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Epidemiology of Bloodstream Infections Caused by *Acinetobacter baumannii* and Impact of Drug Resistance to both Carbapenems and Ampicillin-Sulbactam on Clinical Outcomes

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***Acinetobacter baumannii* has become a leading cause of bloodstream infections (BSI) in health care settings. Although the incidence of infection with carbapenem- and ampicillin-sulbactam-resistant (CASR) *A. baumannii* has increased, there is a scarcity of studies which investigate BSI caused by CASR *A. baumannii*. A retrospective cohort study was conducted on adult patients with BSI caused by *A. baumannii* and who were admitted to the Detroit Medical Center between January 2006 and April 2009. Medical records were queried for patients' demographics, antimicrobial exposures, comorbidities, hospital stay, and clinical outcomes. Bivariate analyses and logistic regression were employed in the study. Two hundred seventy-four patients with BSI caused by *A. baumannii* were included in the study: 68 (25%) caused by CASR *A. baumannii* and 206 (75%) caused by non-CASR *A. baumannii*. In multivariate analysis, factors associated with BSI caused by CASR *A. baumannii* included admission with a rapidly fatal condition (odds ratio [OR] = 2.83, 95% confidence interval [CI] = 1.27 to 6.32, *P* value = 0.01) and prior use of antimicrobials (OR = 2.83, 95% CI = 1.18 to 6.78, *P* value = 0.02). In-hospital mortality rates for BSI caused by CASR *A. baumannii* were significantly higher than those for non-CASR *A. baumannii*-induced BSI (43% versus 20%; OR = 3.0, 95% CI = 1.60 to 5.23, *P* value < 0.001). However, after adjusting for potential confounders, the association between BSI caused by CASR *A. baumannii* and increased risk of in-hospital mortality was not significant (OR = 1.15, 95% CI = 0.51 to 2.63, *P* value = 0.74). This study demonstrated that CASR *A. baumannii* had a distinct epidemiology compared to more susceptible *A. baumannii* strains; however, clinical outcomes were similar for the two groups. Admission with a rapidly fatal condition was an independent predictor for both CASR *A. baumannii* and in-hospital mortality.**

Acinetobacter, a Gram-negative coccobacillus, has become a frequent pathogen in hospitals and other health care settings (1, 2). Found naturally in soil, these bacteria remain stable even under extreme conditions of temperature, humidity, and pH and in the presence of commonly used detergents, such as highly concentrated alcohol preparations, and other antiseptics that normally inhibit the growth of other bacteria (3). This stability offers *Acinetobacter* a growth advantage over other organisms in hospital environments. *Acinetobacter baumannii* is the most prevalent human pathogen among *Acinetobacter* species and utilizes multiple mechanisms of antimicrobial resistance (2). *A. baumannii* presents a challenge to health care personnel in terms of treatment and infection control (4).

A. baumannii is known to cause bacteremia and infections of wounds and the respiratory, gastrointestinal, and genitourinary tracts (2) and has increasingly become a cause of bloodstream infections (BSI) and pneumonia (4, 5). Traditionally, infections caused by *Acinetobacter* species were restricted to the intensive care unit (ICU) setting and to military personnel who have experienced combat-related injuries (6). More recently, these pathogens have spread to other locations in the hospital and to nonhospital populations and health care settings (1, 6, 7). Infections caused by *A. baumannii* are associated with devastating outcomes in terms of morbidity and mortality and contribute to high hospital costs. Among patients with bacteremia caused by *A. baumannii*, overall mortality exceeds 50% (5, 8).

As rates of infection have increased, so has the incidence of infection with multidrug-resistant (MDR) isolates of *Acinetobacter* species, defined as those strains that are resistant to three or more classes of antibiotics (9–13). In fact, many regions of the world now encounter *A. baumannii* strains that are resistant to the two agents most commonly used to treat *A. baumannii*: type 2 carbapenems (such as imipenem, meropenem, and doripenem) and ampicillin-sulbactam. In comparison with susceptible strains of *A. baumannii*, MDR infections are associated with additional increases in morbidity, mortality, length of hospital stay, and health care costs (2, 14–16).

Providing effective treatment for infections caused by MDR *A. baumannii* is a challenge. MDR strains typically require therapy with colistin, an older and relatively toxic polymyxin antimicrobial, and aminoglycosides or with the newer antimicrobial agent tigecycline, which attains poor serum and urine levels and has a limited track record in treating serious infections, including those caused by *A. baumannii* (17). The rise in the incidence of MDR *A. baumannii* is compounded by the lack of new antimicrobials in the pharmaceutical industry research and developmental pipeline (18). While colistin and tigecycline are considered first-line ther-

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apeutics for MDR *A. baumannii*, panresistant isolates have already been described (19), for which there are no available effective therapeutic options (20, 21).

There are limited data pertaining to bloodstream infections caused by MDR *A. baumannii* and no data to our knowledge analyzing bloodstream infections caused by carbapenem- and ampicillin-sulbactam-resistant (CASR) *A. baumannii*. One study which analyzed patients with bloodstream infections caused by carbapenem-resistant *A. baumannii* reported a 30-day mortality rate of 48%. In this study, severity of illness and presence of immunosuppression were the only two predictors of 30-day mortality. Of note, the study did not evaluate ampicillin-sulbactam susceptibilities, and thus, isolates could not be considered CASR (22).

Southeastern Michigan has been particularly affected by MDR *A. baumannii*, which has increased exponentially in prevalence over the past several years and has become a predominant nosocomial pathogen in the region (23). The objectives of this study conducted in Southeastern Michigan were to (i) identify differences between patients with bloodstream infections caused by CASR *A. baumannii* and patients with bloodstream infections caused by more susceptible strains of *A. baumannii* and (ii) determine the impact of CASR on clinical outcomes of patients with bloodstream infections caused by *A. baumannii*.

MATERIALS AND METHODS

Study design and setting. A retrospective cohort study was conducted at the Detroit Medical Center (DMC) from January 2006 to April 2009. The DMC is a metropolitan hospital system consisting of eight hospitals with over 2,200 beds in the greater metro-Detroit area. The study was approved by the institutional review board at DMC and Wayne State University.

Patient population. Patients with blood cultures positive for *A. baumannii* were identified from laboratory data. Patients with bloodstream infections caused by CASR *A. baumannii*, defined as one or more blood cultures positive for a strain of *A. baumannii* resistant to ampicillin-sulbactam (MIC of ≥16/8 μg/ml) and to meropenem and/or imipenem (MIC of ≥8 μg/ml), were compared to patients with bloodstream infections caused by non-CASR *A. baumannii*, defined as one or more positive blood cultures with an *A. baumannii* strain susceptible to meropenem and/or imipenem (MIC of ≤4 μg/ml) and/or ampicillin-sulbactam (MIC of ≤8/4 μg/ml). If patients had more than one episode of bloodstream infection caused by *A. baumannii* during the study period, only the initial episode was included.

Variables and definitions. Variables abstracted from the patient chart included demographics, comorbid conditions, functional status, hospital mortality, and antimicrobials. The number of positive blood cultures was also recorded. All antimicrobial drug use data were abstracted from electronic medication administration records. Therapy was divided into three categories based on the timing of therapy in relation to the initial positive blood culture: prior, empirical, and consolidative. The day on which the initial positive blood culture was obtained was considered day 0. Prior therapy included antimicrobials administered from 30 days prior to 3 days prior to culture (i.e., days −30 to −3). Empirical therapy included antimicrobials administered between days −5 and +3, and consolidative therapy was defined as antimicrobials administered from days +4 to +7.

The number of days to initiation of appropriate antimicrobial therapy was also analyzed. Appropriate therapy was defined as the use of a systemic agent which demonstrated *in vitro* activity against the *A. baumannii* pathogen.

Statistical analysis. All analyses were performed using SAS software (version 9.2; Cary, NC). The *t* test and Wilcoxon rank sum test were used to analyze continuous variables, the chi-square and Fisher exact tests were used for bivariate analyses, and logistic regression was used for multivariate analyses. For multivariate model building, variables with a *P* value of <0.10 in the bivariate analyses were included as candidate variables. Logistic regression with backwards selection was used to select for variables in the final model. Final models included variables with an adjusted *P* value of <0.05. All candidate variables that were not selected for final model inclusion were checked for confounding. Confounders were defined as variables that changed the β-coefficients of selected variables by >10% when added back to the model. Confounding variables were incorporated into the final model. All *P* values were two sided.

RESULTS

Two hundred seventy-four patients with bloodstream infections caused by *A. baumannii* were included in the study: 68 (25%) with bloodstream infection caused by CASR *A. baumannii* and 206 (75%) caused by non-CASR *A. baumannii*. The mean age of the entire cohort was 55 ± 25 years, 133 subjects (48.5%) were males, and 204 (74.5%) were African-Americans. Of the 274 patients with bloodstream infections caused by *A. baumannii*, 78 (28.5%) had multiple positive sets of blood cultures and 196 (71.5%) had a single positive set of blood cultures. There were no significant differences in characteristics or clinical outcomes of patients with multiple positive sets of blood cultures compared to patients with a single set of positive blood cultures.

Comparative analysis of patients with CASR *A. baumannii* and non-CASR *A. baumannii*. The mean age of patients with bloodstream infections caused by CASR *A. baumannii* was 62.5 ± 16 years, and the mean age of patients with bloodstream infection caused by non-CASR *A. baumannii* was 52 ± 25 years (*P* value < 0.001) (Table 1). Patients with CASR *A. baumannii* were predominantly males as opposed to patients with non-CASR *A. baumannii* (57% versus 46%, odds ratio [OR] = 1.60, 95% confidence interval [CI] = 0.92 to 2.78, *P* value = 0.094). Twenty-five percent of patients with bloodstream infections caused by CASR *A. baumannii* had a rapidly fatal condition on admission compared to 8% of patients with bloodstream infections caused by non-CASR *A. baumannii* (OR = 3.70, 95% CI = 1.76 to 7.76, *P* value < 0.001). A greater proportion of patients with CASR *A. baumannii* infections had positive blood cultures after 72 h of admission (hospital-acquired infection) than did patients with non-CASR *A. baumannii* infection (74% versus 48%, OR = 2.97, 95% CI = 1.62 to 5.44, *P* value < 0.001). A greater percentage of patients infected with CASR *A. baumannii* than of patients infected with non-CASR *A. baumannii* were admitted directly to the intensive care unit (ICU) (44% versus 27%, OR = 2.16, 95% CI = 1.22 to 3.83, *P* value = 0.007). Other differences between the two patient groups included mechanical ventilation (57% of patients with CASR *A. baumannii* versus 30% of patients with non-CASR *A. baumannii* [OR = 3, 95% CI = 1.77 to 5.50, *P* value < 0.001]) and ICU admission prior to having a positive *A. baumannii* blood culture (62% versus 35%, OR = 2.94, 95% CI = 1.67 to 5.19, *P* value < 0.001). A history of chronic obstructive pulmonary disease (COPD) was more common in patients with bloodstream infection caused by CASR *A. baumannii* than in patients without CASR *A. baumannii* (29% and 15%, respectively; OR = 2.35, 95% CI = 1.23 to 4.49, *P* value = 0.008). Patients with CASR *A. baumannii* were more likely to have peripheral vascular disease than were patients without CASR *A. baumannii* (37% versus 20%, OR = 2.27, 95% CI = 1.25 to 4.13, *P* value = 0.006).

A much greater proportion of patients with bloodstream infections caused by CASR *A. baumannii* than patients with infections caused by non-CASR *A. baumannii* had prior exposure to several different antimicrobials. Differences in antimicrobial exposures

TABLE 1 Demographics and risk factors in patients with bloodstream infections caused by *Acinetobacter baumannii*

Patient characteristic	Data for subjects with BSI caused by: CASR *A. baumannii* ($n = 68$)	Non-CASR *A. baumannii* ($n = 206$)	OR	95% CI	*P* value
Demographics					
Mean age, yr, ±SD	62.5 ± 16.4	52 ± 25			<0.001
Age of >60 yr, no. (%)	37 (54)	81 (39)	1.84	1.06–3.20	0.029
Sex (male), no. (%)	39 (57)	94 (46)	1.60	0.92–2.78	0.094
Primary infection, no. (%)	67 (98.5)	203 (98.5)	1	0.10–9.87	1
Ethnicity (African American), no. (%)	53 (78)	151 (73)	1.30	0.67–2.46	0.44
Admitted from home, no. (%)	43 (63)	127 (62)	1.07	0.60–1.88	0.81
Comorbidity scores, median (interquartile range)					
Charlson's index on admission	6 (2.5–8.5)	4 (2–7)			0.08
McCabe score on admission	2 (1.5–2)	2 (2–3)			0.0001
Comorbid conditions, no. (%)					
Admitted in rapidly fatal condition	17 (25)	17 (8)	3.70	1.76–7.76	<0.001
Positive *A. baumannii* blood culture after 72 h of admission	50 (74)	99 (48)	2.97	1.62–5.44	<0.001
Directly admitted to ICU	30 (44)	55 (27)	2.16	1.22–3.83	0.007
Central line insertion prior to PBC[a]	11 (16)	49 (24)	0.62	0.30–1.27	0.18
Foley catheter insertion prior to PBC	9 (13)	19 (9)	1.5	0.64–3.50	0.34
Intubation prior to PBC	39 (57)	62 (30)	3	1.77–5.50	<0.001

[a] PBC, positive blood culture.

included exposures to ampicillin-sulbactam (12% versus 4%, *P* value = 0.04), cefepime (54% versus 30%, *P* value < 0.001), linezolid (21% versus 5%, *P* value < 0.001), meropenem (19% versus 5%, *P* value < 0.001), and piperacillin-tazobactam (25% versus 6%, *P* value < 0.001) (Table 2).

In multivariate analysis, variables independently associated with bloodstream infections caused by CASR *A. baumannii* (compared to bloodstream infections caused by non-CASR *A. baumannii*) were determined. Independent predictors included the presence of a rapidly fatal condition at admission (OR = 2.83, 95% CI = 1.27 to 6.32, *P* value = 0.01) and prior use of antimicrobials (OR = 2.83, 95% CI = 1.18 to 6.78, *P* value = 0.02). The final model was controlled for the confounding effect of Charlson's index on admission (score, >3), age (>60 years), ICU admission prior to positive *A. baumannii* culture, and hospital-acquired infection.

TABLE 2 Prior use of antimicrobials (30 days to 3 days prior to culture)

Antimicrobial	No. (%) of patients with BSI caused by: CASR *A. baumannii* ($n = 68$)	Non-CASR *A. baumannii* ($n = 206$)	OR	95% CI	*P* value
Amikacin	2 (3)	0			0.06
Amphotericin B	0	3 (1.5)			1
Ampicillin-sulbactam	8 (12)	9 (4)	3	1.08–7.89	0.04
Aztreonam	3 (4)	7 (3)			0.71
Cefepime	37 (54)	62 (30)	2.77	1.58–4.86	<0.001
Colistin (inhalational)	1 (1.5)	1 (0.5)			0.43
Colistin (intravenous)	3 (4)	3 (1.5)			0.16
Ertapenem	1 (1.5)	0			0.25
Gentamicin	10 (15)	13 (6)	2.56	1.07–6.14	0.03
Imipenem	0	4 (2)			0.57
Linezolid	14 (21)	11 (5)	4.60	2.0–10.7	<0.001
Meropenem	13 (19)	11 (5)	4.20	1.78–9.87	<0.001
Piperacillin-tazobactam	17 (25)	13 (6)	5	2.26–10.85	<0.001
Rifampin	3 (4)	2 (1)			0.1
Tigecycline	6 (9)	6 (3)			0.08
Any antibiotic exposure prior to positive blood culture	67 (89)	126 (63)	4.85	2.21–10.67	<0.0001

Antimicrobial treatment regimens. The median duration from time of specimen collection to time of initiation of appropriate therapy was 2 days in both groups (interquartile ranges [IQRs] of 0 to 3 days in CASR *A. baumannii* versus 1 to 3 days in non-CASR *A. baumannii* bloodstream infections, *P* value = 0.63) (Table 3).

In the empirical phase of therapy, there were significant differences in the use of various antimicrobials between the two groups, i.e., CASR *A. baumannii* and non-CASR *A. baumannii*. Empirical antimicrobials most frequently administered to patients with CASR *A. baumannii* included cefepime (47%), colistin (29%), and meropenem (16%). In patients with BSI caused by non-CASR *A. baumannii*, the most frequently administered antimicrobials included cefepime (48%), ampicillin-sulbactam (20%), ceftriaxone (19%), and meropenem (17%). There were significant differences in the frequency of receipt of two antimicrobial agents, amikacin and colistin (*P* value = 0.025 and *P* value < 0.001, respectively). There was no difference in the appropriateness of empirical therapy between patients with CASR *A. baumannii* and those with non-CASR *A. baumannii* (*P* value = 0.86).

In the consolidative phase of therapy, antimicrobials most frequently administered to patients with CASR *A. baumannii* included colistin (43%), tobramycin (41%), ampicillin-sulbactam (31%), and meropenem (26.5%). In patients with more susceptible strains of *A. baumannii* (i.e., non-CASR strains), the most frequently administered antimicrobials included meropenem (23%), tobramycin (20%), ampicillin-sulbactam (18%), and colistin (16.5%). There were significant differences between the

TABLE 3 Outcomes in patients with bloodstream infections caused by *A. baumannii*

Outcome or length of stay	Data for subjects with BSI caused by: CASR *A. baumannii* ($n = 68$)	Non-CASR *A. baumannii* ($n = 206$)	OR	95% CI	*P* value
Outcome event, no. (%) of patients					
In-hospital mortality	29 (43)	42 (20)	2.90	1.61–5.23	<0.001
Emergency room visits within 60 days of discharge	10 (15)	35 (17)	0.84	0.39–1.80	0.65
Readmission within 60 days of discharge	15 (22)	49 (24)	0.91	0.47–1.75	0.87
Length of stay, days, median (interquartile range)					
Days from initial PBC[a] to initiation of appropriate therapy	2 (0–3)	2 (1–3)			0.63
Length of stay after PBC	9 (3–16.5)	9 (5–16)			0.33

[a] PBC, positive blood culture.

two groups (i.e., CASR and non-CASR) in the frequency of use of various consolidative antimicrobial therapies, including amikacin (*P* value = 0.004), ampicillin-sulbactam (*P* value = 0.024), colistin (*P* value < 0.001), and tobramycin (*P* value = 0.001). Consolidative therapy was appropriate in similar proportions of patients with CASR *A. baumannii* and of patients with non-CASR *A. baumannii* (*P* value = 0.85).

Clinical outcomes of patients with BSI caused by CASR and non-CASR *A. baumannii*. Twenty-nine (43%) patients with BSI caused by CASR *A. baumannii* died in the hospital compared to 42 (20%) of those with BSI caused by non-CASR *A. baumannii* (OR = 2.90, 95% CI = 1.61 to 5.23, *P* value < 0.001) (Table 3). However, in the multivariate model, the relationship between infection caused by CASR *A. baumannii* and increased mortality risk became nonsignificant (OR = 1.15, 95% CI = 0.51 to 2.63, *P* value = 0.74). Independent predictors of in-hospital mortality included admission with a rapidly fatal condition (OR = 53, 95% CI = 13.8 to 204, *P* value < 0.001), Charlson's score of >3 (OR = 3.45, 95% CI = 1.36 to 8.72, *P* value < 0.001), ICU admission prior to positive *A. baumannii* culture (OR = 6.16, 95% CI = 2.52 to 15, *P* value = 0.0001), and hospital-acquired infection (OR = 3.25, 95% CI = 1.14 to 9.22, *P* value = 0.026) (Table 4). The final model was adjusted for age of >60 years, prior use of antimicrobials, and direct admission to ICU.

TABLE 4 Multivariate analysis predicting in-hospital mortality among CASR and non-CASR *A. baumannii* patients[a]

Predictor	Coefficient (β)	SE	OR	95% CI
Age of >60 yr	0.74	0.39	2	0.98–4.51
McCabe score on admission	3.97	0.68	53***	13.8–204
Charlson's score of >3	1.24	0.47	3.45***	1.36–8.72
Prior use of antimicrobials	−0.28	0.52	0.75	0.27–2.09
ICU admission prior to positive *A. baumannii* culture	1.82	0.46	6.16***	2.52–15
Direct admission to ICU	−0.47	0.43	0.62	0.27–1.44
Positive culture after 72 h of admission	1.18	0.53	3.25*	1.14–9.22

[a] The model was adjusted for age of >60 years, prior use of antimicrobials, and direct admission to ICU. Significance: *, *P* value < 0.05; **, *P* value < 0.01; ***, *P* value < 0.001.

Among patients who survived hospitalization, the median durations of hospital stay after the date when blood culture was obtained were similar for patients with BSI caused by CASR *A. baumannii* and patients with BSI caused by non-CASR *A. baumannii* (median stay = 9 days, IQR = 3 to 16.5 days versus median stay = 9 days, IQR = 5 to 16 days; *P* value = 0.33) (Table 3). After discharge, a greater percentage of surviving patients with BSI caused by non-CASR *A. baumannii* (24%) than of subjects with BSI caused by CASR *A. baumannii* (22%) were readmitted to the hospital in the 60 days following discharge (OR = 0.91, 95% CI = 0.47 to 1.75, *P* value = 0.87) (Table 3). Also, a higher percentage of surviving patients with BSI caused by non-CASR *A. baumannii* (17%) than of those with BSI caused by CASR *A. baumannii* (15%) were reevaluated in the emergency department (OR = 0.84, 95% CI = 0.39 to 1.80, *P* value = 0.65) (Table 3). However, these differences were not statistically significant.

DISCUSSION

To our knowledge, this is the largest cohort of patients described with BSI caused by *A. baumannii* and also is the largest cohort of patients with BSI caused by CASR *A. baumannii*. While some published reports have analyzed cohorts of patients with infection caused by MDR *A. baumannii* and others have analyzed patients with either carbapenem- or ampicillin-sulbactam-resistant *A. baumannii*, there have been no prior published reports detailing the epidemiology and outcomes of patients with invasive infection caused by CASR *A. baumannii* (7, 8, 12, 24, 25).

In this study, there were several differences between the patients with BSI caused by CASR *A. baumannii* and those with BSI caused by non-CASR *A. baumannii*. Patients with CASR *A. baumannii* were significantly older than patients with more susceptible strains of *A. baumannii*. Possible reasons for this might have included more frequent health care and antimicrobial exposures among older adults (26). A greater percentage of patients with BSI caused by CASR *A. baumannii* were male in comparison with patients with BSI caused by more susceptible strains of *A. baumannii*, although the reasons for this association remain unclear.

Although the majority of infections were hospital acquired (after 72 h of hospitalization), a greater proportion of CASR *A. baumannii* bloodstream infections than non-CASR *A. baumannii* bloodstream infections were hospital acquired. This finding is not surprising, as *A. baumannii* is primarily a health care-associated pathogen, as are antimicrobial-resistant pathogens, and patients

with MDR pathogens often have had extensive prior health care exposures (2).

Interestingly, patients with CASR *A. baumannii* were more likely to be admitted to the hospital with a rapidly fatal condition and were more likely to be directly admitted to the ICU than were patients with non-CASR *A. baumannii*. This highlights the increased severity of illness among patients with invasive infections caused by CASR *A. baumannii* strains compared to those with more susceptible strains. Another potential explanation might be related to increased virulence of the strains of CASR *A. baumannii*, but virulence factors of study isolates were not analyzed.

The current study demonstrated that, after adjustment for severity of illness, the odds of dying in the hospital were similar for patients with CASR *A. baumannii* and those with non-CASR strains. However, hospital admission with a highly fatal condition was found to predict both BSI with CASR *A. baumannii* and in-hospital mortality. This indicates that, although CASR *A. baumannii* is not independently associated with mortality, patients with BSI caused by this type of pathogen present to the hospital with higher severity of acute illness and are more likely to die. Efforts must be focused on limiting the emergence and spread of CASR *A. baumannii* and also on developing better therapeutic strategies to manage invasive infections caused by CASR *A. baumannii*. Colistin and tigecycline are agents commonly used to treat CASR *A. baumannii*, but the efficacy of these agents and the role of monotherapy compared to combination therapy for treatment of invasive infections caused by CASR *A. baumannii* remain unknown.

This study had several strengths and addresses limitations of prior studies. Prior studies analyzing the impact of MDR *A. baumannii* infection have been limited by several factors. Many studies have analyzed patients with infection caused by MDR *A. baumannii* (8, 27), but none to our knowledge have systematically studied a cohort of patients with invasive infections caused by CASR *A. baumannii*. Additionally, previous studies have included patients who might have been colonized (and not necessarily infected) with *A. baumannii*. Because this study cohort consisted exclusively of patients with BSI caused by *A. baumannii*, only true *A. baumannii* infections were included. This analysis demonstrated similar epidemiology and outcomes among patients with a single set and multiple sets of positive blood cultures, suggesting that patients with a single set of positive blood cultures for *A. baumannii* had infection as opposed to colonization. Because of a low prevalence of MDR *A. baumannii* infections in many hospitals, prior studies have been limited by small study size. The current study is, to our knowledge, the largest published study on the epidemiology and risk factors of BSI caused by *A. baumannii* and includes the largest number of patients with CASR *A. baumannii*.

MDR *A. baumannii* is becoming more common in several parts of the world (28, 29). This study demonstrated that CASR *A. baumannii* has an epidemiology distinct from that of more susceptible *A. baumannii* strains. The results from this study can be used to facilitate preemptive identification and targeting of patients who are at increased risk for CASR *A. baumannii*. This study also demonstrated that patients with BSI caused by CASR *A. baumannii* are more likely to present to the hospital with a rapidly fatal condition and are more likely to die in the hospital. Thus, more experience and better infection control strategies are needed to more effectively limit the spread of CASR *A. baumannii* and to better treat invasive infections caused by this pathogen. Moreover, prospective, controlled studies are needed to examine the impact of BSI caused by CASR *A. baumannii* and to evaluate optimal therapeutic approaches.

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