Infection and Drug Resistance

Dovepress
Taylor & Francis Group

Open Access Full Text Article

CASE REPORT

Successful Treatment of OXA-23 *Acinetobacter baumannii* Pneumonia with Sulbactam-Durlobactam in a Liver Transplant Recipient

Guangjiao Yin[1,2], Zhongzhong Liu[1,2], Zhen Fu[1,2], Zibiao Zhong[1,2], Shaojun Ye[1,2], Huaqin Pan[1–3]

[1]Department of Organ Transplantation, Zhongnan Hospital of Wuhan University, Institute of Hepatobiliary Diseases of Wuhan University, Wuhan, 430071, People's Republic of China; [2]Department of Transplant Intensive Care Unit, Zhongnan Hospital of Wuhan University, Transplant Center of Wuhan University, Wuhan, 430071, People's Republic of China; [3]Department of Critical Care Medicine, Zhongnan Hospital of Wuhan University, Wuhan, 430071, People's Republic of China

Correspondence: Shaojun Ye, Department of Organ Transplantation, Zhongnan Hospital of Wuhan University, Institute of Hepatobiliary Diseases of Wuhan University, 169 Eastlake Road, Wuhan, Hubei, 430071, People's Republic of China, Email 86987100@qq.com; Huaqin Pan, Department of Transplant Intensive Care Unit, Zhongnan Hospital of Wuhan University, Transplant Center of Wuhan University, 169 Eastlake Road, Wuhan, Hubei, 430071, People's Republic of China, Email phq2012@whu.edu.cn

Abstract: Liver transplant recipients face a higher risk of multidrug-resistant (MDR) infections because of preoperative comorbidities, extensive antibiotic use, immunosuppressive therapy, and prolonged mechanical ventilation. Carbapenem-resistant *Acinetobacter baumannii* (CRAB) remains one of the most challenging pathogens in this group. We report a case of a liver transplant recipient with OXA-23-producing *Acinetobacter baumannii* pneumonia. Initial treatment with polymyxin B and eravacycline produced unsatisfactory results and worsened renal dysfunction during treatment. Later, administration of sulbactam-durlobactam (SUL-DUR) combined with meropenem resulted in significant clinical improvement. Follow-up CT scans showed notable resolution, and the patient successfully recovered, with renal function restored and eventual discharge. This case highlights the difficulty of managing OXA-23-producing *Acinetobacter baumannii* in liver transplant patients, especially those with kidney impairment. SUL-DUR showed effective therapy with a much lower risk of nephrotoxicity compared to previously used agents.

Keywords: liver transplantation, carbapenem-resistant *Acinetobacter baumannii*, sulbactam-durlobactam, multidrug-resistant infection

Introduction

Liver transplantation (LT) is the most effective therapeutic option for patients with end-stage liver disease. Due to preoperative comorbidities, repeated antibiotic exposure, immunosuppressant therapy, and prolonged mechanical ventilation, the risk of multidrug-resistant (MDR) bacterial infections increases in LT recipients.[1,2] MDR infections have emerged as a significant threat to this group and are linked to adverse outcomes.[3,4] These infections are associated with a high 30-day mortality rate following LT, especially within the first 5 days of onset.[5] Furthermore, mortality can reach 50% when complicated by septic shock.[4] One of the most challenging pathogens faced in LT recipients is carbapenem-resistant *Acinetobacter baumannii* (CRAB), a gram-negative bacilli known for its extensive antibiotic resistance and its role in severe nosocomial infections, including ventilator-associated pneumonia (VAP), bloodstream infections, and surgical site infections.[5]

CRAB exhibits resistance to nearly all β-lactam antibiotics, including carbapenems, mainly through the production of carbapenem-hydrolyzing class D β-lactamases.[6] Among these, OXA-23 is the most common carbapenemase genotype, with detection rates reaching up to 99% in China.[7] The emergence of antimicrobial resistance mechanisms, combined with limited treatment options, has created significant challenges for clinical management. The 2024 Infectious Diseases Society of America (IDSA) guidance for CRAB infections first recommend combination therapy based on sulbactam-durlobactam (SUL-DUR).[8]

Received: 22 June 2025
Accepted: 9 October 2025
Published: 17 October 2025

As noted, there is still limited data on the role of CRAB in pulmonary infections after LT. Here, we share a case of a liver transplant recipient with OXA-23-producing *Acinetobacter baumannii* pulmonary infection who was effectively treated with SUL-DUR in combination with meropenem.

Case Presentation

A 55-year-old male patient with a history of chronic hepatitis B and post-hepatitic cirrhosis was diagnosed with hepatocellular carcinoma (HCC) in December 2024. He underwent two sessions of transarterial chemoembolization (TACE). Positron emission tomography/Computed tomography (PET/CT) showed multiple nodules and masses in the right hepatic lobe with low metabolic activity, consistent with post-treatment changes, indicating significant tumor suppression or necrosis. No signs of malignancy or metastasis were observed elsewhere. Subsequently, the patient developed complications including portal hypertension, splenomegaly, ascites, hepatorenal syndrome, and cirrhotic cardiomyopathy. In March 2025, he presented at our transplantation center for liver transplantation. He also had bilateral emphysema, pulmonary bullae, and secondary pulmonary tuberculosis. On admission (Day 0), the clinical outcome data were summarized as follows.

- Transaminase levels were within normal limits; total bilirubin was mildly elevated at 31 μmol/L, and blood ammonia was 60 μmol/L.
- Hepatitis B viral load (high-sensitivity quantification) was 120 IU/mL, with hepatitis B surface antigen >250 IU/mL. Alpha-fetoprotein (AFP) was 22 ng/mL, and protein induced by vitamin K absence/antagonist-II (PIVKA-II) was 120 mAU/mL.
- The patient was successfully matched, with minimal risk of an immune response due to human leukocyte antigen (HLA) mismatch. Preoperative panel reactive antibody (PRA) and donor-specific antibodies (DSA) were both negative.
- Detailed laboratory values are summarized in Table 1.

He underwent a classic allogeneic liver transplant with an organ from a brain-dead donor. The donor was a 44-year-old male who suffered a brainstem hemorrhage leading to brain death, with a documented history of fatty liver. Pathological examination of the donor liver showed approximately 30% macrovesicular steatosis in hepatocytes. Immunosuppressants were administered to prevent organ rejection. Corticosteroids and intravenous immunoglobulin (IVIG) were used for

Table 1 Clinical Laboratory Results

Measure	Normal range	Day 0	POD 1	POD 5	Poly B Treatment		Mero-Sul-Dur Treatment		
					POD 10	POD 15	POD 20	POD 25	POD 30
White blood cell (10^9/L)	3.5–9.5	2.7	13.5	12.8	20.3	17.4	7.0	10.5	4.5
Procalcitonin (ng/mL)	<0.05	5.83	12.70	4.80	7.13	3.66	1.41	0.69	0.15
Interleukin-6 (ng/mL)	0–7	452	42	15	1357	161	160	23	12
Urea nitrogen (mmol/L)	2.8–7.6	4.0	11.3	12.3	23.3	55.5	44.0	14.3	7.0
Creatinine (μmol/L)	64–104	60	169	241	506	609	424	153	80
ALT (U/L)	9–50	30	2204	477	52	10	26	19	13
AST (U/L)	15–40	38	4505	91	19	23	19	15	20
TBIL (μmol/L)	5–21	32	107	124	121	114	59	19	15
Ammonia (μmol/L)	18–72	61	76	62	-	-	-	-	-
PT (s)	9.4–12.5	15.9	32.5	17.2	16.5	16.5	15.7	16.4	14.7
APTT (s)	25.1–36.5	37.6	91.3	89.4	44.5	44.4	60.5	38.7	40.5
PTTA (%)	80–130	57	23	51	52	54	56	53	66
D-dimer (ng/mL)	0–500	757	4285	1810	6142	5327	4831	3822	3452
Fibrinogen (mg/dL)	238–498	160	149	225	299	204	199	257	367

Abbreviations: ALT, alkaline phosphatase; AST, aspartate transaminase; TBIL, total bilirubin; PT, prothrombin time; APTT, activated partial thromboplastin time; PTTA, prothrombin time activity.

induction of immunosuppression during surgery. For postoperative maintenance therapy, three immunosuppressive drugs- including prednisolone, tacrolimus (TAC), and mycophenolate mofetil (MMF) were prescribed. On postoperative day (POD) 1, transaminase levels rose to 4500 U/L, and total bilirubin increased to 106 μmol/L. Simultaneously, the patient remained unconscious, with signs suggesting ischemia-reperfusion injury and delayed graft function. Plasma exchange was initiated. Doppler ultrasound was used to monitor graft size, blood flow, resistance index, fluid accumulation, and thrombosis. By POD 3, the patient was conscious and weaned off mechanical ventilation successfully. As per our transplant center's antimicrobial prophylaxis protocol, the patient received piperacillin/tazobactam 4.5 g intravenously (IV) every 12 hours and caspofungin 50mg IV daily for post-transplant infection prevention.

One week postoperatively, the patient developed a fever and excessive airway secretions, which quickly advanced to acute respiratory distress and septic shock. His oxygen needs increased, requiring high-flow nasal oxygen (HFNO) therapy with a fraction of inspired O2 (FiO2) of 60%. He also needed vasopressor support with norepinephrine at a dose of 0.2 mcg/kg/min. Procalcitonin levels rose to 7.13 ng/mL, and interleukin-6 levels surged to 1357 pg/mL, indicating a strong inflammatory response. Chest CT showed worsening of the infection compared to earlier imaging (Figure 1). Transnasal bronchoscopy was performed under conscious sedation, during which airway secretion samples were collected for laboratory testing. Empirical antimicrobial treatment with imipenem/cilastatin, linezolid, and caspofungin was started. On POD 9, metagenomic next-generation sequencing (mNGS) of bronchoalveolar lavage fluid (BALF) detected 1,651,915 copies/mL of *Acinetobacter baumannii* gene fragments (platform: Illumina, manufacturer: Hangzhou Jieyi Biotechnology). The results confirmed that the carbapenem-resistant *Acinetobacter baumannii* isolates carried an OXA-23-producing class D β-lactamase. Additionally, a small number of carbapenem-resistant Klebsiella pneumoniae (CRKP) gene reads (13,915 copies/mL) were identified, indicating the presence of a Klebsiella pneumoniae carbapenemase (KPC)-producing class A β-lactamase. Subsequently, only CRAB was grown in BALF cultures. In vitro antimicrobial susceptibility testing showed that CRAB was only sensitive to polymyxin, eravacycline, and tigecycline (Table 2). The antibiotic regimen was adjusted to polymyxin B 50 mg IV every 12 hours combined with eravacycline 50 mg IV every 12 hours. Despite five days of treatment, there was minimal improvement in respiratory secretions and septic shock. Infection markers remained high, and norepinephrine dependence persisted, with progressively increasing doses needed to maintain hemodynamic stability, indicating inadequate infection control. The patient had a history of preoperative hepatorenal syndrome and developed postoperative acute kidney injury (AKI), which was worsened by

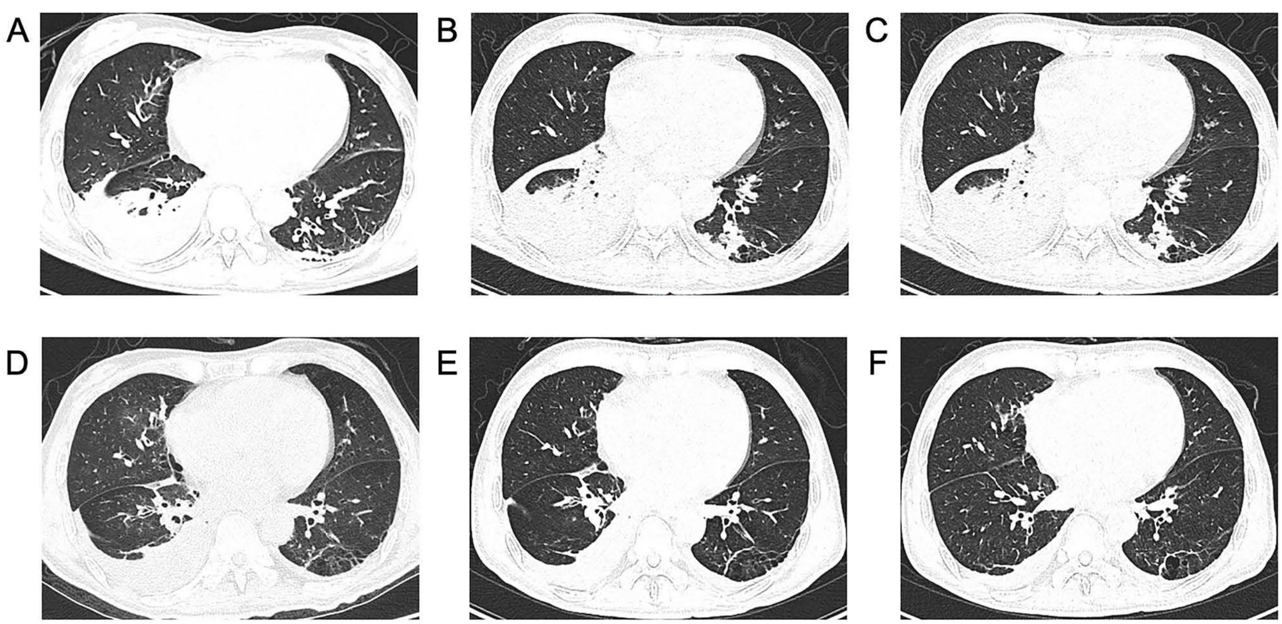

Figure 1 Comparison of CT scans of the patient's lungs before and after treatment. (**A**) (POD 2), (**B**) (POD 12), (**C**) (POD 20), (**D**) (POD 30), (**E**) (POD 36), (**F**) (POD 44). (**B** and **C**) were obtained following polymyxin B therapy, while (**D**–**F**) were taken during the subsequent meropenem-sulbactam-durlobactam therapy. The POD 44 image shows significant resolution of pneumonitis.

 https://doi.org/10.2147/IDR.S548066

Table 2 Antimicrobial Susceptibility Profile of the OXA-23 Producing *Acinetobacter baumannii* Isolate

Antimicrobial	MIC (mg/L)	Sensitivity	S	I	R
Cefoperazone/sulbactam	≥64	R	≤16	32	≥64
Cefepime	≥32	R	≤8	16	≥32
Ceftazidime	≥64	R	≤8	16	≥32
Ciprofloxacin	≥4	R	≤1	2	≥4
Imipenem	≥16	R	≤2	4	≥8
Levofloxacin	≥8	R	≤2	4	≥8
Piperacillin/Tazobactam	≥128	R	≤16/4	32/4–64/4	≥128/4
Tobramycin	≥16	R	≤4	8	≥16
SMZ-TMP	≥320	R	≤2/38		≥4/76
Tigecycline	2	S	≤2	4	≥8
Polymyxin	2	S	≤2		≥4
Meropenem	≥16	R	≤2	4	≥8
Ticarcillin/clavulanic acid	≥128	R	≤16/2	32/2-64/2	≥128/2
Minocycline	8	I	≤4	8	≥16
Doxycycline	≥16	R	≤4	8	≥16
Eravacycline	0.19	S	≤1		

Abbreviations: MIC, minimal inhibitory concentration; I, intermediate; R, resistant; S, sensitive.

polymyxin therapy. About 15 days post-surgery, serum creatinine rose sixfold from baseline, and urine output declined to anuria, requiring continuous renal replacement therapy (CRRT).

On POD20, a repeat chest CT showed persistent infectious consolidation in the lower lobe without radiologic improvement (Figure 1). The patient's sputum volume had not decreased, so pulmonary bronchoscopy was performed again. BALF-mNGS analysis still detected a large number of CRAB gene reads (502,701 copies/mL). CRAB was also isolated from BALF culture. The antibiotic regimen was changed to SUL-DUR 1 g/1 g IV every 8 hours, combined with meropenem 1 g IV every 8 hours. After 5 days of treatment, the patient's oxygenation improved, allowing successful liberation from HFNO therapy. The patient was transitioned to low-flow nasal oxygen at 2 L/min, with oxygen saturation maintained at 96–100%. On day 10 of the SUL-DUR and meropenem regimen, norepinephrine was discontinued, and procalcitonin levels gradually decreased to near-normal levels.

At the end of a 2-week course of SUL-DUR, the patient showed significant clinical improvement, including the resolution of fever, decreased sputum production, and better respiratory function. Renal function gradually improved, enabling the cessation of dialysis, while hepatic allograft function stayed stable. The timeline of the hospital stay with antibiotic treatment is shown in Figure 2. The patient was discharged on POD 44 without any complications.

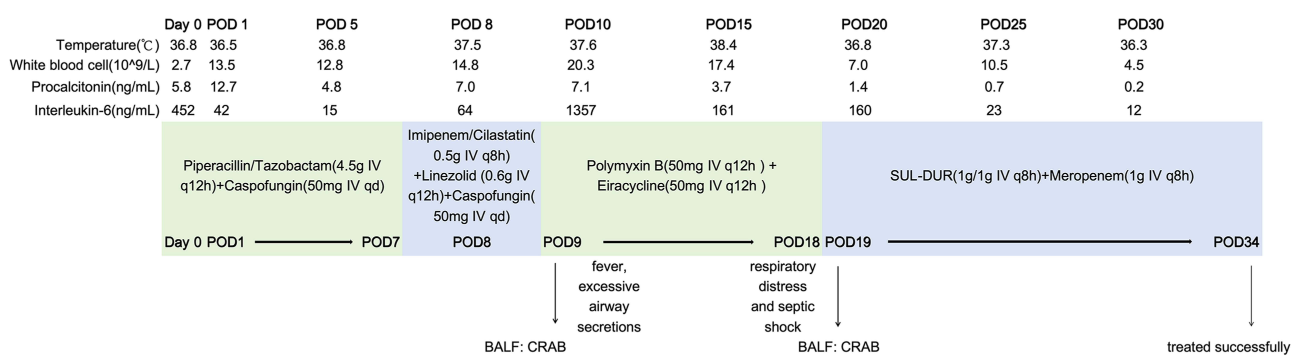

Figure 2 Timeline diagram of disease and treatment course.

Discussion

Infections caused by CRAB have become a serious threat to LT recipients. A systematic review and meta-analysis reported consistently high global detection rates of CRAB, with resistance to imipenem and meropenem averaging 44.7% and 59.4%, respectively.[9] The management of CRAB infections remains especially difficult because of their intrinsic multidrug resistance patterns and the limited effectiveness of current antimicrobial agents. SUL-DUR is a novel anti-CRAB agent, demonstrating potent inhibitory activity against the vast majority of class D carbapenemases (OXA-type β-lactamases), including OXA-23. In this report, we described a liver transplant recipient who developed OXA-23-producing *Acinetobacter baumannii* pneumonia complicated by respiratory failure and septic shock, and was successfully treated with a SUL-DUR-based combination of meropenem.

The main resistance mechanisms of CRAB include β-lactamase production, efflux pump overexpression, target-site modification, and changes in membrane permeability.[6] The predominant mechanism in carbapenem-resistant isolates involves enzymatic hydrolysis of β-lactam antibiotics by β-lactamases, specifically class D carbapenemases of the OXA family, such as OXA-23, OXA-24/40, OXA-51, and OXA-58, which hydrolyze carbapenems through a serine-active site.[10] OXA-23 has the highest global prevalence.[7,11] This Ambler class D β-lactamase confers high-level resistance to imipenem and meropenem by enzymatic hydrolysis and works together with outer membrane protein (OMP) loss, leading to typical MICs higher than 16 mg/L, which exceeds clinical breakpoints for carbapenem susceptibility.[12]

Current treatment strategies for CRAB infections mainly depend on traditional last-resort agents like colistin and tigecycline, but these face limitations such as reduced effectiveness, serious nephrotoxicity, and rising resistance.[13,14] A multicenter study conducted in the intensive care unit (ICU) found that 32.9% of polymyxin-sensitive strains developed resistance to polymyxin during treatment of CRAB with it, with a high rate of treatment failure (death or persistent bacteria).[15] Polymyxin's high nephrotoxicity restricts its use. A multicenter, randomized, active-controlled, Phase 3, non-inferiority clinical trial (ATTACK) showed that SUL-DUR is more effective and safer than polymyxin in treating severe infections like hospital-acquired pneumonia (HAP), VAP, and bloodstream infections caused by CRAB, with significantly lower nephrotoxicity rates (13% vs 38%).[16]

SUL-DUR, a novel β-lactam-β-lactamase inhibitor combination, targets CRAB through synergistic molecular interactions and optimized pharmacokinetic/pharmacodynamic (PK/PD) profiles.[17,18] SUL, a semi-synthetic penicillanic acid derivative, exhibits weak intrinsic inhibitory activity against class A serine β-lactamases but lacks activity against class C and D β-lactamases. Its primary antibacterial action is mediated through high-affinity binding to penicillin-binding protein (PBP) 1 and PBP3 in Acinetobacter baumannii, preventing their involvement in bacterial cell wall synthesis and exerting bactericidal activity against this pathogen.[18,19] DUR, a diazabicyclooctane inhibitor, exhibits potent inhibition of Ambler class A, C, and D enzymes.[20] It demonstrates potent activity against currently studied class D carbapenem-hydrolyzing OXA-type β-lactamases. Meropenem enhances this antibacterial effect by selectively targeting PBP2 and blocking its biological function in cell wall assembly. By blocking enzymatic degradation of SUL and Meropenem, DUR improves their penetration to PBP targets, increasing bactericidal effectiveness against *Acinetobacter baumannii*.[18,21] The combination of SUL-DUR with carbapenem shows significant synergistic bactericidal effects against carbapenemase-producing strains.[22]

A growing body of clinical reports demonstrates the successful use of SUL-DUR-based combinations, often with a carbapenem, in treating critically ill patients with extensively drug-resistant *Acinetobacter baumannii* infections. These include cases of nosocomial pneumonia,[23,24] severe burn injury,[11,25] and meningitis.[26,27] All other patients received a carbapenem during SUL-DUR therapy to cover potential co-infecting pathogens. The 2024 IDSA guidance recommends SUL-DUR, combined with meropenem or imipenem-cilastatin, as the preferred treatment for CRAB infections.[8] Given the complexity of this patient group, data on its use in solid organ transplantation are very limited. To date, successful treatment of CRAB infections with sulbactam-durlobactam has been reported in kidney and lung transplant recipients.[28,29] To our knowledge, we first present the successful use of an antimicrobial regimen combining SUL-DUR and meropenem in a liver transplant recipient.

In our case, the patient developed hepatorenal syndrome before LT. After surgery, acute renal failure complicated the course and was attributed to hypoxic-ischemic reperfusion injury. Due to limited treatment options, polymyxin was used for CRAB infection. However, the pulmonary infection remained uncontrolled, and polymyxin worsened AKI. The antimicrobial

 https://doi.org/10.2147/IDR.S548066

therapy was then changed to a SUL-DUR-based regimen. After two weeks of targeted treatment, the infection was successfully controlled, and kidney function returned to normal.

Conclusion

We present a case of a liver transplant recipient with OXA-23-producing *Acinetobacter baumannii* infection who failed initial therapy with polymyxin and eravacycline, then developed worsening renal insufficiency. Successful eradication was achieved after switching to a combination of SUL-DUR and meropenem. This case highlights the potential clinical utility of SUL-DUR-based combination therapy for managing CRAB infections in solid organ transplantation, especially liver transplant recipients for whom conventional treatments may be contraindicated due to toxicity or resistance. Currently, real-world clinical data on SUL-DUR for CRAB infections in liver transplant recipients remain limited, emphasizing the need for large-scale clinical studies.

Data Sharing Statement

The datasets used and/or analyzed during the current study are available from the corresponding author, Huaqin Pan, upon reasonable request.

Ethics Approval and Informed Consent

Ethics approval was not required for the publication of case report because all patient-related data were collected retrospectively and anonymously. Liver donation was performed in accordance with the Declaration of Istanbul. The brain-dead donor's voluntary consent for organ donation was obtained either from the donor before death or through authorized family members, with proper documentation of informed consent. China's national organ allocation protocols strictly enforce a double-blind process between donor and recipient teams. As members of the transplant surgical team, we do not have access to the donor's informed consent documents nor permission to view donor-identifiable information, as these details are protected by the institution. Written informed consent was obtained from the recipient for this case report's publication.

Acknowledgments

The authors thank the patient who kindly volunteered to participate in this study.

Author Contributions

All authors made a significant contribution to the work reported, whether in conception, study design, execution, data acquisition, analysis, and interpretation, or in all these areas; participated in drafting, revising, or critically reviewing the article; gave final approval of the version to be published; agreed on the journal where the article was submitted; and commit to being responsible for all aspects of the work.

Funding

This research is supported by the Medical Sci-Tech innovation platform of Zhongnan Hospital, Wuhan University (grant no. PTXM2025019 [H.Q. Pan]).

Disclosure

The authors report no conflicts of interest in this work.

References

1. Silva JT, Fernandez-Ruiz M, Aguado JM. Multidrug-resistant Gram-negative infection in solid organ transplant recipients: implications for outcome and treatment. *Curr Opin Infect Dis*. 2018;31(6):499–505. doi:10.1097/QCO.0000000000000488
2. Timsit JF, Sonneville R, Kalil AC, et al. Diagnostic and therapeutic approach to infectious diseases in solid organ transplant recipients. *Intensive Care Med*. 2019;45(5):573–591. doi:10.1007/s00134-019-05597-y
3. Freire MP, Pierrotti LC, Oshiro IC, et al. Carbapenem-resistant Acinetobacter baumannii acquired before liver transplantation: impact on recipient outcomes. *Liver Transpl*. 2016;22(5):615–626. doi:10.1002/lt.24389
4. Liu AJ, Dennis ASM, Fariha Z, et al. Multidrug-resistant organism bloodstream infections in solid organ transplant recipients and impact on mortality: a systematic review. *JAC Antimicrob Resist*. 2024;6(5):dlae152. doi:10.1093/jacamr/dlae152

5. Min E-K, Yim SH, Choi MC, et al. Incidence, mortality, and risk factors associated with carbapenem-resistant Acinetobacter baumannii bacteremia within 30 days after liver transplantation. *Clin Transplant*. 2023;37(5):e14956. doi:10.1111/ctr.14956
6. Marino A, Augello E, Stracquadanio S, et al. Unveiling the Secrets of Acinetobacter baumannii: resistance, Current Treatments, and Future Innovations. *Int J Mol Sci*. 2024;25(13):6814. doi:10.3390/ijms25136814
7. Wang M, Ge L, Chen L, et al. Clinical Outcomes and Bacterial Characteristics of Carbapenem-resistant Acinetobacter baumannii Among Patients From Different Global Regions. *Clin Infect Dis*. 2024;78(2):248–258. doi:10.1093/cid/ciad556
8. Tamma PD, Heil EL, Justo JA, et al. Infectious Diseases Society of America 2024 Guidance on the Treatment of Antimicrobial-Resistant Gram-Negative Infections. *Clin Infect Dis*. 2024. ciae403. doi:10.1093/cid/ciae403
9. Ghahramani A, Naghadian Moghaddam MM, Kianparsa J, et al. Overall status of carbapenem resistance among clinical isolates of Acinetobacter baumannii: a systematic review and meta-analysis. *J Antimicrob Chemother*. 2024;79(12):3264–3280. doi:10.1093/jac/dkae358
10. Docquier JD, Mangani S. Structure-Function Relationships of Class D Carbapenemases. *Curr Drug Targets*. 2016;17(9):1061–1071. doi:10.2174/1389450116666150825115824
11. Tiseo G, Giordano C, Leonildi A, et al. Salvage therapy with sulbactam/durlobactam against cefiderocol-resistant Acinetobacter baumannii in a critically ill burn patient: clinical challenges and molecular characterization. *JAC Antimicrob Resist*. 2023;5(3):dlad078. doi:10.1093/jacamr/dlad078
12. Evans BA, Amyes SG. OXA beta-lactamases. *Clin Microbiol Rev*. 2014;27(2):241–263. doi:10.1128/CMR.00117-13
13. Iovleva A, Fowler Jr VG, Doi Y. Treatment Approaches for Carbapenem-Resistant Acinetobacter baumannii Infections. *Drugs*. 2025;85(1):21–40. doi:10.1007/s40265-024-02104-6
14. Zhang S, Di L, Qi Y, et al. Treatment of infections caused by carbapenem-resistant Acinetobacter baumannii. *Front Cell Infect Microbiol*. 2024;14:1395260. doi:10.3389/fcimb.2024.1395260
15. Qureshi ZA, Hittle LE, O'Hara JA, et al. Colistin-resistant Acinetobacter baumannii: beyond carbapenem resistance. *Clin Infect Dis*. 2015;60(9):1295–1303. doi:10.1093/cid/civ048
16. Kaye KS, Shorr AF, Wunderink RG, et al. Efficacy and safety of sulbactam-durlobactam versus colistin for the treatment of patients with serious infections caused by Acinetobacter baumannii-calcoaceticus complex: a multicentre, randomised, active-controlled, phase 3, non-inferiority clinical trial (ATTACK). *Lancet Infect Dis*. 2023;23(9):1072–1084. doi:10.1016/S1473-3099(23)00184-6
17. Fu Y, Asempa TE, Kuti JL. Unraveling sulbactam-durlobactam: insights into its role in combating infections caused by Acinetobacter baumannii. *Expert Rev Anti Infect Ther*. 2025;23(1):67–78. doi:10.1080/14787210.2024.2440018
18. McLeod SM, O'Donnell JP, Narayanan N, et al. Sulbactam–durlobactam: a β-lactam/β-lactamase inhibitor combination targeting Acinetobacter baumannii. *Future Microbiol*. 2024;19(7):563–576. doi:10.2217/fmb-2023-0248
19. Penwell WF, Shapiro AB, Giacobbe RA, et al. Molecular mechanisms of sulbactam antibacterial activity and resistance determinants in Acinetobacter baumannii. *Antimicrob Agents Chemother*. 2015;59(3):1680–1689. doi:10.1128/AAC.04808-14
20. Durand-Reville TF, Guler S, Comita-Prevoir J, et al. ETX2514 is a broad-spectrum beta-lactamase inhibitor for the treatment of drug-resistant Gram-negative bacteria including Acinetobacter baumannii. *Nat Microbiol*. 2017;2(9):17104. doi:10.1038/nmicrobiol.2017.104
21. Anand A, Verma A, Kaur S, et al. An overview of sulbactam-durlobactam approval and implications in advancing therapeutics for hospital-acquired and ventilator-associated pneumonia by acinetobacter baumannii-calcoaceticus complex: a narrative review. *Health Sci Rep*. 2024;7(9):e70066. doi:10.1002/hsr2.70066
22. Veeraraghavan B, Shin E, Bakthavatchalam YD, et al. A microbiological and structural analysis of the interplay between sulbactam/durlobactam and imipenem against penicillin-binding proteins (PBPs) of Acinetobacter spp. *Antimicrob Agents Chemother*. 2025;69(4):e0162724. doi:10.1128/aac.01627-24
23. Holger DJ, Kunz Coyne AJ, Zhao JJ, et al. Novel Combination Therapy for Extensively Drug-Resistant Acinetobacter baumannii Necrotizing Pneumonia Complicated by Empyema: a Case Report. *Open Forum Infect Dis*. 2022;9(4):ofac092. doi:10.1093/ofid/ofac092
24. Zaidan N, Hornak JP, Reynoso D. Extensively Drug-Resistant Acinetobacter baumannii Nosocomial Pneumonia Successfully Treated with a Novel Antibiotic Combination. *Antimicrob Agents Chemother*. 2021;65(11):e0092421. doi:10.1128/AAC.00924-21
25. VanNatta M, Grier L, Khan MH, et al. In Vivo Emergence of Pandrug-Resistant Acinetobacter baumannii Strain: comprehensive Resistance Characterization and Compassionate Use of Sulbactam-Durlobactam. *Open Forum Infect Dis*. 2023;10(10):ofad504. doi:10.1093/ofid/ofad504
26. Tamma PD, Immel S, Karaba SM, et al. Successful Treatment of Carbapenem-Resistant Acinetobacter baumannii Meningitis With Sulbactam-Durlobactam. *Clin Infect Dis*. 2024;79(4):819–825. doi:10.1093/cid/ciae210
27. Snowdin JW, Mercuro NJ, Madaio MP, et al. Case report: successful treatment of OXA-23 Acinetobacter baumannii neurosurgical infection and meningitis with sulbactam-durlobactam combination therapy. *Front Med Lausanne*. 2024;11:1381123. doi:10.3389/fmed.2024.1381123
28. Webb AR, Fernandez A, Piccicacco N. Successful Combination Therapy with Cefiderocol and Sulbactam-Durlobactam for Donor-Derived Carbapenem-Resistant Acinetobacter baumannii in a Kidney Transplant Recipient. *Transpl Infect Dis*. 2025;27(4):e70048. doi:10.1111/tid.70048
29. Zhang P, Wu J, Xu Y, et al. Sulbactam-durlobactam treatment for pulmonary infections caused by carbapenem-resistant Acinetobacter baumannii in lung transplant recipients. *J Infect*. 2025;91(3):106596. doi:10.1016/j.jinf.2025.106596