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Case report

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

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ABSTRACT

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.

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.

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Table 1
Lab parameters since admission to the hospital.

Investigations	At admission	At 6 h	At 12 h	At 18 h
Hemoglobin (g/dl)	6.2	6.5	6.3	4.7
Total leukocyte count (cells/μL)	3950	1250	460	380
Absolute neutrophil count (cells/μL)	395	87	46	57
Platelet count (cells/μL)	10,000	8000	41,000	51,000
Total bilirubin (mg/dl)	3	–	–	3.7
Albumin (g/dl)	2.55	2.39		
SGOT (U/L)	486	827	–	664
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	–	–
INR	1.76	–	–	1.9
Fibrinogen (mg/dl)	92.9	–	89	–
D-dimer (mg/L FEU)	1.95	–	1.88	–
Ammonia (μmol/L)	–	–	**133**	–
Ferritin (ng/ml)	15,100	–	**84,800**	–
Lactate (mmol/L)		**27**	**15**	**21**
pH	7.0	7.32	7.5	7.52

Case report

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. On examination, he was afebrile, drowsy, peripheral pulses were not palpable. His heart rate was 190/min, blood pressure was 70/30 mmHg with oxygen saturation of 92% on room air. He received intravenous fluid boluses, supplemental oxygen by mask and empirical broad-spectrum intravenous antibiotics. Due to persistent hypotension inotropes (epinephrine and norepinephrine) were started in emergency department and blood culture was sent from peripherally inserted central catheter (PICC)/ central venous catheter (CVC). He was shifted to pediatric intensive care unit, put on oxygen support by high flow nasal cannula with 20 liter/minute oxygen flow and 60% FiO_2. Inotropes were escalated and Injection hydrocortisone was given 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 soda bicarbonate infusion was started. In view of suspected decompensated shock, antibiotics were further escalated to Injection Ceftazidime-avibactam, Injection Aztreonam, Injection Teicoplanin and Injection Fluconazole. 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 μmol/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 cardio-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 patents. 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 cerulide (emetic toxin). Cerulide 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 phased

Table 2
Summary of cases of Bacillus cereus reported in Pediatric Oncology patients.

S. no	Author (ref no.)	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	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 a[15]	1989	Neuroblastoma	NA	2	M	URI,fever, swelling and vesicles in left hand	No	No	NA	No	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	No	Low	NA	NA	NA	NA	Recovery
6	Jenson et al.[16]	1989	ALL	Induction	3	M	Fever, lethary, obtundation	No	Yes	Multiple brain abscesses	No	20	NA	Yes	Yes	NA	Recovery
7	Yoshida et al.[5]	1993	AML (M5b)	Induction	15	M	Fever, headache, vomiting, diarhoea	Yes	Yes	Subarachanoid hemorrhage	NA	NA	NA	NA	NA	NA	Death
8	Musa et al.[8]	1999	ALL	Induction	14	M	Fever, seizure	No	Yes	Brain herniation	Coma, liver enzymes derrangement	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	Intravascular hemolysis	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	Yes	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	NA Seizure, altered	Yes	No	NA Diffuse cerebral edema, acute	NA	NA	Yes	No	No	NA	Recovery
18	Gaur et al.[11]	2001	ALL	NA	13	F	sensorium	Yes	Yes	hydrocephalus	NA	0	Yes	Yes	Yes	NA	Recovery
19	Gaur et al.[11]	2001	Histiocytosis	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	NA	0	Yes	Yes	Yes	NA	Death
21	Leonard et al.[18]	2002	Alveolar RMS	NA	11	M	Fever, seizures	No	Yes	Brain abscesses	Coinfection with Aspergillus like molds	Low	Yes	NA	NA	NA	Recovery
22	Saleeby et al.[19]	2004	ALL	Induction	17	F	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	Brain abscess	Critical illness polyneuropathy	Low	Yes	Yes	Yes	NA	Recovery
24	Uchino et al.[12]	2012	AML	HiDAC	18	F	Seizures	No	No	NA	Central diabetes insipidus, DIC, grade 3 elevation of liver enzymes	3	Yes	NA	NA	NA	Recovery
25	Sharma et al.[21]	2013	ALL	maintenanc e	5	F	Fever, vomiting	Yes	No	NA	Endocarditis	NA	Yes	NA	NA	CVC	Recovery

(*continued on next page*)

Table 2 (*continued*)

S. no	Author (ref no.)	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
							Fever, headache,			Multiple brain	Ascending cholangitis, left homonymous						
26	Hansford et al.[22]	2014	ALL	Induction	8	M	pain abdomen	Yes	Yes	abscesses	hemianopia	30	Yes	Yes	Yes	CVC	Recovery
27	Dabscheck et al.[23]	2015	ALL	Induction	5	M	Fever	No	Yes	Brain abscess	No	Low	NA	NA	NA	NA	Recovery
							Fever, vomiting,				Acute renal failure, acute pulmonary						
28	Chou et al.[24]	2016	ALL	Induction	15	F	drowsiness	Yes	Yes	No	edema	Low	NA	Yes	Yes	NA	Death
29	Nath et al.[6]	2017	ALL	NA	3	M	NA	NA	NA	NA	Fulminant sepsis	500	NA	NA	No	NA	Death
30	Nath et al.[6]	2017	ALL	NA	11	F	NA	NA	NA	NA	NA	400	NA	NA	No	NA	Recovery
31	Nath et al.[6]	2017	ALL	NA	3	F	NA	NA	NA	NA	NA	300	NA	NA	No	NA	Recovery
32	Nath et al.[6]	2017	AML	NA	14	M	NA	NA	NA	NA	NA	400	NA	NA	No	NA	Recovery
33	Nath et al.[6]	2017	ALL	NA	15	M	NA	NA	NA	NA	Meningitis	1900	NA	NA	No	NA	Recovery
34	Nath et al.[6]	2017	Burkitt's lymphoma	NA	4	M	NA	NA	NA	NA	Cellulitis	800	NA	NA	No	NA	Recovery
35	Nath et al.[6]	2017	ALL	NA	8	F	NA	NA	NA	NA	Cellulitis	1700	NA	NA	No	NA	Recovery
36	Present case	2022	ALL relapse	Induction	15	M	Hematemesis, drowsiness	Yes	Yes	No	DIC, multiorgan dysfunction	395	Yes	Yes	Yes	NA	Death

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
	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%)	15/15 (100%)	1.0
	Absent	0	0	

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/uL 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

lymph histiocytosis 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 fluroquinolones. 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 Ceftazidime-avibactam, Injection Aztreonam, injection Teicoplanin), he progressed to multiorgan dysfunction and died. This shows that B. Cereus septicemia can be lethal in immunocompromised patients and positive blood culture should be flagged immediately.

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

Ethical approval

Not Applicable.

Consent

Informed consent of the parents was obtained.

Disclosure

All authors have nothing to declare.

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