Bacillus Cereus Meningoencephalitis in a Neonate

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Abstract   Neonatal meningoencephalitis caused by Bacillus cereus is rare. In most cases, the infection is fatal because of extensive damage and necrosis of infected tissue caused by the toxins produced by B. cereus. We report on a 17-day-old preterm neonate who developed B. cereus bacteraemia and severe haemorrhagic meningoencephalitis. The infant survived. The ultrasound, CT and MRI images showed pattern of haemorrhagic and early cavitating, selective white matter destruction. The interval from the time of recognition of illness to irreversible damage of the central nervous system was short which demonstrates a need for increase awareness, early diagnosis, and more-effective therapy. We describe the clinical course and a distinct neuroimaging characteristic for this organism.

Key words   Bacillus cereus; Meningoencephalitis; Neonate; White matter injury

Introduction

Bacillus cereus is a motile, aerobic or facultatively anaerobic, spore-forming, gram-positive or gram-variable bacterium of the family Bacillaceae that is found worldwide in dust, air, and water. The organism is often regarded as a contaminant of specimens received by the microbiology laboratory, and a positive growth of B. cereus is often disregarded by clinicians and microbiologists. As a human pathogen, the organism is perhaps best known for its role as a mediator of self-limited food borne illness. Among members of this genus, only B. anthracis[1] potentially more significant as a cause of human disease.[1] The first report of neonatal meningitis and bacteraemia due to B. cereus occurred in 1989. A review of the English-language literature yielded 8 reports of B. cereus meningoencephalitis.[2] Most neonatal case reports showed high incidence of haemorrhagic necrosis and poor prognosis. We report a preterm neonate who survived with mild sequalae at 10 month follow-up. We also reviewed the literature on neonatal B. cereus meningoencephalitis.

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Case Report

A 1425-g male infant was born to a 39-year-old Hispanic female G6 P1414 by Cesarean section after 29-weeks' gestation because of placental abruption. Apgar scores were 5 and 8 at 1 and 5 minutes respectively. The pregnancy was uneventful. The infant had respiratory distress immediately after birth and was treated with exogenous surfactant and mechanical ventilation. Umbilical artery and vein catheters were placed after birth and removed after 72 hours of life. A negative blood culture was obtained on admission.

Mechanical ventilation was discontinued after 3 days. Intravenous hyperalimentation including lipids were started on day 2 and enteral feeding began on day 5. On day 17, the infant became lethargic, pale, tachycardic and hyperglycemic. He was again intubated and ventilated. After a blood culture was obtained antibiotic therapy with vancomycin and amikacin was initiated. Lumbar puncture was performed 2 hours after antibiotic treatment.

A sample of cerebrospinal fluid (CSF) showed WBC 0.376×10^6/L (93% neutrophils, 3% lymphocytes, and 4% monocytes); red blood cell 1.2×10^6/L, 3 g/L of protein, 5.44 mmol/L of glucose (serum glucose 11.93 mmol/L). CSF culture was negative. On day 18, seizure activities with stiffening of the limbs and desaturations were noted. Neurosonogram showed bilateral periventricular echodense regions (Figure 1). A neurosonogram on day 20 showed liquefactive necrosis in the periventricular areas and the presence of leukomalacia. On day 25, a CT scan of the head showed extensive areas of encephalomalacia. A blood culture grew a gram positive bacilli organism with the bacterium being identified as Bacillus cereus. Susceptibility tests indicated susceptibility to vancomycin, clindamycin and gentamicin. A ventricular tap 10 days after the treatment was started showed bloody CSF with 0.11 mmol/L of glucose, 4.84 g/L of protein and a WBC of 15.4×10^6/L (97% neutrophils). The neonate received 21 days of vancomycin. Additional blood and CSF cultures performed after completion of the antimicrobial chemotherapy were negative. On day 44, a MRI of the head showed extensive encephalomalacia (Figures 2 & 3). Hydrocephalus developed and on day 65 he underwent ventriculostomy placement. He was discharged after ventriculoperitoneal shunt placement at 90 days of life. At 10 months of age, he showed mild developmental delay.
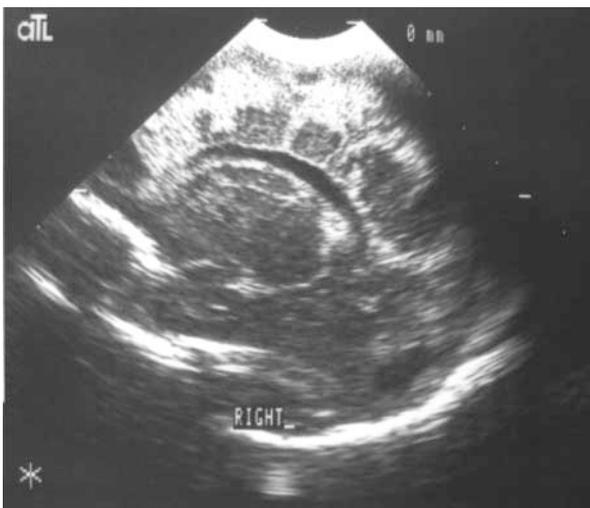

Figure 2   Axial MRI on day 44 showing diffuse cystic encephalomalacia and prominent CSF-containing spaces.

Figure 1   Sagittal sonography (7.5 MHz) on day 18 showed increased echogenicity radiating along the entire length of the lateral ventricles.
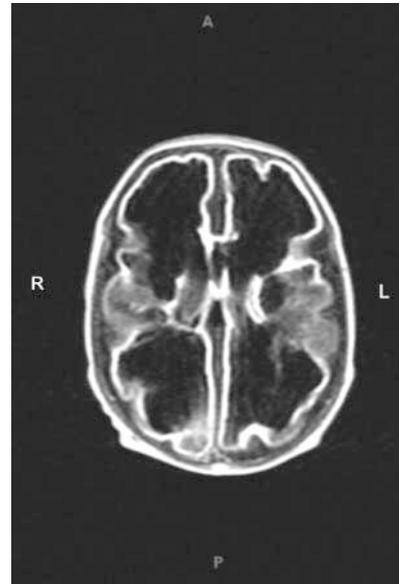

Figure 3   Sagittal MRI on day 44 showing diffuse cystic encephalomalacia and prominent CSF-containing spaces.

Discussion

Although neonatal meningoencephalitis caused by B. cereus and extensive damage and necrosis of the white matter has been previously reported, there is only one report of an MRI in neonates. In most cases, this is fatal due to toxins produced by B. cereus that induced necrosis of infected tissue. The enterotoxin, phospholipases, proteases, and haemolysins induce a widespread liquefactive necrosis.[3,4] Our case emphasizes the neuroimaging characteristics of extensive damage and necrosis of subcortical and periventricular white matter, sparing the cortex, similar to the previous report.[3] This preference could be explained by spread of the infection along white matter tracks.[2,3]

In our case, there was a rapid change from hyperechogenicity to cystic destruction within 24 to 48 hours. We emphasize the importance of serial brain sonography in preterm neonates with sign of sepsis and convulsions. Clinicians need to be made aware of the rapid progression of the infection and the apparent inability of antibiotics to halt the disease process which is typical of infection with B. cereus.

In recent literature, few reports documented imaging in patients with neonatal (mainly gram-negative: Proteus, Serratia, Citrobacter) bacterial meningoencephalitis.[5,6] Serratia marcescens the most common cause of haemorrhagic meningoencephalitis in infants.[7] When gram positive bacilli are found in the blood and CSF, it is frequently regarded as contamination by Bacillus species. Even if the presence of the bacilli is considered important, as in the setting of neonatal meningitis, the infection with Listeria monocytogenes is usually presumed. The standard empirical treatment with high doses of ampicillin and cefotaxime covers Listeria species but not B. cereus. The possibility of B. cereus septicemia and meningitis needs to be considered.

Bacillus cereus is a ubiquitous, gram positive organism that commonly induces food poisoning. B. cereus can readily contaminate the hospital environment, including the uniforms of health care workers, patients' dressings, or intravenous catheters. Despite the widespread distribution of the Bacillus genus, they rarely cause systemic infections. This organism has also been described as causing localised infections, specifically destructive eye infections that lead to orbital abscesses and endophthalmitis. Little is known about the source of transmission of neonatal B. cereus infection. Diligent analyses of possible risk factors must be considered. Common risk factors include bowel perforation,

ventriculoperitoneal shunting, intravenous or arterial catheterisation, neonatal leukaemia, mechanical ventilation, and bronchopulmonary dysplasia.[8,9] Clinicians need to be made aware of B. cereus as a potential pathogen in predisposed patients such as premature babies. Assessment of the origin of the infections is difficult due to the wide dissemination of B. cereus in the environment.

A report from The Netherlands[10] described 35 neonates in a hospital unit that were colonised in the respiratory tract with a single B. cereus clone. The epidemic strain of B. cereus was detected on the hands of nursing staff and in mechanical ventilation equipment. Other report documented intravenous catheters and ventriculoperitoneal shunts as modes of transmission.[4] We did not perform an epidemiologic investigation to find the source of B. cereus in our case. The predisposing factors in our patient were prematurity, mechanical ventilation and indwelling peripheral catheter.

Clinicians should be attentive to the risk factors and increasing awareness of the potential role of B. cereus as human pathogen especially in premature neonates. Whenever Bacillus species of possible clinical significance are encountered, prompt notification with clinicians are extremely important. Most strains of B. cereus are resistant to penicillins and cephalosporins. Combination therapy with vancomycin or clindamycin and aminoglycoside given empirically while awaiting susceptibility data has been recommended for systemic infections.[11] In the literature, most of central nervous system infection with B. cereus in very low birth weight premature followed a devastating course. Of the 8 reported cases, one survived and had cerebral palsy.[2,3] Our patient survived probably because of a prompt administration and correct combination of antibiotics. Outcome in our patient is still too early to established.

In conclusion, we described a preterm neonate with disseminated B. cereus infections and a distinct MRI neuroimaging characteristics. Though it occurs infrequently in neonates, it is not a minor concern because of the fatal clinical cause. For preliminary empiric therapy, the paediatricians should be reminded of the possibility of B. cereus infection, and delay in appropriate treatment can easily lead to significant mortality in systemic disease.

Acknowledgements

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