Infection and Drug Resistance

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ORIGINAL RESEARCH

Clinical Characteristics and Independent Risk Factors for Multidrug-Resistant *Klebsiella pneumoniae* Bloodstream Infections: A Retrospective Analysis from China

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Purpose: This study aimed to analyze the clinical characteristics and risk factors of multidrug-resistant *Klebsiella pneumoniae* bloodstream infections (MDR KP-BSI) compared to non-MDR KP-BSI among adult patients in China, providing guidance for clinicians to prevent MDR KP-BSI.

Patients and Methods: A retrospective analysis of 240 adult patients with KP-BSI (2019–2023) was conducted. Clinical data were analyzed using multivariable logistic regression to identify risk factors.

Results: MDR KP-BSI prevalence was 22.5% (54/240). The MDR KP-BSI group had higher rates of comorbidities (hemiplegia, COPD/severe asthma, chronic cardiac insufficiency, cerebrovascular accident) and higher disease severity scores (APACHE II, SOFA, Pitt bacteremia, Charlson index, all $P<0.05$). Treatment-related factors (antibiotic exposure, ICU admission, nutrition support, invasive procedures) were more frequent in the MDR KP-BSI group ($P<0.05$). Pulmonary origin was significantly more common (42.6% vs 12.4%, $P<0.05$), while liver origin was less common (1.9% vs 24.2%, $P<0.05$) in MDR versus non-MDR KP-BSI. MDR KP-BSI patients had significantly worse outcomes: higher 7-day (40.7% vs 11.8%, $P<0.001$), 14-day (35.2% vs 10.8%, $P<0.001$), and 28-day mortality (27.8% vs 8.6%, $P<0.001$), and prolonged hospitalization [26.5 days (14.0, 64.5) vs 13.0 days (8.0, 23.0), $P<0.001$]. Multivariable analysis identified independent risk factors: recent antibiotic exposure (adjusted OR [aOR] 7.025; 95% CI 2.695–18.313), cerebrovascular accident history (aOR 3.095; 95% CI 1.054–9.903), and pulmonary infection source (aOR 2.941; 95% CI 1.101–7.895).

Conclusion: These predictors emphasize the need for antibiotic stewardship, infection control, and early interventions in high-risk patients to reduce MDR KP-BSI incidence.

Keywords: *Klebsiella pneumoniae*, multidrug-resistant, bloodstream infections, risk factors

Introduction

Bloodstream infections (BSI) have emerged as a major challenge in global public health due to their increased treatment costs and diagnostic uncertainty.[1] *Klebsiella pneumoniae* (KP) is a leading pathogen of BSI and ranks as the second most common pathogen globally, according to epidemiological data.[2] KP infections are associated with high all-cause mortality, especially in cases of multidrug-resistant (MDR) strains, where the mortality rate is significantly elevated.[3]

Received: 16 May 2025
Accepted: 2 August 2025
Published: 10 August 2025

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Notably, the drug resistance progress of KP in Enterobacteriaceae is particularly prominent, making it a pathogen of great concern in clinical practice.[4]

According to the SENTRY Antimicrobial Surveillance Program (2013–2019) analysis of 1882 global BSI cases, KP is the most common pathogen causing MDR BSI.[1] Further studies have shown that patients with MDR-KP infections have significantly higher mortality rates than those with other Gram-negative bacterial infections, especially in cases of BSI, where the mortality rate can be as high as 40%-50%.[5] The drug resistance progression of KP and its limited antibiotic treatment options have made it a major public health threat to global healthcare systems over the past decade.[6] Therefore, a comprehensive analysis of the risk factors for KP, particularly MDR KP-BSI, is essential for optimizing infection control strategies, improving clinical outcomes, and reducing mortality rates.

Clinical practitioners face a dual challenge in treating MDR KP-BSI: the complexity of treatment and delayed decision-making. The primary cause of treatment delays is delayed surveillance culture results, typically requiring 5 days to obtain final results.[7] This delay not only hinders the timely implementation of infection control measures but also increases treatment costs and further complicates the diagnosis. Consequently, MDR KP-BSI has become an increasingly serious issue in global public health. Despite existing research on bloodstream infections caused by multidrug-resistant bacteria, these studies have not specifically focused on KP.[8,9] Currently, there is insufficient evidence-based guidance for clinicians to tailor treatment strategies for patients with MDR KP-BSI based on their clinical characteristics. This knowledge gap limits the comprehensive understanding of risk factors for MDR KP-BSI and hinders the development of effective interventions for high-risk populations.

Our study is the first to focus on KP-BSI and systematically explore the clinical risk factors for MDR KP-BSI, aiming to identify key factors linked to its occurrence. We hope our findings will help clinicians assess the risk of MDR in KP when initial blood culture results indicate a KP infection but before antibiotic susceptibility results are available. Our findings provide essential decision-making support for clinicians, helping to optimize empirical antibiotic treatment strategies, reduce mortality rates, and alleviate the burden on public health systems.

Materials and Methods

Clinical Data

This single-center retrospective cohort study was conducted from January 2019 to December 2023 at Taizhou Municipal Hospital (Taizhou University Affiliated Municipal Hospital), School of Medicine, Taizhou University, a 1200-bed teaching hospital in Taizhou, China. The present study received human research ethics approval (No. LWYJ2023021) from its Ethics Committee and maintained patient confidentiality. Due to the retrospective nature of the study, the Ethics Committee waived the requirement for patient consent. Additionally, patient consent for publication was not required, as the study did not collect personal information.

Patients included in the study were required to meet the BSI definition. For cases in which the same patient had multiple positive blood cultures, only data from the first positive result were recorded for subsequent statistical analysis. The timestamp for blood culture recording refers to the initial blood sample collection time, not the report issuance time. Exclusion criteria were as follows: a) age<18 years; b) incomplete or missing case data; c) discharge within 24 hours; d) non-pathogenic bacteria considered. Of the 526 blood culture results initially screened, 240 met the inclusion criteria. Of these, 54 were MDR KP-BSI cases and 186 were non-MDR KP-BSI cases (Figure 1).

Data Collection

This study collected clinical data from patients via the hospital's electronic medical record system. Baseline characteristics included age, sex, comorbidities, ward of hospitalization, Charlson Comorbidity Index (CCI), Sequential Organ Failure Assessment (SOFA) score, Acute Physiology and Chronic Health Evaluation II (APACHE II) score, and Pitt bacteremia score (all assessed within 24 hours of BSI onset), and routine laboratory parameters. We additionally recorded clinical operations, previous treatments within 2 weeks prior to infection, occurrence of septic shock at BSI onset, bloodstream infection sources, and KP antimicrobial susceptibility profiles. Data collection was performed by a single physician to ensure consistency and reliability.

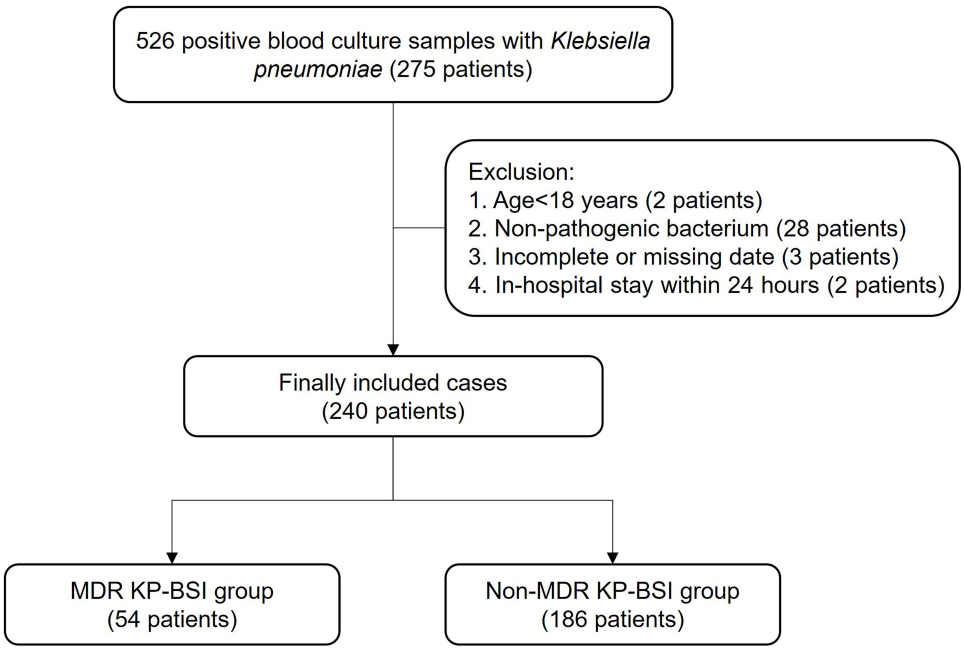

Figure 1 Patient screening and enrollment flowchart for the retrospective cohort study of MDR KP-BSI. Final cohort (n=240) stratified after exclusions detailed in the diagram.
Abbreviations: MDR, multidrug-resistant; KP-BSI, *Klebsiella pneumoniae* bloodstream infections.

Species Identification and Antibiotic Sensitivity Test

Blood cultures were processed using the BacT/ALERT 3D system (Becton-Dickinson, Sparks, MD, USA). Species identification was conducted with Bruker Daltonics DataAnalysis. Antibiotic susceptibility testing was performed using either the VITEK 2 system (AST-GN334/AST-GP67 cards) or the Kirby–Bauer disk diffusion method (Oxoid, UK), following Clinical and Laboratory Standards Institute (CLSI) guidelines.

Definitions

KP-BSI was defined as the isolation of KP in a blood culture accompanied by at least one of the following: fever, chills, or hypotension, combined with other corresponding clinical symptoms/signs, while excluding specimen contamination.[10,11] MDR-KP is defined as KP strains isolated from blood cultures that are resistant to three or more antimicrobial agents.[12] Septic shock was defined as the need for vasopressors to maintain a mean arterial pressure (MAP) of >65 mmHg after fluid resuscitation and a serum lactate level of >2 mmol/L in patients with KP-BSI.[13] Definitive central venous catheter (CVC)-related BSI[14] is defined as the isolation of the same microorganism from at least one percutaneous blood culture and the catheter tip, or from two blood samples (one from the catheter hub and another from a peripheral vein) that meet quantitative blood culture or differential time to positivity criteria for Catheter-Related BSI. If no other major sources of infection are identified for KP-BSI, it is classified as unknown origin KP-BSI. KP-BSI originating from a liver abscess is defined as a blood infection of hepatic origin. Antibiotic exposure was operationally defined as the administration of any systemic antimicrobial agent(s) within 2 weeks preceding blood culture collection, categorized as: (i) BL/BLI: β-lactam/β-lactamase inhibitors; (ii) BL: β-lactam antibiotic; or (iii) Other: fluoroquinolones or aminoglycosides - including monotherapy, combination therapies, or sequential use of agents across these categories.

Statistical Analysis

Statistical analysis was performed with SPSS 26.0 (IBM Corp, Armonk, NY, USA) software. All graphical representations were generated using GraphPad Prism 8.0, with the exception of Figure 2, which was created using R software version 4.3.3. Continuous variables were presented as mean ± standard deviation if normally distributed, and as median and interquartile range (IQRs) if nonnormally distributed. Continuous variables were compared by Student *t* test or

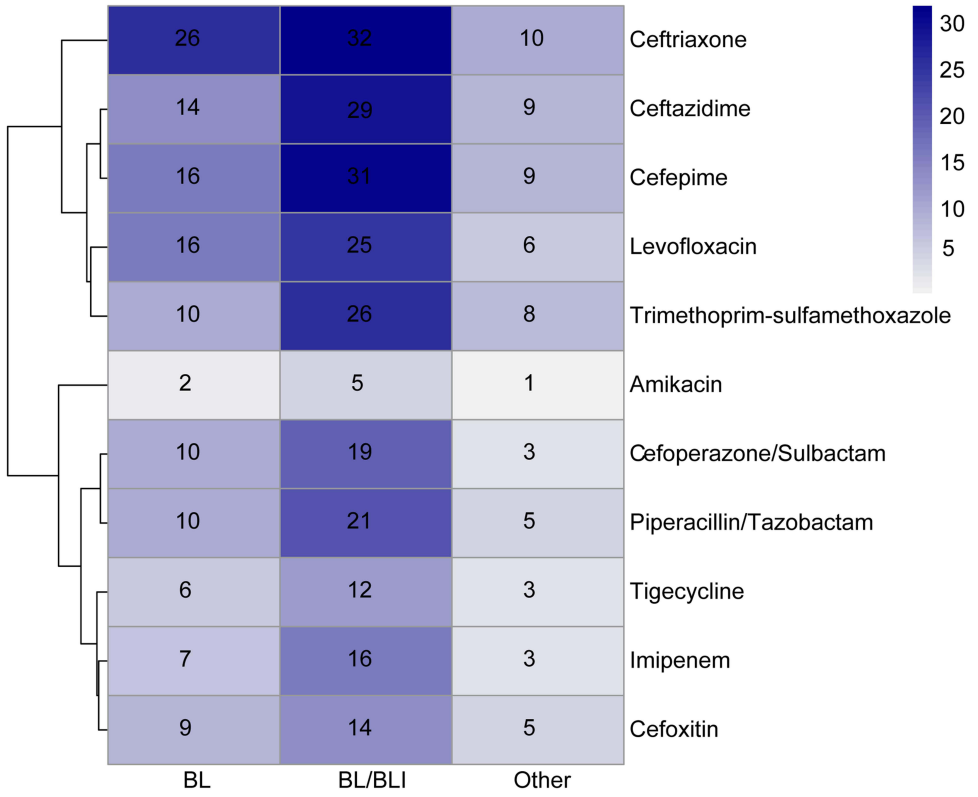

Figure 2 Prior Antibiotic Exposure and Resistance in KP-BSI Isolates. Heatmap stratifies resistance frequencies to 11 antimicrobials (y-axis) by pre-culture exposure categories: β-lactams (BL), β-lactam/β-lactamase inhibitors (BL/BLI), or other antibiotics (fluoroquinolones/aminoglycosides). Color intensity correlates with resistance frequency.

Abbreviations: KP-BSI, *Klebsiella pneumoniae* bloodstream infections; BL/BLI, β-lactam/β-lactamase inhibitors; BL, β-lactam antibiotics.

Mann–Whitney *U*-test and enumeration variables were compared by Pearson χ^2 or Fisher exact test, where appropriate. Variables in the univariate analysis with $P<0.05$ were considered candidates for building multivariate stepwise logistic regression models. Two-tailed $P<0.05$ was considered statistically significant.

Results

Demographics and Clinical Characteristics

In this study, the median age of patients was 70 years (IQR, 58.3–81.0 years), and 56.3% (135/240) of the patients were male. Of the 240 cases, 54 (22.5%) were MDR KP-BSI and 186 (77.5%) were non-MDR KP-BSI. The most common comorbidity was hypertension (42.5%), followed by diabetes (32.1%). The MDR KP-BSI group had higher rates of hemiplegia (22.2% vs 9.1%, $P<0.05$), chronic obstructive pulmonary disease (COPD) or severe asthma (13.0% vs 4.3%, $P<0.05$), chronic cardiac insufficiency (22.2% vs 5.9%, $P<0.05$), and cerebrovascular accident (40.7% vs 14.7%, $P<0.05$) compared to the non-MDR KP-BSI group. Compared with the non-MDR KP-BSI group, the MDR KP-BSI group had significantly higher severity-of-illness scores, as indicated by the APACHE II score [19.0 (11.0, 15.0) vs 11.0 (8.0, 17.0), $P<0.05$], SOFA score [8.0 (3.0, 12.0) vs 3.0 (1.0, 6.0), $P<0.05$], Pitt bacteremia score [3.5 (1.0, 6.3) vs 1.0 (0.0, 2.0), $P<0.05$], and CCI [4.0 (2.0, 5.0) vs 3.0 (2.0, 4.0), $P<0.05$]. Patients with MDR KP-BSI group had significantly higher overall antibiotic exposure within the past two weeks (66.7% vs 22.7%, $P<0.05$), but showed no significant differences in the distribution of specific antibiotic classes (BL, BL/BLI, or other) between groups. Compared to the non-MDR KP-BSI group, the MDR KP-BSI group had more patients admitted to the intensive care unit (ICU) (53.7% vs 24.7%, $P<0.05$), and more patients received parenteral nutrition (33.6% vs 8.6%, $P<0.05$) and enteral nutrition (61.1% vs 24.7%, $P<0.05$). The proportion of patients with central venous catheters was higher in the MDR KP-BSI group than in the non-MDR KP-

BSI group (51.9% vs 20.4%, $P<0.05$). Similarly, the MDR KP-BSI group had a higher proportion of patients with indwelling urinary catheters (59.3% vs 25.8%, $P<0.05$). The MDR KP-BSI group required more life-support measures, such as mechanical ventilation (35.2% vs 11.3%, $P<0.05$) and continuous renal replacement therapy (18.5% vs 4.8%, $P<0.05$), compared to the non-MDR KP-BSI group. Although these differences were observed, there were no significant differences between the two groups in the placement of abdominal drainage tubes or the proportion of surgical procedures (Table 1).

Biological Indicators

The study found that patients with MDR KP-BSI had significantly lower hemoglobin (Hb) levels [89.50 (77.00, 113.00) vs 116.50 (95.75, 133.00), $P<0.05$] and hematocrit (HCT) levels (30.21±11.56 vs 34.17±7.45, $P<0.05$) compared to

Table 1 Baseline Characteristics of Patients with MDR and Non-MDR KP-BSI

Characteristics	Total (n=240)	MDR KP-BSI (n=54)	Non-MDR KP-BSI (n=186)	*P* vale
Age, median years, (IQR)	70.0(58.3, 81.0)	75.5(62.3, 84.0)	69.0(56.8, 79.3)	**0.029**
Sex, n (%)				
Male	135(56.3%)	36(66.7%)	99(53.2%)	0.080
Comorbidities, n (%)				
Diabetes mellitus	77(32.1%)	17(31.5%)	60(32.3%)	0.914
Severe pneumonia	5(2.1%)	3(5.6%)	2(1.1%)	0.137
Myocardial infarction	9(3.8%)	4(7.4%)	5(2.7%)	0.230
Hemiplegia	29(12.1%)	12(22.2%)	17(9.1%)	**0.009**
Hematological disorders	6(2.5%)	3(5.6%)	3(1.6%)	0.255
Chronic kidney disease	16(6.7%)	6(11.1%)	10(5.4%)	0.239
Chronic liver disease	18(7.5%)	5(9.3%)	13(7.0%)	0.577
COPD or severe asthma	15(6.3%)	7(13.0%)	8(4.3%)	**0.046**
Chronic cardiac insufficiency	23(9.6%)	12(22.2%)	11(5.9%)	**<0.001**
Hypertension	102(42.5%)	30(55.6%)	72(38.7%)	**0.027**
Solid tumor	28(11.7%)	6(11.1%)	22(11.8%)	0.885
Gastrointestinal hemorrhage	8(3.3%)	4(7.4%)	4(2.2%)	0.143
Cerebrovascular accident	48(20.0%)	22(40.7%)	26(14.0%)	**<0.001**
CCI, (IQR)	3.0(2.0, 4.0)	4.0(2.0, 5.0)	3.0(2.0, 4.0)	**0.007**
APACHE II score, (IQR)	12.0(8.0, 20.0)	19.0(11.0, 25.0)	11.0(8.0, 17.0)	**<0.001**
SOFA score, (IQR)	4.0(2.0, 7.8)	8.0(3.0, 12.0)	3.0(1.0, 6.0)	**<0.001**
Pitt bacteremia score, (IQR)	1.0(0.0, 4.0)	3.5(1.0, 6.3)	1.0(0.0, 2.0)	**<0.001**
Septic shock, n (%)	81(33.8%)	32(59.3%)	49(26.3%)	**<0.001**
Hospitalization ward, n (%)				
ICU stay	75(31.3%)	29(53.7%)	46(24.7%)	**<0.001**

(*Continued*)

https://doi.org/10.2147/IDR.S535018

Table 1 (Continued).

Characteristics	Total (n=240)	MDR KP-BSI (n=54)	Non-MDR KP-BSI (n=186)	*P* vale
Previous treatment, n (%)				
Parenteral nutrition	34(14.2%)	18(33.3%)	16(8.6%)	**<0.001**
Enteral nutrition	79(32.9%)	33(61.1%)	46(24.7%)	**<0.001**
Mechanical ventilation	40(16.7%)	19(35.2%)	21(11.3%)	**<0.001**
Antibiotic exposure	78(32.5%)	36(66.7%)	42(22.6%)	**<0.001**
BL	84(35.0%)	14(25.9%)	70(37.6%)	0.112
BL/BLI	123(51.2%)	31(57.4%)	92(49.5%)	0.304
Other[a]	33(13.8%)	9(16.7%)	24(12.9%)	0.408
CRRT	19(7.9%)	10(18.5%)	9(4.8%)	**0.001**
Clinical operation, n (%)				
Indwelling central venous catheter	66(27.5%)	28(51.9%)	38(20.4%)	**<0.001**
Indwelling urinary catheter	80(33.3%)	32(59.3%)	48(25.8%)	**<0.001**
Indwelling abdominal drainage catheter	19(7.9%)	6(11.1%)	13(7.0%)	0.323
Surgery	64(26.7%)	16(29.6%)	48(25.8%)	0.576

Notes: Bold indicates $P<0.05$; [a]Fluoroquinolones, Aminoglycosides.

Abbreviations: KP-BSI, *Klebsiella pneumoniae* bloodstream infections; MDR, multidrug-resistant; COPD, chronic obstructive pulmonary disease; CCI, Charlson Comorbidity Index; SOFA, sequential organ failure assessment; APACHE, acute physiology and chronic health; IQR, interquartile range; ICU, intensive care unit; CRRT, continuous renal replacement therapy; BL/BLI, β-lactam/β-lactamase inhibitors; BL, β-lactam antibiotics; KP-BSI, *Klebsiella pneumoniae* bloodstream infections; MDR, multidrug-resistant.

patients with non-MDR KP-BSI. No statistically significant differences were observed between MDR and non-MDR KP-BSI groups in liver and kidney function markers (ALT, AST, ALP, TBi, albumin, SCr), blood routine tests (white blood cell count, platelet count), or inflammatory biomarkers (CRP, PCT) (Figure 3).

Sources of KP-BSI

The primary source of KP-BSI was urinary tract infection (22.1%, 53/240), followed by pulmonary infection (19.2%, 46/240). For MDR KP-BSI cases, the primary source was pulmonary infection (42.6%, 23/54), followed by urinary tract infection (25.9%, 14/54). Compared to the non-MDR KP-BSI group, the MDR KP-BSI group had a significantly higher proportion of infections originating from the lungs (42.6% vs 12.4%, $P<0.05$) and a significantly lower proportion from the liver (1.9% vs 24.2%, $P<0.05$). No significant differences were found between the two groups in other infection sources, such as those originating from central venous catheters, the biliary tract, or the abdominal cavity (Figure 4).

Antibiotic Resistance and Appropriate Therapy

In this study, among patients with KP BSI, the resistance rate to Ceftriaxone was the highest at 31.8% (68/240), followed by Cefepime at 23.8% (56/235). In the MDR KP-BSI group, the resistance rates of KP strains were as follows: Ceftriaxone (92.9%), Levofloxacin (82.7%), Cefepime (81.5%), Ceftazidime (75.9%), Piperacillin/Tazobactam (67.9%), Cefoperazone/Sulbactam (61.5%), Cefoxitin (55.0%), Trimethoprim/Sulfamethoxazole (50.9%), Imipenem (48.1%), Tigecycline (33.3%), and Amikacin (15.7%). These rates showed significant differences compared to the non-MDR KP-BSI group. The MDR KP-BSI group exhibited a significantly higher extended-spectrum beta-lactamases (ESBL) positivity rate (51.3% vs 16.9%, $P<0.05$) compared to the non-MDR KP-BSI group (Table 2).

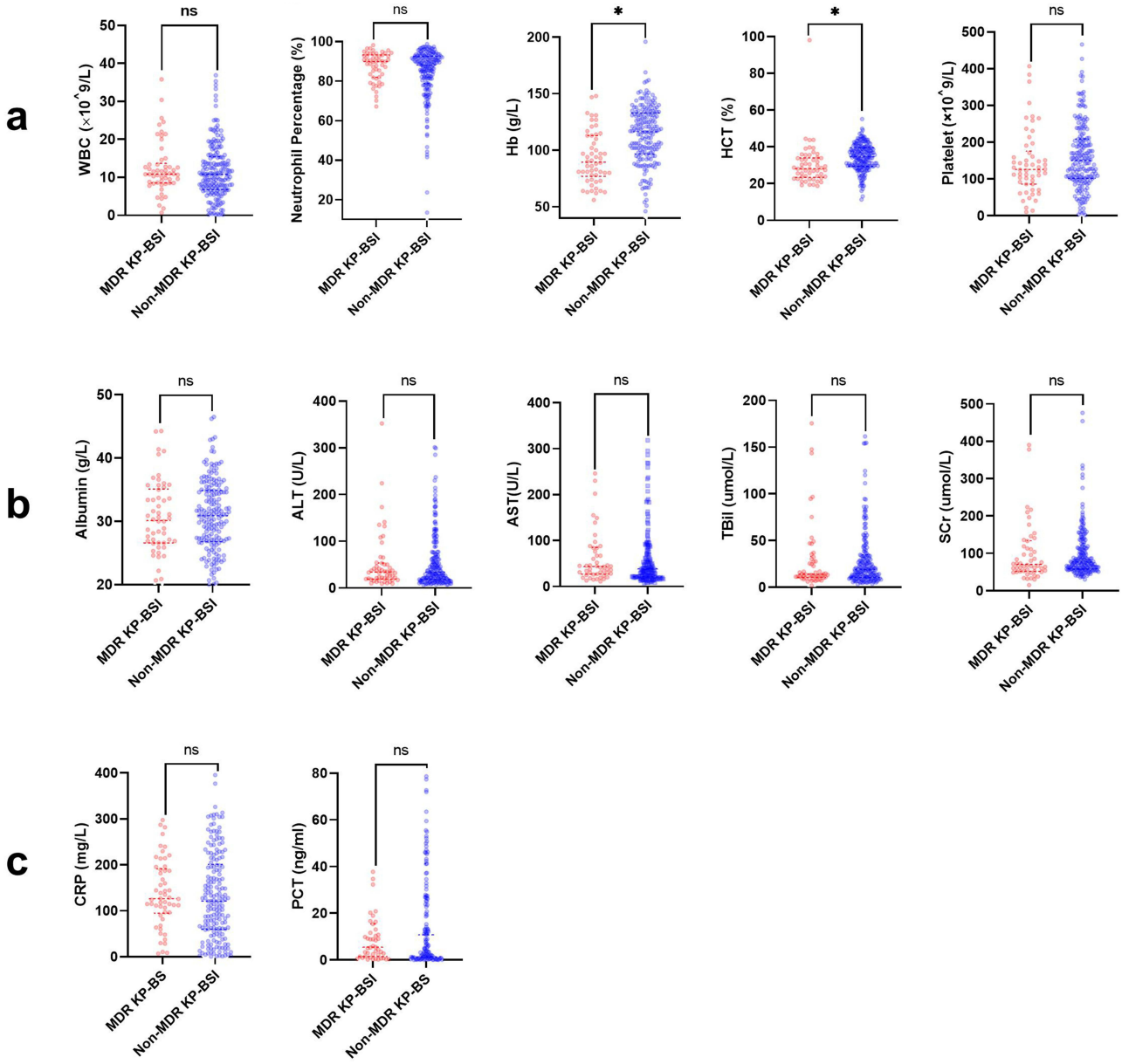

Figure 3 Comparative scatter plots of clinical indicators between MDR KP-BSI and non-MDR KP-BSI cohorts. (**a**) Blood routine tests: WBC, Hb ($P<0.001$), HCT ($P=0.003$), Platelet; (**b**) Liver and kidney function markers: ALT, AST, ALP, TBil, Albumin, SCr; (**c**) Inflammatory biomarkers: CRP, PCT. X-axis: Patient cohorts; Y-axis: Individual patient measurements. Red circles indicate the MDR cohort; blue circles indicate the non-MDR cohort. *Statistical significance: $P<0.05$; ns: not significant.

Abbreviations: ALP, alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; CRP, C-reactive protein; Hb, hemoglobin; HCT, hematocrit; KP-BSI, *Klebsiella pneumoniae* bloodstream infections; MDR, multidrug-resistant; PCT, procalcitonin; SCr, serum creatinine; TBil, total bilirubin; WBC, white blood count.

Given these notable differences in resistance profiles, we further explored potential contributing factors, particularly the role of prior antibiotic exposure. To this end, a heatmap (Figure 2) was generated to illustrate the relationship between different classes of previously administered antibiotics and the resistance frequencies of KP isolates to 11 commonly used antimicrobial agents. The results revealed that isolates from patients with prior exposure to β-lactam/β-lactamase inhibitor combinations (BL/BLI) exhibited significantly higher resistance rates to multiple antibiotics, especially ceftriaxone (n=32) and cefepime (n=31). Similarly, prior use of β-lactam monotherapy (BL) was associated with increased resistance to ceftriaxone (n=26) and levofloxacin (n=16). In contrast, isolates from patients who had received antibiotics categorized as "Other" showed relatively lower resistance frequencies.

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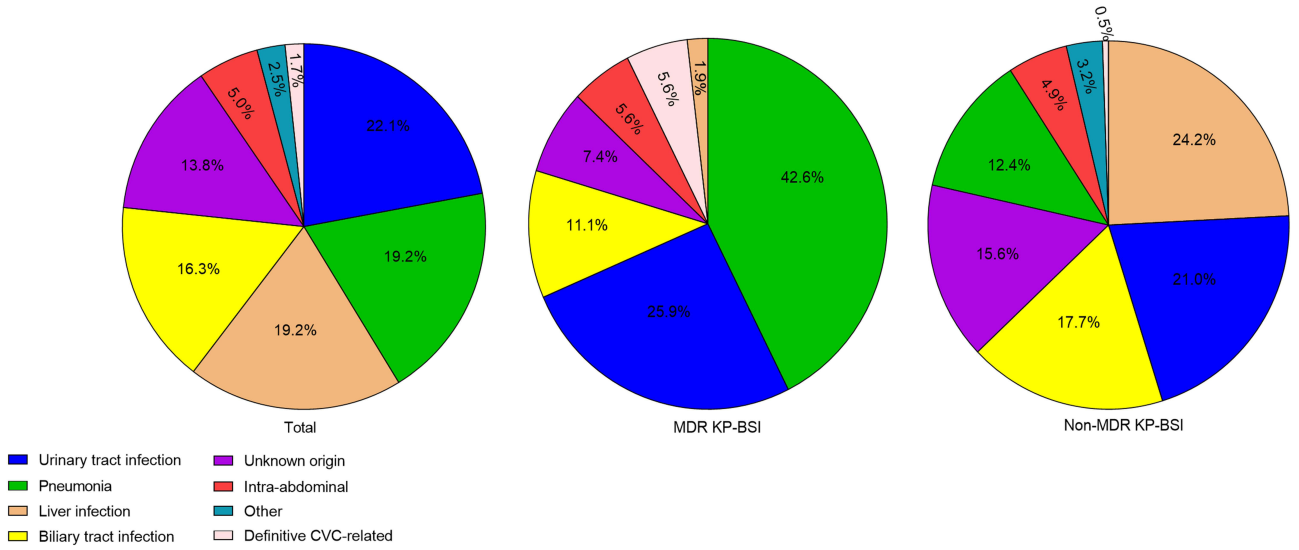

Figure 4 Comparative distribution of infection sources in MDR versus non-MDR KP-BSI cohorts. Pie charts depict source proportions for: Total cohort (n=240), MDR KP-BSI (n=54), and non-MDR KP-BSI (n=186). Pneumonia predominates in MDR KP-BSI, while liver infections are more frequent in non-MDR KP-BSI. Other sources (skin/soft tissue, intracranial, bone/joint infections) occur exclusively in non-MDR cases.
Abbreviations: KB-BSI, *Klebsiella pneumoniae* bloodstream infections; CVC, central venous catheter; MDR, multidrug-resistant.

Clinical Outcomes of MDR KP-BSI

Patients with MDR KP-BSI exhibited significantly worse clinical outcomes compared to non-MDR KP-BSI patients. Mortality rates were substantially higher in the MDR KP-BSI group at all time points: 7-day mortality (40.7% vs 11.8%, $P<0.001$), 14-day mortality (35.2% vs 10.8%, $P<0.001$), and 28-day mortality (27.8% vs 8.6%, $P<0.001$). Hospital length of stay was markedly prolonged [26.5 (14.0, 64.5) vs 13.0 (8.0, 23.0), $P<0.001$] (Table 3).

Table 2 Comparison of the Microbiological Characteristics Between the Groups of MDR and Non-MDR KP-BSI

Antibiotic Resistance[a]	Total (n=240)	MDR KP-BSI (n=54)	Non-MDR KP-BSI (n=186)	*P* vale
Cefoperazone/Sulbactam (52 vs 177)[b]	32(14.0%)	32(61.5%)	0(0.0%)	**<0.001**
Ceftazidime (41 vs 11)[b]	52(22.2%)	41(75.9%)	11(6.1%)	**<0.001**
Imipenem (54 vs 183)[b]	26(11.0%)	26(48.1%)	0(0.0%)	**<0.001**
Ceftriaxone (42 vs 172)[b]	68(31.8%)	39(92.9%)	29(16.9%)	**<0.001**
Cefepime (54 vs 181)[b]	56(23.8%)	44(81.5%)	12(6.6%)	**<0.001**
Tigecycline (51 vs 184)[b]	21(8.9%)	17(33.3%)	4(2.2%)	**<0.001**
Amikacin (51 vs 181)[b]	8(3.4%)	8(15.7%)	0(0.0%)	**<0.001**
Piperacillin/Tazobactam (53 vs 182)[b]	36(15.3%)	36(67.9%)	0(0.0%)	**<0.001**
Levofloxacin (52 vs 179)[b]	47(20.3%)	43(82.7%)	4(2.2%)	**<0.001**
Cefoxitin (40 vs 172)[b]	28(13.2%)	22(55.0%)	6(3.5%)	**<0.001**
Trimethoprim-sulfamethoxazole (53 vs 174)[b]	44(19.4%)	27(50.9%)	17(9.9%)	**<0.001**
ESBL (39 vs 160)[b]	47(23.6%)	20(51.3%)	27(16.9%)	**<0.001**

Notes: Bold indicates $P<0.05$; [a]Not all agents listed tested in all isolates; [b]The numbers in parentheses represent the total numbers of *Klebsiella pneumoniae* isolates that performed the susceptibility test.
Abbreviations: KP-BSI, *Klebsiella pneumoniae* bloodstream infections; MDR, multidrug resistance; ESBL, Extended-spectrum beta-lactamases.

Table 3 Comparison of Clinical Outcomes Between Patients with MDR-KP and Non-MDR KP-BSI

Outcome	Total (n=240)	MDR KP-BSI (n=54)	Non-MDR KP-BSI (n=186)	*P* vale
7-day mortality, n (%)	44(18.3%)	22(40.7%)	22(11.8%)	**<0.001**
14-day mortality, n (%)	39(16.3%)	19(35.2%)	20(10.8%)	**<0.001**
28-day mortality, n (%)	31(12.9%)	15(27.8%)	16(8.6%)	**<0.001**
Hospital length of stay (days), median (IQR)	15.0(9.0, 28.0)	26.5(14.0, 64.5)	13.0(8.0, 23.0)	**<0.001**

Note: Bold indicates *P*<0.05.
Abbreviations: KP-BSI, *Klebsiella pneumoniae* bloodstream infections; MDR, multidrug resistance.

Independent Risk Factors for MDR KP-BSI

Variables with a P-value <0.05 in the univariate analysis of this study were entered into a multivariable logistic regression model to identify factors associated with MDR KP-BSI. As shown in Figure 5, independent risk factors for MDR KP-BSI were a history of cerebrovascular accident (adjusted odds ratio [OR], 3.095; 95% confidence interval [CI], 1.054–9.903), antibiotic exposure within 2 weeks (adjusted odds ratio [OR], 7.025; 95% confidence interval [CI], 2.695–18.313), and pulmonary infection source (adjusted odds ratio [OR], 2.941; 95% confidence interval [CI], 1.101–7.895).

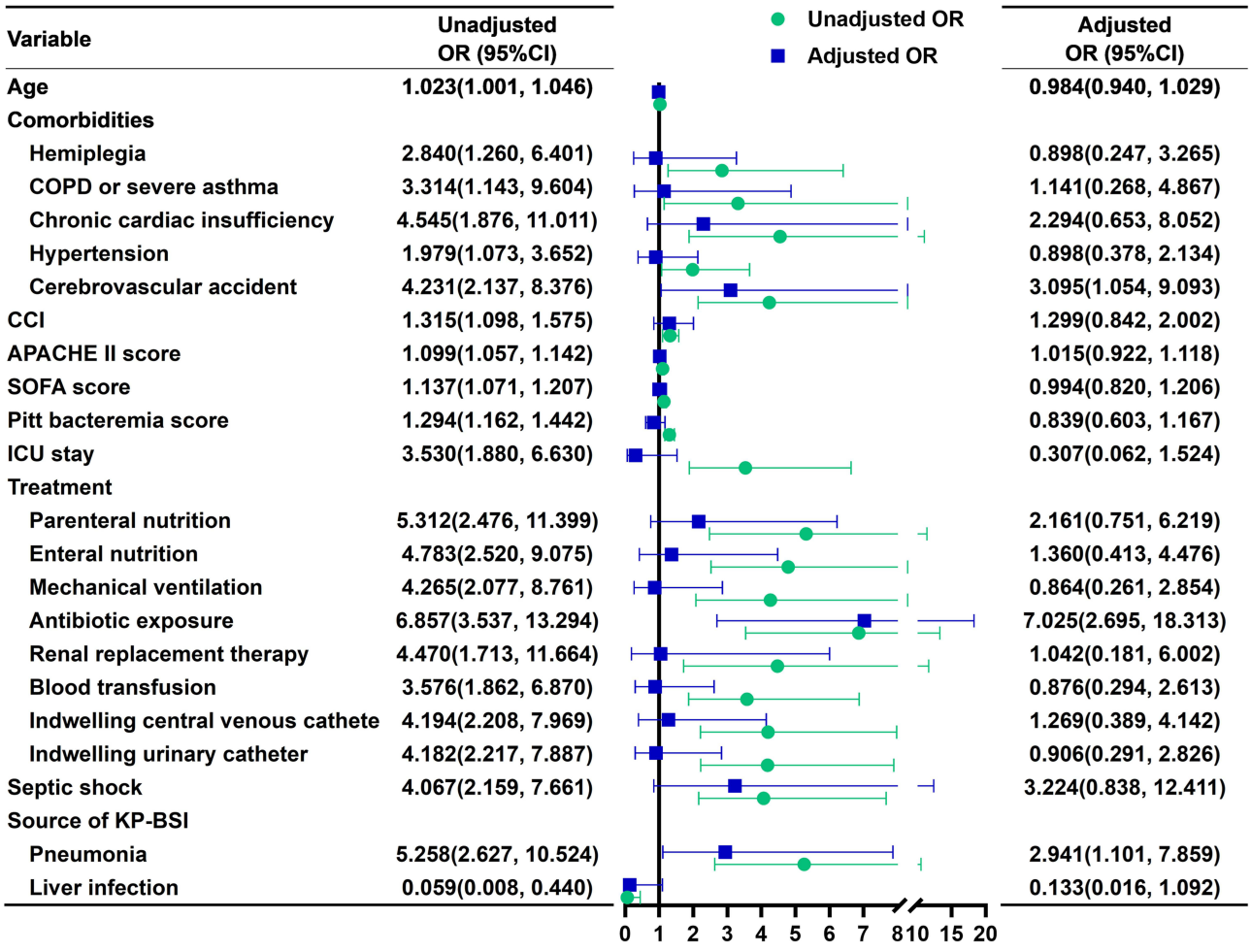

Figure 5 Multivariable logistic regression identifying independent risk factors for MDR-KP BSI. Forest plot displays significant predictors with adjusted odds ratios (95% CIs): antibiotic exposure within 2 weeks, cerebrovascular accident history, and pulmonary infection source.
Abbreviations: CI, confidence interval; KP-BSI, *Klebsiella pneumoniae* bloodstream infections; MDR, multidrug-resistant; ICU, intensive care unit; CCI, Charlson Comorbidity Index; COPD, chronic obstructive pulmonary disease.

https://doi.org/10.2147/IDR.S535018

Discussion

Our key findings are as follows: (1) MDR KP-BSI accounted for 22.5% (54/240) of all KP-BSI cases. KP showed high antibiotic resistance rates, particularly to Ceftriaxone. In MDR-KP strains, the highest resistance rates occurred against Ceftriaxone, Levofloxacin, and Cefepime. (2) Compared to non-MDR KP-BSI cases, patients with MDR KP-BSI demonstrated significantly poorer clinical outcomes, including elevated mortality at the 7-, 14-, and 28-day measurement timepoints and prolonged hospital length of stay. (3) The primary source of MDR-KP strains was the lungs, in contrast to non-MDR KP strains, which mainly originated from the liver. (4) Cerebrovascular accident, recent antibiotic exposure, and a pulmonary infection source were identified as independent risk factors for MDR KP-BSI.

Published studies indicate an increasing prevalence of multidrug-resistant bacteria in the general population.[15] Specifically, reported MDR-KP prevalence ranges from 0% to 33.9%, with a mean of 8.2%.[16] In our cohort, MDR-KP accounted for 22.5% of KP-BSI, within the range reported in the literature. This finding underscores the persistent presence of MDR-KP in BSI. This trend warrants attention, as the high proportion of MDR-KP in clinical settings poses a serious public health threat. Consequently, strengthening surveillance, prevention, and control measures is urgently needed. This is crucial for mitigating its impact on healthcare systems and protecting patients from infection risks.

This study, in line with previous research,[17] found that patients with MDR KP-BSI exhibited worse clinical outcomes compared to those with non-MDR KP-BSI. The extended length of hospitalization was closely associated with infections caused by drug-resistant bacteria, which complicates the clinical condition and increases the difficulty of treatment. The elevated mortality rate was primarily attributed to the following factors: First, MDR-KP is resistant to multiple commonly used antibiotics, which significantly restricts clinical treatment options, reduces the effectiveness of empirical therapy, and increases the risk of treatment failure.[11] Second, this study found that patients with MDR KP-BSI had more severe comorbidities, as evidenced by higher APACHE II and SOFA scores, indicating that patients were in a more critical condition at the onset of bloodstream infection. Additionally, the study observed a higher incidence of septic shock among patients with MDR KP-BSI. Septic shock is a life-threatening condition that further exacerbates the severity of illness and leads to higher mortality rates. In summary, the worse clinical outcomes in patients with MDR KP-BSI are mainly due to the limited treatment options caused by drug resistance, the severity of underlying diseases, and the high incidence of septic shock. These factors collectively contribute to the significant increase in mortality and hospitalization duration among patients with MDR KP-BSI. These findings highlight the urgent need for clinicians to recognize the impact of MDR KP-BSIs and implement targeted interventions to address these challenges.

Our analysis revealed high resistance levels in MDR-KP to widely used antibiotics, including penicillins and cephalosporins, consistent with previous studies.[18] Notably, 51.3% of MDR-KP strains in this cohort produced ESBLs, a primary mechanism of drug resistance in Enterobacteriaceae.[19] The high prevalence of ESBL-producing strains among MDR-KP isolates contributes to their high resistance to β-lactam antibiotics. Furthermore, the resistance mechanisms of MDR-KP may involve multiple factors, including the presence of blaKPC-2 and blaNDM-5 genes,[20] which are key contributors to carbapenem resistance in MDR-KP. In addition to the acquisition of resistance genes, drug resistance can also arise from multiple mechanisms, including alteration of drug targets, drug inactivation, reduced cell permeability, and increased activity of efflux pump.[21] The combined action of these mechanisms contributes to the broad-spectrum resistance of MDR-KP to multiple antibiotic classes, presenting significant challenges for clinical management.

Because of the limited antibiotic treatment options, MDR-KP has been recognized as a hazardous pathogen for nosocomial infections.[22] Amikacin and tigecycline were once effective antibiotics for treating MDR-KP.[23] However, our susceptibility testing revealed high resistance rates to both amikacin and tigecycline in MDR-KP isolates. Recent studies have indicated that KP has shown a significant increase in resistance to antibiotics, including amikacin and tigecycline, which may be associated with the overuse of antibiotics during treatment.[24,25] Critically, we identified prior antibiotic exposure within the 2 weeks preceding BSI as an independent risk factor for MDR-KP development. This finding is supported by a prospective observational study demonstrating that patients with prior antibiotic exposure had a higher risk of earlier-onset infections caused by MDR-KP.[26] Moreover, the selective pressure exerted by antimicrobials represents a primary factor contributing to the development of MDR. Antibiotic use targeting susceptible bacteria creates an environment in which drug-resistant bacteria are left to proliferate and dominate.[27] Antibiotic-induced depletion of short-chain fatty acid-producing bacteria impairs the bactericidal activity of inflammatory monocytes, thereby

undermining the body's antimicrobial immune defense and leading to associated infections.[28] Given this, clinicians should pay close attention to MDR-KP, strictly adhere to the indications for antibiotic use, and use antibiotics appropriately. Implementing antimicrobial stewardship programs based on current guidelines and restricting unnecessary antibiotic use can help limit the emergence of antibiotic resistance in bacteria.[29] These measures are of critical importance for controlling the spread of MDR-KP and reducing hospital-acquired infections.

Pulmonary infection was identified as an independent risk factor for MDR-KP BSI patients. The formation of MDR-KP in pulmonary infections involves several key factors: Firstly, respiratory infections are often caused by the aspiration of bacteria colonizing the upper respiratory tract.[17] MDR-KP enhances its adhesion to the respiratory epithelial surface by expressing fimbrial genes.[30] Additionally, the capsular polysaccharide on the surface of MDR-KP interferes with the host's clearance mechanisms, while the production of extracellular polysaccharides promotes biofilm formation.[31] This further enhances the persistence of bacteria in the lungs and limits contact with phagocytes, antibodies, and antibiotics.[31,32] Finally, MDR-KP is relatively non-immunogenic, enabling it to evade host immune surveillance without triggering a strong immune response. In patients with pulmonary infections, effective source control is often challenging, and the pulmonary penetration of some antimicrobial agents is limited.[33] The interplay of these factors enables MDR-KP to colonize the lungs and cause infections, thereby complicating treatment efforts. In contrast, the majority of KP strains isolated from liver abscesses were non-MDR KP strains. This observation is consistent with previous studies, which have demonstrated that KP strains isolated from liver abscess pus cultures are susceptible to most commonly used clinical antibiotics.[34,35] The reason is that KP strains in liver abscesses typically enter the bloodstream through direct dissemination and the portal circulation.[35] This mode of spread reduces their exposure to antibiotics in the environment, thereby decreasing the emergence and development of drug resistance.[36] The relatively enclosed infection environment of liver abscesses may limit contact between KP strains and other resistant strains, thereby reducing the spread of resistance genes.[37]

Previous research has found that being bedridden is an independent risk factor for MDR bacterial infections,[38] which is consistent with our finding that patients with hemiplegia are more prone to MDR KP-BSI. Specifically, cerebrovascular accident was identified as an independent risk factor for MDR KP-BSI patients, consistent with prior studies confirming the susceptibility of patients with cerebrovascular accident to MDR bacterial infections.[39] Patients with cerebrovascular accident are prone to infection-related complications, with an occurrence rate of bloodstream infections of 1.79%.[40] In patients with cerebrovascular accidents, the risk of infection is increased, which is related to the secondary brain tissue damage caused by the immune response following central nervous system injury and the subsequent activation of the sympathetic nervous system leading to immunosuppression. These factors together increase the risk of infection complications.[41] In patients with cerebrovascular accidents, brain-derived damage-associated molecular patterns enter the bloodstream, activating the systemic immune system and mobilizing immune cells from lymphoid organs and the gut. This process may increase gut permeability, releasing bacteria and their metabolites into the bloodstream and thereby increasing the risk of infection.[42] Moreover, in the pathogenesis of MDR-KP infections, the transition of bacteria from colonization to infection is a critical step.[29] Notably, the colonization rate of MDR-KP in our patient cohort was not low. According to the study,[43] which screened 1687 patients at a tertiary hospital in Italy, 65 patients (3.9%) tested positive for MDR-KP. Under these circumstances, when hosts develop bloodstream infections, the presence of host immune dysregulation and immunosuppression makes MDR bacterial colonization more likely to transition from colonization to pathogenicity.[44]

This study has several limitations. First, as a single-center study, results may be affected by local infection control policies, limiting generalizability. Second, the retrospective design may lead to information bias, such as data loss, hindering comprehensive evaluation. Third, the small sample size may limit statistical power and increase the chance of random results. Moreover, the lack of recorded prior KP colonization in patients may introduce bias. Additionally, the failure to monitor KP virulence limits understanding of infection mechanisms. Finally, inadequate treatment descriptions, without detailed discussions on treatment-regimen selection, affect a comprehensive understanding of treatment outcomes and resistance development. Thus, future studies should adopt multicenter, prospective designs, ensure complete data collection, including virulence monitoring, and enhance result robustness and extrapolation.

Conclusion

This study aimed to analyze the prevalence, risk factors, and prognosis of MDR KP-BSI. We found that the prevalence of MDR KP-BSI is not low. It was associated with higher rates of comorbidities, greater disease severity scores, and more frequent treatment - related factors like antibiotic exposure and ICU admission. The MDR KP-BSI group more commonly had pulmonary infection sources but less commonly hepatic ones. MDR KP-BSI patients had worse prognoses, with higher mortality rates and longer hospital stays. Multivariable analysis revealed that recent antibiotic exposure, a history of cerebrovascular accident, and pulmonary infection source were independent risk factors for MDR KP-BSI. The findings highlight the need for better clinical prevention and control of MDR KP-BSI by managing these risk factors.

Abbreviations

BSI, bloodstream infections; KP, *Klebsiella pneumoniae*; MDR, multidrug-resistant; CCI, Charlson comorbidity index; SOFA, sequential organ failure assessment; APACHE, acute physiology and chronic health evaluation; MAP, mean arterial pressure; Hb, hemoglobin; HCT, hematocrit; CRP, c-reactive protein; PCT, procalcitonin; CVC, central venous catheter; WBC, white blood count; ESBL, extended-spectrum beta-lactamases; OR, adjusted odds ratio; CI, confidence interval; MDR, multidrug-resistant; COPD, chronic obstructive pulmonary disease; IQR, interquartile range; ICU, intensive care unit; CRRT, continuous renal replacement therapy; ALT, alanine aminotransferase; AST, aspartate aminotransferase; TBil, total bilirubin; SCr, serum creatinine; CLSI, Clinical and Laboratory Standards Institute.

Ethics Approval

Our study received human research ethics approval from the Medical Ethics Committee of Taizhou Municipal Hospital (Taizhou University Affiliated Municipal Hospital), School of Medicine, Taizhou University (Approval No. LWYJ2023021). We make sure to keep patient data confidential and compliance with the Declaration of Helsinki.

Informed Consent

Due to the retrospective nature of the study, the Ethics Committee determined that no patient consent was required.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Funding

This work was supported in part by grants from the Medical Health Science and Technology Project of Zhejiang Provincial Health Commission [No. 2025KY1871, Cheng Zheng; No. 2024KY1811, Panpan Xu; No. 2024KY1824, Qingqing Chen]; The Science and Technology Project of Taizhou [No. 23ywb70, Qingqing Chen; No. 24ywa44, Cheng Zheng].

Disclosure

The authors declare that they have no competing interests in this work.

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