

Maternal Colonization and Neonatal Sepsis Caused by *Edwardsiella tarda*

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ABSTRACT. A case of neonatal sepsis caused by *Edwardsiella tarda*, a bacterium usually associated with freshwater ecosystems, is described. The infant's mother was immersed in lake water during the sixth month of pregnancy and had vaginal and gastrointestinal colonization with the same strain of *E tarda* as the infant at the time of delivery. This case suggests that maternal exposures to contaminated bodies of water during pregnancy may represent a risk to newborns. *Pediatrics* 2003;111:e296–e298. URL: <http://www.pediatrics.org/cgi/content/full/111/3/e296>; water-borne infection, Gram-negative bacteria, neonatal sepsis.

ABBREVIATIONS. CSF, cerebrospinal fluid; rep-PCR, repetitive extragenic palindromic polymerase chain reaction.

Edwardsiella tarda is a Gram-negative bacterium associated with freshwater ecosystems.¹ Known to colonize a wide variety of amphibians, reptiles, and fish, *E tarda* can also cause disease in these animals, as exemplified by emphysematous putrefactive disease of catfish.² Human disease is unusual: a 1993 review³ cited approximately 300 reports in the English-language literature, most of them describing patients with gastroenteritis (83%), wound infection (8%), or bacteremia (5%). As might be expected, contact with marine environments was a common theme in these reports, but other risk factors were apparent, including penetrating trauma, age extremes, underlying hepatobiliary disease, iron overload states, and immune incompetence. To our knowledge, only 2 previous cases of neonatal *E tarda* sepsis have been reported.^{4,5} The present case is unique in that maternal colonization with the infecting organism was demonstrated and plausible risk factors for exposure were present.

CASE REPORT

A 6-day-old white boy presented with a 1-day history of lethargy, decreased feeding, and emesis. He was hypotonic and moderately jaundiced. Temperature was 38.7°F, pulse was 187 beats/min, respiratory rate was 28/min, and blood pressure was 72/53 mm Hg. White blood cell count was $5.2 \times 10^9/L$ ($5200/mm^3$) with 1% juvenile forms, 20% bands, 30% neutrophils, 32% lymphocytes, 9% monocytes, 3% eosinophils, and 5% atypical lymphocytes; hemoglobin was 143 g/L (14.3 g/dL), platelet count was $130 \times$

$10^9/L$ ($130\,000/mm^3$), and the C-reactive protein level was 11.0 mg/dL. Cerebrospinal fluid (CSF) white cell count was $2 \times 10^6/L$ ($2/mm^3$), red blood cell count was $2 \times 10^6/L$ ($2/mm^3$), and no organisms were seen on Gram stain. Chemistry tests were remarkable for a total bilirubin level of 253 $\mu\text{mol/L}$ (14.8 mg/dL). The patient was admitted to an intensive care unit, given intravenous fluids, and empirically treated with ampicillin (100 mg/kg every 6 hours) and gentamicin (2.5 mg/kg every 8 hours).

By hospital day 2, he was afebrile, was feeding well, and had a normal physical examination. The initial blood culture grew *E tarda* within 12 hours, susceptible to ampicillin (minimum inhibitory concentration $\leq 2 \mu\text{g/mL}$) and gentamicin (minimum inhibitory concentration $\leq 1 \mu\text{g/mL}$); urine and CSF cultures were negative. On hospital day 5, gentamicin was discontinued and the infant was treated with ampicillin alone until the 10th day, when he was discharged in good health with no detectable sequelae. At 5 months of age, he was well but had mild to moderate left-sided and severe to profound right-sided hearing loss demonstrated by auditory brainstem response audiometry. Tympanostomy tubes were placed at 7 months, and at 8 months he had normal hearing sensitivity on the left but severe hearing loss on the right as demonstrated by auditory brainstem response audiometry and distortion-product otoacoustic emissions. At 11 months of age, he was developmentally normal and the cause of unilateral hearing loss remained undetermined.

The infant was born at term by spontaneous vaginal delivery to a healthy mother. There were no complications during pregnancy, labor, or delivery, and the patient went home on the second day of life. The mother did not work outside the home. Cleansing and ingestion of fish were not common in the household, but the father worked as a maintenance worker on boats that were docked at a freshwater lake. The mother had been baptized by immersion in the same lake in June, during the sixth month of pregnancy. Cultures of the lake water, obtained in the fall shortly after the infant was admitted, were negative. Stool cultures from the father and the patient's older brother, obtained at the same time, did not grow *Edwardsiella* species. However, *E tarda* was isolated from the mother's stool, and a vaginal swab culture yielded *E tarda* as well as normal vaginal flora.

The maternal and infant strains of *E tarda* had identical antibiotic susceptibility patterns. These strains were compared using repetitive extragenic palindromic polymerase chain reaction (rep-PCR), wherein noncoding repetitive bacterial DNA sequences lying between genomic coding regions are amplified.^{6–8} Isolates were grown overnight in trypticase soy broth then lysed by microbead disruption. DNA was isolated (UltraClean Microbial Genomic DNA Isolation Kit; Mo Bio Laboratories, Inc, Solana Beach, CA), and rep-PCR was performed using 2 different primers (Uprime-B1 and Uprime-RI) at Bacterial BarCodes, Inc (Houston, TX). Amplification products were then separated on 1.5% agarose gels containing 3 $\mu\text{g/mL}$ ethidium bromide. The banding pattern for both strains was identical (Fig 1).

DISCUSSION

Neonatal *E tarda* sepsis was first reported in 1968 from Nigeria.⁴ The female infant was born at term and presented on the 10th day of life with fever, poor feeding, and weight loss. CSF showed 9625 white blood cells (90% neutrophils), and the organism was isolated from both CSF and blood. The infant ultimately died despite antibiotic treatment, and the authors speculated that the source of infection was

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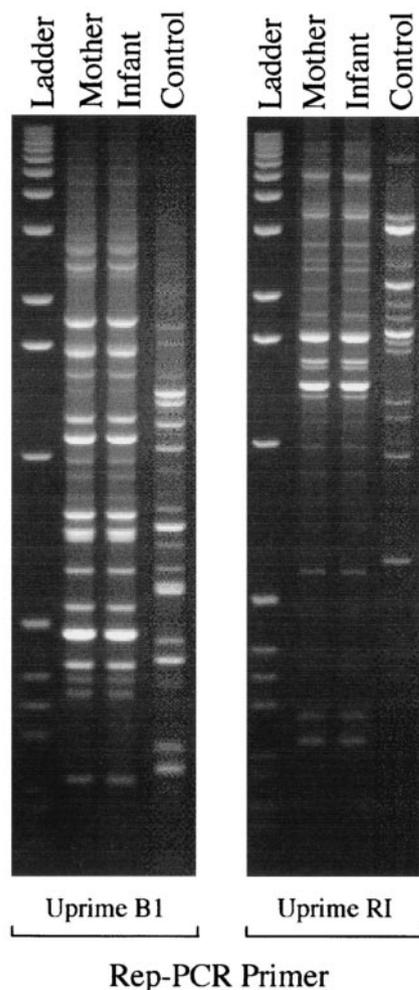


Fig 1. DNA fingerprint of maternal (vaginal) and infant (blood) isolates of *E tarda* using 2 different rep-PCR primers. Control organism was group A streptococcus. Ladder refers to standard DNA molecular weight markers.

snake flesh eaten by the mother. A second case was reported from Brooklyn, NY, in 1988.⁵ This was an 1820-g premature female infant born vaginally to a mother who experienced a self-limited diarrheal illness 5 days before delivery. Lethargy, hypotonia, apnea, cyanosis, and bradycardia developed on the third day of life, and the infant died shortly thereafter despite treatment with penicillin and gentamicin. At autopsy, meningitis was apparent and *E tarda* was isolated from CSF, blood, brain, and heart. There was no mention of maternal exposure to marine animals or environments, and cultures from the parents' stools, the pet bird's excrement, and household tap water failed to yield *Edwardsiella* species.

The present case is unique in that meningitis was absent and the infant survived. More important, the case comes close to providing a complete paradigm for neonatal *Edwardsiella* sepsis. The infant most likely acquired the infection from the mother, because the strains isolated from the infant's blood and the mother's vagina were phenotypically and genetically similar and cultures from the father and brother were negative. Perinatal (vertical) transmission is suggested by onset of illness on the fifth day of life, although postnatal (horizontal) transmission

cannot be excluded. Given the rapidity with which the organism grew from the infant's blood (suggesting a high inoculum) and his clinical presentation, it is unlikely that the isolate represented contamination from skin colonization. It is possible that the mother became colonized with *E tarda* after immersion in the shallow part of the freshwater lake in the summertime, although there is no proof that the water was contaminated (cultures were done several months later after the weather had turned colder). Other studies point to associations between *E tarda* infections and marine exposures during warm-weather months,⁹ and *E tarda* colony counts in bodies of freshwater are known to increase with warm weather.¹⁰ It remains possible that the father was the first to acquire the organism through his work on boats at the same location. However, because he had no evidence of gastrointestinal colonization at the time of delivery, he would have been an unlikely source for the infant's infection.

E tarda is known to cause gastroenteritis, particularly in tropical and subtropical countries where dietary habits include raw fish.³ Disease manifestations include acute secretory enteritis, bacillary dysentery, and chronic gastroenteritis. It is noteworthy that the mother in this case did not have a diarrheal illness during pregnancy. Whereas asymptomatic carriage of *E tarda* has been documented, it is extremely rare; 1 study in Japan found only 26 cases in >353 600 individuals.¹¹ This mother also had no symptoms of vaginal infection. Little is known about asymptomatic vaginal colonization with *E tarda*, but at least 5 cases of gynecologic infection have been reported, including tubo-ovarian abscess,⁹ Bartholin cyst abscess,^{9,12} salpingitis,¹³ and uterine abscess.¹⁴

Edwardsiella species are oxidase-negative, catalase-positive, motile, Gram-negative bacilli.¹ They can ferment glucose like other members of the family Enterobacteriaceae and, like salmonella and shigella species, are unable to ferment lactose. In fact, *E tarda* closely resembles salmonella biochemically, particularly in its ability to produce hydrogen sulfide on common laboratory media. This may lead to misidentification, although indole production is a distinguishing feature of *E tarda*. Most strains of *E tarda* are susceptible to ampicillin, gentamicin, and cefotaxime,¹⁵ antibiotics that are commonly included in empiric treatment of neonatal sepsis.

E tarda seems to be capable of colonizing the maternal birth canal and causing neonatal infection. The recent case of neonatal sepsis and meningitis caused by *Pseudomonas aeruginosa* acquired from a hot tub¹⁶ raised concerns about maternal exposures to contaminated bodies of water during pregnancy. Although the current case might raise similar concerns with respect to freshwater lakes, the risk is likely to be small because bathing is probably common during pregnancy and neonatal sepsis caused by contaminating organisms is rare.

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