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CASE REPORT

Morganella morganii: a rare cause of early onset neonatal sepsis and meningitis

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Introduction

Early onset neonatal sepsis is defined by the National Institute for Health and Care Excellence (2012) guidelines as infection occurring within 72 hours of birth. In the UK, data available from the neonatal infection surveillance network (NeonIN) from 2005–14 show that the incidence of early onset neonatal sepsis was 0.7/1000 live births, which accounted for 5.6 neonatal admissions per 1000 live births (Cailes et al, 2018). Group B streptococcus (43%) and *Escherichia coli* (18%) are the most commonly isolated pathogens (Cailes et al, 2018). According to the UK-based NeonIN study, *Morganella morganii* is a rare cause of early onset neonatal sepsis accounting for 1/124 (0.8%) cases (Vergnano et al, 2011). This article describes a case of early onset neonatal sepsis in a pre-term infant caused by *M. morganii* that most likely occurred via vertical transmission.

Case report

A male infant, weighing 1.6 kg, was delivered by emergency caesarean section as a result of fetal distress at 32^{+5} weeks. A single dose of betamethasone was administered to the mother before delivery to expedite fetal lung maturation. The pregnancy had been complicated by pregnancy-induced hypertension.

The infant was born in good condition with no resuscitation required. Owing to the pre-term onset of labour, intravenous benzylpenicillin and gentamicin were started empirically after collecting blood culture. The initial blood glucose level was 0.8 mmol/litre and, following a 10% dextrose bolus, the hypoglycaemia resolved. The initial C-reactive protein level was 44 mg/litre, which peaked at 52 mg/litre on day 2. The white cell count was 18.0×10^9/litre, neutrophil count was 0.9×10^9/litre and platelet count was 130×10^9/litre. Gram negative rods were grown in blood culture on day 1 and *Morganella morganii* was identified on subculture. In view of bacteraemia and deranged inflammatory markers, lumbar puncture was performed at 24 hours. CSF microscopy showed a white cell count of 942×10^6/litre (90% polymorphs) and red cell count was 2240×10^6/litre. CSF glucose was 1.9 mmol/litre (concurrent blood glucose: 3.0 mmol/litre) and protein was 2.59 g/litre. CSF culture also grew *M. morganii*.

As expected, the isolate showed reduced susceptibility to amoxicillin and co-amoxiclav as a result of AmpC beta-lactamase production but no extended-spectrum beta-lactamase production. As AmpC-producing coliforms have the potential to become resistant to cephalosporins through induction or selection of derepressed mutants, antibiotic therapy was adjusted to meropenem for 3 weeks and gentamicin was discontinued after 7 days. Cranial ultrasound scans on day 9 and day 25 both showed normal intracranial appearances and the ventricles were not enlarged. A hearing test was reported as normal.

He remained stable and was discharged home at a corrected gestational age of 37^{+3} weeks. The infant had a normal examination at discharge. At clinic follow up 6 months later, the neutropaenia had resolved, and the infant remained well and was achieving age-appropriate milestones.

Discussion

Early onset neonatal sepsis caused by *M. morganii* is rare. Over the last two decades, only 11 cases have been reported and, of these, two had meningitis (Rowen and Lopez, 1998; Maheshwari et al, 2001; Casanova-Román et al, 2002; Boussemart et al, 2004; Dutta and Narang, 2004; Sinha et al, 2006; Ovalle et al, 2009; Chang et al, 2011; Vergnano et al, 2011; Tratsaert et al, 2019).

M. morganii is a Gram-negative coliform (family *Enterobacteriaceae*) that is found in the digestive system and the environment (Tratsaert et al, 2019). Although early onset neonatal sepsis with *M. morganii* is extremely rare, it may present asymptomatically or

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Learning points

- *Morganella morganii* is a rare but significant cause of neonatal sepsis, which can lead to high mortality and morbidity.
- Early recognition and treatment remains the best practice for improving outcomes.
- Early liaison with a microbiologist or infection specialist is recommended to ensure appropriate antibiotic treatment.

with non-specific clinical signs, with prematurity being a significant risk factor (nine out of 11 cases). The most common findings were respiratory distress, tachypnoea and perinatal depression (Dutta and Narang, 2004; Sinha et al, 2006; Milligan and Barenkamp, 2013; Tratsaert et al, 2019). The case fatality rate in the literature for *M. morganii* was 27% (three out of 11 cases). Maternal risk factors identified included chorioamnionitis, sepsis and urinary tract infection, with *M. morganii* grown from blood culture (n=1), urine (n=1), placenta (n=1) and vagina (n=1) (Rowen and Lopez, 1998; Maheshwari et al, 2001; Casanova-Román et al, 2002; Boussemart et al, 2004; Milligan and Barenkamp, 2013).

All isolates showed reduced susceptibility (resistance) to ampicillin or amoxicillin. Furthermore, many isolates showed resistance to first and second generation cephalosporins (Rowen and Lopez, 1998; Casanova-Román et al, 2002; Sinha et al, 2006; Chang et al, 2011). Depending on susceptibilities, treatment consisted of either a cephalosporin or carbapenem usually combined with an aminoglycoside. However, given that *M. morganii* can produce chromosomal beta-lactamase (AmpC) through either induction or via derepression of mutants, treatment of meningitis caused by this and similar organisms may fail with standard empirical therapy for meningitis (e.g. cephalosporins) as a result of intrinsic mechanisms of resistance. In this setting meropenem is recommended as the antibiotic of choice.

Conclusions

This case reinforces the importance of remaining aware of this rare pathogen, which can cause early-onset sepsis and/or meningitis in neonates. Early onset *M. morganii* infection is rare; it is not susceptible to commonly used beta-lactam antibiotics, although is usually susceptible to aminoglycosides. Early suspicion, diagnosis, pathogen detection and appropriate antimicrobial treatment based on local epidemiology and confirmation of sensitivities for neonates remains the best therapeutic approach.

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