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ORIGINAL RESEARCH

Risk and Prognostic Factors for Bloodstream Infections Due to Clonally Transmitted *Acinetobacter baumannii* ST2 with *armA*, *bla*OXA-23, and *bla*OXA-66: A Retrospective Study

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Background: Multidrug-resistant *Acinetobacter baumannii* (MDR-AB) is a major cause of bacterial bloodstream infections (BSIs), associated with high morbidity and mortality. The risk and prognostic factors for BSIs caused by clonally transmitted *A. baumannii* ST2, carrying *armA, bla*OXA-23 and *bla*OXA-66, remain unclear.

Methods: We retrospectively analyzed 97 hospitalized patients with *A. baumannii* BSI (January 2019–May 2022). Whole-genome sequencing and bioinformatic analysis characterized the strains. Clinical data were reviewed to identify risk factors for secondary BSIs, *A. baumannii* BSIs with mixed infections involving extra-bloodstream pathogens, and mortality predictors.

Results: High-risk clone sequence type (ST) 2 was identified in 87 isolates (89.7%), with 86 exhibiting clonal dissemination. Carbapenems and aminoglycosides resistance occurred in 78.4% of strains, linked to *armA, bla*OXA-23, and *bla*OXA-66. Patients' median age was 56.6 years (range: 11–93), with males comprising 62.9%. Elderly patients (>65 years) accounted for 40.2%, 85.6% had hospital stays >10 days, and 84.5% had ICU admissions. Adverse outcomes were observed in 55.7% of cases. ICU admission (OR = 5.144, 95% CI: 1.290–20.511, P = 0.020) and open injury (OR = 5.998, 95% CI: 1.164–30.892, P = 0.032) were specific risk factors significantly associated with BSIs, while the presence of three or more underlying diseases (OR = 6.419, 95% CI: 2.074–19.866, P = 0.001) was significantly associated with increased mortality risk.

Conclusion: The majority of *A. baumannii* strains causing BSIs in this study belonged to multidrug-resistant ST2 lineage, harboring *armA, bla*OXA-23 and *bla*OXA-66. Risk factors for secondary and mixed infections included prolonged ICU stays, mechanical ventilation (≥7 days), and open injuries, while poor prognosis was linked to severe comorbidities and extended invasive ventilation. Targeted infection control strategies are critical to reducing mechanical ventilation duration and managing open injuries in high-risk patients.

Keywords: *Acinetobacter baumannii*, bloodstream infection, risk factors, molecular epidemiology, secondary infections, mixed infections

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Background

Acinetobacter baumannii infections pose a significant challenge in healthcare due to their extraordinary survival capabilities and rapid development of resistance to major antibiotic classes. This adaptability facilitates clonal transmission within healthcare settings, leading to severe infections, increased mortality, prolonged hospital stays, and escalated healthcare costs.[1,2] Among various *A. baumannii* clones, the ST2 lineage has been identified as the most prevalent globally, contributing disproportionately to hospital outbreaks and carbapenem-resistant infections.[3]

Resistance in *A. baumannii* is primarily driven by carbapenem-hydrolyzing β-lactamases (CHβLs), with *bla*OXA-23 and *bla*OXA-66 recognized as the predominant CHβLs responsible for carbapenem resistance.[4] Furthermore, the presence of the 16S rRNA methyltransferase gene *armA* confers high-level resistance to aminoglycosides, further limiting treatment options and exacerbating clinical outcomes.[5] These resistance determinants are often carried by clonally transmitted strains, particularly ST2, highlighting the critical need to better understand their epidemiology and clinical impact.

Bloodstream infections (BSIs) caused by *A. baumannii* are among the most frequent and severe manifestations of *Acinetobacter* infections.[6] These infections often originate as secondary infections from primary sites, including the lower respiratory tract, urinary tract, and intravascular devices, with wounds being less common sources.[7] Critically ill patients in intensive care units (ICUs) are particularly vulnerable, with BSIs frequently occurring alongside infections in other body sites.[8] Key risk factors for BSIs include ICU stays, mechanical ventilation, broad-spectrum antibiotic use, central venous catheterization, invasive procedures, and prolonged hospital stay.[9–11] However, the specific factors contributing to secondary *A. baumannii* BSIs, particularly those involving clonally transmitted strains and concurrent infections with other pathogens, remain inadequately understood.

In addition, the prognostic factors associated with *A. baumannii* BSIs, such as imipenem resistance, ICU admission, pneumonia, diabetes, and septic shock, have been studied to some extent.[12] Yet, a comprehensive understanding of the impact of specific resistance genes (*armA, bla*OXA-23, and *bla*OXA-66) and clonal transmission patterns on clinical outcomes is lacking.

This study addresses these critical gaps by employing whole-genome sequencing (WGS) to investigate the molecular epidemiology of clonally transmitted *A. baumannii* ST2 strains harboring *armA, bla*OXA-23, and *bla*OXA-66. Through a retrospective analysis of clinical data from 97 patients diagnosed with *A. baumannii*-caused BSIs, we sought to identify specific risk factors for secondary infections, *A. baumannii* BSIs accompanied infection with other pathogens, and prognostic indicators associated with mortality. This exploration provides valuable insights into the mechanisms driving clonal transmission, resistance, and their clinical implications, offering a foundation for targeted infection control strategies and improved patient outcomes.

Materials and Methods

Study Design

This retrospective, single-center study was conducted at Nanjing Drum Tower Hospital, a 3800-bed tertiary comprehensive hospital in Nanjing, China, from January 1, 2019, to May 31, 2022. Ninety-seven patients diagnosed with *A. baumannii* BSIs were included. Inclusion criteria were as follows: (1) Positive blood culture for *A. baumannii*, (2) Clinical symptoms consistent with BSIs (eg, fever, chills, or hemodynamic instability), and (3) Only the first episode was included for patients with multiple episodes. Exclusion criteria were as follows: (1) Patients with blood cultures indicating colonization or contamination, based on clinical assessment and microbiological criteria (see definitions below). (2) Absence of clinical signs or symptoms suggestive of BSI. (3) Patients receiving palliative care or with incomplete medical records. This study was performed in accordance with the principles of the Declaration of Helsinki. Approval was granted by the Ethics Committee of Nanjing Drum Tower Hospital (2023–390). Informed consent for participation was waived by the Regional Ethics Committee of Nanjing Drum Tower Hospital.

Definitions

BSI was defined based on clinical and microbiological criteria, including a positive blood culture for *A. baumannii* and compatible clinical symptoms such as fever, chills, or hemodynamic instability. Colonization was defined as the presence of *A. baumannii* in blood cultures without accompanying clinical symptoms or signs of infection. Contamination was defined as a single positive blood culture for *A. baumannii* in the absence of clinical symptoms or when an alternative source of infection was identified. *A. baumannii* BSIs with mixed infections involving extra-bloodstream pathogens were defined as an *A. baumannii* BSI occurring concurrently with other pathogens identified from blood or other clinical specimens (eg, sputum, urine, or wound cultures). The 30-day all-cause mortality rate refers to the proportion of patients who die from any cause within 30 days of the diagnosis of *A. baumannii* BSI. While the 90-day all-cause mortality rate refers to the proportion of patients who die from any cause within 90 days after the diagnosis. These definitions align with the guidelines outlined in the CDC manual (https://www.cdc.gov/nhsn/pdfs/pscmanual/4psc_clabscurrent.pdf accessed December 22 2019).

Data Collection

Clinical data were retrospectively collected from electronic medical records, including demographic characteristics (age, sex, comorbidities, and recent surgical history), Laboratory findings (infection indicators such as CRP and PCT, antimicrobial susceptibility results, and culture findings from various specimens collected before and after the first positive *A. baumannii* blood culture), hospitalization details (length of hospital stay, ICU admission, and invasive procedures such as mechanical ventilation, and central venous catheterization performed within 7 days of BSI diagnosis), as well as information on underlying diseases and the 30-day and 90-day all-cause mortality rates. This study was conducted in accordance with the principles of the Declaration of Helsinki and which was approved by the Ethics Committee of Nanjing Drum Tower Hospital (2023–390) and an exemption from the informed consent was obtained. All data were anonymized before the analysis to safeguard patient privacy.

Microbiology Identification and Susceptibility Testing

Initially, bacterial identification was performed using Vitek 2.0 and matrix-associated laser desorption ionization-time of flight mass spectrometry (MALDI-TOF MS) (BioMerieux, Craponne, France). Confirmation was achieved via average nucleotide identity (ANI) based on whole-genome sequencing (WGS).

Antimicrobial susceptibility testing was conducted using the micro-broth dilution method, using the following agents: imipenem, cefepime, ceftazidime, ceftriaxone, ampicillin/sulbactam, piperacillin/tazobactam, amikacin, gentamicin, tobramycin, sulfamethoxazole-trimethoprim, ciprofloxacin, levofloxacin, tigecycline, and polymyxin B. Results were interpreted according to CLSI 2022 guidelines,[13] with *Pseudomonas aeruginosa* ATCC 27853 used as a quality control strain.

Whole-Genome Sequencing and Phylogenetic Analysis

Genomic DNA was extracted using a commercial kit (Tiangen Biochemical Technology Co., Ltd., Beijing, China) and sequenced on the Illumina MiSeq platform (Illumina, San Diego, CA, USA) provided by Beijing Tiangen Biochemical Technology (Beijing, China). *De novo* assembly was performed using CLC Genomics Workbench version 21.0.4 (Qiagen, Hilden, Germany).

Mobile genetic elements and their relationships with antimicrobial resistance genes were identified using MobileElementFinder (https://cge.food.dtu.dk/services/MobileElementFinder/). Phylogenetic analysis was conducted using Prokka, Roary, jModelTest 2, and RaxML ng, along with iTOL Version 6.5.8 software, as detailed in a previous study.[14] The latest version of the Virulence Factor Database (VFDB) (http://www.mgc.ac.cn/VFs/download.htm) was used to compare nucleotide coding sequence files extracted from 97 genomes in batches with BLAST software to obtain a detailed distribution of virulence factors (VFs) in all genomes, with the e-value set at "1e-5, identity ≥ 70%, coverage ≥ 90%, and match length ≥ 30%.

Statistics

Data analysis was performed using SPSS 27.0 (IBM, Armonk, NY, USA). Categorical variables were analyzed using the Pearson chi-square test or Fisher's exact test, while continuous variables were analyzed using *t*-tests or the Mann–Whitney *U*-tests. Odds ratios (ORs) and 95% confidence intervals (CIs) were calculated, with significance set at $p < 0.05$. Univariate and multivariate logistic regression analyses were used to identify risk and prognostic factors.

Data Availability

Draft genome assemblies have been deposited in the NCBI database under BioProject number PRJNA989674.

Results

Clonal Transmission and Antimicrobial Resistance

Of the 97 *A. baumannii* strains analyzed, 89.7% (n = 87) were identified as ST2 (Figure 1). High resistance to imipenem was observed in 87 strains (89.7%), and over 78.0% exhibited resistance to aminoglycosides. However, susceptibility to tigecycline and polymyxin B remained. Phylogenetic analysis revealed a close genetic relationship among 86 of the 87 ST2 strains, suggesting clonal dissemination within the healthcare setting. In contrast, non-ST2 strains demonstrated greater genetic diversity, underscoring the unique clonal nature of ST2 dissemination (Figure 1).

Resistance Determinants and Virulence Factors

All ST2 strains harbored *bla*OXA-23, with the majority also carrying *bla*OXA-66 and *bla*ADC-25 (Figure 2A). Notably, *armA* was detected in 78.4% (n = 76) of strains (Figure 2B). Genomic analysis identified various insertion sequences (ISs) and transposons co-located with *armA* on the same contigs, forming diverse mobile elements (Figures 3 and 4). Additionally, eight categories of VFs were identified, with adherence and effector delivery systems being most prevalent.

Clinical Epidemiology

The patient cohort had a median age of 56.6 years (range: 11–93), with 62.9% being male and 40.2% classified as elderly (aged >65 years). Prolonged hospital stays (>10 days) were common (85.6%), and 84.5% of patients required ICU admission. Secondary infections were reported in 68.0% of cases, primarily originating from the lower respiratory tract (46.4%) and associated with catheterizations (13.4%) (Figure 5A). Organ failure was the most common underlying condition, affecting 66% of patients (Figure 5C). Invasive procedures were frequently performed, with 47.4% of patients undergoing intravenous catheterization and 43.3% having requiring continuous urinary catheterization for more than a week (Figure 5D). The 30-day and 90-day all-cause mortality rates post-index culture were 34.0% and 53.6%, respectively.

Risk Factor Analysis

Univariate and multivariate logistic regression analyses identified several independent risk factors. ICU admission (OR = 5.144, 95% CI: 1.290–20.511, P = 0.020), open injuries (OR = 5.998, 95% CI: 1.164–30.892, P = 0.032), and prolonged venous catheterization (≥7 days) (OR = 4.703, 95% CI: 1.217–18.181, P = 0.025) were linked to secondary *A. baumannii* BSIs (Table 1). Similar risk factors, including ICU admission (OR = 7.025, 95% CI: 1.485 ~ 33.220, P = 0.014), length of hospitalization (OR = 1.093, 95% CI: 1.015–1.177, P = 0.018) and open injury (OR = 5.928, 95% CI: 1.130–31.083, P = 0.035), were linked to BSIs with *A. baumannii* BSIs concurrent with infections caused by other pathogens at different sites (Table 2). Poor prognosis was predicted by the presence of three or more underlying diseases (OR = 6.419, 95% CI: 2.074–19.866, P = 0.001) and invasive mechanical ventilation lasting more than a week (OR = 4.402, 95% CI: 1.142–14.308, P = 0.030) (Table 3).

Discussion

A. baumannii is globally recognized for its formidable multidrug resistance and capacity for clonal dissemination, particularly in hospital environments.[1,15] Our comprehensive genomic and epidemiological analysis of 97 *A. baumannii* strains from BSIs aimed to elucidate risk factors associated with secondary *A. baumannii* BSIs, *A. baumannii* BSIs concurrent with infections caused by other pathogens at different sites, and prognostic factors over a three-year observational period.

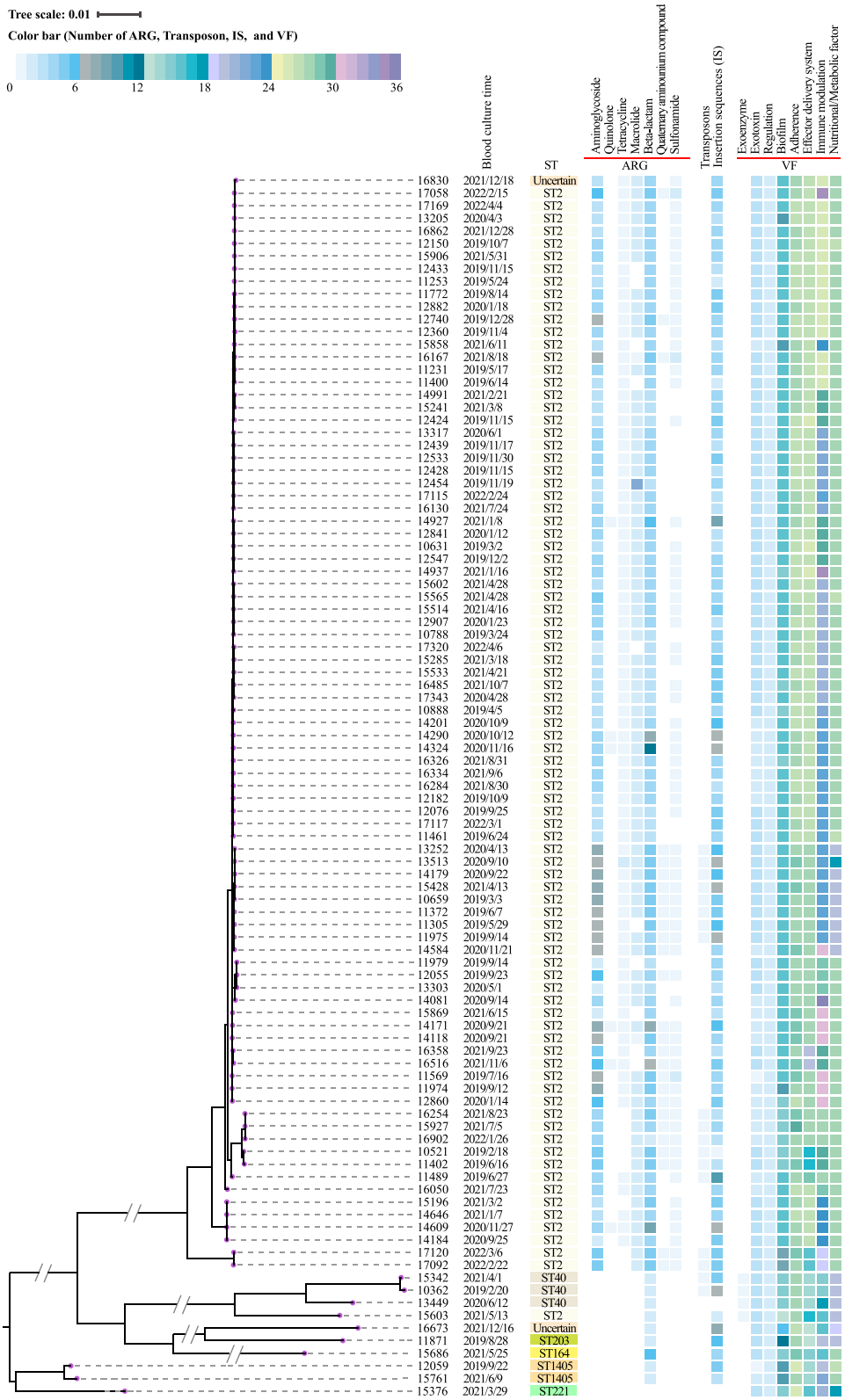

Figure 1 Phylogenetic tree and heatmap analysis of 97 *Acinetobacter baumannii* isolates causing bloodstream infections. The phylogenetic tree of 97 *A. baumannii* isolates was annotated with corresponding dates of blood culture isolation and sequence types (STs). The phylogenetic tree scale indicates genetic distance (0.01). The heatmap highlights the presence of antimicrobial resistance genes (ARGs), insertion sequences (IS), and virulence factors (VFs) across the isolates. The color bar at the bottom represents the abundance of ARGs, IS elements, and VFs in individual isolates, with values ranging from 0 to 36. Key resistance mechanisms include aminoglycosides, quinolones, tetracyclines, macrolides, β-lactams, and sulfonamides, alongside virulence traits such as biofilm formation, adherence, and effector delivery systems.

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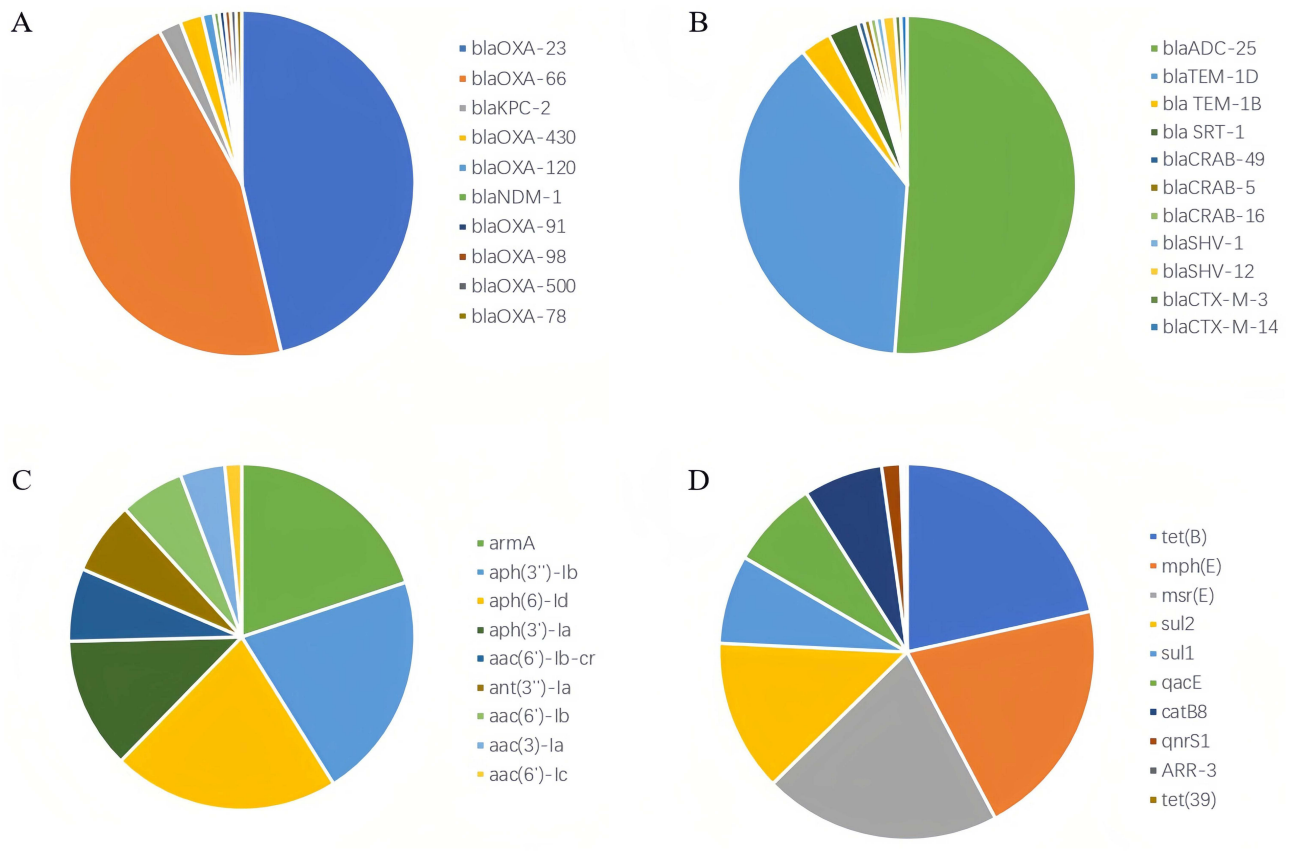

Figure 2 The Distribution of various antimicrobial resistance determinants among 97 *Acinetobacter baumannii* isolates associated with bloodstream infections. (**A**) The distribution of carbapenem hydrolyzing ß-lactamase encoding genes; (**B**) The distribution of other ß-lactamase encoding genes; (**C**) The distribution of aminoglycoside resistant determinants. (**D**) the distribution of the remaining resistant determinants, including genes responsible for resistance to other antibiotic classes such as tetracyclines, macrolides, and sulfonamides.

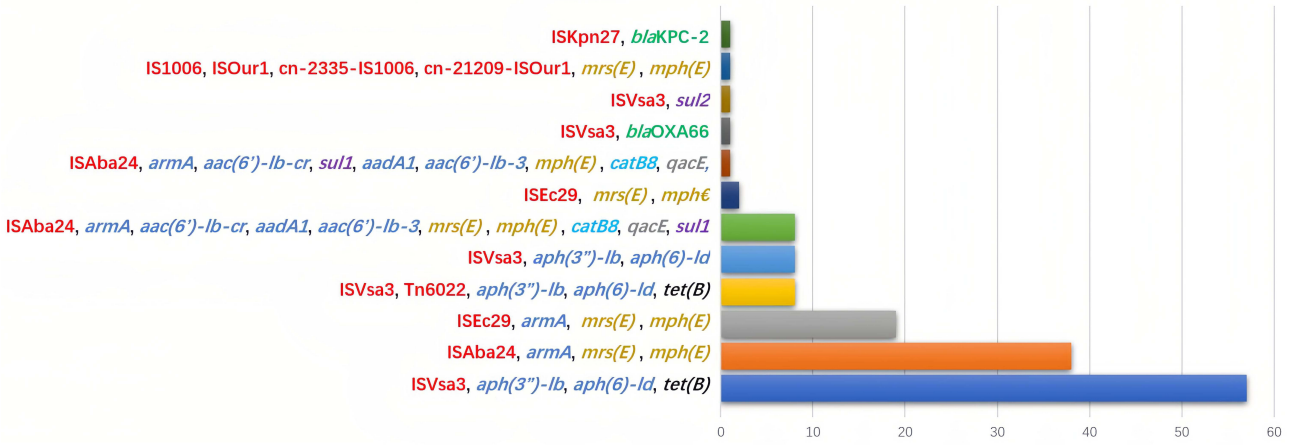

Figure 3 Co-Localization of Resistance Determinants and Insertion Sequences within the same contigs of *Acinetobacter baumannii*. Left array represents the composition of resistance determinants and insertion sequences on individual contigs; Red, Insertion sequences (IS), indicating mobile genetic elements; Green, carbapenem-hydrolyzing β-lactamase genes; Light Blue, aminoglycoside resistance genes; Black, tetracycline resistance genes; Purple, sulfonamide resistance genes. Dark Yellow, erythromycin resistance genes; Gray: quaternary amine disinfectant resistance genes.

Our findings indicated that over 89.0% of the strains were resistant to carbapenems, consistent with the high prevalence of *bla*OXA-23 and *bla*OXA-66 in our study, which is similar to the previous report.[3] This resistance profile mirrors global patterns observed in ST2 clones of *A. baumannii*. Notably, our study revealed a significant prevalence of

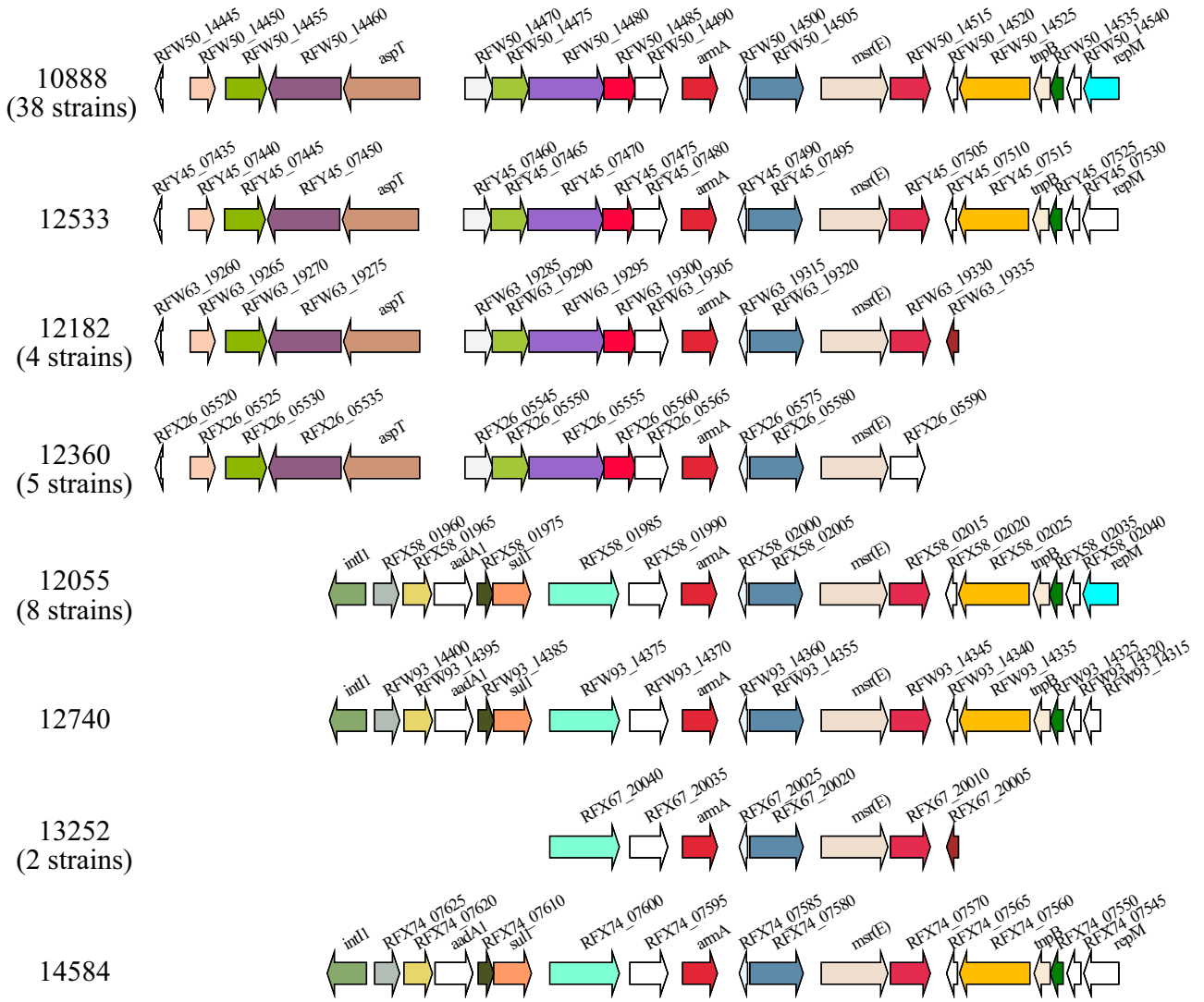

Figure 4 The flanking sequence around *armA* gene within *Acinetobacter baumannii* causing bloodstream infections. Genes around *armA* were shown by different blocks; The same color represents homologous genes and unique genes are shown in white.

armA in conjunction with *bla*OXA-23, *bla*OXA-66, and *bla*ADC-25, resulting in a predominant resistance gene profile. Similar combinations have been documented in various global ST2 clones, including reports from Switzerland,[16] South America,[17] Pakistan,[18] Yemen,[19] Vietnam,[20] and Korea.[21] Studies from Europe and the United States have also identified widespread carbapenem resistance in ST2 clones, particularly linked to *bla*OXA-23.[22,23] Likewise, data from Europe and the United States indicate the clonal dissemination of ST2 strains, highlighting the global significance of this clone.[24] Thus, the frequent co-occurrence of *armA* with *bla*OXA-23, *bla*OXA-66, and *bla*ADC-25 in these clones suggests a robust mechanism driving clonal dissemination, particularly in China, where such a comprehensive report of clonal transmission within a single institution is unprecedented. Therefore, our study provides novel insights into the clonal dissemination of ST2 *A. baumannii* strains in a healthcare setting, particularly the widespread presence of *armA* and its association with mobile genetic elements, which has not been thoroughly explored in previous studies. Furthermore, our phylogenetic analysis revealed that 86 out of 87 ST2 strains displayed close genetic relatedness, indicating clonal spread within our facility. This genetic similarity suggests a common ancestral origin for these strains, pointing to multiple outbreaks during the study period which underscore the urgent need for enhanced infection control measures to mitigate the persistent threat posed by clonal dissemination of multidrug-resistant (MDR) *A. baumannii* in healthcare settings.

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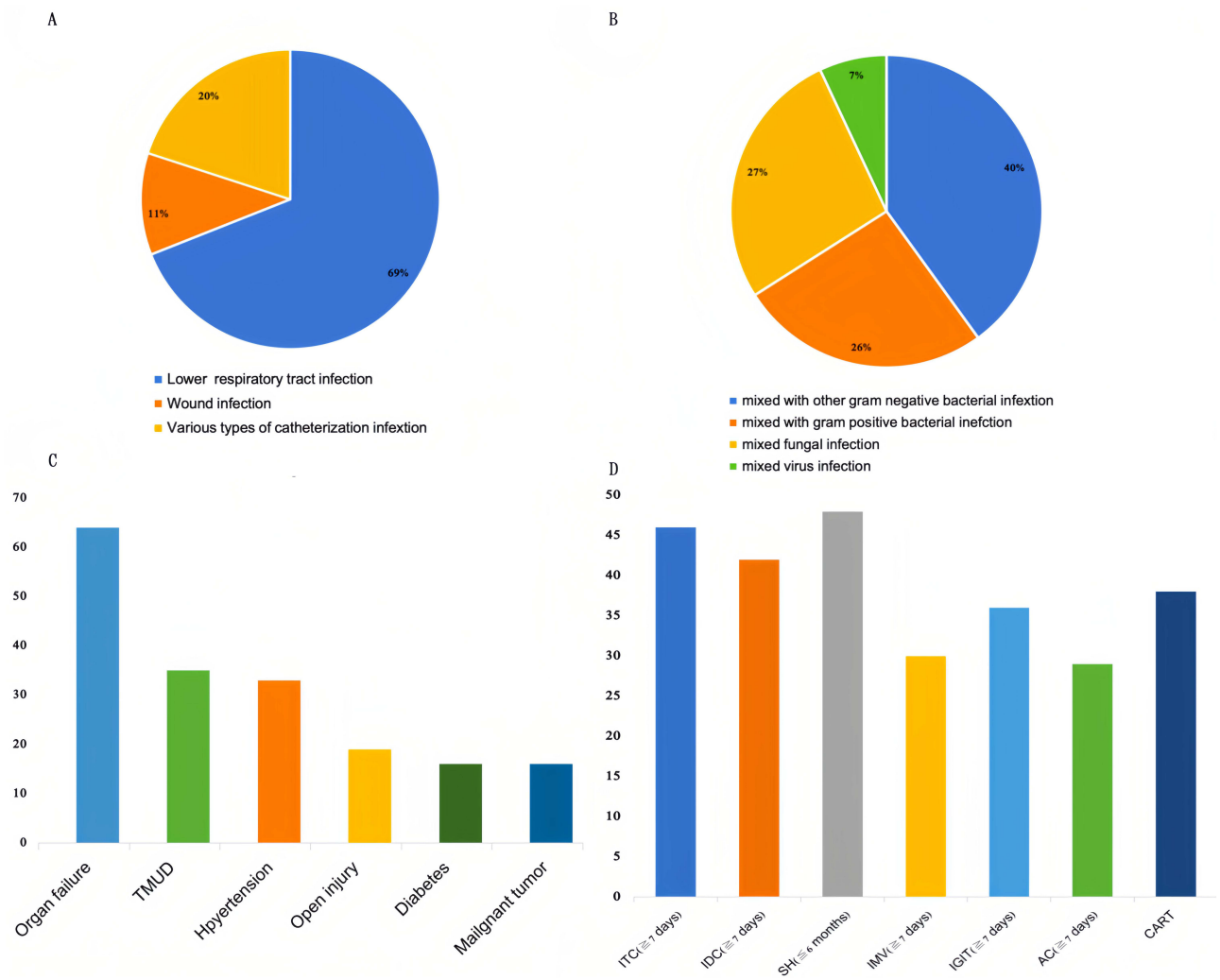

Figure 5 The clinical characterization of the 97 patients diagnosed with *Acinetobacter baumannii* causing bloodstream infections. (**A**) The primary resources of the 97 patients diagnosed with *Acinetobacter baumannii* bloodstream infections. (**B**) The mixed infections of the 97 patients diagnosed with *A. baumannii* bloodstream infections. (**C**) The underlying diseases of the 97 patients diagnosed with *A. baumannii* bloodstream infections. (**D**) The invasive procedures of the 97 patients diagnosed with *A. baumannii* bloodstream infections.

Abbreviations: TMUD, three or more underlying diseases; ITC, intravenous catheterization (≥7 days); IDC, indwelling catheter (≥7 days); SH, surgical history (≦6 months); IMV, invasive mechanical ventilation (≥7 days); IGIT, indwelling gastrointestinal tube (≥7 days); AC, arterial catheterization (≥7 days); CART, continuous renal placement therapy; F/CB, fiber/electronic bronchoscopy/CB.

The patient cohort in this study revealed critical clinical and epidemiological trends. The predominance of male patients and the high rate of ICU admissions reflect known vulnerabilities among critically ill populations.[25,26] The observed 55.7% mortality rate, higher than reported in other studies,[27,28] may be attributable to the prevalence of co-infections and secondary BSIs, as well as the widespread resistance patterns observed in this cohort.

Interestingly, most BSIs were secondary infections originating from the lower respiratory tract, consistent with hospital-acquired infection pathways.[29,30] This may relate to tracheal intubation or tracheostomy, performed in all patients, likely contributed to impaired airway secretion clearance, elevating the risk of secondary BSIs.[16] Furthermore, over 70% of *A. baumannii* BSIs were associated with co-infections, primarily involving other gram-negative bacteria such as *Escherichia coli*. This prevalence aligns with prior reports,[31] highlighting the complexity of managing mixed infections, which exacerbate disease severity and likely contribute to the high mortality rates observed in our study. In addition, our study identified several key risk factors for secondary *A. baumannii* BSIs, including ICU admission, open injuries, and prolonged venous catheterization (≥7 days). These factors are consistent with those observed in other critical conditions, such as COVID-19-associated acute respiratory distress syndrome, where ICU

Table 1 Univariate- and Multivariate and Analysis for the Risk Factors of Secondary Bloodstream Infections Caused by *Acinetobacter baumannii*

Variables	Secondary BSIs (n = 66)	Primary BSIs (n = 31)	Chi-Square		Univariate Analysis		Multivariate Analysis	
			t/X^2	P Value	Risk Ratio (%95 CI)	P Value	Risk Ratio (%95 CI)	P Value
General Information								
Agender (male) (%)	39 (59.1%)	22 (71.0%)	1.275[a]	0.259				
Age	54.70 ± 19.656	60.65 ± 17.891	−1.427	0.157				
Department (ICU) (%)	61 (92.4%)	21 (67.7%)	9.830[a]	0.002	5.810 (1.781~18.954)	0.004	5.144 (1.290~20.511)	0.020
Length of Hospitalization	13.5 (20.5)	10 (7)	1.569	0.121				
Underlying disease								
Hypertension (%)	21 (31.8%)	12 (38.7%)	0.466[a]	0.504				
Diabetes (%)	10 (15.2%)	6 (19.4%)	0.271[a]	0.603				
Organ failure (%)	47 (71.2%)	17 (54.8%)	2.519[a]	0.112				
Open injury (%)	17 (25.8%)	2 (6.5%)		0.029	5.031 (1.083~23.359)	0.039	5.998 (1.164~30.892)	0.032
Three or more basic diseases (%)	24 (36.4%)	11 (35.5%)	0.007[a]	0.933				
Invasive Operations								
Continuous renal replacement therapy (%)	28 (42.4%)	10 (32.3%)	3.600[a]	0.058				
Fiber/electronic Bronchoscopy (%)	18 (27.3%)	6 (19.4%)	0.710[a]	0.399				
Surgical history (<=6 months) (%)	34 (51.5%)	14 (45.2%)	11.722[a]	0.001	1.900 (0.791~4.536)	0.148		
Invasive mechanical ventilation (≥7 days) (%)	25 (37.9%)	5 (16.1%)	4.671[a]	0.031	3.171 (1.078~9.324)	0.036	0.611 (0.134~2.783)	0.524
Venous catheterization (≥7 days) (%)	39 (59.1%)	7 (22.6%)	11.277[a]	0.001	4.952 (1.869~13.123)	0.001	4.703 (1.217~18.181)	0.025
Arterial intubation (≥7 days) (%)	25 (37.9%)	4 (12.9%)	6.278[a]	0.012	4.116 (1.288~13.154)	0.017	1.552 (0.310~7.772)	0.593
Indwelling catheter (≥7 days) (%)	33 (50.0%)	9 (29.0%)	3.777[a]	0.052				
Indwelling gastrointestinal tube (≥7 days) (%)	26 (39.4%)	10 (32.3%)	0.460[a]	0.498				

Notes: The value of $p < 0.05$ was set as the significance threshold (in italics).
Abbreviation: CI, confidence interval.

interventions significantly increase the risk of MDR *A. baumannii* outbreaks.[32] As we know that the widespread use of venous catheterization in ICU settings creates a favorable environment for bacterial colonization and subsequent bloodstream invasion.[33] In addition, extended catheterization often leads to the formation of a loose fibrin sheath on the catheter surface, providing a pathway for bacterial proliferation.[34] Interestingly, our findings reveal that not all invasive procedures confer the same level of risk. While prolonged catheterization and mechanical ventilation were significant risk factors, short-term arterial catheterization and thoracoabdominal drainage did not significantly impact infection likelihood, emphasizing the importance of procedure duration and context.[35]

The overlap between risk factors for secondary BSIs and mixed infections highlights shared vulnerabilities across infection scenarios. ICU stays and prolonged hospitalization were the most significant contributors, with invasive procedures playing a critical role in facilitating bacterial entry.[10,36,37] In fact, ICU stays were the most significant risk factor, with extended hospitalization also substantially increasing the carriage and consequent infection rates of *A. baumannii*.[30] These findings reinforce the critical role of hospital environment and patient management in the spread of this pathogen. Invasive procedures were notably implicated in facilitating the entry of *A. baumannii* into the bloodstream, particularly during interventions that breach the skin's integrity, such as catheter insertions.[26] The extended use of invasive mechanical ventilation disrupts the airway's normal microbial environment, significantly increasing the risk of infection.[38] This is particularly evident when the ventilation period exceeds 7 days, leading to the formation of a loose fibrous protein sheath on invasive devices, which serves as a breeding ground for *A. baumannii* colonization and subsequent infection.[37] Furthermore, the identification of specific risk factors, such as open injuries and prolonged catheterization, offers new perspectives on secondary BSIs and mixed infections.

Mortality rates in this study, particularly among patients undergoing prolonged mechanical ventilation, align with previous studies demonstrating a direct correlation between invasive interventions and poor outcomes.[39] Notably, the incidence of pandrug-resistant *A. baumannii* infections increased from 15.0% to 24.0% with more than 7 days of mechanical ventilation, and exceeded 50.0% when the duration extends beyond 14 days.[40] This correlation underscores the severe impact of prolonged mechanical support on patient outcomes, emphasizing the need for stringent infection

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Table 2 Univariate and Multivariate Analysis for the Risk Factors of *Acinetobacter baumannii* Bloodstream Infections Mixed with Other Pathogenic Infections

Variables	*A. baumannii* BSIs Accompanied Infection with Other Pathogens (n = 68)	Single *A. baumannii* BSIs (n = 29)	Chi-Square Test		Univariate Analysis		Multivariate Analysis	
General information			t/X^2	P values	Risk ratio (%95 CI)	P value	Risk ratio (%95 CI)	P value
Agender (male) (%)	42 (61.8%)	19 (65.5%)	0.123[a]	0.726				
Age	54.84 ± 18.991	60.72 ± 19.539	1.386	0.169				
Department (ICU) (%)	62 (91.2%)	20 (69.0%)	7.672[a]	0.006	4.650 (1.473~14.677)	0.009	7.025 (1.485~33.220)	0.014
Length of hospitalization	14 (20.25)	8 (6.75)	−1.855	0.064	1.084 (1.023~1.148)	0.007	1.093 (1.015~1.177)	0.018
Underlying disease								
Hypertension (%)	21 (30.9%)	12 (41.4%)	0.998[a]	0.318				
Diabetes (%)	12 (17.6%)	4 (13.8)	0.029	0.865				
Malignant tumors (%)	10 (14.7%)	6 (20.7%)	0.528[a]	0.467				
Organ failure (%)	49 (72.1%)	15 (51.7%)	3.745[a]	0.053	2.407 (0.978~5.924)	0.056		
Open injury (%)	17 (25.0%)	2 (6.9%)	4.230[a]	0.040	4.500 (0.967~20.941)	0.050	5.928 (1.130~31.083)	0.035
Three or more underlying disease (%)	25 (36.8%)	10 (34.5%)	0.046[a]	0.830				
Invasive operation								
Continuous renal replacement therapy (%)	33 (33.8%)	5 (17.2%)	2.722[a]	0.099	2.453 (0.828~7.272)	0.106		
Surgical history (<=6 months) (%)	35 (51.5%)	13 (44.8%)	0.359[a]	0.549				
Fiber/electronic bronchus (%)	20 (29.4%)	4 (13.8%)	1.891	0.169				
Invasive mechanical ventilation (≥7 days) (%)	25 (36.8%)	5 (17.5%)	3.627[a]	0.057	2.791 (0.946~8.237)	0.063		
Venous catheterization (≥7 days) (%)	38 (55.9%)	8 (27.6%)	6.528[a]	0.011	3.325 (1.293~8.551)	0.013	0.574 (0.113~2.922)	0.504
Arterial intubation (≥7 days) (%)	23 (33.8%)	6 (20.7%)	1.673[a]	0.196				
Indwelling catheter (≥7 days) (%)	35 (51.5%)	7 (24.1%)	6.186[a]	0.013	3.333 (1.258~8.862)	0.015	1.542 (0.334~7.121)	0.579
Indwelling gastrointestinal tube (≥7 days) (%)	30 (44.1%)	6 (20.7%)	4.781[a]	0.029	3.026 (1.093~8.376)	0.033	1.295 (0.332~5.202)	0.716

Note: The value of $p < 0.1$ was set as the significance threshold (in italics).
Abbreviation: CI, confidence interval.

Table 3 Univariate and Multivariate Analysis of Prognostic Risk Factors for *Acinetobacter baumannii* Bloodstream Infections

Variables	Death Group (n = 54)	Survival Group (n = 43)	x^2/t Values	P Values	Univariate Analysis		Multivariate Analysis	
General information					**Risk ratio (%95 CI)**	**P value**	**Risk ratio (%95 CI)**	**P value**
Agender (male) (%)	33 (61.1%)	28 (65.1%)	0.165[a]	0.685				
Age	58.87 ± 19.625	53.74 ± 18.592	1.308	0.194				
Department (ICU) (%)	50 (92.6%)	32 (74.4%)	6.048[a]	0.014	4.297 (1.259~14.662)	0.020	1.737 (0.375~8.038)	0.480
Underlying disease								
Hypertension (%)	19 (35.2%)	14 (32.6%)	0.074[a]	0.786				
Diabetes (%)	11 (20.4%)	5 (11.6%)	1.328[a]	0.249				
Malignant tumors (%)	11 (20.4%)	5 (11.6%)	1.328[a]	0.249				
Open injury (%)	8 (14.8%)	11 (25.6%)	1.762[a]	0.184				
Organ failure (%)	41 (75.9%)	23 (53.5%)	5.369[a]	0.020	2.742 (1.155~6.514)	0.022	2.370 (0.820~6.850)	0.111
Three or more underlying disease (%)	26 (48.1%)	9 (20.9%)	7.689[a]	0.006	3.508 (1.415~8.697)	0.007	6.419 (2.074~19.866)	0.001
Invasive operation								
Electronic/fiberoptic bronchoscopy (%)	18 (33.3%)	6 (14.0%)	3.457[a]	0.063				
Continuous renal replacement therapy (%)	25 (46.3%)	13 (30.2%)	2.451[a]	0.117				
Invasive mechanical ventilation (≥7 days) (%)	22 (40.7%)	8 (18.6%)	7.132[a]	0.034	3.110 (1.366~7.243)	0.009	4.042 (1.142~14.308)	0.030
Venous catheterization (≥7 days) (%)	32 (59.3%)	14 (32.6%)	6.532[a]	0.011	2.993 (1.275~7.026)	0.012	2.074 (0.597~7.204)	0.251
Arterial intubation (≥7 days) (%)	20 (37.0%)	9 (20.9%)	5.167[a]	0.023	2.701 (1.133~6.428)	0.025	0.842 (0.224~3.165)	0.799
Indwelling catheter (≥7 days) (%)	23 (42.6%)	19 (44.2%)	0.289[a]	0.591				
Indwelling gastrointestinal tube (≥7 days) (%)	22 (40.7%)	14 (32.6%)	1.058[a]	0.304				

Note: The value of $p < 0.05$ was set as the significance threshold (in italics).
Abbreviation: CI, confidence interval.

control and management strategies in settings with high-risk patients. The findings also highlight unique prognostic factors that could guide targeted clinical interventions, thereby contributing to a more comprehensive understanding of *A. baumannii*-related BSIs.

While this study provides valuable insights into the epidemiology and risk factors of *A. baumannii* BSIs, several limitations should be acknowledged. First, the retrospective design may lead to selection bias, potentially affecting the generalizability of the results. Second, the single-center setting and relatively small sample size restrict the extrapolation of our findings to broader populations. Third, the exclusion of antibiotic exposure data from the risk factor analysis is a significant limitation, given that all participants had received extensive antibiotic treatment prior to the onset of BSIs. This could have influenced both the microbial landscape and the outcomes of the infections, underscoring the need for cautious interpretation of the associated risk factors and their impacts.

Conclusion

This study demonstrated that the majority of *A. baumannii* strains causing BSIs in our cohort belonged to the ST2 lineage, a globally prevalent clone known for its multidrug resistance and clonal dissemination. These ST2 strains harbored multiple resistance genes, including *armA, bla*OXA-23, *bla*OXA-66, and *bla*ADC-25, contributing to their extensive resistance profiles. Most BSIs were secondary infections, frequently originating from the lower respiratory tract and co-occurring with other pathogenic infections, particularly gram-negative bacteria. Key factors associated with the occurrence of secondary and mixed infections included prolonged ICU stays, extended mechanical ventilation (≥7 days), and the presence of open injuries, while poor prognosis was linked to three or more comorbidities and prolonged use of invasive mechanical ventilation. To mitigate the incidence of such infections and improve patient outcomes, infection control strategies should focus on reducing the duration of mechanical ventilation and ICU stays, alongside rigorous management of open injuries and tailored care for high-risk patients with severe comorbidities or prolonged invasive interventions.

Data Sharing Statement

Draft genome assemblies have been deposited in the NCBI database under BioProject number PRJNA989674 and accession numbers SAMN36186295 to SAMN36186405.

Other data used and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Ethics Approval and Consent to Participate

This study was approved by the Ethics Committee of Nanjing Drum Tower Hospital (2023-390) in accordance with the principles of the Declaration of Helsinki, and an exemption from informed consent was obtained. All data were anonymized before the analysis to safeguard patient privacy.

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Disclosure

The authors declare that they have no competing interests.

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