Congenital sepsis caused by *Eikenella corrodens*

Brittany L. Hu¹, Julie-Ann M. Crewalk², David P. Ascher³

¹School of Medicine, University of Virginia, Charlottesville, USA
²Pediatric Infectious Disease Group, Inova Health System Hospital for Children, Fairfax, USA
³Department of Pediatrics, Inova Health System Hospital for Children, Fairfax, USA

Email: jcrewalk@kidspid.com

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ABSTRACT

*Eikenella corrodens* is a part of normal human oral flora and a rare cause of intrauterine and neonatal infections. We describe a case of congenital *E. corrodens* sepsis with positive blood cultures at birth in the setting of low maternal risk factors for infection. Our case is one of two reported cases of congenital *E. corrodens* sepsis resulting in newborn survival.

Keywords: Neonatal Sepsis; Intrauterine Infection; Chorioamnionitis

1. INTRODUCTION

*Eikenella corrodens* is a slow-growing, facultative anaerobic gram-negative bacillus that is known to cause serious human illnesses. Known cases include head and neck infections, meningitis, endocarditis, intra-abdominal infections, skin infections, and infection after human bite wounds [1]. The bacterium is a part of human oral, upper respiratory and gastrointestinal flora, and as such, it has classically been associated with exposure to human oral secretions. It is susceptible to a wide range of antibiotics, such as ampicillin, gentamicin, penicillin G, trimethoprim-sulfamethoxazole, amoxicillin-clavulanic acid, cefotaxime, and ceftriaxone [2]. Interestingly, it is typically resistant to metronidazole and clindamycin, which are usually effective in treating infections caused by human oropharyngeal flora. Co-infection is also observed, especially with streptococci species [1]. While *E. corrodens* may cause a variety of infections, obstetric and neonatal infections are much less common.

Materno-fetal infections caused by *E. corrodens* have been associated with preterm labor and neonatal morbidity and mortality. Reports of such cases have typically involved chorioamnionitis [3]. Different routes of infection have been proposed, including hematogenous spread from the oral cavity or from bite wounds, as well as ascending vaginal infection following oral-genital sex during pregnancy [4]. While more recent studies have shown an increase in gram-negative bacteria as a cause of neonatal infections, *E. corrodens* remains a rare etiology of neonatal sepsis [5]. We searched the literature for reported cases of congenital *Eikenella* sepsis with positive blood cultures at birth. We describe the fourth case.

2. CASE

A 30 year-old woman, gravida II para I, was admitted at 33 weeks and 2 days gestation for preterm labor. On the day of delivery, the mother was afebrile, although she had an elevated white blood cell count of 19,900/mm³. The mother was otherwise healthy and had received prenatal care throughout her pregnancy. Prenatal testing was negative for human immunodeficiency virus, hepatitis B virus, rubella, and syphilis. The mother denied periodontal disease or any history of a human bite. For a living, she trained German Shepherds, but denied any bites or wounds from the dogs. She also denied any oral-genital contact, dental work, or drug use during her pregnancy.

Rupture of membranes occurred at delivery, and a male infant was born vaginally. The delivery was complicated by cord prolapse. The amniotic fluid and placenta were not grossly inflammatory, however maternal blood and placental cultures were not sent, and the placenta was not examined histologically. Other than the increased white blood cell count, the mother had no signs of infection and was not treated with antibiotics.

After delivery, the infant showed signs of respiratory distress and was given a dose of surfactant. The infant weighed 1395 grams, with Apgar scores of 5, 8, and 9 at 1, 5, and 10 minutes respectively. His heart rate at birth was 80 beats per minute, which increased to 120 beats per minute after respiratory support via bag valve mask. His temperature was 36.7°C rectal and his blood pressure was 61/31 mmHg. He was subsequently placed on continuous positive airway pressure and intravenous (IV) fluids. The infant was initially started on IV ampicillin and gentamicin. When preliminary culture results showed gram-negative rods, cefotaxime was added on day of life (DOL) 2 for double gram-negative coverage. A chest x-ray demonstrated diffuse haziness but no focal infil-
trate. On DOL 1, his white blood cell count was 2990/mm³. His differential showed 12% neutrophils, 1% bands, 47% lymphocytes, 31% monocytes, and 2% eosinophils. He was neutropenic with an absolute neutrophil count (ANC) of 388. His lumbar puncture was traumatic with 3 white blood cells/mm³, 6100 red blood cells/mm³, glucose 36 mg/dl, and protein 210 mg/dl. The cerebrospinal fluid culture was negative.

Blood cultures obtained at 30 minutes of life eventually grew *E. corrodens* and coryneform gram-positive rods. *E. corrodens* was reported as susceptible to trimethoprim-sulfamethoxazole and ceftriaxone. Repeat blood cultures were negative. The infant’s white blood cell count increased over the next few days, peaking at 28,000/mm³ with an ANC of 11,700 on DOL 5. He maintained a normal core body temperature during the duration of his hospital stay.

The infant improved clinically on antimicrobial therapy. By DOL 4, he was weaned from continuous positive airway pressure to high flow nasal cannula without complications. By DOL 13 he was weaned to room air. He received nasogastric tube feeds during the first four weeks of life due to oxygen desaturation while breast-feeding. He showed no evidence of brain abscess or other lesions on head ultrasound. Repeated surveillance cultures of his nasopharynx, skin, and rectum were negative for Methicillin Resistant *Staphylococcus aureus* (MRSA), Vancomycin Resistant *Enterococcus* (VRE), and *Serratia* and *Pseudomonal* species. Cefotaxime was discontinued on DOL 5. He otherwise completed a 10-day course of gentamicin and 14-day course of ampicillin. As the positive blood cultures were obtained at 30 minutes of life, it is most likely that the infection was acquired in utero.

### Table 1. Reported cases of congenital sepsis caused by *Eikenella corrodens*.

<table>
<thead>
<tr>
<th>Case</th>
<th>Maternal risk factors</th>
<th>Maternal signs and symptoms</th>
<th>Maternal treatment</th>
<th>Gestational age</th>
<th>Birth weight</th>
<th>Newborn signs and symptoms</th>
<th>Newborn treatment</th>
<th>Newborn outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hu et al.</td>
<td>None</td>
<td>White blood cell count (WBC) 19,000/mm³</td>
<td>None</td>
<td>33 weeks</td>
<td>1395 g</td>
<td>Positive blood culture at 30 min. of life; WBC 2990/mm³; ANC 388; respiratory distress</td>
<td>Ampicillin 100 mg/kg IV every 12 hours for 14 days; Gentamycin 4 mg/kg IV every 36 hours for 10 days; Cefotaxime 50 mg/kg every 12 hours for 5 days</td>
<td>Infant survived and improved clinically on antimicrobial therapy.</td>
</tr>
<tr>
<td>Andrés et al. [2]</td>
<td>None reported</td>
<td>Temperature 38°C; positive cervical and vaginal cultures</td>
<td>None</td>
<td>28 weeks</td>
<td>1035 g</td>
<td>Positive cultures from blood, pharynx, nose, ear, and umbilical cord; WBC 16,000/mm³ (80% neutrophils)</td>
<td>Ampicillin 50 mg/kg IV every 8 hours for two weeks</td>
<td>Infant survived.</td>
</tr>
<tr>
<td>Kostadinov &amp; Pinar [6]</td>
<td>Bleeding gums</td>
<td>Placental histology showed acute necrotizing chorioamnionitis</td>
<td>None</td>
<td>23 weeks</td>
<td>590 g</td>
<td>Positive blood and lung cultures post-mortem; bronchopneumonia and funisitis on autopsy</td>
<td>None</td>
<td>Infant died shortly after birth.</td>
</tr>
<tr>
<td>Sporken et al.[9]</td>
<td>Oral sex during pregnancy</td>
<td>WBC 15,200/mm³; temperature 38.9°C; positive cervical and amniotic fluid cultures</td>
<td>Cefotaxime 4 g per day and metronidazole 2 g per day started 18 hours prior to delivery</td>
<td>24 weeks</td>
<td>570 g</td>
<td>Positive cultures from blood, axilla, groin, mouth, throat, nose, ear, and anus; intra-cerebral hemorrhage, bronchopneumonia, and acute enteritis on autopsy</td>
<td>None</td>
<td>Infant died shortly after birth.</td>
</tr>
</tbody>
</table>
Two additional cases of neonatal *E. corrodens* infection have been described. In 2007, Bueno *et al.* [7] reported the case of a full-term neonate who presented on DOL 5 with fever and lethargy. He was found to have a positive blood culture and was successfully treated with antibiotics. More recently in 2009, Jadhav *et al.* [4] reported a case of *Eikenella* chorioamnionitis associated with frequent oral sex during pregnancy and tongue piercing in both the mother and her partner. Their case suggested ascending vaginal infection and hematogenous spread from tongue piercing as potential routes for *E. corrodens* intrauterine infection.

We describe a case in which preterm labor associated with congenital *E. corrodens* sepsis occurred in the absence of overt chorioamnionitis. Allaker *et al.* [8] found *E. corrodens* in the dental plaque of 62% of the healthy dogs studied, which may have been a potential risk factor. However, the mother denied any dog bites or injuries. Aside from this, the mother did not have any apparent risk factors for *Eikenella* bacteremia or ascending infection. Her only sign of possible infection was an elevated white blood cell count. This may have been an indication of early chorioamnionitis, which may explain the route of fetal infection.

Most notably, our case is one of only two reported cases of congenital *E. corrodens* sepsis associated with survival of the newborn. This is likely related to the fact that two of the prior cases of congenital sepsis occurred at a very early gestational age. However, early recognition and treatment with effective antibiotics should not be underemphasized. In the case reported by Sporken *et al.* [9], the mother received a first-generation cephalosporin and metronidazole prior to delivery, which were not effective against *E. corrodens*. Her cultures grew a strain of *Eikenella* that was susceptible to most other antibiotics. In the case described by Andrés *et al.* [2], the newborn was treated appropriately with ampicillin beginning on day 1 of life. The infant in their case survived.

In our case, a high index of suspicion for infection as the cause of preterm delivery led to the finding of bacteremia caused by an unexpected pathogen. This demonstrates that *E. corrodens*, while an uncommon cause of materno-fetal infection, may cause congenital sepsis and preterm delivery in the absence of clinical maternal infection. The infant in our case survived secondary to increased gestational age, probable early onset of chorioamnionitis, and the prompt administration of appropriate antibiotics.

REFERENCES


