminemia gradually improved, the dose of PSL was reduced to 40 mg/day on day 43 from the start of PSL treatment.

On day 22, chest roentgenogram (Fig. 1) showed infiltrates in the left lower lung field. Due to the absence of fever, cough or sputum and normal physical examination findings, no antibiotics were administered. On day 46, chest roentgenogram (Fig. 2A) and chest computed tomography (CT) (Fig. 2B) revealed a cavitary lesion surrounded by infiltrates in the left lower lung lobe. On day 47, bronchoscopic examinations were performed, followed by antibiotic therapy with intravenous meropenem (2.0 g/day). No pathogenic organisms were identified by sputum cultures. Seven days later, B. cereus was isolated from the bronchial lavage fluid and transbronchial lung biopsy specimens. After undergoing eight days of intravenous meropenem treatment, the patient received 400 mg/day of moxifloxacin and 900 mg/day of clindamycin orally for 15 days. Chest roentgenogram and chest CT performed three months after discharge showed a reduction in the size of the cavity.

To identify B. cereus toxin genes, we performed multiplex polymerase chain reaction (PCR). The strain isolated from our patient exhibited the presence of nhe and cesB and the absence of nblA, hblC, hblD, cap, cya, lef, pag and Ba813, which indicates that the strain possessed strong cytotoxicity among the various strains of B. cereus (4) and was different from outbreak strains (5).

Discussion

B. cereus plays a well-known pathogenic role in food poisoning. It leads to toxin-mediated (6, 7), self-limited illness characterized by emetic or diarrheal syndromes. Systemic or localized infections encompass bacteremia (8), endocarditis (9), meningitis (10) and pneumonia (11-23). In this report, we described a case of B. cereus necrotizing pneumonia in a patient with nephrotic syndrome under treatment with high-dose corticosteroids. Previously reported cases of B. cereus pneumonia in adults are summarized in Table. Most of these cases occurred in patients with hematological disorders (e.g., leukemia and aplastic anemia) or alcohol abuse; however, four lethal cases of B. cereus pneumonia in immunocompetent welders and metalworkers have been recently reported (12, 15). Our patient was not neutropenic, although he could have been immunocompromised owing to hypogammaglobulinemia and the use of corticosteroids.

B. cereus is generally considered to be a saprophytic contaminant when isolated from clinical samples. Therefore, detection of the microorganism in multiple samples is necessary to make a definitive diagnosis of B. cereus pneumonia. In the present case, the pathogenic role of B. cereus in pneumonia was indicated because the bacteria were isolated from two different specimens that were sampled aseptically. However, the two specimens were obtained with the same day.
infection, we were unable to identify the route of the infection and the possibility for contamination cannot be ruled out.

As in most of the reported cases of *B. cereus* pulmonary infection, we were unable to identify the route of the infection (18). We observed no respiratory symptoms such as cough, hemoptysis or chest pain, whereas gastrointestinal symptoms (diarrhea and emesis) were present. Therefore, in the present case, it was indicated that the *B. cereus* pulmonary infection might have resulted from transient bacteremia from a gastrointestinal infection.

*B. cereus* produces β-lactamase and is therefore resistant to penicillin and cephalosporins (1). *B. cereus* is usually susceptible to clindamycin, vancomycin, fluorquinolones, carbapenems and aminoglycosides. After isolating *B. cereus*, we changed the antibiotics to a combination of clindamycin and moxifloxacin, which was effective.

A multiplex PCR analysis revealed that the strain of *B. cereus* isolated from our patient was different from outbreak strains possessing Ba813 (5). The present strain, which was positive for *nhe* and *ces* and negative for *hbl*, possesses strong cytotoxic capacity and accounts for 28.6% of *B. cereus* strains (4). These results are compatible with the development of necrotizing pneumonia in our patient and did not present a life-threatening condition.

In summary, this is a rare case of *B. cereus* necrotizing pneumonia in a patient with nephrotic syndrome. The present case indicates the importance of performing a bronchoscopic assessment of lung infiltrates, especially in immunocompromised hosts, even if the inflammatory reaction is not significant.

The authors state that they have no Conflict of Interest (COI).

### References


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