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Emerging Problems in Infectious Diseases

Clinical features and risk factors for mortality in patients with *Klebsiella pneumoniae* bloodstream infections

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Abstract

Introduction: Concern about *Klebsiella pneumoniae* (*K. pneumoniae*) bloodstream infections (KP-BSIs) is widespread because of their high incidence and lethality. The aim of this study was to investigate the clinical features of, and risk factors for mortality caused by KP-BSIs.
Methodology: This was a single-center retrospective observational study performed between 1 January 2019 and 31 December 2021, at a tertiary hospital. All patients with KP-BSIs were enrolled and their clinical data were retrieved from electronic medical records.
Results: A total of 145 patients were included (121 in the survival group and 24 in the non-survival group). There was a higher proportion of lower respiratory tract infections in the non-survival group than in the survival group (33.3% vs. 12.4%) ($p < 0.05$). There was a higher proportion of multi drug resistant (MDR) strains of *K. pneumoniae* in the non-survival group than in the survival group (41.7% vs. 16.5%) ($p < 0.05$). Multivariate analysis revealed that sequential organ failure assessment (SOFA) score > 6.5 (OR, 13.71; 95% CI, 1.05–179.84), admission to the intensive care unit (ICU) (OR, 2.27; 95% CI, 0.26–19.61) and gastrointestinal bleeding (OR, 19.97; 95% CI, 1.11–361.02) were independent risk factors for death in patients with KP-BSIs.
Conclusions: Among all KP-BSIs, a high proportion of *K. pneumoniae* originated from lower respiratory tract infections, and a high proportion of *K. pneumoniae* were MDR; however, mortality was not influenced. SOFA score > 6.5, admission to the ICU, and gastrointestinal bleeding were independent risk factors for death in patients with KP-BSI.

Key words: *Klebsiella pneumoniae*; mortality; bloodstream infections; clinical features; risk factor.

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Introduction

Bloodstream infections (BSIs) are one of the leading causes of death in hospitalized patients [1] and are a worldwide concern [2]. Among the bacteria that cause bloodstream infections, *Klebsiella pneumoniae* (KP) ranks second after *Escherichia coli* [3], and notably, the incidence of BSIs is increasing year by year [4]. KP-BSIs have a high mortality rate, reportedly ranging from 20% to 40% [5,6]. Moreover, according to data from the China Antimicrobial Surveillance Network (CHINET, www.chinets.com) and a Chinese retrospective study, the drug resistance of KP is also increasing year by year [7,8]. The high drug resistance of KP not only threatens human health but also creates challenges for public health [9] and increases the mortality rate of patients [10,11]. However, there is no consensus regarding the risk factors for mortality in patients with KP-BSIs. Zhang *et al.* showed that high acute physiology and chronic health evaluation (APACHE) II scores, high sequential organ failure assessment (SOFA) scores, and invasive surgery were risk factors for mortality in patients with KP-BSI, but the clinical laboratory and score data were collected before the diagnosis [12]. A previous study [13] reported that positive blood culture results usually require up to 2–4 days; therefore, there is a certain deviation and time lag for the results. Girometti *et al.* found that among the factors tested, APACHE II scores were most accurate in predicting patient survival prognosis [14]. Meatherall *et al.* reported that chronic liver disease and cancer were significant risk factors for KP-BSI, but these studies were not recent [15]. Therefore, it is necessary to reanalyze the clinical features of and risk factors for patients with KP-BSI to establish criteria for the early identification of KP-BSI,

which would have a positive impact on the prognosis and treatment of patients.

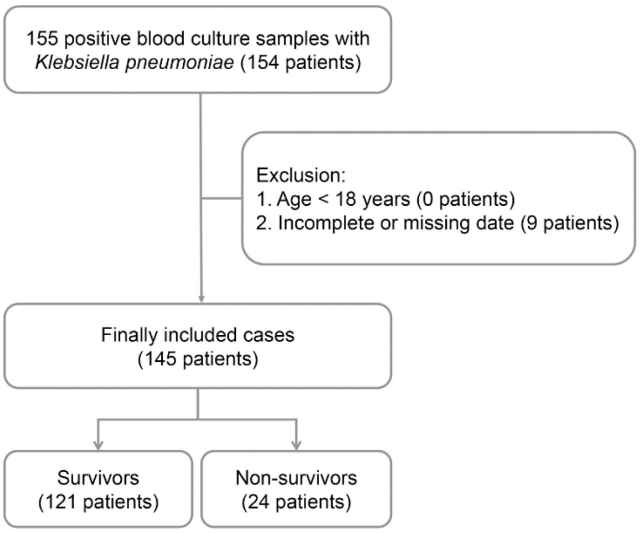
To this end, we conducted a retrospective analysis of cases in our center from 2019-2021 to reanalyze the clinical characteristics of KP-BSI and identify risk factors for mortality, with the aim of providing a reference for early medical intervention by clinicians, thereby reducing the morbidity and mortality of KP-BSI and improving clinicians' ability to make a differential clinical diagnosis.

Methodology

This was a single-center retrospective study in which the data of all cases of KP-BSI from 1 January 2019 to 31 December 2021 at Taizhou Municipal Hospital were collected. Taizhou Municipal Hospital is an affiliated, 1200-bed, comprehensive tertiary teaching hospital that serves a broad population in the local region of Taizhou—a city with a subtropical climate and a population of 6 million—China. This study was approved (No. LWYJ2023021) by the Ethics Committee of Taizhou Municipal Hospital, which determined that patient consent was not needed because this was a retrospective study.

The inclusion criteria were as follows: 1) KP-BSI, defined as the isolation of KP in at least one blood culture bottle, in accordance with the criteria of the US Centers for Disease Control and Prevention (CDC) [16]; 2) when there were multiple blood culture results, the first positive blood culture of KP-BSI was selected; and 3) the clinical indexes and blood chemistry indexes needed for calculating the APACHE II score and SOFA score were obtained on the day of blood culture extraction. The exclusion criteria were as follows: 1) age less than 18 years; and 2) incomplete data or information.

Figure 1. Flowchart of participant enrollment.



The patients with KP-BSI were divided into survival and non-survival groups based on whether they survived for 28 days or died, respectively (Figure 1).

Evaluation metrics

Patient data were extracted from electronic medical records. The baseline characteristics included age and gender, and the clinical data included underlying diseases, SOFA score, Pitt bacteremia score, Charlson comorbidity index (CCI) score, APACHE II score 24 h after BSI onset, hospitalization ward, previous exposure, and nosocomial infection. Data on possible sources of BSI and sensitivity to antibiotics were also collected. These data were collected by the same clinician to ensure data consistency and reliability. Septic shock was defined in accordance with septic shock guidelines [17]. Multidrug resistance was defined as resistance to three or more classes of antimicrobial drugs [18]. Antibiotic classes included carbapenems, β-lactamase inhibitors, cephalosporins, fluoroquinolones, and aminoglycosides. Long-term corticoid treatment was defined as prescription of a continuous dose of glucocorticoids for ≥ 30 days [19].

Statistical analysis

Continuous variables were compared using Student's t test or the Mann–Whitney U test, and count variables were compared using Pearson's χ^2 test. 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. All data were statistically analyzed using SPSS 26.0 software (IBM Corp, Armonk, NY, USA).

Results

Demographic and clinical characteristics

In this study, the mean age of the 145 patients with KP-BSIs was 68.24 years, and 53.8% (78/145) of the patients were male. The most frequent comorbidities were hypertension (37.9%, 55/145) and diabetes (28.3%, 41/145). There were no statistically significant differences in age and gender between the two groups. However, chronic cardiac insufficiency (29.2% vs. 5.8%, $p < 0.05$), gastrointestinal bleeding (16.7% vs. 2.5%, $p < 0.05$), and long-term corticoid treatment (16.7% vs. 0.8%, $p < 0.05$) were more likely in the non-survival group than in the survival group. The APACHE II scores (25.5 vs. 11.0, $p < 0.05$) and SOFA scores (13.5 vs. 3.0, $p < 0.05$) were higher in the non-

Table 1. Baseline characteristics of patients with KP-BSI.

Characteristics	Total (n = 145)	Survival group (n = 121)	Non-survial group (n = 24)	*p* value
Age	68.2 ± 15.0	65.7 ± 17.5	68.7 ± 14.6	0.376
Male	78 (53.8%)	63 (52.1%)	15 (62.5%)	0.349
Comorbidities				
Trauma	5 (3.4%)	5 (4.1%)	0 (0.0%))	0.688
Hypertension	55 (37.9%)	48 (39.7%)	7 (29.2%)	0.333
Diabetes	41 (28.3%)	34 (28.1%)	7 (29.2%)	0.916
Solid tumor	20 (13.8%)	16 (13.2%)	4 (16.7%)	0.902
Cerebrovascular accident	27 (18.6%))	21 (17.4%)	6 (25.0%)	0.379
Chronic kidney disease	7 (4.8%)	5 (4.1%)	2 (8.3%)	0.722
Chronic cardiac insufficiency	14 (9.7%)	7 (5.8%)	7 (29.2%)	0.002*
Gastrointestinal bleeding	7 (4.8%)	3 (2.5%)	4 (16.7%)	0.015*
Chronic obstructive pulmonary disease or Severe Asthma	9 (6.2%)	7 (5.8%)	2 (8.3%)	0.992
Myocardial infarction	5 (3.4%)	3 (2.5%)	2 (8.3%)	0.410
Long-term corticoid treatment	5 (3.4%)	1 (0.8%)	4 (16.7%)	0.001*
APACHE II score	11.0 (8.0,19.0)	11.0 (8.0,16.0)	25.5 (17.5,30.5)	< 0.001*
SOFA score	3.0 (1.0,7.0)	3.0 (1.0,5.0)	13.5 (8.25,15.0)	< 0.001*
Charlson Comorbidity Index	2.0 (1.0,3.5)	2.0 (2.0,3.5)	2.5 (1.0,3.8)	0.660
Hospitalization ward				
ICU	46 (31.7%)	26 (21.5%)	20 (83.3%)	< 0.001*
Clinical operation before blood sampling within 48 hours				
Surgery	42 (29.0%)	23 (19.0%)	3 (12.5%)	0.089
Indwelling central venous catheter	41 (28.3%)	23 (19.0%)	18 (75.0%)	< 0.001*
Parenteral nutrition	8 (5.5%)	6 (5.0%)	2 (8.3%)	0.863
Mechanical ventilation	7 (4.9%)	2 (1.7%)	5 (20.8%)	0.001*
Blood transfusion	30 (20.7%)	18 (14.9%)	12 (50.0%)	< 0.001*
Bronchoscopy therapy	4 (2.8%)	4 (3.3%)	0 (0.0%)	0.825
Indwelling urinary catheter	60 (41.4%)	40 (33.1%)	20 (83.3%)	< 0.001*
Mixed-type BSI	14 (9.7%)	11 (9.1%)	3 (12.5%)	0.890
Septic shock	52 (35.9%)	31 (25.6%)	21 (87.5%)	< 0.001*

APACHE: acute physiology and chronic health evaluation; BSI: bloodstream infection; ICU: intensive care unit; KP: *Klebsiella pneumoniae*; SOFA: sequential organ failure assessment; *Significant.

survival group than the survival group, indicating greater disease severity. Additionally, a higher proportion of non-surviving patients than surviving patients was admitted to the intensive care unit (ICU) (83.3% vs. 21.5%, $p < 0.05$). Before BSI onset, the percentages of patients with indwelling urinary catheters (83.3% vs. 33.1%), indwelling central venous catheters (75% vs. 19.0%), blood transfusions (50% vs. 14.9%), and septic shock (87.5% vs. 25.6%) were higher in the non-survival group than in the survival group (all $p < 0.05$) (Table 1).

Biological indicators

Compared with patients in the survival group, those in the non-survival group had lower hemoglobin levels, hematocrit levels, platelet counts and serum albumin

Table 2. Comparison of biological indicators between two groups of KP-BSI.

Biological indicators	Total (n = 145)	Survival group (n = 121)	Non-survial group (n = 24)	*p* value
Blood routine test				
White blood cells ($\times 10^9$/L) (IQR)	10.7 (6.9, 15.3)	10.8 (7.6, 15.3)	9.0 (2.8, 19.3)	0.208
Percentages of neutrophils (%) (IQR)	87.6 (78.3, 92.7)	87.8 (78.9, 92.0)	86.3 (71.2, 94.6)	0.890
Hemoglobin (g/L) (mean ± SD)	108.3 ± 24.5	110.8 ± 24.5	95.5 ± 20.2	0.005*
Hematocrit (%) (mean ± SD)	33.0 ± 6.9	33.7 ± 7.0	29.6 ± 5.7	0.008*
Platelet ($\times 10^9$/L) (IQR)	138.0 (102.0, 208.0)	152.0 (107.0, 224.5)	87.5 (42.0, 115.3)	< 0.001*
Liver and kidney function				
Albumin (g/L) (mean ± SD)	30.5 ± 6.0	31.3 ± 5.8	26.1 ± 5.1	< 0.001*
ALT (U/L) (IQR)	35.0 (18.0, 95.8)	32.0 (16.5, 73.5)	65.0 (21.0, 184.0)	0.008*
AST (U/L) (IQR)	43.0 (21.5, 95.0)	37.0 (21.0, 80.8)	109.0 (44.0, 436.0)	< 0.001*
ALP (U/L) (IQR)	105.0 (73.5, 158.0)	104.5 (73.8, 149.5)	150.0 (58.0, 313.0)	0.296
γ-GT (U/L) (IQR)	63.0 (30.5, 156.0)	61.5 (30.8, 147.3)	66.0 (29.0, 159.0)	0.662
LDH (U/L) (IQR)	244.0 (185.0, 360.3)	222.0 (177.0, 306.0)	570.0 (354.5, 782.5)	< 0.001*
Tbil (μmol/L) (IQR)	17.9 (10.1, 33.4)	16.85 (10.0, 30.1)	31.9 (12.4, 63.5)	0.037*
SCr (μmol/L) (IQR)	74.0 (56.5, 120.5)	69.0 (56.0, 99.0)	150.0 (88.25, 210.0)	< 0.001*
PCT (ng/mL)	6.4 (1.3, 37.1)	6.6 (0.9, 37.1)	5.0 (1.4, 36.4)	0.546
CRP (mg/l) (mean ± SD)	133.7 ± 90.3	130.3 ± 89.4	150.3 ± 94.5	0.322

ALT: alanine aminotransferase; ALP: alkaline phosphatase; AST: aspartate transaminase; BSI: bloodstream infection; CRP:C-reactive protein; γ-GT: γ-gamma glutamyl transpeptidase; IQR: interquartile range; KP: *Klebsiella pneumoniae*; LDH: lactate dehydrogenase; PCT: procalcitonin; SD: standard deviation; SCr: Serum creatinine; Tbil: total bilirubin. *Significant.

Table 3. Comparison of the sources of KP-BSI.

	Total (n = 145)	Survival group (n = 121)	Non-survival group (n = 24)	*p* value
Primary site of infection				
Lower respiratory tract infections	23 (15.9%)	15 (12.4%)	8 (33.3%)	0.010*
Intra-abdominal infection	5 (3.4%)	4 (3.3%)	1 (4.2%)	> 0.999
Urinary tract infection	26 (17.9%)	22 (18.2%)	4 (16.7%)	> 0.999
Liver infection	35 (24.1%)	31 (25.6%)	4 (16.7%)	0.500
Biliary tract infection	25 (17.2%)	24 (19.8%)	1 (4.2%)	0.119
Bloodstream infection of unknown origin	24 (16.6%)	18 (14.9%)	6 (25.0%)	0.223
Other[a]	6 (4.1%)	6 (5.0%)	0 (0.0%)	0.265

[a] Skin and soft tissue infections; central venous catheter infections: and intracranial infections; *Significant. BSI: bloodstream infection; KP: *Klebsiella pneumoniae*.

levels. Regarding liver and kidney function, alanine aminotransferase (ALT), aspartate transaminase (AST), lactate dehydrogenase (LDH), total bilirubin (TBil), and serum creatinine (SCr) levels were higher in the non-survival group than in the survival group. However, the procalcitonin levels, C-reactive protein levels, white blood cell counts and neutrophil percentages were not significantly different between the two groups (all $p < 0.05$) (Table 2).

Isolates and sources of KP-BSI

The main source of KP-BSI was liver infection (24.1%), followed by urinary tract infection (17.9%), and biliary tract infection (17.2%). There was a higher proportion of lower respiratory tract infections in the non-survival group than in the survival group (33.3% vs. 12.4%, $p < 0.05$). There were no significant differences in the case of other sources of infection between the two groups (Table 3).

Antibiotic resistance rate and antibiotic treatment

The drug sensitivity results for the two groups were incomplete. The analysis of the available drug sensitivity results showed that the KP strain had the highest rate of resistance to ticarcillin (53.8%), followed by ciprofloxacin (43.8%). The proportion of patients with KP resistance to cefoperazone/sulbactam (30.4% vs. 8.7%, $p = 0.004$), ceftazidime (29.1% vs. 16.4%, $p = 0.028$), imipenem (29.1% vs. 7.4%, $p = 0.002$), piperacillin/tazobactam (29.2% vs. 9.44%, $p =$

Table 4. Comparison of the microbiological characteristics with two groups.

	Total (n = 145)	Survival group (n = 121)	Non-survival group (n = 24)	*p* vale
Antibiotic resistance [a]				
Cefoperazone/sulbactam(115 vs. 23)[b]	17 (12.3%)	10 (8.7%)	7 (30.4%)	0.004*
Ceftazidime (116 vs. 23)[b]	28 (20.1%)	19 (16.4%)	9 (39.1%)	0.028*
Meropenem (9 vs. 7)[b]	5 (31.3%)	1 (11.1%)	4 (57.1%)	0.154
Imipenem (121 vs. 24)[b]	16 (11.0%)	9 (7.4%)	7 (29.2%)	0.002*
Ertapenem (121 vs. 24)[b]	8 (6.6%)	6 (5.7%)	2 (13.3%)	0.673
Ceftriaxone (110 vs. 17)[b]	41 (32.3%)	35 (31.8%)	6 (35.3%)	0.775
Cefepime (117 vs. 24)[b]	30 (21.3%)	22 (18.8%)	8 (33.3%)	0.113
Cefsulodin (107 vs. 17)[b]	16 (12.9%)	13 (12.1%)	3 (17.6%)	0.811
Cefuroxime sodium (112 vs. 17)[b]	42 (32.6%)	36 (32.1%)	6 (35.3%)	0.796
Cefuroxime axetil (112 vs. 17)[b]	44 (34.1%)	38 (33.9%)	6 (35.3%)	0.912
Tigecyclinec (118 vs. 22)[b]	12 (8.6%)	8 (6.8%)	4 (18.2%)	0.181
Amikacin (118 vs. 21)[b]	6 (4.3%)	4 (3.4%)	3 (9.5%)	0.489
Piperacillin/tazobactam(117vs. 24)[b]	19 (13.5%)	11 (9.4%)	8 (33.3%)	0.002*
Levofloxacin (114 vs. 24)[b]	27 (19.6%)	17 (14.9%)	10 (41.7%)	0.003*
Ciprofloxacin (9 vs. 7)[b]	7 (43.8%)	3 (33.3%)	4 (57.1%)	0.657
Minocycline (9 vs. 7)[b]	4 (25.0%)	1 (11.1%)	3 (42.9%)	0.383
Ticarcillin (7 vs. 6)[b]	7 (53.8%)	3 (42.9%)	4 (66.7%)	0.764
Amoxicillin (7 vs. 6)[b]	16 (13.0%)	13 (12.1%)	3 (18.8%)	0.739
Cotrimoxazole (114 vs. 24)[b]	26 (18.8%)	18 (15.8%)	8 (33.3%)	0.046*
MDR	30 (20.7%)	20 (16.5%)	10 (41.7%)	0.005*
The antibiotypes prior to KP-SBI onset				
BL/BLI	74 (51.0%)	66 (54.5%)	8 (33.3%)	0.058
BL	50 (34.5%)	38 (31.4%)	12 (50.0%)	0.080
Other[c]	21 (10.3%)	8 (6.6%)	7 (29.2%)	0.001*
Target therapy				
Resistance of KP to target antibiotic	15 (50.0%)	5 (45.5%)	2 (66.7%)	> 0.999
Targeted treatment for co-pathogens	7 (50.0%)	5 (45.5%)	3 (66.7%)	> 0.999
Delayed antibiotic therapy	1 (0.7%)	1 (0.8%)	0 (0.0%)	> 0.999

[a]Not all listed drugs have been tested in all isolates. [b]The numbers in parentheses indicate the total number of *Klebsiella pneumoniae* isolates tested for drug sensitivity. [c] Fluoroquinolones:Aminoglycosides. *Significant. BL/BLI:β-lactam/β-lactamase inhibitors; BL:β-lactam antibiotics; BSI:bloodstream infection; KP: *Klebsiella pneumoniae*; MDR: multidrug resistance.

0.002), levofloxacin (41.7% vs. 14.9%, $p = 0.003$) and cotrimoxazole (33.3% vs. 9.4%, $p = 0.046$) was significantly higher in the non-survival group than in the survival group (all $p < 0.05$). Additionally, the proportion of causative organisms that were multidrug resistant was higher in the non-survival group than in the survival group (41.7% vs. 16.5%, $p = 0.005$) (Table 4).

Independent risk factors for mortality in patients with KP-BSI

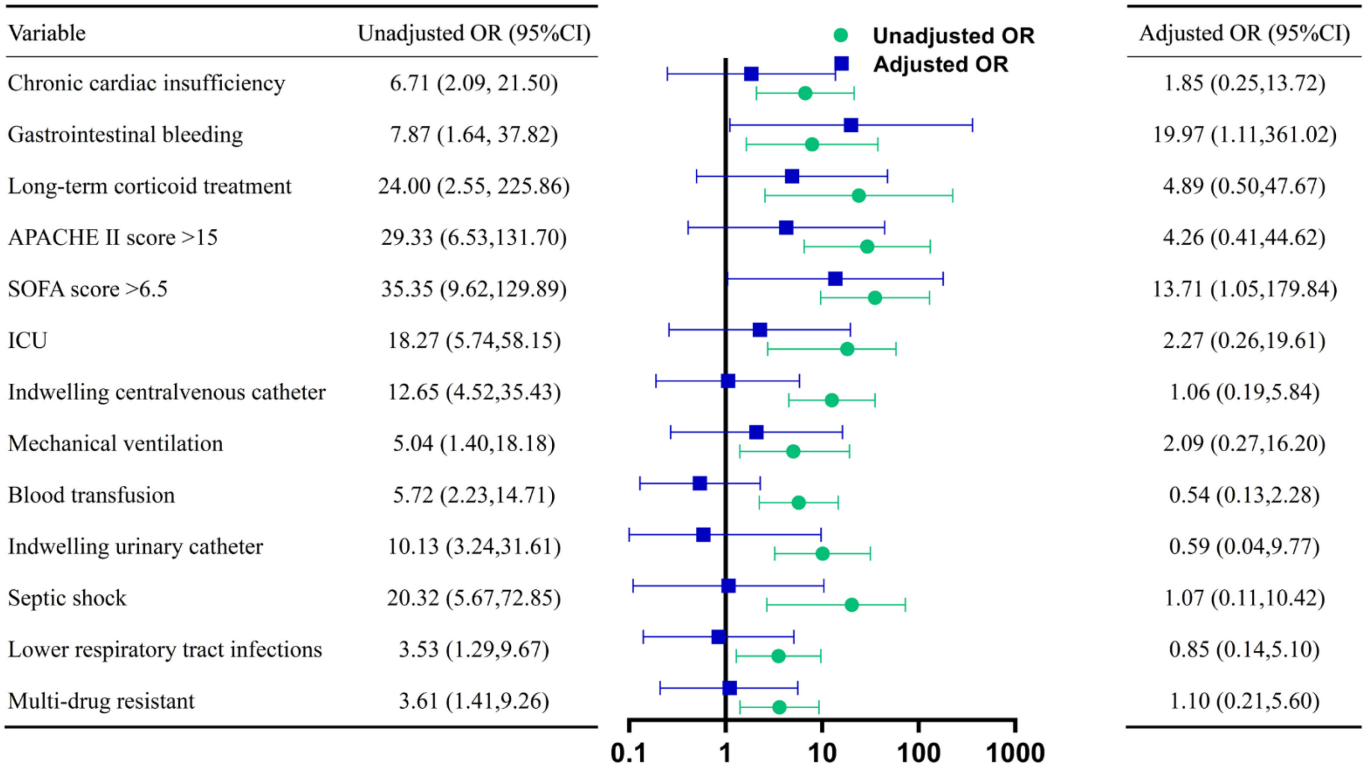
Multivariate logistic regression analysis indicated that SOFA score > 6.5 (OR, 13.71; 95% CI, 1.05–179.84), ICU admission (OR, 2.27; 95% CI, 0.26–19.61) and gastrointestinal bleeding (OR, 19.97; 95%, CI, 1.11–361.02) were independent risk factors for death in patients with KP-BSIs (Figure 2).

Discussion

We analyzed the clinical characteristics of patients with KP-BSIs and their risk factors for death to facilitate early detection and intervention, which are of great value for clinical diagnosis, treatment and evaluation.

The findings of our study indicate that higher APACHE II and SOFA scores are associated with poorer prognosis in patients with KP-BSIs; these results are consistent with those of previous research. Specifically, a SOFA score greater than 6.5 was an independent risk factor for death in these patients [20–22]. Interestingly, the CCI scores were not significantly different between the survival and non-survival groups; however, it is possible that a larger sample size could have yielded different results. In previous studies, ICU admission was shown to be an independent risk factor for KP-BSIs [23–25]. ICU patients often have a worse prognosis due to the severity of their condition and immunodeficiency; an observation that is generally consistent with the results of this study. In our study, a large proportion of poor prognosis was associated with surgery performed within 48 hours of blood collection. KP strains can form biofilms on and attach to catheters, as reported by Schroll *et al.* [26], providing a possible explanation for the high proportion of indwelling catheters in the non-survival group. The results of this study suggest that medical staff should prioritize aseptic technique and hand hygiene to reduce the risk of colonization and infection. Overall, our study highlights the importance of the early identification and

Figure 2. Multivariable logistic regression of factors associated with KP-BSIs.



APACHE: acute physiology and chronic health evaluation; CI: confidence interval; KP-BSI: Klebsiella pneumoniae bloodstream infection; ICU: intensive care unit; OR: odds ratio; SOFA: sequential organ failure assessment.

management of KP-BSIs, particularly in ICU patients and those undergoing surgery. By addressing the underlying risk factors and implementing appropriate prevention measures, healthcare professionals can improve patient outcomes and reduce the burden of these infections.

KP is a conditionally pathogenic bacterium that is widely present in the natural environment and that can colonize the human respiratory and digestive tracts. Epidemiological studies have shown that KP strains have a high colonization rate in the digestive tract, with one study isolating 592 KP strains from stool samples of 954 healthy individuals in Asian countries; a rate of 62.1% [27]. Moreover, several studies have reported an 80% concordance between KP infections and colonizing strains [28,29]. These findings suggest that KP colonization in the digestive tract can lead to BSIs under certain disease conditions. In fact, Micozzi *et al.* analyzed the relationship between KP intestinal colonization and BSI rates, reporting rates as high as 58% [30]. This underscores the importance of preventing the spread of KP from the digestive tract to the bloodstream, as BSIs can lead to sepsis and an increased risk of patient mortality. Notably, the use of proton pump inhibitors, which are often prescribed for gastrointestinal bleeding, has been associated with an overgrowth of intestinal bacteria [31]. This could explain why patients with gastrointestinal bleeding and KP-BSIs have poorer outcomes, as observed in our study. Therefore, clinicians must remain vigilant in identifying and treating KP-BSIs in patients with gastrointestinal bleeding, as this complication typically portends a poor prognosis.

In recent years, drug-resistant KP has become a growing concern due to the widespread use of antibiotics. BSIs caused by drug-resistant KP are particularly worrisome because of limited antibiotic options and high mortality rates [32,33]. In this study, we observed that the most commonly used antibiotics before KP-BSI onset were β-lactam/β-lactamase inhibitors, followed by β-lactam antibiotics, with fluoroquinolones and aminoglycosides being the least used. Our findings reflect the prescribing practices of physicians at our center. Importantly, we also found that patients who had used fluoroquinolones or aminoglycosides before KP-BSI onset had a worse prognosis. This may be related to the use of quinolone antibiotics, as previous reports identified the use of quinolones as an independent risk factor for carbapenem-resistant KP (CRKP) [34,35]. Therefore, the use of quinolone antibiotics before disease onset is highly likely to contribute to the development of CRKP BSIs, which are associated with poor treatment outcomes and a worse prognosis. The limited antibiotic options and poor therapeutic effects in the treatment of multidrug-resistant KP likely contributed to the worse prognosis observed in patients with KP-BSIs [36]. Therefore, clinicians should exercise caution when prescribing antibiotics and consider the potential risks associated with antibiotic use.

Our study had some limitations. First, it was a single-center study, and we collected data on KP-BSIs across a 3-year period in our hospital; but the number of patients was still relatively small. Second, this was a retrospective study with inherent selection bias, confounding factors, and certain limitations. We reviewed and analyzed electronic medical record information, but some information was not available, such as the virulence and genotype of KP strains, which could not be further verified and analyzed in depth, thus leading to certain limitations. Third, we had no control over variables such as the type and timing of antibiotic administration, which prevented us from providing appropriate treatment recommendations.

Conclusions

In summary, the independent risk factors for death in patients with KP-BSIs were SOFA score > 6.5, admission to the ICU, and gastrointestinal bleeding. Clinicians need to be vigilant in the case of patients with KP-BSIs and concurrent gastrointestinal bleeding; and especially with patients who have multiple organ failure and are admitted to the ICU. They need to perform early pathogenic investigations and administer appropriate empirically based treatment.

Authors' contributions

PX: collected data collection and drafted manuscript; QC, CZ, XZ: conceived the research and designed the study; XL: collected blood culture samples; RL: supervised the research; CZ: statistical analysis and revised the manuscript. All authors were responsible for acquiring, analyzing, or interpreting the data; and critically revising the manuscript for important intellectual content. Panpan Xu, Xijiang Zhang, and Qingqing Chen contributed equally to this work.

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