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*Original Article*

# Differences in clinical characteristics of bloodstream infections caused by *Escherichia coli* and *Acinetobacter baumannii*

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**Abstract:** Background: Bloodstream infections (BSI) caused by *Escherichia coli* (*E. coli*) and *Acinetobacter baumannii* (*A. baumannii*) are on the rise, accompanied by high morbidity and mortality. The aim of this study was to investigate the differences between BSI caused by *E. coli* and *A. baumannii*. Methods: This was a retrospective study. A total of 118 patients were enrolled, including 63 cases of BSIs caused by *E. coli* and 55 cases of BSIs caused by *A. baumannii*. Clinical data were collected. Sources of infection, risk factors, drug resistance, mortality, and clinical characteristics were compared. Results: Sources of infection, including the pancreatic duct, lungs, urinary tract, and other sites between BSIs caused by *E. coli* and *A. baumannii,* showed statistical significance. Risk factors, including multiple infections, ICU acquired infections, time to blood culture positivity, antibiotic treatment, and antifungal treatment, between the two groups showed statistical differences. Treatment outcomes, including empiric antibiotic treatment, total hospital stay, in-hospital mortality, attributable 28-day mortality, and attributable in-hospital mortality, between the two groups were statistically different. Resistance of *E. coli* to carbapenems was low and resistance of *A. baumannii* to prostacyclin was low. ICU acquired infections, time to blood culture positivity, acute kidney damage, renal replacement therapy, mechanical ventilation, and SOFA scores were statistically different between death and survival groups. Conclusion: BSIs caused by *E. coli* and *A. baumanii* showed significant differences in sources of infection, 28 days of mortality, time to blood culture positivity, and antibiotic resistance.

**Keywords:** Bloodstream infections, Escherichia coli, Acinetobacter baumannii

## Introduction

In recent years, Enterobacteriaceae infections have increased, becoming the main source of nosocomial infections. *Escherichia coli* (*E. coli*) and *Klebsiella pneumoniae* are the most common pathogens of nosocomial infections [1, 2]. *E. coli* belongs to the normal flora in human body. When the body defense system weakens, *E. coli* shows pathogenicity and becomes a conditional pathogen. The Chinese bacterial resistance surveillance network [3] revealed that *E. coli* became the most important bacterium causing bloodstream infections (BSI) during the period of 2011-2012. *E. coli* has become a representative strain producing extended spectrum beta-lactamases (ESBLs) [4]. *E. coli* has a high resistance and can induce severe BSI with poor prognosis, presenting a great challenge to clinical diagnosis and treatment. *Acinetobacter baumannii* (*A. baumannii*), a conditional pathogen, is a gram-negative non-fermentative bacterium that widely distributes in the natural environment. *A. baumannii* can survive for a long time in a hospital environment. It invades the skin, respiratory tract, digestive tract, and urogenital tract, leading to a variety of serious infections in patients with severely impaired immune function, such as patients with ventilator-associated pneumonia, BSIs, urinary tract infections, and meningitis [5-8]. *A. baumannii* also has a strong drug resistance. Multi-drug resistance, pan-resistant, and all-resistant *A. baumannii* are prevalent in the world [8]. *A. baumannii*-BSI often occurs in critically ill patients, seriously threatening lives [9].

Incidence of BSI is increasing all over the world, accompanied by higher fatality rates and mortality rates [10]. Each year, 80 to 257 infections in 100 thousand people are reported [11-18]. In Europe, incidence and mortality of BSIs are

estimated at 1,200,000 and 157,000, respectively [10]. Incidence and mortality of hospital acquired BSIs are 240,000 and 29,000, respectively [10]. Therefore, early and effective targeted treatment for BSI is needed. According to clinical data, BSIs caused by *E. coli* and *A. baumannii* are on the rise [19]. However, whether there are differences in clinical characteristics between the two has not been reported.

In this study, risk factors of BSI, clinical features of 28-day mortality, and differences in clinical characteristics between BSIs caused by *E. coli* and *A. baumannii* were analyzed. Present findings may provide clues and direction for clinical treatment of BSIs.

## Materials and methods

### *Patients*

This was a retrospective study. A total of 118 patients, including 87 males and 31 females, with an average age of 52 ± 10.66, ranging from 25 to 78 years old, were collected, between January 2007 and January 2014, from the People's Hospital of Xinjiang Uygur Autonomous Region in Xinjiang China. There were 63 cases of BSIs caused by *E. coli* and 55 cases of BSIs caused by *A. baumannii*. Inclusion criteria were as follows: 1) At more than 48 hours after admission, patients had body temperatures of more than 38°C or less than 36°C; 2) Patients had clinical manifestations, such as chills or hypotension; and 3) *E. coli* or *A. baumannii* was isolated from blood cultures of the patients more than one time. Exclusion criteria were as follows: 1) Patients had no obvious fever or the fever could be explained by other reasons; and 2) Only one time was the blood culture positive, with the following blood cultures negative or found with other pathogens. Informed consent was obtained from every patient. The study was approved by the Ethics Review Board of the People's Hospital of Xinjiang Uygur Autonomous Region.

### *Data collection*

Clinical features of patients were analyzed and compared, including demography (gender, age), comorbidities (diabetes, chronic renal failure, biliary tract disease, congestive heart failure, and chronic obstructive pulmonary disease), results of time to blood culture positivity, suspected infections, previous medical exposure (over 48 hours of antibiotic treatment, especially antifungal treatment over the past 30 days), and infections in other sites besides the bloodstream. General conditions of the patients with BSI were evaluated, including acute renal impairment, septic shock, mechanical ventilation, renal replacement therapy, and removal of suspicious foci of infections. Severity of disease was assessed using APACHE II scores and SOFA scores [20, 21]. Original and attributable mortality, 28 day mortality, total hospital stay, and hospital stay after BSI were assessed.

### *Definitions*

Intensive care unit (ICU) acquired BSI was defined as the first positive blood culture determined more than 2 days after admission to ICU. Possible sources of infection were identified by microbiological results and analysis of 2 physicians. Appropriate empiric antimicrobial therapy refers to the management of the *in vitro* antimicrobial activity of the strain within 24 hours after onset of BSI [16]. Septic shock was defined as sepsis associated with organ dysfunction and persistent hypotension after volume resuscitation. Established criteria were used to define acute kidney injury [21, 22]. Original mortality was defined as death after the first positive blood culture of *E. coli* or *A. baumannii*. Attributable mortality was defined by clinical evidence of active infections and positive cultures or death due to organ failure during development or exacerbation of infections [23].

### *Antimicrobial resistance*

Isolation, identification, and sensitivity monitoring of microbiology were performed using the Vitek 2 automatic system (Meriere, France). Double-disc confirmation testing was used to detect $ESBL^+$. *E. coli* ATCC 25922 and *A. baumannii* ATCC 19606 were used as controls. K-B agar diffusion method was used to test resistance of ampicillin, ampicillin/sulbactam, piperacillin/tazobactam, cefoperazone/sulbactam, cefazolin, aztreonam, ceftazidime, ceftazidime, meropenem, imipenem, tigecycline, gentamicin, ciprofloxacin Star, levofloxacin (LAX), and compound sulfamethoxazole. The minimum inhibitory concentration was classified according to cut-off points established by the clinical and laboratory bodies (CLSI2014) [24].

**Table 1.** Sources of infection in the two groups (n, %)

| Source of bacteremia | *E. coli* (n=63) | *A. baumannii* (n=55) | *P* |
|---|---|---|---|
| Pancreaticobiliary tract | 1 (19.0) | 17 (30.9) | 0.042* |
| Pneumonia | 8 (12.7) | 20 (36.4) | <0.001** |
| Urinary tract | 13 (20.6) | 4 (7.3) | 0.001** |
| Intra-abdomina | 7 (11.1) | 12 (21.9) | 0.008** |
| Catheter-related infection | 2 (3.2) | 8 (14.5) | <0.001** |
| Surgical wound | 3 (4.8) | 3 (5.5) | 0.871 |
| Gastrointestinal tract | 3 (4.8) | 2 (3.6) | 0.811 |
| Liver abscess | 3 (4.8) | 2 (3.6) | 0.811 |
| Skin or soft tissue | 1 (1.6) | 3 (5.5) | 0.015* |
| Unknown | 5 (7.9) | 3 (5.5) | 0.445 |

Note: * and **, P<0.05 and <0.01, respectively.

*Statistical analysis*

Data was analyzed using IBM SPSS 17.0 software. Quantitative variables of normal and abnormal distribution are expressed as mean ± SD and median, respectively. Differences among groups were examined with Chi-squared test. If the continuous variables were normally distributed, average values of the two different groups were evaluated by the two-sample unpaired t test. For continuous variables that were not normally distributed, the nonparametric Mann-Whitney U-test was employed. Categorical variables were compared using the $x^2$ test or Fisher's test, as appropriate. Odds ratios (ORs) and 95% confidence intervals (CIs) were also calculated. Regarding multivariate analysis, hospital events at the onset of BSI were scored using APACHE II and SOFA scores. *P* values less than 0.05 indicate statistical significance.

## Results

*Comparison of infection sources between BSI patients caused by E. coli and A. baumannii*

As shown in **Table 1**, sources of BSI caused by *E. coli* usually came from the urinary tract (20.6%). However, those caused by *A. baumannii* mainly came from pneumonia (36.4%), followed by pancreaticobiliary duct (30.9%) and intra-abdomina (21.9%). Infection sources between the two groups were compared. Results showed statistical differences between blood flow infections caused by *E. coli* and *A. baumannii* in the pancreaticobiliary tract (19.0% and 30.9%, P<0.05), pneumonia (12.7% and 36.4%, P<0.01), urinary tract (20.6% and 7.3%, P<0.01), intra-abdomina (11.1% and 21.9%, P<0.01), catheter-related infection sites (3.2% and 14.5%, P<0.01), and skin or soft tissue (1.6% and 5.5%, P<0.05). Surgical wounds, gastrointestinal tract, liver abscess, and unknown sites showed no significant statistically significant differences between the two groups.

*Comparison of risk factors associated with progression of BSIs caused by E. coli and A. baumannii*

As shown in **Table 2**, there were statistically significant differences in multiple-infections, ICU acquired infections, rates of diabetes, time to blood culture positivity, antibiotics, and antifungal therapy between BSIs caused by *E. coli* and *A. baumannii.* In general, rates of multiple-infections in BSIs caused by *E. coli* and *A. baumannii* were 6.3% and 18.2%, respectively. Rates of ICU acquired infections caused by *E. coli* and *A. baumannii* were 15.9% and 42%, respectively. There were statistically significant differences in the two factors between the two groups. For complications, rates of diabetes in BSIs caused by *E. coli* and *A. baumannii* were 43% and 9.0%, respectively. Differences were statistically significant (P<0.01). Moreover, chronic renal failure, biliary tract disease, congestive heart failure, chronic obstruction, and pulmonary disease between the two groups showed no significant differences. In addition, the time to blood culture positivity, antibiotics, and antifungal therapy between the two groups showed statistical significance.

*Comparison of antibiotic treatment between BSIs caused by E. coli and A. baumannii*

As shown in **Table 3**, rates of appropriate empirical antimicrobial therapy of *E. coli*-BSI and *A. baumanii*-BSI patients were 54% and 15%, respectively. Differences were statistically significant (*P*<0.01). This led to longer hospital stays for *A. baumanii*-BSI patients (50%) than *E. coli*-BSI patients (24%). In terms of mortality, original in-hospital mortality, attributable 28-day mortality, and attributable in-hospital mortality rates of *A. baumanii*-BSI patients were 23%, 18%, and 23%, respectively, higher than those of *E. coli*-BSI patients (12%, 10%, and 15%). Differences were statistically significant.

**Table 2.** Risk factors associated with progression of BSIs caused by *E. coli* or *A. baumannii*

| Variable | Total (n=118) | *A. baumannii* (n=55) | *E. coli* (n=63) | Univariate analysis | | Multivariate analysis | |
|---|---|---|---|---|---|---|---|
| | n (%) | n (%) | n (%) | OR (95% CI) | *P*-Value | OR (95% CI) | *P*-Value |
| General variables | | | | | | | |
| Male sex | 76 (64.4) | 41 (65) | 35 (63.6) | 1.11 (0.48-2.52) | 0.801 | | |
| Age, mean ± SD | 55.98±16.24 | 51.58±15.99 | 58.19±16.08 | NA | 0.052 | | |
| Multiple-infection | 101 (85.6) | 10 (18.2) | 4 (6.3) | NA | 0.005** | | |
| ICU acquired infection | 26 (22) | 23 (42) | 10 (15.9) | 4.25 (1.71-10.78) | 0.001** | 5.83 (2.1-17.26) | 0.002** |
| Comorbidities | | | | | | | |
| Diabetes mellitus | 30 (25.4) | 5 (9.0) | 27 (43) | 0.21 (0.11-0.76) | 0.008** | | |
| Chronic renal failure | 8 (6.8) | 5 (9.0) | 4 (6.3) | 1.51 (0.31-6.82) | 0.688 | | |
| Biliary tract disease | 36 (30) | 15 (27.2) | 2 (3.2) | 0.78 (0.31-1.88) | 0.615 | | |
| Congestive heart failure | 9 (7.6) | 6 (11) | 4 (6.3) | 2.11 (0.49-8.45) | 0.283 | | |
| Chronic obstructive | 3 (2.5) | 2 (3.6) | 2 (3.2) | 1.22 (0.11-14.1) | 0.99 | | |
| Pulmonary disease | | | | | | | |
| Time to blood culture positivity | | | | | | | |
| Less than 7 days | 77 (65.2) | 26 (47.2) | 34 (54) | 8.42 (2.3-29.9) | 0.001** | | |
| More than 7 days | 12 (10.1) | 17 (31) | 2 (3.2) | 17.16 (3.5-84.2) | 0.001** | | |
| Probable source of infection | | | | | | | |
| Lung | 16 (13.6) | 8 (14.5) | 9 (14.3) | 1.15 (0.4-3.7) | 0.775 | | |
| Urinary | 1 (0.8) | 0 (0) | 1 (1.6) | NA | 1 | | |
| Catheter | 1 (0.8) | 8 (14.5) | 4 (6.3) | 2.71 (0.7-10.2) | 0.14 | | |
| Hospital events priorto onset of BSI | | | | | | | |
| Exposure to antimicrobial Therapy | 76 (64.4) | 50 (90.9) | 32 (51) | 8.40 (2.4-29.9) | 0.051 | 4.03 (1.0-16.4) | 0.04* |
| Antifungal drugs | 24 (20.3) | 20 (36.4) | 9 (14.3) | 4.19 (1.7-10.68) | 0.001** | 5.75 (2.0-17.22) | 0.002** |
| Events on the onset of BSI | | | | | | | |
| APACHE II score, median (IQR) | 3 (1-6) | 4 (1-7) | 3 (1-5) | NA | 0.085 | | |
| SOFA score, median (IQR) | 5 (2-8) | 3 (1-6) | 4 (1-7) | NA | 0.091 | | |

Note: ICU, Intensive care unit; BSI, Blood stream infection; APACHE II score, Acute physiology and chronic health evaluation II score; SOFA score, Sequential Organ Failure Assessment score. * and **, P<0.05 and <0.01, respectively.

**Table 3.** Comparison of antimicrobial therapies and treatment outcomes between *E. coli*-BSI and *A. baumannii*-BSI patients

| Treatment and outcomes | Total (n=118) | *A. baumannii* (n=55) | *E. coli* (n=63) | *P* value |
|---|---|---|---|---|
| Appropriate empirical antimicrobial therapy | 80 (67.8) | 15 (27.3) | 54 (85.7) | <0.001** |
| Appropriate empirical antimicrobial therapy | 80 (67.8) | 15 (27.3) | 54 (85.7) | <0.001** |
| LOS after BSI onset, Days, median (IQR) | 18.5 (9-31) | 24 (9.5-51) | 15 (8.5-28) | 0.066 |
| Total LOS, days, median (IQR) | 31.5 (19.75-62.75) | 50 (28-83) | 24 (16.5-51) | 0.001** |
| Mortality rate | | | | |
| Crude 28-day mortality n (%) | 27 (22.9) | 18 (32.8) | 12 (19) | 0.087 |
| Crude in-hospital mortality n (%) | 31 (26.3) | 23 (41.8) | 12 (19) | 0.013** |
| Attributable 28-day mortality n (%) | 25 (21.2) | 18 (32.8) | 10 (15.9) | 0.04* |
| Attributable in-hospital mortality n (%) | 29 (24.6) | 23 (41.2) | 15 (23.8) | 0.005** |

Note: BSI, Blood stream infection; LOS, Total length of hospital stay. * and **, P<0.05 and <0.01, respectively.

*Comparison of antimicrobial resistance between BSIs caused by E. coli and A. baumannii*

As shown in **Table 4**, the two groups had high resistance to narrow spectrum antibiotics. For *E. coli*-BSIs, the resistance rates of $ESBL^-$, $ESBL^+$ ampicillin were 66.7% and 100%, respectively. For *A. baumanii*-BSIs, the resistance rates of MDRAB and XDRAB ampicillin were 35% and 60%, respectively. Drug resistance showed a downward trend in both groups with the upgrading of antibiotics, including ceftriaxone, cephalosporin, and cefepime, especially for *E. coli*-BSIs. For broad-spectrum antibiotics,

**Table 4.** Antimicrobial resistance comparison between *E. coli* and *A. baumannii* isolates (n, %)

| Antibacterial drugs | *E. coli* (n=63) | | *A. baumannii* (n=55) | |
|---|---|---|---|---|
| | ESBL$^-$ (n=27) | ESBL$^+$ (n=36) | MDRAB (n=20) | XDRAB (n=35) |
| Ampicillin | 18 (66.7) | 36 (100) | 7 (35) | 21 (60) |
| Ampicillin/sulbactam | 10 (37) | 18 (50) | 6 (30) | 19 (54.2) |
| Piperacillin/tazobactam | 6 (22.2) | 13 (36.1) | 5 (25) | 25 (71.4) |
| Cefoperazone/sulbactam | 4 (14.8) | 16 (44.4) | 6 (30) | 18 (51.4) |
| Cefazolin | 8 (29.6) | 36 (100) | 20 (100) | 35 (100) |
| Aztreonam | 4 (14.8) | 24 (66.7) | 3 (15) | 30 (85.7) |
| Ceftriaxone | 4 (14.8) | 33 (91.7 | 4 (20) | 28 (80) |
| Ceftazidime | 4 (14.8) | 16 (44.4) | 6 (30) | 24 (68.6) |
| Cefepime | 2 (7.4) | 12 (33.3) | 6 (30) | 25 (71.4) |
| Meropenem | 2 (7.4) | 1 (0.3) | 6 (30) | 24 (68.6) |
| Imipenem | 1 (3.7) | 1 (0.3) | 6 (30) | 25 (71.4) |
| Tigecycline | - | - | 2 (10) | 12 (34.3) |
| Gentamicin | 8 (29.6) | 19 (52.8) | 6 (30) | 23 (65.7) |
| Ciprofloxacin | 14 (51.9) | 27 (75) | 5 (25) | 25 (71.4) |
| Levofloxacin | 12 (44.4) | 27 (75) | 4 (20) | 10 (28.6) |
| Sulfamethoxazole | 10 (37) | 24 (66.7) | 5 (25) | 24 (68.6) |

Note: ESBLs, Extended spectrum β-lactamases; MDR, Multidrug resistant; XDR, Extensively drug resistant.

resistance of *E. coli* to meropenem and imipenem was low.

Resistance rates of ESBL$^-$, ESBL$^+$ meropenem for *E. coli*-BSIs were 7.4% and 0.3%, respectively. Those of ESBL$^-$, ESBL$^+$ imipenem were 3.7% and 0.3%, respectively. Resistance rates of *A. baumanii* to meropenem and imipenem did not decrease obviously. Resistance rates of MDRAB, XDRAB meropenem for *A. baumanii*-BSI were 30% and 68.6%, respectively. Those of MDRAB, XDRAB imipenem were 30% and 71.4%, respectively. Resistance of *E. coli* to meropenem and imipenem was very low, but that of *A. baumanii* was still high. Resistance of *A. baumanii* to tigecycline showed a downward trend, compared to other drugs shown in the form. Resistance rates of MDRAB and MDRAB tigecycline were 10% and 34.3%, respectively. Drug resistance showed a downward trend in both groups with the upgrading of antibiotics. Resistance of *E. coli* to carbapenem and *baumanii* to tigecycline was low.

*Clinical and microbiological features associated with original 28-day mortality*

As shown in **Table 5**, rates of ICU acquired infections in death and survivor groups were 36% and 18.1%, respectively. Differences were statistically significant ($P<0.05$). The time to blood culture positivity showed statistical significance between the two groups both for less than 7 days and greater than 7 days. Additionally, events on the onset of BSI, including acute renal damage, use of renal replacement therapy, and mechanical ventilation, as well as SOFA scores between the two groups, showed statisticalsignificance.However,comorbidities, the probable source of infection, and hospital events prior to onset of BSI showed no statistical significance.

## Discussion

BSI is a serious infectious disease. It can cause multiple organ dysfunction syndrome, which has a high mortality rate [25]. Although new broad-spectrum antibiotics have been used in clinic, morbidity and mortality rates of BSI have not been controlled [26]. ICU patients have the highest incidence of BSI because of their primary diseases, long stay in hospital, long-term use of antimicrobial agents, and various invasive procedures [27]. This retrospective study revealed differences in infection source, risk factors, drug resistance, mortality, and related clinical features between BSIs caused by *E. coli* and *A. baumannii.*

Present results showed that sources of BSIs caused by *E. coli* mainly came from the urinary tract, while those caused by *A. baumannii* mainly came from pulmonary infections, in accord with a previous study [28]. According to the characteristics of sources of infection, clinicians can choose the appropriate empirical anti-infection treatment for the corresponding BSIs, then analyze the risk factors for BSIs. In BSIs caused by *A. baumannii*, multiple infections and ICU acquired infections accounted for a higher proportion, which further increased the use of antibiotics, antifungal drugs, and catheters, compared with those of *E. coli.* When the mucosal barrier is destroyed and immunity decreases, *A. baumannii* becomes a pathogenic bacterium and increases the chances of BSI [29]. When the time to blood-culture positivity

**Table 5.** Identification of clinical and microbiological characteristics associated with crude 28-day mortality

| Variable | Total (n=118) | Death (n=25) | Survivors (n=88) | Univariate analysis | | Multivariate analysis | |
|---|---|---|---|---|---|---|---|
| | n (%) | n (%) | n (%) | OR (95% CI) | *P*-Value | OR (95% CI) | *P*-Value |
| General variables | | | | | | | |
| Male sex | 76 (64.4) | 19 (76) | 54 (61.4) | 2.1 (0.79-5.8) | 0.143 | | |
| Age, mean ± SD | 55.98±16.24 | 51.55±16.01 | 56.23±16.35 | NA | 0.76 | | |
| multiple-infectionl | 101 (85.6) | 3 (12) | 8 (9.1) | 1.28 (0.3-5.27) | 0.710 | | |
| ICU acquired infection | 26 (22) | 9 (36) | 16 (18.1) | 2.81 (1.11-7.28) | 0.029* | | |
| Comorbidities | | | | | | | |
| Diabetes mellitus | 30 (25.4) | 4 (16) | 30 (34.1) | 0.41 (0.11-1.38) | 0.14 | | |
| Chronic renal failure | 8 (6.8) | 1 (4) | 7 (8) | 0.46 (0.4-3.85) | 0.681 | | |
| Biliary tract disease | 36 (30) | 8 (32) | 27 (31) | 0.99 (0.38-2.57) | 1 | | |
| Congestive heart failure | 9 (7.6) | 5 (20) | 4 (4.5) | 4.9 (1.18-19.98) | 0.26 | | |
| Chronic obstructive Pulmonary disease | 3 (2.5) | 0 (0) | 3 (3.4) | NA | 1 | | |
| Time to blood culture positivity | | | | | | | |
| Less than 7 days | 77 (65.2) | 12 (48) | 49 (55.7) | 8.41 (2.2-29.6) | 0.001** | | |
| More than 7 days | 12 (10.1) | 8 (32) | 26 (29.5) | 17.2 (3.4-83.5) | 0.001** | | |
| Probable source of infection | | | | | | | |
| Lung | 16 (13.6) | 9 (36) | 7 (8) | 6.11 (1.9-18.5) | 0.002** | 4.23 (1.0-17.3) | 0.045 |
| Urinary | 1 (0.8) | 0 (0) | 1 (1) | NA | 1 | | |
| Catheter | 1 (0.8) | 2 (8) | 8 (9.1) | 0.82 (0.18-4.1) | 1 | | |
| Hospital events prior to onset of BSI | | | | | | | |
| Exposure to antimicrobial Therapy | 76 (64.4) | 19 (76) | 54 (61.4) | 2.1 (0.75-5.7) | 0.144 | | |
| Antifungal drugs | 24 (20.3) | 9 (36) | 13 (14.8) | 5.25 (2.3-11.23) | 0.001** | | |
| Events on the onset of BSI | | | | | | | |
| Acute kidney injury | 17 (14.4) | 12 (48) | 4 (4.5) | 18.01 (5.2-63.9) | 0.001** | | |
| Use of renal replacement therapy | 8 (6.7) | 5 (20) | 3 (3.4) | 6.75 (1.6-30.6) | 0.001** | | |
| Use of mechanical ventilation | 25 (21.2) | 14 (56) | 12 (10.2) | 11.96 (4.1-33.8) | 0.001** | | |
| Removal of the infectious source | 34 (28.8) | 6 (24) | 27 (30.1) | 0.67 (0.2-1.88) | 0.453 | | |
| APACHE II score, median (IQR) | 3 (1-6) | 4 (1-7) | 3 (1-5) | NA | 0.085 | | |
| SOFA score, median (IQR) | 5 (2-8) | 8 (3-12.5) | 2 (1-4) | NA | 0.001** | 1.40 (1.2-1.6) | 0.001** |

Note: ICU, Intensive care unit; BSI, Blood stream infection; APACHE II score, Acute physiology and chronic health evaluation II score; SOFA score, Sequential Organ Failure Assessment score. * and **, P<0.05 and <0.01, respectively.

was within one week, *E. coli* was superior to *A. baumannii*. When it was more than one week, *A. baumannii* was superior. This result provides clues and direction for clinical antibiotic selection.

This study showed that empiric antibiotic therapy was more effective against *A. baumannii* than *E. coli.* Drug resistance showed a downward trend in both groups with the upgrading of antibiotics. Resistance of *E. coli* to carbapenems was very low and the resistance of *A. baumannii* to prostacyclin showed a downward trend. The generation of drug resistance may be related to the destruction of the balance of normal bacterial flora caused by the use of a variety of antimicrobial agents [30]. Repeated use of broad-spectrum antibiotics can increase the selective pressure of antibiotics, resulting in the emergence of drug-resistant strains [30]. *A. baumannii* resistance generally increases. According to the 2012 CHINET Chinese fine resistance monitoring network data [31], resistance rates of *A. baumannii* to imipenem and meropenem were greater than 60%, Cefoperazone/sulbactam and minocycline were 39.1% and 27.3%, other test drugs were more than 50%, and polymyxin B and polymyxin E were more than 90%. At present, polymyxin has no listed products in China and it has nephrotoxicity and neurotoxicity. Therefore, tigecycline is considered to be one of the few drugs for the treating carbapenem resistant *A. baumannii* [32]. It has good effects on pulmonary infections of carbapenem resistant *A. baumannii* and has less adverse drug reactions [33]. In addition, the effects of cyclosporine in the treatment of BSI are satisfactory [34]. Poulakou

[35] found that the clinical success rate of cyclosporine for BSI was 80% and the bacterial clearance rate was 80%. To improve the blood concentration of tegafur, combination therapy can be attempted [36]. In 2012, the expert consensus on the diagnosis, treatment, and prevention of *A. baumannii* infections [37] pointed out that multidrug resistant or extensively drug-resistant *A. baumannii* infections could be treated by Cefoperazone/sulbactam combined with tigecycline. Therefore, for treatment of multidrug resistant or extensively drug-resistant *A. baumannii* infections, prostacyclin or combination drugs may be used to reduce drug resistance, increase effectiveness, and improve the cure rate and clinical prognosis.

This study showed that rates of ICU acquired infections, the time to blood culture positivity, acute renal damage, use of renal replacement therapy and mechanical ventilation, and SOFA scores in death and survivor groups had statistical significance. The above indicators were closely related to the original 28-day mortality and similar with risk factors associated with progression of BSIs. Results indicate that these factors not only complement each other to accelerate the occurrence of BSIs but are also closely related to prognosis. Present results imply that ICU patients that are critically ill, use invasive ventilators for a long time, retain deep vein catheterization, and use large number of broad-spectrum antibiotics, may experience microecological imbalance and an increase of pathogenic bacteria infections [38].

## Conclusion

The present study found that BSIs caused by *E. coli* mainly came from the urinary tract, while those cause by *A. baumannii* mainly came from the lungs and the pancreas bile duct. Therefore, the choice of antibiotics can be preliminarily judged by the site of infection. ICU acquired infection, antifungal therapy, acute kidney injury, renal replacement therapy, mechanical ventilation, and other risk factors are closely related to the progression of accelerated BSIs and the 28-day mortality rate. This makes it worthy to clinical attention. The time to blood culture positivity for less than one week was more common in *E. coli*, while more than one week was more common in *A. baumannii*, which has certain guiding function to clinical antibiotic treatment. *E. coli* has a low resistance to carbapenem. The use of tigecycline or combination therapy for multidrug resistant or extensively drug-resistant *A. baumannii* infections can reduce resistance, increase efficiency, and improve cure rates and clinical prognosis.

## Disclosure of conflict of interest

None.

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