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Propensity score matched analysis comparing the clinical outcome of *Klebsiella pneumoniae* and *Escherichia coli* causing community-onset monomicrobial bacteremia

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Abstract

Bacteremia is a life-threatening condition that is associated with substantial healthcare costs. *Escherichia coli* and *Klebsiella pneumoniae* are the leading causes of community-onset gram-negative bacteremia. However, a comprehensive comparison between these pathogens involved in bacteremia episodes has yet to be reported.

In this retrospective cohort study, adults with community-onset monomicrobial bacteremia caused by *E coli* or *K pneumoniae* were recruited in the emergency department of a medical center during a 6-year period, and the clinical variables were collected retrospectively from medical records. The complicated abscess occurrence was determined through imaging studies, according to the opinion of an infectious disease consultant. According to the independent predictors of 28-day mortality identified through multivariate regression analyses, patients in the *E coli* group were propensity score matched (PSM) in a 1:1 ratio to those in the *K pneumoniae* group.

A total of 274 and 823 adults with *K pneumoniae* and *E coli* bacteremia were included in the present study. The *K pneumoniae* group had more patients with fatal comorbidities (McCabe classification), critical illness (Pitt bacteremia score ≥ 4) at bacteremia onset, and initial syndrome (e.g., severe sepsis and septic shock) as well as a higher crude mortality rate than did the *E coli* group. After appropriate matching, no significant differences were observed in the critical illness at bacteremia onset, initial syndrome, major comorbidities, and comorbidity severity of the 2 groups (*E coli*, n = 242; *K pneumoniae*, n = 242). Furthermore, despite similar 14- and 28-day crude mortality rates between the 2 PSM groups, more frequent abscess occurrences and a longer length of hospitalization were observed in the *K pneumoniae* group than in the *E coli* group.

Conclusively, numerous clinical features at initial presentations varied between the *E coli* and *K pneumoniae* groups. Despite conducting a PSM analysis to control the differences in the baseline characteristics, a longer length of hospitalization and more frequent abscess occurrences were observed in the *K pneumoniae* group than in the *E coli* group.

Abbreviations: CLSI = Clinical and Laboratory Standards Institute, CT = computed tomography, ED = emergency department, ESBL = extended-spectrum-β-lactamase, ICU = intensive care unit, PSM = propensity score matched.

Keywords: bacteremia, community-onset, *Escherichia coli*, *Klebsiella pneumoniae*

1. Introduction

Bacteremia is a life-threatening condition because of substantial healthcare costs and in-hospital mortality rates of up to 30%.[1] *Escherichia coli* and *Klebsiella pneumoniae* are the leading causes of community-onset gram-negative bacteremia[2] and are the major causes of liver abscess, intra-abdominal infection, and complicated soft-tissue infection.[3–6] Several studies have reported that a new *K pneumoniae*-related invasive pyogenic liver abscess syndrome, occasionally accompanied by

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septic metastases with abscess occurrences, is a substantial public health problem worldwide.[3–5] In addition, several reports have mentioned the occurrence of various types of abscesses complicated by *E coli* bloodstream infections.[7–9] However, the clinical effect of these abscess occurrences complicated with *E coli* or *K pneumoniae* on patient outcomes has yet to be reported in the literature. Furthermore, although *E coli* and *K pneumoniae* bacteremia have been described individually in several studies,[4,6,10,11] a comprehensive comparison of these 2 pathogens involved in bacteremic episodes has not been conducted. Therefore, we compared the clinical characteristics, initial presentations, frequencies of abscess occurrences, and clinical outcomes of the adults with community-onset bacteremia caused by *E coli* and those by *K pneumoniae* in the present study.

2. Materials and methods

2.1. Study design, setting, and population

This was a retrospective cohort study conducted during a 6-year period, between January 2008 and December 2013, at the emergency department (ED) of a medical center in southern Taiwan. The study was approved by the hospital's institutional review board (ER-100–182), and the requirement for informed consent was waived. This analysis was reported using the format recommended by STROBE (Strengthening the Reporting of Observational Studies in Epidemiology). Partial clinical information in this cohort has been published.[12–15]

During the study period, the blood cultures of patients who underwent blood culture sampling at the ED were screened for bacterial growth by using a computer database. Adults with monomicrobial *E coli* or *K pneumoniae* bacteremia at the ED were investigated. Their medical records were reviewed for clinical characteristics, source of transfer, vital signs, severity of bacteremia (e.g., Pittsburgh bacteremia score), comorbidities, laboratory data, and initial syndrome (e.g., septic shock and severe sepsis) immediately following each patient's visit to the ED. At that time, records of prior hospitalization, antimicrobial therapies and their duration, microbiological results, imaging studies, the source of bacteremia, further hospitalization, the length of the hospital stay, and clinical outcomes were obtained from the charts.

The study excluded those having incomplete chart records, hospital-onset bacteremia, or in whom bacteremia was diagnosed prior to the ED visits. For eligible patients, medical records were reviewed for the above clinical information by 2 of the authors, and if any discrepancy was found, the medical records were inspected by the 2 authors together. For patient having more than 1 focus of infections, they were all assigned to the final diagnoses of infectious focus. In cases with multiple bacteremic episodes, only the first episode was included for each patient.

The primary outcome was overall mortality during the 28 days following ED arrival (e.g., bacteremia onset). If a patient was discharged within 28 days after ED arrival and was not followed-up at our hospital, he/she was excluded.

2.2. Microbiological methods

E coli and *K pneumoniae* was then identified using biochemical tests and confirmed with a Vitek system (BioMerieux, Lyon, France) and a Gram-Negative Identification card. These isolates in the study period were prospectively collected. Antimicrobial susceptibility was determined by the disk diffusion method, and the interpretation followed the breakpoints recommended by the Clinical and Laboratory Standards Institute (CLSI) in 2016.[16] The tested drugs included ampicillin/sulbactam, piperacillin/tazobactam, cefazolin, cefuroxime, cefotaxime, ceftazidime, cefepime, ertapenem, imipenem, and levofloxacin. If patient empirically treated by other agents, the susceptibility of the indicated agent was measured. Extended-spectrum β-lactamase (ESBL) production was detected by the phenotypic confirmatory test with the cephalosporin–clavulanate combination disks recommended by the previous CLSI guidelines.[17]

2.3. Definitions

As previous definitions,[14,18] community-onset bacteremia indicates that the place of onset of the bacteremic episode is the community and includes long-term healthcare facility-acquired and community-acquired bacteremia. Bacteremia sources were determined clinically on the basis of the presence of an active infection site comorbid with bacteremia or the isolation of a microorganism from other clinical specimens before or on the same date as that of bacteremic onset. If the source of bacteremia could not be assigned to a specific site, it was classified as primary bacteremia. As previously described,[14,15] antibiotic therapy was considered to be appropriate, if the route and dosage of an antimicrobial agent was administered as recommended in the Sanford Guide[19] and bacteremic pathogens were in vitro susceptible to the prescribed agent based on the contemporary breakpoints recommended by CLSI.[16] The time to appropriate antibiotic therapy was defined as the period between appropriate antimicrobial administration and bacteremia onset (ED arrival). Inappropriate empirical antibiotic therapy was defined as the time to appropriate antibiotic therapy >24 h.[14,15]

Bacteremic patients were assigned to the complicated abscess occurrence if the bacteremic episode occurred in conjunction with hepatic and/or extra-hepatic abscesses (such as lung abscesses, empyema, pyomyositis or necrotizing soft-tissue infections, epidural abscesses, renal or para-renal abscesses, mycotic aneurysm or brain abscesses) that were diagnosed by imaging studies. A complete imaging study included chest X-ray plus either abdominal sonography or abdominal computed tomography (CT) performed within 5 days after bacteremic onset, according to the opinion of an infectious disease consultant. The severity of bacteremia was graded using the Pittsburgh bacteremia score and a previously validated scoring system based on vital signs, mental status, receipt of mechanical ventilation, and recent cardiac arrest.[20] Malignancy included both hematological malignancies and solid tumors, while comorbidities were defined as previously described.[21] The prognosis of preexisting diseases was assessed using a previous delineated classification system (McCabe classification).[22] Crude mortality was used to define death by all causes.

2.4. Statistical analysis

Statistical analyses were performed using the Statistical Package for the Social Sciences for Windows (Chicago, IL, Version 20.0). Continuous variables were expressed as the means ± standard deviations and compared using Student *t* tests. Categorical variables, expressed as numbers and percentages, were compared using a Chi-squared test or Fisher exact test. All variables with *P* values <.1 by univariate analysis were considered for the stepwise, backward logistic regression model to obtain the independent risk factors of 28-day mortality. A *P* value <.05 was considered significant.

To study the adverse impact of the different causative microorganism on clinical outcome, a propensity-score-matched (PSM) analysis was performed to control for baseline variables

(e.g., bacteremia onset) linked to 28-day mortality between the *E coli* and *K pneumoniae* groups. The propensity score was calculated by the independent predictors of 28-day crude mortality assessed in a multivariable logistic regression model. Patients in the *E coli* group were matched at a ratio of 1:1 with those in the *K pneumoniae* group by individual propensity scores. The matching scheme used the closest total scores manually. As previously described,[23] the matching tolerance was a propensity score difference of 0.2.

We did not conduct formal sample size calculations, and all available data were used to maximize the power. As previous studies' suggestion, at least 8 to 10 events per variable are needed for reliable multiple logistic regression analysis.[24] As for missing values, we planned to conduct a complete case analysis if the missing values were below 5%, as such an analysis might have been feasible in that case. If the missing values were at or above 5%, we planned to perform the appropriate imputation.[25]

3. Results

3.1. Baseline demographics and clinical characteristics of the study patients

Of the 1292 adults with community-onset monomicrobial *Enterobacteriaceae* bacteremia, a total of 274 (21.2%) and 825 (63.9%) adults with *K pneumoniae* and *E coli* bacteremia, respectively, were selected according to the inclusion and exclusion criteria (Fig. 1). Table 1 shows a comparison of the baseline characteristics of the *K pneumoniae* and *E coli* groups. The number of female patients and those with comorbid hypertension and bacteremia because of urosepsis was significantly lower in the *K pneumoniae* group than in the *E coli* group. In addition, the *K pneumoniae* group had more patients with comorbid malignancies or liver cirrhosis, bacteremia caused by pneumonia or liver abscess, primary bacteremia, and low platelet count at bacteremia onset than did the *E coli* group.

A comparison of the bacteremia severity, comorbidity severity, and clinical outcomes of the 2 groups is outlined in Table 2. The number of patients with ultimately or rapidly fatal comorbidities (McCabe classification), critical illness (Pitt bacteremia score ≥ 4) at bacteremia onset, and initial syndrome (e.g., severe sepsis and septic shock) was significantly higher in the *K pneumoniae* group than in the *E coli* group. Furthermore, the *K pneumoniae* group had a shorter period of the time to appropriate antibiotic therapy and a higher crude mortality rate than did the *E coli* group. Notably, the *K pneumoniae* group had more abscess occurrences and longer lengths of total hospitalization (15.1 days vs. 11.6 days; $P < .001$) and intensive care unit (ICU) stays (2.8 days vs. 0.5 days; $P < .001$) than did the *E coli* group (Fig. 2A).

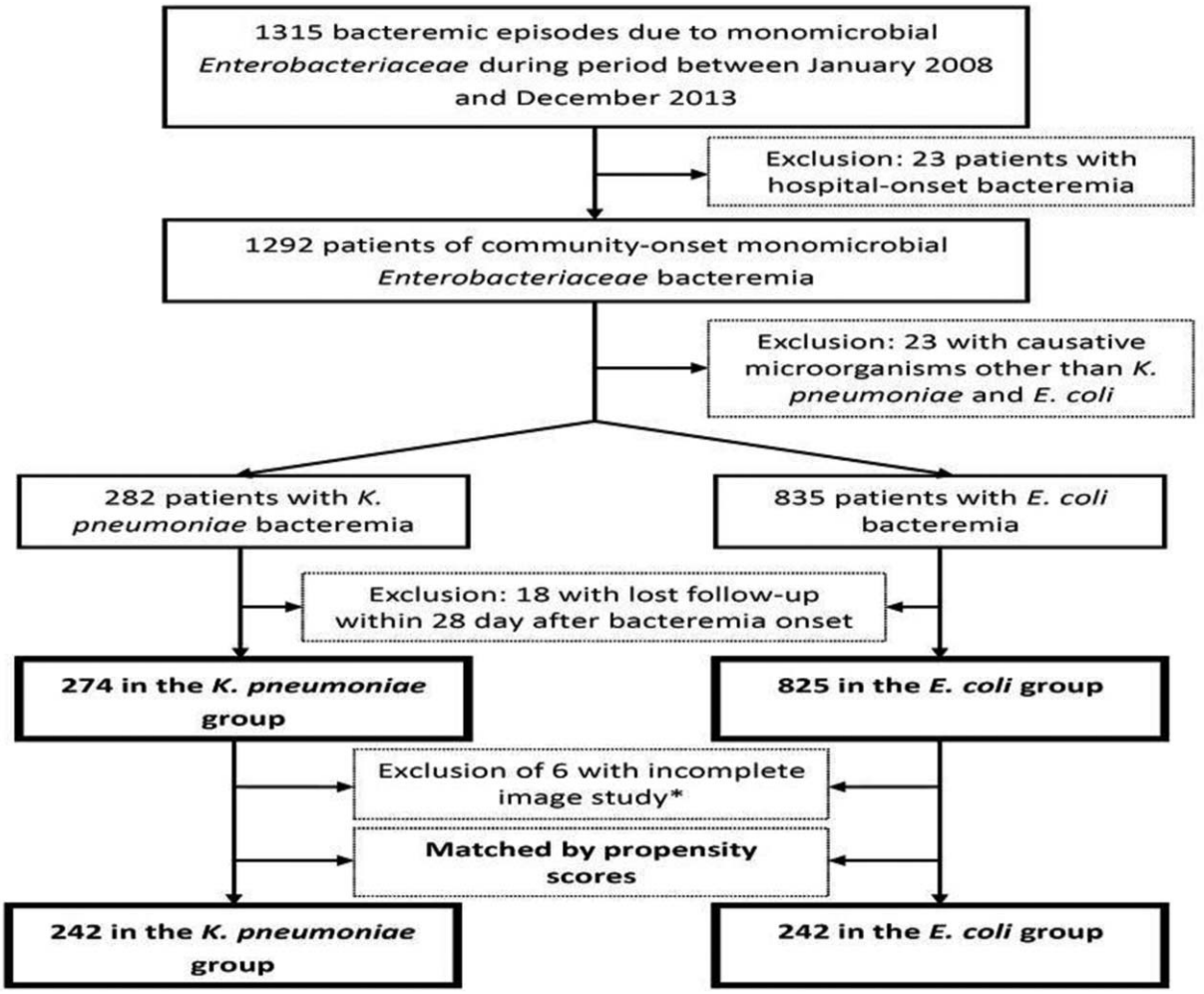

Figure 1. Patient selection flowchart. *A complete image study included chest X-ray plus either abdominal sonography or abdominal computed tomography, according to the opinion of an infectious disease consultant.

Table 1

Difference of baseline characteristics at bacteremia onset between adults with community-onset monomicrobial bacteremia caused by *Klebsiella pneumoniae* and those by *Escherichia coli*.

Characteristics	Patient number (%)		
	K pneumoniae (n = 274)	*E coli* (n = 825)	*P*
Age, year, mean ± standard deviation	66.9 ± 14.3	69.0 ± 15.7	.05
Gender, female	95 (34.7)	593 (65.3)	<.001
Nursing-home residents	14 (5.1)	39 (4.7)	.80
Major comorbidities			
Diabetes mellitus	120 (43.8)	312 (37.8)	.08
Hypertension	112 (40.9)	431 (52.2)	.001
Malignancy	87 (31.8)	208 (25.2)	.03
Neurological disorder	61 (22.3)	164 (19.9)	.40
Liver cirrhosis	53 (19.3)	102 (12.4)	.004
Chronic kidney disease	37 (13.5)	119 (14.4)	.71
Coronary artery diseases	29 (10.6)	84 (10.2)	.85
Urologic disease	22 (8.0)	46 (5.6)	.14
Major bacteremia source			
Pneumonia	62 (22.6)	38 (4.6)	<.001
Urinary tract	58 (21.2)	513 (62.2)	<.001
Liver abscess	57 (20.8)	5 (0.6)	<.001
Biliary tract	28 (10.2)	103 (12.5)	.32
Primary bacteremia	28 (10.2)	40 (4.8)	.001
Intra-abdominal	26 (9.5)	101 (12.2)	.22
Laboratory examination at the bacteremic onset			
Leukocytes > 9000/mm^3	167 (60.9)	555 (67.3)	.06
Platelets < 150,000/mm^3	132 (48.2)	300 (36.4)	.001
Serum creatinine > 1.5 mg/dL	74 (27.0)	258 (31.3)	.18
C-reactive protein > 100 mg/L	135 (49.3)	296 (35.9)	<.001

3.2. Susceptibility of the E coli and K pneumoniae isolates

Figure 3 presents the susceptibility rates of 835 *E coli* isolates and 282 *K pneumoniae* isolates to ampicillin/sulbactam, piperacillin/tazobactam, cefazolin, cefuroxime, cefotaxime, ceftazidime, cefepime, ertapenem, imipenem, and levofloxacin. The *E coli* and *K pneumoniae* isolates exhibited the lowest susceptibility (55.7% and 78.0%, respectively) to cefazolin and the highest susceptibility (100% and 100%, respectively) to imipenem.

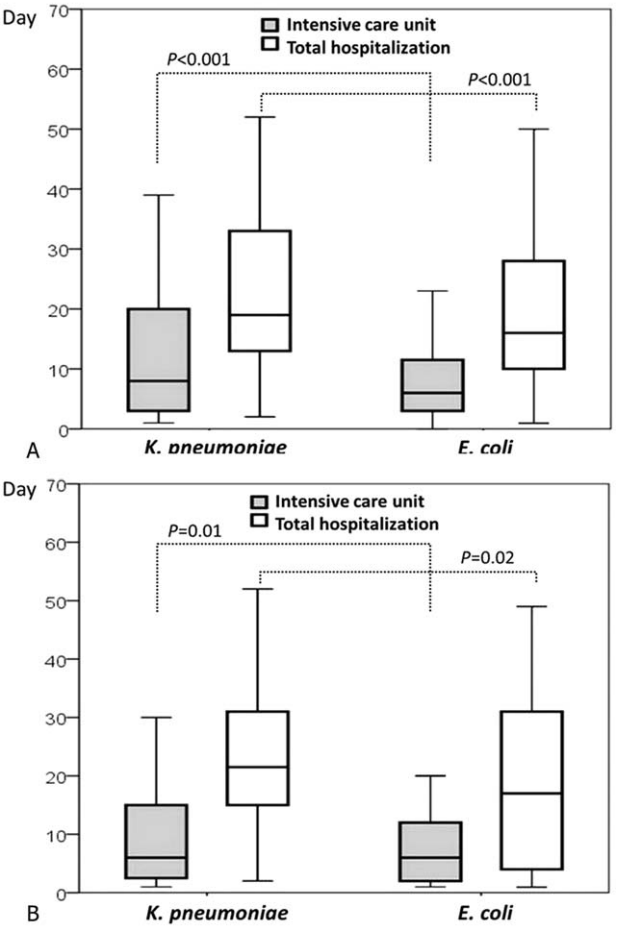

Figure 2. Comparisons of the length of stay in intensive care units and total hospitalization in the *Klebsiella pneumoniae* and *Escherichia coli* groups, among the total 1099 patients (A) and the 484 matched patients (B).

Table 2

Time to appropriate antibiotic therapy, bacteremia severity, comorbidity severity, abscess occurrence, and mortality rate in adults with community-onset monomicrobial *Klebsiella pneumoniae* or *Escherichia coli* bacteremia.

Characteristics	Patient number (%)		*P*
	K pneumoniae (n = 274)	*E coli* (n = 825)	
Time to appropriate antibiotic therapy, d, mean ± standard deviation	0.2 ± 0.8	0.4 ± 1.3	<.001
Severity-of-illness marker at the bacteremia onset			
Pitt bacteremia score ≥ 4	68 (24.8)	112 (13.6)	<.001
Initial syndrome			
Severe sepsis	139 (50.7)	321 (38.9)	.001
Septic shock	70 (25.5)	115 (13.9)	<.001
Severity of comorbidity (McCabe classification)			<.001
Rapidly or ultimately fatal	79 (28.8)	153 (18.5)	
Nonfatal	195 (71.2)	672 (81.5)	
Complicated abscess occurrence*	89/273 (32.6)	47/820 (5.7)	<.001
Crude mortality rate			
14-d	38 (13.9)	42 (5.1)	<.001
28-d	54 (19.7)	62 (7.5)	<.001
At discharge	57 (20.8)	65 (7.9)	<.001

* Six patients having an incomplete image study were excluded.

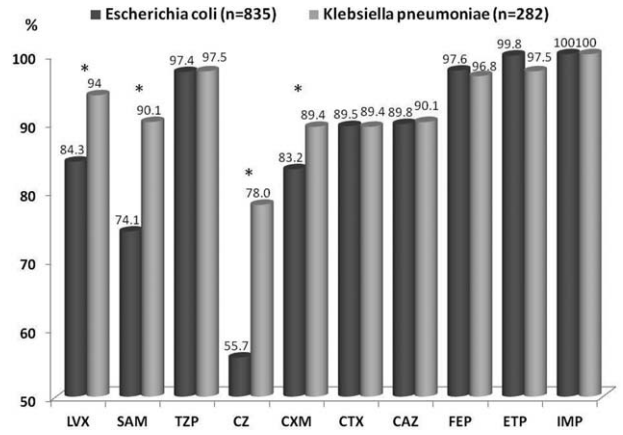

Figure 3. Susceptibility of *Escherichia coli* and *Klebsiella pneumoniae* causing community-onset bacteremia. CAZ = ceftazidime, CRO = cefotaxime, CXM = cefuroxime, CZ = cefazolin, ETP = ertapenem, FEP = cefepime, IMP = imipenem, LVX = Levofloxacin, SAM = ampicillin/sulbactam, TZP = piperacillin/tazobactam. *Indicated a significant difference ($P < .05$) between *E coli* and *K pneumoniae*.

Levofloxacin, ampicillin/sulbactam, piperacillin/tazobactam, cefuroxime, cefotaxime, ceftazidime, cefepime, and ertapenem were effective against 74.1% to 99.8% and 89.4% to 97.5% of *E coli* and *K pneumoniae* isolates, respectively. Notably, the *E coli* isolates had a significantly lower susceptibility to levofloxacin, ampicillin/sulbactam, cefazolin, and cefuroxime than did the *K pneumoniae* isolates (Fig. 3). A similar proportion of ESBL producers in the 2 groups (*K pneumoniae*, 14/274, 5.1% vs. *E coli*, 46/825, 5.6%; $P = .77$) was observed.

3.3. Predictors of 28-day mortality

The association of several clinical variables with 28-day mortality, in terms of age, gender, Pitt bacteremia score at bacteremia onset, bacteremia source, comorbidity severity, major comorbidities, resistant pathogens, inappropriate empirical antibiotic therapy, and abscess occurrence, was examined using univariate analysis (Table 3). The following variables were positively associated with 28-day mortality: critical illness (Pitt bacteremia score ≥4) at bacteremia onset, bacteremia because of pneumonia or intra-abdominal infections, inappropriate empirical antibiotic therapy, causative microorganisms with ESBL producers, ultimately or rapidly fatal comorbidities (McCabe classification), and comorbidities with liver cirrhosis or malignancies. In addition, female, bacteremia because of urosepsis, causative microorganisms with levofloxacin-susceptible isolates, and comorbid hypertension was negatively associated with 28-day mortality. Of note, the complicated abscess occurrence was not linked to 28-day mortality.

Consequently, only 6 independent predictors, ultimately or rapidly fatal comorbidities (McCabe classification), critical illness at bacteremia onset, bacteremia of pneumonia or urosepsis, ESBL producers, and underlying liver cirrhosis, were included in the multivariate regression model (Table 3).

3.4. Baseline characteristics and clinical outcomes after PSM analysis

Of the 825 patients in the *E coli* group, 242 patients were matched with 242 patients in the *K pneumoniae* group who had the closest propensity scores, according to 6 independent predictors of 28-day mortality (e.g., fatal comorbidities, critical illness at bacteremia onset, ESBL producers, bacteremia caused

Table 3

Association of 28-day mortality and clinical variables in adults with community-onset monomicrobial *Escherichia coli* or *Klebsiella pneumoniae* bacteremia.

Variables	Patients number (%)		Univariate analysis		Multivariate analysis	
	Fatal case (n = 116)	Survivor (n = 983)	OR (95% CI)	*P*	Adjusted OR (95% CI)	*P*
The elderly, ≥65 y	72 (62.1)	619 (63.0)	0.96 (0.65–1.43)	.85	—	—
Gender, female	43 (37.1)	591 (60.1)	0.43 (0.30–0.62)	<.001	NS	NS
Pitt bacteremia score ≥4 at bacteremia onset	71 (61.2)	109 (11.2)	12.65 (8.29–19.32)	<.001	11.19 (6.69–18.70)	<.001
Ultimately and rapidly fatal comorbidity (McCabe classification)	61 (52.6)	171 (17.4)	5.27 (3.53–7.86)	<.001	4.31 (2.56–7.28)	<.001
Major source of bacteremia						
Pneumonia	43 (37.1)	57 (5.8)	9.57 (6.03–15.19)	<.001	4.47 (2.41–8.28)	<.001
Urinary tract	24 (20.7)	547 (55.6)	0.21 (0.13–0.33)	<.001	0.46 (0.25–0.85)	.01
Intra-abdominal	20 (17.2)	107 (10.9)	1.71 (1.01–2.87)	.04	NS	NS
Biliary tract	8 (6.9)	123 (12.5)	0.52 (0.25–1.09)	.08	—	—
Bacteremic isolates						
Levofloxacin-susceptible isolate	83 (71.6)	872 (88.7)	0.32 (0.20–0.50)	<.001	NS	NS
Cefazolin-susceptible isolate	63 (54.3)	524 (55.1)	0.97 (0.66–1.42)	.87	—	—
ESBL producers	21 (18.1)	39 (4.0)	5.35 (3.02–9.47)	<.001	7.69 (3.56–16.61)	<.001
Inappropriate empirical antibiotic therapy	27 (18.2)	89 (9.4)	2.16 (1.35–3.46)	.001	NS	NS
Complicated abscess occurrence*	14/114 (12.3)	122/979 (12.5)	0.99 (0.55–1.78)	.96	—	—
Major comorbidities						
Malignancy	55 (47.4)	240 (24.4)	2.79 (1.89–4.13)	<.001	NS	NS
Liver cirrhosis	37 (31.9)	118 (12.0)	3.43 (2.22–5.31)	<.001	2.45 (1.33–4.51)	.004
Hypertension	36 (31.0)	507 (51.6)	0.42 (0.28–0.64)	<.001	0.60 (0.35–1.02)	.06
Diabetes mellitus	36 (31.0)	396 (40.3)	0.67 (0.44–1.01)	.05	—	—
Neurological disorder	30 (25.9)	195 (19.8)	1.41 (0.90–2.20)	.13	—	—
Chronic kidney disease	17 (14.7)	139 (14.1)	1.04 (0.61–1.80)	.88	—	—

CI = confidence interval, ESBL = extended-spectrum-β-lactamase, NS = nonsignificance (after processing the backward multivariate regression), OR = odds ratio.
* Six patients having an incomplete image study were excluded.

Table 4

Comparison of baseline characteristics, comorbidity severity, bacteremia severity, and crude mortality between patients with community-onset monomicrobial bacteremia caused by *Klebsiella pneumoniae* and those by *Escherichia coli*, matched by the propensity scores.

Characteristics	Patient number (%)		*P*
	K pneumoniae (n = 242)	*E coli* (n = 242)	
The elderly, ≥65 y	129 (53.3)	177 (73.1)	<.001
Gender, female	154 (63.6)	122 (50.4)	.03
Severity-of-illness marker at the bacteremia onset			
Pitt bacteremia score ≥ 4	46 (19.0)	46 (19.0)	1.00
Initial syndrome			
Severe sepsis	113 (46.7)	100 (41.3)	.23
Septic shock	52 (21.5)	56 (23.1)	.66
Major comorbidities			
Diabetes mellitus	105 (43.4)	89 (36.8)	.14
Hypertension	100 (41.3)	119 (49.2)	.08
Malignancy	70 (28.9)	81 (33.5)	.28
Neurological disorder	49 (20.2)	50 (20.7)	.91
Liver cirrhosis	44 (18.2)	44 (18.2)	1.00
Chronic kidney disease	28 (11.6)	36 (14.9)	.28
Major source of bacteremia			
Liver abscess	57 (23.6)	4 (1.7)	<.001
Urinary tract	55 (22.7)	55 (22.7)	1.00
Pneumonia	36 (14.9)	36 (14.9)	1.00
Biliary tract	27 (11.2)	57 (23.6)	<.001
Primary bacteremia	27 (11.2)	19 (7.9)	.22
Intra-abdominal	25 (10.3)	55 (22.7)	<.001
Skin and soft-tissue	11 (4.5)	6 (2.5)	.22
Complicated abscess occurrence	85 (35.1)	18 (7.4)	<.001
Comorbidity severity (McCabe classification)			1.00
Rapidly or ultimately fatal	56 (23.1)	56 (23.1)	
Nonfatal	186 (76.9)	186 (76.9)	
Crude mortality rate			
14–d	23 (9.5)	19 (7.9)	.52
28–d	34 (14.0)	30 (12.4)	.59

by pneumonia or urosepsis, and underlying liver cirrhosis). Although the number of the elderly and female patients and those with bacteremia caused by liver abscess, intra-abdominal or biliary tract infections differed between the 2 groups, appropriate matching was evident because no significant differences were observed between the 2 groups in bacteremia severity at onset, comorbidity severity, or major comorbidities (Table 4). Consequently, the crude mortality rates did not significantly differ between the 2 groups. Notably, more frequent abscess occurrences and longer lengths of total hospitalization (15.4 days vs. 12.8 days; $P = .02$) and ICU stays (2.0 days vs. 0.8 days; $P = .01$) were observed in the *K pneumoniae* group than in the *E coli* group (Fig. 2B).

4. Discussion

Several clinical characteristics at initial presentations, in terms of baseline demographics, bacteremia severity, bacteremia source, comorbidity severity, and types of comorbidities, differed between the *E coli* and *K pneumoniae* groups. Notably, the *K pneumoniae* group had a higher number of critical illness at bacteremia onset and poorer outcomes than did the *E coli* group. Using the PSM analysis, the clinical outcomes of these 2 groups became similar after adjustment for baseline characteristics, particularly for the comorbidity severity, bacteremia severity, and major comorbidities. Consequently, the distribution of bacteremia sources remained different between the 2 groups; more importantly, patients in the *K pneumoniae* group were more frequently complicated with abscess occurrences and had longer lengths of hospital stays than did the *E coli* group.

E coli and *K pneumoniae* are the leading species of community-onset *Enterobacteriaceae* bacteremia. Although several studies have described their characteristics individually,[4,6,10,11] a comprehensive comparison of these 2 pathogens has yet to be reported. Only a few studies have investigated the differences in the clinical presentations and outcomes between patients with liver abscesses caused by *E coli* and those by *K pneumoniae*.[26,27] In accordance with the previous studies that reported no differences in the short-term mortality rates of the aforementioned groups,[26,27] the present study demonstrated similar mortality rates in the PSM patients of the *E coli* and *K pneumoniae* groups. However, in contrast to the previous reports,[26,27] the severity of bacteremia and comorbidity at initial presentations were higher in the *K pneumoniae* group than in the *E coli* group.

To our knowledge, a distinct *K pneumoniae*-related invasive syndrome, which involved hepatic and extra-hepatic abscesses, has been increasingly reported in Asia[4] and has emerged as a global disease.[5,28] Because *K pneumoniae* harbors a virulence gene, *mag*A, and is hypermucoviscous, it has been associated with complications, such as abscess occurrence and septic metastasis.[3,29] Otherwise, because of a lack of hypermucoviscosity-related genes in *E coli*, no studies have reported septic metastasis secondary to liver abscess or bacteremia. This cohort study is the first to demonstrate that the frequency of abscess complications to bacteremia differed between these 2 microorganisms.

According to our review of the relevant literature, pneumonia is the most common infection among patients with community-acquired *K pneumoniae* bacteremia worldwide,[4] and urosepsis is the leading source of *E coli* bacteremia.[10] Thus, it was reasonable that the distribution of bacteremia source differed considerably between the 2 study groups despite PSM analysis. Because of the dissimilar distribution of bacteremia sources and frequencies of abscess occurrences, a longer length of hospitalization was observed in the *K pneumoniae* group than in the *E coli* group here.

Several limitations are inherent in the study design. First, the vast association of clinical outcomes and clinical variables at initial presentations, such as severity of bacteremia and comorbidity, were successfully established among bacteremic patients.[18,30] Because baseline characteristics significantly differed in the *E coli* and *K pneumoniae* groups, to overcome the difference of baseline variables and thus to compare the adverse impact of the different microorganism on patient outcome, we performed a PSM analysis to control these confounding factors (e.g., baseline variables) linked to mortality. Second, to assess the primary endpoint, patients with uncertain 28-day mortality were excluded, which may have resulted in selection bias. However, to reduce this bias, patient lacking the fatality information was retrieved through telephone interviews. Consequently, only a few patients (18/1117, 1.6%) were excluded from our study, and this bias was considered to have exerted negligible influence on the results. Third, to assess the relationship between the clinical outcomes and the causative microorganism, the occurrence of complicated abscesses was evaluated as a covariate here; only 6 patients who underwent limited imaging studies were excluded. The measure bias might exist because only half study patients underwent abdominal CT scans, but a similar proportion of those received the abdominal CT scans between the 2 groups (*E coli*, 406/825, 49.2% vs. *K pneumoniae*, 142/274, 51.8%; $P=.45$) was observed. Accordingly, the frequencies of abscess occurrences could be reasonably compared between the 2 groups. Based on our finding, a well-designed prospective study, which enrolls adults with bloodstream infections and conducts CT examinations, is warranted to validate our findings. Finally, no analyses for source control were performed in the present study. However, take the cases of urosepsis as the example. In those with or without interventions for source control, such as percutaneous nephrolithotomy or nephrectomy, the 28-day crude mortality rate was not different (2/36, 5.6% vs. 22/535, 4.1%; $P=.66$), indicative of standard quality of clinical care in the study hospital.

5. Conclusion

The present study indicates that several clinical characteristics differ between the adults with community-onset bacteremia caused by *E coli* and *K pneumoniae*. Patients in the *K pneumoniae* group had higher antimicrobial susceptibility, more critical illness at bacteremia onset, and poorer short-term prognosis than did those in the *E coli* group. In addition, after adjustment for the baseline characteristics associated with short-term mortality in the 2 groups, longer lengths of hospitalization and more complicated abscess occurrences were observed in the *K pneumoniae* group than in the *E coli* group.

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