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Leuconostoc mesenteroides Bacteremia in a Patient With Lower Extremity Cellulitis

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A 51-year-old woman with a history of obesity, bilateral lower-extremity edema, type 1 diabetes mellitus, rheumatoid arthritis, multiple sclerosis, and spinal stenosis presented to the emergency department (ED) with left leg swelling, pain, redness, and abnormal warmth.

She had previously received a diagnosis of cellulitis and had been treated with amoxicillin clavulanate and cephalexin as an outpatient. She was not on any disease-modifying antirheumatic drugs (DMARDs) due to her having lost her health insurance coverage. Her medications were acidophilus-sporogenes, 1 tablet orally twice daily; cyclobenzaprine, 10 mg orally 3 times daily; gabapentin, 400 mg orally 3 times daily; hydralazine, 25 mg orally twice

daily; insulin lispro subcutaneously 4 times daily before meals and at bedtime; lisinopril, 5 mg orally daily; and topical nystatin cream twice daily for a rash on her thigh and under her breasts. She also had been taking morphine extended release, 100 mg orally 3 times a day for more than 1 year.

Her vital signs were normal, and the results of a complete blood cell count (CBC) and basic metabolic panel were unremarkable, except for hyperglycemia. She was admitted to the hospital for recalcitrant cellulitis and was treated with intravenous (IV) piperacillin-tazobactam and linezolid.

Four days later, the patient developed new-onset diarrhea without fever, along with leukocytosis. Polymerase chain reaction stool tests results were positive for *Clostridium difficile*, and the colitis was treated with a 10-day course of oral vancomycin. She was discharged on oral linezolid for an additional 3 days.

Three weeks later, the patient was readmitted to the ED due to recurrent cellulitis. At this presentation, she denied having any other associated symptoms, a history of recent travel, dental work, or zoonotic exposures. Her vital signs were stable, and her leukocyte count was within normal limits. Her erythrocyte sedimentation rate was noted to be elevated at 41 mm/h. Laboratory test results were significant for a white blood cell count of 6000/μL, neutrophils of 3700/μL, and a hemoglobin level of 10.5 g/dL. Blood cultures were drawn, and she was treated with oral doxycycline and discharged. However, 1 of 4 blood cultures grew gram-positive cocci in an anaerobic blood culture 2 days later.

The patient was readmitted to the hospital for treatment of bacteremia with IV vancomycin and piperacillin-tazobactam. Her vital signs and CBC results were normal. The patient said that she had been feeling better, and the cellulitis had improved after outpatient antibiotic treatment. Three days later, the infectious agent was identified as *Leuconostoc mesenteroides*.

She was then started on IV ampicillin and gentamicin. However, the gentamicin caused intolerable nausea and subjective fevers after the second infusion. Therefore, gentamicin was switched to ceftriaxone, 2 g/d IV. After 7 days of this treatment, the patient felt better, the cellulitis had resolved, and she had had no further fevers; she was discharged home with a peripherally inserted central catheter to finish a total of a 14-day course of antibiotics. The bacterium was sensitive to ampicillin, chloramphenicol, penicillin, and minocycline and had an intermediate sensitivity to gentamicin and ceftriaxone.

DISCUSSION

Species of the *Leuconostoc* genus are gram-positive, catalase-negative, microaerophilic cocci.1 Rarely, they can be found in gastric or vaginal fluids, but they are not considered to be part of

normal human flora.2 Due to some shared biological features, *Leuconostoc* species might be misidentified as *Lactobacillus*, *Streptococcus* (particularly the viridans group), *Pediococcus*, or *Enterococcus*.3 The bacteria also play a role in the fermentation of milk, wine, and pickles.4

Clinically, *Leuconostoc* species are characterized by their intrinsic resistance to vancomycin due to a lack of a target in the cell wall.5 Nevertheless, the recommended antibiotic treatment includes penicillin G, ampicillin, clindamycin, carbapenems, and aminoglycosides.5 Successful treatment with tigecycline has also been reported.6

Cases of infection with *Leuconostoc* species are rarely reported. Risk factors for infection with *Leuconostoc* species include immunosuppressed states, central venous line insertion, and recent vancomycin therapy. For example, a case was reported of *L mesenteroides* meningitis associated with HIV and central nervous system tuberculosis.7 However, *L mesenteroides* has also been described in several immunocompetent patients, including empyema in a healthy man producing and selling sauerkraut and pickled vegetables.4 One group of authors reported a case of endophthalmitis in a healthy man after phacoemulsification and posterior chamber implantation.1

In one instance, *Leuconostoc lactis* bacteremia was diagnosed in a patient with gastrointestinal (GI) tract amyloidosis secondary to rheumatoid arthritis and tuberculous arthritis.8 *L mesenteroides* bacteremia was also reported in a patient with partial gastrectomy secondary to Chagas disease.9

Regarding the route of infection, many authors speculate that disruption of the GI barrier may be a risk factor for *Leuconostoc* bacteremia, given that it is more commonly seen in neonates with short bowel syndrome.2,7 Additionally, some authors have suggested the skin to be a possible source for this pathogen, since several cases have been diagnosed after central venous catheter insertion or the initiation of total parenteral nutrition (TPN).9,10 In the largest nosocomial outbreak reported so far, 40 of 42 patients with *Leuconostoc* bacteremia were on TPN.10 Nevertheless, there is no well-established theory regarding the route of infection.

In our patient's case, the primary risk factor was chronic immunosuppression. Other risk factors included type 1 diabetes mellitus, a chronic inflammatory condition that alters the duodenal mucosa microbiome,11 and that the patient was on long-term opioids, which also can disrupt the GI flora.12 However, because *L mesenteroides* is not part of the normal flora, the impact of diabetes and chronic opiate use in its pathogenesis needs more epidemiological study.

We suspect that the pathogenesis in this case was cellulitis, which resulted in microscopic skin breaks and chronic edema induced by venous stasis, leading to the development of bacteremia. Another possibility is that the GI barrier was disrupted by her chronic autoimmune disease, leading to bacteremia. Vancomycin may have played a role in the growth of *L mesenteroides*.

In conclusion, *Leuconostoc* bacteremia should be suspected in a patient with immunosuppression, prolonged use of vancomycin, skin trauma, and/or GI tract pathology. Because of the widespread use of vancomycin, *Leuconostoc* infection may be a cause for increasing concern in the infectious diseases specialty.

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