Characteristics and Outcomes of Polymicrobial Bloodstream Infections in the Emergency Department: A Matched Case–control Study

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Abstract

Objectives: Polymicrobial bloodstream infection (BSI) is a critical condition and has been increasingly reported; however, the authors were unable to find an emergency department (ED) patient-based study in the literature.

Methods: A retrospective matched case–control study with a ratio of 1:3 among patients with polymicrobial BSIs in an ED was conducted. The case group was patients aged > 16 years with polymicrobial BSIs. Patients matched for age and sex with monomicrobial BSIs were sampled as the control group. Demographic information, underlying conditions, microbiologic data, and outcomes were collected for further analysis.

Results: From January 2005 to December 2007, a total of 112 episodes of polymicrobial BSIs among 109 patients were included. Two pathogens were isolated among 87 (77.7%) episodes and three were found among 25 (22.3%) episodes. A history of hospitalization within 90 days was an independent risk factor for polymicrobial BSIs ($p = 0.003$). Intraabdominal infection ($p < 0.001$) and respiratory tract infection ($p = 0.017$) were more likely to be associated with polymicrobial BSIs. Gram-negative and Gram-positive bacteria were documented in 95.5 and 46.4% episodes of polymicrobial BSIs, respectively. Inappropriate antimicrobial treatment was observed in 53.6% of polymicrobial BSIs, but only accounted for 23.8% of monomicrobial BSIs ($p < 0.001$). The overall 30-day mortality rate of the polymicrobial group was significantly higher than those with monomicrobial BSIs (30.3 and 11.6%, respectively; $p < 0.001$).

Conclusions: Patients with polymicrobial BSIs had a high mortality rate. Acknowledgment of the clinical and microbiologic characteristics and recognition of patients at risk for polymicrobial BSIs are critical in EDs.

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Blood cultures are frequently used to evaluate patients with fever in emergency departments (EDs). More than 3.1 million blood cultures, associated with 2.8% of all ED visits, are ordered annually in the United States.[1] Although almost half of all blood cultures ordered in the ED may not be necessary, patients with positive blood cultures more often than not have a serious infection and need admission for further treatment.[1,2]

Polymicrobial bloodstream infection (BSI), defined as the presence of at least two different microorganisms found from the blood cultures, has been reported increasingly, with rates ranging from 6% to 32% of all BSI episodes.[3–7] The mortality rate of hospitalized patients with polymicrobial BSIs ranged from 21% to 63%, approximately twice the rate of those with monomicrobial infections.[3,4,8,9] Most patients with

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polymicrobial BSIs have underlying medical conditions including malignancy, neutropenia, gastrointestinal disease, genitourinary disease, recent surgical procedures, or the presence of central venous catheters.[3–7,10,11] Nevertheless, the majority of investigations of polymicrobial BSIs are limited to unselected populations or neutropenic patients with underlying malignancy.

Despite the critical nature of polymicrobial BSIs in EDs, to our knowledge, no emergency patient-based study for polymicrobial BSIs could be found in the literature. Therefore, we performed a retrospective matched case–control study among patients with polymicrobial BSIs with the following objectives: 1) to explore clinical characteristics and underlying diseases, 2) to identify isolated microorganisms, 3) to define the sources of infections, 4) to evaluate empirical antimicrobial treatment, and 5) to compare their survival rates.

METHODS

Study Design

We conducted a retrospective matched case–control study to compare polymicrobial with monomicrobial BSIs in patients arriving at an ED. This study was approved by the institutional review board of E-Da Hospital in Taiwan.

Study Setting and Population

This study was carried out at E-Da Hospital, a 1,000-bed university-affiliated hospital in southern Taiwan with approximately 60,000 annual ED visits. The records of all positive blood cultures collected at an ED between January 2005 and December 2007 from patients aged > 16 years were reviewed.

Study Protocol

The medical records of included patients were each reviewed by two authors. If any discrepancy was found, the medical records were inspected again by these two authors together. Demographic information of age and sex, underlying illness, clinical condition, microbiology, source of bacteremia, and outcome were collected from the medical records. Clinical data were analyzed by case episodes. All patients with polymicrobial BSIs were enrolled into the case group, whereas those with monomicrobial BSIs were enrolled in the control group. For the purpose of comparison, we performed matching by a blinded observer unaware of the clinical outcomes. Patients with polymicrobial BSIs were matched by sex and age (±3 years) with a ratio of 1:3 to those with monomicrobial BSIs during the same study period.

Episodes were considered distinct if separated by at least 7 days and if the reason for visiting the ED was different. Monomicrobial and polymicrobial BSIs were defined as one species and two or more species that were isolated from blood cultures, respectively. Shock was defined as systolic pressure less than 90 mm Hg or requiring inotropic agents to maintain blood pressure during the ED stay. The sources of BSIs were determined clinically on the basis of the presence of an active infection site coincident with BSIs or isolation of the organism from other clinical specimens prior to or on the same date as the onset of BSIs. If a polymicrobial BSI was presumed to be due to two different sources, the episode was excluded from our study. If the source of a BSI could not be attributed to any known source, it was classified as a primary BSI.

According to the blood culture collection guidelines of E-Da Hospital, two sets of blood cultures collected 30 minutes apart with proper site preparation and aseptic technique were ordered routinely by physicians if infection was suspected. If patients received antibiotics within 24 hours, BACTEC resin-containing blood culture bottles were used (Becton Dickinson Diagnostic Instrument System, Sparks, MD). All blood culture samples were processed by the BACTEC 9240 system (Becton Dickinson). Susceptibilities to antimicrobial agents were determined according to the Clinical and Laboratory Standards Institute criteria of the year.[12–14] If the blood cultures yielded skin flora, including coagulase-negative staphylococci, micrococci species, diphtheroids, *Bacillus* species, or *Propionibacterium acnes* in only one culture, these microorganisms were regarded as contaminated,[15] and these episodes were excluded from our study.

Antimicrobial therapy was considered inappropriate if the regimen included antimicrobial agents that were not effective against all of the pathogens isolated from the blood cultures via in vitro susceptibility testing or the lack of antibiotic therapy during the ED stay. We classified the inappropriate antibiotics therapy into four groups: 1) use of improper kinds of antibiotics (such as enterococci, which should be treated with penicillin but were treated with a cephalosporin); 2) use in resistant microorganisms (such as extended-spectrum β-lactamase-producing *Enterobacteriaceae* and methicillin-resistant *Staphylococcus*); 3) combination therapy needed but not received (such as two or three isolates in polymicrobial BSIs, which cannot be treated by only one antibiotic); and 4) no antibiotics use during the ED stay.

Outcome Measures

We used the overall 30-day mortality for outcome analysis. The causes of mortality were determined by the medical records. If patients were discharged within 30 days after admission and were not followed up at our hospital, telephone contact was made to collect the required information. Patients who were lost to follow-up were excluded from our study.

Data Analysis

We used the SPSS software package (version 14.0, SPSS Inc., Chicago, IL) to analyze the results. Categorical variables were analyzed using the chi-square test or Fisher exact tests, as appropriate. Continuous variables were analyzed using the Student's t-test. Univariate odds ratios (ORs) were computed by the Mantel-Haenszel test. To identify the risk factors for polymicrobial BSIs, underlying conditions that could contribute to polymicrobial BSIs and were associated with a level of significance of less than 0.20 in univariate analyses were included in a logistic regression model for multivariate analysis (conditional backward stepwise model). The OR, 95% confidence interval (CI), and p-value

were calculated for each factor. The Hosmer-Lemeshow goodness-of-fit test was used to assess the fitness of the model. Survival data were analyzed by Kaplan-Meier plots. Associations between variables and survival were compared using the log-rank tests. All p-values were two-tailed and a p-value < 0.05 was considered statistically significant.

RESULTS

Characteristics of Enrolled Episodes

During the study period, a total of 4,290 positive blood cultures were documented among 162,760 ED visits (Figure 1). A total of 112 episodes of polymicrobial BSIs among 109 patients were included in our study. Three patients had two episodes of polymicrobial BSIs. Two pathogens were isolated among 87 (77.7%) episodes, and three pathogens were found among 25 (22.3%) episodes. Sixty patients were male and 52 were female, and their median age was 65 years (mean ± SD = 64.0 ± 15.7; range = 18–89 years).

Underlying Conditions

After matching, no significant differences by age or sex were noted between case and control groups. The prevalence of underlying conditions is shown in Table 1. The presence of malignancy (p = 0.018) and a history of hospitalization within 90 days (p = 0.003) were associated with polymicrobial BSIs. However, multivariate regression analysis showed that only patients with a history of hospitalization within 90 days were predisposed to polymicrobial BSIs (OR = 1.92; 95% CI = 1.25 to 2.97; p = 0.003).

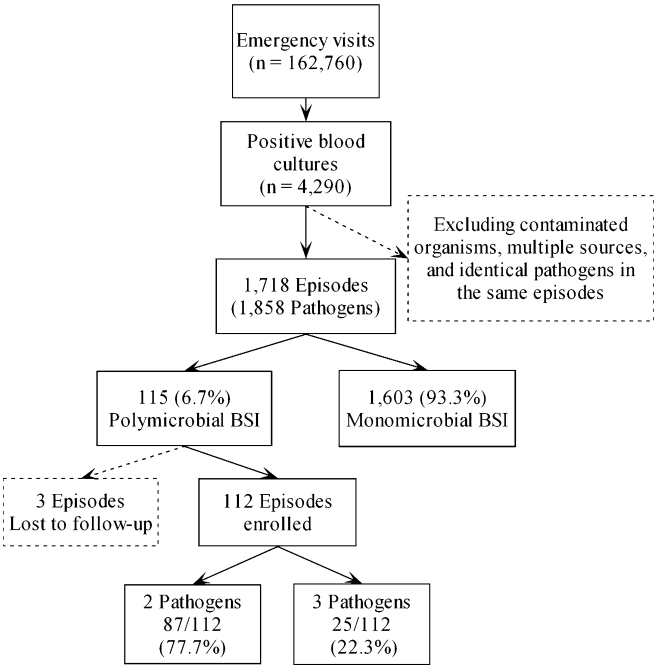

Figure 1. Patient enrollment, number of bacteremic episodes, and number of isolates in polymicrobial BSIs. BSI = bloodstream infection.

Table 1
Demographic Characteristics, Underlying Conditions, and Clinical Manifestations of 112 Polymicrobial and 336 Monomicrobial BSI Episodes

Characteristics	No. (%) of Episodes		OR (95% CI)
	Polymicrobial (*n* = 112)	Monomicrobial (*n* = 336)	
Age (year, mean ± sd)	64.0 ± 15.7	64.4 ± 15.5	—
Sex, male	60 (53.6)	180 (53.6)	1.0 (0.65–1.54)
Clinical condition			
Diabetes mellitus	35 (31.3)	135 (40.2)	0.68 (0.43–1.07)
End-stage renal disease	3 (2.7)	18 (5.4)	0.49 (0.14–1.68)
Malignancy	36 (32.1)	71 (21.1)	1.77 (1.10–2.84)
Liver cirrhosis	18 (16.1)	47 (14.0)	1.18 (0.65–2.13)
HIV infection	0	3 (0.9)	—
COPD	7 (6.3)	13 (3.9)	1.66 (0.64–4.26)
Nosocomial infection	8 (7.1)	22 (6.5%)	0.91 (0.39–2.11)
Hospitalized within 90 days	56 (50.0)	115 (34.2)	1.92 (1.25–2.97)
Catheter/tube			
Port-A Cath	15 (13.4)	33 (9.8)	1.42 (0.74–2.73)
Bile drainage tube	0	2 (0.6)	—
Urinary catheter	3 (2.7)	7 (2.1)	1.29 (0.33–5.09)
Clinical manifestation			
Anemia*	35 (31.3)	89 (26.5)	1.26 (0.79–2.01)
Neutropenia†	5 (4.5)	6 (1.8)	2.57 (0.77–8.59)
Thromocytopenia‡	36 (32.1)	78 (23.2)	1.57 (0.98–2.51)
Shock	18 (16.1)	33 (9.8)	1.76 (0.95–3.27)
Inappropriate antimicrobial therapy	60 (53.6)	80 (23.8)	3.69 (2.36–5.78)
30-day mortality	34 (30.4)	39 (11.6)	3.32 (1.97–5.60)

BSI = bloodstream infection; COPD = chronic obstructive pulmonary disease; HIV = human immunodeficiency virus.
*Hemoglobin < 10 g/dL.
†Absolute neutrophil count < 0.5×10^9/L.
‡Platelet count < 100×10^9/L.

Table 2
Sources of 112 Polymicrobial and 336 Monomicrobial BSIs

Source of BSI	No. (%) of Episodes: Polymicrobial (n = 112)	No. (%) of Episodes: Monomicrobial (n = 336)	OR (95% CI)	p-value
Urinary tract infection	19 (17.0)	131 (39.0)	0.32 (0.19–0.55)	<0.001
Lower respiratory tract infection	20 (17.9)	32 (9.5)	2.07 (0.02–1.23)	0.017
Catheter-related infection	3 (2.7)	8 (2.4)	1.23 (0.29–4.33)	1
Intraabdominal infection	38 (33.9)	64 (19.0)	2.18 (1.36–3.52)	0.001
Skin and soft tissue infection	19 (17.0)	36 (10.7)	1.70 (0.93–3.11)	0.081
Bone/joint infection	0	5 (1.5)	—	0.338
Intravascular infection	1 (0.9)	8 (2.4)	0.37 (0.05–2.99)	0.461
Primary bacteremia	12 (10.7)	48 (14.3)	0.72 (0.37–1.41)	0.337
Others*	0	4 (1.2)	—	—

BSI = bloodstream infection.
*Including central nervous system infection and ear, nose, and throat infection.

Sites of Infections

The sources of infections could be identified in 89.3% of polymicrobial BSIs and in 85.7% of monomicrobial BSIs. The most common source of polymicrobial BSIs was intraabdominal infection (33.9%). Comparing with monomicrobial BSIs, sources of polymicrobial BSIs were significantly more likely attributed to intraabdominal infection (p = 0.001) and lower respiratory tract infection (p = 0.017). On the contrary, monomicrobial episodes were significantly more likely to arise from the urinary tract infection (p < 0.001; Table 2).

Pathogens of Bacteremia

The isolates of polymicrobial and monomicrobial BSIs are presented in Table 3. Only one episode of *Candida albicans* was found in the group of polymicrobial BSIs. Gram-positive bacteria were found in 95.5% of polymicrobial BSIs, and Gram-negative bacteria were identified in 46.4%. The most frequent isolates of Gram-negative and Gram-positive bacteria in polymicrobial BSIs were *Escherichia* species (57.1%) and streptococci (26.8%), respectively.

Table 4 shows the microorganisms isolated from patients with polymicrobial BSIs according to their infection sites. Gram-negative bacteria could be recognized in the majority of urinary tract infections (73.8%), lower respiratory tract infections (86.7%), catheter-related BSIs (85.7%), and intraabdominal infections (78.4%). In contrast, Gram-negative bacteria could be identified in only half of skin and soft tissue infections. The most common polymicrobial combination in both lower respiratory tract infection and intraabdominal infection was *Escherichia coli* and *Klebsiella pneumoniae* coinfection (25 and 23.7%, respectively).

Antimicrobial Therapy

Inappropriate antimicrobial treatment was found in 53.6% of patients with polymicrobial BSIs and was significantly more frequent than in those with monomicrobial BSIs (23.8%; OR = 3.69; 95% CI = 2.36 to 5.78; p < 0.001; Table 1). Figure 2 shows the proportions of inappropriate antimicrobial therapy in polymicrobial BSIs by different sites of infections. The types of inappropriate antibiotics use are demonstrated in Figure 3. One-third of polymicrobial bacteremic patients should have received combination therapy of antibiotics but did not.

Survival Analysis

The overall 30-day mortality rates of patients with polymicrobial and monomicrobial BSIs were 30.3 and 11.6%, respectively. Patients receiving inappropriate antibiotics had a higher mortality rate than those who were treated with appropriate antibiotics (p = 0.011). Among these mortality cases, 70 died directly due to infections, and three patients died of other etiologies (hepatocellular carcinoma rupture, spontaneous subarachnoid hemorrhage, and respiratory failure due to pulmonary metastasis in a hepatocellular carcinoma patient). In the survival analysis, patients with polymicrobial BSIs had a significantly higher overall mortality rate than those with monomicrobial BSIs (p < 0.001; Figure 4). Patients infected with two or three microorganisms had a significantly higher overall 30-day mortality rate than those with only one isolate (p < 0.001 and p = 0.001, respectively). However, there was no statistical difference between the group of two-pathogen and three-pathogen polymicrobial BSIs (p = 0.234).

DISCUSSION

Our study found that polymicrobial BSIs accounted for 6.7% of all BSI episodes in the ED, which is similar to the previous reports of general population studies.[4,16] We also identified that a history of hospitalization within 90 days was an independent risk factor for polymicrobial BSIs, as was bacteremia due to lower respiratory tract infection or intraabdominal infection. Polymicrobial BSIs are known to be associated with hospital-acquired infections in several studies.[8,9,17] Intraabdominal infection is also a well-known cause of polymicrobial bacteremia.[9] Lower respiratory tract infections have also been associated with multiple pathogens. Lauderdale et al.[18] reported that polymicrobial infection occurred in 4% to

Table 3
Microorganisms Among 112 Polymicrobial and 336 Monomicrobial BSIs

Microorganism	No. (%) of Episodes: Polymicrobial (*n* = 112)	No. (%) of Episodes: Monomicrobial (*n* = 336)	OR (95% CI)	p-value
Gram-positive bacteria*	60 (53.6)	80 (23.8)		
Staphylococcus spp.	15 (13.4)	38 (11.3)	1.21 (0.64–2.30)	0.554
S. aureus	14 (12.5)	36 (10.7)	1.19 (0.62–2.30)	0.603
MRSA	9 (8.0)	15 (4.5)	1.87 (0.80–4.40)	0.146
Streptococci	30 (26.8)	29 (8.6)	3.87 (2.20–6.82)	<0.001
S. pneumoniae	1 (0.9)	2 (0.6)	1.51 (0.14–16.75)	1
Enterococcus spp.	14 (12.5)	7 (2.1)	6.71 (2.64–17.10)	<0.001
Other‡	0	4 (1.2)	—	—
Gram-negative bacteria†	189 (168.8)	255 (75.9)		
Escherichia spp.	64 (57.1)	136 (40.5)	1.96 (1.27–3.02)	0.002
E. coli	63 (56.3)	136 (40.5)	1.89 (1.23–2.91)	0.004
ESBL production	3 (2.7)	5 (1.5)	1.82 (0.43–7.75)	0.417
Klebsiella spp.	50 (44.6)	52 (15.5)	4.40 (2.74–7.09)	<0.001
K. pneumoniae	46 (41.1)	52 (15.5)	3.81 (2.36–6.14)	<0.001
ESBL production	3 (2.7)	0	—	—
Pseudomonas spp.	19 (17.0)	15 (4.5)	4.37 (2.14–8.94)	<0.001
Proteus spp.	11 (9.8)	7 (2.1)	5.12 (1.93–13.55)	0.001
Enterobacter spp.	10 (8.9)	5 (1.5)	6.49 (2.17–19.42)	0.001
Aeromonas spp.	4 (3.6)	6 (1.8)	2.04 (0.56–7.35)	0.277
Salmonella spp.	0	7 (2.1)	—	0.200
Acinetobacter spp.	4 (3.6)	8 (2.4)	1.52 (0.45–5.14)	0.505
Citrobacter spp.	6 (5.4)	4 (1.2)	4.70 (1.30–16.96)	0.019
Morganella morganii	5 (4.5)	2 (0.6)	7.80 (1.49–40.81)	0.012
Other‡	15 (13.4)	13 (3.9)	3.84 (1.77–8.35)	0.001
Fungi				
Candida albicans	1 (0.9)	1 (0.3)	3.02 (0.19–48.65)	0.438

BSI = bloodstream infection; ESBL = extended-spectrum β-lactamase; MRSA = methicillin-resistant *S. aureus*.
*The rate of Gram-positive bacteria in one episode of polymicrobial BSI was 46.4% (52/112), significantly higher than monomicrobial BSI (OR = 2.77; 95% CI = 1.77 to 4.34; p < 0.001).
†The rate of Gram-negative bacteria in one episode of polymicrobial BSI was 95.5% (107/112), significantly higher than monomicrobial BSI (OR = 6.80; 95% CI = 2.68 to 17.25; p < 0.001).
‡Including *Providencia alcalifaciens, Chryseobacterium meningosepticum, Serratia marcescens, Moraxella* spp., *Plesiomonas shigelloides, Weeksella virosa, Vibrio cholerae, Vibrio vulnificus, Flavobacterium* spp., *Shewanella putrefaciens* Bv. 1, *Stenotrophomonas maltophilia*, *Pasteurella multocida*, *Methylobacterium* spp., *Pantoea agglomerans*, *Lactobacillus* spp., *Leclercia adecarboxylata*, *Listeria monocytogenes*, *Stomatococcus mucilaginosus*, and *Roseomonas* spp.

39% of patients with community-acquired pneumonia. *Streptococcus pneumoniae* is found to be the most common pathogen mixed with other pathogens in western countries.[19–21] However, our study revealed that *K. pneumoniae* is the most common isolate in the polymicrobial bacteremic patients. This may be due to the high prevalence rate of *K. pneumoniae* infection in Taiwan.[22]

With regard to the microbiology of polymicrobial BSIs, we noted that nearly all episodes included at least one isolate of Gram-negative bacilli, but only around half of episodes involved Gram-positive bacteria. In a study of inpatient polymicrobial BSIs, 44% of the infections involved at least one Gram-negative bacterium and 43% involved Gram-positive bacteria.[4] In another large study of polymicrobial BSIs among malignancy patients, 76% of the infections involved at least one Gram-negative bacillus, and 50% involved Gram-positive organisms.[16] The culture rate of Gram-positive bacteria was similar to our study of emergency-based patients, but the yielding rate of Gram-negative bacteria has differed in the literature. Because these results are important to provide the information for empirical antimicrobial treatment in clinical practice, more studies involving patients with different underlying diseases and different populations are necessary.

Empirical treatment is given before results of blood cultures are available, and the prescription of empirical antibiotics may be dependent on a clinical basis alone. Our study showed that the empirical antibiotics treatment was inappropriate in 53.6% of patients with polymicrobial BSIs, which is much higher than those with monomicrobial BSIs. Regarding the causes of inappropriate use of antibiotics, we found that no combination therapy occurred in one-third of episodes. In contrast to monomicrobial BSIs, treating multiple pathogens with only one antibiotic could be inadequate, even when a broad-spectrum antibiotic was prescribed. For example, enterococci are intrinsically resistant to cephalosporins. If a broad-spectrum cephalosporin is used for polymicrobial infections including enterococci, the treatment will be inadequate. So acknowledging the common causative microorganisms in each site of infection is important to guide empirical antibiotics treatment.

Our study also found that almost a fourth of bacteremic patients received no antibiotics during their ED stay, neither for polymicrobial nor for monomicrobial

BSIs. Indeed, there is no indicator available to precisely predict the presence of bacteremia.[23,24] Delayed or absent antibiotic treatment is most often due to inaccurate clinical evaluation of the risk factors for severe infection or bacteremia.[25] Adequate education and interdisciplinary coordination may improve clinical performance.

The mortality rate of patients with polymicrobial BSIs ranged from 14% to 43%, approximately two times the mortality rate of those with monomicrobial BSIs.[26] The higher mortality in polymicrobial BSIs was reported to be related to inappropriate antimicrobial treatment.[4,16,17,27,28] As a result, the empirical antimicrobial therapy that could cover the possible pathogens is

Table 4
Pathogens Isolated from 112 Polymicrobial BSIs

Pathogens	UTI (*n* = 19)	LRI (*n* = 20)	CRBSI (*n* = 3)	IAI (*n* = 38)	SSTI (*n* = 19)	Intravascular Infection (*n* = 1)	Primary (*n* = 12)
Gram-positive bacteria	11	6	1	19	20	0	3
Staphylococcus spp.	2	2	1	2	8	0	0
S. aureus	2	2	1	2	7	0	0
MRSA	2	1	1	1	4	0	0
Streptococci	4	2	0	11	10	0	2
S. pneumoniae	0	1	0	0	0	0	0
Enterococcus spp.	5	1	0	6	2	0	1
Gram-negative bacteria	31	39	6	69	21	0	20
Escherichia spp.	11	7	1	32	10	0	4
E. coli	11	7	1	31	10	0	4
ESBL production	1	0	0	1	1	0	1
Klebsiella spp.	5	11	0	23	2	1	5
K. pneumoniae	5	10	2	21	1	1	5
ESBL production	1	1	0	0	0	0	0
Pseudomonas spp.	4	10	0	2	0	0	4
Proteus spp.	3	2	0	2	4	0	0
Enterobacter spp.	1	1	3	1	0	1	3
Aeromonas spp.	0	0	0	2	0	0	2
Acinetobacter spp.	1	0	0	1	2	0	0
Citrobacter spp.	3	1	0	2	0	0	0
Morganella morganii	2	0	0	1	1	0	1
Other*	1	7	0	3	2	0	1
Fungi							
C. albicans	0	0	0	0	0	0	1

CRBSI = catheter-related bloodstream infection; ESBL = extended-spectrum β-lactamase; IAI = intraabdominal infection; LRI = lower respiratory tract infection; MRSA = methicillin-resistant *S. aureus*; SSTI = skin and soft tissue infection; UTI = urinary tract infection.
*Including *P. alcalifaciens, C. meningosepticum, S. marcescens, Moraxella osloensis, P. shigelloides, Flavobacterium* spp., *Roseomonas* spp., and *V. cholerae.*

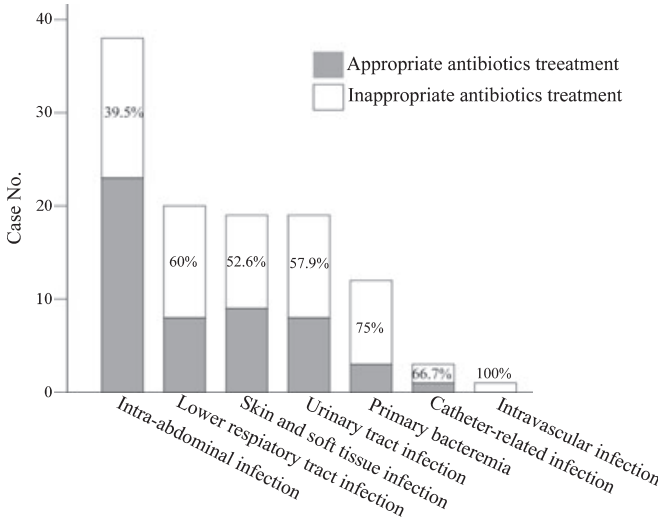

Figure 2. Proportions of inappropriate antimicrobial therapy in polymicrobial BSIs by different sites of infections. BSI = bloodstream infection.

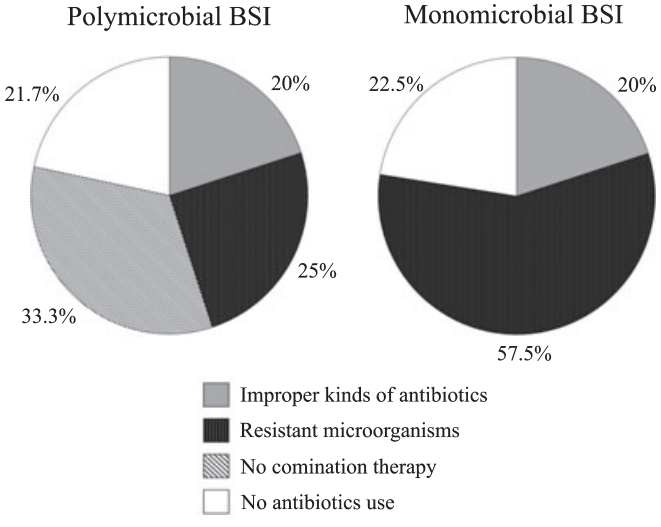

Figure 3. Types of inappropriate antimicrobial therapy in polymicrobial and monomicrobial BSIs. BSI = bloodstream infection.

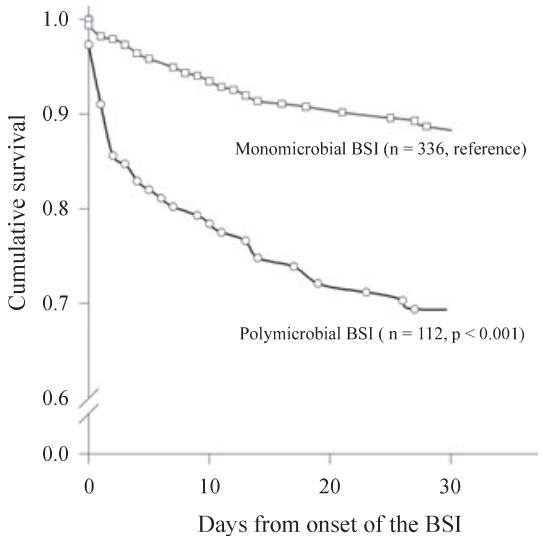

Figure 4. Kaplan-Meier survival curves of the 30-day cumulative survival rate for patients with polymicrobial and monomicrobial BSIs in the ED. BSI = bloodstream infection.

essential for treating patients at risk for polymicrobial BSIs. Effective and early antimicrobial therapy administered during an ED stay for BSI has been demonstrated to improve survival rates.[25,28,29] Although the organisms and the number of causative isolates will not be known at the time of blood cultures, recognizing patient-specific risk factors, the suspicious infection sources, and the most common pathogens, as well as the regional and institutional patterns of antibiotic resistance, will help physicians to provide adequate empirical antibiotics therapy.

LIMITATIONS

First, our study was conducted at a single hospital in southern Taiwan, limiting its generalizability. Second, although there is a standard practice regarding the use of blood cultures, we could not ascertain that all emergency physicians adhered to established guidelines. Therefore, omission of patients with polymicrobial BSIs could exist. Third, while there is an antibiotics use policy in our hospital, the prescription of empirical antibiotics may be dependent on the personal experiences of physicians. Fourth, the data used in our analysis were collected from medical records. There may be inconsistencies among the completeness of these data. Finally, because all of the univariate and multivariate analyses were carried out in an exploratory fashion, some risk factors may not have been detected or explored in our study. Further prospective studies with specific hypotheses are necessary to accurately determine these risk factors.

CONCLUSIONS

Our study provides important information about the clinical and microbiologic manifestations of ED patients with polymicrobial BSIs. Inasmuch as the mortality rate of patients with polymicrobial BSIs is significantly higher than those with monomicrobial infection, identifying patients at risk for polymicrobial BSIs, knowing the most common microorganisms in different infection sites, and being familiar with the epidemiology of regional and institutional patterns of antibiotic resistance are of paramount importance to provide adequate antimicrobial therapy in EDs.

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