

Pancreatic Necrosis Infection due to *Lactobacillus paracasei* in an Immunocompetent Patient

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Key Words

Acute pancreatitis · *Lactobacillus paracasei* subspecies *tolerans* · 16S rDNA sequencing · Antibiotic prophylaxis, acute pancreatitis · Antibiotic sensitivity, *Lactobacillus* spp. · Pancreatic necrosis infection

Abstract

An immunocompetent patient recovering from gallstone-induced pancreatitis had to be readmitted due to abdominal pain, fever, and rapid deterioration. Computed tomography guided needle aspiration established the diagnosis of pancreatic necrosis infection; microbiological investigations revealed mono-infection with *Lactobacillus paracasei* subspecies *tolerans*. To our knowledge, this is the first description of a pancreatic necrosis infection due to *L. paracasei* in an immunocompetent patient without any known risk factors for *Lactobacillus* infections.

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Introduction

Any acute pancreatitis (AP) progresses in about 15–20% of the patients to severe pancreatic necrosis which is prone to infection. The infection rate is related to the size

of pancreatic and peripancreatic necrosis, typically occurs during the 2nd or 3rd week after onset of AP, and is associated with a high mortality (20–30%). The final diagnosis of a pancreatic necrosis infection is based on computed tomography (CT) guided needle aspiration and subsequent microbiological workup which identifies predominantly gram-negative bacteria deriving from the intestine, such as *Staphylococcus aureus* or *Staphylococcus epidermidis*, but also *Candida* spp. We report an unusual case of pancreatic necrosis infection caused by *Lactobacillus paracasei* in an immunocompetent patient.

Case Report

After a previous transient episode of colic-like epigastric pain, an otherwise healthy 52-year-old man developed persistent epigastric pain, nausea, and vomiting, leading to hospital admission. Based on physical examination, laboratory investigations, and CT, the diagnosis of gallstone-induced pancreatitis was made. Acute endoscopic retrograde cholangiopancreatography and papillotomy were not advisable due to an edematous ampulla, and conservative treatment consisting of parenteral fluid and electrolyte replacement, food restriction, and analgesia was started. On the 8th day of hospitalization, the patient became subfebrile paralleled by leukocytosis. Based on the presumptive diagnosis of an early pancreatic infection, empiric intravenous antibiotic therapy with imipenem was started. Over the next 8 days, the patient became asymptomatic, and the laboratory parameters normalized, so that imipenem was stopped. Before hospital discharge, abdominal ultrasonography was performed, demonstrating

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gallstones without evidence of pancreatic necrosis, calcifications, or a dilated pancreatic duct. A contrast-enhanced CT scan, which might have provided further information about presence or absence of pancreatic necrosis, was not available at this time.

Five days later, the patient was readmitted because of severe abdominal pain. On physical examination, the patient was febrile (tympenic temperature 39.6°C) and had tachycardia (104/min) and abdominal tenderness. Laboratory investigations disclosed leukocytosis of 30.1 G/l, a slightly reduced sodium level of 132 mmol/l, and elevations of α -amylase to 166 IU/l and of C-reactive protein to 283 mg/l. The CT scan was repeated and showed pancreatic necrosis of 10 × 4 × 10 cm in the region of the pancreas corpus, fluid collections, and peripancreatic inflammation. CT-guided needle aspiration of the necrotic area was performed. Microbiological investigations revealed numerous gram-positive rods. Based on these findings, the diagnosis of a pancreatic necrosis infection was confirmed, and antibiotic therapy with imipenem was restarted. In culture, *L. paracasei* subspecies *tolerans* was identified which was further confirmed by sequence analysis of the 16S rDNA. Antibiotic sensitivity testing showed resistance to penicillinase-resistant penicillins, clindamycin, and clarithromycin. Under imipenem, the patient made a good recovery over the following 3 weeks and could be discharged from the hospital. Débridement was planned 6 weeks later.

Discussion

Lactobacilli are anaerobic or facultative anaerobic gram-positive rods that belong to the normal flora of gastrointestinal tract and vagina. Infections are most often seen in patients with an immunosuppressive disease, and,

so far, only very few cases have been reported in immunocompetent patients [1]. Besides immunosuppression, previous treatment with antibiotics ineffective against lactobacilli and previous surgery [2–4] represent known risk factors for *Lactobacillus* infections. Reported infectious manifestations include bacteremia, endocarditis, meningitis, pneumonia, liver and splenic abscesses, urinary tract infections, chorioamnionitis, and chondritis [1–9]. The presented case is unique for several reasons: it is the first report of a pancreatic necrosis infection due to *L. paracasei*, the patient was not immunocompromised, and none of the other known risk factors for *Lactobacillus* infections was present. Since antibiotic sensitivity testing confirmed susceptibility of the isolated *L. paracasei* to imipenem, infection on the basis of germ selection induced by the first antibiotic treatment course is highly unlikely. However, antibiotic prophylaxis in severe AP remains controversial. While several previous studies [10–14] suggested that prophylaxis using broad-spectrum antibiotics significantly reduces the infection rate in severe AP, a recent study [15] showed no benefit of antibiotic prophylaxis.

In conclusion, the case presented demonstrates that also in immunocompetent patients unusual causes of pancreatic necrosis infection, even by a relatively avirulent germ, such as *L. paracasei*, have to be considered in the differential diagnosis.

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