Meningitis Caused by Vancomycin-Resistant *Leuconostoc* sp.  

YACOOB M. COOVADIA, ZAREENA SOLWA, AND JAN VAN DEN ENDE*  
Department of Microbiology, University of Natal Medical School and King Edward VIII Hospital, Durban, South Africa  

Received 26 February 1987/Accepted 5 June 1987

A rare case of purulent meningitis caused by vancomycin-resistant *Leuconostoc* sp. is reported. The patient was treated successfully with penicillin G, and there were no neurological sequelae. We recommend that all gram-positive cocci isolated from cerebrospinal fluid and blood be fully identified so as not to confuse *Leuconostoc* sp. with more commonly isolated pathogens such as pneumococci and other alpha-hemolytic streptococci.

*Leuconostoc* spp. are gram-positive, nonmotile, nonsporing facultative anaerobes found on plants and dairy products (7). They are generally considered nonpathogenic and of little or no importance in clinical microbiology. However, Buu-Hoi et al. (4) recently reported the isolation of *Leuconostoc* spp. from blood cultures obtained from two immunocompromised patients and suggested that these organisms can cause serious infections, especially in the immunocompromised. Like Shlaes et al. (8), they recommended that all gram-positive bacteria be routinely tested against vancomycin, so as not to miss strains of streptococci or *Leuconostoc* spp. resistant to this antibiotic. The present report documents the isolation of a *Leuconostoc* sp. from the cerebrospinal fluid (CSF) of a 16-year-old woman with pyogenic meningitis. As far as we could ascertain, this is the first reported case of meningitis caused by this organism.

**Case report.** A previously healthy 16-year-old woman was admitted to the medical wards of King Edward VIII Hospital with a 1-day history of severe headache, painful eyes, vomiting, and neck stiffness. She also admitted to having a nonproductive cough, dysuria, loss of appetite, and constipation, but she specifically denied any history of trauma.

The only relevant past history included hospitalization as a child for generalized body pains and drinking of river water. Examination revealed a well-nourished young woman with a temperature of 37.8°C, herpes labialis, and marked neck stiffness with positive Kernig’s and Brudzinski’s signs. No other abnormalities were noted on examination. CSF obtained on admission was turbid with 640 × 10⁶ leukocytes per liter (75% neutrophils and 25% lymphocytes), 5.3 g of total protein per liter, and 0.3 mmol of glucose per liter (blood glucose, 6.6 mmol/liter). Gram staining of the CSF revealed numerous gram-positive diplococci. Other relevant laboratory results were: hemoglobin, 13.1 g/dl; leukocytes, 18.2 × 10⁶/liter; total serum protein, 67 g/liter (albumin, 34 g/liter; globulin, 33 g/liter); and liver enzymes within normal limits. A chest radiograph showed inflammatory changes consistent with bronchopneumonia in the right lung, but a skull radiograph was reported as normal. Latex agglutination antigen studies on CSF were negative for *Haemophilus influenzae* type b, *Streptococcus pneumoniae*, Neisseria meningitidis groups A, B, C, Y, and W135, and *Streptococcus agalactiae* (Wellcome; Wellcome Research Laboratories, Beckenham, England). Initial management included intravenous penicillin G (2 million units per 2 h) and intravenous chloramphenicol (1 g/6 h). Culture of the CSF yielded heavy growth of a facultatively anaerobic, alpha-hemolytic, gram-positive diplococcus which was presumptively identified as *S. pneumoniae*. The organism was susceptible to penicillin, ampicillin, chloramphenicol, cotrimoxazole, and cefotaxime. On day 3, the patient was noted to have a sixth left cranial nerve palsy and depressed reflexes in the left leg. A computer-assisted tomographic brain scan did not reveal any intracranial lesions or hydrocephalus. The cranial nerve palsy resolved after the institution of dexamethasone therapy, and her progress thereafter was uncomplicated. Chloramphenicol was stopped on day 5. Repeat examination of the CSF after 10 days of therapy revealed a total leukocyte count of 20 × 10⁶/liter with equal numbers of neutrophils and lymphocytes; total protein, 0.56 g/liter; and glucose, 5.3 mmol/liter. After 2 weeks of penicillin therapy, the patient was discharged well with no evidence of neurological deficit.

**Microbiology.** Blood cultures obtained at admission yielded no growth. The CSF isolate was shown to be catalase and oxidase negative but did not agglutinate with pneumococcal antisera (Statens Seruminstitute, Denmark). It also could not be identified by the API 20 Strep (API, France) and was shown to be resistant to vancomycin, against which we routinely test all gram-positive isolates. We then tested for gas production in MRS broth (6) containing glucose. A positive result was obtained, which thereby excluded identification of the isolate as a streptococcus. *Leuconostoc* spp. were then considered, and this was supported by results obtained with the API 50 CH system together with a characteristic antimicrobial resistance pattern (4). The organism was resistant to optochin, vancomycin, trimethoprim, sulfamethoxazole, teichomycin, and cefoxitin but susceptible to penicillin, ampicillin, tetracycline, erythromycin, chloramphenicol, clindamycin, cephalaxin, cefotaxime, cefotaxime, gentamicin, and amikacin as determined by the Stokes and Waterworth agar disk diffusion method (E. J. Stokes and P. M. Waterworth, Assoc. Clin. Pathol. Broadsheet 55, 1972). The strain fermented glucose, sucrose, maltose, ribose, L-arabinose, D-xylene, galactose, D-fructose, D-mannose, salicin, cellobiose, and N-acetylglucosamine but did not ferment mannitol, dulcitol, L-xylene, sorbitol, inositol, inulin, L-sorbose, rhamnose, and erythritol. Tests for nitrite reduction, gelatin liquefaction, and hydrolysis of esculin were also positive. These results are in complete agreement with those described by Bridge and Sneath (3) for inclusion in the genus *Leuconostoc*. MICs (in milligrams per liter) determined on Mueller-Hinton agar
by the agar dilution method (5) were: vancomycin, ≥256; penicillin, 1; chloramphenicol, 4; erythromycin, ≤0.01; methicillin, 8 (32°C); fusidic acid, 8; rifampin, 1; and imipenem, ≤0.01.

The case described here lends further support to the report of Buu-Hoï et al. (4) that *Leuconostoc* spp. can cause infections in humans. However, in contrast to the two cases described by them, our patient did not have any obvious underlying disease which could have predisposed her to infection. In addition, the patient was successfully treated with penicillin and at the time of discharge was completely normal physically and neurologically. Antimicrobial susceptibility testing revealed that the isolate was susceptible to most of the antibiotics tested, except vancomycin, trimethoprim, sulfamethoxazole, teicoplanin, and cefoxitin. These results are in complete agreement with these reported by Buu-Hoï et al. (4).

Although C. Thornsberry and R. R. Facklam (Antimicrob. News1. 1:63–64, 1984) have indicated that many vancomycin-resistant streptococci are in fact lactobacilli, other reports indicate that vancomycin resistance does indeed occur among streptococci, although this is rare (1, 2, 8). Serious infection caused by a vancomycin-resistant strain of *Streptococcus sanguis* II was documented by Shlaes et al. (8). We are therefore in agreement that all gram-positive isolates from blood and other sterile body fluids be routinely tested with vancomycin. Buu-Hoï et al. (4) have recommended that vancomycin-resistant strains should, in addition, be tested for gas production in glucose-MRS broth (6), which if positive is suggestive of *Leuconostoc* spp. However, it should be emphasized that heterofermentative lactobacilli may also produce gas from glucose and be vancomycin resistant. Thus, full characterization of such strains is still necessary. By using this approach, we have recently recognized three additional strains of gram-positive cocci in blood cultures that are vancomycin resistant, produce gas, and are at present being further characterized. Although *Leuconostoc* spp. may be only rare causes of human infections, we believe that laboratories should be on the alert for these organisms, especially in light of our experience that they may initially be confused with a more common pathogen, such as *S. pneumoniae*.

Y. M. C. is in receipt of a South African Medical Research Council grant.

We are grateful to the physician in charge for allowing us access to her patient’s records and the Medical Superintendent, King Edward VIII Hospital, for permission to publish.

**LITERATURE CITED**


