CASE REPORT

Neonatal meningitis associated with osteomyelitis and epidural empyema

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SUMMARY

Neonatal meningitis is a serious disease with significant mortality and morbidity. Its signs and symptoms are subtle, non-specific, atypical or absent.
Cephalohaematoma is frequent in newborns and complications are uncommon, including local infection after haematogenous spread in the setting of bacteraemia or meningitis with a possibility of osteomyelitis, epidural abscess and subdural empyema.
We report the case of a late preterm newborn, with an unremarkable pregnancy, born by vacuum-assisted vaginal delivery that presented in the fifth day of life with irritability, fever and grunting. Cerebrospinal fluid and blood cultures were positive for *Escherichia coli*. The patient had neurological deterioration despite adequate antibiotic therapy and brain MRI showed a right parietal epidural empyema, subcutaneous abscess, osteomyelitis and supratentorial hydrocephalus. The culture of the cephalohaematoma's abscess material was positive for *E coli*. Antibiotic therapy was continued for 8 weeks. The child, now 2 years old, has spastic tetraparesis with global development delay.

BACKGROUND

Neonatal meningitis is an important cause of mortality and morbidity. Newborns (NB) have a higher risk of meningitis and neurological complications.[1 2] Over the last decades there as been a decline in mortality, which is about 10–15%, but morbidity remains high, up to 20–50%.[1 2]

The epidemiology of meningitis in the neonatal period is similar to that of neonatal sepsis.[1 2] Gram-negative enteric bacilli account for 30–40% of neonatal meningitis cases. *Escherichia coli* is the most common organism, accounting for up to 50% of Gram-negative isolates during the neonatal period.[1 2]

In neonatal meningitis, a repeated cerebrospinal fluid (CSF) evaluation is recommended after 48–72 h of treatment. This follow-up is of therapeutic and prognostic significance as the delay in CSF sterilisation may suggest antimicrobial resistance or an infectious meningeal or para-meningeal focus, such as obstructive ventriculitis, abscess, subdural empyema or multiple small vessel thrombi.[1 2] A delay in CSF sterilisation is a feature of Gram-negative meningitis, and may be responsible for its higher mortality.[1 2]

Cephalohaematomas occur in 1–2% of spontaneous vaginal deliveries and in approximately 4% of forceps or vacuum-assisted deliveries.[3–5] The frequency is higher in male newborns and in primiparous mothers.[6] Complications of cephalohaematomas are uncommon and include infection. Bacterial infection of a cephalohaematoma is usually associated with needle aspiration, scalp infection, scalp electrodes, sepsis or meningitis.[3–6] The most common infecting organism are *E coli* and *Staphylococcus aureus*; other reported organisms include *Staphylococcus epidermidis*, *Pseudomonas*, *Proteus*, *Salmonella*, *Gardnerella* species, group B *Streptococcus* and anaerobes.[4 5]

CASE PRESENTATION

A female, late preterm newborn, second birth from an unremarkable pregnancy, with normal prenatal ultrasound, irrelevant serological maternal investigation and no risk factors for infection was born by vacuum-assisted vaginal delivery to a 30-year-old gravida 2, para 2 mother, at a gestational age of 36 weeks and 6 days, with good adaptation to extrauterine life. Somatometry was appropriate for the gestational age. Her physical examination was normal with the exception of a right parietal cephalohaematoma. She became jaundiced on the second day of life with hyperbilirubinaemia of 236 µmol/L, for which she underwent phototherapy for 24 h. She was discharged home on the fourth day of life, clinically well. Twelve hours postdischarge, she was admitted to the neonatal intensive care unit (NICU) for fever (38°C), irritability and grunting.

INVESTIGATIONS

Initial blood tests showed leukopaenia (white cell count 1.9×10^9/L) with neutropenia (1.1×10^9/L neutrophils), anaemia (haemoglobin 9.5 g/dL) and thrombocytopaenia (platelet count 64×10^9/L). A peripheral blood smear showed neutrophils with hyposegmentation, cytoplasmic vacuolation and toxic granulation. C reactive protein was 17.1 mg/dL.

A lumbar puncture (LP) was performed and CSF analysis revealed hypoglycorrhachia (glucose <1 mmol/L) and elevated proteins (>900 mg/dL); the CSF sample was insufficient for cytological examination and an antigen detection test.

Blood and CSF cultures were positive for *E coli*, resistant to β-lactams and sensitive to gentamycin.

TREATMENT

Antibiotic therapy with ampicillin (200 mg/kg/day), gentamicin (4 mg/kg/day) and cephotaxime (100 mg/kg/day) was promptly initiated. After the blood and CSF culture results, ampicillin was suspended.

Red blood cell transfusion was performed on day 2 and plasma transfusions from days 3 to 6.

To cite: Vale B, Morais S, Resende C, *et al*. *BMJ Case Rep* Published online: [*please include* Day Month Year] doi:10.1136/bcr-2013-009149

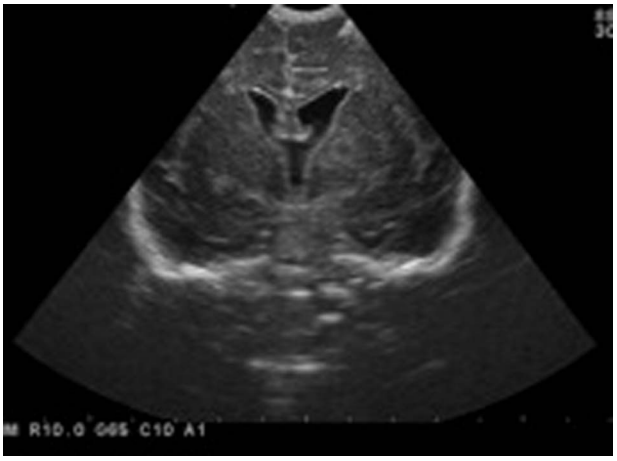

Figure 1 Cranial ultrasound with ventriculitis, dilation of the lateral ventricles and third ventricle.

OUTCOME AND FOLLOW-UP

The patient underwent clinical deterioration despite adequate antibiotic therapy. On the first day of admission, after a complicated episode of apnoea with bradycardia, she was intubated and ventilated with ventilatory support parameters for 7 days. Neurologically, on the second day of admission, she showed a generalised hypertonia, an opisthotonus position with internal rotation of the upper limbs when manipulated and initiated seizures. She started on phenobarbital that was stopped on the ninth day of admission, after 2 days without seizures. Because of a persistent position in opisthotonos and hypertonia, the LP was repeated on the 10th day of admission, with a negative CSF culture. On the same day, a cranial ultrasound was performed, which showed ventriculitis and moderate ventricular dilation with enlargement of the third ventricle (figure 1). The brain MRI showed mild-to-moderate supratentorial hydrocephalus and, on the right superior parietal level, an organised collection suggestive of epidural empyema in communication with a subcutaneous abscess and osteomyelitis of the skull (figure 2). At this time, an increase in the dimensions and consistency of the right parietal cephalohaematoma was noticeable. A skull X-ray showed thinning in the right parietal region (figure 3).

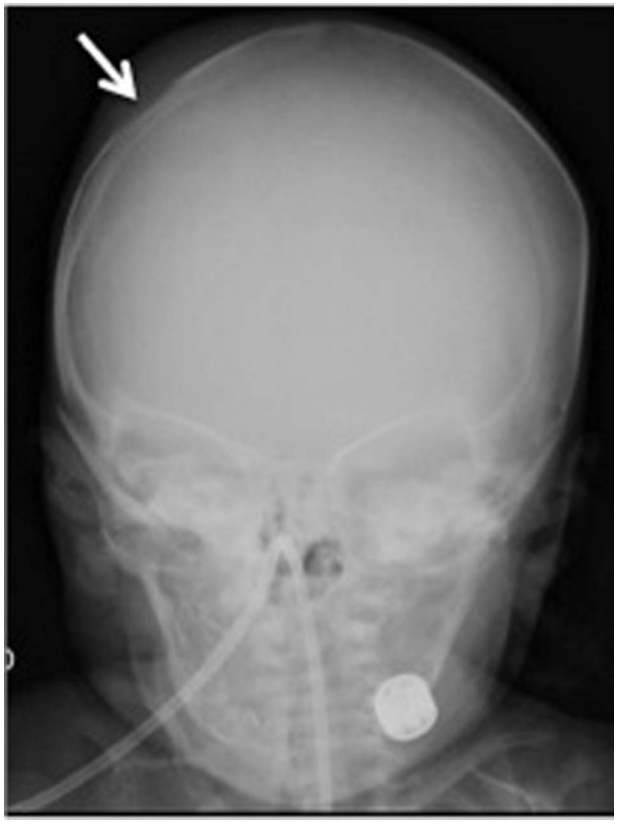

Figure 3 Skull X-ray with thinning in the right parietal region.

Abscess and epidural empyema drainages were performed and an Ommaya ventricular reservoir was placed on the left ventricle. The abscess exudate culture of the cephalohaematoma was positive for *E coli*. Owing to clinical and laboratory deterioration (table 1), cephotaxime was changed to meropenem (40 mg/kg/day) and an aminoglycoside was maintained. For dehiscence and persistent CSF drainage from the surgical sutures since the fifth day after surgery, the ventricular derivation system was withdrawn 15 days later. Vancomycin was then added to treatment (15 mg/kg/day) as she maintained fever, irritability, continuous opisthotonos and horizontal nystagmus.

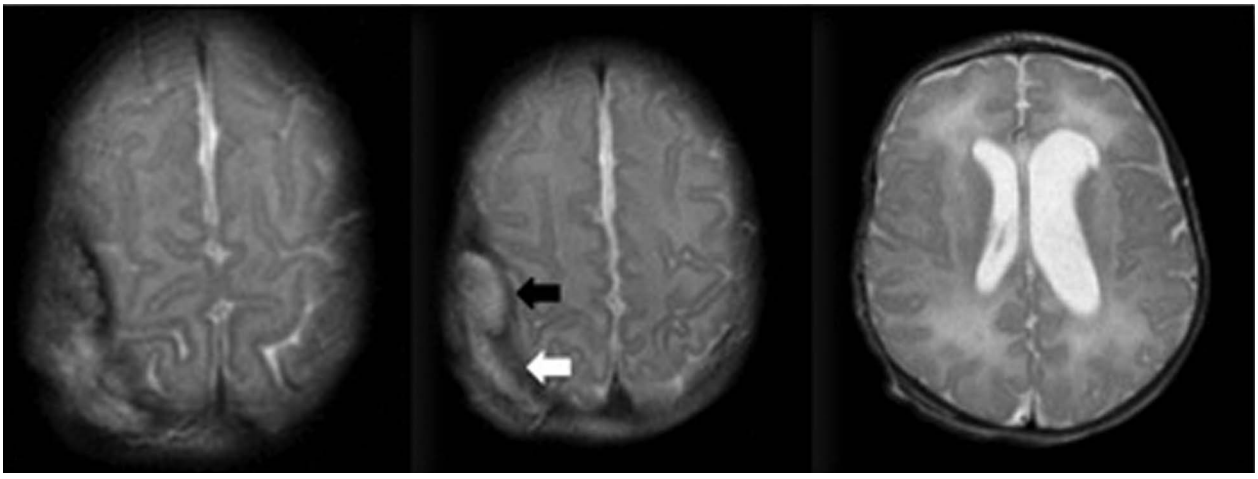

Figure 2 Brain MRI with epidural empyema (black arrow), in communication with a subcutaneous abscess (white arrow) with the location of the cephalohaematoma, and moderate ventriculomegaly.

Table 1 Analytical evolution during hospitalisation

	D1	D3	D4	D5	D6	D16	D17	D24
Hb (g/dL)	9.5	10.1	11.5	13.8	13.1	11.2	9.8	10.1
WCC (/μL)	1900	17 210	22 520	24 710	27 670	13 500	20 500	16 200
Platelets (/μL)	64 000	5000	15 000	40 000	37 000	42 8000	31 4000	64 4000
CRP (mg/dL)	17.1	29.2	19.4	8	4.6	1	8.8	0.8

CRP, C reactive protein; Hb, haemoglobin; WCC, white cell count.

After the reservoir withdrawal, for moderate hydrocephalus, evident in the cranial ultrasound and head CT scan, a new external reservoir was placed on day 34 of admission (figures 4 and 5), that was changed to a definitive ventricular derivation system due to sustained ventricular dilation requiring frequent puncture of the reservoir. The CSF cultures were always negative (table 2).

Meropenem, vancomycin and aminoglycoside were stopped after 46 days of antibiotic therapy and monotherapy with cephotaxime was reinitiated (150 mg/kg/day), for a total of 8 weeks of antibiotic therapy. Galactosaemia screening was negative. This child is now 2 years old, ophthalmological evaluation is normal, has an auditory deficit with auricular prosthesis, global developmental delay, spastic tetraparesia and epilepsy and is followed by a multidisciplinary team.

DISCUSSION

Cephalohaematoma is a subperiosteal haemorrhage that does not cross suture lines, is most often found in the parietal, followed by the occipital region of the skull.[3 4] It is usually a benign and self-limited condition and reabsorption occurs within a few weeks to months after birth, not requiring specific therapy. Focal infection should be suspected if local signs of infection develop over the cephalohaematoma (increased size, overlying cutaneous erythema, tenderness with or without fluctuation), erythematous scalp lesions and osteolytic changes of the underlying skull. It should also be suspected when, during a systemic infection, there is a sudden enlargement of a stationary cephalohaematoma associated with meningitis relapse or sepsis after antibiotic therapy.[3 6 7] Complications of infected cephalohaematomas include cranial osteomyelitis, epidural abscess and subdural empyema.[3–5]

In the case presented, it was not the development of local signs of infection that contributed to the diagnosis of infected cephalohaematoma, despite the report of increased dimensions and consistency, but the persistent neurological deterioration with hydrocephalus in spite of an appropriate antibiotic therapy. Brain imaging showed a cephalohaematoma complicated by a subcutaneous abscess, osteomyelitis and epidural empyema of the skull. The question that persists is what was the cause and what was the consequence: if bacteraemia and meningitis were responsible for the secondary infection of the cephalohaematoma, or if the infected cephalohaematoma was responsible for meningitis and bacteraemia, by contiguous spread.

Children with acute neurological complications have more adverse outcomes. Low birth weight, low Apgar score at 1 min, history of symptoms for longer than 24 h, focal neurological deficits, requirement for mechanical ventilatory support or inotropes, delayed sterilisation of the CSF leukopaenia, thrombocytopaenia, high CSF protein and low CSF glucose concentration, seizures duration longer than 72 h, coma, no response after anticonvulsant therapy, abnormal cerebral ultrasound findings and status epilepticus are important prognostic factors of complications in neonatal meningitis.[8] In this case, there are a number of risk factors related to adverse outcome.

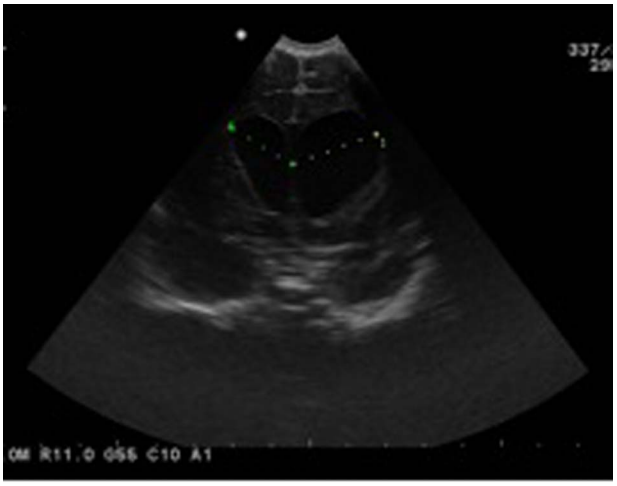

Figure 4 Cranial ultrasound with moderate hydrocephalus.

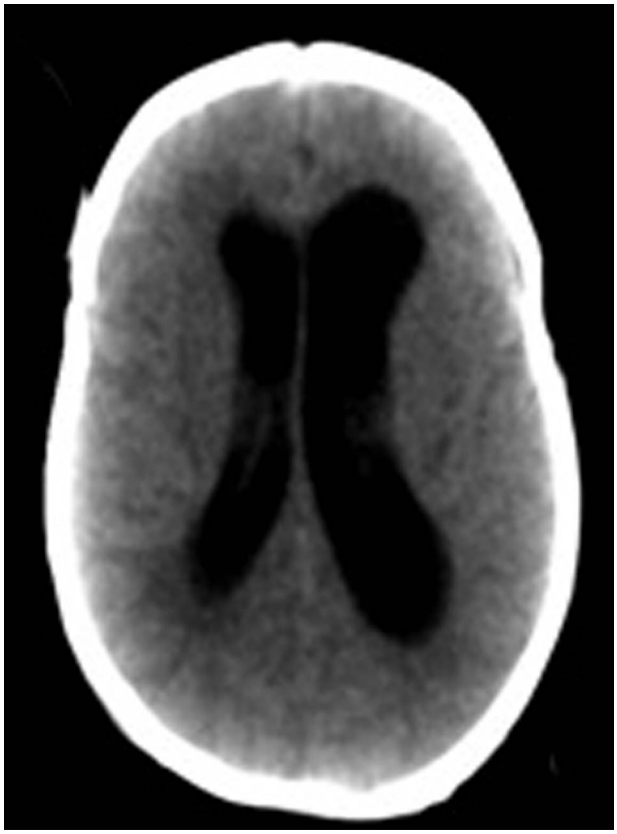

Figure 5 Head CT scan with moderate hydrocephalus.

Table 2 Cerebrospinal fluid analysis during hospitalisation

	D1	D10	D15	D17	D31	D34
Glucose (mmol/L)	<1	–	<1	–	1	1
Proteins (mg/dL)	>900	–	523	–	559	265
Citology	–	–	400 (polymorphonuclear)	–	545 (60%: polymorphonuclear)	54 (75%: mononuclear)
Culture	*Escherichia coli*	Negative	Negative	Negative	Negative	Negative

This case intends to enhance that not all cephalohaematomas have a benign course and that infection of a cephalohaematoma may be associated with severe complications. A delay in the sterilisation of CSF in the context of bacteraemia/meningitis and neurological deterioration in spite of appropriate antibiotic therapy should lead to suspicion of a meningeal or parameningeal infection.

The diagnosis of an infected cephalohaematoma requires an aspirate and culture and the exclusion of systemic infection with blood and CSF cultures, as sepsis or meningitis can be associated.[3–5] In the case presented, the abscess exudate culture of the cephalohaematoma was also positive for *E coli*. Treatment consists of incision and drainage and debridement should be performed if empyema and osteomyelitis are associated. Empiric antibiotic therapy should cover *E coli* and *S aureus*.[3–5] In this case, the duration of treatment was determined by concomitant infection, namely osteomyelitis.

Learning points

- Cephalohaematoma complications should be suspected if it develops inflammatory signs or in the presence of unexplained or recurrent fever.
- A delay in cerebrospinal fluid sterilisation and clinical deterioration in an NB with meningitis may indicate an infectious meningeal or parameningeal focus.
- Sepsis or meningitis may be associated with infected cephalohaematomas.
- Empirical antimicrobial treatment of an infected cephalohaematoma should be directed toward *Escherichia coli* and *Staphylococcus aureus*.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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