Case report

Bacillus cereus infection in pediatric oncology patients: A case report and review of literature

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A B S T R A C T

Introduction: Bacillus Cereus infection can be life-threatening in immunocompromised patients. We report here a case of Bacillus Cereus septicemia in a child with relapsed acute lymphoblastic leukemia (ALL) and present review of literature.

Methods: We collected clinical, laboratory and outcome data of our patient with relapsed ALL and Bacillus Cereus infection. We reviewed literature for Bacillus Cereus infection in pediatric oncology patients by searching MED-LINE/PubMed/Google/Google Scholar/Cochrane and summarized the data obtained. Various risk factors like presence of gastrointestinal or central nervous system (CNS) symptoms, neutropenia, central venous catheter in-situ, corticosteroids use, intrathecal chemotherapy and outcomes were analyzed using Fisher Exact Chi Square test.

Results: A 15-years-old boy with relapsed ALL on induction chemotherapy presented with giddiness and difficulty in breathing. He had an episode of hematemesis followed by fainting at home. He had refractory shock which did not respond to fluid boluses, inotropes and hydrocortisone. He had severe metabolic acidosis with high lactate and ammonia and died within 36-hours of onset of symptoms. His blood culture was positive for Bacillus Cereus. We came across 36 published cases of Bacillus Cereus in children with cancer including present case. Of these, 28 had acute leukemia and rest 8 had other cancers. CNS symptoms were present in 13 patients. Overall mortality was 25%. Patients with multisystem involvement had significantly higher mortality compared to those having localized disease (p-value 0.033).

Conclusion: In pediatric oncology patients on chemotherapy, cultures positive for Bacillus Cereus should be considered significant. Mortality is higher in those with multisystem involvement.

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Introduction

Bacillus Cereus (B. Cereus) is a gram positive aerobic or facultative anaerobic spore forming rod. It is a frequent cause of food poisoning. Although it is considered a contaminant and non-pathogenic in immune-competent individuals, it can cause serious manifestations in neutropenic and immunocompromised patients. It has been shown to cause septicemia, central nervous system (CNS) infections, respiratory infections, endocarditis as well as local wound, burn and ocular infections [1–4]. Although rare but can be highly fatal in patients with hematological malignancies [5–7]. Fulminant course has been reported with multisystem involvement [8–10]. We report here a case of B. Cereus septicemia in a child with relapsed acute lymphoblastic leukemia (ALL) and also present review of literature.

Method

We collected clinical, laboratory and outcome data of our patient with relapsed ALL who had Bacillus Cereus infection. We also reviewed the published cases of B. Cereus infection in pediatric oncology patients and summarized the data obtained. For the review of literature, we searched published data on MED-LINE/ PubMed/Google/Google Scholar/Cochrane and summarized the data obtained. Various risk-factors like presence of gastrointestinal or central nervous system symptoms, neutropenia, central venous catheter in-situ, corticosteroids use, intrathecal chemotherapy and outcome were analyzed using Fisher Exact Chi Square test. P value < 0.05 was considered significant.
and 60% FiO\textsubscript{2} (CVC). He was shifted to pediatric intensive care unit, put on oxygen peripherally inserted central catheter (PICC)/central venous catheter support by high flow nasal cannula with 20 liter/min oxygen flow. Specific broad-spectrum intravenous antibiotics. Due to persistent intravenous fluid boluses, supplemental oxygen by mask and emergency intubation for refractory shock. Blood workup was sent which included complete blood count, differential count, coagulation profile, liver and renal function tests. Results are shown in Table 1 (at admission). His initial investigations revealed pancytopenia, 50% blasts in peripheral smear, raised C-reactive protein (14 mg/L) with deranged liver and renal functions. His serum ammonia level was high. Venous blood gas analysis showed high anion gap, metabolic acidosis and pH of 7.0 with very high lactate levels (27 mmol/L). Injection sodium bicarbonate infusion was started. In view of suspected uncomplicated shock, antibiotics were further escalated to Injection Cefazidime-avibactam, Injection Aztreonam, Injection Teicoplanin. He received multiple transfusions of packed red cell, platelet concentrate, fresh frozen plasma and cryoprecipitate. His repeat investigations sent 6-hours later showed further worsening of liver and renal functions along with hyperphosphatemia. N-acetyl cysteine was started for deranged liver function tests and phosphate binder and allopurinol were given for hyperphosphatemia.

A femoral central line was inserted for intravenous access other than the existing PICC line. He started bleeding from femoral central line site, which continued despite blood products support, manual compression and pressure bandage application. Another 12 h later, he became drowsy, supplemental oxygen requirement increased and blood pressure started to fall again. Repeat investigations were sent as shown in Table 1. Inotropes were further escalated. His ammonia level was 133 \(\mu\)mol/L and ferritin increased to 84,800 ng/mL. In view of low Glasgow Coma Scale score (GCS=8) and persistently low blood pressure, he was intubated and started on mechanical ventilation. He had an episode of endotracheal bleeding after few hours followed by cardiac arrest. Immediate cardiac-pulmonary resuscitation was started and child was revived after sustained efforts. After another 30 min, he again had cardiac arrest and despite all resuscitative measures he could not be revived. The child died within 36 h of onset of symptoms. His blood culture sent at admission revealed aerobic spore bearer which was identified by VITEK2 system (bio-Merieux)-BCL card as B. cereus.

### Review of literature

Based on our literature search, total 19 articles were found. 14 papers were exclusively about pediatric patients and 5 papers included both adults and pediatric patients. We have included only pediatric patients in our review. The total number of pediatric oncology patients with B. cereus infection published so far and included in this review are 36 children [5,6,8,9,11–24]. The demographic profile, disease spectrum, clinical presentation, relevant associated factors like steroid use, recent previous intrathecal chemotherapy, presence of CVC, absolute neutrophil count (ANC), outcome are summarized in Table 2. Age ranged from 2 to 18 years with median of 10 years. There were 22 males and 14 females in this cohort. Majority of children (28/36) had acute leukemia and remaining 8 patients had other childhood cancers. CNS symptoms were present in 13 patients, absent in 13 and data was not available for 9 patients. Corticosteroids were administered in past one month to 11 patients while data was not available for the same in 20 patients. Similarly, 9 patients received intrathecal chemotherapy in past 1-week, 13 patients did not receive any and there was no data for rest of the 13 patients. CVC was in situ in 19 patients but source of B. cereus could be identified as CVC in only 5 patients. Localized disease like brain abscess, cellulitis, endocarditis was seen in 11 patients and 14 patients had multisystem involvement. In our review, culture-sensitivity data was available in 17 patients. Majority of isolates were sensitive to vancomycin, carbapenem and resistant to penicillin. We evaluated the risk factors for mortality due to B. cereus infection among these patients, as shown in Table 3. A total of 9 patients (25%) died. Based on the available data, we found that patients with multisystem affection, had higher mortality as compared to those having localized disease (\(p = 0.033\)).

### Discussion

B. Cereus is a known cause of self-limiting food poisoning requiring only symptomatic treatment in immunocompetent patients. It produces exotoxins, including enterotoxin (diarrheal toxin) and ceruleoid (emetic toxin). Ceruleoid is usually resistant to heat and proteolysis. It inhibits hepatic mitochondrial fatty acid oxidation, causes swelling of mitochondria and results in cell death [2,3]. B. Cereus also produces proteases, hemolysins and phospholipases causing tissue damage and multiorgan dysfunction [1]. In addition to contaminated meat-based products, rice and pasta dishes, other important sources of B. Cereus are intravascular catheters, open wounds, drug abuse and contaminated bed linen [4]. Definite source of this infection in our case is difficult to pin-point.

B. Cereus is of significant concern in immunocompromised patients. As per published literature, the occurrence of B. Cereus in hematological malignancies ranges from 0.07% to 2% [5,6]. Mortality can be as high as 52% [7]. Hence growth of B. Cereus in blood culture of an immunocompromised patient should be flagged and that too urgently instead of the common practice of ignoring it as a contaminant. This can be a useful clue for instituting appropriate antibiotics at the earliest.

Our patient presented with vomiting and later developed encephalopathy. This appears to be in accordance with two phases of infection.

### Table 1

| Investigations             | At admission | At 6 h | At 12 h | At 18 h |
|----------------------------|--------------|--------|---------|---------|
| Hemoglobin (g/dl)          | 6.2          | 6.5    | 6.3     | 4.7     |
| Total leucocyte count (cells\(\mu\)L) | 3950         | 1250   | 460     | 380     |
| Absolute neutrophil count (cells\(\mu\)L) | 395          | 87     | 46      | 57      |
| Platelet count (cells\(\mu\)L) | 10,000       | 8000   | 41000   | 51000   |
| Total bilirubin (mg/dl)    | 3            | –      | –       | 3.7     |
| Albumin (g/dl)             | 2.55         | 2.39   | –       | –       |
| SGOT (U/L)                 | 486          | 827    | –       | –645    |
| SGPT (U/L)                 | 511          | 381    | –       | –310    |
| Blood urea (mg/dl)         | 99           | 117    | –       | –133    |
| S. creatinine (mg/dl)      | 1.5          | 2.1    | –       | –2.5    |
| S. uric acid (mg/dl)       | 13.4         | 15.4   | –       | –18.1   |
| S. calcium (mg/dl)         | 7            | 6.7    | –       | –6.0    |
| S. phosphorus (mg/dl)      | –            | 10.6   | –       | –1.9    |
| INR                        | 1.76         | –      | –       | –1.9    |
| Fibrinogen (mg/dl)         | 92.9         | –      | –       | –89     |
| d-dimer (mg/L FEU)         | 1.95         | –      | –       | –1.88   |
| Ammonia (\(\mu\)mol/L)    | –            | –      | –       | –133    |
| Ferritin (ng/ml)           | 15.100       | –      | –       | 84,800  |
| Lactate (mmol/L)           | 27           | 15     | 21      | –       |
| pH                         | 7.0          | 7.32   | 7.5     | 7.52    |
| S. no | Author(s) | Year | Diagnosis | Stage of Rx | Age | Sex | Symptoms on presentation | GI symptom | CNS symptom | CNS lesions | Other features | ANC | CVC | Steroid | IT chemo | Source | Outcome |
|-------|-----------|------|------------|------------|-----|-----|--------------------------|------------|-------------|-------------|---------------|-----|-----|---------|---------|--------|---------|
| 1     | Feldman et al.[13] | 1974 | ALL refractory/relapse | NA | 17 | M | Fever, chest pain | NA | No | NA | Pneumonia, pulmonary infarct | 36 | NA | Yes | Yes | NA | Death |
| 2     | Guiot et al.[14] | 1986 | ALL | Induction | 18 | M | Wound on forearm | NA | Yes | CT-damaged BBB | Fever, drowsiness | 20 | NA | NA | NA | NA | Wound Death |
| 3     | Henrickson et al.[15] | 1989 | ALL | Remission | 5 | M | Swelling, redness in left 4th finger | No | No | NA | Cellulitis | Low | NA | NA | NA | NA | Recovery |
| 4     | Henrickson et al.[15] | 1989 | Neuroblastoma | NA | 2 | M | URI, fever, swelling and vesicles in left hand | No | No | NA | Low | NA | NA | NA | NA | Recovery |
| 5     | Henrickson et al.[15] | 1989 | ALL | Induction | 8 | M | Eschar-like lesion on toe, fever | No | No | NA | None | Low | NA | NA | NA | NA | Recovery |
| 6     | Jensen et al.[16] | 1989 | ALL | Induction | 3 | M | Fever, lethargy, obtundation | No | Yes | Multiple brain abscesses | No | 20 | NA | Yes | Yes | Recovery |
| 7     | Yoshida et al.[5] | 1993 | AML (M5b) | Induction | 15 | M | Fever, headache, vomiting, diaphoresis | Yes | Yes | NA | Subarachnoid hemorrhage | NA | NA | NA | NA | NA | Death |
| 8     | Musa et al.[8] | 1999 | ALL | Induction | 14 | M | Fever, seizure | No | Yes | NA | Brain herniation | Coma, liver enzymes derangement | 100 | Yes | Yes | NA | NA | Death |
| 9     | Arnaout et al.[9] | 1999 | ALL relapse | Induction | 10 | F | Abdominal pain, lethargy | No | Yes | Multiple brain infarcts | Intraocular hemorrhage | 0 | Yes | Yes | Yes | CVC | Recovery |
| 10    | Christenson et al.[17] | 1999 | ALL relapse | Induction | 10 | F | Abdominal pain, headache, diarrhea | Yes | No | NA | Shock | Low | Yes | NA | NA | CVC | Recovery |
| 11    | Christenson et al.[17] | 1999 | Acute leukemia | Induction | 5 | F | Abdominal pain, seizures | Yes | No | NA | Shock | Low | Yes | NA | NA | NA | Death |
| 12    | Christenson et al.[17] | 1999 | AML relapse | post BMT day 311 | 6 | M | Fever, abdominal pain, diarrhea | Yes | No | NA | No | NA | Yes | NA | NA | NA | Recovery |
| 13    | Gaur et al.[11] | 2001 | Nasopharyngeal carcinoma | NA | 16 | F | NA | No | No | NA | NA | NA | Yes | No | No | NA | Recovery |
| 14    | Gaur et al.[11] | 2001 | ALL | NA | 4 | M | NA | No | No | NA | NA | NA | Yes | Yes | No | NA | Recovery |
| 15    | Gaur et al.[11] | 2001 | NHL | NA | 17 | M | NA | No | No | NA | NA | NA | Yes | No | No | NA | Recovery |
| 16    | Gaur et al.[11] | 2001 | MDS | NA | 7 | M | NA | No | No | NA | NA | NA | Yes | No | No | NA | Recovery |
| 17    | Gaur et al.[11] | 2001 | Yolk sac tumor | NA | 5 | M | Seizure, altered | No | No | NA | NA | NA | Yes | No | No | NA | Recovery |
| 18    | Gaur et al.[11] | 2001 | ALL | NA | 13 | F | Sensorium altered | Yes | Yes | NA | Diffuse cerebral edema, acute hydrocephalus | NA | 0 | Yes | Yes | Yes | NA | Recovery |
| 19    | Gaur et al.[11] | 2001 | Histioctosis | NA | 10 | M | NA | Yes | No | NA | NA | NA | Yes | No | No | CVC | Recovery |
| 20    | Gaur et al.[11] | 2001 | ALL relapse | NA | 15 | F | Altered sensorium | Yes | Yes | Multiple brain infarcts, hydrocephalus | Brain abscesses | 0 | Yes | Yes | Yes | NA | Death |
| 21    | Leonard et al.[18] | 2002 | Alveolar RMS | NA | 11 | M | Fever, seizures | No | Yes | NA | Coccinfection with Aspergillus like molds | Low | Yes | NA | NA | NA | Recovery |
| 22    | Saleeb et al.[19] | 2004 | ALL | Induction | 17 | F | NA | NA | NA | NA | NA | NA | Low | NA | NA | NA | Tea bag | Recovery |
| 23    | Nishikawa et al.[20] | 2009 | ALL | Induction | 16 | F | Fever, disorientation | No | Yes | NA | Brain abscess | Low | Yes | Yes | Yes | NA | Recovery |
| 24    | Uchino et al.[12] | 2012 | AML | HiDAC | 18 | F | Seizures | No | No | NA | Multiple brain infarcts, central diabetes insipidus, DIC, grade 3 elevation of liver enzymes | 3 | Yes | NA | NA | NA | Recovery |
| 25    | Sharma et al.[21] | 2013 | ALL maintenance | NA | 5 | F | Fever, vomiting | Yes | No | NA | Endocarditis | NA | Yes | NA | NA | CVC | Recovery |
pathogenesis of fulminant septicemia syndrome of B. Cereus disease as described in literature [8]. First phase usually consists of mild febrile illness with sympathetic nervous systemic over-activity lasting 6–14 h and a short fulminant phase marked by high fever accompanied by major CNS disturbances, resulting in deep coma and brainstem dysfunction. We think that our patient did not have fever since he was on dexamethasone as part of his chemotherapy. Similar to our case, there are 2 reported cases of fulminant B. Cereus sepsis in acute leukemia who were on dexamethasone, remained afebrile, presented with massive intravascular hemolysis and died shortly. Hence, B. Cereus infection should be kept as a possible differential in immunocompromised patients even in absence of fever while on steroids if he/she presents with vomiting and CNS symptoms like fainting, altered sensorium [9].

Various risk factors have been identified for severe and fatal B. cereus infection in immunocompromised patients like corticosteroids, neutropenia, preceding intrathecal chemotherapy, CNS symptoms and presence of CVC [7,10,11,12]. Our patient was on dexamethasone (20 mg/m2/day), received intrathecal chemotherapy 4 days before onset of symptoms, had CVC in-situ and had ANC of 395/μL on admission. In our review of published literature, it was found that patients who presented with localized disease like cellulitis, brain abscess, endocarditis had significantly better outcome than those who had multisystem involvement. Some differences in age, gender, type of disease, presence of GI or CNS symptoms were noted between patients who survived and those who died. Moreover, the patients with poorer outcome were noted to have low ANC, received steroids in the month preceding the infection, received intrathecal chemotherapy in the preceding week and had a CVC in situ however the difference was not found to be statistically significant in our analysis of published cases (Table 3) [5,6,8,9,11–24].

Some of the striking features of our case were persistent high lactate, hyperammonemia and high ferritin. Since our patient had vomiting and no diarrhea, we can assume that emetic toxin (cerulide) induced dysfunction of mitochondrial beta oxidation could have caused acute liver failure that led to hyperammonemia and acute encephalopathy. Similar to our case, there is a case report of food poisoning by emetic toxin of B. Cereus in 11 years old child leading to acute encephalopathy, liver failure and systemic organ damage involving hyperammonemia, lactic acidosis and hypoglycemia [2]. Thus, hyperlactatemia and hyperammonemia can be considered as predictors of poor outcome. The raised ferritin levels signify the ongoing inflammation and possibly hemophagocytic

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**Table 3**

Factors affecting mortality due to B. cereus in Pediatric Oncology Patients.

| Characteristic               | Death (n = 9) | Recovery (n = 27) | P value |
|-----------------------------|---------------|------------------|---------|
| **Age**                     |               |                  |         |
| <5 year                     | 2/9 (22%)     | 9/27 (33%)       | 0.690   |
| >5 year                     | 7/9 (78%)     | 18/27 (67%)      |         |
| **Gender**                  |               |                  |         |
| Male                        | 6/9 (67%)     | 16/27 (60%)      | 1.0     |
| Female                      | 3/9 (33%)     | 11/27 (40%)      |         |
| **Disease**                 |               |                  |         |
| Acute leukemia              | 9/9 (100%)    | 19/27 (70%)      | 0.160   |
| Leukemia                    | Others        | 0                | 8/27 (30%) |
| **GI symptoms**             |               |                  |         |
| Present                     | 5/6 (83%)     | 8/20 (40%)       | 0.160   |
| Absent                      | 1/6 (17%)     | 12/20 (60%)      |         |
| **CNS symptoms**            |               |                  |         |
| Present                     | 6/8 (75%)     | 7/18 (39%)       | 0.202   |
| Absent                      | 2/8 (25%)     | 11/18 (61%)      |         |
| **Local vs systemic**       |               |                  |         |
| Localized                   | 1/9 (11%)     | 10/16 (63%)      | 0.033   |
| Systemic                    | 8/9 (89%)     | 6/16 (37%)       |         |
| **Low ANC**                 |               |                  |         |
| Present                     | 8/9 (89%)     | 16/18 (89%)      | 1.0     |
| Absent                      | 1/9 (11%)     | 2/18 (11%)       |         |
| **Use of steroid**          |               |                  |         |
| Present                     | 5/5 (100%)    | 6/11 (55%)       | 0.119   |
| Absent                      | 0             | 5/11 (45%)       |         |
| **IT chemotherapy**         |               |                  |         |
| Present                     | 4/5 (80%)     | 5/17 (30%)       | 0.116   |
| Absent                      | 1/5 (20%)     | 12/17 (70%)      |         |
| **CVC**                     |               |                  |         |
| Present                     | 4/4 (100%)    | 15/19 (100%)     | 1.0     |
| Absent                      | 0             | 0                |         |
lymph histiocytes due to infection or underlying malignancy. The clinical course in our patient was fulminant. He succumbed within 36 h of onset of symptoms.

B. Cereus produces beta lactamase and is resistant to penicillin, cephalosporins and trimethoprim-sulfamethoxazole and susceptible to aminoglycosides, carbapenems, vancomycin and fluoroquinolones. However, there are many reports of fatal B. Cereus sepsis despite giving appropriate antibiotics [7]. Even though our patient was given Injection Meropenem, Injection Amikacin and other antibiotics (Injection Cef-

appropriately antibiotics [7]. Even though our patient was given Injection aminoglycosides, carbapenems, vancomycin and fluoroquinolones.

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Ethical approval

Not Applicable.

Consent

Informed consent of the parents was obtained.

Disclosure

All authors have nothing to declare.

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