CONCISE COMMUNICATION

Multidrug Resistant *Acinetobacter baumanii*: A 15-Year Trend Analysis

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From 2000 to 2009, rates of multidrug-resistant *Acinetobacter baumanii* increased 10-fold to 0.2 per 1,000 patient days. From 2010 to 2015, however, rates markedly declined and have stayed below 0.05 per 1,000 patient days. Herein, we present a 15-year trend analysis and discuss interventions that may have led to the decline.

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By 1990, *Acinetobacter baumanii* was recognized as a healthcare-associated pathogen of increasing significance, with several hospital outbreaks reported elsewhere.[1] Though no clear source of *A. baumanii* was found in many outbreaks, investigators implicated the hospital environment as a potential reservoir, and *A. baumanii* was found to persist for up to 4 weeks on dry surfaces.[2] By 2003, *A. baumanii* caused 7% of pneumonia in intensive care units (ICUs) and large hospitals, reaching endemic status. Control measures such as hand hygiene, environmental cleaning, contact isolation, targeted screening, and cohorting were implemented.[3,4] By 2007, *A. baumanii* was the ninth most common pathogen overall for device-associated and surgical site infections (SSIs) among facilities reporting to the National Healthcare Safety Network (NHSN), and 30% of isolates were resistant to carbapenems.[5]

By 2010, *A. baumanii* ranked 14th overall for device-associated and SSIs among facilities reporting to the NHSN, and >60% of isolates were resistant to carbapenems.[6] In 2013, the Centers for Disease Control and Prevention (CDC) categorized *A. baumanii* as a serious threat and reported a national total of 12,000 infections annually, of which 7,300 were multidrug resistant.[7] By 2014, however, *A. baumanii* no longer ranked among the top 15 pathogens, and the magnitude of resistance was declining.[8] The reasons for this marked decline are unclear.

The purpose of this study was to determine institutional temporal trends for both community- and hospital-onset infections (CO and HO, respectively) and to examine the reasons for the trends.

METHODS

We performed a retrospective analysis of patients with multidrug-resistant *A. baumanii* (MDR-Ab) who presented to 1 of 2 hospitals in our health system from 2000 to 2015. MDR-Ab was defined according to NHSN's Lab ID Event Reporting protocol and was included any clinical *Acinetobacter* spp testing nonsusceptible to ≥1 agent in at least 3 of 6 antimicrobial classes.[9] Hospital A is a tertiary-care academic teaching facility with 540 beds. Hospital B is a community hospital with 265 beds.

In phase 1 of our analysis, we calculated the health system-level CO and HO annual infection rates using the incident MDR-Ab Lab ID event for each patient. We defined CO as any specimen collected ≤3 days after admission and HO as any specimen collected >3 days after admission (Figure 1).[9] A visual assessment of the rate trend directed phase 2 of our analysis, in which we created CO and HO rate-based subgroups and examined infection rates, patient characteristics (Table 1), and hospital infection prevention interventions (Figure 1) in those groups. Patient characteristics included well-known risk factors for infection plus those defined by prior internal analyses (eg, referral by skilled nursing facilities (SNFs, data not shown).[2] Patient characteristics were summarized using the mean (min, max) for continuous variables and frequency (percentages) for categorical variables. Comparisons between CO and HO subgroups were carried out using *t* tests for continuous measures and χ^2 or the Fisher exact test for categorical measures. Statistical analyses were conducted using SPSS version 24 software (IBM, Armonk, NY). *P* values < .05 were considered statistically significant.

The year of implementation for infection prevention interventions (eg, hand hygiene campaign, chlorhexidine gluconate (CHG) bathing, ultraviolet-C (UV-C) disinfection) were added to Figure 1.

RESULTS

From 2000 to 2015, 568 patients had positive MDR-Ab cultures using the NHSN surveillance definition,[9] and 100% were resistant to meropenem. Overall, 258 patients had CO infections (45.4%) and 310 had HO infections (55.6%). Moreover, 64% of specimens were from sputum, 14% were from skin and soft tissue, and 8% were from blood. For both CO and HO groups, rates increased roughly 10-fold over the first half of the 15-year period. The HO rate peaked in 2008; the CO rate peaked in 2009. For both, rates were steady until 2011, when they began to decline continuously. Cases used to calculate annual rates were temporally spaced and were not part of any obvious outbreaks. There was a clear change from 2008 to 2009 where the rates of both CO and HO infections ceased to further increase, so we conducted further analysis using 2009 as a cut-off to create subgroups (ie, cultures up to and including 2009 [PRE] and after 2009 [POST]).

Overall, patients were >60 years old (64.2 ±20.2), male (57%), had neurologic conditions (46%), were diabetic (34%), presented to the emergency department (ED) from SNFs (42%), and had tracheostomies (52%) and percutaneous endoscopic gastronomy (PEG) tubes (49%). We compared the

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TABLE 1. Characteristics of Patients with Community- and Hospital-Onset MDR *A. baumanii* Clinical Isolates, Comparing Two Periods (2000–2009 and 2010–2015)

	Community Onset			Hospital Onset		
Variable	PRE-2009 (N = 100)	POST-2009 (N = 158)	*P* Value	PRE-2009 (N = 164)	POST-2009 (N = 146)	*P* Value
Age, mean y (range)	66.2 (1.22–94.6)	66.6 (0.46–96.8)	.874	60.5 (0.02–95.8)	64.5 (0.65–94.1)	.08
Male sex, no. (%)	58 (58)	92 (62)	.519	90 (54.9)	83 (56.8)	.727
Entry point, no. (%)						
Direct admission	38 (38)	38 (24)	.017	81 (49.4)	50 (34.2)	.007
Emergency department	62 (62)	120 (76)		…	…	
Referral, no. (%)						
Home	15 (15)	34 (21.5)	.108	58 (35.4)	39 (26.7)	<.001
Hospital	31 (31)	32 (20.3)		73 (44.5)	35 (24)	
Skilled nursing facility	54 (54)	92 (58.2)		22 (20.1)	72 (49.3)	
Comorbidities, no. (%)						
Cancer	17 (17)	35 (22.2)	.315	28 (17.1)	32 (21.9)	.811
Cardiovascular	14 (14)	13 (8.2)	.140	74 (44.5)	35 (24)	.554
Decubitus ulcer	26 (26)	47 (29.7)	.515	31 (18.9)	30 (20.5)	.716
Diabetes	40 (40)	62 (39)	.903	46 (27)	47 (32.2)	.427
ESRD on dialysis	0 (0)	1 (0.6)	>.99	7 (4.3)	9 (6.2)	.451
Lung disease (eg, COPD)	16 (16)	24 (15.2)	.861	25 (15.2)	30 (20.5)	.222
Neurologic (eg, Parkinson's)	57 (57)	90 (57)	.995	58 (35.4)	58 (39.7)	.428
Paraplegic or quadriplegic	8 (8)	14 (8.9)	.809	10 (6.1)	15 (10.3)	.981
Psychiatric (eg, MDD)	4 (4)	2 (1.3)	.211	11 (6.7)	16 (11)	.185
Transplant	10 (10)	9 (6)	.197	10 (6.1)	15 (10.3)	.178
Hospital days to onset, mean (min, max)	…	…	…	26.6 (4, 240)	18.3 (4, 91)	.03
Specimen type, no. (%)						
Blood	10 (10)	10 (6.3)	.306	18 (11)	8 (5.5)	.210
Drainage, unspecified	3 (3)	8 (5.1)		6 (3.7)	5 (3.4)	
Skin/soft tissue	13 (13)	30 (19)		20 (12.2)	17 (11.6)	
Sputum	67 (67)	95 (60)		102 (62.2)	100 (68.5)	
Urine	4 (4)	13 (8.2)		14 (8.5)	16 (11)	
Invasive devices, no. (%)						
Central line	…	…	…	154 (93.9)	118 (80.8)	<.001
Endotracheal tube	…	…	…	94 (57.3)	56 (38.4)	<.001
PEG tube	51 (51)	89 (56.3)	.403	63 (38.4)	77 (52.7)	.011
Tracheostomy	54 (54)	83 (52.5)	.818	87 (53)	73 (50)	.592
Urinary catheter, indwelling	4 (4)	8 (5.1)	.771	43 (26.2)	52 (35.6)	.073
ICU stay, no. (%)	…	…	…	139 (84.8)	112 (76.7)	.072
Surgery, no. (%)	…	…	…	68 (41.5)	40 (27.4)	.010
Mechanical ventilation, no. (%)	…	…	…	134 (81.7)	111 (76)	.220
Time to discharge, mean (range)	13 (0–141)	12 (0–175)	.085	20 (0–384)	15 (0–1,023)	.142
Discharge status, no. (%)						
Alive	76 (76)	121 (76.6)	.915	102 (62.2)	96 (65.8)	.515
Died	24 (24)	37 (23.4)		62 (37.8)	50 (34.2)	

NOTE. ESRD, end-stage renal disease; COPD, chronic obstructive pulmonary disease; MDD, major depressive disorder; PEG, percutaneous endoscopic gastronomy; ICU, intensive care unit.

patient characteristics of the 2 CO and HO subgroups, PRE and POST (Table 1). PRE and POST patients in the CO group were similar except more came through the ED in the POST period (62% vs 76%; $P = .017$). The in-hospital mortality for CO patients was 24%. Comparing PRE and POST patients in the HO group, the percentage of patients from SNFs more than doubled (20% vs 49%; $P < .001$), more had PEG tubes (38% vs 53%; $P = .011$), the mean number of days to onset decreased by 31% ($P = .03$), fewer had endotracheal tubes (57% vs 38%; $P < .001$) and central lines (94% vs 80%; $P < .001$), and there was less exposure to surgery (41% vs 27%; $P = .01$). The in-hospital mortality for HO patients was 36%.

The years of implementation for infection prevention interventions are shown in Figure 1. Active surveillance testing (AST) and contact precautions for MDR-Ab began before 2005. In 2008, hospital A moved to a new

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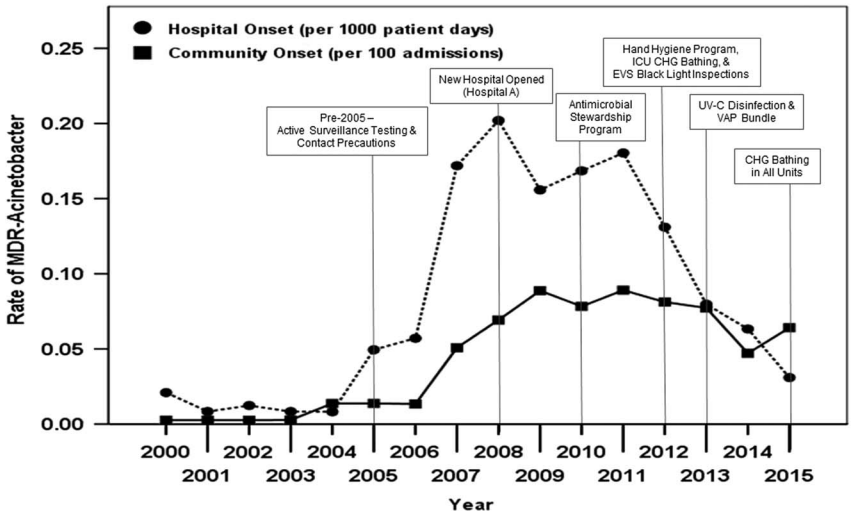

FIGURE 1. Infection prevention interventions. NOTE. ICU, intensive care unit; CHG, chlorhexidine gluconate; UV-C, ultraviolet-C; VAP, ventilator-associated pneumonia.

building with all private rooms. In 2010, the antimicrobial stewardship program formed. Through various interventions, including prospective audit and feedback, the team targeted the unnecessary use of multiple broad-spectrum antibiotics, including carbapenems. In 2012, daily chlorhexidine gluconate (CHG) bathing began in ICUs; the institutional hand hygiene program expanded to include more observations, feedback, and direct education; and blacklight inspections for surface disinfection were adopted by environmental services personnel. In 2013, UV-C disinfection was added to the discharge cleaning protocol, and the ventilator-associated pneumonia (VAP) bundle was fully implemented. By 2015, CHG bathing expanded to all hospital units.

DISCUSSION

From 2000 to 2009, our health system witnessed a sharp increase in both CO and HO MDR-Ab rates. Following 2009, CO and HO rates both became steady and then declined and have remained low. We hypothesize that this overall trend is the due to a combination of institutional factors, community trends, and the emergence of more common pathogens *Clostridium difficile* and carbapenem-resistant Enterobacteriaceae (CRE).

Possibly, moving to a new hospital with all private rooms in 2008 was our initial hard-hitting defense against MDR-Ab. At this point, the HO rate stopped increasing. This move, followed by more robust surface disinfection, antimicrobial stewardship, and the adoption of the VAP bundle, likely drove rates down. Notably, in the POST period, HO patients had fewer endotracheal tubes and central lines, which certainly could result in fewer infections. Based on these data, no one specific intervention appears to have led to the decline.

Our key limitations were ecological study design and, thus, the inability to establish causation. We did not identify the reason for the sharp increase; therefore, we do not know whether a major system stressor was applied and then removed. Also, our data are limited to a single health system, and these findings may not be similar in other regions.

In summary, we performed a 15-year retrospective review of CO and HO MDR-Ab rates across our health system. We observed a sharp increase followed by a steady period, then a decrease in HO MDR-Ab coupled with a similar broader community trend. We have not identified the primary drivers for rate trends in either direction. As postulated by others,[10] there does not appear to be a single dominant intervention but rather a number of infection prevention strategies and antimicrobial stewardship practices that have contributed to the decline of MDR-Ab in our health system.

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