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Leclercia adecarboxylata Bacteremia without a Focus in a Non-Immunosuppressed Patient

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Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Patient: Female, 74-year-old
Final Diagnosis: Leclercia adecarboxylata bacteremia
Symptoms: Cough • fever • shock • shortness of breath
Medication: —
Clinical Procedure: —
Specialty: Infectious Diseases • Microbiology and Virology

Objective: Rare disease

Background: *Leclercia adecarboxylata* is a gram-negative rod, which is normally found in water and food. It is an emerging pathogen that affects immunocompromised patients, including patients with hematological malignancies or those receiving chemotherapy. Generally, *L. adecarboxylata* is considered a low-virulence pathogen with an excellent susceptibility profile, but some strains may be resistant to multiple antibiotics, such as β-lactams. Moreover, *L. adecarboxylata* is usually isolated as a part of polymicrobial cultures in immunocompetent individuals, but there have been cases where it was the only isolate.

Case Report: A 74-year-old woman who was non-immunosuppressed and had multiple comorbidities was admitted with acute decompensated heart failure due to pneumonia. She was treated with multiple courses of antibiotics including amoxicillin-clavulanate and ciprofloxacin for pneumonia, but her infection worsened, and she had cardiopulmonary arrest. After resuscitation, she was stable for several days but suddenly became confused and hypotensive. The septic screen showed *L. adecarboxylata* bacteremia without a clear source, which was treated successfully with meropenem for 14 days. After the meropenem course, the patient developed diarrhea and was found to have severe *Clostridium difficile* infection. She did not respond to oral vancomycin and intravenous metronidazole and died.

Conclusions: This case illustrated an infection in a non-immunosuppressed individual by an organism that is considered an opportunistic pathogen, mainly affecting immunocompromised patients. The patient's blood culture grew *L. adecarboxylata*, which was sensitive to all antibiotics but resolved with meropenem treatment. Owing to increasing *L. adecarboxylata* infections, we recommend further studies to understand the organism's pathogenesis, risk factors, and resistance pattern.

Keywords: Bacteremia • Bacterial Infections • Carbapenems • *Clostridium difficile* • Immunocompetence

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Background

Leclercia adecarboxylata is a motile, gram-negative rod, which was formerly known as *Escherichia adecarboxylata* [1]. However, deoxyribonucleic acid homology studies led to reclassifying the organism in a new group, and it was named *L. adecarboxylata* [2]. Most commonly, *L. adecarboxylata* is found in water and soil [3]. Generally, *L. adecarboxylata* is sensitive to most antibiotics; however, antibiotic resistance has been reported [4,5].

L. adecarboxylata infections are most often nonfatal owing to its low virulence and good antibiotic susceptibility [6]. The most identifiable risk factor for infection is immunosuppression; however, it was found that *L. adecarboxylata* could be part of polymicrobial infections in immunocompetent patients [7], particularly when the culture is taken from a wound [8]. Conversely, *L. adecarboxylata* has been reported as a sole isolate from blood, sputum, urine, and peritoneal fluid [9-12]. *L. adecarboxylata* might be disregarded in the context of polymicrobial infection because of its low virulence and ubiquity.

There are a wide variety of treatment options for *L. adecarboxylata*. However, its resistance to fosfomycin is commonly reported [4]. Resistance to other antibiotics such as trimethoprim-sulfamethoxazole, aminoglycosides, penicillin, and cephalosporins have also been reported [7]. Almost all cases of *L. adecarboxylata* infection are sensitive to carbapenems, amikacin, tetracycline, and tigecycline [7].

Here, we are reporting a case of isolated *L. adecarboxylata* bacteremia in a non-immunosuppressed patient. The aim of this study is to narrate the course, management, and outcome of such cases.

Case Report

A 74-year-old woman presented to the emergency department with progressive shortness of breath and productive cough for 1 week, along with increasing abdominal distention. She had a history of diabetes mellitus (with latest A1C 7.1%), hypertension, chronic kidney disease, heart failure with preserved ejection fraction, coronary artery bypass graft, peripheral vascular disease, and right above-knee amputation.

The patient had progressive dyspnea, associated with orthopnea, paroxysmal nocturnal dyspnea, and lower-extremity swelling. Her cough was productive with yellowish sputum, but she had no fever. The patient had recurrent abdominal ascites secondary to heart failure (serum ascites albumin gradient of 1.5 g/L and ascitic protein of 26 g/L) for 6 months prior to presentation, which required weekly abdominal paracentesis. She was conscious and oriented, and her vital signs were stable. The examination did not show any stigmata of chronic liver disease. A cardiac examination revealed a pansystolic murmur in the left lower sternal border, while the lung examination showed fine bilateral lung crepitations. She had generalized abdominal tenderness, with tense ascites and bilateral lower-extremity pitting edema. The patient also had a stage 2 sacral ulcer, which was not infected.

The initial laboratory test results showed a normal white blood cell (WBC) count of 5.9×10^9 g/L. The chest X-ray at admission showed bilateral perihilar and right basilar airspace opacities, with right pleural effusion. She was admitted for acute decompensated heart failure secondary to pneumonia.

The patient was admitted to the cardiac care unit and was administered furosemide 40 mg IV twice per day and amoxicillin-clavulanate. Six days after admission, the patient had a temperature of 38.9°C, with diffuse abdominal pain. Blood laboratory results showed an elevated WBC count of 13.7×10^9 g/L (reference range, 4.0-11.0×10^9), neutrophil count of 11.0×10^9 (reference range, 2.0-7.5×10^9), lymph count of 0.96×10^9 (reference range, 1.5-4.0×10^9), C-reactive protein level of 31.3 mg/L (reference range, 0.0-5.0 mg/L), and procalcitonin level of 0.42 ug/L (reference range, <0.25 ug/L). The patient was still febrile and not responding to amoxicillin-clavulanate, and was therefore switched to IV ciprofloxacin to treat the sepsis. A few hours later, the patient went into cardiac arrest. Cardiopulmonary resuscitation was performed for 3 minutes, a peripherally inserted central catheter (PICC) was inserted, and the patient was intubated.

After 3 days, the patient was extubated and paracentesis was performed. Ascitic fluid culture results were negative but had a cell count of 196 leukocytes/µL. Two days later, the patient became confused and hypotensive and a laboratory examination showed that the inflammatory markers were elevated. A full septic screen was done and exhibited no organisms in the urine, sputum, or cerebrospinal fluid; however, a superficial swab from the sacral ulcer, which did not show signs of infection (**Figure 1**), grew *Escherichia coli* and *Klebsiella pneumoniae*, which were considered contaminants. A blood culture was taken using aerobic and anaerobic BACT/ALERT blood culture bottles (BioMérieux, Marcy l'Étoile, France) and was incubated in the BACT/ALERT3D blood culture machine. The culture was detected as positive after 18 h of incubation. A Gram stain from blood culture demonstrated gram-negative rods (**Figure 2**). The blood was subcultured on blood, MacConkey, and chocolate agar, and incubated at 37°C overnight. The agar plate showed grey, large mucoid colonies with weak lactulose fermentation (**Figure 3**). The organism was identified as *L. adecarboxylata* using the Vitek-MS microbial identification system (BioMérieux, France), and sensitivity was performed on the Vitek-2 sensitivity machine (BioMérieux, France) following

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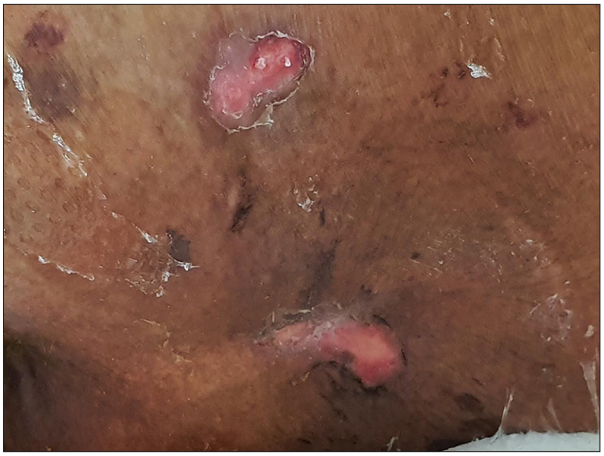

Figure 1. Stage 2 sacral ulcer without signs of an infection.

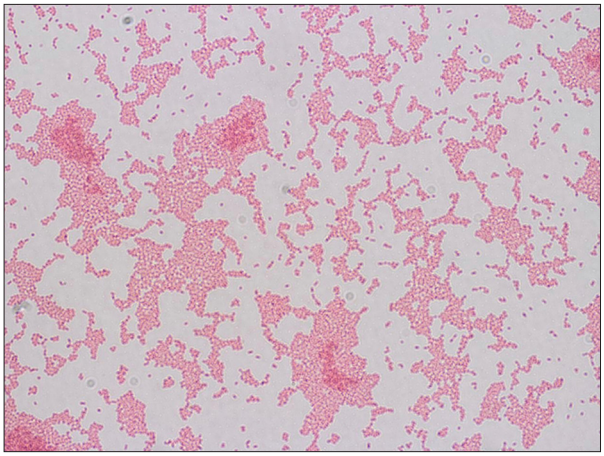

Figure 2. Gram stain: Gram-negative rods.

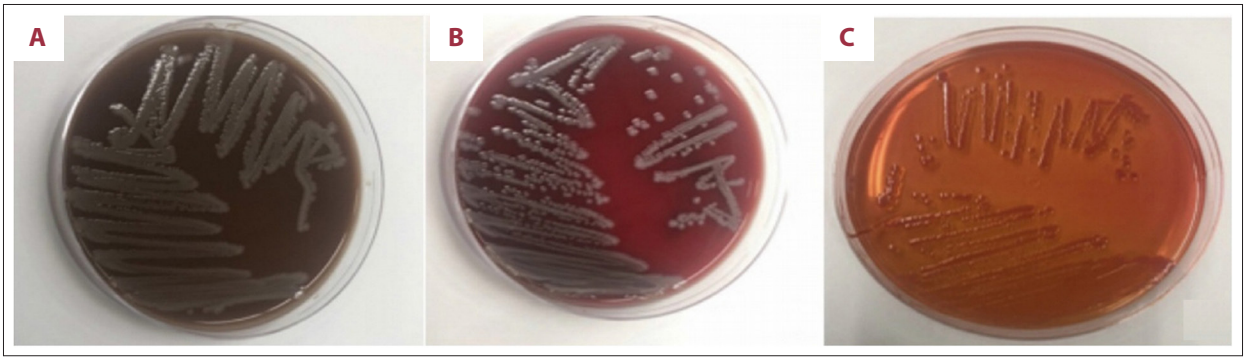

Figure 3. (**A**) Chocolate and (**B**) blood agar: mucoid, grey large colonies. (**C**) MacConkey agar: weak lactose fermentation.

Table 1. Antibiotics sensitivity to *Leclercia adecarboxylata* in the blood culture.

Antibiotics	Sensitivity	MIC
Ampicillin	Sensitive	≤2
Ciprofloxacin	Sensitive	≤0.25
Gentamicin	Sensitive	≤1
Meropenem	Sensitive	≤0.25
Trimethoprim/ sulfamethoxazole	Sensitive	≤20

MIC – minimum inhibitory concentration.

the manufacturer's instructions (**Table 1**). The abdominal pigtail catheter and PICC line were removed after the blood culture results were obtained, and ciprofloxacin was changed to meropenem, as the patient was not improving.

After 48 h of meropenem, her fever abated, inflammatory markers improved, and *L. adecarboxylata* bacteremia cleared. The patient completed 14 days of meropenem, and blood cultures were negative for organisms at the end of treatment. Five days before finishing the course of meropenem, the patient developed watery diarrhea, and *Clostridium difficile* was detected. The patient was administered oral vancomycin, and later IV metronidazole was added. Her diarrhea did not improve, her WBC count reached 49.7×10^9 g/L, and she required inotropic support to maintain her blood pressure. An abdominal computed tomography (CT) scan showed a circumferential thickening involving the distal transverse colon to the rectum, indicating colitis (**Figure 4**). The patient's condition worsened over the following days (22 days after detecting *C. difficile*) until she died due to severe colitis.

Discussion

L. adecarboxylata is a flora of the gastrointestinal tract [13]. Initial isolates by Leclerc were from water, but later, *L. adecarboxylata* was commonly reported in the environment, especially in food and water [14-16].

In case reports and series, *L. adecarboxylata* was isolated from a large variety of specimens, including wounds, feces, urine, gallbladder, peritonsillar and periovarian abscesses, synovial fluid, peritoneal fluid in peritoneal dialysis, nosocomial

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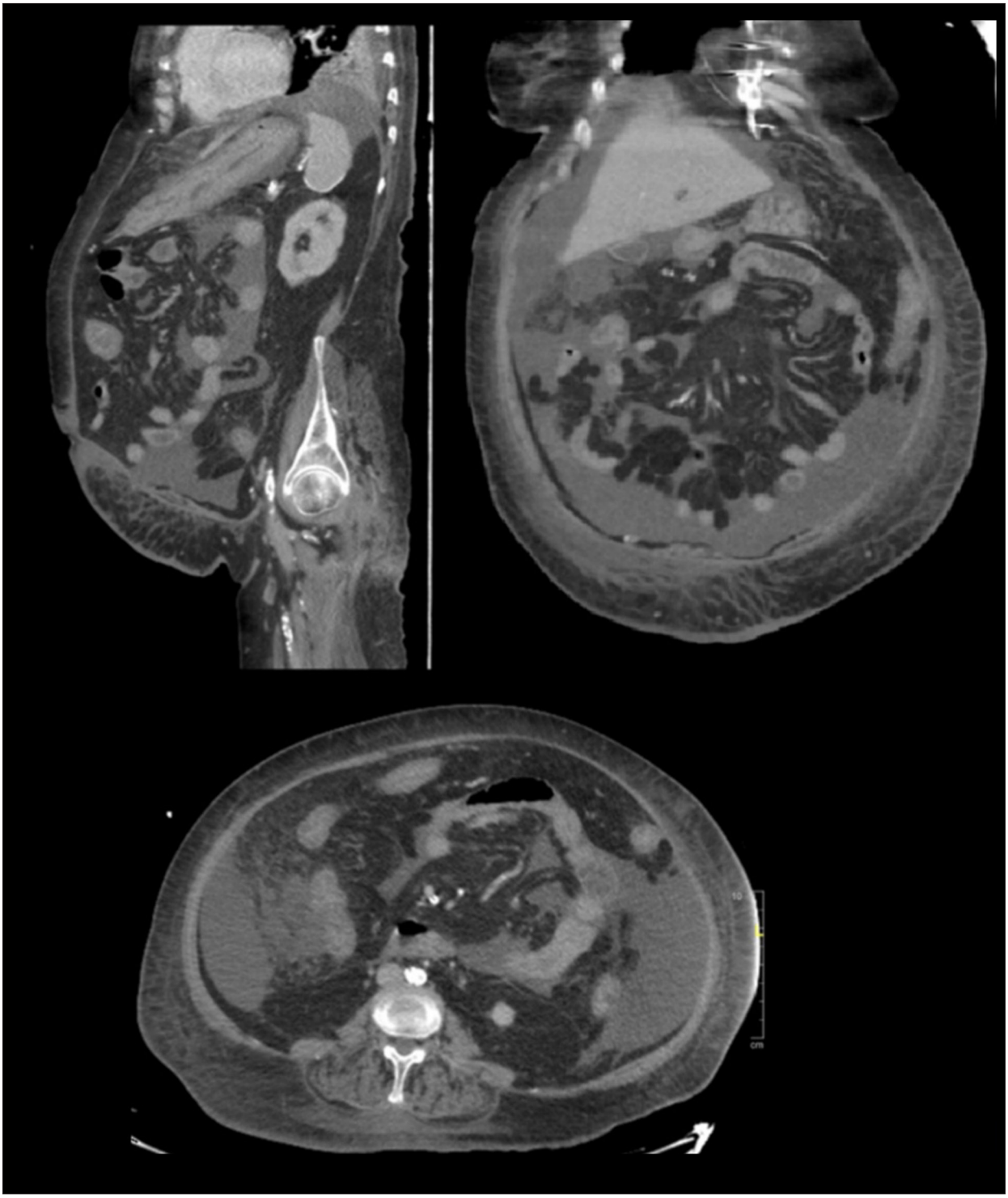

Figure 4. Abdominal and pelvis computed tomography scans showing circumferential wall thickening of the colon and rectum, prominent in the left transverse colon, and abdominopelvic free fluid with no free air.

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pneumonia, and bacteremia [17-19]. *L. adecarboxylata* is an uncommon pathogen and is usually isolated as a part of polymicrobial wound cultures; it is postulated that it needs other bacteria to facilitate infection [13,17,20]. It might be the only isolate in immunocompromised patients, as Hess et al reported in patients with cirrhosis and hematological malignancies, and in those receiving chemotherapy [13].

Bacteremia caused by *L. adecarboxylata* is usually associated with immunosuppression and the presence of central lines. Monomicrobial bacteremia in the case of our patient was puzzling because her controlled diabetes and other comorbid conditions were not consistent with the pattern of immunosuppression found in the literature. Although diabetes may affect the body's immunity, that usually occurs in the context of poor glycemic control [21]. In the literature, patients with *L. adecarboxylata* infection usually also have severe immunosuppression, for example, through the use of immunomodulators or chemotherapy [7,22,23]. On the other hand, few cases with diabetes as the only risk factor had skin and soft tissue infection [24,25]. Our patient had a PICC line for a few days, which could have been the source; however, only a peripheral blood culture was positive, contradicting this theory. Another possible source could have been iatrogenic peritonitis; however, all patients reported to have *L. adecarboxylata* peritonitis had peritoneal dialysis catheters, which our patient did not have. Although, skin commensals are much more likely to present in this setting. The third possibility is translocation from the gut, secondary to the patient's *C. difficile* infection, which became symptomatic after the positive culture. The source of bacteremia in a 51-year-old woman who presented with nausea and vomiting and had small bowel thickening on an abdominal CT was presumed to be the gastrointestinal tract. That patient, however, had end-stage renal disease and was on hemodialysis [26]. Nonetheless, a lack of focus has been described in cases of *L. adecarboxylata* bacteremia [27]

L. adecarboxylata isolates are reported to have a good sensitivity profile, and, in general, its resistance pattern is similar to that of other Enterobacteriaceae. However, fosfomycin resistance is more common with *L. adecarboxylata*. There are reports of resistant isolates exhibiting SHV-12 β-lactamase production; thus, they are behaving like an extended-spectrum β-lactamase organism [5]. Another study looked at hospital staff hand hygiene compliance and isolated VIM-1 Metallo-β-Lactamase producers [28]. The isolates were resistant to β-lactams, including carbapenems, and were susceptible to aztreonam. However, these were not clinical samples from patients.

There are no guidelines discussing treatment, but good response to β-lactams and fluoroquinolones was observed in multiple reports [7,12]. Our patient cleared her bacteremia with a brief course of meropenem, suggesting this antibiotic might be a good option to treat *L. adecarboxylata* infection. Nonetheless, the lack of identifiable source of the bacteremia can be considered as a limitation of the presented case.

Conclusions

L. adecarboxylata infection is not exclusive to immunocompromised patients. We reported a case of *L. adecarboxylata* bacteremia in a non-immunosuppressed patient. The organism was detected in the blood despite the patient being on multiple antibiotics, to which the organism was sensitive. Meropenem showed good activity against *L. adecarboxylata*. Further studies to understand the pathogenesis, risk factors, and resistance pattern of *L. adecarboxylata* are recommended, as it has become an emerging pathogen in the last decade.

Conflicts of Interest

None.

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