**Case Report**

*Bacillus cereus* Bacteremia and Meningoencephalitis in a Twin-Preterm Neonate: A Case Report and Review of the literature

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**Abstract**

*Bacillus cereus* is a Gram-positive spore-forming, motile and rod-shaped bacterium that produces tissue destructive toxins and it is commonly found in the environment. As a human pathogen, it is known for self-limited acute gastroenteritis after food poisoning. But it is also a rare cause of neonatal sepsis, highly aggressive and often fatal. Herein, we describe a case of *Bacillus cereus* bacteremia and meningoencephalitis in a 4-day-old female twin-preterm neonate. The neonate in only 12 hours after the initial clinical deterioration developed irreversible brain damage and fell to coma. The infant died on her sixth day of life due to cardiorespiratory deterioration and brain stem, central arrest. This case report aims to highlight the importance of clinical suspicion, early diagnosis and effective treatment.

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**Case Presentation**

Female premature, IVF twin B, GA: 33+2-week, BW 1925 gr, born by cesarean section with normal Apgar scores. On postnatal day 4, previously asymptomatic N/G fed with IV fluid supplementation, the newborn presented with apnea, fever and tachycardia. The haematology study showed leucopenia with normal platelets and hemoglobin levels. The first sample of cerebral spinal fluid (CSF) was normal and, CRP initially, negative. The blood and CSF cultures were both positive for *Bacillus cereus*. Twelve hours after the initial signs and symptoms of sepsis, the neonate showed acute clinical deterioration and need of mechanical support: grunting, poor perfusion, tachycardia and opisthotonos. Elevated CRP and persistent leucopenia, as well as, lactic acidosis: pH 7.046, Lac 108 mg/dl were noted. To the broad spectrum –ampicillin and gentamycin-antibiotic treatment, cefazidime and vancomycin was added at high doses. The ultrasound of the brain, only hours from the symptoms’ initiation, showed echogenicity of the choroid plexus and cysts in deep white matter around the ventricles (Figure 1 & 2).

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The clinical deterioration with cerebellar coma was accompanied by U/S progression to parenchymal subcortical and cortical echogenicity (Figure 3 & 4).

The neonate remained intubated, apneic, floppy and unresponsive with no signs of brainstem function. For a short period of time, focal rhythmic spasms of the jaw were noted probably reflexing primitive circuit enrolment, in the absence of upper neuron control. CFM followed suppression pattern (Figure 5 & 6). CSF repeated, direct indicative of meningitis, but sterile. At the end of the 6th day of life, the patient died. The other twin baby is healthy, growing and asymptomatic after a short course of antibiotics which was discontinued with negative infection control.

**Discussion**

The volatile progression of the clinical signs of the CNS infection by *B. cereus* is probably depicting the typical pathological findings described in the literature [7], as the bacilli seem to penetrate the vascular endothelium and also affect the luminal structure, causing the so named “liquefactive necrosis”, following a spatial distribution from central brain vessels to outer layers. A typical disease progression extends from the periventricular white matter, to the deep gray matter and finally the meninges. Probably the case in the vignette had the respiratory center being affected with initial apneic episodes but negative direct indexes for meningitis at the first stages (1st CSF: normal). Thus, evolved to irritability in a few hours, spasms and finally coma (2nd CSF: hemorrhagic/ infectious), after that no respiratory effort was detected on mechanical ventilation. Studies that correlate the pathophysiology with other strains e.g. *B. anthracis* include suffocation at an end point stage of hemorrhagic meningoencephalitis [8].

The strains’ virulence is often attributed to the toxin production. Hemolytic and non-hemolytic gene products act either directly to the host peripheral organs affecting the end tissues either making pores onto cell membranes [9] or utilizing oxidative enzymes, such as enterotoxin, cytoxin K and aerolysin O, which in the adult gut may cause diarrhea, or hemolysin BL strongly related to necrotic lesions [10].

Another proposed mechanism involves the toxins’ role against the host defense system, by increasing oxidative stress, which might have played a major role in our neonate. It has been described that macrophages insult may be the first target [11]. Early-leukopenia was noted in our case, with normal initially platelets, impoverishing the naïve innate immunity system, allowing the bacilli spread, even more effectively, at the blood rich regions, such as the subarachnoid vasculature of the premature brain. Intrinsic individual features of the primary innate immunity seem to have determined the completely different clinical appearance of the IVF separate unaffected twin [12].

Hence the ubiquity of Bacilli spores in the environment is well known, the penetrate in the various nosocomial hosts can hardly been predicted [1,13]. Certainly, parts of the hospital equipment, previously thought as sterilized, can hardly been intact from latent microorganism forms. There are NICUs that after Bacillus’ contamination outbreak have altered the method of sterilization of the ventilator sensor [2], in the case described no respiratory support was being needed or so offered before the complete clinical
deterioration. Though, “spore-form-bacteria” have been found to remain longitudinally over some surfaces. According to recent Space Stations Studies reference [3], when detected, quarantine remains for months or years. Nonetheless, biofilm formation in contact with inorganic material, may cause hardly any sign or mild symptoms as such in a healthy host. The potentially long latent period of the disease has not been extensively described in the literature. One of the most recent extensive surveys [13], including neonatal infected nosocomial population of various hospitals in France concludes that the same strain may remain longer than a two-year period in the same hospital, suggesting that in susceptible populations, having positive sample test for B. cereus should rather be considered as pathogenic rather than a contamination and imposes prompt treatment.

In contrast to acutely developed diarrheic syndrome, spores may last for long but also, the period of inoculation in the same host occasionally cannot be determined. In the case described, after the procedure of amniocentesis, the mother had mild bowel disturbances with diarrhea and was under broad spectrum antibiotic treatment. The twin B sac was diagnosed with 040.3 Polyhydramnios, third trimester, which was the indication for the antibiotic treatment. The twin B sac was diagnosed with 040.3 Polyhydramnios, third trimester, which was the indication for the antibiotic treatment. The twin B sac was diagnosed with 040.3 Polyhydramnios, third trimester, which was the indication for the antibiotic treatment. The twin B sac was diagnosed with 040.3 Polyhydramnios, third trimester, which was the indication for the antibiotic treatment. The twin sibling in our vignette was healthy and asymptomatic despite the first-days-similar conditions in the neonatal department. The two similar neonatal cases referred from the same period and hospital had shown different outcome [13]. Even, the total perinatal incubation period of time remains provocative. Impressively, a premature neonate, as described in literature, had shown latent carriage for weeks since a treated blood stream infection presided weeks before the lethal CNS infection [4].

The difference between the twin siblings, as well as the perinatal period individual events may impose variable vulnerability to the specific bacterial and response. Thus, infection from Bacillus cereus, an opportunistic pathogen may possibly be related to the host’s immune compromised response [5]. In most of the neonatal cases referred, were meningocerebralitis was involved, the outcome was lethal. Among the eight deaths, in total with patients of all ages, reported in the French Study, the four were premature neonates [13]. To our knowledge only a few neonatal cases, did rescue after appropriate treatment. They were term neonates and longitudinal follow up is not further described [6] (Table 1). In table 1, cases of neonates, both, premature and full term with CNS infection are depicted, though it is not extensive it exposes most of those which are referred as case reports.

### Table 1

<table>
<thead>
<tr>
<th>Reference</th>
<th>Year</th>
<th>Neonate/Cns Infection Bacillus cereus</th>
<th>Treatment</th>
<th>Outcome</th>
<th>Predisposing Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Turnbull et al</td>
<td>1977</td>
<td>Female/ 4 days 32 weeks Necrotising enterococatitis Meningitis, cerebral haemorrhage</td>
<td>Ampicillin, gentamicin</td>
<td>Died</td>
<td></td>
</tr>
<tr>
<td>2 Hendricks et al</td>
<td>1981</td>
<td>8d/F Complete hemorrhagic necrosis of the brain</td>
<td>Ampicillin, gentamicin</td>
<td>Died</td>
<td>Ventricular puncture</td>
</tr>
<tr>
<td>3 Feder et al</td>
<td>1988</td>
<td>47d/F Sequelae included Brain damage, hydrocephalus, hypotonia and hyper-reflexia</td>
<td></td>
<td>poor</td>
<td></td>
</tr>
<tr>
<td>4 Patrick et al</td>
<td>1989</td>
<td>26 weeks/ Female/ 7 days Bilateral thalamic haemorrhage, Encephalitis</td>
<td>Vancomycin, amikacin</td>
<td>Died</td>
<td></td>
</tr>
<tr>
<td>5 Weisse et al</td>
<td>1991</td>
<td>5d/M36 weeks/ 21d/M term Meningitis</td>
<td>Vancomycin, gentamicin, chloramphenicol</td>
<td>Recovered</td>
<td>Myelomeningocele; none</td>
</tr>
<tr>
<td>6 Tokieda et al</td>
<td>1999</td>
<td>4d/F 5d/F Multiple brain parenchyma, subdural, epiderumaLand subarachnoid hemorrhage; and widespread softening and hemorrhagic necrosis of the brain</td>
<td>Ampicillin, gentamicin, cefotaxime</td>
<td>Died</td>
<td>Peripheral venous catheter; nasal feeding tube</td>
</tr>
<tr>
<td>7 Tuladhar et al</td>
<td>2000</td>
<td>14d/M Intraventricular hemorhage</td>
<td>Vancomycin, gentamicin, imipenem, clindamycin, ciprofloxacin, igh</td>
<td>Died</td>
<td>Not identified</td>
</tr>
<tr>
<td>8 Chu et al</td>
<td>2001</td>
<td>28d/M Meningitis, Infarction and liquefactive necrosis of the whole brain</td>
<td>Vancomycin, amikacin</td>
<td>Died</td>
<td>Bronchopulmonary dysplasia,- decamethasone used</td>
</tr>
<tr>
<td>9 Heep et al</td>
<td>2004</td>
<td>14d/M Ventriculitis, hemorrhagic necrotizing lesions</td>
<td>Vancomycin, gentamicin, meropenem</td>
<td>N/A</td>
<td>Ventriculostomy tube</td>
</tr>
</tbody>
</table>
In conclusion, *B. cereus* infection may be rare but there must be awareness concerning its toxicity and high mortality among neonates. It should always be suspected especially in preterm and extremely low birth weight infants with symptoms of sepsis.

**Acknowledgement**

None.

**Conflict of Interest**

No Conflict of Interest.

**References**