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Early onset meningitis due to *Morganella morganii*: a case report

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Summary

We describe a case of a term newborn who developed *Morganella morganii* meningitis. *M. morganii* is a rare perinatal pathogen causing neonatal sepsis and chorioamnionitis. Prompt management of chorioamnionitis and prevention of early neonatal infection are imperative to reduce morbidity and mortality in both newborn and mother.

Keywords: Morganella morganii; Neonatal sepsis; Meningitis

Introduction

Reports of central nervous system (CNS) infections due to *Morganella morganii* are scarce with only some cases reported in the literature [1,2,12,15,24,33,35]. *M. morganii* in newborns is an opportunistic pathogen which can cause neonatal infections and sepsis.

The presence of a chromosomally encoded inducible *ampC* β -lactamase challenges the clinician in the selection of a targeted treatment and repeated microbiological cultures are mandatory in patient's follow-up [3,10,32]. Several reports describe severe neurologic sequelae or fatal outcome in neonates suffering from *M. morganii* meningitis or bacteremia due to delay of appropriate treatment [8,23].

We discuss a case of a term newborn with early onset meningitis due to *M. morganii* successfully treated with a third-generation cephalosporin associated with an aminoglycoside.

Case report

After a rapid labor, a term female baby was born to a 32-year-old mother by spontaneous vaginal delivery. Rupture of membranes occurred 8 hours before delivery with malodorous amniotic fluid. The mother was afebrile during both labor and delivery and C-reactive protein (CRP) was negative. The pregnancy had been uneventful and group B *Streptococcus* (GBS) vaginal screening was negative. The mother didn't receive any antibiotics during pregnancy neither before delivery.

Physical examination at birth showed transient respiratory distress but 3 hours later the patient presented with a severe apnea requiring her admission to the neonatal intensive care unit. CRP was slightly elevated at 23 mg/L. Chest X-ray was normal. Empirical antibiotherapy was initiated including ampicillin 100mg/kg/day and amikacin 15 mg/kg/day. After 26 hours, the newborn presented irritability and a lumbar puncture was performed. Cerebral spinal fluid microscopy (CSF) showed 1760 white blood cells/mm^3 with neutrophilic pleiocytosis, decreased glycorrachia (34 mg/dL) and increased protein level (1.90 g/L).

Ampicillin was switched to cefotaxime (25 mg/kg every 6 hours for 21 days). A blood test was repeated 18 hours later and showed thrombocytopenia and increased CRP at 146 mg/L. CSF culture grew for *M. morganii* Manual antimicrobial susceptibility testing with disks performed according to the CLSI 2017 guidelines showed susceptibility to cefotaxime, ceftazidime, cefepime, piperacillin/tazobactam, meropenem, aztreonam, amikacin, ciprofloxacin and trimethoprim/sulfamethoxazole.

Susceptibility to cefotaxime was alternatively confirmed with a minimal inhibition concentration (MIC) value of 0,094µg/ml. Three blood cultures remained sterile. Urine culture was also sterile. Surface swabs from the ear grew for the same strain. No placenta culture was performed.

Her clinical condition improved after 24 hours of treatment and CRP declined steadily. Repeated CSF sample performed 9 days after the initiation of the antibiotics was sterile and subsequent brain imaging (RMN and ultrasound) was normal.

Discussion

M. morganii is a gram-negative facultative anaerobe rod commonly found in the environment and in the intestinal tract of humans (as part of the substitution flora), mammals, and reptiles [25,35]. It is an uncommon cause of community-acquired infection and is most often found in adult nosocomial infections such as urinary tract infections [32], hepatobiliary tract and surgical wound infections [21,23,24,27]. Necrotizing fasciitis, pericarditis [31], septic arthritis and endophthalmitis [13] have been rarely reported.

Early onset *M. morganii* neonatal sepsis is rare but may cause serious invasive disease. The clinical signs are non-specific, but the most common symptoms are respiratory distress and tachypnea. There is no sex predilection [8] but this infection mostly affects premature babies [28, 29, 30]. The mortality and morbidity rates are even higher in lower gestational and smaller birth weight preterm infants.

The most common antenatal risk is maternal chorioamnionitis [6,7,15,18,22] but also the use of antibiotics before delivery [29]. It is suggested that the use of intrapartum ampicillin prophylaxis for group β-streptococcal infection is a risk factor for the natural selection of *M. morganii* strains [32]. For this reason, *M. morganii* should be considered, despite its rarity, as a potential pathogen in sepsis caused by vertical transmission [5,32].

M. morganii has an inducible *ampC* β-lactamase which initially confers resistance to certain narrow spectrum β-lactam antibiotics (e.g. penicillins, first- and second-generation cephalosporins) [20,23,25,27,33]. Moreover, the over-production of the enzyme can ultimately lead to third-generation cephalosporin resistance explained by a reversible inducible expression or a de-repressed constitutive expression of the *ampC* gene. Various

β-lactams including third-generation cephalosporins are considered as inducers of this *ampC* expression mechanism. However, evidence is still lacking on the frequency of mutation towards constitutive resistance [3,15,25,33]. To prevent this phenomenon, confirmed *M. morganii* sepsis should be treated with a combination of third-generation cephalosporin and aminoglycoside. [19,22,29].

Conclusion

M. morganii is a rare cause of neonatal sepsis and meningitis. Knowing its extended resistances profile, appropriate antibiotics should be initiated as soon after bacteriological identification. Genome sequencing of *M. morganii* could further provide important information concerning virulence and determinants of fitness.

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