MAJOR ARTICLE

Acinetobacter Infections and Outcomes at an Academic Medical Center: A Disease of Long-Term Care

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Background. Our study aims to describe the epidemiology, microbial resistance patterns, and clinical outcomes of *Acinetobacter* infections at an academic university hospital. This retrospective study analyzed all inpatient clinical isolates of *Acinetobacter* collected at an academic medical center over 4 years. The data were obtained from an Academic tertiary referral center between January 2008 and December 2011. All consecutive inpatients during the study period who had a clinical culture positive for *Acinetobacter* were included in the study. Patients without medical records available for review or less than 18 years of age were excluded.

Methods. Records were reviewed to determine source of isolation, risk factors for acquisition, drug resistance patterns, and clinical outcomes. Repetitive sequence-based polymerase chain reaction of selected banked isolates was used to determine patterns of clonal spread in and among institutions during periods of higher infection rates.

Results. Four hundred eighty-seven clinical isolates of *Acinetobacter* were found in 212 patients (in 252 admissions). Patients with *Acinetobacter* infections were frequently admitted from healthcare facilities (HCFs) (59%). One hundred eighty-three of 248 (76%) initial isolates tested were resistant to meropenem. One hundred ninety-eight of 249 (79.5%) initial isolates were multidrug resistant (MDR). Factors associated with mortality included bacteremia (odds ratio [OR] = 1.93, $P = .024$), concomitant steroid use (OR = 2.87, $P < .001$), admission from a HCF (OR = 6.34, $P = .004$), and chronic obstructive pulmonary disease (OR = 3.17, $P < .001$).

Conclusions. *Acinetobacter* isolates at our institution are frequently MDR and are more common among those who reside in HCFs. Our findings underline the need for new strategies to prevent and treat this pathogen, including stewardship efforts in long-term care settings.

Keywords. communicable diseases; drug resistance, microbial; long-term care.

Bacteria in the *Acinetobacter* spp are small, aerobic, Gram-negative, nonfermenting coccobacilli that have increased in medical importance over the last 2 decades [1]. This is in part due to their impressive level of antimicrobial resistance. They are resistant to heat and disinfection, and they can spread both by airborne and person-to-person transmission, making it a highly effective nosocomial pathogen [2–6].

Recent epidemiologic studies have uncovered concerning colonization and infection rates of long-term care residents with this pathogen. A recent point prevalence study among acute and long-term care facilities in Maryland discovered that 100% of the long-term care facilities surveyed harbored *Acinetobacter*, compared with 31% of acute care hospitals, and up to 63% of residents of these facilities screened positive for the organism [7]. More concerning is that the endemic strains in these facilities are either carbapenem-resistant *Acinetobacter* (CRAB) or multidrug resistant (MDR) [7]. In

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contrast, facilities in Taiwan and Brazil have reported relatively low rates of CRAB and MDR *Acinetobacter* [8]. These studies emphasize the need for local surveillance of *Acinetobacter* sensitivities, because these data impact empiric antibiotic choices for at-risk patients.

Our institution is a tertiary care university hospital that cares for a sizeable number of patients with a high burden of illness. Many patients are admitted from surrounding nursing homes and long-term care facilities. In this study, we describe the burden of *Acinetobacter* infections at our center including epidemiology, drug resistance, and outcomes. In addition, we sought to determine how related some of these isolates were at a molecular level.

METHODS

This retrospective study was approved by the University of Texas Southwestern (UTSW) Institutional Review Board. We created a list of all *Acinetobacter* isolates (regardless of species) isolated from hospitalized patients (including Emergency Department collections before admission) between January 2008 and December 2011 at any UTSW Medical Center inpatient facility. The facilities included St. Paul University Hospital, a comprehensive tertiary care center, and Zale Lipshy Hospital, which includes a mixture of acute care beds for surgical subspecialty patients, a surgical intensive care unit, and subacute rehabilitation beds. St. Paul Hospital and Zale Liphsy Hospital are across the street from one another and patients are transferred freely between them. Bacteria were identified biochemically using the Siemen's Microscan Walkaway 96 system, which reports *Acinetobacter baumannii* and *Acinetobacter haemolyticus* as a single group. Data were collected for every patient admission in which *Acinetobacter* was isolated. If multiple cultures were positive for *Acinetobacter* during a single admission, the first positive isolate was used for data analysis. Data collected included the following: place of residence before admission (home, outside hospital, other healthcare facilities (HCFs) including skilled nursing facilities, nursing homes, long-term acute care centers, and rehabilitation centers); age, comorbidities, previously described risk factors for *Acinetobacter* infection (neutropenia, chemotherapy, prolonged ventilation, and others); hospitalization in the past 30 days; antibiotic exposure in the past 30 days (≥72 hours in past 30 days, or any exposure within last 14 days); and prior infection or colonization with *Acinetobacter* or other pathogens during a previous hospital stay.

Patients were classified as infection versus colonization using previously described clinical criteria [9]. The definition of colonization was the presence of *Acinetobacter*, without signs or symptoms indicating active infection due to *Acinetobacter*. Infection was defined as the presence of an *Acinetobacter* isolate with clinical evidence of an active, continuing infection attributable to *Acinetobacter*. Clinical evidence included, for example, hyperthermia, leukocytosis, abscess, and nonspecific evidence of multisystem involvement [10]. The updated 2013 National Healthcare Safety Network (NHSN) surveillance definitions were used to classify types of *Acinetobacter* infections [11, 12]. Main NHSN criteria applied included those pertaining to the lower respiratory tract, soft tissue infection, bone, decubitus ulcers, surgical sites, urinary tract, bloodstream, intra-abdominal compartment, or indwelling catheters, with the following caveats: for respiratory infections, if no bronchoscopy was performed, sputum samples were accepted as laboratory evidence of infection if they had >25 leukocytes and <10 epithelial cells/high-powered field. For nonverbal or intubated patients, diagnostic criteria for sepsis were used in place of urinary symptoms if no other source was apparent [13]. In addition, the presence of altered mental status and acute hematuria without other apparent cause were included as signs of catheter-associated urinary tract infection (UTI) per Infectious Diseases Society of America guidelines [14]. In cases in which more than 1 pathogen was isolated, *Acinetobacter* was only considered significant if it was present on more than 1 urine specimen at $>10^5$ colony-forming units and other criteria for UTI were met. In the presence of copathogens or in ambiguous cases, clinical judgment was used to determine whether an infectious syndrome was attributable to *Acinetobacter*. Cases were reviewed by 2 Infectious Diseases Fellows (A. P. and J. T.). In cases of disagreement, *Acinetobacter* infection versus colonization was decided by an Infectious Diseases attending physician (D. E. G.).

For each initial *Acinetobacter* isolate, we collected data on the species, site of infection, time to isolation, hospital floor where patient was located at time of isolation, site of acquisition (hospital acquired if ≥72 hours after admission vs community acquired), presence of copathogens, antimicrobial sensitivities, treatment course, and outcomes. In cases of patients with multiple positive cultures for *Acinetobacter*, only the most invasive isolate was considered. Sources were ranked from most invasive to least invasive in the following order: blood >abdominal fluid >bone >respiratory tract >wound >catheter tip >urine. For instance, if a patient grew *Acinetobacter* from the urine, but also from the blood, the case was categorized as a bacteremia. Multidrug resistant *Acinetobacter* was defined as resistance to 3 or more classes of antimicrobials, and extensively drug-resistant *Acinetobacter* (XDR) was defined as resistance to at least 1 agent in all but 2 or fewer antimicrobial categories, according to consensus definitions [15, 16]. The clinical endpoints assessed were sepsis, cardiac arrest, requirement for intensive care unit (ICU) stay, length of stay in hospital and ICU, and all-cause mortality during hospitalization.

During data analysis, we generated an epidemic curve to determine whether there were peak times of *Acinetobacter* infection (Supplementary Figure 1). A peak was defined as a month with a 1.5-fold or greater incidence in initial *Acinetobacter* isolates over previous months, with the average being 5.1 initial

Table 1. Clinical Features of Patients Admitted From Home vs From a Healthcare Facility[a]

Unique Patients (n = 212)	Admitted From Home (n = 80)	Admitted From Healthcare Facility (n = 132)	Odds Ratio (95% CI)	*P* Value
Demographics and comorbidities				
Male	43 (53.8)	69 (52.3)	—	NS
Age, year, (median, IQR)	56 (41–67)	63 (51–72)	—	.009
Diabetes mellitus	26 (32.5)	63 (47.7)	—	NS
COPD	8 (10.0)	19 (14.4)	—	NS
Malignancy	21 (26.3)	26 (19.7)	—	NS
End-stage renal disease	11 (13.8)	27 (20.5)	—	NS
Transplant recipient	9 (11.3)	4 (3.0)	0.25 (0.07–0.83)	.016
Cirrhosis	2 (2.5)	2 (1.5)	—	NS
HIV	1 (1.3)	1 (0.8)	—	NS
Chronic steroid use	13 (16.3)	14 (10.6)	—	NS
Splenectomy	3 (3.8)	0 (0.0)	—	NS
All admissions (n = 252)	Admitted from home (n = 103)	Admitted from healthcare facility (n = 149)	Odds ratio (95% CI)	*P* value
Infection (vs colonization)	70 (68.0)	95 (63.8)	—	NS
MDR Acinetobacter (vs non-MDR)	63 (61.8)	135 (91.8)	6.96 (3.41–14.21)	<.001
Source of most invasive[b] *Acinetobacter* isolate				
Respiratory	29 (28.2)	46 (30.9)	—	NS
Wound	17 (16.5)	44 (29.5)	2.12 (1.13–3.97)	.018
Urine	30 (29.1)	21 (14.1)	0.40 (0.21–0.75)	.004
Blood	19 (18.1)	21 (14.1)	—	NS
Bone	4 (4.9)	10 (6.7)	—	NS
Abdomen	4 (4.9)	4 (2.7)	—	NS
Catheter tip	0 (0.0)	2 (1.3)	—	NS
Exposures				
Antibiotics in past 30 days	56 (54)	107 (73)	2.25 (1.32–3.82)	.003
Intravascular catheter in past 30 days	27 (26)	110 (74)	7.39 (4.48–14.06)	<.001
UTSW admission in past 30 days	42 (41)	74 (50)	3.5 (1.51–8.16)	NS
Urinary catheter in past 30 days	34 (33)	64 (43)		NS
Mechanical ventilation in past 30 days	6 (6)	53 (36)	8.92 (3.67–21.74)	<.001
PEG tube in past 30 days	10 (10)	44 (30)	3.90 (1.86–8.18)	<.001
ICU stay in past 30 days	12 (26)	31 (55)		NS
Acinetobacter infection in past 30 days	12 (12)	23 (15)		NS
Surgery in past 30 days	10 (10)	24 (16)		NS
Indwelling HD device	2 (2)	24 (16)	9.70 (2.24–42.01)	<.001
Outcomes				
All-cause mortality during admission	6 (5.8)	31 (20.8)	4.25 (1.70–10.60)	0.001
Length of stay after *Acinetobacter* isolation	11 (5–24)	15 (6–34)	—	NS

Abbreviations: CI, confidence interval; COPD, chronic obstructive pulmonary disease; HD, hemodialysis; ICU, intensive care unit; IQR, interquartile range; HIV, human immunodeficiency virus; MDR, multidrug resistant; NS, nonsignificant; PEG, percutaneous endoscopic gastrostomy; UTSW, University of Texas Southwestern.

[a] Data are presented as No. (%) unless otherwise specified.

[b] If the patient grew *Acinetobacter* from more than 1 site during admission, only the most invasive isolate was considered. Sources were ranked from most invasive to least invasive in the following order: blood >abdominal fluid >bone >respiratory tract >wound >catheter tip >urine.

isolates per month. We analyzed these peaks with respect to time and space to identify instances of hypothetical person-to-person transmission. We then selected isolates from our banked specimens for strain typing to determine whether clonal intrahospital spread was occurring during these times of higher-than-average rates of isolation.

Statistics

Descriptive statistics and univariate analyses were used to identify associations between exposures and death. A multivariable generalized estimating equations model for predictors of MDR *Acinetobacter* was generated using a repeated-measures logistic regression method, including nonmissing data on all predictors.

Table 2. Summary of *Acinetobacter* Cultures Obtained From Patients During Hospital Admissions, January 2008–December 2011[a]

Overview of Patients with *Acinetobacter*	N
Total patients	212
Total admissions during which *Acinetobacter* was isolated	252
Total isolates of *Acinetobacter* spp	487
Average *Acinetobacter* isolates per admission	1.9 (range, 1–19)
Acinetobacter baumannii/haemolyticus	242 (96%)
Infection (vs colonization)	165 (65%)
Hospital-acquired *Acinetobacter* infections	75 (30%)
Admissions from another healthcare facility	149 (59%)
Admissions with *Acinetobacter* per 1000 admissions	
2008	2.74
2009	2.88
2010	2.31
2011	2.37

[a] Data are presented as No. (%) unless otherwise specified.

Forty-four variables were entered into the univariate model, including demographic, microbiologic, and treatment variables. To avoid an overspecified model, very strict variable reduction was used. A univariate P value < 0.05 was required for inclusion in the model, and manual backwards selection was used to arrive at a final model [17]. Variables included in the final multivariate model were number of isolates per patient, admission from HCF, chronic obstructive pulmonary disease (COPD), steroids, urinary catheter in past 30 days, and *Acinetobacter* bacteremia. All analyses were performed using SAS 9.3 (SAS Inc., Cary, NC).

DNA Fingerprinting by Repetitive Sequence-Based Polymerase Chain Reaction

To investigate the possibility of clonal relationships between isolates during peak times of *Acinetobacter* infection, repetitive sequence-based polymerase chain reaction (rep-PCR) was performed on selected isolates that were banked as part of infection control practices. This method was used as described by Misbah et al [18], and strains were analyzed using DiversiLab technology. This technology has been widely studied in *Acinetobacter* epidemiology and has been shown to be reliable for typing strains within and among hospitals with good resolution [19–21]. Isolates that clustered at 95% or greater similarity were considered related and were defined as rep-PCR clusters. Strain designations are specific to our institution. The DiversiLab library did not contain international strains for comparison.

RESULTS

Epidemiology

The mean age of the patients was 57 years and 52.8% were male. Patients admitted from HCFs were slightly older, less likely to

Table 3. Antibiogram of *Acinetobacter* Susceptibilities From January 2008 to December 2011

	Colistin	Minocycline	Tobramycin	Amp/ Sulbactam	Tigecycline	Meropenem	Ceftazidime	TMP/ sulfa	Gentamicin	Levooflaxacin	Amikacin	Cefepime	Ceftriaxone	Cefotaxime
Number tested (n)	124	60	250	73	131	248	250	245	248	250	183	240	38	41
Susceptible[a]	119	50	105	25	32	60	58	54	54	47	33	42	4	3
Intermediate	0	8	9	25	0	5	7	0	4	5	32	15	3	3
Resistant	6	2	136	23	89	183	185	191	190	198	118	183	31	35
Percent Susceptible	96	83.3	42	34.2	24.4	24.2	23.2	22	21.8	18.8	18	17.5	10.5	7.3

Abbreviations: Amp, ampicillin; FDA, US Food and Drug Administration; MIC, minimum inhibitory concentration; sulfa, sulfamethoxazole; TMP, trimethoprim.

[a] Sensitivities determined by MicroScan and reported per FDA breakpoints. E test for colistin, minocycline, and tigecycline performed by Mayo Reference Laboratory. For tigecycline, an MIC ≤2 was considered sensitive per the package insert (Pfizer).

be transplant recipients, and exposed to more antibiotics and invasive devices in the preceding 30 days compared with those admitted from home (Table 1). All patients had a high burden of comorbid illnesses including diabetes, end-stage renal disease, and malignancy. The respiratory tract, wounds, and UTI were the most common sites of isolation (Table 1). During admission, 40 patients developed bacteremia. Sources of secondary bacteremia included pneumonia (5 patients), wound infections (3 patients), UTIs (3 patients), line infections (2 patients), and osteomyelitis (1 patient). The remaining 27 patients with bacteremia did not grow *Acinetobacter* from another site. The highest mortality was seen among patients with bacteremia (30%) followed by pneumonia (24%). No patients with only a urinary isolate died.

In the time period specified, *Acinetobacter* was isolated from 212 unique patients during 252 admissions, with a total of 487 *Acinetobacter* isolates among all patients (Table 2). The incidence of *Acinetobacter* cases per 1000 admissions remained fairly constant over the years of the study period (Supplementary 1). According to NHSN criteria, a majority of admissions (165 of 252) represented true *Acinetobacter* infections. The overwhelming majority of initial isolates were *A baumannii* or *A haemolyticus* (96%). A minority of isolates (30%) were hospital acquired, with the majority being present on admission from the community. Considering only the patients with community-acquired *Acinetobacter* isolates (n = 177), 100 (56%) were from HCFs and 77 (44%) were from home. When considering all patients who had *Acinetobacter* isolated during their admission (n = 252), both community- and hospital-acquired isolates, most patients were admitted from HCFs (149 of 252, 59%). The number of total hospital wide *Acinetobacter* isolates per year did not change substantially over this 4-year period (total annual isolates ranged from 128 to145 from 2008 to 2011).

Antibiotic Susceptibility

Analyzing only the first isolate for each admission, we found that the vast majority of our isolates were carbapenem-resistant (n = 183 of 248, 74%) as well as MDR (n = 198 of 249, 79.5%). Forty-six isolates were determined to be XDR as defined under Methods. Most active drugs overall in these isolates were colistin (119 isolates) and tobramycin (105 isolates) (Table 3). By percentage of isolates tested, colistin and minocycline displayed the highest level of in vitro sensitivity at 96% and 83%, respectively. All other drugs tested were not reliably active including trimethoprim-sulfamethoxazole, tigecycline, ampicillin/sulbactam, ceftazidime, and ciprofloxacin. Six of the initial isolates demonstrated colistin resistance, and a majority of isolates tested (98 of 118, 97%) were not susceptible to tigecycline, with a minimum inhibitory concentration (MIC) of ≥1 per EUCAST guidelines [22]. Using a cutoff of MIC ≤2 as suggested by the package insert yielded an 89% resistance rate for tigecycline [23]. Sensitivities for tigecycline, colistin, and minocycline were performed by E-test (Mayo Reference Laboratories), which may overestimate tigecycline resistance [24].

The most important risk factor for infection or colonization with MDR *Acinetobacter* was admission from a HCF. Over 90% (n = 135 of 147) of patients admitted from HCFs had MDR isolates, compared with 62% of patients from home (OR = 6.2, $P < .001$).

Antibiotic Treatment and Outcomes

We analyzed the most common antimicrobials that were used for these infections (data not shown). A total of 16 of 165 infected patients received a combination of antimicrobials predicted to be active by in vitro sensitivities, whereas 60 patients did not receive any active antibiotics. Carbapenems were the most common component of definitive treatment, although 66% of isolates treated with carbapenems were resistant. Thirty-eight of

Table 4. Clinical and Microbiologic Factors Associated With Death Among Patients With *Acinetobacter* Infection (n = 165): Univariate and Multivariate Analysis

			Univariate Analysis		Multivariate Analysis	
Variable	Dead (n = 29)	Alive (n = 136)	Odds Ratio (95% CI)	*P* Value	Adjusted Odds Ratio (95% CI)	*P* Value
Age, per year increase (median, IQR)	62.0 (23.0)	58.5 (21.0)	—	NS	—	NS
Number of isolates, per additional isolate	3.0 (4.0)	1 (1.0)	1.52 (1.18–1.95)	.001	1.53 (1.12–2.10)	.008
Admitted from healthcare facility	23 (79.3)	72 (52.9)	3.41 (1.28–9.08)	.014	6.34 (1.82–22.03)	.004
COPD	9 (31.0)	14 (10.3)	3.92 (1.50–10.27)	.005	3.17 (1.75–5.74)	.0001
Steroids	8 (27.6)	13 (9.6)	3.60–1.33–9.76)	.012	2.87 (1.58–5.20)	.0005
Urinary catheter in past 30 days	6 (20.7)	59 (43.4)	0.34 (0.13–0.91)	.032)	0.56 (0.34–0.93)	.025
Acinetobacter bacteremia	12 (41.4)	28 (20.6)	2.73 (1.15–6.43)	.022	1.93 (1.09–3.41)	.024
No active treatment	6 (20.7)	12 (8.8)	—	NS		
Treatment with >1 active drug	1 (4.2)	16 (13.1)	—	NS		

Abbreviations: CI, confidence interval; CKD, chronic kidney disease; COPD, chronic obstructive pulmonary disease; ESRD, end-stage renal disease; HCF, healthcare facility; ICU, intensive care unit; IQR, interquartile range; MDR, multidrug resistant.

the infected patients were treated with colistin-containing regimens (38 of 165, 23.0%), and 11 of 38 (29.7%) of these died. There was a trend toward lower mortality among infected patients treated with 2 or more active drugs compared with zero or no active drugs (1 of 23 [4.3%] vs 16 of 122 [13.1%]), although this was not statistically significant.

Over half of the patients spent time in the ICU. The mean length of stay was 27 days with a range of 0–324 days. A majority of patients (155 of 252, 61.5%) remained in the hospital 10 days or longer. Twenty-five percent of patients experienced sepsis or septic shock, and 37 patients (17.5%) died during the admission. Risk factors for death for infected patients (univariate and multivariate analysis) are shown in Table 4. When controlling for the other variables, admission from a healthcare facility, COPD, multiple positive cultures for *Acinetobacter*, steroid use, and bacteremia remained significant predictors of death.

Repetitive Sequenced-Based Polymerase Chain Reaction Analysis

For 2 of the peak months of *Acinetobacter* isolation, January and June of 2011, the laboratory had banked *Acinetobacter* isolates. Eighteen of 32 patients had isolates available for typing. Repetitive sequenced-based PCR was used to analyze 22 isolates from these 18 patients. Typing revealed 3 dominant clusters of *Acinetobacter* circulating within our hospital (Figure 1), along with 2 outlier subtypes. Of the 18 patients typed, 11 (61%) arrived from HCFs with their isolates, 6 (33%) acquired the isolates in the hospital, and 1 (6%) came from home. The isolates did not cluster according to facility of origin, and patients from the same facility often had different clones of *Acinetobacter*. Two episodes of possible interhospital spread were noted. In case 1, Patient N had been hospitalized in the ICU for 34 days without infection when Patient J was admitted to the neighboring room from a

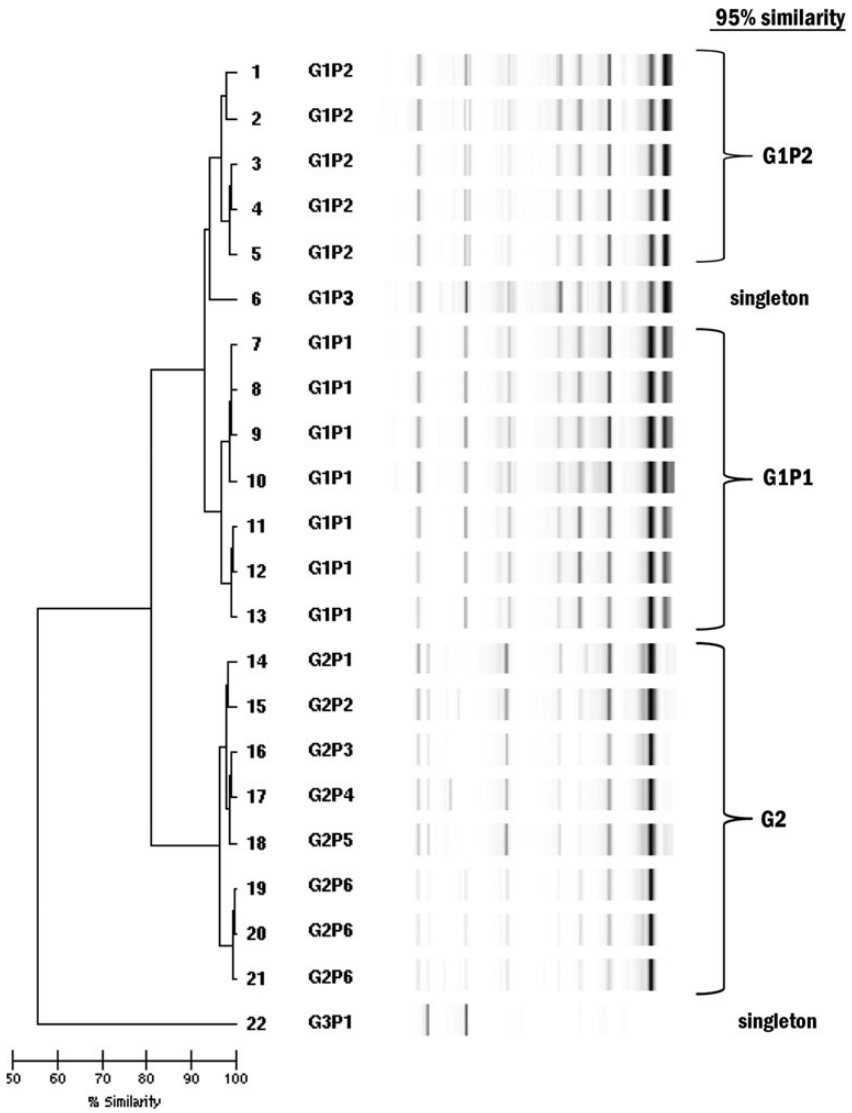

Figure 1. Dendrogram representing relationships between *Acinetobacter* isolates cultured during peak times of hospital infections.

HCF with an *Acinetobacter* infection (cluster G1P2). Ten days after Patient J's admission, Patient N grew cluster G1P2 *Acinetobacter* from the sputum. Patient N went on to die from *Acinetobacter* pneumonia and intra-abdominal infection 17 days later. In a second episode, Patient T was admitted from home to the ICU. Four days later, Patient L was admitted from a nursing facility with a cluster G1P1 *Acinetobacter* growing from sputum. Nine days later, Patient T grew a cluster G1P1 *Acinetobacter* from his blood. The infections were treated successfully. Also of interest was a patient who grew different clones of *Acinetobacter* during his hospital stay. He arrived with a G1P2 *Acinetobacter*, and then 17 days later he also grew a G2P6 *Acinetobacter*. Whether he had different colonizing clones on admission or acquired a new one in the hospital is not known.

DISCUSSION

Our study uncovered several unexpected features of *Acinetobacter* infections in our hospital setting. First, the rates of antibiotic resistance, particularly among residents of HCFs, were alarmingly high. The antibiotics typically selected for empiric coverage for serious infections among those with healthcare exposure, namely extended-spectrum penicillins and carbapenems, did not have reliable activity against the *Acinetobacter* strains seen in this population.

Second, patients with *Acinetobacter* in our study had high levels of comorbid illness and protracted hospital stays, but they had relatively low mortality compared with other studies (Figure 2). Predictors of mortality in our cohort were similar to what has been seen in previous studies (lung disease, steroid use), but after controlling for other exposures, residence in a HCF was an independent predictor of death in our population. It is interesting to note that MDR infection was not associated with death

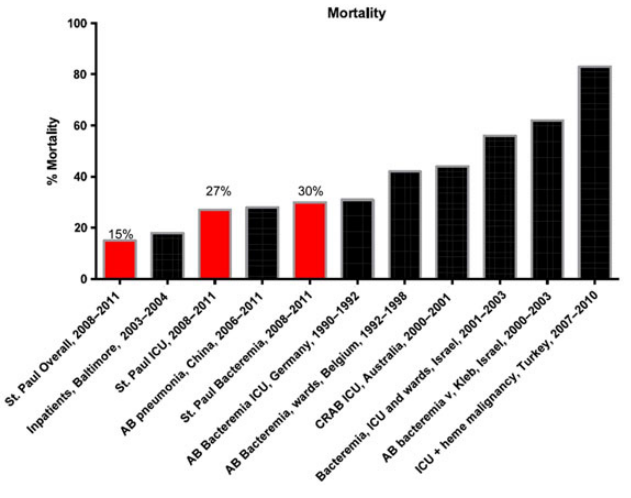

Figure 2. Comparative mortality of *Acinetobacter* infections in various settings worldwide.

in the multivariate analysis. In our cohort, the mortality of *Acinetobacter* bacteremia was 30%, compared with 50%–60% in earlier studies [25, 26]. Improvements in mortality may be due to advancements in the care of critically ill patients, rather than in antimicrobials, because no new drugs have yet become available for this pathogen since tigecycline in 2005.

Another surprising finding was that a majority of patients arrived with their *Acinetobacter* isolate either from home or a long-term care facility. Less than 30% were hospital acquired, which means that strategies for prevention may need to focus on prehospital risk factors, such as reducing inappropriate antibiotic use in the community, avoiding unnecessary catheter placement, and shortening hospital stays whenever possible. Unfortunately, these strategies mandate interinstitutional collaborations, which can be difficult to implement.

From the rep-PCR analysis, we were able to identify 3 clusters of *Acinetobacter* isolates and 2 outliers. A recent study of the epidemiology of *Acinetobacter* in Iran using rep-PCR as well as sequencing of 70 isolates demonstrated 5–7 clusters per hospital, whereas a hospital in Helsinki found 9 clusters among 55 isolates [27]. In comparison, our hospital demonstrated less diversity with only 3 clusters, which may suggest a high degree of clonal sharing among a small number of facilities, or a recent start to our epidemic relative to other cities.

Treatment of *Acinetobacter* in the setting of high institutional rates of carbapenem resistance presents the clinician with an intractable problem. Although colistin remains the most active antibiotic for *Acinetobacter* in vitro, physicians hesitate to use it for empiric therapy given its nephrotoxicity and lack of mortality benefit in retrospective studies. When evaluating the impact of drugs predicted to be active using in vitro susceptibility data, there was a suggestion that use of 2 or more active drugs may be of benefit, although the sample size was too small to show significance. This finding highlights the need for larger prospective trials of combination therapy.

As a retrospective and nonrandomized study, this observational dataset cannot be used to make firm connections between treatments and outcomes. The observed event rate was too small to generate a robust multivariate model of predictors of death. In particular, the impact of *Acinetobacter* on mortality could not be reliably adjusted for the overall level of illness using the APACHE or Charlson score, because not enough variables were collected to perform these calculations. In addition, we did not have access to records outside of our hospital system, so exposures and admissions at outside facilities in the past 30 days may be incomplete, and the distinctions between residents of HCFs and home may be overestimated.

CONCLUSIONS

Our study underlines the success of *Acinetobacter* as a nosocomial pathogen. Patients with extended stays in hospitals and HCFs

can serve as a reservoir for MDR *Acinetobacter* dissemination, which may be lethal in some cases. In our institution, rising drug resistance has highlighted the need for integrated surveillance, stewardship, and infection control efforts between hospitals and feeder facilities [7].

Supplementary Material

Supplementary material is available online at *Open Forum Infectious Diseases* (http://OpenForumInfectiousDiseases.oxfordjournals.org/).

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Potential conflicts of interest. All authors: No reported conflicts.

All authors have submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest.

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