

Recurrent Group B Streptococcal Septicemia in a Very Low Birth Weight Infant with Infective Endocarditis and Submandibular Cellulitis

Dear Editor,

Maternal Group B streptococcal colonisation can lead to neonatal pneumonia, meningitis or sepsis. Neonatal Group B Streptococcal (GBS) sepsis is common, but infective endocarditis is rare.

Case Report

Our patient, the second of a set of twins, was a Chinese baby girl delivered small for gestational age at 28+3 weeks via emergency caesarian section with a birth weight of 730 g. The pregnancy was complicated by twin-twin transfusion with discrepant growth and maternal hyperthyroidism. A previous sibling had no history of GBS infection but the mother was not screened for GBS via vaginal swab.

She was ventilated at birth for poor respiratory effort and her neonatal course was complicated by pneumothorax, meconium plug syndrome and large patent ductus arteriosus. Pulmonary valve stenosis was found on echocardiography on day 20 of life.

First Episode of GBS Sepsis

At birth, the baby had blood culture as well as ear surface and gastric cultures all growing GBS. Lumbar puncture was performed, and cerebrospinal fluid (CSF) culture did not grow any bacteria. Chest radiography was unremarkable. Intravenous ampicillin was given for 3 days, then subsequently changed to intravenous crystalline penicillin for another 11 days when the blood culture result was known. Gentamicin was administered for 7 days.

Second Episode of GBS Sepsis

The baby was well until day 38 of life when she presented with multiple episodes of apnoea and desaturations, necessitating intubation. A repeat blood culture again grew GBS while CSF and urine cultures were both negative. In view of the recurrence of GBS sepsis, echocardiography was performed showing vegetations at the pulmonary valve consistent with infective endocarditis. Chest radiography was normal. Hepatobiliary, renal and spleen ultrasound were performed showing no foci of infection. Subsequent

blood cultures repeated on days 44, 48 and 50 of life were negative. Repeat echocardiography on day 55 revealed resolution of vegetation. Intravenous ceftriaxone was given for 4 days followed by intravenous crystalline penicillin for 28 days. Amikacin was also administered for 10 days. Another repeat blood culture was performed on the day of completion of all antibiotics and showed no bacterial growth.

Third Episode of GBS Sepsis

However, on day 78 of life, 7 days after the cessation of the second course of antibiotics, right submandibular cellulitis was noted. Blood culture again grew GBS, while urine and CSF cultures were negative. Chest radiography was normal. Echocardiography was repeated and showed no vegetation. Nasal swab culture did not show GBS but grew *Staph aureus* instead. Intravenous crystalline penicillin was administered for 21 days, together with amikacin for 7 days. Intravenous metronidazole was also given empirically for 6 days but was stopped when all culture results was back.

Discussion

This case manifests several unusual presentations for GBS sepsis. Firstly, infective endocarditis of pulmonary valves is aggressive and rare.¹ In adults, it is associated with a 15% to 20% mortality and the need for surgery in at least 30%. Furthermore, to the best of our knowledge, it had never been reported before in preterm or very low birth weight (VLBW) infants. Neonatal infective endocarditis has non-specific symptoms and signs, unlike in older children and adults.²

Secondly, the same patient has submandibular cellulitis, which is highly associated with late-onset GBS infection, predominantly in premature male infants 2 to 10 weeks of age.³

Thirdly, it is unusual for 3 episodes of GBS sepsis to occur in the same child during the same episode of hospitalisation. Recurrent neonatal GBS sepsis is uncommon, and it occurred despite penicillin sensitivity of the GBS [minimum inhibitory concentration (MIC) for penicillin: 0.125 mcg/ml].

Although a case reporting recurrent GBS sepsis in

infancy had identified exposure to maternal breast milk as a possible source,⁴ our patient never received breast milk. Immunodeficiency was also considered unlikely as there was no family history of immunodeficiency, nor were there any abnormal clinical signs, such as dysmorphic facies, abnormal eczematous rashes and intractable hepatosplenomegaly, to suggest this. Full blood count, immunoglobulin M, A, G and E were done, with results normal for her age.

We could only perform serotyping when the specimen was retained when recurrent sepsis occurred, which was during the second episode of GBS sepsis. The same serotype III was found in the blood cultures for the second and third episodes. Serotype III is the commonest strain in early neonatal sepsis,⁵ although it is also the commonest strain in the late onset sepsis.

We suspect that the infection was likely to be incompletely eradicated or may be nosocomially transmitted through skin contact of healthcare providers. If the bacteria was incompletely eradicated, the duration of antibiotic given may not be of sufficient length to eradicate the bacteria. This may be due to various reasons. Certain virulence factors could explain this, for example, translation initiation factor 2 (*infB*) C allele found in restriction digest pattern (RDP) type III-3 strains.⁶

Conclusion

GBS sepsis rarely presents with infective endocarditis, especially of the pulmonary valves. Facial submandibular cellulitis is a rare but a classical presentation for the late onset GBS sepsis. The recurrent nature of the GBS sepsis may be either due to incomplete eradication or nosocomial transmission.

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