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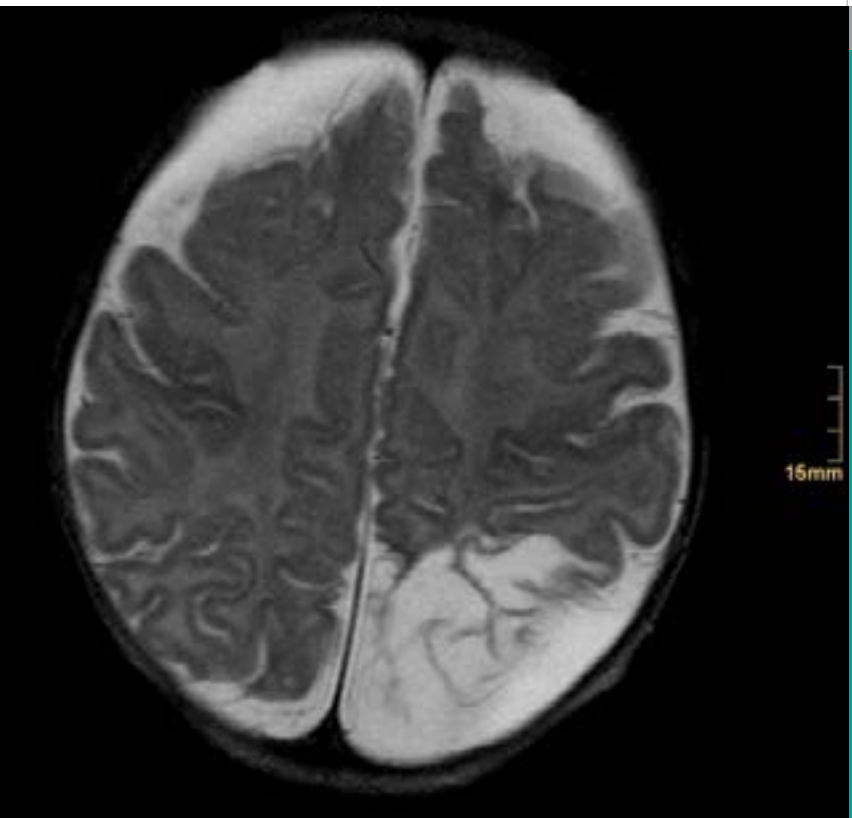
Group B streptococcal meningitis in a 7-day-old neonate: early- versus late-onset disease?



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Based on the postnatal age when clinical signs of infection become apparent, neonatal infections with group B streptococci (GBS) are classified into early-onset and late-onset infections. Early-onset infections almost always manifest within 24 hours of birth, but may appear in the second 24 hours or at any time during the first 7 days of life (1). In contrast, late-onset infections occur at 7 to 90 days of age. Early-onset infections are normally acquired from the mother's genital tract, whereas late-onset disease is believed to reflect delayed infection after early colonization or cross infection. Cases of ingestion of contaminated mother's milk followed by an infection of the neonate have been reported.

GBS meningitis more often occurs in late-onset disease (35-40%) than in early-onset disease (5-10%). In early-onset infections, serotype Ia is isolated most frequently (38%), followed by serotype III (31%), V (14%) and II (10%). This contrasts with a predominance of serotype III strains in late-onset cases, irrespective of the focus of infection. Meningitis can occur with all strains, but serotype III strains have a particular propensity for meningeal invasion.

Because the pathogenesis of late-onset infections is not fully understood, it is unknown if and how these infections can reliably be prevented. Specifically, it is not clear whether late-onset infections after early colonization can be prevented by intrapartum chemoprophylaxis (4, 5).

This male infant was delivered vaginally at 38 5/7 weeks of gestation (birth weight 3540 g) to a healthy 24-year-old G2/P2. At 32 weeks of gestation, a vaginal smear was positive for GBS, and the mother was treated with oral antibiotics (amoxicillin/clavulanic acid). When the mother was admitted, the results of the vaginal smear were not communicated to the treating team, and no intrapartum chemoprophylaxis was administered. Rupture of membranes occurred two hours before birth and the amniotic fluid was clear. The mother was afebrile during labor and delivery.

Postnatal adaptation was uncomplicated with Apgar scores of 8, 9, 10 at 1, 5 and 10 minutes, respectively. On the fifth day of life, after an uneventful postpartum period, mother and infant were discharged from the hospital.

On the seventh day of life, the infant was admitted to the hospital because of fever, irritability and poor feeding. On admission, there was a shrill cry, increased muscle tone with a tendency for opisthotonus and the anterior fontanel was slightly bulging. His white blood cell count was 3.1 G/l with a marked left shift, the C-reactive protein concentration was 19 mg/l, hemoglobin concentration and platelet count were normal. CSF analysis revealed a white blood cell count of 1005/µl with a predominance of polymorphnuclear cells, protein and glucose concentrations of 3.72 g/l and 0.5 mmol/l, respectively. Group B streptococci were ulti-

mately grown in blood, CSF and breast milk cultures.

Appropriate antibiotic treatment was administered for two weeks. Because of the GBS-positive breast milk sample, the mother was treated with oral penicillin for 10 days. Repeat breast milk cultures were negative and breast feeding was restarted.

On the 14th day of life, cerebral ultrasound showed increased extracerebral fluid in the fronto-parietal region bilaterally, consistent with subdural hygromas. Nine days later (23rd day of life), parenchymal lesions in the left frontal lobe were demonstrated (Fig. 1), probably representing infarctions. The left parieto-occipital lobe was slightly hyperechoic without the usual crisp gyral pattern (Fig. 2).

An initial EEG revealed marked depression of background activity, focal amplitude depression posteriorly on the left side as well as multifocal sharp-slow-waves. Clinical seizures were treated with phenobarbitol.

During regular follow-up visits, distinctive features of cerebral palsy became evident. A follow-up EEG showed no evidence of epileptogenic potentials, and after a seizure-free period of two months, the antiepileptic medication could be stopped. At the age of nine months, there were both cognitive and

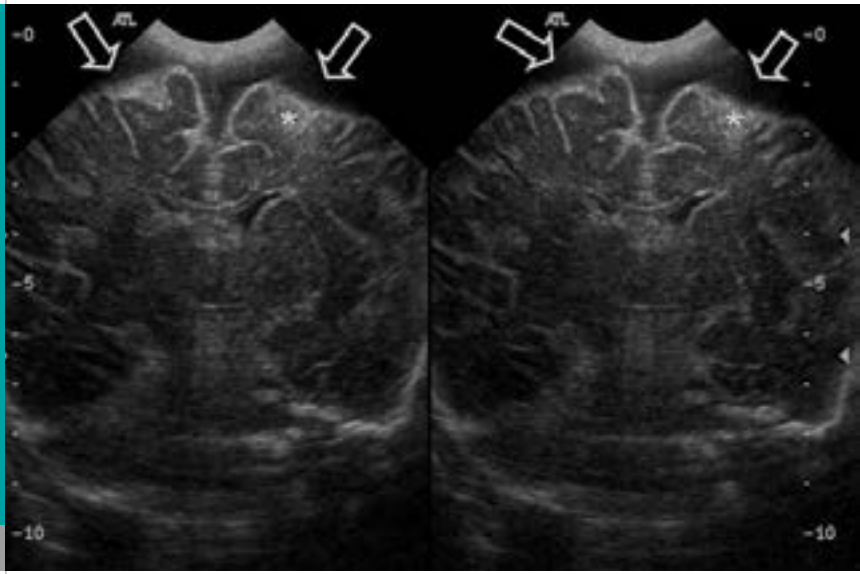

Fig. 1

Sonography of the brain (23rd day of life): coronal view at the level of the foramina of Monroi demonstrates bilateral hygromas (arrows) and a small parenchymal lesion in the frontal lobe on the left (asterisk).

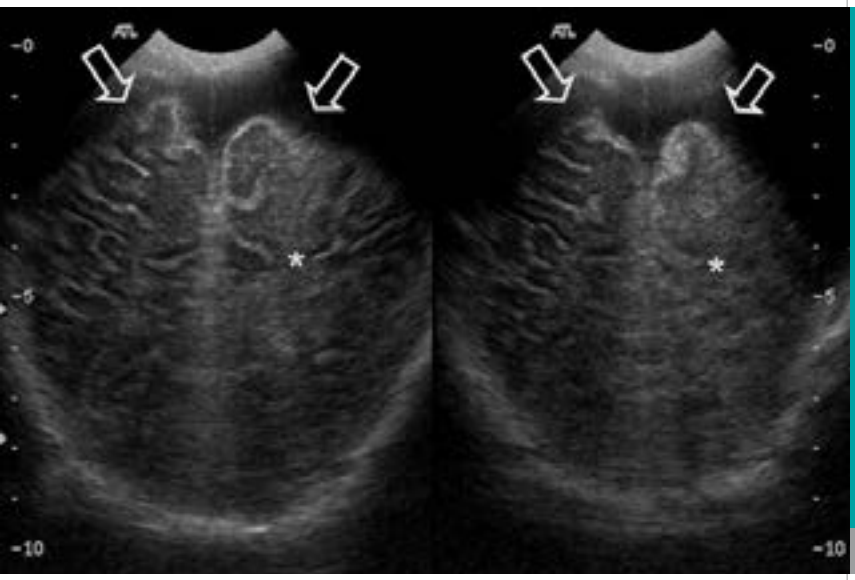

Fig. 2

Sonography of the brain (23rd day of life): posterior coronal view demonstrates a markedly less well defined gyral pattern of the parieto-occipital lobe on the left (asterisk). Bilateral hygromas extend into this region (arrows).

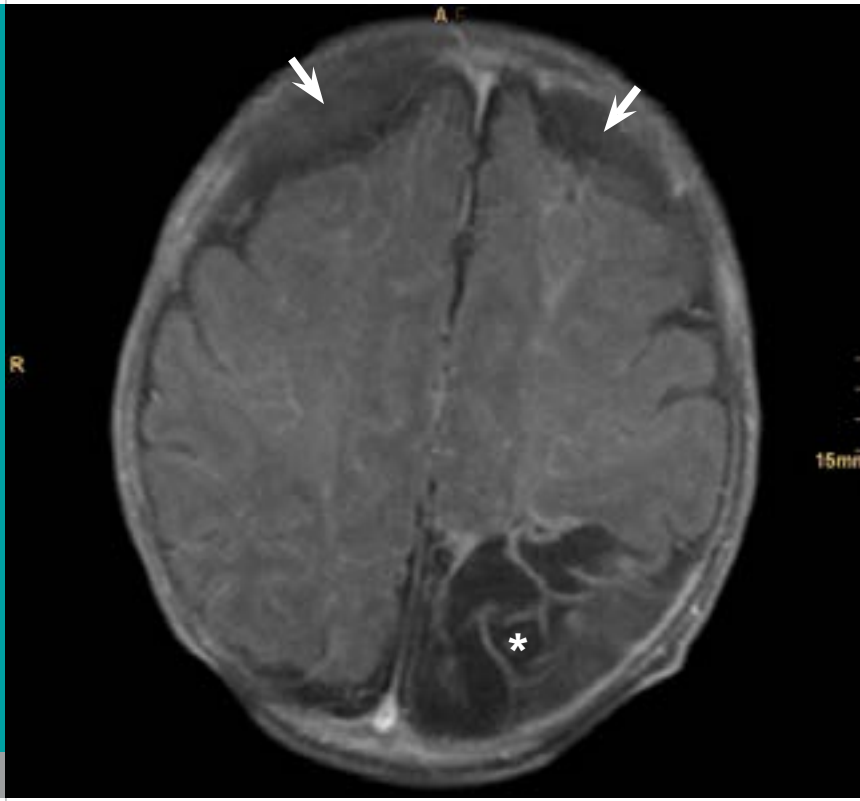

Fig. 3

Axial T1-weighted MR image (42nd day of life): marked parenchymal destruction in the parieto-occipital lobe on the left (asterisk) and bilateral hygromas (arrows).

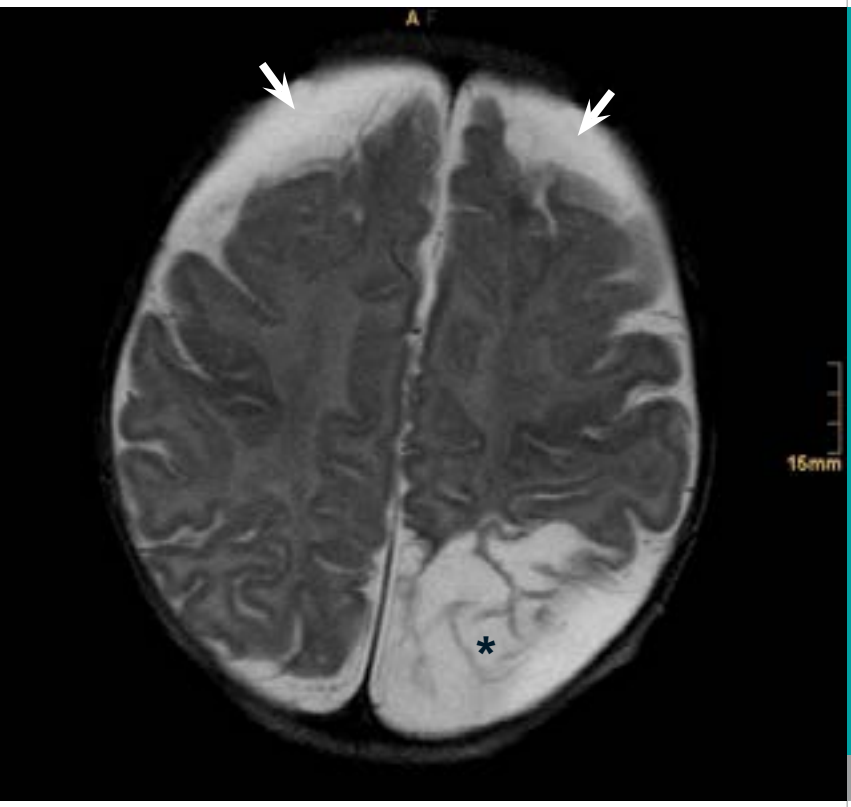

Fig. 4

Axial T2-weighted MR image (42nd day of life) at the same level demonstrating the same findings as in figure 3.

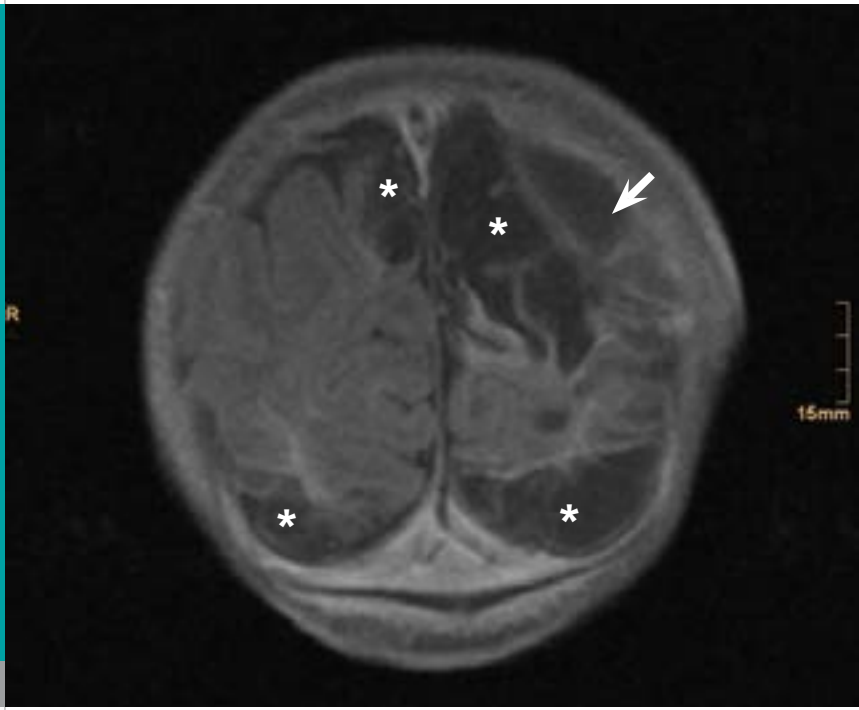

Fig. 5

Coronal T1-weighted MR image (42nd day of life): extensive parenchymal destruction in both parietal-occipital lobes, mainly on the left side (asterisks) and bilateral hygromas (arrows).

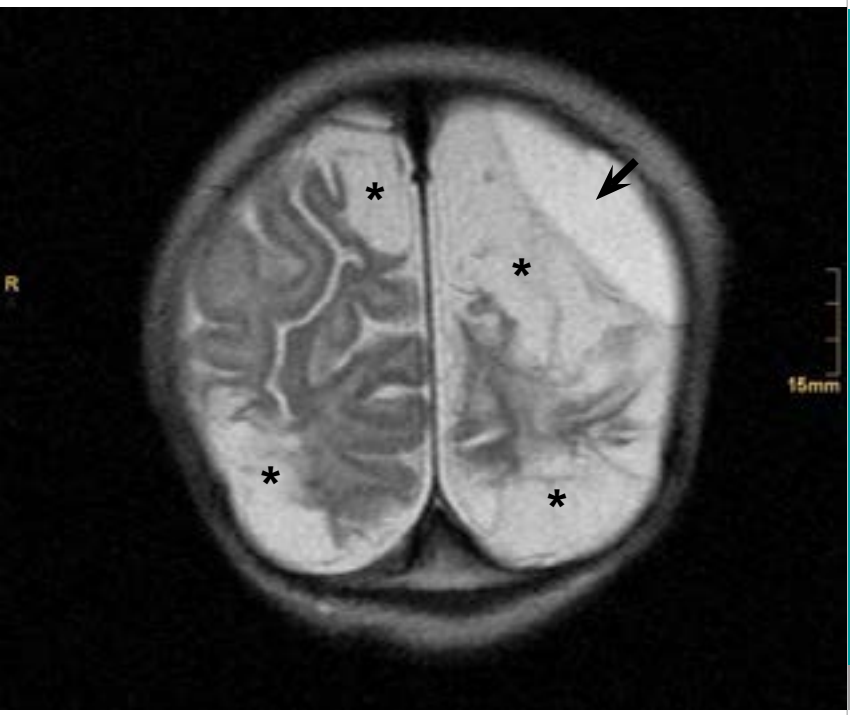

Fig. 6

Coronal T2-weighted MR image (42nd day of life) at the same level demonstrating the same findings as in figure 5.

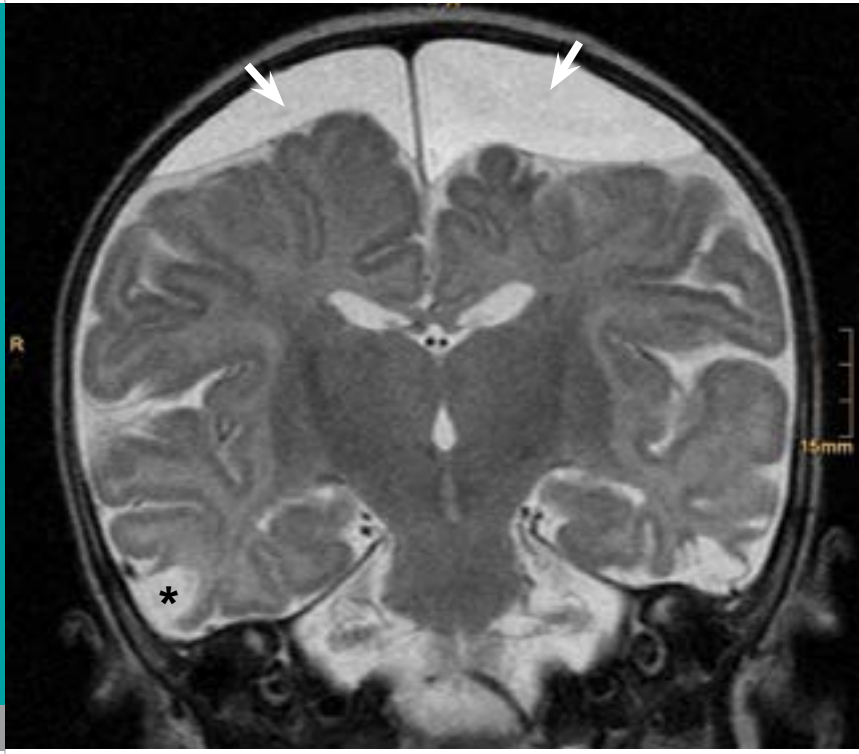

Fig. 7

Coronal T2-wt MR image (42nd day of life): illustrative view of marked bilateral hygromas (arrows) and small parenchymal lesions (asterisks).

motor developmental delays of approximately 2-3 months. Follow-up cerebral ultrasound demonstrated slightly dilated lateral ventricles, marked parenchymal lesions in the left cerebral hemisphere. The subdural hygromas were unchanged on the left side and decreasing in size on the right

On the 42nd day of life, an MRI showed multiple areas of destruction of the parenchyma, mainly in the left parieto-occipital lobe (Fig. 3-6) with residual hemorrhagic and necrotic changes.

DISCUSSION

It is unclear whether this case should be classified as early- or late-onset GBS sepsis. Therefore, we can only speculate if an appropriate intrapartum chemoprophylaxis could have prevented this serious infection. It must be emphasized, however, that the detection of GBS during pregnancy usually does not require immediate antibiotic therapy but should always prompt intrapartum chemoprophylaxis. Many women who are treated during pregnancy will become GBS carriers again prior to delivery. Therefore, GBS screening is recommended between 35-37 weeks of gestation (5).

If our case represents late-onset GBS sepsis with meningitis, the contaminated breast milk might have been the source of infection (6, 7). GBS may be present in breast milk without causing clinical mastitis.

Some case reports of assumed transmission of GBS through breast milk have suggested the use of rifampicin and amoxicillin to eradicate carriage of the organism in mother and neonate (8). Currently, the clinical significance of GBS positive breast milk remains unclear.

The prognosis of neonatal GBS meningitis is severe. Reported mortality rates range between 8.5 to 12.4% (9). Permanent neurologic sequelae occur in about one third of the survivors. The most severely affected infants may suffer from global mental retardation, cortical blindness and/or spasticity.

REFERENCES

1. Baker CJ, Edwards MS. Group B streptococcal infections: perinatal impact and prevention methods. Ann N Y Acad Sci 1988;549:193-202

2. Edwards MS, Baker CJ. Group B Streptococcal Infections. In: Remington J, Klein J. Infectious diseases of the Fetus and Newborn Infant. W.B. Saunders, 5th ed. (2001):1091-1156

3. Edwards MS, Rench MA, Haffar AAM, et al. Long-term sequelae of group B streptococcal meningitis in infants. J Pediatrics 1985;106:717-722 *(Abstract)*

4. Kind C. Care of neonates whose mothers are colonised with group B streptococci. Recommendations by the Swiss Society of Neonatology (revised version, see *www.neonet.ch*)

5. Prevention of Perinatal Group B Streptococcal Disease. Revised guidelines from CDC. MMWR Recomm Rep 2002 Aug 16;51(RR-11):1-22 *(Abstract)*

6. Dinger J, Müller D, Pargac N. et al. Breast milk transmission of group B streptococcal infection. Pedatr Infect Dis J 2002;21:567-568 *(Abstract)*

7. Olver W, Bond D, Boswell S et al. Neonatal group B streptococcal disease associated with infected breast milk. Arch Dis Child Fetal Neonatal Ed 2000;83:48-49 *(Abstract)*

8. Atkins J, Heresi G, Coque T et al. Recurrent group B streptococcal disease in infants: Who should receive rifampicin? J Pediatrics 1998;132:537-539 *(Abstract)*

9. Heath PT, Yusoff NK, Baker CJ. Neonatal meningitis. Arch Dis Child Fetal Neonatal Ed 2003;88;F173-F178 *(Abstract)*

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