A case of postpartum Group B streptococcal meningitis

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Summary: A case of postpartum Group B streptococcal meningitis, a rare complication of an invasive infection by a common maternal commensal bacterium, which demonstrates the need to develop rapid and accurate antepartum and intrapartum screening methods for this organism.

Keywords: Group B streptococcus, post partum, meningitis, screening

INTRODUCTION

Group B streptococcus (GBS, *Streptococcus agalactiae*) is a well-known pathogen of the neonatal period, which is usually managed as a benign commensal organism in women. We present a case of maternal GBS meningitis diagnosed day 1 postpartum, highlighting a rare, but serious, complication of maternal GBS carriage in the perinatal period.

CASE REPORT

A 30-year-old Sudanese immigrant was seen for her sixth pregnancy. She had previously had five normal vaginal births at term. Her antenatal course was uncomplicated. At 36 weeks of pregnancy, a self-collected low vaginal swab was cultured onto layered blood agar and Agar chromID Strepto B agar plates, and also inoculated to a colistin and nalidixic acid broth; the blood agar plate was incubated in a CO₂ incubator for 18–24 hours at approximately 35°C; the Agar chromID Strepto B agar plate and the broth were incubated at approximately 35°C in O₂ conditions for 18–24 hours: no evidence of GBS was detected.

She was admitted at 38⁺⁵ weeks gestation for induction of labour. Following vaginal dinoprostone overnight, artificial rupture of the membranes (ARM) was performed in the morning. Labour was augmented with syntocinon and she progressed to a normal vaginal birth 11 hours and 10 minutes after ARM. No antibiotics were administered during labour, nor was epidural anaesthesia required.

At 27 hours after delivery, she complained of a headache, which soon became severe with photophobia, tachycardia, confusion, nuchal rigidity and positive Brudzinski's and Kernig's signs. There were no localizing neurological signs, but she became combative and uncooperative. Her Glasgow coma score ranged from 8 to 11. Petechial haemorrhages were seen on her limbs. It was then noted that her platelet count had

been $95 \times 10^9/L$ on admission. An urgent computed tomography brain scan showed no abnormality.

Three hours after the onset of headache she became febrile (38.5°C). She was given intravenous benzylpenicillin 1.2 g, gentamicin 320 mg and metronidazole 500 mg, and was then retrieved to a level 3 adult intensive care unit. Her infant remained well with no signs of sepsis.

Lumbar puncture showed $2800 \times 10^6/L$ polymorphonuclear cells, $120 \times 10^6/L$ mononuclear cells, $200 \times 10^6/L$ red blood cells and no bacteria in the cerebrospinal fluid (CSF); protein was 2.26 g/L and glucose <0.6 mmol/L. A diagnosis of acute bacterial meningitis was made and treatment was changed to intravenous ceftriaxone 2 g two times daily and benzylpenicillin 2.4 g four hourly. Blood and CSF cultures subsequently grew *S. agalactiae* (GBS). Intravenous benzylpenicillin 2.4 g four hourly was maintained for 14 days.

Day 1 post-transfer, she had four generalized tonic-clonic seizures, which resolved after phenytoin loading. Following this she recovered well, but continued to have a headache for seven days. She was transferred back to the obstetric hospital on day 7 postnatal. Testing for immunodeficiency disorders showed no abnormality. She subsequently made a complete recovery.

DISCUSSION

S. agalactiae is a Gram-positive coccus, which colonizes the genital and gastrointestinal tracts of up to 35% of pregnant women.¹ The average duration of colonization in women is approximately 13 weeks.²

Antepartum GBS-screening programmes are commonly used in Australian maternity units, with the intention of detecting maternal colonization, so as to reduce neonatal morbidity and mortality with intrapartum therapy to the mother. The aim is not primarily to protect women from perinatal infection, but such treatment has a beneficial side-effect of administering chemoprophylaxis to women most at risk of developing such infections. The fact that our patient had screened negative suggests that either she had returned a false-negative culture, or that she had acquired the organism between screening and the onset of

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meningitis. It is possible to increase the sensitivity of the screening by the addition of a rectal as well as a vaginal swab.³ The development of polymerase chain reaction (PCR) and optical immunoassay tests for GBS may result in a test being able to be performed at the time of onset of labour, thus providing a more accurate reflection of true colonization, and enable targeted intrapartum antibiotic prophylaxis.⁴ PCR assays have been shown to increase the probability of GBS colonization from 21% to 65%,⁴ but their use requires 24-hour clinical microbiology services in order to be effective. Even with a PCR assay, intrapartum screening may be too late to obtain the full effect of antibiotic prophylaxis for up to 50% of women.⁴

The incidence of invasive GBS disease in pregnant women was reported as 12:100,000 live births in the USA.⁵ Half of these infections involved the upper genital tract, placenta or amniotic membranes. The rest involved bacteraemia without focus, pneumonia and puerperal sepsis. In all, 79% occurred in the absence of additional underlying medical conditions. In this study, invasive GBS disease in non-pregnant adults steadily increased by 32% between 1999 and 2005, but despite the introduction of guidelines for chemoprophylaxis against neonatal disease during the same time period, the rate of invasive disease in pregnant women did not decrease in line with early-onset neonatal disease.⁵ This raises the question of whether either the prevalence and/or the virulence of GBS are increasing with time.

Postpartum GBS meningitis is extremely rare: we believe that this is the first case reported in Australia. A literature review revealed 10 previous cases after vaginal birth, none after caesarean section and two following elective termination of pregnancy.^{6,7} None was associated with epidural anaesthesia.⁷ In a review reporting complications of GBS meningitis in adults from a general medical population,⁸ the overall case fatality rate was 34.4%, and of the survivors 7% had neurological sequelae, always deafness. Of the reported cases in pregnant women,^{6–8} two died, a case fatality rate of 20%. Deafness was also the only ongoing sequela among survivors. Our patient suffered a neurological complication of seizures, but she has not had a recurrence since day 2 of her illness. She has not complained of hearing loss.

In conclusion, we report an extremely rare complication of GBS colonization in the postpartum period. The literature raises the question of whether GBS is increasing in virulence among adults, given that the incidence of invasive disease has been steadily rising in the USA in this population. This

highlights the importance of developing and using accurate antepartum and, even better, intrapartum screening methods in order to reduce the burden of disease among both neonates and mothers.

DECLARATIONS

Competing interests: None.

Guarantor: WMH.

Contributorship: KG, AM and WMH all cared for the patient. KG researched the literature and wrote the first draft of the manuscript. All authors reviewed and edited the manuscript and approved the final version of the manuscript.

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