Invasive Bacillus cereus infection in a renal transplant patient: A case report and review

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CASE REPORT


Bacillus cereus is a common cause of gastrointestinal diseases. The majority of individuals with B cereus-related food poisoning recover without any specific treatment. It can, however, rarely cause invasive disease in immunocompromised patients.

Key Words: Bacillus cereus; Food poisoning; Septicemia; Rhabdomyolysis

CASE PRESENTATION

A 65-year-old man presented to the acute tertiary care centre of St Joseph’s Healthcare (Hamilton, Ontario) with vomiting and fever. His medical history consisted of microscopic polyangiitis, a perinuclear antineutrophil cytoplasmic antibody vasculitis and related chronic renal failure, which was managed with azathioprine and prednisone. His baseline creatinine levels ranged between 160 µmol/L and 170 µmol/L. He was previously treated with cyclophosphamide and was switched to azathioprine two months before presentation. He also had a history of coronary artery bypass, hypertension and dyslipidemia. One day before his presentation, he had ingested a salami and bologna sandwich he had purchased from a local grocery store. Two hours later, he developed vomiting and generalized weakness. The next day, he was admitted with a diagnosis of food poisoning when he presented with fever and persistent vomiting. Twenty-four hours following his admission, he developed diffuse nonpruritic, erythematous papular and vesiculopustular rash and continued to experience fever.

He was noted to have elevated creatinine and creatine kinase (CK) levels, consistent with rhabdomyolysis and was transferred to the coronary care unit. He was initiated on piperacillin-tazobactam.

His temperature was 39.8°C, blood pressure was 109/75 mmHg, heart rate was 87 beats/min, respiratory rate was 20 breaths/min and his oxygen saturation was 90% on 2 L of oxygen via nasal prong. Other than the presence of diffuse rash, the physical examination was normal. Dermatological examination revealed diffuse, discrete erythematous papular and vesiculopustular lesions involving the face, anterior and posterior trunk and bilateral arms, hands and legs as shown in Figure 1. There was no conjunctival, mucosal, palm or sole involvement. The lesions were non-tender.

Laboratory investigations revealed a white blood cell count of 3.5×10⁹/L, a hemoglobin level of 93 g/L and a platelet count of 3.5×10⁹/L, suggestive of relative pancytopenia. Blood film demonstrated toxic granulation. His CK level on admission was 806 U/L, which increased to 32,924 U/L within 48 h. Uribalysis showed blood and protein. A test to detect myoglobin in his urine was not performed. He had metabolic acidosis with a venous pH of 7.26, PCO₂ of 33 mmHg and a bicarbonate level of 14 mmol/L. His lactate, aspartate aminotransferase (AST) and alanine aminotransferase (ALT) levels were 271 µmol/L and 15.3 mmol/L, respectively. His CK level on admission was 806 U/L, which increased to 32,924 U/L within 48 h. Urinalysis showed blood and protein. A test to detect myoglobin in his urine was not performed. He had metabolic acidosis with a venous pH of 7.26, PCO₂ of 33 mmHg and a bicarbonate level of 14 mmol/L. His lactate, aspartate aminotransferase (AST) and alanine aminotransferase (ALT) levels were 271 µmol/L and 15.3 mmol/L, respectively. His CK level on admission was 806 U/L, which increased to 32,924 U/L within 48 h. Urinalysis showed blood and protein. A test to detect myoglobin in his urine was not performed.

He received intravenous normal saline and bicarbonate for the management of rhabdomyolysis. Antibiotics were switched from...
piperacillin-tazobactam to vancomycin at a dose of 1 g every 48 h and meropenem 500 mg every 12 h for possible disseminated \textit{B. cereus} infection.

The patient's family brought in the components of the sandwich ingested by the patient. Within 24 h of incubation, the meat grew \textit{B. cereus}. A portion of the meat was also sent to the public health laboratory for the detection of \textit{B. cereus} toxin. The toxin testing was not performed because less than 100 count/g of \textit{B. cereus} was isolated from the food. The patient improved significantly within 72 h of initiating intravenous vancomycin and meropenem, with resolution of the fever and skin lesions. The CRP and the serum creatinine levels improved to 489 \text{µmol/L} and 219 \text{µmol/L}, respectively. He completed a two-week course of vancomycin and meropenem. At six months' follow-up, the patient was doing well and his serum creatinine level had returned to baseline.

**DISCUSSION**

We report a case of sepsis with both rhabdomyolysis and poplar, vesicular skin rash due to \textit{B. cereus} infection. The combination of these two features occurring in a single patient has not been described previously.

\textit{B. cereus} is a ubiquitous organism found in soil, vegetation, dairy products and rice, and causes either an emetic or diarrheic syndrome. The emetic syndrome, which resembles \textit{Staphylococcus aureus} enterotoxin foodborne illness, develops after a short incubation period (0.5 h to 6 h) due to the emetic toxin, cereulide (7,8). The toxin is resistant to heat, cold, pH variations and proteolysis and, as a result, is often seen in food poisoning outbreaks (7,8). The diarrheic syndrome is caused by heat labile enterotoxins produced in the gut following the consumption of contaminated food. The incubation period of diarrheic syndrome is longer, ranging from 6 h to 24 h (1,2).

\textit{B. cereus} can cause local skin and wound infections, and invasive diseases such as bacteremia, central intravascular catheter-associated infection, endocarditis, osteomyelitis and meningocerephalitis. \textit{B. cereus} endophthalmitis can occur following a penetrating ocular trauma or injection drug use (1,2).

Rhabdomyolysis is a syndrome characterized by elevated serum concentrations of creatine phosphokinase and myoglobin leading to renal dysfunction. Rhabdomyolysis can be precipitated by trauma, ischemia, metabolic and electrolyte abnormalities, drugs and a variety of infectious diseases. The proposed mechanism of muscle damage includes toxin generation and direct bacterial invasion (9).

\textit{B. cereus} bacteremia associated with rhabdomyolysis has been reported in two patients (3,4). Both patients were immunocompromised and experienced a fatal outcome directly due to \textit{B. cereus} infection. In the first case, postmortem examination revealed diffuse muscle necrosis with infiltration of Gram-positive bacilli in the quadriceps. Infiltration of Gram-positive bacilli was also found in the lung, spleen and liver with acute tubular necrosis of the kidney.

There have also been two reported cases of fulminant liver failure associated with elevated CK levels in immunocompetent patients infected with \textit{B. cereus} following the ingestion of contaminated food (10,11). The emetic toxin cereulide was implicated in both cases, potentially as a mitochondrial toxin. The patient described in the first case died, whereas the second patient had a good outcome, with normalization of liver function following appropriate therapy.

We believe our patient's skin lesions were likely due to \textit{B. cereus} infection. According to a review of 10 pediatric cases of primary cutaneous \textit{B. cereus} infection in neutropenic patients treated for cancer or aplastic anemia, vesicles or pustules were seen only on the limbs (12). Culture of the vesicle fluid or wound drainage grew \textit{B. cereus}, but blood cultures did not grow the bacteria. In each case, the vesicles responded promptly to appropriate antibiotics including vancomycin, as was the case with our patient.

The majority of cases of \textit{B. cereus}-related food poisoning require supportive management only. Individuals with invasive disease require antimicrobial therapy. \textit{Bacillus} species produce beta-lactamases and are usually resistant to penicillins and cephalosporins. Vancomycin, aminoglycosides, carbapenem, clindamycin and ciprofloxacin are usually active in vitro (1,2).

\textit{B. cereus} should be considered as a serious pathogen in both immunocompromised and immunocompetent patients because it can cause sepsis, rash, rhabdomyolysis and acute fulminant liver failure. Prompt recognition and treatment with an appropriate antibiotic are essential, especially in immunocompromised patients, to prevent significant morbidity and mortality.

**REFERENCES**


