Infectious Endocarditis Caused by *Lactobacillus acidophilus* in a Patient with Mistreated Dental Caries

Takeshi Nishijima, Katsuji Teruya, Mikio Yanase, Yuichi Tamori, Kazuhiro Mezaki and Shinichi Oka

**Abstract**

We present a rare case of infectious endocarditis caused by *Lactobacillus acidophilus* in a patient on long-term steroid use for autoimmune hepatitis. *In vitro* susceptibility-guided antibiotics with benzylpenicillin plus clindamycin and successive mitral annuloplasty resulted in a favorable outcome. Infectious endocarditis was suspected to be a complication of mistreated periodontal infection. Maintenance of oral hygiene is important in immunocompromised patients.

**Key words:** infectious endocarditis, lactobacillus species, immunocompromised, dental infection

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**Introduction**

*Lactobacilli* are commensal bacteria in the human oral cavity, gastrointestinal tract, and female genital tract. Lactobacilli are widely used in food and as probiotics, and are effective in the treatment of infantile and adult diarrhea and antibiotic-associated diarrhea (1, 2). However, rare cases of lactobacilli-induced bacteraemia, meningitis, or endocarditis have been reported particularly in immunocompromised patients (3, 4). Here we report a case of infectious endocarditis caused by *Lactobacillus acidophilus* in a patient with autoimmune hepatitis.

**Case Report**

A 28-year-old female of East Asian origin was hospitalized in our clinic with a 3-month history of fever and pain in the right foot. She had a history of autoimmune hepatitis and had been taking prednisolone (2.5 mg/day) for more than one year. She had also been treated for dental caries, but she stopped seeing the dentist six months before admission. There was no history of cardiac disease, intravenous drug use or intake of probiotics. Since the appearance of fever, the patient was treated with levofloxacin (500 mg/day). On admission, the patient was alert and oriented. The body temperature was 38.3°C. Physical examination showed a painful nodule in the 5th toe of the right foot. Oral examination showed multiple caries. Cardiac auscultation revealed grade 3/6 systolic murmur loudest over the apex, which was not evident during hospitalization 3 years earlier. Blood test showed leukocytosis (8,910/μL, neutrophil 87.4 %), microcytic anaemia with hemoglobin of 9.9 g/dL, thrombocytopenia (platelet count: 102,000/μL), and elevated C reactive protein of 3.91 mg/dL. Liver function was relatively maintained with serum albumin 3.9 g/dL, total bilirubin 1.1 mg/dL, and prothrombin activity 75.1%. Liver transaminase and lactate dehydrogenase were within the normal ranges. Rheumatoid factor was positive, and anti-phospholipid antibody, anti-nucleus antibody, and anti-neutrophil cytoplasmic antibody were negative. Erythrocyte segmentation rate was 51 mm/hr.

Computed tomography of the abdomen showed a hyperdense, wedge-shaped splenic lesion, suggestive of splenic infarction. There were heterogeneous hypointensities areas in the liver, but no ascites. Whole-body fluorodeoxyglucose positron emission tomography revealed high uptake in the 5th toe of the right foot, but no uptake in the splenic infarct.
area. Transesophageal echocardiography showed mitral prolapse with third-degree mitral regurgitation but no valvular vegetation was found. Dental consultation indicated multiple apical periodontitis and dental treatment was commenced.

All four culture bottles of two sets of blood samples drawn the day before admission showed Gram positive rods (GPR) on day 3. The patient was treated with intravenous antibiotics (meropenem 2 g plus clindamycin 2,400 mg). Another set of four culture bottles of two blood samples taken on day 3 also revealed GPR. \textit{L. acidophilus} was identified on day 8, and the isolate was susceptible to benzylpenicillin (minimum inhibitory concentration [MIC] 0.12 μg/mL), ampicillin (MIC 0.25 μg/mL), imipenem (MIC ≤ 0.25 μg/mL) and clindamycin (MIC ≤0.12 μg/mL) (Fig. 1).

MIC was measured with Etest® for penicillin according to the Clinical and Laboratory Standards Institute guidelines, and others with the microbroth dilution method [ (5); Fig. 2]. On day 9, the antibiotics were replaced with benzylpenicillin (24 million units) plus clindamycin (2,400 mg). The final diagnosis was infectious endocarditis caused by \textit{L. acidophilus} with Osler’s node in the right foot and splenic infarction.

One anaerobic culture bottle of two sets of blood samples taken on day 6 revealed \textit{L. acidophilus}, but no growth was observed in blood cultures prepared on day 14 and thereafter. Computed tomography on day 32 confirmed the disappearance of the nodule in 5th toe of the right foot. She was discharged on day 42 after 6 weeks of intravenous antibiotics treatment, and continued to take clindamycin 1,800 mg orally for 6 more weeks. Mitral annuloplasty was performed one year later, and no relapse has been observed for 2 years.

**Discussion**

We reported a rare case of infectious endocarditis caused by \textit{L. acidophilus} in a patient on long-term steroid use for autoimmune hepatitis. To our knowledge, only 28 cases (3 cases in Japan), including this case, of infectious endocarditis caused by lactobacilli have been reported since 1992 after the publication of Duke’s criteria (4, 6-9).

Endocarditis was diagnosed based on modified Duke’s criteria (9). This patient fulfilled one major criterion: new valvular regurgitation, and four minor criteria: fever, major arterial embolus (splenic infarction), immunologic phenomenon (Osler’s node in one toe), and microbiological evidence (positive blood culture).

Since the data for treatment and susceptibility of lactobacillus is scarce, there is no recommended empiric therapy (4). Salminen et al. indicated that the choice of antibiotics should depend on the susceptibility testing of lactobacilli, and that therapy guided by \textit{in vitro} susceptibility tests significantly reduced mortality (10). Accordingly, we chose the combination therapy of intravenous benzylpenicillin plus clindamycin for 6 weeks and thereafter, clindamycin orally for 6 weeks. Mitral annuloplasty was conducted for severe mitral regurgitation, and no relapse was observed during the 2-year post-discharge period.

Maintenance of oral hygiene is important in immunocompromised patients in order to prevent systemic infection (11). Cannon et al. reported that dental procedures or oral diseases are the predisposing condition in almost half of the patients with lactobacillus-induced endocarditis (3). The present patient developed endocarditis caused by \textit{L. acidophilus} after she discontinued dental treatment. Thus, dental infection was highly likely the predisposing factor for endocarditis.

In summary, we presented a case of infectious endocarditis caused by \textit{L. acidophilus} in a patient with autoimmune hepatitis. Long-term use of \textit{in vitro} susceptibility-guided antibiotics and mitral annuloplasty led to a favorable outcome. Maintenance of oral hygiene is important in immunocompromised patients.

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

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