Early onset meningitis due to Morganella morganii: a case report
Valérie Tratsaert1,2,*
Verroken Alexia3
Diane Stroobant2,4
Aude Helsmoortel1
Anne Charon1
Dimitri Van der Linden4
1Neonatology, GHDC Charleroi, Belgium
2Pediatric Infectious Diseases, General Pediatrics, Pediatric Department, Cliniques universitaires Saint-Luc, Brussels, Belgium
3Microbiology Department, Cliniques universitaires Saint-Luc, Brussels, Belgium
4Pediatrics, Pediatric Department, GHDC Charleroi, Belgium
*Corresponding author: Valérie Tratsaert, Pediatric Infectious Diseases, General Pediatrics, Pediatric Department, Cliniques universitaires Saint-Luc, Brussels, Belgium; E-mail: valerie.tratsaert@uclouvain.be
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Summary
We describe a case of a term newborn that developed meningitis to Morganella morganii. He was vaginal delivered after rapid labor. Respiratory distress occurred 3 hours after birth. Blood culture and lumbar puncture were positive for M. morganii. It is a rare perinatal pathogen, causing severe disease in association with maternal chorioamnionitis and premature rupture of the membranes. Recognition of chorioamnionitis infection and the prevention of early neonatal infection are very important to reduce the morbidity and mortality for the newborn and the mother.

Keywords: Morganella morganii; Neonatal sepsis; Meningitis
**Introduction**

Central nervous system (CNS) infections due to *M. morganii* are scarce with only some cases reported in the literature [1,2,12,15,24,33,35] *M. morganii* seems to be 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 of each *M. morganii* infection and repeated microbiological cultures are mandatory in patient’s follow-up [3,10,32]. Several articles describe severe neurologic sequelae or fatal outcome in neonates with *M. morganii* meningitis or sepsis due to treatment failure [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 and an aminoglycoside.

**Case report**

A term female infant of 38 weeks’ gestation, weighing 3170 g was born from a 32-year-old multigravida, G2P2, by spontaneous vaginal delivery after rapid labor of 2 hours. Rupture of membranes occurred 8 hours before delivery with malodorous amniotic fluid. The mother was afebrile during both labor and delivery and the C-reactive protein (CRP) was negative. The pregnancy had been uneventful and vaginal culture performed at 34 weeks of gestation to screen carriage of group B Streptococcus (GBS) was negative. The mother didn’t receive antibiotics during pregnancy and before delivery.

The newborn’s Apgar scores were 8 and 9 at 1 and 5 min, respectively. Physical examination at birth showed respiratory distress with a favorable and rapid resolution but 3 hours later the patient presented with a severe apnea requiring her admission in the NICU. CRP was slightly increased (23 mg/L) chest X-ray was normal. Empirical antibiotic therapy was initiated with ampicillin 100mg/kg/day and amikacin 15 mg/kg/day. After 26 hours, the newborn presented meningitis signs and a lumbar puncture was performed with microscopy showing a white blood cell count of 1760/mm³ and a red blood cell count of 5520/mm³.

Ampicillin was switched to cefotaxime (25 mg/kg every 6 hours for 21 days) still in combination with amikacin (15 mg/kg every 24 hours for 2 days). The blood test was repeated and showed thrombocytopenia and increased CRP at 146 mg/L.

The cerebrospinal fluid (CSF) culture grew for *M. morganii*. Manual antimicrobial susceptibility testing with disks performed according to the CLSI 2017 guidelines showed susceptibility to cefotaxime, cefazidime, cefepime, piperacillin/tazobactam, meropenemaztreonam, amikacin, ciprofloxacin, and trimethoprim/sulfamethoxazole.

Susceptibility to cefotaxime was alternatively confirmed with a minimal inhibition concentration value of 0,094 µg/ml. 3 blood cultures remained sterile. Urine culture was sterile. Surface swabs from the ear grew with the same strain. There was no placenta culture.

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

**Discussion**

*M. morganii* is a gram-negative facultatively anaerobic 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 surgery 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 rate 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. For this reason, *M. morganii* should be considered, despite its previous scarcity, as a pathogen that may be increasingly implicated in sepsis caused by vertical transmission [5,32]. It is recommended to administer antibiotics if chorioamnionitis is suspected because it decreases the rate of neonatal bacteremia, pneumonia, and sepsis[13].

*M. morganii* is naturally resistant to colistin. It 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]. However, 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, the evidence is still lacking on the frequency of mutation towards constitutive resistance [3,15,25,33]. Aiming to prevent this phenomenon, and sepsis with *M. morganii* should
be treated with a combination of third-generation cephalosporin in combination with an aminoglycoside [19,22,29].

**Conclusion**

*M. morganii* has been recognized as an increasingly important pathogen. The disease spectrum associated with *M. morganii* is broad and includes neonatal sepsis and meningitis. Knowing their extended resistances profile, the appropriate antibiotics should be initiated as soon as identification is known. Considering the increased frequency of *M. morganii* infections, enhanced research on this pathogen is important. Genome sequence of *M. morganii* could further provide important information concerning virulence and determinants of fitness. Recognition of chorioamnionitis infection and the prevention of early neonatal infection are very important to reduce the morbidity and mortality for the newborn and the mother.

**References**


