Acinetobacter baumannii Infections in a Tertiary Care Hospital in Mexico over the Past 13 Years

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Key Words

Resistance patterns · *Acinetobacter baumannii* · Antimicrobial susceptibility

Abstract

Background: *Acinetobacter baumannii* has evolved from an opportunistic pathogen into a common and persistent nosocomial bacterium capable of causing severe infections during endemic and epidemic periods. **Methods:** The study period extended from January 1999 to December 2011 and involved patients hospitalized at the Hospital Civil de Guadalajara, Fray Antonio Alcalde, Jalisco, Mexico. From each patient, a single isolate was obtained, and a total of 3,680 unique isolates were collected. Susceptibility tests were performed according to the guidelines of the Clinical and Laboratory Standards Institute. **Results:** *A. baumannii* has disseminated throughout the Hospital Civil de Guadalajara, Fray Antonio Alcalde, since 1999. *A. baumannii* isolates obtained from patients treated in the adult intensive care unit represent the majority of the isolates that have been collected. In addition, *A. baumannii* was isolated from the adult

neurosurgical ward and the adult internal medicine ward, and these isolates were frequently obtained from secretions. A persistent decrease in the susceptibility of *A. baumannii* isolates to meropenem (92% in 1999 to 12% in 2011), imipenem and amikacin has been observed. **Conclusions:** *A. baumannii* became an endemic nosocomial pathogen during the study period at the Hospital Civil de Guadalajara, Fray Antonio Alcalde, and has exhibited a persistent decrease in susceptibility to all categories of antimicrobial agents over the past 13 years.

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Introduction

Acinetobacter baumannii is a nosocomial pathogen found worldwide that is responsible for a diverse set of serious infections that include bacteremia, ventilator-associated pneumonia, postsurgical meningitis and skin and skin structure infections [1–3]. Moreover, *A. baumannii* has evolved from a nosocomial bacterium that primarily affects immunocompromised patients in hos-

pital intensive care units (ICUs) into a pathogen capable of infecting children, neonates, patients with diabetes mellitus and older adults in community hospitals and nursing homes [4, 5]. Furthermore, *A. baumannii* can also spread from adult patients in the ICU to other hospital wards via direct person-to-person (contact) transmission involving an infected patient, a staff member or noninfected patients across a hospital system. As a result, sequential outbreaks, clonal spread, endemicity and dispersal between hospitals and cities can occur [6–8].

The aim of this study is to report the incidence of *A. baumannii* in a tertiary care teaching hospital over the past 13 years, the distribution of *A. baumannii* within the hospital and selected microbiological and molecular characteristics of the isolates identified.

Methods

Setting and Study Period

The study period extended from January 1999 to December 2011 and involved surveillance of *A. baumannii* isolates obtained from patients treated at the Hospital Civil de Guadalajara, Fray Antonio Alcalde, a 1,000-bed, tertiary care teaching hospital. The hospital provides services to the Jalisco state and its capital, Guadalajara, the second largest city in western Mexico, as well as to surrounding states. The hospital consists of adult and pediatric beds distributed among 31 wards in three connected buildings. All of the pediatric units are located in one building, while adult services are distributed between the two other buildings. There is one general adult ICU, two neonatal ICUs and an Obstetrics and Gynecology Service. In 2011, there were 651 patients discharged from the adult ICU, 1,692 patients from the medicine ward, 7,002 from the surgery ward, 2,040 from the neurosurgery ward and 7,398 from the pediatric ward.

Study Design

The study includes a retrospective review of our hospital clinical microbiology database for all *A. baumannii* isolates recovered during 13 years. All duplicate *A. baumannii* isolates were eliminated to ensure that only the first isolate, irrespective of the clinical source, was included in the analysis. All isolates from secondary cases and clusters were also entered in the database. Data regarding hospital areas where *A. baumannii* was recovered, isolation sites and *A. baumannii* resistance patterns were recorded. The dataset contained no patient's demographic information. The Centers for Disease Control and Prevention definition for nosocomial infections was used [9]. Whonet 5.6 (World Health Organization, Geneva, Switzerland) software was used for descriptive statistics. Linear trend analysis for seasonal trend over time was performed using SPSS statistical software, version 17.0.

Bacterial Collection and Microbiological Methods

All *A. baumannii* strains collected between January 1999 and December 2011 were available, and these included 3,680 unique

isolates. Originally, a single isolate for each patient was obtained from each culture-positive collection site. Species identification and antibiotic susceptibility for each isolate were analyzed by Sensititre ARIS® 2X (TREK Diagnostic Systems Inc., Westlake, Ohio, USA). The antimicrobial agents tested included amikacin, ceftazidime, imipenem and meropenem. Classification of isolates as susceptible or resistant followed Clinical and Laboratory Standards Institute guidelines. *Escherichia coli* ATCC 25922 and *Pseudomonas aeruginosa* ATCC 27853 were employed for quality control tests for susceptibility test reagents and methods. A subset of 98 isolates (from 2006–2007) was also analyzed using the commercial assay API 20 NE (Biomerieux®), in addition to biochemical tests including catalase, oxidase and malonate and growth at 37, 40 and 42°C. All of the strains were identified as belonging to the *A. baumannii-calcoaceticus* complex. The identification of *A. baumannii* was confirmed by determining the presence of the *bla*_{OXA-51} gene by PCR [Alcantar-Curiel, unpubl. data, 10].

Analysis of Chromosomal DNA by Pulsed Field Gel Electrophoresis

Genomic DNA was isolated from overnight cultures and prepared as described previously [11]. Briefly, genomic DNA was digested with *Apa* I (New England Biolabs, Frankfurt, Germany, or Promega, Madison, Wisc., USA), and the resulting restriction digest products were resolved by pulsed field gel electrophoresis (PFGE) using a contoured clamped homogeneous electric field Gene Path system (Bio-Rad). Restriction fragment analysis was interpreted according to criteria suggested by Tenover et al. [12]. Accordingly, a difference of 6 bands or fewer was used to define epidemiological relatedness [11].

Production of Metallo- β -Lactamases

Detection by E-Tests. To screen for the presence of metallo- β -lactamases (MBLs), E-test strips (AB Biodisk, Solna, Sweden) were used according to the manufacturer's instructions. A suspension of each isolate in sterile saline solution was adjusted to the density of a 0.5 McFarland standard, then swabbed onto Müller-Hinton agar plates in three directions to ensure uniform growth. Once the agar surface was completely dry, an E-test strip containing imipenem and imipenem plus EDTA was applied to each plate with sterile forceps. The plates were subsequently incubated at 35°C for 48 h. A reduction in the minimum inhibitory concentration of imipenem by ≥ 3 twofold dilutions in the presence of EDTA was interpreted as an indicator of MBL production. Equally, the presence of a 'phantom' zone between the two gradient sections or deformation of the imipenem ellipses was also considered indicative of MBL production [13].

Double-Disk Sensitivity Test. Two 10- μ g imipenem and meropenem disks (Becton Dickinson) were placed on a plate, and appropriate amounts of an EDTA solution were added to one of them to obtain the desired concentration. The inhibition zones of the imipenem/meropenem and imipenem/meropenem-EDTA disks were compared after 16–18 h of incubation in air at 35°C. The inhibition zone diameter for MBL-positive *Acinetobacter* spp. increased by 6–13 mm, while that for MBL-negative isolates increased by 1–7 mm [14].

Table 1. Distribution of *A. baumannii* isolates by hospital ward

Ward	Year											
	2000 (n = 105)	2001 (n = 106)	2002 (n = 201)	2003 (n = 166)	2004 (n = 147)	2005 (n = 244)	2006 (n = 288)	2007 (n = 411)	2008 (n = 472)	2009 (n = 379)	2010 (n = 537)	2011 (n = 606)
ICU	20	31.1	29.3	25.3	20.4	22.9	24.6	30.6	36.2	34	28.4	22.4
Medicine	23.8	31.1	16.9	21.6	23.1	16.8	19	27.4	21.3	28.2	28.3	26.4
Surgery	7.6	2.8	23.3	27.7	23.1	22.5	26.3	12.6	23.5	17.9	13.9	18.8
Neurosurgery	32.3	20.7	18.4	19.8	17.6	24.1	19	17.2	9.1	13.4	15.8	19.8
Pediatric	16.1	14.1	11.9	5.4	15.6	13.5	10.7	11.9	9.7	6.3	13.4	12.5

Results are shown as percentages. In 1999, a total of 18 isolates were found, with the following distribution: Surgery, 6; Pediatric, 5; Medicine, 4; Neurosurgery, 2; ICU, 1.

Table 2. Distribution of *A. baumannii* isolates by isolation site

Isolation site	Year											
	2000 (n = 105)	2001 (n = 106)	2002 (n = 201)	2003 (n = 166)	2004 (n = 147)	2005 (n = 244)	2006 (n = 288)	2007 (n = 411)	2008 (n = 472)	2009 (n = 379)	2010 (n = 537)	2011 (n = 606)
Secretions	59	53.7	49.7	59.6	52.3	52.4	54.5	56.6	52.7	51.1	50.4	38.9
Vascular catheter	12.3	15	13.4	13.2	14.9	15.5	13.1	10.7	15.4	13.9	16.7	12
Blood	4.7	15	16.4	9	10.2	7.7	6.5	8.5	9.7	10.5	7.6	8.5
Respiratory sources	13.3	4.7	6.4	7.2	9.5	11	18.7	17.2	15	13.9	11.7	30.5
Urine	7.6	4.7	8.4	6.6	7.4	7.3	2.4	4.8	4	5.8	9.4	6.9
Cerebral spinal fluid	0	6.6	3.4	2.4	4.7	1.2	2.7	0.4	2.3	1.8	3.1	1.8
Pleural fluid	2.8	0	1.9	1.8	0.6	4.5	1.7	1.4	0.6	2.6	0.7	1.1

Results are shown as percentages. Respiratory sources include tracheal and bronchial specimens. In 1999, a total of 18 isolates were found, with the following distribution: secretions, 9; respiratory sources, 3; blood, 3; urine, 3.

Results

Evolution of A. baumannii

The first *A. baumannii* isolate detected at the Hospital Civil de Guadalajara (Jalisco, Mexico) was obtained from a patient treated in the adult ICU. From this ward, *A. baumannii* was found to spread to the adult medicine ward and the neurosurgical ward, then to the rest of the hospital. In 1999, there were 18 *A. baumannii* isolates identified, then between 2000 and 2001, this number increased to >100. In 2010, >500 *A. baumannii* isolates were collected. The adult ICU had the highest percentage of isolates in the years 2001, 2002, 2005 and 2007–2010. The second highest percentage of isolates was detected in the surgical ward in the years 2003, 2004 and 2006 (table 1). Between 2000 and 2001, *A. baumannii* isolates were frequently found in the adult neurosurgical service. More-

over, during the last 10 years of observation, this ward was the fourth most common hospital area for *A. baumannii* isolation (table 1). In contrast, the percentage of *A. baumannii* isolates obtained from the pediatric ward remained stable (e.g. <15%) throughout the observation period (table 1). In 2011, the prevalence of *A. baumannii* isolates was 20.8% for the ICU, 9.4% for the medicine ward, 1.6% for the surgery ward, 5.8% for the neurosurgery ward and 1% for the pediatric ward.

A. baumannii Isolation Sources, Ranking and Seasonal Distribution

Since 2000, *A. baumannii* has frequently been isolated from secretions, with this site accounting for >50% of all isolation sites monitored through 2011 (table 2). Isolation of *A. baumannii* from vascular catheters was the second most common site, but was surpassed by isolates from

Fig. 1. Seasonal distribution of *A. baumannii*. Linear trend analysis was not significant.

Table 3. The 5 most frequently isolated bacterial pathogens over time

Isolation rank	Year				
	2002	2005	2008	2011	
1	<i>Escherichia coli</i>	<i>Escherichia coli</i>	<i>Escherichia coli</i>	<i>Escherichia coli</i>	
2	<i>Staphylococcus epidermidis</i>	<i>Staphylococcus aureus</i>	<i>Staphylococcus aureus</i>	<i>Acinetobacter baumannii</i>	
3	<i>Staphylococcus aureus</i>	<i>Staphylococcus epidermidis</i>	<i>Staphylococcus epidermidis</i>	<i>Staphylococcus aureus</i>	
4	<i>Klebsiella pneumoniae</i>	<i>Pseudomonas aeruginosa</i>	<i>Pseudomonas aeruginosa</i>	<i>Pseudomonas aeruginosa</i>	
5	<i>Pseudomonas aeruginosa</i>	<i>Klebsiella pneumoniae</i>	<i>Acinetobacter baumannii</i>	<i>Staphylococcus epidermidis</i>	

respiratory sources in 2000, 2006, 2007 and 2011 (table 2). The fourth and fifth most common isolation sites were blood and urine, respectively, with a small number of isolates obtained from cerebral spinal fluid and pleural fluid (table 2). In 2007, *A. baumannii* became, for the first time, 1 of the 5 most frequent bacterial pathogens isolated. In 2009, it was the fourth most frequent bacterial pathogen, displacing other nosocomial pathogens such as *P. aeruginosa* and becoming the second most common Gram-negative pathogen after *E. coli* in 2011 (table 3). In addition, *A. baumannii* was more frequently isolated during the dry, hot months between April and June and during the hot, rainy season between July and September, although this trend was not statistically significant (fig. 1).

PFGE Analysis and Susceptibility of *A. baumannii* Isolates to Selected Antibiotics

Thirteen types of *A. baumannii* clones were identified from the PFGE patterns observed. Multiple *A. baumannii* clones were present in some samples, and clones L and D were predominant among the ICU isolates. Clones L and D were also found to be the cause of nosocomial outbreaks (fig. 2). During the study period, a persistent decrease in the susceptibility of *A. baumannii* isolates to meropenem, imipenem and amikacin was observed (table 4). During 2006–2007, there was an increase in the percentage of isolates susceptible to cefepime, imipenem and meropenem, probably related to predominant clones with different sensitivities. However, a subset of *A. baumannii* clones were susceptible to amikacin, imipenem and me-

Fig. 2. Dendrogram obtained by PFGE analysis. PFGE was run in 65 selected isolates, and 12 clones were detected (A–L). Representative patterns of the 12 clones are shown.

Table 4. Susceptibility of *A. baumannii* to selected antibiotics

Antibiotic	Year												
	1999 (n = 18)	2000 (n = 105)	2001 (n = 106)	2002 (n = 201)	2003 (n = 166)	2004 (n = 147)	2005 (n = 244)	2006 (n = 288)	2007 (n = 411)	2008 (n = 472)	2009 (n = 379)	2010 (n = 537)	2011 (n = 606)
Amikacin	33.3	31.7	39.6	22.4	29.6	25.9	23	21.5	19	41.5	35.9	15.5	20.8
Cefepime	45.5	51.6	64.2	66.2	70.7	68	47.9	72	74.6	46.3	38	35	26.2
Ceftazidime	11.1	21.2	18.3	11.4	15.1	25.9	16.5	21.8	26.3	15.9	13.5	5.6	8.6
Ciprofloxacin	11.1	22.5	28.3	17.5	15.1	23.1	14	17.2	13.7	12.3	12.1	4.5	8.1
Imipenem	88.2	86.5	82.1	80.5	90	87.8	80.9	84.2	73.7	44.1	21.1	8.8	13.9
Meropenem	91.7	63.2	80.2	73.5	74.1	80	58.5	70.5	60.4	41.2	19.8	9.9	11.8

Results are shown as percentages.

roopenem. The most predominant clone, L, was characterized to be 47% resistant and 53% intermediate to meropenem, and to be 71% susceptible and 29% intermediate to imipenem. In contrast, the second most frequent clone type, clone D, was 100% resistant to meropenem and 91% resistant to imipenem (table 5).

MBL producers were also screened using the double-disk diffusion method with meropenem and EDTA. Analysis of a subset of 98 isolates revealed that 48% (47 out of 98) were resistant to either meropenem or imipenem or both. Of these, 83% (39 out of 47) were positive for MBLs on 1 of the 2 assays. The remaining 17% were

Table 5. Susceptibility patterns in representative *A. baumannii* clones

Clone	Isolate number	MIC, mg/ml							
		amikacin	cefepime	ceftazidime	cefotaxime	ciprofloxacin	tetracycline	imipenem	meropenem
L (n = 17)	764	128 (R)	8 (S)	32 (R)	>256 (R)	>20 (R)	32 (R)	4 (S)	>16 (R)
	765	64 (R)	24 (I)	48 (R)	>256 (R)	>20 (R)	32 (R)	6 (I)	8 (I)
	777	64 (R)	8 (S)	32 (R)	>256 (R)	>20 (R)	>48 (R)	4 (S)	6 (I)
	781	128 (R)	10 (I)	32 (R)	>256 (R)	>20 (R)	>48 (R)	6 (I)	>16 (R)
	782	>256 (R)	6 (R)	48 (R)	>256 (R)	>20 (R)	32 (R)	4 (S)	8 (I)
	787	>256 (R)	12 (I)	64 (R)	>256 (R)	>20 (R)	48 (R)	4 (S)	8 (I)
	789	128 (R)	16 (I)	48 (R)	>256 (R)	>20 (R)	>48 (R)	6 (I)	8 (I)
	788	>256 (R)	12 (I)	64 (R)	>256 (R)	>20 (R)	32 (R)	6 (I)	>16 (R)
	793	128 (R)	10 (I)	48 (R)	>256 (R)	>20 (R)	32 (R)	6 (I)	8 (I)
	814	96 (R)	6 (S)	16 (I)	>256 (R)	>20 (R)	8 (I)	4 (S)	>16 (R)
	816	96 (R)	8 (S)	24 (I)	>256 (R)	>20 (R)	16 (R)	4 (S)	8 (I)
	821	>256 (R)	8 (S)	32 (R)	>256 (R)	>20 (R)	32 (R)	4 (S)	>16 (R)
	818	128 (R)	6 (S)	20 (I)	>256 (R)	>20 (R)	20 (R)	4 (S)	8 (I)
	819	96 (R)	6 (S)	16 (I)	>256 (R)	>20 (R)	16 (R)	4 (S)	>16 (R)
	820	96 (R)	4 (S)	12 (I)	>256 (R)	>20 (R)	16 (R)	4 (S)	8 (I)
	822	48 (I)	8 (S)	32 (R)	>256 (R)	>20 (R)	32 (R)	4 (S)	>16 (R)
	067	24 (I)	6 (S)	16 (I)	>256 (R)	>20 (R)	16 (R)	4 (S)	>16 (R)
D (n = 11)	041	>256 (R)	20 (I)	48 (R)	>256 (R)	>20 (R)	>48 (R)	>24 (R)	>16 (R)
	042	>256 (R)	24 (I)	128 (R)	>256 (R)	>20 (R)	>48 (R)	>24 (R)	>16 (R)
	045	>256 (R)	20 (I)	96 (R)	>256 (R)	>20 (R)	>48 (R)	16 (R)	>16 (R)
	051	>256 (R)	12 (I)	128 (R)	>256 (R)	>20 (R)	>48 (R)	>24 (R)	>16 (R)
	058	>256 (R)	8 (S)	48 (R)	>256 (R)	>20 (R)	>48 (R)	>24 (R)	>16 (R)
	059	10 (S)	16 (I)	>128 (R)	>256 (R)	>20 (R)	>48 (R)	4 (S)	>16 (R)
	060	>256 (R)	24 (I)	64 (R)	>256 (R)	>20 (R)	>48 (R)	>24 (R)	>16 (R)
	062	>256 (R)	10 (I)	64 (R)	>256 (R)	>20 (R)	>48 (R)	>24 (R)	>16 (R)
	063	>256 (R)	16 (I)	96 (R)	>256 (R)	>20 (R)	>48 (R)	>24 (R)	>16 (R)
	064	>256 (R)	20 (I)	>128 (R)	>256 (R)	>20 (R)	>48 (R)	>24 (R)	>16 (R)
	066	>256 (R)	12 (I)	48 (R)	>256 (R)	>20 (R)	>48 (R)	>24 (R)	>16 (R)

MIC = Minimum inhibitory concentration; R = resistant; S = susceptible; I = intermediate.

negative, suggesting another mechanism of resistance such as mutation of porins or efflux pumps. Correspondingly, these isolates appear to produce MBLs. This was corroborated by the detection of carbapenemase genes by PCR [Alcantar-Curiel, unpubl. data].

Discussion

Currently, *A. baumannii* is considered to be one of the most difficult bacterial species to contain and treat in a hospital setting. Accordingly, it is critical to be able to document and understand the ability, dissemination pathways and resistance patterns of *A. baumannii* clones. Therefore, we report our 13-year experience with identifying *A. baumannii* isolates at our hospital.

The sustained, endemic nature of *A. baumannii* described in the present report illustrates what is now recognized as an important characteristic of *A. baumannii*. That is, once *A. baumannii* is introduced into a hospital, it may persist and become endemic [15, 16]. After disseminating throughout our hospital system from the adult ICU, *A. baumannii* became 1 of the 5 most frequent bacterial pathogens isolated in 2007. Furthermore, between 2009 and 2011, it was the fourth most frequent bacterial pathogen detected, displacing other nosocomial pathogens, including *P. aeruginosa*, and becoming the second most common Gram-negative pathogen only after *E. coli*. While there are several possible causes for the dissemination of *A. baumannii* that were observed, we hypothesize that the most likely explanation involves patients who were admitted to the ICU for stabilization after a surgical procedure and required mechanical ventila-

tion. While in the ICU, these patients acquired *A. baumannii*, which was then transferred to other areas as patients were moved to the neurosurgical ward or the adult internal medicine ward. As such, this situation would have facilitated the dissemination of *A. baumannii* in our hospital [17]. Patients in ICU are usually sicker, have more invasive procedures, receive prolonged antibiotic therapy and are in close contact with similar patients. The combination of all these factors compromises the immune system of a patient, facilitating initial colonization and subsequent progression to severe infection. *A. baumannii* colonizes the respiratory tract, where it can advance to nosocomial pneumonia [18, 19]. In addition, the endemicity and nosocomial outbreak of multidrug-resistant *A. baumannii* at our hospital is predominantly related to the coexistence of multiple clones and the spread of 2 carbapenem-resistant clones. Similar to other reports, it was found that during the epidemic periods, 2 epidemic *A. baumannii* clones coexisted with other related *A. baumannii* strains [15, 20].

Throughout the study period, the adult ICU was found to be an environment where the risk of *A. baumannii* transmission between colonized or infected patients was higher than other clinical areas [21]. Moreover, *A. baumannii* was found to be a nosocomial pathogen frequently isolated from all types of secretions at our hospital, including those from the respiratory tract [22]. Accordingly, medical personnel in these departments have been advised to be aware of any patient with an abnormal secretion. Once identified, these patients are to be considered potential carriers of *A. baumannii*, and contact precautions are strictly enforced.

A. baumannii was more frequently isolated during the summer months at our hospital. Although the correlation between season and frequency was not statistically significant, given that this phenomenon has also been observed in other areas of the world that have similar temperature increases during the summer months, we consider that this pattern should be further investigated [23].

In an attempt to control the spread of *A. baumannii* in our hospital, we have moved *A. baumannii*-positive patients to an adult infectious diseases ward. In addition, during outbreaks of *A. baumannii* detected in the Neurosurgical Unit, all patients were moved to this infectious diseases ward. During these periods, no dispersal occurred within the ward. During periods of peak admission to the ICU, patients at risk also had their length of stay shortened and were subsequently moved to the infectious diseases ward. A similar process has been implemented at other hospitals [24, 25].

During the period studied, some *A. baumannii* clones were found to become increasingly resistant to carbapenems. For example, some *A. baumannii* isolates developed the ability to produce MBLs, including blaVIM-4, blaIMP-1 and the carbapenemase blaOXA-24 [26]. The persistent decrease in susceptibility to imipenem is also occurring in other Latin America countries [27]. A recent report regarding the evolution of antimicrobial resistance among Gram-negative bacilli in Latin America documented that imipenem-resistant *Acinetobacter* spp. rates increased from 6.4, 12.6 and 0.0% in the 1997–1999 period to 84.9, 71.4 and 50.0% in 2008–2010 in Argentina, Brazil and Chile, respectively. Further, oxacillinase (OXA)-producing *Acinetobacter* spp. were documented in Argentina (OXA-23 and -24), Brazil (OXA-23), Chile (OXA-58) and Mexico (OXA-24) [27].

A delay in the administration of tigecycline or colistin, alone or in combination, has the potential to increase the risk of mortality [28]. In late 2010, colistin was introduced in Mexico. Correspondingly, when an *A. baumannii* infection was encountered outside of the ICU, which has frequently occurred in recent years, the majority of patients are started on an empirical regimen that includes tigecycline or colistin. Furthermore, these two drugs have been reported to be administered for the treatment of severe *A. baumannii* infections [29, 30]. For example, administration of tigecycline in conjunction with an aggressive infection control strategy has been employed successfully to control ICU outbreaks. However, *A. baumannii* can develop resistance to both colistin and tigecycline, and thus, extreme vigilance is required to diagnose the development of resistance during treatment [31].

Although our study includes a significant span of time and number of isolates, it has several limitations. The findings from our tertiary care teaching hospital may not be pertinent to other hospitals. In terms of the samples analyzed, no environmental or healthcare worker cultures were included in the study, and no matched cohort or control cases were involved. In relation to the data, patient data did not include the severity of present illness or the patient comorbidities, both of which could provide important context for infection, nor did it distinguish between colonization or infection so it was impossible to discern the progression of colonization to infection. The semiautomated identification system used cannot differentiate all of the members of the *A. calcoaceticus*-*A. baumannii* complex and cannot properly break down other *Acinetobacter* species, i.e. *Acinetobacter* genomic species 3 and *Acinetobacter* genomic species 13TU. Further, the sensitivity panels used did not include tigecycline or co-

listin, preventing us from evaluating the evolution of susceptibility of *A. baumannii* to those antibiotics. The detection of MBL production using E-test strips can produce false negative results. Finally, only some of the mechanisms involved in *A. baumannii* resistance were investigated in selected isolates, with some chosen *A. baumannii* isolates shown to have MBL genes including the *bla*_{OXA-51} gene.

In conclusion, the epidemiology of *A. baumannii* in a tertiary care teaching hospital in Mexico over the past 13

years is described. In this hospital, *A. baumannii* has become an endemic nosocomial pathogen that has not been eradicated from its main reservoir. Currently, the transfer of infected patients to an infectious diseases ward and the early treatment of *A. baumannii*-infected patients have shown limited success at our institution. It is anticipated that adequate antimicrobial stewardship and improved control and prevention measures will further help decrease the spread and impact of *A. baumannii* at our hospital.

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Erratum

In the article by Matsunaga et al., entitled ‘The polyketide synthase-associated multidrug tolerance in *Mycobacterium intracellulare* clinical isolates’ [*Chemotherapy* 2012;58:341–348, DOI: [10.1159/000343311](https://doi.org/10.1159/000343311)], the second author’s name is misspelled and should read Shinji Maeda.