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CASE REPORTS

Early-onset Neonatal Sepsis Caused by Vertical Transmission of Morganella morganii

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The onset of Morganella morganii infection within the first 72 h after birth has only been reported on 1 occasion. The authors describe a second case in which M. morganii was cultured from both the neonate's blood and the mother's lochia. The infant was successfully treated with cefotaxime and gentamicin.

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INTRODUCTION

Morganella morganii is a Gram-negative bacillus most often isolated as a nosocomial pathogen. It is a rare cause of infection in the pediatric population (1). Early-onset neonatal sepsis occurs as a multisystemic illness in the first 3 d of life. Bacteria responsible for early-onset disease include group B Streptococci, Escherichia coli and Listeria monocytogenes. We report a case of early-onset neonatal sepsis caused by M. morganii. As far as we know, this is only the second such case reported in the English language literature.

CASE REPORT

A pre-term male infant weighing 2,315 g was delivered to a 25-y-old primigravida by spontaneous vaginal delivery after a 14-h labor. The woman was afebrile during both labor and delivery. Membranes were artificially ruptured 6 h before delivery, revealing clear amniotic fluid. The pregnancy had been uneventful and cultures performed in search of group B Streptococci were negative.

The infant's Apgar scores were 9 and 10 at 1 and 5 min, respectively. Physical examination was normal at birth and his gestational age, estimated by means of the Ballard score, was 35 weeks. By 28 h of age, grunting, lethargy and poor appetite were noted. Body temperature was 38.1°C, respiratory rate 58 breaths/min and heart rate 150 beats/min. Arterial blood gases revealed metabolic acidosis. The white blood cell (WBC) count was 5.65×10^3/ml, with 29% neutrophils, 37% band forms, 12% lymphocytes and 22% monocytes. The CRP level was 22 mg/l. Lumbar puncture yielded normal cerebrospinal fluid (CSF): 8 WBCs/ml, normal glucose and protein levels and no organisms on Gram stain. Red blood cell count, platelet count, serum electrolyte concentrations, urinalysis and chest radiography were normal. Culture of CSF was negative. The blood culture grew M. morganii, which was identified using the Vitek System (BioMérieux Vitek, Hazelwood, MO) and confirmed with the API 20E system (BioMérieux). The organism was resistant to ampicillin, amoxicillin, amoxicillin–clavulanic acid and cefazolin but susceptible to cefotaxime and gentamicin. Empirical antibiotic therapy was started with i.v. cefotaxime (50 mg/kg every 12 h) and ampicillin (50 mg/kg every 12 h). Once the culture results were known, ampicillin was stopped and gentamicin (2.5 mg/kg every 12 h) was added. Blood culture performed after 24 h of antibiotic therapy was negative and the infant's clinical status improved during this period. The total duration of therapy was 10 d.

The mother became febrile 4 h postpartum and was treated empirically with clindamycin and gentamicin. She remained afebrile for the first 48 h following the start of treatment. Urine, blood and rectal cultures were sterile but M. morganii was isolated from a cervical swab culture. The culture was pure and the amount of growth on agar was reported semiquantitatively as moderate (2 +). The organism was susceptible to cefotaxime and gentamicin but resistant to ampicillin, amoxicillin, amoxicillin–clavulanic acid and cefazolin.

DISCUSSION

M. morganii is a cause of nosocomial infections in adults and a rare cause of bacteremia. These infections are related to postoperative wound infections and infections of the urinary tract (2). As M. morganii infection typically does not cause bacteremia (2), neonatal bacteremia is even more rare. Rowen and Lopez (3) reported a case of early-onset sepsis; the organism isolated from the blood of the neonate was resistant to ampicillin and cefazolin and susceptible to cefotaxime and gentamicin. However, a positive culture from the mother's urine at 3 d postpartum was also resistant to gentamicin. In our case, the isolates from the neonate's blood and cervical culture demonstrated the same resistances to antibiotics. These two isolates had the same biotype and antibiotic sensitivities; therefore it is probable that the infant acquired the pathogen via exposure to the infected maternal tissues. Nevertheless we were not able to perform a genetic analysis to prove that the infant and mother's isolates were identical.

The epidemiologic risk factors for M. morganii infection are advanced age, the presence of serious underlying diseases, hospitalization, recent surgery and concurrent antibiotic use (2). In the case described by Rowen and Lopez (3),

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the mother had been receiving monthly injections of benzathine penicillin for prophylaxis of rheumatic fever, together with a course of amoxicillin 1 month prepartum for pharyngitis and ampicillin with gentamicin on the day of the birth. In our case, the mother had not received any antibiotics during either the pregnancy or the birth. In a case of late neonatal sepsis reported by Salen and Eppes (1) and a case of neonatal cerebral abscess reported by Verboon-Marciolek et al. (4), no antibiotics had been administered and there were no other risk factors present. Antibiotics had also not been administered to the mother in a case of chorioamnionitis reported by Carmona et al. (5), although the infant had not been affected.

The widespread use of ampicillin for the prophylaxis of neonatal sepsis caused by group B Streptococci could be responsible for the increased incidence of early-onset neonatal sepsis caused by non-group B streptococcal organisms resistant to ampicillin (6). 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.

REFERENCES

1. Salen PN, Eppes S. Morganella morganii: a newly reported, rare cause of neonatal sepsis. Acad Emerg Med 1997; 4: 711–4.
2. Mylotte JM, McDermott C. Morganella morganii: epidemiology of bacteremic disease. Infect Control 1984; 5: 131–7.
3. Rowen JL, Lopez SM. Morganella morganii early onset sepsis. Pediatr Infect Dis J 1998; 17: 1176–7.
4. Verboon-Marciolek M, Vandertop WP, Peters ACB, Roord JJ, Geelen SPM. Neonatal brain abscess caused by Morganella morganii [Letter]. Clin Infect Dis 1995; 20: 271.
5. Carmona F, Fábregues F, Alvarez R, Vila J, Cararach V. A rare case of chorioamnionitis by Morganella morganii complicated by septicemia and adult respiratory distress syndrome. Eur J Obstet Gynecol Reprod Biol 1992; 45: 67–70.
6. Towers CV, Carr MH, Padilla G, Asrat T. Potential consequences of widespread antepartal use of ampicillin. Am J Obstet Gynecol 1998; 179: 879–83.

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Severe Thrombocytopenic Purpura Due to Brucellosis

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A case of severe thrombocytopenic purpura as the sole manifestation of brucellosis in an 11-y-old boy is presented. Clinical examination was negative and laboratory tests revealed anemia, leukopenia and severe thrombocytopenia. The initial diagnosis was idiopathic thrombocytopenic purpura and intravenous gamma globulin was given. A prompt hematological response was observed. However, on the fifth day after admission, blood culture yielded Brucella which could not be serotyped. The boy was subsequently treated with intravenous gentamicin, oral doxycycline and rifampicin and was discharged in good health. Brucellosis has occasionally been associated with mild hematological abnormalities such as anemia and leukopenia. Thrombocytopenia is rare and only in very rare cases it is severe enough to cause bleeding. Prompt recognition of this complication of brucellosis and aggressive therapy are essential, especially if there is a family history of brucellosis or if there is suspicion of exposure to infected food products.

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INTRODUCTION

Very few data on the frequency and diversity of hematological abnormalities occurring during brucellosis in children have been reported. The commonest are anemia, leukopenia and mild thrombocytopenia (1). Severe thrombocytopenic purpura as the first manifestation of the disease is extremely rare (1–4).

CASE REPORT

The patient was an 11-y-old boy, the third of 4 siblings from a rural area of Northwestern Greece. He was admitted to the Pediatric Department of the General Hospital of Ioannina with a petechial rash on the trunk and neck, as well as ecchymoses on the upper and lower extremities, which had appeared 3 d previously. He also had a 1-d history of mild fever (38 °C) before the onset of the rash. On admission, the spleen, liver and lymph nodes (cervical, axillary and inguinal) were not palpable.

Laboratory tests revealed leukocytes 5.99×10^9/l (26% neutrophils), mild anemia (hematocrit level 33.2%) and severe thrombocytopenia (8×10^9/l platelets). Fibrinogen levels, prothrombin time and activated partial thromboplastin time were normal. Antibodies to platelets were negative. The initial diagnosis was idiopathic thrombocytopenic purpura and intravenous gamma globulin was given, with a prompt hematological response. On the