Case Report

# Leuconostoc bacteremia in a child with short-gut syndrome

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## ABSTRACT

In the last decade Leuconostoc species have been reported with increasing frequency as human pathogens, causing bacteremia, meningitis and peritonitis. We report here a child with short-bowel syndrome who developed bacteremia following multiple surgeries for necrotizing enterocolitis. Leuconostoc species was isolated from the blood cultures. The child was successfully treated with ampicillin and gentamycin. He however remained total parenteral nutrition dependent due to his multiple abnormalities. We call the attention of microbiologists and pediatricians to this emerging pathogen, which is intrinsically resistant to vancomycin and can be misidentified in the microbiology laboratory as *Viridans streptococci* or *Enterococci*. Increased awareness by clinicians of this organism is called for, if it is to be recognized and appropriately treated.

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Leuconostoc species (spp) were, until recently, regarded as non-pathogenic for humans. In 1985, the first cases of Leuconostoc bacteremia were reported in France and the potential pathogenicity of the species became recognized.[1] Since then varying systemic infections have been reported in the literature, such as bacteremia,[2-4] meningitis,[5] spontaneous bacterial peritonitis,[6] multiple liver abscesses[7] and bacteremia following liver transplantation.[8] Leuconostoc spp belong to the group of *Streptococcaceae* and are facultative anaerobic gram-positive coccal and coccobacillary bacteria resembling *Viridans streptococci* in colonial morphology, but differing from other Streptococci in their resistance to vancomycin.[9] We report a 10-month old baby boy who developed short-bowel syndrome following severe necrotizing enterocolitis, for which he had surgical removal of a large part of the small intestine. He continued to develop several episodes of sepsis and on one occasion, Leuconostoc spp was recovered in his blood culture.

Case Report. This is a 10-month old male born by cesarean section at 28 weeks gestation. The weight at birth was 800 grams. He was then admitted to the neonatal intensive care unit (NICU). During his prolonged stay in the NICU, he developed the following complications; intraventricular hemorrhage grade III, arrested post-hemorrhagic hydrocephalus; severe respiratory distress syndrome; bronchopulmonary dysplasia and continued to be oxygen dependent; patent ductus arteriosus closed by surgery; and severe necrotizing enterocolitis (NEC), for which he had multiple surgeries. The major part of the small intestine was resected and removed but excluding the ileocecal valve. He was labeled as a case of "short-bowel syndrome" (**Figure 1**). The patient became total parenteral nutrition-dependent (TPN) and developed

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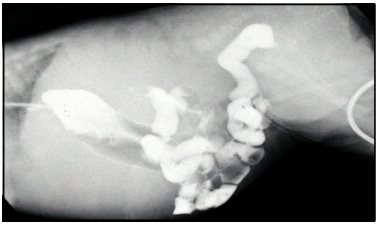

Figure 1 - Postoperative barium study showing the residual short bowel.

liver complications, manifested by increased liver enzymes and jaundice. His general status was stable, and he was gaining weight, so he was transferred from the NICU to the Pediatric ward. At the age of 3 months, his weight was 2 kg. On the Pediatric ward, he continued to be TPN dependent, growing slowly, but with little weight gain, and not gaining normal milestones well. At age 10 months, he could not control his head well, but could only roll over on his side. He was spastic, mainly in the lower limbs, with a little following of close objects with his eyes. He now weighed 5.7 kg, looked well nourished and oriented. During his prolonged hospital stay he had 2 episodes of coagulase negative staphylococci line sepsis, for which he was treated with intravenous vancomycin, to which he responded satisfactorily. He then had final surgery to close the colostomy, after which he developed a fever and routine blood cultures taken grew Leuconostoc spp. On the plate, the organism was alpha hemolytic after growth at 37°C, producing grayish colonies and on gram stain was coccoid, arranged in pairs or short chains. It was thought at first to be *Viridans streptococci*, but its resistance to vancomycin prompted further investigations, namely, fermentation of glucose with gas, bile-aesculin test positivity, and growth in MRS (deMann, Rogosa, Sharpe) medium, which confirmed its identity.[9] He was treated with ampicillin (100mg/kg/day) and gentamycin (2.5mg/kg/dose).

**Discussion.** Until 1985, Leuconostoc spp were regarded as non-pathogens in man. However, in 1985, Leuconostoc spp were isolated from the blood of 2 immunocompromised patients. Since then, they have been isolated from cases of meningitis, peritonitis, and liver abscesses.[1,2] Leuconostoc are members of the *Streptococcaceae* and are usually found in soil, dairy products and vegetable matter.[1] They are gram-positive coccoid bacteria, non-motile and facultatively anaerobic. They usually appear on initial isolation as *Viridans streptococci* but on susceptibility testing are resistant to vancomycin, which fact prompts further investigations into their characteristic features.[9] The isolate in this case produced alpha hemolysis on blood agar. Its resistance to vancomycin by Kirby Bauer technique prompted further investigations. The isolate hydrolyzed esculin and produced no ammonia from arginine. It was susceptible to penicillin and ampicillin.[9,10] Further confirmation of the isolate was carried out using the Microscan (Microscan Walkaway 40 SI. Dade Behring Inc. 1584 Enterprise Blvd., West Sacramento, CA 95691, USA), which showed the isolate to be Leuconostoc spp.

Our patient had all the characteristic features described in the literature for the development of Leuconostoc infection, such as a short-bowel syndrome, severe host-defense impairment, presence of central venous catheter, general poor clinical status. Besides these, he had patent ductus arteriosus, hydrocephalus, multiple episodes of sepsis and bowel surgery.

The portal of entry of Leuconostoc to produce disease in man is uncertain and undetermined.[1] However, since they are usually found in vegetable matter and food stuffs, it has been suggested that the gastrointestinal tract is a potential reservoir, from which they may gain access to the blood stream especially in immunocompromised patients, whose integumentary barrier provided by the intestinal lining, may be deficient.[1] In our case, we speculate that the organism gained access into the blood stream through the gastrointestinal tract. The predisposing factors for the development of Leuconostoc infection have been reported to be prematurity, short-gut syndrome, prior vancomycin therapy and central venous catheter insertion.[2] All these predisposing factors were present in our patient.

Some reports have suggested that since the organism is not normally present in the gastrointestinal flora, they may be present in infant feeding and the organism may gain access into the blood stream, especially in patients with underlying gastrointestinal disorders.[1,2,10] Nosocomial bacteremia caused by Leuconostoc has been described, resulting from extrinsic contamination of infant formula.[11,12] The possibility of this exists in our patient, as we did not culture the patient's infant feed.

The usual therapy of Leuconostoc infection is with the penicillins, but other agents such as erythromycin, cephalosporins, and aminoglycosides are also effective as the organism is susceptible to these agents in vitro.[13] However, aminoglycosides are not recommended for monotherapy, but when combined with beta-lactam agents effect good synergistic activity.[2] Our patient was treated with a

combination of ampicillin and gentamycin and he responded very well.

Leuconostoc spp are now to be regarded as emerging pathogens and the attention of clinicians and microbiologists is called to this organism. They should be suspected when *Viridans streptococci* are cultured from blood or sterile body fluids and are vancomycin resistant, particularly in immunocompromised patients, and more so in those with central venous catheters and predisposing factors for Leuconostoc infections detailed above.

## References


1. Handwerger S, Horowitz H, Coburn K, Kolokathis A, Wormser GP. Infection due to Leuconostoc species: six cases and review. ***Rev Infect Dis*** 1990; 12: 602-610.
2. Dhodapkar KM, Henry NK. Leuconostoc bacteremia in an infant with short-gut syndrome: case report and literature review. ***May Clin Proc*** 1996; 71: 1171-1174.
3. Bernaldo de Quiros JC, Munoz P, Cercenado E, Hernandez Sampelayo T, Moreno S, Bouza E. Leuconostoc species as a cause of bacteremia: two case reports and a literature review. ***Eur J Clin Microbiol Infect Dis*** 1991; 10: 505-509.
4. Ling ML. Leuconostoc bacteremia. ***Singapore Med J*** 1992; 33: 241-243.
5. Coovadia YM, Solwa Z, van den Ende J. Meningitis caused by vancomycin–resistant Leuconostoc sp. ***J Clin Microbiol*** 1987; 25: 1784-1785.
6. Templin KS, Crook T, Riley T 3rd, Whitener C, Aber RC. Spontaneous bacterial peritonitis and bacteremia due to Leuconostoc species in a patient with end-stage Liver disease: a case report. ***J Infect*** 2001; 43: 155-157.
7. Vagiakou-Voudris E, Myolona-Petropoulou D, Kalogeropoulou E, Chantziz A, Chini S, Tsiodra P, et al. Multiple liver abscesses associated with bacteremia due to Leuconostoc lactis. ***Scand J Infect Dis*** 2002; 34: 766-767.
8. Espinoza R, Kusne S, Pasculle AW, Wada S, Fung J, Rakela J. Leuconostoc bacteremia after liver transplantation: another cause of vancomycin resistant gram-positive infection. ***Clin Transplant*** 1997; 11: 322-324.
9. Facklam RR, Hollis D, Collins MD. Identification of gram-positive coccal and coccobacillary vancomycin-resistant bacteria. ***J Clin Microbiol*** 1989; 27: 724-730.
10. Isenberg HD, Vellozzi EM, Shapiro J, Rubin LG. Clinical laboratory challenges in the recognition of Leuconoctoc spp. ***J Clin Microbiol*** 1988; 26: 479-483.
11. Noriega FR, Kotloff KL, Martin MA, Schwalbe RS. Nosocomial bacteremia caused by Enterobacter sakazaki and Leuconostoc mesenteroides resulting from extrinsic contamination of infant formula. ***Pediatr Infect Dis J*** 1990; 9: 447-449.
12. Carapetis J, Bishop S, Davis J, Bell B, Hogg G. Leuconostoc sepsis in association with continuous enteral feeding: case reports and a review. ***Pediatr Infect Dis J*** 1994; 13: 816-823.
13. Swenson JM, Facklam RR, Thornsberry C. Antimicrobial suspectibility of vancomycin-resistant Leuconostoc, Pediococcus and Lactobacillus species. ***Antimicrob Chemother*** 1990; 34: 543-549.