Case report

**Bacillus cereus** meningoencephalitis in an immunocompetent patient

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**ABSTRACT**

Central nervous system (CNS) infection from Bacillus cereus (B. cereus) is rare and usually occurs in immunosuppressed patients or in a presence of invasive CNS devices. Our case reported here is a very rare case of an immunocompetent elderly patient without any CNS devices who was diagnosed with B. cereus meningoencephalitis and bacteremia. According to our patient, preceding gastrointestinal (GI) symptoms and trans-sphenoidal hypophysectomy could be the precipitating factors. A positive blood culture should not be concluded as a contamination but prompt repeating another set of blood culture for a better clinical judgment. Given its abrupt clinical course and high mortality rate, high index of suspicion for rapid detection and management is needed for a preferable clinical outcome. Empiric treatment with intravenous vancomycin is reasonable before a susceptibility result becomes available.

**Background**

*Bacillus cereus* (B. cereus) is facultatively anaerobic, spore-forming, Gram-positive, rod-shaped bacteria that present in the environment. It is one of the most common causes of food poisoning but infrequently can result in invasive extra-intestinal infections such as wound, eye, and respiratory tract infection [1–3]. It can also cause systemic infections including bacteremia and endocarditis [4,5]. Central nervous system (CNS) infection from *B. cereus* is rare and usually associated with invasive CNS devices or immunosuppressed status. Herein, we report a case of an elderly patient without any invasive CNS devices or immunosuppressive agents who was diagnosed with *B. cereus* meningoencephalitis and bacteremia.

**Case presentation**

An 80-year-old patient with history of irritable bowel syndrome (IBS) and secondary adrenal insufficiency resulting from trans-sphenoidal hypophysectomy of a pituitary tumor eight years ago was re-admitted to the hospital with an acute onset of altered mental status and high-grade fevers 12 hours after being recently discharged from the previous hospitalization.

The patient was previously hospitalized for four days with fatigue, abdominal pain, diarrhea, nausea, and vomiting, which were attributed to acute adrenal insufficiency resulting from poor compliance to corticosteroid treatment. During the past two years, the patient was admitted several times with the same diagnosis and clinical presentations. The patient’s medications included daily 20-milligram hydrocortisone and aspirin. The patient was originally from Mexico and moved to the United States 20 years ago. The patient denied using tobacco products, ethanol, and recreational drugs. There were no recent history of travel, sick contact, animal contact, and recent consumption of deli meats or unpasteurized dairy products. The patient was afebrile, however, the emergency room provider thought that this could be early symptoms and signs of sepsis, thus blood cultures were obtained and they did not grow any organisms. All symptoms improved after correction of hyponatremia and administration of systemic corticosteroid. The patient was feeling well before being discharged.

The patient presented to the emergency department 12 hours later. On examination, the temperature was 40 °C, the pulse rate 108 per minute, the blood pressure 131/78 mmHg, the respiratory rate 13 breaths per minute, and the oxygen saturation 99% while the patient was breathing on ambient air. The patient appeared confused and would not answer questions or follow commands. Complete neurological assessment was difficult to perform due to altered mental status, however, only rigid neck was observed and the patient was able to move all extremities. The rest of the exam was otherwise unremarkable. Blood tests showed leukocytosis with white blood cell (WBC) count of 16,990 cells/µL with polymorphonuclear cells (PMN) of 87.5%. Meningoencephalitis was suspected, thus empirical antimicrobial therapy with

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vancomycin, cefepime, ampicillin, and acyclovir was initiated. The computed tomography (CT) of the head without contrast and CT angiography of the head and neck revealed no evidence of acute intracranial pathology. Unfortunately, given the presence of a pacemaker, a magnetic resonance imaging (MRI) of the brain could not be performed for further information. Lumbar puncture was performed and the cerebrospinal fluid (CSF) analysis revealed WBC of 1317 cells/µL (86% neutrophils, 10% monocytes, and 3% lymphocytes), protein of 496 mg/dL, and glucose of less than 5 mg/dL; the result was consistent with a bacterial infection. Gram stain of the CSF showed no organisms. CSF polymerase chain reaction (PCR) did not detect any of the following pathogens: Escherichia coli K1, Haemophilus influenzae, Listeria monocytogenes, Neisseria meningitidis, Streptococcus agalactiae, Streptococcus pneumoniae, cytomegalovirus (CMV), enterovirus, herpes simplex virus (HSV) type 1 and 2, varicella zoster virus (VZV), human herpesvirus (HHV) type 6, human parechovirus, and Cryptococcus neoformans/gatti. The antimicrobial regimen was de-escalated to intravenous ceftriaxone 2 g every 12 h. Bacterial and mycobacterial cultures from the CSF were negative. Two sets of blood cultures were obtained on two consecutive days of hospitalization due to no clinical improvement after initiation of intravenous antibiotics. One out of two blood culture bottles drawn on admission and a day after admission grew Bacillus cereus. Given the fact that there were two positive blood cultures, it was considered the most likely etiology of meningoencephalitis in this patient. Transthoracic echocardiogram did not reveal any vegetations of the native valves and pace maker leads. Intravenous vancomycin 0.75 g (adjusted dose) every 12 h was added. The CSF was subsequently sent for broad-range 16s rDNA PCR for definitive diagnosis. B. cereus was only susceptible to vancomycin and gentamicin, thus ceftriaxone was discontinued. The patient was treated with intravenous vancomycin for a total of 14 days without any adverse reactions. The patient had complete resolution of all the symptoms and subsequently was discharged nine days after the presentation. The result of the broad-range 16s rDNA PCR of the CSF came back positive for B. cereus confirming the diagnosis of CNS infection.

Methods

Our literature review of B. cereus was based on PubMed database. English keywords were used. The first search term "Bacillus cereus meningocoeaphalitis" yielded 7 articles (6 items are available in English). The second search term "Bacillus cereus meningitis" yielded 22 articles (17 items are available English). The abstracts of the articles retrieved from all two searches were reviewed, and the references from pertinent articles were reviewed to identify additional case reports. A total of 31 relevant publications were identified and the full articles on 41 patients with CNS infection from B. cereus were available for review.

Discussion and conclusions

Bacillus cereus is a Gram-positive, facultatively anaerobic, spore-forming bacilli which is widespread in the environment especially in soil or dust. Consequently, the contamination of this organism in hospital environment is inevitable. Study from Barrie et al. [6] showed association between B. cereus infection in patients and contamination of the organism in hospital environment. It can usually cause self-limited toxin-mediated gastrointestinal (GI) disease but the incidence of serious extra-intestinal infections has been increasingly reported according to previous published studies [7].

CNS infection with B. cereus is infrequent. It can present with various manifestations including ventriculitis, meningitis, meningocoeaphalitis, brain abscess, hydrocephalus, intraparenchymal, subarachnoid, and subdural hemorrhage (Table 1). The condition usually occurs in patients with defect in host defense mechanisms either from underlying primary or secondary immunodeficiency disorders, or invasive CNS procedures or devices. According to our literature review of the previous published adult and pediatric cases of B. cereus CNS infections (Table 1), chemotherapy may be associated with development of B. cereus CNS infections. Preterm delivery could also be one of the predisposing factors in pediatric patients [8]. To our knowledge, our case does not have an overt immunosuppressive condition. The daily dose of glucocorticoid replacement therapy for adrenal insufficiency is 20 mg of hydrocortisone daily (equivalent to 5 mg of prednisolone or prednisone), which is considered a physiologic dose. However, we did not do extensive workups for immunodeficiency disorders in this case, and immune dysfunction occurs with age. Immunosenescence could be a risk factor for B. cereus bacteremia and CNS infections in our case. Provided that there is no comparable case in the literature, it is difficult to predict the potential risk factors in this case. However, Berke et al. [9] reported a case of postoperative B. cereus meningitis after trans-septal trans-sphenoidal excision with retained ventriculostomy tube in a 25-year-old patient. Our patient’s history of trans-sphenoidal hypophysectomy was eight years ago, true correlation of the procedure with the development of meningocoeaphalitis in our patient is questionable. The source of the infection in our patient has not been elucidated. One of the possibilities could be from GI tract since the patient presented with diarrhea during the previous hospitalization. The diarrhea could either be from the underlying condition of IBS or from possible prior bacterial infection. A report by Gaur et al. [10] showed that in three out of four patients with B. cereus bacteremia, the organism was isolated in cultures of their stools or rectal swabs obtained within 72 h of presentation of the infection. However, it is still not clear if rectal colonization could be the source of the bacteremia. Contaminated intravenous fluid during the previous admission could be another possible source of bacteremia in our patient.

Given its ubiquitous presence, the isolation of B. cereus from sterile clinical specimens is usually considered as a contamination, especially in immunocompetent patients. The positive blood culture for B. cereus in our patient was initially interpreted as a contamination considering the patient’s immune status, an absence of invasive CNS devices, and a report of one positive blood culture out of the two bottles on the first day. However, one positive bottle for two consecutive days had pointed the positive blood cultures towards being from a true pathogen. Subsequently, the positive broad-range 16s rDNA PCR from the CSF for B. cereus confirmed the diagnosis.

B. cereus CNS infection usually has an abrupt clinical course and a high mortality rate despite aggressive treatment with broad-spectrum antimicrobial therapy. High index of suspicion is needed for rapid detection and management [11]. To date, there is still no standard guideline of treatment for B. cereus CNS infection, thus guidance on management comes solely from previously reported cases. The production of beta-lactamases makes B. cereus resistant to beta-lactam antimicrobial agents including third-generation cephalosporins but the organism is known to be susceptible to aminoglycosides, clindamycin, erythromycin, vancomycin, and chloramphenicol [7]. Vancomycin is generally considered the preferred agent for empiric therapy and was used in many reported cases including in our patient [12]. On the other hand, due to the outright and inducible resistance of B. cereus against clindamycin, the agent should not be used before the susceptibility results become available [13]. In our case, after the susceptibility report showed susceptibility to vancomycin, the treatment with intravenous vancomycin monotherapy for 14 days resulted in complete resolution of the clinical symptoms. The patient felt lucky that the condition was diagnosed correctly and that the patient got better with the treatment received.

This presenting case adds new learning points to the body of literature on B. cereus CNS infection. We believe that B. cereus should be included in one of the differential pathogens causing CNS infections in patients who had preceding GI signs and symptoms and with a history of trans-sphenoidal hypophysectomy regardless of duration after the procedure. A positive blood culture of B. cereus in one out of two obtained bottles should not be concluded as a contamination but prompt repeat another set of blood cultures for a better clinical judgment. The empiric
### Table 1
Summary of previously reported cases of *Bacillus cereus* CNS infection.

| Year | Author [ref] | Clinical diagnosis | Age/Sex | Outcomes     | Predisposing conditions | Treatments                           |
|------|--------------|--------------------|---------|--------------|--------------------------|--------------------------------------|
| 1970 | Leffert et al. [14] | Meningitis | 18 weeks /M | recovered | - Dandy-Walker cyst       | - Ampicillin                          |
| 1977 | Turnbull et al. [15] | Encephalitis with extensive necrosis of brain | 1 day /F | died       | - Ventriculo-arterial shunt revision | - Gentamicin                          |
| 1981 | Colpin et al. [16] | Meningitis | 19 years /M | died       | - Preterm delivery        | - Ampicillin                          |
| 1981 | Berke et al. [9] | Ventriculitis and meningitis | 25 years /F | recovered | - Necrotizing enterocolitis | - Gentamicin                          |
| 1991 | Weisse et al. [17] | Meningoencephalitis with complete hemorrhagic necrosis of the brain | 8 days /F | died       | - Umbilical catheter      | - Penicillin                          |
| 1989 | Jenson et al. [18] | Leptomeningitis | 32 years /M | recovered | - Post chemotherpay       | - Gentamicin                          |
| 1998 | Feder et al. [19] | Meningitis | 47 days /F | recovered | - Central line            | - Penicillin                          |
| 1987 | Garcia et al. [20] | Leptomeningitis | 1 year /F | recovered | - Trans-septal trans-sphenoidal excision of pituitary adenoma | - Chloramphenicol                     |
| 1999 | Musa et al. [21] | Meningitis | 5 days /M | recovered | - Ventricular punction    | - Vancomycin                          |
| 1997 | Barrie et al. [6] | Hydrocephalus | 55 years /F | died        | - Post chemotherpay + intrathecal methotrexate | - Vancomycin                          |
| 1995 | Marley et al. [22] | Meningoencephalitis, subarachnoid hemorrhage, liver and myocardium necrosis | 26 years /M | died       | - Ventricular drain       | - Chloramphenicol                     |
| 1997 | Akiyama et al. [23] | Leptomeningitis, subarachnoid hemorrhage, liver and stomach necrosis | 64 years /M | died       | - Ventricular drain       | - Chloramphenicol                     |
| 1997 | Berner et al. [24] | Meningitis | 18 months /M | recovered | - Ventricular hemorrhage | - Chloramphenicol                     |
| 1999 | Tokieda et al. [8] | Multiple brain parenchyma, subdural, epidural, subarachnoid hemorrhage | 4 days /F | died       | - Hydrop fetalis          | - Chloramphenicol                     |
| 1999 | Musa et al. [25] | Leptomeningeal and neural necrosis | 30 years /M | died       | - Neuraxial catheter      | - Chloramphenicol                     |
| 2000 | Tuladhar et al. [26] | Intraventricular hemorrhage | 14 days /M | died       | - Perterm delivery        | - Chloramphenicol                     |
| 2000 | Marshman et al. [27] | Fulminant meningitis | 41 years /F | recovered | - Post chemotherpay       | - Chloramphenicol                     |
| 2001 | Gaur et al. [10] | Right basal ganglia infarction | 20 years /F | died       | - Intrathecal chemotherpay | - Chloramphenicol                     |
| 2001 | Chu et al. [28] | Meningitis with liquefactive necrosis of the brain | 30 days /M | died       | - Neutropenia             | - Chloramphenicol                     |
| 2003 | de Almeida et al. [29] | Meningitis | 16 years /F | died       | - Post bone marrow transplant | - Chloramphenicol                     |

(continued on next page)
treatment with intravenous vancomycin is reasonable before a susceptibility result becomes available.

Ethics approval

Not applicable.

Consent

Written informed consent was obtained from the patient for publication of this case report. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

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Author’s contributions

The manuscript was read and approved by all the authors. Pichaya Tao Worapongsatitaya: writing original draft. Jakrapun Pupaibool: reviewing, editing, and supervising. All authors contributed to the work in this manuscript.

CRediT authorship contribution statement

Pichaya Tao Worapongsatitaya: writing-original draft preparation, software, investigation, Jakrapun Pupaibool: conceptualization, methodology, software, validation, writing, reviewing, editing, supervision, All authors contributed to the work in this manuscript.

Conflicts of interests

The authors have no conflicts of interests to declare.

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