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Highlights

- Eravacycline shows emerging activity against CRAB in real-world settings
- Eravacycline monotherapy cleared cefiderocol-resistant CRAB bloodstream infection
- Treatment was effective in a critically ill patient with concurrent C. difficile colitis

Successful eravacycline monotherapy for cefiderocol-resistant *Acinetobacter baumannii* bloodstream infection in a critically ill patient with concomitant *Clostridioides difficile* colitis

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Keywords

CRAB; *Acinetobacter baumannii*; Eravacycline; bacteremia; Clostridioides difficile; C. difficile.

Abstract

Carbapenem-resistant *Acinetobacter baumannii* (CRAB) bloodstream infections (BSIs) are associated with high mortality and limited therapeutic options, particularly when resistance to novel agents such as cefiderocol emerges. We report the case of an 87-year-old critically ill woman with multiple comorbidities who developed cefiderocol-resistant CRAB BSI during hospitalization, concomitant with *Clostridioides difficile* colitis. The patient had previously received broad-spectrum antimicrobial therapy and ventilatory support. Cefiderocol resistance was detected by gradient diffusion (MIC Test Strip) on iron-depleted Mueller–Hinton agar, showing an MIC of 12 mg/L. Considering the resistance profile and concurrent *C. difficile* infection, antimicrobial therapy was switched to eravacycline monotherapy (1 mg/kg every 12 hours). Eravacycline susceptibility testing by gradient diffusion (ETEST) demonstrated an MIC of 0.75 mg/L. Treatment was administered for 14 days, resulting in clinical improvement and blood culture sterilization within 96 hours.

Introduction

Carbapenem-resistant *Acinetobacter baumannii* (CRAB) is a critical pathogen responsible for healthcare-associated infections in severely debilitated patients, causing bloodstream infections (BSIs) with high mortality rates [1]. Therapeutic options for CRAB infections remain extremely limited and include cefiderocol, sulbactam/durlobactam, and colistin; however, robust clinical data and randomized trials are scarce, particularly for new agents [2]. Eravacycline, a novel fluorocycline antibiotic approved for complicated intra-abdominal infections, shows emerging activity against CRAB in real-world settings [3–5]. We report a case of cefiderocol-resistant CRAB BSI successfully treated with eravacycline monotherapy in a critically ill patient with concurrent *Clostridioides difficile* colitis.

Case report

In November 2025, an 87-year-old woman was admitted to the sub-intensive care unit for acute dyspnea. Chest computed tomography (CT) revealed bilateral pleural effusions, more pronounced on the right side. Her medical history was significant for chronic obstructive pulmonary disease (COPD), chronic kidney disease, chronic heart failure, and diabetes mellitus.

Laboratory investigations showed severe anemia, with a hemoglobin level of 6.3 g/dL (reference range 12–16 g/dL). NT-proBNP was markedly elevated at 23,534 ng/L (reference <125 ng/L). High-sensitivity troponin T was 552 ng/L (reference <14 ng/L), with a slight increase to 580 ng/L after 2 hours. C-reactive protein (CRP) was elevated at 65.8 mg/L (reference <5 mg/L). The white blood cell (WBC) count was 6,710 cells/µL (reference range 4,000–11,000 cells/µL).

A diagnosis of acute-on-chronic heart failure associated with COPD exacerbation was established. Non-invasive ventilation (NIV) was initiated together with intravenous diuretic therapy. Owing to concomitant anemia, red blood cell transfusion was performed. Rectal swab screening at admission was negative for multidrug-resistant organisms.

After initial stabilization with NIV, the patient was transitioned to Venturi mask oxygen therapy; however, recurrence of dyspnea necessitated reinstitution of NIV, followed by alternating support with NIV and high-flow nasal oxygen (HFNO). Due to elevated CRP levels and suspected COPD exacerbation, empirical piperacillin/tazobactam was initiated on hospital day 3, despite the absence of radiological evidence of infection on chest CT. However, sputum testing using a multiplex molecular panel (FilmArray Pneumonia Plus Panel, BioFire, bioMérieux®) detected *Klebsiella pneumoniae* and Influenza A virus. Antimicrobial therapy was escalated to meropenem, and antiviral treatment with oseltamivir was commenced. However, conventional sputum cultures did not confirm the growth of *K. pneumoniae*. Oseltamivir was discontinued after 5 days, and meropenem after 7 days. Ventilatory support was continued, alternating between NIV and HFNO.

On hospital day 25, for fever, increased respiratory secretion, and increased CRP, due to clinical suspicion of hospital-acquired pneumonia (HAP), repeat sputum molecular testing identified CRAB. Culture confirmed CRAB, which was susceptible only to colistin; cefiderocol susceptibility had not been tested at that time. The patient was treated with ampicillin–sulbactam (12 g/24 h) and cefiderocol (1 g every 8 hours administered as an extended infusion), adjusted for an estimated glomerular filtration rate of 16 mL/min, for a total of 7 days.

On hospital day 42, due to progression of the right-sided pleural effusion, which had become massive, thoracic drainage with chest tube placement was performed and maintained for 7 days. On hospital day 48, the patient developed a low-grade temperature elevation (37.8°C). Oxygen saturation was 99% on HFNO (FiO_2 65%), blood pressure was 145/48 mmHg, and laboratory investigations revealed a WBC count of 9,700/μL, neutrophils 88%, and CRP 318 mg/L. Blood and sputum cultures were obtained. Concurrently, she developed diarrhea and rectal pain with frequent bowel movements; stool testing was positive for *C. difficile* toxin B.

Peripheral blood cultures yielded CRAB in three bottles, as detected by rapid molecular assay (BioFire), and sputum culture grew CRAB. No central venous catheter was inserted in the patient.

Fidaxomicin was initiated for *C. difficile* infection, and a new course of cefiderocol in combination with ampicillin–sulbactam was started. After 48 hours, antimicrobial susceptibility testing demonstrated that the CRAB isolate was resistant to cefiderocol. MIC was determined by gradient diffusion (MIC Test Strip) on iron-depleted, cation-adjusted Mueller–Hinton agar (ID-CAMHA), following European Committee on Antimicrobial Susceptibility Testing (EUCAST) recommendations. After incubation at 35 ± 1°C for 16–20 h, the MIC was read at the intersection between the inhibition ellipse and the strip scale, yielding a value of 12 mg/L, consistent with resistance according to EUCAST breakpoints for *A. baumannii*.

Considering recent extensive antibiotic exposure, the updated resistance profile, and the presence of concomitant *C. difficile* colitis, antimicrobial therapy was modified to eravacycline monotherapy (1 mg/kg every 12 hours). Eravacycline was administered for 14 days, resulting in clinical improvement and microbiological eradication, with follow-up blood cultures turning negative 96 hours after treatment initiation. Susceptibility testing performed by gradient diffusion (ETEST, bioMérieux®) showed an eravacycline minimum inhibitory concentration (MIC) of 0.75 mg/L.

Fidaxomicin was administered for 10 days, with progressive resolution of gastrointestinal symptoms. No recurrence of CRAB bacteremia or *C. difficile* infection was observed during hospitalization.

After 60 days of hospitalization, the patient was successfully weaned from HFNO and NIV, initially transitioning to low-flow oxygen via nasal cannula at 3 L/min and subsequently to room air on hospital day 65.

The hospital course was further complicated by a urinary tract infection caused by carbapenem-resistant *Klebsiella pneumoniae*. Management included replacement of the indwelling urinary catheter and a 5-day course of gentamicin, resulting in clinical resolution. The patient was discharged home after a total hospital stay of 78 days.

The patient's clinical course during hospitalization and the trends in inflammatory markers are summarized in Figures 1 and 2, respectively.

Discussion

CRAB BSIs present formidable treatment challenges, particularly when cefiderocol resistance emerges, and alternative agents are unavailable. This case highlights the potential effectiveness of eravacycline as monotherapy for the treatment of CRAB BSI in a complex clinical scenario characterized by advanced age, multiple comorbidities, critical illness, and concurrent *C. difficile* infection (CDI).

The favorable course of colitis observed during eravacycline therapy suggests that this agent is unlikely to worsen CDI and may confer an ancillary benefit due to its intrinsic activity against *C. difficile*. Recent limited evidence supports the use of adjunctive eravacycline with the standard of care for severe or fulminant colitis [6].

The successful clinical and microbiological outcome observed in this patient is notable given the limited therapeutic options available, documented resistance to cefiderocol, and the absence of standardized susceptibility EUCAST or CLSI (Clinical and Laboratory Standards Institute) clinical breakpoints for eravacycline against *A. baumannii*. Epidemiological cut-off (ECOFF) value analyses have suggested that wild-type CRAB isolates typically have MICs <0.25 mg/L, whereas more recent studies using FDA- or EUCAST-derived interpretative criteria have generally considered isolates with MICs ≤0.5 mg/L susceptible [7]. Alternative criteria, such as those proposed by the Chinese Committee on Antimicrobial Susceptibility Testing, define susceptibility at MIC <1 mg/L; however, these lack universal validation [8].

Eravacycline exhibits enhanced in vitro activity against multidrug-resistant CRAB and overcomes common tetracycline resistance mechanisms; nevertheless, its use in BSIs remains off-label and is supported mainly by in vitro data and limited and discordant real-world evidence [4, 9].

The IGNITE trials documented only one case of CRAB bacteremia responsive to eravacycline [10]. Most published experiences describe eravacycline as part of combination regimens, whereas reports of successful monotherapy remain exceedingly rare, making the present observation particularly relevant.

Wang et al., in a retrospective multicenter study, reported a clinical response rate of 84.0% among hematologic patients with BSIs treated with eravacycline either as monotherapy or in combination therapy, including cases caused by CRAB [3].

Melchio et al. described a case of CRAB BSI in a patient with previous ventilator-associated pneumonia. The patient was initially treated with cefiderocol plus high-dose sulbactam; after achieving hemodynamic stabilization within 48 hours, treatment was de-escalated to eravacycline monotherapy [4].

More recently, Hu et al. published the results of a retrospective real-world multicenter cohort study in China showing that eravacycline-based regimens were associated with numerically higher clinical success rates (63.2% vs 55.1%) and lower mortality rates (40.1% vs 42.0%) in patients with CRAB pneumonia, compared with best available therapy consisting of sulbactam-, colistin-, or tigecycline-based regimens administered either as monotherapy or combination therapy. In addition, eravacycline-based therapy achieved significantly higher microbiological eradication rates at the end of treatment (45.1% vs 32.0%, p = 0.009) [11]. Notably, these findings were confirmed in the subgroup of patients with concomitant bacteremia (23/204, 11.3%). None of the enrolled patients received cefiderocol, a drug widely used in Europe and the United States for the treatment of CRAB.

In our case, eravacycline was administered before antimicrobial susceptibility results were available, resulting in clinical improvement. Susceptibility testing for eravacycline was performed using the gradient diffusion (ETEST) method. While widely implemented in routine clinical microbiology laboratories, gradient diffusion methods may yield MIC values that differ from those

obtained by broth microdilution, the reference standard, potentially affecting the accuracy of susceptibility categorization. Therefore, the observed MIC of 0.75 mg/L should be interpreted cautiously, given inter-method variability.

Earlier studies suggested that MIC test strips may slightly underestimate eravacycline MIC values for *A. baumannii* compared with the reference broth microdilution method, although categorical agreement between the applied methodologies remained high [12]. Furthermore, more recent findings have demonstrated a high level of concordance between the two methods, supporting the reliability of the ETEST in routine clinical practice [13].

Nevertheless, interpretation of antimicrobial susceptibility is complicated by the current lack of validated clinical breakpoints for eravacycline against CRAB. In our case, application of ECOFF values would have discouraged the use of eravacycline, as the isolate exhibited an MIC >0.5 mg/L. Nevertheless, the favorable clinical outcome observed in our patient suggests that ECOFF-based interpretation alone may not adequately predict clinical efficacy in CRAB infections. On the contrary, our findings highlight the urgent need for clinically validated breakpoints for eravacycline against CRAB, given the extremely limited therapeutic options available for this pathogen and its high resistance to most antibiotics currently used in clinical practice.

Likewise, cefiderocol MIC was determined by gradient diffusion (MIC Test Strip) on ID-CAMHA. According to current EUCAST recommendations, the reference method for cefiderocol susceptibility testing is broth microdilution performed in ID-CAMHB. Given the drug's siderophore-mediated uptake and its dependence on iron-restricted conditions, agar-based methods may show inter-method variability, particularly in *A. baumannii* [14]. Although the observed MIC of 12 mg/L clearly falls within the resistant range according to EUCAST breakpoints [15], the absence of confirmation by broth microdilution should be acknowledged as a methodological limitation.

Eravacycline and cefiderocol differ substantially in both their mechanisms of action and their resistance pathways. Cefiderocol is a siderophore cephalosporin that exploits bacterial iron transport systems to achieve intracellular penetration and inhibits cell wall synthesis through binding to penicillin-binding proteins [16]. Resistance mechanisms described in *A. baumannii* include alterations in iron transport pathways, β-lactamases, and heteroresistance phenomena [17]. In contrast, eravacycline is a synthetic fluorocycline that inhibits bacterial protein synthesis through binding to the 30S ribosomal subunit and retains activity against many isolates carrying common tetracycline resistance determinants, including efflux pumps and ribosomal protection proteins [16]. The different mechanisms of action and resistance profiles may explain preserved eravacycline activity despite cefiderocol resistance in the present case.

Combination therapy is frequently preferred for severe CRAB infections due to concerns about rapid resistance emergence and limited clinical evidence supporting monotherapy [2]. However, in the present case, several factors supported a monotherapy approach. First, therapeutic alternatives were extremely limited because of cefiderocol resistance. Second, avoidance of additional broad-spectrum agents was considered desirable in the setting of active *C. difficile* colitis. Third, the patient achieved rapid microbiological clearance and progressive clinical stabilization after initiation of eravacycline, supporting continuation of a targeted monotherapy strategy.

In conclusion, this case suggests eravacycline may be effective and safe as monotherapy for cefiderocol-resistant CRAB BSI in complex clinical settings with severe comorbidities and concurrent CDI. While these findings must be interpreted cautiously, given the single-case nature of the observation, they support further investigation of eravacycline monotherapy as a salvage strategy in selected patients with extensively drug-resistant *A. baumannii* infections and underscore the need for dedicated clinical studies to better define its role, optimal use, and potential added value in patients with concurrent CDI. Interpretation of eravacycline susceptibility remains challenging because standardized clinical breakpoints for *A. baumannii* are currently lacking.

Consent

Informed consent was obtained from the patient for the use of clinical anonymous data.

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This study did not receive any funding.

Conflict of interest

Nothing to declare

Author contribution

L.P. writing original draft and conceptualization; A.A. clinical management and visualization; G.D.A., visualization; M.T., visualization; T.F. microbiological investigations and visualization; G.M.G., microbiological investigations and visualization; A.C. supervision and revision.

Ethical Approval statement

This study was approved by the Ethics Committee "Palermo 1", Palermo, Italy (DTR-MDR DIMUNA-Verbal n.11 22 December 2022).

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1&search%5Bspecies%5D=4&search%5Bdisk_content%5D=-1&search%5Blimit%5D=50
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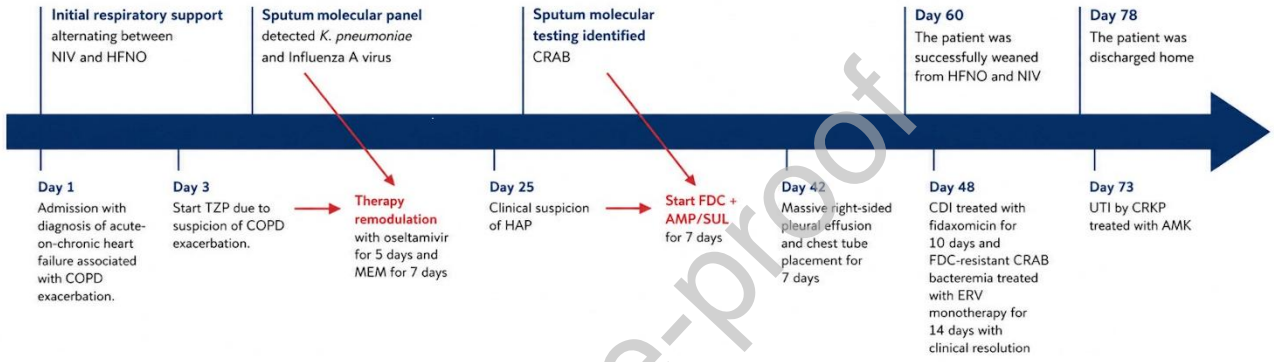

Figure 1. Timeline of the patient's hospital course, including clinical events, diagnostic procedures, treatments, and outcomes. NIV: Non-invasive ventilation. HFNO: high-flow nasal oxygen. CRAB: carbapenem-resistant *A. baumannii*. COPD: chronic obstructive pulmonary disease. TZP: piperacillin-tazobactam. MEM: meropenem. HAP: hospital acquired pneumonia. FDC: cefiderocol. AMP/SUL: ampicillin/sulbactam. CDI: *Clostridioides difficile* infection. ERV: eravacycline. UTI: urinary tract infection. CRKP: carbapenem resistant *K. pneumoniae*. AMK: amikacin.

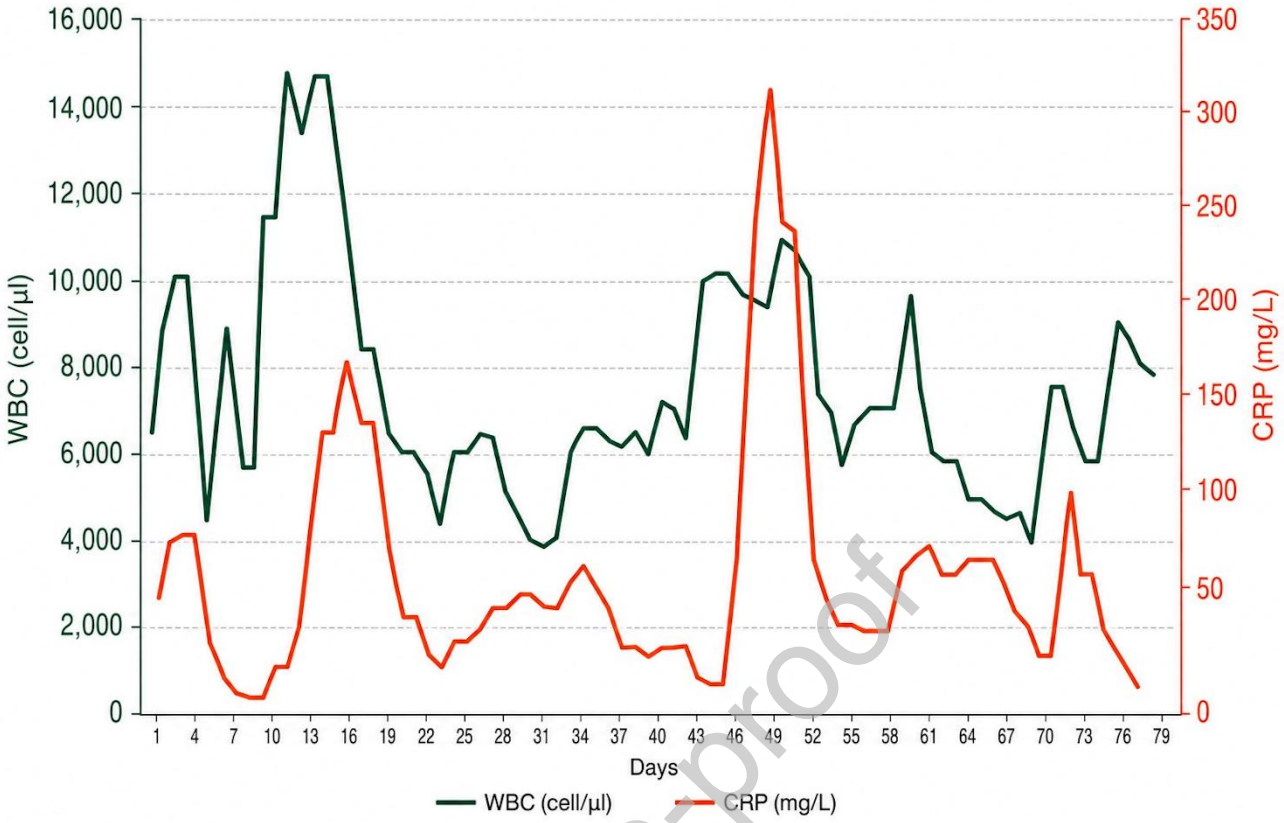

Figure 2. Trends in inflammatory markers during hospitalization. CRP: C-reactive protein. WBC: White blood cells.

Table 1. Antibiogram of *A. baumannii* isolated from blood cultures, including cefiderocol susceptibility testing performed by gradient diffusion (MIC Test Strip) on iron-depleted, cation-adjusted Mueller–Hinton agar.

Antibiotics	Minimum inhibitory concentration (mg/L)	Interpretation according to EUCAST breakpoint
Amikacin	> 32	Resistant
Ciprofloxacin	> 1	Resistant
Colistin	≤ 1	Susceptible
Gentamicin	> 4	Resistant
Imipenem	>8	Resistant
Levofloxacin	> 8	Resistant
Meropenem	> 8	Resistant
Trimethoprim/sulfamethoxazole	>8/152	Resistant
Cefiderocol	12	Resistant
Eravacycline	0.75	Non-interpretable*

* No clinical breakpoint for *A. baumannii.* Eravacycline MIC values ≤0.5 mg/L have been considered susceptible in several recent studies using FDA- or EUCAST-derived interpretative criteria, although no validated EUCAST/CLSI clinical breakpoint for *A. baumannii* currently exists.

Conflict of Interes

The authors have declared no conflict of interest