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Case Report

Cedecea Lapagei Bacteremia Resistant to Carbapenem and Co-Infection with Acinetobacter Baumannii: A Rare Case Report from Taiwan

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SUMMARY

Cedecea is a rare genus of bacteria within the Enterobacteriaceae family. We reported a case of an 81-year-old man with jejunal obstruction caused by adenocarcinoma, who developed septic shock and hypercapnic respiratory failure after surgery. Cultures from his blood and central venous catheter revealed Acinetobacter baumannii complex and Cedecea lapagei.

While the Acinetobacter baumannii complex showed no resistance to the tested antibiotics, the Cedecea lapagei was resistant to polymyxin E and imipenem but sensitive to ertapenem, fluoroquinolones, aminoglycosides, and novel β-lactam/β-lactamase inhibitor combinations. The patient's persistent fever was effectively treated with ciprofloxacin and cefoperazone/sulbactam.

This case is the first report of Cedecea lapagei bacteremia in Taiwan, highlighting its potential as a nosocomial opportunistic pathogen with multiple antimicrobial resistance.

1. Introduction

Cedecea is a genus of rare bacteria within the Enterobacteriaceae family. The name of this genus is derived from the letters CDC, which stands for the Centers for Disease Control and Prevention in Atlanta, where the organism was originally discovered in 1981.[1] These bacteria are characterized as Gram-negative, non-encapsulated, non-sporing, lipase-positive, catalase-positive, oxidase-negative, lactose-negative, and facultative anaerobic.

Currently, the Cedecea genus includes three recognized species of clinical significance: C. davisae, C. lapagei, and C. neteri, as well as three unnamed species.[2] C. lapagei can be distinguished from other Cedecea strains by its ability to grow in media that lacks thiamine and can be rapidly identified in the Voges–Proskauer test using the O'Meara method.[2]

There have been few reported cases of Cedecea infections, which exhibit varying clinical presentations, drug susceptibilities, and treatment responses. As of January 2023, there have been twenty reported cases of human infections with C. lapagei worldwide.[3] To the best of our knowledge, no clinical cases of Cedecea infection have been reported in Taiwan.

Acinetobacter baumannii complex and C. lapagei are opportunistic pathogens commonly linked to hospital-acquired infections, particularly in critically ill or immunocompromised patients. Concurrent infections with both pathogens are rare and present significant clinical challenges. This case report describes an elderly patient who developed bacteremia due to these two pathogens following surgery for jejunal obstruction caused by adenocarcinoma. The article also reviews the existing literature on the management of C. lapagei infections.

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2. Case report

An 81-year-old male with no significant medical history was admitted to the hospital due to postprandial nausea, vomiting, significant weight loss (from 85 kg to 65 kg), and poor appetite over the past two months. Imaging revealed a jejunal obstruction likely caused by malignancy.

The patient was diagnosed with adenocarcinoma at the junction of the fourth portion of the duodenum and jejunum, with liver metastasis. On the 18th day of admission, he underwent surgery, including enterolysis, tumor resection, intestinal anastomosis, and a liver biopsy. His tumor staging was pT3N1M1, stage IV.

Despite being placed on Flomoxef for surgical prophylaxis, he developed septic shock and hypercapnic respiratory failure on day 4, with leukopenia (white blood cell 2.4×10^3/uL, band 2%, segment 78%). He required mechanical ventilation and intensive care unit (ICU) admission. Blood and central venous catheter tip cultures identified Acinetobacter baumannii complex and Cedecea lapagei, the latter resistant to polymyxin E and imipenem but sensitive to ertapenem, fluoroquinolones, aminoglycosides, and novel β-lactam/β-lactamase inhibitor combinations. The Acinetobacter baumannii complex showed no resistance to the tested antimicrobials (Table 1). Our bacterial identification was conducted using the VITEK® MS PRIME Matrix Assisted Laser Desorption Ionization-Time of Flight (MALDI-TOF) system, while antimicrobial susceptibility testing was performed with the VITEK® 2 automatic system (BioMerieux, USA). However, 16S ribosomal RNA sequencing for further identification was not available at our hospital.

The patient was initially treated in the ICU with Meropenem and Teicoplanin; however, his fever persisted. Two days later, upon receiving the antimicrobial sensitivity test results, Ciprofloxacin was prescribed. When this treatment did not lead to improvement, a

Table 1
Antimicrobial susceptibility testing using the VITEK® 2 COMPACT system (bioMérieux, France) followed CLSI guidelines (M100, 2024).

Result of antimicrobial susceptibility tests		
Blood culture – for catheter lines		
1. Acinetobacter baumannii complex 2. Cedecea lapagei		
	1. MIC	2. MIC
Ceftazidime	S (2)	S (≤ 0.12)
Ciprofloxacin	S (0.25)	S (≤ 0.06)
Colistin	S (≤ 0.5)	R (≥ 16)
Cefpirome	S (4)	S (≤ 1)
Gentamicin	S (≤ 1)	S (≤ 1)
Imipenem	S (≤ 0.25)	R (8)
Tigecycline	S (≤ 0.5)	S (≤ 0.5)
Cefoperazone/Sulbactam	S (≤ 8)	S (≤ 8)
Ampicillin	-	R (≥ 32)
Amikin	-	S (≤ 2)
Cefotaxime	-	S (≤ 0.25)
Cefuroxime	-	I (16)
Ertapenem	-	S (≤ 0.12)
Flomoxef	-	I (32)
Cefoxitin	-	R (≥ 64)
Levofloxacin	-	S (≤ 0.12)
Trimethoprim/Sulfamethoxa	-	S (≤ 20)
Cefazolin (urine)	-	R (≥ 64)
Cefazolin (others)	-	R (≥ 64)
Ceftolozane/Tazobactam	-	S (≤ 0.25)
Ceftazidime/Avibactam	-	S (≤ 0.12)

MIC (Minimum inhibitory concentration) (unit: ug/ml).
Abbreviations: CLSI, Clinical & Laboratory Standards Institute; I, intermediate; R, resistant; S, susceptible.

combination of Ciprofloxacin and Cefoperazone/Sulbactam was initiated due to a suspected anaerobic infection resulting from intra-abdominal complications.

Within 48 hours of starting this combination therapy, the patient's fever subsided, and his clinical condition showed significant improvement. He was successfully weaned off the mechanical ventilator after 10 days in the ICU and subsequently transferred to the general ward after a total of 12 days in the ICU. The patient was discharged after spending 15 days in the general ward, during which he received chemotherapy with 5-Fluorouracil and Cisplatin.

3. Discussion

In this case report, we describe a patient with rare adenocarcinoma of the small intestine, which occurs at an incidence of 0.7 cases per 100,000 individuals in the United States. The tumor was located in the jejunum, accounting for 11–25% of rare small bowel adenocarcinomas.[4] The patient experienced significant weight loss and poor oral intake, which severely affected his nutritional status and immune function. Additionally, his advanced age and late-stage tumor increased his risk of surgical complications and nosocomial infections.[5] Notably, this is the first reported case in Taiwan of a concurrent nosocomial infection involving both C. lapagei and the Acinetobacter baumannii complex, highlighting the challenges of managing infections in patients with advanced cancer.

The detection of two Gram-negative pathogens in the blood and at the tip of the central venous catheter led us to suspect that the source of the bacteremia was related to the intestinal surgery rather than a catheter-related skin infection. This may explain why the patient's fever did not improve after the removal of the central venous catheter and why combination therapy with Ciprofloxacin and Cefoperazone/Sulbactam was necessary.

Although it was discovered more than 40 years ago, C. lapagei was designated a human pathogen in 2006.[6] It has been reported to cause community-acquired pneumonia in an immunocompromised patient following a COVID-19 infection.[7] A systemic review conducted in January 2023 revealed 20 clinical infections cause by C. lapagean worldwide.[3] The bacteria were primarily isolated from respiratory specimens, accounting for 40% of the cases (including sputum and bronchoalveolar lavage fluid). Additionally, C. lapagei was found in other sources such as nasal sinuses, blood, lung tissue, knee wound sites, peritoneal dialysis fluid, urine, oral ulcers, bone biopsies, and skin bulla fluid.

Cedecea species are most closely resemble Serratia in terms of lipase positivity and resistance to cephalothin and polymyxin E,[2] with the exception that Cedecea does not hydrolyze deoxyribonucleic acid (DNA) or gelatin. The clinical resistance observed in Cedecea isolates is primarily attributed to the expression of multiple β-lactamases and efflux pumps. Most of the clinical isolates were sensitive to levofloxacin (87.5%), ciprofloxacin (81.3%), cefotaxime (71.4%), trimethoprim/sulfamethoxazole (69.2%), meropenem (61.5%), and amikacin (60%)[3] as reported in previous studies of twenty cases. Cedecea has a natural resistance to colistin (Polymyxin E), likely due to modifications of its lipopolysaccharides resulting from cationic substitutions in its genome, which includes the genes mgrB, phoP, and phoQ. However, no plasmid DNA has been identified, which reduces the likelihood of the presence of MCR-1 (mobilized colistin resistance) originating from plasmids.[8]

Cedecea species are part of the human intestinal microbiome, suggesting a potential endogenous source for infection.[9,10] Their motility is facilitated by peritrichous flagella, which may contribute to adhesion and colonization of the host. The ability to form biofilms is suggested by the presence of quorum sensing (QS), a mechanism that bacteria use to regulate biofilm formation. This has been documented for the C. neteri strain SSMD04.[11,12] These virulence factors may explain why our patient developed Cedecea bacteremia following jejunal resection and adhesion of the bacteria to his central venous catheter.

4. Conclusion

We presented a rare case of bacteremia caused by the Acinetobacter baumannii complex and C. lapagei following the resection of jejunal adenocarcinoma. C. lapagei exhibits intrinsic resistance to polymyxin E and cephalothin, and it should be recognized as an opportunistic pathogen in immunocompromised hosts. Effective treatment requires prompt identification of the pathogens, appropriate microbiological testing, and the use of targeted antibiotic therapy.

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