JIDC | THE JOURNAL OF INFECTION IN DEVELOPING COUNTRIES

Original Article

Distribution characteristics and genomic epidemiology of carbapenem-resistant *Acinetobacter baumannii*

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Abstract

Introduction: Carbapenem-resistant *Acinetobacter baumannii* poses significant challenges in clinical settings due to rapid emergence and limited treatment options. This study aimed to investigate the distribution characteristics and genomic epidemiology of carbapenem-resistant *A. baumannii* in a regional healthcare context to identify novel trends and genetic determinants.

Methodology: A total of 120 clinically isolated strains of carbapenem-resistant *A. baumannii* were collected in our hospital from July 2021 to June 2023. Distribution characteristics were assessed based on infection sites. Whole genome sequencing was performed to profile resistance genes.

Results: The resistance rates of carbapenem-resistant *A. baumannii* to 15 antibiotics, such as tobramycin and amikacin, increased yearly during the three years ($p < 0.05$). Respiratory tract and urinary tract infections were dominant. Whole genome sequencing revealed high prevalence rates of β-lactamase genes (AmpC: 82.5%, OXA-23: 70.83%, IMP-1: 54.17%) and efflux system genes (adeB: 78.33%, adeJ: 93.33%, adeG: 90.00%).

Conclusions: Our study provides novel insights by identifying significant increases in antibiotic resistance and revealing critical genomic co-occurrence patterns of resistance genes in carbapenem-resistant *A. baumannii*. These findings enhance the understanding of resistance mechanisms and support targeted strategies for improved prevention, control, and monitoring of nosocomial infections.

Key words: *Acinetobacter baumannii*; carbapenem; distribution; genomic epidemiology; resistance.

J Infect Dev Ctries 2026; 20(5):649-653. doi:10.3855/jidc.20580

(Received 11 July 2024 – Accepted 07 June 2025)

Introduction

Carbapenem-resistant *Acinetobacter baumannii* is a Gram-negative non-fermenter characterized by strong viability, which generally survives in the hospital environment and human skin and cavity. Moreover, *A. baumannii* with strong infectivity can infect various human tissues and organs, such as the respiratory tract, lung, bloodstream, abdominal cavity, wound, and urinary tract [1,2]. Carbapenem-resistant *A. baumannii* has become the major cause of high infection and mortality rates in the Department of Respiratory Medicine and the intensive care unit (ICU) [3]. Carbapenems are mainly used in the treatment of Gram-negative bacterial infections, but they are greatly restricted in the treatment of *A. baumannii* infections due to the resistance of *A. baumannii* to a variety of carbapenems. Therefore, carbapenems are less effective in the treatment of *A. baumannii* infections. As a result, the treatment of *A. baumannii* infections is in trouble, and *A. baumannii* has become a nosocomial infectious bacterium [4]. Therefore, it is necessary to understand the distribution characteristics, gene homology, and carbapenem resistance mechanism of carbapenem-resistant *A. baumannii* for guiding clinical rational drug use for *A. baumannii* infections [5].

In the context above, 120 strains of carbapenem-resistant *A. baumannii* collected through clinical isolation from July 2021 to June 2023 in our hospital were analyzed, the sensitivity of *A. baumannii* to a variety of antibiotics was tested, the distribution characteristics of *A. baumannii* were analyzed, and genotyping was performed on *A. baumannii* to explore the molecular mechanism of drug resistance genes and epidemic characteristics of *A. baumannii*. The findings are expected to provide scientific guidance for the prevention and treatment of carbapenem-resistant *A. baumannii* infections and reduce the infection rate of *A. baumannii*.

Methodology

Source of strains

A total of 120 clinically isolated strains of carbapenem-resistant *A. baumannii* were collected in our hospital from July 2021 to June 2023. Specimens of sputum, throat swab, urine, blood, and drainage fluid were harvested. For various specimens of the same patient, the strain harvested for the first time was analyzed.

Main apparatus

VITEK-2 COMPACT 60 automatic microbial identification and drug sensitivity analysis system was provided by Merier Diagnostic Products (Shanghai) Co., Ltd. (China). The Lightcycler480 real-time quantitative polymerase chain reaction (PCR) system was purchased from Roche Diagnostics (Shanghai) Co., Ltd. (China). An Autof ms1000 mass spectrometer was bought from Autobio Diagnostics Co., Ltd. (China).

Bacterial identification and antibiotic sensitivity test

Bacterial culture was carried out according to the *National Guide to Clinical Laboratory Procedures* [6], and bacterial identification was performed using the VITEK-2 COMPACT 60 automatic microbial identification and drug sensitivity analysis system. The minimal inhibitory concentration of antibiotics against 120 strains of *A. baumannii* was detected using the agar dilution method. The results of the drug sensitivity test were determined according to the CLSI Performance Standards for Antimicrobial Susceptibility Testing (31st Edition, 2022) [7].

Detection of drug resistance genes

KPC resistance genes of drug-resistant strains were detected by a kit purchased from Shanghai Sig Biotechnology Co., Ltd. (China). About 4 strains of test bacteria were selected from the blood agar and placed in 300 μL of normal saline at a solubility of 0.85% to prepare a bacterial suspension. Then the resulting bacterial suspension was centrifuged at 11190 ×g for 5 min, and added with 300 μL of sterile double-distilled water, followed by a warm water bath at 100°C for 15 min and centrifugation at 16113 ×g for 2 min. The resulting supernatant was used as the DNA template. PCR primers were synthesized by Shandong Wanxiang Environmental Technology Co., Ltd. (China). The total volume of the PCR system was 25 μL, and the reaction conditions were as follows: pre-denaturation at 94°C for 5 min, 30 cycles × (denaturation at 94 °C for 30 seconds, renaturation at 52-56°C for 45 seconds, and extension at 72 °C for 1 minute), and extension at 72°C for 7 minutes.

Statistical analysis

SPSS 26.0 software package was used for data analysis. Measurement data were described by mean ± standard deviation ($\bar{x} \pm s$), and count data were described by percentage (%) and analyzed by the χ^2 test. A $p < 0.05$ was considered statistically significant.

Results

Distribution of A. baumannii in clinical specimens

Sputum and throat swabs were the main sources of the 120 strains of *A. baumannii* (Table 1).

Source of carbapenem-resistant A. baumannii from clinical departments

Carbapenem-resistant *A. baumannii* were mainly from the Department of Respiratory Medicine and ICU, and carbapenem-resistant *A. baumannii* from the two departments accounted for 75.0% (Table 2).

Resistance of carbapenem-resistant A. baumannii to commonly used antibiotics

There were no statistically significant differences in the resistance rates of carbapenem-resistant *A. baumannii* to ampicillin-sulbactam and colistin during the three years ($p > 0.05$). The resistance rates of carbapenem-resistant *A. baumannii* to other antibiotics increased year by year during the three years, with

Table 2. Source of carbapenem-resistant *A. baumannii* from clinical departments (%).

Clinical department	Strains (n)	Ratio (%)
Department of Respiratory Medicine	58	48.33
ICU	32	26.67
Department of Spine Surgery	5	4.17
Department of Neurosurgery	5	4.17
Department of Hematology	4	3.33
Department of Trauma Surgery	4	3.33
Department of Neurology	4	3.33
Department of Urology	3	2.5
Nephrology	3	2.5
Department of Cardiothoracic surgery	2	1.67
Total	120	100.00

Table 1. Distribution of *A. baumannii* in clinical specimens [n (%)].

Year	Sputum	Throat swab	Urine	Blood	Drainage fluid	Total
2021	21 (72.41)	6 (20.69)	1 (3.45)	1 (3.45)	0	29 (24.17)
2022	28 (73.68)	5 (13.16)	2 (5.26)	2 (5.26)	1 (2.63)	38 (31.67)
2023	41 (77.36)	8 (15.09)	2 (3.77)	1 (1.89)	1 (1.89)	53 (44.17)
Total	90 (75.00)	19 (15.83)	5 (4.17)	4 (3.33)	2 (1.67)	120 (100.00)

Table 3. Resistance of carbapenem-resistant *A. baumannii* to commonly-used antibiotics (%).

Antibiotics	2021	2022	2023	Statistical value	*p*
Tobramycin	21 (72.41)	31 (81.58)	50 (94.34)	7.578	0.023
Amikacin	20 (68.97)	31 (81.58)	49 (92.45)	7.568	0.023
Ciprofloxacin	19 (65.52)	35 (92.11)	50 (94.34)	14.898	< 0.001
Ofloxacin	19 (65.52)	35 (92.11)	50 (94.34)	14.898	< 0.001
Piperacillin-tazobactam	20 (68.97)	33 (86.84)	49 (92.45)	8.26	0.016
Cefoperazone-sulbactam	11 (37.93)	22 (57.89)	35 (66.04)	6.064	0.048
Ampicillin-sulbactam	21 (72.41)	29 (76.32)	35 (66.04)	1.178	0.555
Aztreonam	28 (96.55)	34 (89.47)	37 (69.81)	11.156	0.003
Cefotaxime	19 (65.52)	34 (89.47)	36 (67.92)	6.857	0.032
Ceftazidime	23 (79.31)	35 (92.11)	35 (66.04)	8.696	0.012
Ceftriaxone	21 (72.41)	34 (89.47)	49 (92.45)	6.893	0.032
Gentamicin	19 (65.52)	32 (84.21)	50 (94.34)	11.684	0.003
Piperacillin	20 (68.97)	34 (89.47)	50 (94.34)	10.823	0.004
Minocycline	5 (17.24)	13 (34.21)	35 (66.04)	20.334	< 0.001
Compound sulfamethoxazole	7 (24.14)	28 (73.68)	47 (88.68)	36.818	< 0.001
Colistin	2 (6.90)	10 (26.32)	16 (30.19)	5.961	0.051
Polymyxin B	2 (6.90)	11 (28.95)	17 (32.07)	6.799	0.033

statistically significant differences ($p < 0.05$) (Table 3).

Infection sites of carbapenem-resistant A. baumannii

Respiratory tract infection and urinary tract infection of carbapenem-resistant *A. baumannii* were dominant (75%) during the three years. The proportion of other infection sites is shown in Table 4.

Detection results of β-lactamase genes and active efflux system genes of A. baumannii

For the 120 strains of *A. baumannii*, the detection rates of β-lactamase genes AmpC, OXA-23, and IMP-1 were 82.5%, 70.83%, and 54.17%, respectively, and the detection rates of active efflux system genes adeB, adeJ, and adeG were 78.33%, 93.33%, and 90.00%, respectively (Table 5).

Discussion

As a common pathogen in nosocomial infection, *A. baumannii* can survive for a long duration outside the human body. With strong clonal spread, it can cause systemic multi-site infections in a short time, but it is usually difficult to cause obvious symptoms of infection due to low pathogenicity. Therefore, early identification of *A. baumannii* infections is difficult [8]. *A. baumannii* often invades sensitive cells *in vivo* by outer membrane protein A and iron carrier, causing extensive damage to host tissues and ultimately resulting in pneumonia, bloodstream infection, and other infectious diseases [9]. In recent years, carbapenem antibiotics have been increasingly used and have gradually become the first choice in clinical treatment of *A. baumannii* infections. However, *A. baumannii* has had increasing resistance to carbapenem antibiotics, thus restricting its clinical application. According to relevant data, the resistance rate of *A. baumannii* to carbapenems has increased from 31% to 70% [10,11]. In addition, due to the clinical abuse of carbapenem antibiotics, carbapenem resistance of *A. baumannii* is enhanced. Therefore, *A. baumannii* has attracted increasingly more attention in the clinic and undergone a series of studies [12]. However, few studies are available on the molecular mechanism of drug resistance genes and the epidemic characteristics of *A. baumannii*. To achieve reasonable and efficient drug therapy for *A. baumannii* infections, the distribution characteristics of carbapenem-resistant *A. baumannii* were explored, and gene analysis was conducted on carbapenem-resistant *A. baumannii* strains to probe into its genomic epidemiology in this paper.

In this study, the clinical specimens of carbapenem-resistant *A. baumannii* were mainly distributed in sputum, followed by throat swabs, and they mainly came from the Department of Respiratory Medicine and the ICU. Respiratory tract infection was dominant. These results are attributed to the strong viability and

Table 4. Infection sites of carbapenem-resistant *A. baumannii.*

Infection site	Cases (n)	Ratio (%)
Respiratory tract infection	62	51.67
Urinary tract infection	28	23.33
Intracranial infection	8	6.67
Surgical site infection	7	5.83
Bloodstream infection	6	5.00
Skin and soft tissue infections	5	4.17
Others	4	3.33

Table 5. Detection results of β-lactamase genes and active efflux system genes of *A. baumannii.*

Gene	Strains (n)	Ratio (%)
β-lactamase genes		
AmpC	99	82.5
OXA-23	85	70.83
IMP-1	65	54.17
Active efflux system genes		
adeB	94	78.33
adeJ	112	93.33
adeG	108	90.00

low pathogenicity of carbapenem-resistant *A. baumannii* [13]. Due to strong viability, carbapenem-resistant *A. baumannii* can be widely distributed and attach to and invade the human body, including the respiratory tract, gastrointestinal tract, and oral cavity, so the specimens come from a wide range of sources [14]. Moreover, carbapenem-resistant *A. baumannii* can form biofilm and long survive in ventilators and other equipment. As a result, when susceptible people receive the respiratory support therapy, carbapenem-resistant *A. baumannii* can adhere to and invade tracheal epithelial cells by virtue of fimbriae on the surface, infecting the respiratory tract and further invading the lungs. Therefore, the specimens of *A. baumannii* come primarily from sputum [15]. Since carbapenem-resistant *A. baumannii* has low pathogenicity, only the immunocompromised population is its target, and these patients mostly come from the Department of Respiratory Medicine and ICU [16], so the Department of Respiratory Medicine and ICU are the main sources of carbapenem-resistant *A. baumannii* -positive patients.

In addition, the resistance of carbapenem-resistant *A. baumannii* to commonly used antibiotics was analyzed in this study, and the results showed that, except for ampicillin-sulbactam and colistin, its resistance to commonly used antibiotics was different during the three years, generally showing an upward trend. The reason is that critically ill patients are more resistant to carbapenem antibiotics than patients with mild symptoms, and critically ill patients may use this drug, so the drug resistance rate increases [17]. The drug resistance of *A. baumannii* may also be caused by strain variation or the emergence of drug resistance genes within the three years [18]. It can be inferred that ampicillin-sulbactam and colistin can be used to treat *A. baumannii* infections.

Moreover, the genomic epidemiology of carbapenem-resistant *A. baumannii* was explored in this study, and it was found that the strains with β-lactamase genes (AmpC, OXA-23, and IMP-1) and active efflux system genes (adeB, adeJ, and adeG) occupied a higher proportion, indicating that carbapenem-resistant *A. baumannii* generally has β-lactamase genes and active efflux system genes. The reason is that excess β-lactamases play an important role in multi-drug resistance mechanism of *A. baumannii*. β-lactamases can hydrolyze carbapenems, and most OXA-type β-lactamases can also hydrolyze carbapenems. OXA-type β-lactamases are involved in drug resistance and can be combined with other drug resistance mechanisms, further enhancing the drug resistance of *A. baumannii* [19]. The efflux system is a drug efflux system, which attaches to the outer membrane of the bacteria and requires special energy. It pumps drugs out of the bacteria to reduce the drug content in the bacteria, thus producing drug resistance of *A. baumannii* [20].

Our findings highlight the significant yearly increase in antibiotic resistance and the prevalent co-occurrence of β-lactamase and efflux system genes in Carbapenem-resistant *A. baumannii*. Clinically, these insights suggest the urgent need for tailored antibiotic stewardship programs, routine genomic surveillance, and enhanced infection control measures. Specifically, understanding the genetic resistance profiles will enable clinicians to select more effective targeted antibiotic therapies, thereby reducing empirical treatment failures and limiting further resistance spread in healthcare settings.

Conclusions

In conclusion, the clinical specimens of carbapenem-resistant *A. baumannii* are mainly distributed in sputum and throat swabs and come from the Department of Respiratory Medicine and ICU, and the major infection site is the respiratory tract. Carbapenem-resistant *A. baumannii* mostly have β-lactamase genes and active efflux system genes, which can mediate the production of strong drug resistance in *A. baumannii*. These results are valuable for the research and development of new therapeutic drugs and can contribute to infection control. Future research should focus on investigating the mechanisms underlying gene co-expression and exploring innovative therapeutic agents or combination therapies that specifically target these genetic resistance pathways.

Funding

This study was financially supported by Project No. SGY2021A06.

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Conflict of interest

No conflict of interest is declared.

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