Bordetella bronchiseptica Pneumonia in an Extremely-Low-Birth-Weight Neonate

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ABSTRACT

Bordetella bronchiseptica, a gram-negative coccobacillus, is a common veterinary pathogen. In both domestic and wild animals, this bacterium causes respiratory infections including infectious tracheobronchitis in dogs and atrophic rhinitis in swine. Human infections are rare and have been documented in immunocompromised hosts. Here, we describe an extremely-low-birth-weight infant with *B. bronchiseptica* pneumonia. This is the first report that describes the microorganism's responsibility in causing nosocomial infection in a preterm neonate. He recovered uneventfully after a course of meropenem. It is possible that the bacteria colonize the respiratory tracts of our health care workers or parents who may have had contact with pets and then transmitted the bacterium to our patient. Follow-up until 21 months of age showed normal growth and development. He did not suffer from any significant residual respiratory disease.

KEYWORDS: *Bordetella bronchiseptica,* extremely low birth weight, preterm, neonate

Bordetella bronchiseptica, a pleomorphic gram-negative coccobacillus, is a common veterinary pathogen. In both domestic and wild animals, this bacterium causes respiratory infections including infectious tracheobronchitis in dogs and atrophic rhinitis in swine.[1] Case reports suggest that cavitary pneumonia or disseminated infections occur in immunocompromised patients.[2] The risk of acquiring *B. bronchiseptica* in the pediatric population is unclear, although certain children post–lung transplantation have been reported to acquire the infection from their ill pets.[3] Here, we report the first account of *B. bronchiseptica* pneumonia in an extremely-low-birth-weight infant.

CASE HISTORY

This male baby was the second infant from a twin pregnancy. Spontaneous onset of preterm labor developed at 25 weeks of gestation. One course of antenatal steroids was completed. The babies were delivered via emergency lower-segment caesarean section. He was intubated in the delivery room. The Apgar score after 1 minute was 5, and after 5 minutes it was 7. The birth weight was 690 g.

Surfactant (Survanta®, Abbott Labs, North Chicago, IL) was administered soon after admission into our neonatal intensive care unit (NICU) for respiratory distress syndrome. The infant was successfully extubated

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on the third day after birth with application of noninvasive ventilation. The baby remained stable except for an episode of catheter-related methicillin-resistant coagulase-negative staphylococcus (CoNS) bloodstream infection on day of life (DOL) 9. The ventilatory setting was gradually increased by the third week, and reintubation was required on DOL 22. Chest radiography showed an increased haziness over both lung fields (see Fig. 1). The baby had increased respiratory secretions. C-reactive protein (CRP) increased to 1.78 mg/dL (normal is < 0.76 mg/dL) on DOL 22. The complete blood picture suggested leukocytosis (leukocyte count $= 14.7 \times 10^9$/L). Vancomycin was continued for the CoNS bloodstream infection, and ceftazidime was added. Endotracheal aspirate (ETA) culture on the same day revealed growth of *Escherichia coli* sensitive to both first- and second-generation cephalosporins. Blood culture showed no growth. There was no remarkable clinical improvement in the subsequent few days. CRP remained elevated. A high ventilatory setting was needed, with fraction of inspired oxygen remaining up to 0.6. On DOL 26, we repeated the ETA culture, which yielded a pure growth of a gram-negative rod that was identified as *B. bronchiseptica* by colony morphology, oxidase, and the GNI + card (bioMerieux Vitek, Hazelwood, MO). Regarding antimicrobial susceptibility, our microbiology laboratory adopted the guidelines for agar diffusion testing for *Enterobacteriaceae* as references. The strain identified was reported to be sensitive to cefuroxime and ceftazidime. The infant's lung condition remained unchanged, and he required a high ventilatory setting (peak inspiratory pressure was 22 cm H_2O). The repeat ETA culture on DOL 29 revealed growth of both *B. bronchiseptica* (sensitive to cefuroxime and ceftazidime) and CoNS. Fungal culture was negative. Because there was no agreed-upon antibiotic zone size in the routine Clinical and Laboratory Standards Institute testing system, the sensitivity results of *B. bronchiseptica* could only be regarded as tentative. Vancomycin was administrated for 14 days till DOL 27 for coverage of methicillin-resistant CoNS bacteremia. A 12-day course of ceftazidime was administered until DOL 33. In view of the infant's lack of improvement in respiratory status, on DOL 33, we decided to switch the antibiotic treatment to a 7-day course of meropenem. ETA culture on DOL 36 showed scanty growth of CoNS only. CRP normalized on DOL 39, and the infant was extubated on the same day.

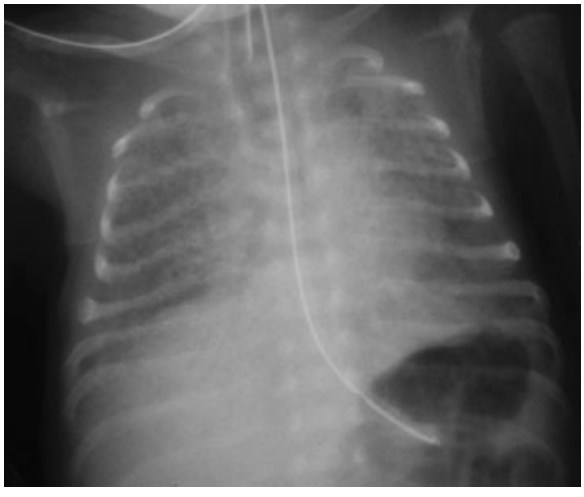

Figure 1 Chest radiograph of our patient showing pneumonic changes.

The baby experienced other complications of extreme prematurity; these included transient hyperglycemia, neonatal jaundice, feeding intolerance, gastroesophageal reflux, and stage 2 retinopathy of prematurity. The infant was weaned off oxygen supplementation at the postconceptual age of 45 weeks because of bronchopulmonary dysplasia. He was ultimately discharged on day 150. Follow-up until 21 months of age showed normal growth and development. There was no active health problem including respiratory aspects.

DISCUSSION

B. bronchiseptica, an obligatory aerobic gram-negative coccobacillus, is small and motile. It is nonfermentative, producing no indole or hydrogen sulfide, but it does produce cytochrome oxidase, lysine decarboxylase, and catalase. It belongs to the genus *Bordetella*, which consists of seven species. Unlike the fastidious *B. pertussis*, it grows on routinely used media, including blood, chocolate, and MacConkey agars. The genotypical approach utilizing the 16S rRNA molecular sequencing method has been developed to allow the rapid identification of *B. bronchiseptica*.[4] The genome of *B. bronchiseptica* has been sequenced, and these advances in molecular biology aid in furthering the understanding of the bacterium.[5]

B. bronchiseptica is responsible for respiratory infections in many different mammals, including mice, rats, cats, dogs, foxes, pigs, horses, and occasionally humans. The most important and best described natural infections cause kennel cough and atrophic rhinitis in dogs and swine, respectively.[2] Studies from animals suggest that the bacteria adhere to the cilia and surface structures of respiratory epithelium and result in the mechanical blocking of respiratory cilia and failure to clear mucus secretions. The production of various virulence factors, such as heat-labile dermonecrotic toxin, adenylate cyclase-hemolysin, and filamentous hemagglutinin, contributes to the disease development.[1,6] In vitro studies reveal that *B. bronchiseptica* demonstrates preferential adherence to the nonhuman mammalian ciliated cells of rabbits, mice, and hamsters, whereas *B. pertussis* better adheres to human ciliated cells.[7] Such differential tropism explains why *B. bronchiseptica* causes infections mainly in animals, whereas *B. pertussis* is strictly a human pathogen.

Human disease due to this bacterium has been reported as early as 1911, but it was not until the 1970s that stringent criteria for differentiating *B. bronchiseptica* from other phenotypically similar nonfermentative gram-negative coccobacilli were followed.[1] The organism is capable of colonizing the human respiratory tract. There have been reports of *B. bronchiseptica* respiratory infections in immunocompromised hosts, such as the malnourished child, those suffering from acute leukemia, cystic fibrosis, and AIDS, as well as those patients who had undergone lung and bone marrow transplantations.[3,4,8–12] In the absence of a suitable selective culture media (such as blood agar supplemented with cephalexin) used in the laboratory, underreporting of the disease caused by this organism is likely.[13]

Some patients had prior contact with dogs or cats.[8,10] Nosocomial transmission of *B. bronchiseptica* was described in bone marrow transplantation setting, as confirmed by pulsed-field gel electrophoresis techniques.[10] Rath et al reported a 6-week-old previously healthy African-American infant who had a *B. bronchiseptica* infection leading to respiratory failure after household contact with a dog recently vaccinated with a live, attenuated vaccine for kennel cough.[14] Airborne transmission was found to be probable in the experimental setting.[15] In our NICU, no similar isolates were found in other babies. It is possible that the bacteria colonize the respiratory tracts of our health care workers or parents who may have had contact with pets and then transmitted the bacterium to our patient.

There have been wide variations in susceptibility results from different studies;, these results are not readily explainable but may reflect differences in the populations of strains tested, methodological variations, or both.[1] Most *B. bronchiseptica* strains are reportedly sensitive to aminoglycosides, extended-spectrum third-generation cephalosporins, tetracyclines, quinolones, and trimethoprim-sulfamethoxazole.[2] Carbapenem and quinolones[10,14,16] were used to save some critically ill patients.

CONCLUSION

This report expands the spectrum of immunocompromised hosts to include preterm, extremely-low-birth-weight infants who develop an infection from *B. bronchiseptica*. Our case recovered after prolonged antibiotic therapy with an eventual switch to a carbapenem. When similar scenarios occur in the future, we may consider conducting surveillance cultures among all contacts to document the route of transmission and prevent potential spread.

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