

[CASE REPORT]

Leuconostoc lactis- A Rare Cause of Bacterial Meningitis in an Immunocompromised Host

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Abstract:

Leuconostoc lactis, often found in fermented dairy products, although considered to have a low pathogenic potential, can cause life-threatening infections in immunocompromised hosts. We herein report a 62-year-old man with a history of alcoholic liver cirrhosis, hepatocellular carcinoma, and diabetes mellitus who developed a very rare case of bacterial meningitis caused by this organism. After we administered antibiotics including ampicillin, he recovered completely within two weeks. This gram-positive coccus (GPC) is sensitive to ampicillin but naturally resistant to vancomycin, while its susceptibility to ceftriaxone has not yet been established. In acute GPC meningitis in immunocompromised hosts, *Leuconostoc lactis* should therefore be considered as a possible pathogen.

Key words: *Leuconostoc lactis*, bacterial meningitis, immunocompromised

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Introduction

Leuconostoc lactis is a catalase-negative Gram-positive coccus (GPC) that is often found in foods, particularly in fermented dairy products (e.g., cheese, yogurt) (1). Although considered to have a low pathogenic potential, it can cause life-threatening infections in immunocompromised hosts. We herein report a case of meningitis caused by *Leuconostoc lactis* in an immunocompromised patient.

Case Report

A 62-year-old Japanese man was brought to the emergency department due to an acute onset of headache and a disturbance of consciousness. He had a past medical history significant for type 2 diabetes mellitus, alcoholic liver cirrhosis (Child-Pugh class B), and hepatocellular carcinoma with intrahepatic metastases treated by transcatheter arterial chemoembolization. He did not complain of any symptoms until he went to bed 6 hours before admission. On arrival, his vital signs were as follows: temperature, 39.7°C; blood pressure, 166/78 mmHg; pulse rate, 122/min; respiration,

36/min. He was lethargic and disoriented with a Glasgow Come Scale score of 6/15. His pupils were equal at 4 mm and reactive to light. Physical examination revealed severe nuchal rigidity and positive for Brudzinski's and Kernig's signs, but no seizures, paralysis, or other neurological deficits were observed. Bilateral mandibular lymphadenopathy, conjunctival icterus, and spider angiomas on his trunk were also noted. Brain magnetic resonance imaging and chest radiographs showed no abnormality. A blood work-up revealed albumin of 3.0 g/dL, total bilirubin of 1.8 mg/dL, serum glucose level of 240 mg/dL, and C-reactive protein of 0.46 mg/dL. The total leucocyte count was $6.9 \times 10^4/\mu\text{L}$ and the differential percentage of neutrophils was 92%. A cerebrospinal fluid (CSF) analysis revealed xanthochromia, pleocytosis (343 cells/ μL , 96% neutrophils), and an elevated protein level (220 mg/dL) with a decreased CSF-to-serum glucose ratio (0.46, CSF glucose level of 112 mg/dL). CSF Gram staining showed Gram-positive cocci in chains (Figure). *Streptococcus pneumoniae* antigen testing of urine and CSF were negative. We diagnosed the patient to have bacterial meningitis and administered intravenous meropenem, vancomycin, and ampicillin immediately. On hospital day 3, *Leuconostoc lactis* was detected in both blood and CSF cul-

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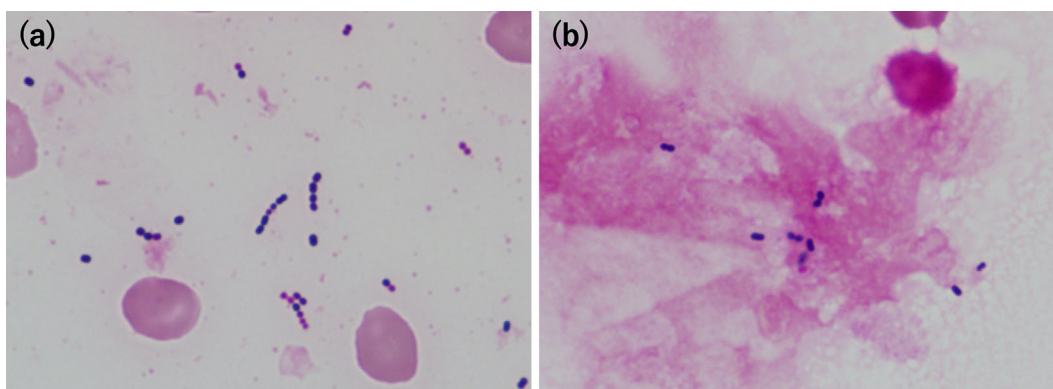


Figure. Gram staining of the cerebrospinal fluid (a) and blood (b). *Leuconostoc lactis* appear as Gram-positive diplococci and chains.

tures. An antibiotic susceptibility test showed this organism was susceptible to ampicillin (minimum inhibitory concentration of 1 µg/mL) according to the Clinical and Laboratory Standards Institute guidelines (2). As a result, we changed the antibiotic regimen to ampicillin monotherapy. His symptoms gradually improved, and a CSF analysis results returned within the normal range on hospital day 9. The patient was discharged home with no symptoms after two weeks of antibacterial treatment.

Discussion

Leuconostoc species are considered to be of low pathogenic potential for healthy individuals; however, they have recently been regarded as pathogens for immunocompromised hosts, causing fatal infections, such as sepsis or meningitis (3, 4). In our case, the patient was thought to be predisposed to this organism because he had diabetes mellitus, severe cirrhosis, and cancer. Although the detailed route of entry is not clear, in patients with liver cirrhosis, bacterial translocation is known to occur due to increased intestinal permeability and intestinal immune abnormality, and this mechanism is one of the major causes of systemic infections (5). Additionally, this patient was accustomed to having yogurt which may have contained this organism. Taken together, we speculate that the entry point was the gastrointestinal tract.

Antibiotic selection is crucial for the treatment of infections caused by *Leuconostoc lactis*. This organism is usually susceptible to penicillin, but unlike other GPCs, it is characteristically resistant to vancomycin because it produces peptidoglycan precursors ending in D-Ala-D-Lac (6). In acute meningitis caused by the GPC chain, ceftriaxone is often administered assuming *S. pneumoniae*. However, the susceptibility of *Leuconostoc species* to ceftriaxone is unestablished, and a ceftriaxone resistant strain has been reported in a case of ventriculitis caused by *Leuconostoc lactis* (7). Therefore,

it may be better to add ampicillin for the treatment of GPC meningitis occurring in immunocompromised patients.

This case highlights the importance of considering *Leuconostoc lactis* as a possible pathogen in cases of acute meningitis caused by GPCs in immunocompromised hosts, since it would enable prompt adequate treatment and lead to better clinical outcomes in these patients.

The patient provided his informed consent for publication of his data.

The authors state that they have no Conflict of Interest (COI).

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