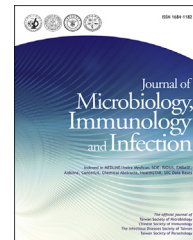




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CASE REPORT

Bacillus cereus septicemia in a patient with acute lymphoblastic leukemia: A case report and review of the literature



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Bacillus cereus is an aerobic Gram-positive, spore-forming, rod-shaped bacterium that is responsible for foodborne illnesses. We report on a 15-year-old girl with B-cell acute lymphoblastic leukemia, who fell into a somnolent state after presenting with a 12-hour history of fever, muscle soreness, myalgia in both calves, sore throat, and vomiting. Fulminant septicemic syndrome caused by *B. cereus* was finally identified. The aim of this work is the introduction of *B. cereus* as a differential diagnosis of sepsis in patients with acute leukemia in induction chemotherapy, to prevent delayed treatment.

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Introduction

Bacillus cereus, an aerobic Gram-positive, rod-shaped, spore-forming bacterium, is widely distributed in the

environment and is usually considered a contaminant when recovered from cultures. Although it commonly causes foodborne gastroenteritis, which is mostly benign and self-limiting, it occasionally causes severe infections in immunocompromised patients, including septic shock, meningitis, brain abscess, colitis, endocarditis, respiratory infections, and infection-related coagulopathy and hemolysis. Inoue et al reviewed 58 case reports of *B. cereus* sepsis in patients with leukemia and found that only 28 of those patients survived.¹ We present a fatal case of *B. cereus* sepsis in a patient with acute leukemia.

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Case report

A 15-year-old girl presented to hospital with a 7-day history of fever, sore throat, abdominal fullness, yellowish skin color, and general weakness. Based on her physical symptoms, clinicians at the hospital diagnosed tonsillitis and the patient received medicine. Her symptoms, however, did not subside, and the patient was taken to a different hospital. Liver function tests performed at that time revealed elevated levels of aspartate aminotransferase (1356 units/L), alanine aminotransferase (1930 units/L), and anemia (hemoglobin = 8.1 g/dL). She was referred to our hospital 3 days later, where an initial diagnosis of acute hepatitis was made.

After admission, complete blood count showed a hemoglobin level of 6.5 g/dL and a platelet count of 19,000/mm³. The white blood cell (WBC) count was 17,570/mm³, with 5% segmented neutrophils, 43% lymphocytes, 9% atypical lymphocytes, and 43% immature cells. After a series of examinations, acute B-cell lymphoblastic leukemia was diagnosed. Based on the TPOG-2002-ALL protocol, the patient was classified as belonging to the very high risk group. The Taiwan Pediatric Oncology Group (TPOG) was formed with the cooperation of all leukemia treatment centers in Taiwan in 1988 and has since initiated national cooperative group studies. A TPOG-2002-ALL VHR induction phase protocol was started 3 days after admission. Prednisolone 40 mg/m² was administered. Within 2 days, there was a marked decrease in blast cell count and WBC count, indicating a good response. However, neutropenic fever was noted. In response, cefepime (1 g by intravenous drip, twice daily) was administered as empiric therapy. Urine culture grew *Escherichia coli* (>10⁵ colonies/mL), which was susceptible to ampicillin, gentamicin, and cefepime.

Four days after induction chemotherapy, the patient experienced a sleepless and restless night, and had a fever of 38–39 °C. Skin rashes over the forehead, a sore throat, and myalgia over both lower limbs, in addition to another fever spike (40.1 °C) and one vomiting incident, were noted the next morning.

Sudden onset of consciousness disturbance, with anisocoric pupils occurred 2 hours later. The Glasgow Coma Scale was 8. Endotracheal intubation was performed. Brain computed tomography showed no intracranial hemorrhage. During the next 2 days, the patient suffered from four episodes of pulseless electrical activity, with a return of spontaneous circulation after resuscitation. Inotropic agents, including dopamine, epinephrine, dobutamine, norepinephrine, empiric antibiotics comprising meropenem (1 g by intravenous drip, 3 times daily), vancomycin (1 g by intravenous drip, 3 times daily), acyclovir (500 mg by intravenous drip, 3 times daily), fluconazole (400 mg by intravenous drip, once daily), and granulocyte colony-stimulating factor, were administered. However, the patient's general condition worsened. There was a rapid onset of epistaxis, gastrointestinal (GI) bleeding, menorrhagia, hematuria, and hypoalbuminemia, in addition to acute renal failure and acute pulmonary edema. The patient died 2 days after onset of the symptoms of septic shock.

Rectal, throat, and urine samples were analyzed to determine the causative agent. The results of all tests

performed (urine culture, blood culture, PCR for enterovirus, influenza, and adenovirus) revealed Gram-positive bacilli in three sets of blood cultures. The isolates were subjected to 16S ribosomal RNA gene sequencing using the broad-range bacterial specific primer pair Bact 5F (GAA GAG TTT GAT CMT GGC TC) and Bact 809R (GCG TGG ACT ACC AGG GTA TC), which is highly conserved throughout the phylogenetic tree and is found in all prokaryotic organisms.^{2,3} The product was compared with known sequences in the National Center for Biotechnology Information (NCBI) blast database and the result confirmed the presence of *B. cereus*. Therefore, *B. cereus*-induced fulminant septicemic syndrome was diagnosed.

Discussion

The incidence of *B. cereus* septicemia accounts for approximately 2% of all cases of bacteremia or fungemia.⁴ There have been several reports of *B. cereus* septicemia in patients receiving cancer chemotherapy, especially in patients with hematologic malignancies.^{5–15} All had severe neutropenia and high fever. The clinical course was fulminant, and death was rapid. In our patient, who had acute lymphoblastic leukemia, and who was severely neutropenic and iatrogenically immunosuppressed due to chemotherapy, fatal septicemic shock, and coma caused by *B. cereus* developed rapidly. The clinical course in our patient was compatible with that reported by Musa et al, which was marked by two phases: a mild febrile illness lasting 6–14 hours, accompanied by subtle symptoms of autonomic sympathetic nervous system overactivity, and a second short fulminant phase, marked by high fever (40–41 °C) accompanied by major central nervous system (CNS) disturbances, resulting in deep coma and brain stem dysfunction.¹⁶

Inoue et al reported on 12 cases and analyzed a total of 58 previously reported cases of *B. cereus* septicemia in patients with hematologic malignancies.¹ They found that acute leukemia, a neutrophil count of near 0/μL, or the presence of CNS symptoms during febrile episodes, were associated with a fatal prognosis. Their findings are compatible with the clinical presentation of our patient.

In our patient, all three sets of blood cultures showed Gram-positive bacilli, and the laboratory staff considered it as a contaminant. However, the three sets of blood cultures were sampled from different sites and at different times using sterile procedures. Therefore, it is unlikely that the samples were contaminated. *B. cereus* was definitively diagnosed after a full PCR workup. According to a previous study, the presence of *B. cereus* in blood cultures should not be regarded as contamination in patients receiving intensive chemotherapy for leukemia.^{1,4,17}

Our patient was treated with meropenem and vancomycin after she manifested symptoms of septic shock. According to a previous study, all of the isolated *B. cereus* strains were sensitive to imipenem, vancomycin, levofloxacin, and gentamicin, and the authors strongly recommend immediate initiation of treatment with meropenem and vancomycin in such situations. However, they also experienced cases of fatal *B. cereus* sepsis, in which appropriate antibiotic treatments were not effective.¹

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