CASE REPORT

Bilateral haemorrhagic basal ganglia infarction associated with early-onset group B streptococcus meningitis

Rajesh Gupta,¹ Taiwo Maraiyesa,¹ Brendon Conry²¹Paediatrics Department, Tunbridge Wells Hospital, Pembury, UK²Radiology Department, Tunbridge Wells Hospital, Pembury, UKCorrespondence to
Dr Rajesh Gupta,
r.gupta@nhs.net

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SUMMARY

A 2-day-old infant presented with poor feeding and grunting. Investigations revealed a raised C reactive protein of 164. Full septic screen was done, which subsequently confirmed a diagnosis of group B streptococcus meningitis. Baby was afebrile and haemodynamically stable. There were no obvious neurological manifestations, and a routine cranial ultrasound scan was done, which revealed echogenic changes in the basal ganglia and thalami. MRI brain showed extensive haemorrhagic infarction within the lentiform and caudate nuclei with involvement of both posterior limbs of the internal capsule. This was followed by triventricular hydrocephalus needing shunt procedure. The clinical course was complicated by infantile spasms, which were treated with vigabatrin and steroids and subsequent global developmental delay and cerebral palsy.

This case is of interest because the baby was afebrile and haemodynamically stable at presentation. Despite early treatment, the baby went on to have very unusual and significant damage seen on neuroimaging subsequently manifesting with developmental delay, infantile spasms and cerebral palsy. Haemorrhagic infarction is well-known sequelae of hypoxic-ischaemic injury; however, in this case, there was no history of hypoxic injury, rather a confirmed case of GBS meningitis.

CASE PRESENTATION

A term infant presented on day 2 of life with grunting respiration and poor feeding. Baby was born at home at term by spontaneous vaginal delivery. Apgars were recorded as 9 and 10 at 1 and 5 min of life. There were no antenatal risk factors for sepsis and no record of group B streptococcus colonisation. Mother had a previous stillbirth. Weight was 2180 g (9% loss of birth weight).

On examination, baby was afebrile, irritable with level anterior fontanelle; there was no evidence of grunting but mild chest recessions. Blood sugar was 1.4 mmol/L following which a bottle-feed was taken and repeat check was satisfactory. Blood gas showed a lactate of 3.4 mmol/L. Baby was admitted and commenced on benzyl penicillin and Gentamicin as inflammatory markers were raised with a C reactive protein (CRP) of 164. Following a full septic screen, blood culture and cerebrospinal fluid (CSF) grew group B streptococcus and baby received a total of 2 weeks of intravenous benzyl penicillin and gentamicin.

BACKGROUND

Group B streptococcus (*Streptococcus agalactiae*) is recognised as the most frequent cause of severe early-onset (less than 7 days of age) infection in newborn infants. However, there is still controversy about its prevention. The incidence of early-onset Group B streptococcus (EOGBS) disease in the UK in the absence of systematic screening or widespread intrapartum antibiotic prophylaxis is 0.5/1000 births.¹ Most infants who develop EOGBS disease present with illness soon after birth, and 90% have presented clinically by 24 hours of age, before culture results become available.²

Figure 1 Day 9 cranial ultrasound scan (coronal view) showing echogenic changes in both basal ganglia.

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Figure 2 Day 14 MRI (axial T1) showing high signal haemorrhage in the lentiform and caudate nuclei bilaterally. (MRI scanner: Philips Achieva 3T).

Figure 3 Day 14 MRI (axial FFE gradient echo sequence) showing haemosiderin staining confirming basal ganglia haemorrhage. FFE, Fast Field Echo.

After 2 weeks of antibiotics, a repeat CSF examination still showed high white cell count of $95 \times 10^6/L$ (20% neutrophils, 80% lymphocytes) and a high protein of 1.79 g and glucose of

Figure 4 Day 32 MRI (axial FLAIR) showing new onset hydrocephalus with triventricular dilatation. FLAIR, fluid attenuated inversion recovery.

1.3. A baseline head ultrasound showed echogenic changes in basal ganglia and thalami (figure 1). MRI of the brain showed bilateral haemorrhagic infarction involving both basal ganglia (figures 2 and 3). In view of this, treatment was continued with intravenous cefotaxime for another 10 days totalling 3.5 weeks of treatment.

Post-treatment CSF examination showed a white cell count of $26 \times 10^6/L$ (10% neutrophils, 90% lymphocytes), protein of 1.83 g/L and glucose of 1.7 g/L. The CRP normalised so no further antibiotics were given. Persistent CSF findings were thought to be a natural resolution phase of adequately treated GBS meningitis. A final CSF microscopy performed 6 months after the initial CSF was normal with a white cell count of $1 \times 10^6/L$. Three serial blood culture specimens taken after the initial positive blood culture were all sterile.

Patient had a follow-up cranial ultrasound at 1 month of age, which showed significant ventriculomegaly (head circumference 37 cm with increasing centiles) confirmed on MRI of the brain (figure 4). Patient was then referred to the neurosurgeons who initially performed an endoscopic third ventriculostomy and subsequently required insertion of a right ventriculoperitoneal shunt 10 days later. Postoperative CT shunt series were satisfactory and OFC stable.

Neurodevelopmental follow-up revealed significant developmental delay and signs of cerebral palsy. He presented at 7 months of age with 3-day history of jerks in keeping with spasms. Electroencephalography (EEG) revealed modified hypsarrhythmia, and baby was treated with prednisolone and vigabatrin with good response. Repeat EEG about 3 weeks later showed significant improvement with resolution of hypsarrhythmia and abolition of spasms.

OUTCOME AND FOLLOW-UP

This term neonate with early-onset GBS meningitis went on to have unusual findings on MRI brain and subsequent neurological sequelae including infantile spasms, developmental delay and has a four-limb motor disorder. Patient also required a shunt procedure and has now been weaned off anticonvulsants and is being followed by the multidisciplinary team including neurosurgeons.

DISCUSSION

Imaging is usually performed in neonates with meningitis frequently for complications. Acute complications of neonatal bacterial meningitis include cerebral oedema (vasogenic and cytotoxic), ventriculitis, cerebritis, hydrocephalus, brain abscess, cerebral infarction and subdural effusion or empyema.³

Awareness of the most common pattern of complications produced by each organism responsible for meningitis aids in management in two ways: it allows the radiologist to suggest a possible diagnosis if none has been made, or if the diagnosis is known, it aids the radiologist in recognising early or subtle evidence of complications.⁴

In a 10-year retrospective review of MRI and microbiology data for all neonates carried out by medical imaging at the Royal Children's Hospital in Australia, each organism responsible for neonatal/infant meningitis produced an identifiable pattern of complications on MRI. *Streptococcus* species (group B streptococcus, pneumococcus or subtype not recorded) meningitis was complicated by ischaemic changes more frequently than meningitis due to other organisms. The pattern of extensive (multiple or diffuse) infarcts was typical of streptococcal meningitis and rarely seen with other organisms.

Our case highlights brain MRI changes in group B streptococcus meningitis that are typically associated with hypoxic-ischaemic injury in the newborn. Severe acute asphyxia is associated with lesions in the basal ganglia, thalami, brain stem, hippocampus and the corticospinal tracts around the central fissure.^{5–8}

Large studies of neonatal stroke (both arterial and venous) confirm an association with bacterial meningitis, and in a case series that looked at stroke patterns specific to GBS meningitis, there was recognisable acute cerebral infarction patterns with adverse neurological outcomes. Two distinct patterns of focal infarction have been identified in neonates with group B streptococcal meningitis: a deep perforator arterial stroke to the basal ganglia, thalamus and periventricular white matter and a superficial injury with patchy, focal infarctions of the cortical surface.⁹

In a retrospective cohort study of one centre in France aimed at determining the complications of neonatal meningitis due to group B streptococcus between 1985 and 2010, only 9 of 25 cases could be analysed showing the main acute neuroimaging complication was ischaemic stroke.³

Our patient's initial brain MRI scan was performed on day 14 of life (day 12 of illness), and it showed extensive haemorrhagic infarction within the lentiform nuclei and caudate nuclei. There was involvement of both posterior limbs of the internal capsule. It also showed moderate dilatation of the lateral ventricles with additional abnormal signal intensity with some probable cystic formation in the superior periventricular white matter. At the time this MRI was performed, the

only abnormal neurology noted was possible spasticity in the lower limbs. Baby was feeding orally at the time; however, the MRI changes were of concern, but it was difficult to predict the outcome with certainty as the aetiology of the changes were not hypoxia–ischaemia related.

His neurological condition however rapidly progressed with increasing head circumference at 1 month of age with confirmed hydrocephalus on cranial ultrasound. A brain MRI scan at 1 month of age showed triventricular hydrocephalus necessitating a shunt procedure.

At 6 months of life, he suffered from infantile spasms confirmed on EEG following which he was commenced on vigabatrin and prednisolone and a repeat brain MRI scan at the time showed persisting abnormal appearance as seen in the initial brain scan. His clinical findings of global developmental delay and four-limb motor disorder are consistent with his MRI changes.

Learning points

- ▶ Although ischaemic infarction is well recognised, haemorrhagic infarction involving the basal ganglia and posterior limbs of the internal capsule is not well described secondary to group B streptococcus meningitis.
- ▶ Brain imaging should be routinely performed on patients with GBS meningitis regardless of whether there is any abnormal neurological finding on examination.
- ▶ MRI changes and neurological sequelae in this case also highlight the complication spectrum of group B streptococcus meningitis and should help clinicians in management and prognosticating families of such children.

Contributors RG managed the case, conducted literature search and finalised the manuscript. TM conducted the literature search and drafted the manuscript. BC assisted with the final draft of manuscript and neuroimaging selection and reporting.

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