Cauliflower in a preterm brain
This male infant was born to a 29-year-old G4/P3 mother at 27 5/7 weeks of gestation. Apart from preeclampsia, which was the reason for the caesarean section, the medical history and the pregnancy were uneventful. Umbilical arterial cord-pH was 7.40, Apgar scores were 3, 6, and 6 at 1, 5 and 10 minutes, respectively. The baby was intubated because of respiratory insufficiency and transported to the neonatal intensive care unit.

His birth weight was 920 g, the length 34.0 cm and the head circumference 24.5 cm (all between the 10th and 25th percentile).

Surfactant was given at the age of 20 minutes and the infant could be rapidly weaned from the ventilator. After receiving caffeine, the baby was extubated at the age of 13 hours and placed on nasal-CPAP with an FiO2 of 23%. The first two cerebral ultrasound scans at the age of 9 and 38 hours were normal.

On the third day of life (44 hours of age), the baby developed sudden respiratory distress. Blood gas analysis showed a mild metabolic acidosis. No further signs of infection were detected at that stage. He was intubated and a cerebral ultrasound was performed. This third ultrasound scan showed multiple cauliflower-like intra-parenchymatous necrotic lesions in the white matter (Fig. 1, 2). The grey matter was normal and no intracerebral hemorrhage was detected.
The baby deteriorated very rapidly and developed fulminant septic shock requiring several vasopressors and volume replacement therapy. A blood culture was obtained and antibiotics, amoxicillin and gentamycin, were started. Despite maximum therapy, the baby died of multiorgan failure within five hours. Blood culture and cerebrospinal fluid culture (which was obtained postmortem) were positive for Bacillus cereus. Even before these results became available, Prof. Linda de Vries had suggested a diagnosis of Bacillus cereus meningoencephalitis based on the cerebral ultrasound images.

DISCUSSION

Bacillus cereus is a gram positive spore forming rod. It is a ubiquitous organism which can contaminate the hospital environment, but is also known to be present in food (non-anthrax Bacillus species) (1). Bacillus cereus produces toxins, including necrotizing enterotoxins, phospholipases, proteases and hemolysins. Local infections, such as destructive eye infections with orbital abscesses and/or endophthalmitis have been described. Systemic infections affecting the central nervous system are less well recognized. This may be because microbiologists often regard Bacillus cereus as a contaminant. In preterm infants, Bacillus cereus may cause devastating systemic infections including severe hemorrhagic meningoencephalitis.
Most infants with Bacillus cereus meningoencephalitis appear to have the same clinical course: an uncomplicated delivery and uneventful first days of life. They present after a symptom-free interval with acute symptoms of meningitis and leukocytosis, hypoglycemia, thrombocytopenia, hypotension and shock. Despite adequate therapy most patients die (2, 3).

The most frequent infection in the newborn caused by gram positive rods is listeriosis. Bacillus cereus is susceptible to aminoglycosides, clindamycin, chloramphenicol and erythromycin, but - in contrast to Listeria monocytogenes - resistant to β-lactam antibiotics such as penicillins and cephalosporins (5).

The diagnosis of a Bacillus cereus meningoencephalitis is made by culture of blood and cerebrospinal fluid, but also by the typical and pathognomonic cauliflower-like pattern on cerebral ultrasound representing hemorrhagic lesions with central necrosis and asymmetric extensive destruction of the white matter.
Cerebral ultrasound examination on 3rd day of life: multiple intraparenchymatous necrotic white matter lesions (coronal views: A) frontal region, B) parietal region, C) occipital region).
Cerebral ultrasound examination on 3rd day of life: multiple intraparenchymatous necrotic white matter lesions (parasagittal views: A) paramedian left, B) lateral left, C) lateral right).


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