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Letter to the Editor

Maternal vaginal colonisation with *Neisseria meningitidis* serogroup B and late onset neonatal invasive meningococcal disease

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Further to a recent report of an outbreak of *Neisseria meningitidis* serogroup Y serotype (ST-) 1466 urogenital infections in Australia,[1] we report a case of late-onset neonatal invasive meningococcal disease (IMD) secondary to maternal vaginal colonisation. The risk associated with vaginal colonisation of *N. meningitidis* for vertical transmission is unknown, but is of heightened concern given the increased detection of urogenital infections and the clinical and public health implications.[1] Isolated cases of neonatal IMD associated with either maternal IMD or colonisation predating the recent outbreak have been reported previously.[2,3]

A 21-year-old woman (G1P0, 38^{+3} weeks gestation) presented with spontaneous rupture of membranes (SROM), afebrile, with clear liquor. A vaginal swab was collected, but results were pending at the time of delivery. She did not meet clinical risk criteria for Group B *Streptococcus* (GBS) prophylaxis,[4] and did not receive antibiotics in labour. She proceeded to emergency caesarean section for foetal distress five hours after SROM. The 3,195 g neonate had APGAR scores of 5 and 6 and was treated empirically with intravenous ampicillin and gentamicin for 48 hours. The neonate had no source of sepsis identified, including a set of negative blood cultures collected prior to antibiotics. They were discharged home well on day four of life.

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On day nine, the neonate was readmitted with fevers. At this time, it was noted that the mother's vaginal swab collected at delivery grew *N. meningitidis* and GBS. Neonatal cerebrospinal fluid grew *N. meningitidis* serogroup B; blood cultures were negative. The neonate received 14 days of antibiotics and was discharged home without evident disability.

Histological examination of the placenta showed evidence of intraamniotic infection with chorioamnionitis, umbilical panvasculitis and funisitis. *N. meningitidis* DNA was detected in placental tissue. Whole genome sequencing of neonatal and maternal isolates found both were Men-B ST-2506 (clonal complex 32) and were identical (1 SNP difference on split kmer analysis).[5]

Late-onset neonatal IMD associated with maternal vaginal *N. meningitidis* has been documented.[2,3] This mother presented with SROM, had histological evidence of ascending intrauterine infection and *N. meningitidis* DNA detected in the placenta. Delayed onset of neonatal sepsis was likely attributable to partial treatment. This case further highlights the need to establish clear risk management strategies for vaginal *N. meningitidis* colonisation in pregnancy for mothers, neonates and their contacts.

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