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ORIGINAL RESEARCH

Comparative Analysis of Clinical Characteristics and Antimicrobial Resistance of IA-BSI Caused by *Escherichia coli* and *Klebsiella pneumoniae*

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Objective: To investigate the clinical characteristics and antimicrobial resistance differences in patients with Intra-abdominal infection-associated bloodstream infection (IA-BSI) caused by *Escherichia coli* (*E. coli*) and *Klebsiella pneumoniae* (*K. pneumoniae*).

Methods: A total of 276 patients with Intra-abdominal infection-associated bloodstream infection (IA-BSI) were retrospectively enrolled from a single center, including 185 cases in the *Escherichia coli* group and 91 cases in the *Klebsiella pneumoniae* group. The chi-square test and Mann–Whitney *U*-test were used to compare clinical characteristics and antimicrobial resistance profiles between the two groups, and Kaplan-Meier curves were plotted to evaluate 30-day prognosis.

Results: (1) The *Klebsiella pneumoniae* group had higher Pitt scores, SOFA scores, and carbapenem-resistant (CR) rates ($P<0.05$); (2) The *Escherichia coli* group showed significantly higher ESBL-positive rates and quinolone resistance rates ($P<0.05$); (3) The CR group had higher Pitt and SOFA scores, with an inapplicability rate of empirical therapy of 90.0% and a mortality rate of 60.0%; (4) No statistically significant difference in 30-day mortality was observed between the *Escherichia coli* and *Klebsiella pneumoniae* groups ($P=0.24$).

Conclusion: There are heterogeneities in clinical characteristics and antimicrobial resistance profiles between patients with IA-BSI caused by *Escherichia coli* and *Klebsiella pneumoniae*. Patients infected with *Klebsiella pneumoniae* present with more severe conditions, higher carbapenem resistance rates and greater treatment difficulty, whereas *Escherichia coli* is characterized by high ESBL-positive rates and high quinolone resistance rates. This study provides evidence for clinical selection of antimicrobial agents, and further multicenter studies are needed for validation.

Keywords: *Escherichia coli*, *Klebsiella pneumoniae*, intra-abdominal infection, bloodstream infection

Introduction

Intra-abdominal infections (IAIs) refer to infections involving any intra-abdominal viscera including the peritoneum, ranging from common acute appendicitis and cholecystitis to postoperative intra-abdominal infections. IAIs mostly occur due to damage to the gastrointestinal mucosal defense barrier and invasion of intestinal flora into the abdominal cavity. They are the second most prevalent infection in intensive care units (ICUs) worldwide, with morbidity and mortality only lower than pneumonia. Moreover, IAIs serve as a critical trigger for bloodstream infection (BSI) in critically ill patients. Differences in prognosis are mainly associated with infection type, underlying diseases, comorbidities, disease severity and organ dysfunction.[1–3] Treatment of intra-abdominal infections mainly consists of anti-infective therapy, source control, nutritional support and organ function support. Timely and rational selection of antimicrobial agents and effective source control are crucial for improving patient prognosis.[3]

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Pathogens of intra-abdominal infections are mostly derived from normal intestinal flora, and infection tends to occur when the gastrointestinal mucosal barrier is impaired. Intra-abdominal infections are predominantly polymicrobial, usually a mixture of Gram-positive and Gram-negative bacteria, accompanied by a certain number of anaerobes, and some cases may also involve fungal infection.[1] Available epidemiological evidence indicates that the predominant pathogens of intra-abdominal infections are *Enterobacteriaceae*, *Enterococcus*, Anaerobes and *Streptococcus*,[4] Among them, Gram-negative bacteria account for the highest proportion, with the top five species being *Escherichia coli, Klebsiella pneumoniae, Pseudomonas aeruginosa, Enterobacter cloacae* and *Acinetobacter baumannii*.[5] *Escherichia coli* (*E. coli*) and *Klebsiella pneumoniae* (*K. pneumoniae*) are the predominant pathogens and also the leading causative agents of bloodstream infection secondary to intra-abdominal infection. Existing studies have confirmed that *Enterobacteriaceae* are the most commonly isolated pathogens in intra-abdominal infection-associated bloodstream infection (IA-BSI), among which *Escherichia coli* and *Klebsiella pneumoniae* predominate. Their infection rates and pathogenic risks are significantly higher than those of other strains such as *Enterococcus*, Anaerobes and *Streptococcus*, further highlighting their core pathogenic roles in IA-BSI. Significant differences exist between the two species in virulence characteristics, antimicrobial resistance phenotypes and clinical prognosis, which directly affect the selection of empirical anti-infective regimens.

In terms of etiological evolution, the species composition of pathogens causing intra-abdominal infections has not changed significantly in recent years, whereas bacterial antimicrobial resistance has become increasingly severe. The main resistance mechanism of Enterobacteriaceae is mediated by β-lactamases, among which extended-spectrum β-lactamase (ESBL)-producing strains are the most prevalent. The detection rates of ESBL-producing *Escherichia coli* and *Klebsiella pneumoniae* in China are markedly higher than those in European and American countries. Due to irrational use of antimicrobials, advanced age, immunocompromise and other factors, patients with intra-abdominal infections are susceptible to secondary infections caused by ESBL-producing *Enterobacterales* (ESBL-E) and carbapenem-resistant *Enterobacterales* (CRE), further increasing the difficulty of clinical diagnosis and treatment.

Epidemiological data show that the mortality burden associated with bacterial infections is substantial, with bloodstream infections contributing a remarkably high proportion of deaths. In 2019, deaths attributable to 33 bacterial pathogens accounted for 13.6% of all global deaths, among which bloodstream infection-related deaths reached 56.2%. Deaths caused by *Escherichia coli* and *Klebsiella pneumoniae* made up 54.9% of total bacterial-associated deaths.[6] Multicenter studies have confirmed that Gram-negative bacteria are the predominant pathogens of bloodstream infections, accounting for 59.8%, with *Escherichia coli* and *Klebsiella pneumoniae* as the dominant strains,[7] heir pathogen spectrum is highly consistent with that of intra-abdominal infections, which suggests intra-abdominal infections are an important source of bloodstream infections induced by these pathogens, further establishing the core pathogenic position of the two strains in IA-BSI.

Although both *Escherichia coli* and *Klebsiella pneumoniae* are core pathogens of IA-BSI, they differ significantly in virulence characteristics, antimicrobial resistance profiles and clinical prognosis, which directly affects the selection of empirical treatment regimens. At present, studies focusing on intra-abdominal-origin bloodstream infections remain scarce. Most existing studies analyze bloodstream infections of all sources collectively, failing to reflect the clinical features and resistance patterns of intra-abdominal-derived infections, and thus providing limited guidance for clinical diagnosis and treatment.[8–11] Therefore, this study comparatively analyzes the clinical characteristics and antimicrobial resistance differences of IA-BSI caused by *Escherichia coli* and *Klebsiella pneumoniae*. It aims to clarify the discrepancies between the two pathogens, guide the selection of empirical antimicrobial agents, optimize clinical stratified management of IA-BSI, reduce the risks of drug-resistant bacterial transmission and mortality, and ultimately improve patient prognosis.

Methods

Subjects and Groups Studied

This study retrospectively analyzed 276 patients with Intra-abdominal infection-associated bloodstream infection (IA-BSI) hospitalized at the First Affiliated Hospital of Dali University from 2022 to 2025. IA-BSI was defined as secondary bloodstream infection confirmed by blood culture with a definite intra-abdominal infectious focus, excluding other primary infection sources such as catheters, urinary tract, lungs, skin and soft tissues. A total of 276 patients were

enrolled in this study, who were divided into the *Escherichia coli* (*E. coli*) group (185 cases) and the *Klebsiella pneumoniae* (*K. pneumoniae*) group (91 cases) according to causative pathogens. Based on 30-day prognosis, each group was further subdivided into non-survivor and survivor subgroups: 22 non-survivors and 163 survivors in the *E. coli* group, and 17 non-survivors and 74 survivors in the *K. pneumoniae* group.Inclusion criteria: ① Age ≥18 years old; ② At least one positive blood culture for *E. coli* or *K. pneumoniae* within 24 hours after confirmation of intra-abdominal infection.Exclusion criteria: ① Patients with incomplete clinical data; ② Bloodstream infection of non-abdominal origin; ③ Concurrent infection at other sites on admission; ④ Pregnant or lactating women.

Data Collection

Clinical data of patients in both groups were collected via the Hospital Information System (HIS), including age, gender, length of hospital stay, underlying diseases (hypertension, diabetes mellitus, coronary heart disease, cerebral infarction), complications (hypokalemia, hypoalbuminemia, anemia), immunosuppressive status, antibiotic use, bacterial resistance phenotypes and antimicrobial susceptibility results. Meanwhile, the Pitt Bacteremia Score (Pitt score) and Sequential Organ Failure Assessment (SOFA) score on admission were calculated, and comorbidities were evaluated using the Charlson Comorbidity Index (CCI). Microbiological Methods:

Strain identification and antimicrobial susceptibility testing were performed using an automated microbial analysis system. Susceptibility results were interpreted according to the standards of the Clinical and Laboratory Standards Institute (CLSI). Appropriateness of empirical antimicrobial therapy was determined based on the *Guidelines for Clinical Application of Antimicrobial Agents (2015 Edition)*. Empirical therapy was defined as appropriate if anti-infective treatment was initiated before blood culture reports were available, the pathogen was susceptible to the prescribed antimicrobial agent in vitro, and clinical symptoms and inflammatory markers improved within 14 days of treatment. Therapy was regarded as inappropriate if the isolate was resistant or intermediate to the agent and there was no improvement in clinical symptoms or inflammatory markers after 14 days of treatment.

Statistical Analysis

Statistical analyses were conducted using SPSS 25.0. Non-normally distributed continuous data were expressed as median (interquartile range) [M (Q1, Q3)], and the Mann–Whitney *U*-test was adopted for between-group comparisons. Categorical data were presented as frequency and percentage [n (%)], with the χ^2-test or Fisher's exact test used for group comparisons. Kaplan-Meier curves were generated to compare survival outcomes between *Klebsiella pneumoniae* and *Escherichia coli*. A P-value of less than 0.05 was considered statistically significant.

Results

Comparison of Clinical Characteristics of Patients with IA-BSI Caused by Escherichia coli and Klebsiella pneumoniae

A total of 276 patients with IA-BSI were enrolled, including 185 (67.0%) infected with *Escherichia coli* and 91 (33.0%) with *Klebsiella pneumoniae*. There were no statistically significant differences between the two groups in age, gender, length of hospital stay, site of infection, CCI score, 30-day prognosis, or the incidence rates of liver cirrhosis, hypertension, diabetes mellitus, coronary heart disease, hypokalemia and anemia. The *Klebsiella pneumoniae* group had significantly higher Pitt score and SOFA score ($P<0.05$), as well as higher incidences of immunosuppression, liver abscess, cerebral infarction and hypoalbuminemia. Antimicrobial resistance analysis revealed that carbapenem resistance rate was significantly higher in the *Klebsiella pneumoniae* group ($P<0.001$), whereas ESBL-positive rate was higher in the *Escherichia coli* group (57.8% vs 31.9%). No significant difference was observed in empirical antimicrobial treatment regimens between the two groups (Table 1). Kaplan-Meier survival analysis showed no statistically significant difference in 30-day prognosis between the two groups ($P=0.24$) (Figure 1).

Table 1 Comparison of Clinical Characteristics of Patients with IA BSI Caused by *Escherichia coli* and *Klebsiella pneumoniae*

Variables	Overall (N = 276)	Intra-Abdominal Infection		*P*-values
		E. coli (N = 185)	*K. pneumoniae* (N = 91)	
Age	63 (54, 70)	63 (54, 71)	61 (53, 69)	0.206
Length of stay	20 (12, 28)	19 (12, 27)	21 (13, 33)	0.229
Gender				
Female	121(43.8%)	86 (46.5%)	35 (38.5%)	0.207
Male	155(56.2%)	99 (53.5%)	56 (61.5%)	
Acquisition				
Community (n, %)	121(43.8%)	77 (41.6%)	44 (48.4%)	0.289
Hospital (n, %)	155(56.2%)	108(58.4%)	47 (51.6%)	
Disease severity				
Pitt score	1.00(0.00,2.00)	1.00(0.00,2.00)	1.00(0.00,3.00)	0.009
Sofa score	4.0 (2.0, 6.0)	4.0 (2.0, 6.0)	5.0 (3.0, 7.0)	0.019
CCI score	4.00(2.00,6.00)	4.00(2.00,6.00)	4.00(3.00,6.00)	0.844
Empiric therapy				
Antibiotics ≥2 during hospitalization	8 (2.9%)	2 (1.1%)	6 (6.6%)	
Carbapenems	49 (17.8%)	31 (16.8%)	18 (19.8%)	
Cefoxitin	5 (1.8%)	4 (2.2%)	1 (1.1%)	
Cephalosporins	83 (30.1%)	62 (33.5%)	21 (23.1%)	
Latamoxef	1 (0.4%)	1 (0.5%)	0 (0.0%)	
No	15 (5.4%)	6 (3.2%)	9 (9.9%)	
Piperacillin	93 (33.7%)	66 (35.7%)	27 (29.7%)	
Quinolones	21 (7.6%)	12 (6.5%)	9 (9.9%)	
Tetracyclines	1 (0.4%)	1 (0.5%)	0 (0.0%)	
Bacterial type				
Carbapenem-resistant strains	10 (3.6%)	0 (0.0%)	10 (11.0%)	<0.001
ESBL-producing strains	136 (49.3%)	107 (57.8%)	29 (31.9%)	
NO	130 (47.1%)	78 (42.2%)	52 (57.1%)	
Outcome				
Death (n, %)	39 (14.1%)	22 (11.9%)	17 (18.7%)	0.128
Survival (n, %)	237 (85.9%)	163 (88.1%)	74 (81.3%)	
Immunosuppression				
No (n, %)	234 (84.8%)	166 (89.7%)	68 (74.7%)	0.001
Yes (n, %)	42 (15.2%)	19 (10.3%)	23 (25.3%)	
Underlying disease				
Liver abscess (n, %)	30 (10.9%)	14 (7.6%)	16 (17.6%)	0.012
Liver cirrhosis (n, %)	43 (15.6%)	33 (17.8%)	10 (11.0%)	0.140
Hypertension (n, %)	49 (17.8%)	33 (17.8%)	16 (17.6%)	0.958
Diabetes mellitus (n, %)	47 (17.0%)	28 (15.1%)	19 (20.9%)	0.233
Coronary heart disease (n, %)	6 (2.2%)	2 (1.1%)	4 (4.4%)	0.094
Cerebral infarction (n, %)	7 (2.5%)	2 (1.1%)	5 (5.5%)	0.041
Hypokalemia (n, %)	17 (6.2%)	11 (5.9%)	6 (6.6%)	0.833
Hypoproteinaemia (n, %)	84 (30.4%)	49 (26.5%)	35 (38.5%)	0.042
Anemia (n, %)	35 (12.7%)	19 (10.3%)	16 (17.6%)	0.086

Comparison of Characteristics Between Non-Survivor and Survivor Groups of IA-BSI Patients Caused by Klebsiella pneumoniae and Escherichia coli

In this study, patients were grouped by pathogen type and further divided into non-survivor and survivor subgroups based on 30-day prognosis to compare baseline characteristics between subgroups. Among patients infected with *Escherichia coli*, there were no statistically significant differences between non-survivors and survivors in age, gender, source of

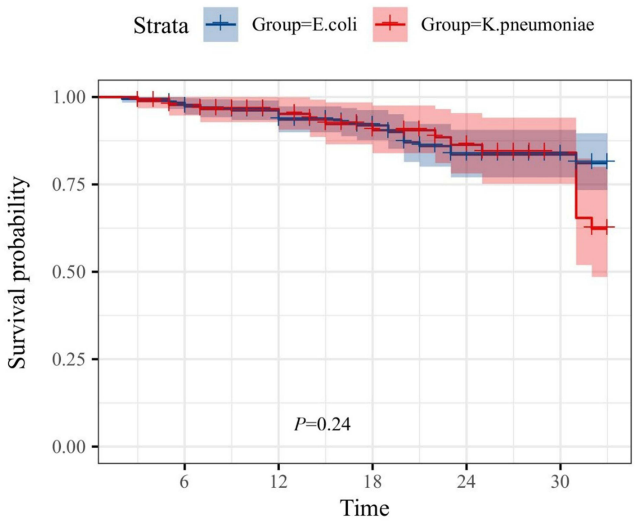

Figure 1 Analysis of 30-day survival curves for patients infected with *Escherichia coli* and *Klebsiella pneumoniae*.

infection, Pitt score, appropriateness of empirical therapy, resistance phenotypes, or incidence of underlying diseases. However, non-survivors had a shorter hospital stay, significantly higher SOFA score (5.0 vs 3.0, *P*=0.048) and CCI score (6.50 vs 4.00, *P*=0.013), as well as markedly higher incidences of hypoalbuminemia (54.5% vs 22.7%, *P*=0.001) and anemia (36.4% vs 6.7%, *P*<0.001).For patients infected with *Klebsiella pneumoniae*, no significant differences were found between non-survivors and survivors in age, gender, length of hospital stay, CCI score, or incidence of underlying diseases and complications. Nevertheless, non-survivors presented with significantly higher Pitt score (3.00 vs 1.00, *P*<0.001) and SOFA score (7.0 vs 5.0, *P*=0.007), a significantly lower rate of appropriate empirical antimicrobial therapy (47.1% vs 89.2%, *P*<0.001), and higher proportions of carbapenem-resistant phenotype (35.3% vs 5.4%, *P*=0.004), nosocomial-acquired infection (76.5% vs 45.9%, *P*=0.023) and hypokalemia (23.5% vs 2.7%, *P*=0.010) (Table 2).

Table 2 To Compare the Characteristics Between the Death Group and the Survival Group of Patients with IA BSI Caused by *Klebsiella pneumoniae* and *Escherichia coli*

Variables	***E. coli* (N=185)**			***K. pneumoniae* (N=91)**		
	Death (N=22)	**Survival (N=163)**	***P*-values**	**Death (N=17)**	**Survival (N=74)**	***P*-values**
Age, Median	65 (46,70)	63 (54,71)	0.747	64 (53,73)	61 (53,67)	0.575
Length of stay	14 (7,20)	20 (12,28)	0.006	23 (14,52)	20 (13,33)	0.370
Gender						
Female (n, %)	9(40.9%)	77(47.2%)	0.576	8(47.1%)	27(36.5%)	0.419
Male (n, %))	13(59.1%)	86(52.8%)		9(52.9%)	47(63.5%)	
Acquisition						
Community (n, %)	11(50.0%)	66(40.5%)	0.396	4(23.5%)	40(54.1%)	0.023
Hospital (n, %)	11(50.0%)	97(59.5%)		13(76.5%)	34(45.9%)	
Disease severity						
Pitt score	1.00(0.00, 2.00)	1.00(0.00, 2.00)	0.755	3.00(2.00, 5.00)	1.00(0.00, 2.00)	<0.001
Sofa score	5.0 (3.0, 8.0)	3.0(2.0,6.0)	0.048	7.0 (5.0, 10.0)	5.0 (3.0, 5.0)	0.007
CCI score	6.50(3.00, 9.00)	4.00(2.00,6.00)	0.013	4.00(3.00,6.00)	4.00(3.00, 5.00)	0.766
Appropriate empirical antibiotic therapy						
No (n, %)	3 (13.6%)	30 (18.4%)	0.770	9 (52.9%)	8 (10.8%)	<0.001
Yes (n, %)	19(86.4%)	133(81.6%)		8 (47.1%)	66(89.2%)	

(*Continued*)

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Table 2 (Continued).

Variables	*E. coli* (N=185)			*K. pneumoniae* (N=91)		
	Death (N=22)	Survival (N=163)	*P*-values	Death (N=17)	Survival (N=74)	*P*-values
Bacterial type						
Carbapenem-resistant strains (n, %)	0 (0.0%)	0 (0.0%)	0.493	6 (35.3%)	4 (5.4%)	0.004
ESBL-producing strains (n, %)	11(50.0%)	96 (58.9%)		5 (29.4%)	24(32.4%)	
No	11(50.0%)	67 (41.1%)		6 (35.3%)	46(62.2%)	
Immunosuppression						
No (n, %)	20(90.9%)	146(89.6%)	>0.999	15(88.2%)	53(71.6%)	0.220
Yes (n, %)	2 (9.1%)	17 (10.4%)		2 (11.8%)	21(28.4%)	
Underlying disease						
Liver abscess (n, %)	2 (9.1%)	12 (7.4%)	0.675	1 (5.9%)	15 (20.3%)	0.288
Liver cirrhosis (n, %)	5 (22.7%)	28 (17.2%)	0.554	3 (17.6%)	7 (9.5%)	0.389
Hypertension (n, %)	5 (22.7%)	28 (17.2%)	0.554	4 (23.5%)	12(16.2%)	0.489
Diabetes mellitus (n, %)	5 (22.7%)	23 (14.1%)	0.339	2 (11.8%)	17(23.0%)	0.509
Coronary heart disease (n, %)	0 (0.0%)	2 (1.2%)	>0.999	1 (5.9%)	3 (4.1%)	0.569
Cerebral infarction (n, %)	0 (0.0%)	2 (1.2%)	>0.999	0 (0.0%)	5 (6.8%)	0.579
Hypokalemia (n, %)	4 (18.2%)	7 (4.3%)	0.029	4 (23.5%)	2 (2.7%)	0.010
Hypoproteinaemia (n, %)	12(54.5%)	37 (22.7%)	0.001	10(58.8%)	25(33.8%)	0.056
Anemia (n, %)	8 (36.4%)	11 (6.7%)	<0.001	6 (35.3%)	10(13.5%)	0.070

Comparison of Characteristics Among Patients in ESBL-Producing, Carbapenem-Resistant (CR), and Non-Resistant Groups

Patients were classified into three groups according to bacterial resistance phenotypes: carbapenem-resistant (CR) group (n=10), extended-spectrum β-lactamase-producing (ESBL) group (n=136), and non-resistant group (n=130). There were no statistically significant differences among the three groups in baseline indicators including age, gender, length of hospital stay, site of infection, CCI score, underlying diseases and complications. The CR group had significantly higher Pitt score and SOFA score than the ESBL and non-resistant groups, with an inappropriate empirical antimicrobial therapy rate of 90.0% ($P<0.001$) and a 30-day mortality rate of 60.0% ($P=0.002$). A statistically significant difference was observed in the incidence of anemia among the three groups ($P=0.031$), with the highest proportion (40.0%) in the CR group. No significant difference was found in the acquisition route of infection among the three groups (Table 3).

Table 3 The Characteristics of ESBL, CR and Non-Drug Resistance Groups Were Compared

Variables	Bacterial Type				*p*-value
	Overall (N=276)	CR (N=10)	ESBL (N=136)	No (N=130)	
Age, Median	63 (54, 70)	59 (53, 64)	64 (54, 71)	62 (53, 69)	0.340
Length of stay	20 (12, 28)	24 (14, 50)	18 (12, 28)	21 (12, 28)	0.356
Gender					
Female (n, %)	121 (43.8%)	4 (40.0%)	60 (44.1%)	57 (43.8%)	>0.999
Male (n, %)	155 (56.2%)	6 (60.0%)	76 (55.9%)	73 (56.2%)	
Acquisition					
Community (n, %)	121 (43.8%)	2 (20.0%)	55 (40.4%)	64 (49.2%)	0.113
Hospital (n, %)	155 (56.2%)	8 (80.0%)	81 (59.6%)	66 (50.8%)	
Disease severity					
Pitt score	1.00 (0.00, 2.00)	4.00 (1.00, 7.00)	1.00 (0.00, 2.00)	1.00 (0.00, 2.00)	0.047
Sofa score	4.0 (2.0, 6.0)	7.0 (5.0, 11.0)	4.0 (2.0, 6.0)	4.0 (2.0, 5.0)	0.021
CCI score	4.00 (2.00, 6.00)	3.50 (2.00, 4.00)	4.00 (2.00, 6.00)	4.00 (2.00, 6.00)	0.372

(*Continued*)

Table 3 (Continued).

Variables	Bacterial Type				*p*-value
	Overall (N=276)	CR (N=10)	ESBL (N=136)	No (N=130)	
Appropriate empirical antibiotic therapy					
No (n, %)	50 (18.1%)	9 (90.0%)	26 (19.1%)	15 (11.5%)	<0.001
Yes (n, %)	226 (81.9%)	1 (10.0%)	110 (80.9%)	115 (88.5%)	
Outcome					
Death (n, %)	39 (14.1%)	6 (60.0%)	16 (11.8%)	17 (13.1%)	0.002
Survival (n, %)	237 (85.9%)	4 (40.0%)	120 (88.2%)	113 (86.9%)	
Immunosuppression					
No (n, %)	234 (84.8%)	8 (80.0%)	121 (89.0%)	105 (80.8%)	0.127
Yes (n, %)	42 (15.2%)	2 (20.0%)	15 (11.0%)	25 (19.2%)	
Underlying disease					
Liver abscess (n, %)	30 (10.9%)	0 (0.0%)	13 (9.6%)	17 (13.1%)	0.462
Liver cirrhosis (n, %)	43 (15.6%)	0 (0.0%)	25 (18.4%)	18 (13.8%)	0.247
Hypertension (n, %)	49 (17.8%)	0 (0.0%)	21 (15.4%)	28 (21.5%)	0.144
Diabetes mellitus (n, %)	47 (17.0%)	0 (0.0%)	21 (15.4%)	26 (20.0%)	0.249
Coronary heart disease (n, %)	6 (2.2%)	0 (0.0%)	4 (2.9%)	2 (1.5%)	0.748
Cerebral infarction (n, %)	7 (2.5%)	0 (0.0%)	3 (2.2%)	4 (3.1%)	0.782
Hypokalemia (n, %)	17 (6.2%)	1 (10.0%)	6 (4.4%)	10 (7.7%)	0.299
Hypoproteinaemia (n, %)	84 (30.4%)	4 (40.0%)	42 (30.9%)	38 (29.2%)	0.708
Anemia (n, %)	35 (12.7%)	4 (40.0%)	18 (13.2%)	13 (10.0%)	0.031

Comparative Analysis of Antimicrobial Resistance Between Klebsiella pneumoniae and Escherichia coli

This study compared the resistance profiles of 185 *Escherichia coli* isolates and 91 *Klebsiella pneumoniae* isolates against 12 commonly used antimicrobial agents, with susceptibility results interpreted as susceptible (S), intermediate (I), and resistant (R). Inter-group comparisons showed no significant differences in resistance rates to amikacin, cefepime, and piperacillin (P=0.483, 0.438, 0.473, respectively). *Escherichia coli* exhibited significantly higher resistance rates to amoxicillin-clavulanate (44.3% vs 34.1%, P<0.001), aztreonam (43.8% vs 30.8%, P=0.038), ciprofloxacin (63.8% vs 47.3%, P<0.001), gentamicin (35.1% vs 19.8%, P=0.024), ceftriaxone (63.2% vs 37.4%, P<0.001), cefoxitin (23.2% vs 19.8%, P=0.048), tobramycin (11.4% vs 9.9%, P=0.016), and levofloxacin (58.9% vs 29.7%, P<0.001). In contrast, *Klebsiella pneumoniae* had a significantly higher imipenem resistance rate than *Escherichia coli* (11.0% vs 0.5%, P<0.001) (Table 4).

Table 4 Comparative Analysis of Drug Resistance of *Klebsiella pneumoniae* and *Escherichia coli*

Antimicrobials (n, %)	*E. coli* (N=185)			*K. pneumoniae* (N=91)			*p*-value
	S	I	R	S	I	R	
Amikacin	180(97.3%)	-	5 (2.7%)	87 (95.6%)	-	4 (4.4%)	0.483
Amoxicillin clavulanate potassium	103(55.7%)	-	82 (44.3%)	53 (58.2%)	7 (7.7%)	31 (34.1%)	<0.001
Aztreonam	104(56.2%)	-	81 (43.8%)	63 (69.2%)	-	28 (30.8%)	0.038
Ciprofloxacin	51 (27.6%)	16 (8.6%)	118(63.8%)	47 (51.6%)	1 (1.1%)	43 (47.3%)	<0.001
Gentamycin	116(62.7%)	4 (2.2%)	65 (35.1%)	71 (78.0%)	2 (2.2%)	18 (19.8%)	0.024
Ceftriaxone	68 (36.8%)	-	117(63.2%)	57 (62.6%)	-	34 (37.4%)	<0.001
Cefoxitin	118(63.8%)	24 (13.0%)	43 (23.2%)	69 (75.8%)	4 (4.4%)	18 (19.8%)	0.048
Cefepime	144(77.8%)	-	41 (22.2%)	67 (73.6%)	-	24 (26.4%)	0.438
Tobramycin	115(62.2%)	49 (26.5%)	21 (11.4%)	71 (78.0%)	11 (12.1%)	9 (9.9%)	0.016
Imipenem	184(99.5%)	-	1 (0.5%)	80 (87.9%)	1 (1.1%)	10 (11.0%)	<0.001

(*Continued*)

Table 4 (Continued).

Antimicrobials (n, %)	*E. coli* (N=185)			*K. pneumoniae* (N=91)			*p*-value
	S	I	R	S	I	R	
Levofloxacin	31 (16.8%)	45 (24.3%)	109(58.9%)	48 (52.7%)	16 (17.6%)	27 (29.7%)	<0.001
Piperacillin	161(87.0%)	13 (7.0%)	11 (5.9%)	75 (82.4%)	7 (7.7%)	9 (9.9%)	0.473

Notes: Drug sensitivity results were determined as S (sensitive), I (intermediate resistance), R (resistance).

Discussion

Intra-abdominal infection is a common severe clinical infection and a major predisposing factor for secondary bloodstream infection, with the mortality rate of bloodstream infections reaching 20%–40%. *Escherichia coli* and *Klebsiella pneumoniae* are the predominant pathogens of IA-BSI. With the extensive use of antimicrobial agents, their antimicrobial resistance continues to evolve, and the prevalence of multidrug-resistant and carbapenem-resistant strains markedly increases treatment difficulties. Rising antimicrobial resistance in IA-BSI poses severe challenges to empirical antimicrobial therapy and infection prevention and control.At present, comparative studies focusing on these two pathogens in IA-BSI are scarce. Clarifying their differences is of great clinical value for stratified diagnosis-treatment and antimicrobial selection. This study specifically focused on patients with IA-BSI and compared clinical characteristics and resistance profiles between IA-BSI caused by *E. coli* and *K. pneumoniae*. Although no significant difference in 30-day prognosis was found between the two groups, remarkable heterogeneity existed in disease severity, resistance phenotypes and prognostic factors. These findings compensate for the limitations of studies on bloodstream infections of all origins, and can provide evidence for clinical stratified management, antimicrobial selection and nosocomial infection prevention and control.

This study demonstrated that the *Klebsiella pneumoniae* group had significantly higher Pitt score, SOFA score and carbapenem resistance rate, which represents the core clinical value of comparing these two pathogens in the present research. *Klebsiella pneumoniae* possesses high-virulence biological characteristics. Its capsular polysaccharides (K-antigens) effectively inhibit phagocytosis by host phagocytes, its fimbrial structures enhance adhesion to host mucosa, and the pathogen readily forms biofilms to resist antimicrobial penetration and reduce therapeutic efficacy,[12] resulting in more severe conditions and more difficult treatment. In contrast, as a dominant commensal bacterium in the human intestinal tract, *Escherichia coli* infection is mostly associated with gastrointestinal mucosal barrier damage and bacterial translocation. It exhibits relatively mild virulence phenotypes, with most strains lacking high-virulence plasmids and exerting weak virulence effects.[13] Significant differences in virulence, antimicrobial resistance and disease severity between the two strains confer clear clinical significance to their comparison, which can guide stratified treatment and empirical antimicrobial selection according to different pathogens.[14] Kaplan-Meier survival analysis revealed no statistically significant difference in 30-day survival rate between the two groups (P=0.24), which may be attributed to factors such as underlying diseases and short-term follow-up.

In recent years, the antimicrobial resistance rate of *Klebsiella pneumoniae* has increased year by year, markedly narrowing therapeutic options for clinical anti-infective treatment. With the extensive clinical application of carbapenem antibiotics, the detection rate of carbapenem-resistant *Klebsiella pneumoniae* (CRKP) has also shown a significant upward trend.[12,15–18] In this study, 19.8% of patients in the *Klebsiella pneumoniae* group received empirical carbapenem therapy, which may intensify selective pressure for carbapenem-resistant strains. This also reflects a real-world issue in clinical practice: clinicians tend to prefer high-grade antimicrobials for potential highly resistant pathogens. Although this strategy may control infection and relieve symptoms in the short term, it masks the underlying risks of high virulence and high resistance of the strains. The carbapenem resistance rate of *Klebsiella pneumoniae* in our study (11.0%) was higher than that reported in previous literature,[11] mainly because our cohort was dominated by intra-abdominal-origin and hospital-acquired infections. Cross-transmission and invasive procedures in the ICU further increase the risk of antimicrobial resistance.[11,19,20]

In this study, the mortality rate of patients infected with carbapenem-resistant strains reached 60.0%, and the rate of inappropriate empirical therapy was as high as 90.0%. Carbapenem-resistant *Klebsiella pneumoniae* often carries multiple resistance genes simultaneously, leaving limited therapeutic options. In addition, most such patients have

hospital-acquired infections complicated with underlying conditions such as immunosuppression and hypoalbuminemia, resulting in impaired host anti-infection defense capacity. Failure to integrate local antimicrobial resistance surveillance data into empirical treatment decisions may easily lead to inappropriate regimens, thereby delaying disease management and worsening prognosis. As one of the major carbapenemase-producing Enterobacterales species and a key multidrug-resistant pathogen in ICUs, elevated resistance levels of *Klebsiella pneumoniae* directly affect patient outcomes.[21–23]

One previous study reported ESBL detection rates of 27.2% and 14.9%, and ciprofloxacin resistance rates of 38.7% and 21.0% for *Escherichia coli* and *Klebsiella pneumoniae*, respectively.[24] Our study showed a similar trend: *E. coli* had a higher ESBL-positive rate (57.8% vs 31.9%) and significantly higher quinolone resistance rates than *K. pneumoniae*, with ciprofloxacin resistance at 63.8% vs 47.3% and levofloxacin at 58.9% vs 29.7%. These findings differ from those reported by Kim et al[24] Studies have confirmed that *Enterobacterales* are prone to produce β-lactamases, which are associated with plasmid-borne ESBL genes such as $bla_{\text{CTX-M}}$. Some isolates also carry the plasmid-mediated quinolone resistance gene *qnrB55*. Since these resistance genes are frequently located on the same transmissible plasmid, multidrug-resistant phenotypes readily emerge, further limiting clinical anti-infective therapeutic options.[25,26] In our region, cephalosporins and quinolones have long been the main empirical agents for intra-abdominal infections. Under antimicrobial selective pressure, co-existence and horizontal transmission of ESBL and quinolone resistance genes among *Enterobacterales* have been promoted. Differences in local antimicrobial prescribing patterns and resistance surveillance backgrounds may explain the relatively high quinolone resistance rates observed in our study. In addition, as a single-center retrospective analysis including only patients from one hospital, our results have limitations when generalized to other medical institutions.

Our study showed that both strains exhibited susceptibility rates to amikacin exceeding 90%, which provides important evidence for the clinical management of IA-BSI in this region. Amikacin combined with β-lactams can be prioritized for community-acquired infections, whereas amikacin plus tigecycline may be used for hospital-acquired infections. This regimen can cover resistant strains and reduce the overuse of carbapenems. It is recommended to establish a regional antimicrobial resistance surveillance system for IA-BSI to dynamically optimize empirical therapeutic regimens. Accordingly, we propose constructing a local IA-BSI surveillance system that regularly updates data on ESBL prevalence in *Escherichia coli* and carbapenem resistance in *Klebsiella pneumoniae*, so that empirical anti-infective strategies can be timely adjusted according to local resistance trends.

Despite the findings of this study, several limitations remain: (1) This is a single-center retrospective study with a limited sample size, which may affect the results; multicenter studies are needed to validate the conclusions. (2) Only short-term prognosis was evaluated, without long-term follow-up or in-depth investigation of molecular resistance mechanisms. (3) The impact of secondary infections at other sites during hospitalization could not be fully excluded. (4) As single-center data, the generalizability is restricted.

Notwithstanding these limitations, this study specifically focused on patients with IA-BSI, distinct from previous generalized studies on bloodstream infections of all origins. It clarified the heterogeneity between the two pathogens in terms of disease severity and resistance profiles, and proposed practical combination regimens based on local resistance data, which has practical reference value for primary hospitals with relatively scarce new antimicrobial agents.

In conclusion, there are significant differences in clinical characteristics and antimicrobial resistance profiles between IA-BSI caused by *Escherichia coli* and *Klebsiella pneumoniae. Klebsiella pneumoniae* presents a higher carbapenem resistance rate, whereas *Escherichia coli* is characterized by high rates of ESBL production and quinolone resistance. Clinically, strengthened resistance surveillance and rational antimicrobial use based on epidemiological data and individual patient conditions are warranted.

Data Sharing Statement

All data generated or analyzed during this study are included in this published article.

Ethics Approval and Consent to Participate

This study conformed to the guidelines of the Helsinki Declaration. Ethics approval was obtained by the Research Ethics Committee of the first Affiliated Hospital of Dali University. And the Ethics Committee waived the requirement for

informed consent due to the retrospective and observational nature of the investigation, as well as the anonymity of the data. Consent for Publication Written informed consent for publication was obtained from all participants.

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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, have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors report no conflicts of interest in this work.

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