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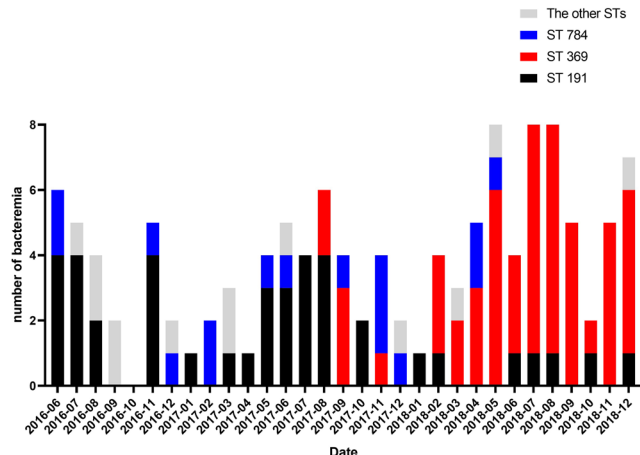
Session: 60. HAI: MDRO – GNR Epidemiology, Acinetobacter
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Background. The dissemination of carbapenem-resistant *Acinetobacter baumannii* (CRAB) became an urgent public health concern. A specific sequence type (ST) of *A. baumannii* has been reported to be associated with severity of disease or mortality. This study aimed to determine the genetic relatedness of CRAB blood isolates cultured from patients at a tertiary care hospital and to investigate clinical characteristics and outcome of CRAB bacteremia.

Methods. CRAB blood isolates were collected between June 2016 and December 2018, and their clinical data were obtained. Multi-locus sequence test (MLST) was performed using the Oxford scheme, and the STs were assigned using the MLST database.

Results. Of the 126 CRAB blood isolates, 123 isolates which could be typed by MLST all belonged to clonal complex (CC) 92. During the entire period, ST369 (42.3%) was the most dominant, followed by ST191 (32.5%), ST784 (13.8%) and ST451 (4.1%). ST369 was firstly introduced in August 2017. ST191 (61.4%) was the most abundant during June 2016 to July 2017, whereas ST369 (65.8%) replaced ST191 (16.5%) since August 2017. The time interval between intensive care unit admission and bacteremia was shorter in ST369 than ST191 in multivariate analysis (day, median (Q1, Q3), ST369 6 (3, 9.8), ST191 9 (6, 17), Odds Ratio 0.87 (95% CI 0.76–0.99) $P = 0.048$ logistic regression). According to the ST, the 7-day and 30-day mortality rates were as follows; 46% and 65% in ST191, 50% and 62% in ST369, and 10.7% and 46.4% in the other STs. Patients infected by ST191 or 369 had significant higher 7-day mortality rates (ST191/369, 48.3% vs. the other STs 10.7%, $P = 0.001$ by log-rank test) and 30-day mortality rates (ST191/369, 63.2% vs. the other STs, 46.4%, $P = 0.045$ by log-rank test).

Conclusion. This study demonstrates the clonal spread of two STs at a tertiary care hospital in South Korea over 2.5 years. After the introduction of ST369, it replaced ST 191 and widely disseminated within a hospital. Two predominant STs were associated with poor outcome. Continuous surveillance are necessary to monitor the dissemination of these strains.



Disclosures. All authors: No reported disclosures.

545. Incidence of Carbapenem Non-Susceptible *Acinetobacter* spp. and Carbapenem-Resistant *Pseudomonas aeruginosa* Clinical Cultures among Patients in US Acute Care Hospitals, 2012–2017

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Background. Carbapenem-nonsusceptible *Acinetobacter* spp. (CNAB) and carbapenem-resistant *Pseudomonas aeruginosa* (CRPA) are recognized causes of severe and difficult to treat healthcare-associated infections. This study estimated and compared the incidence of CNAB and CRPA among patients admitted to US acute care hospitals in 2012–2017.

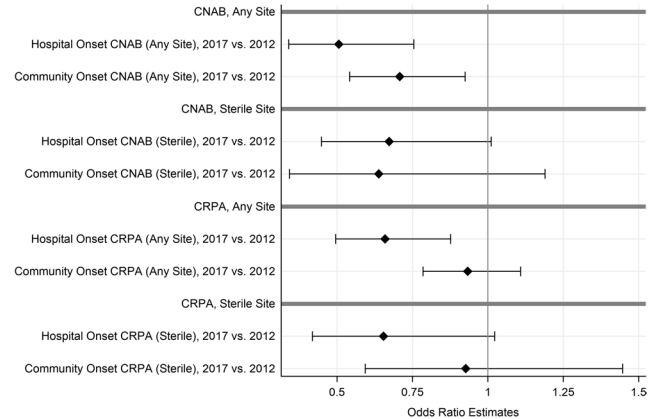
Methods. We measured the incidence of positive clinical cultures from inpatient encounters in a cohort of over 300 hospitals submitting data to the Premier Healthcare Database and Cerner Health Facts in 2012–2017. We included clinical cultures from any body site yielding *Acinetobacter* spp./*P. aeruginosa* non-susceptible/resistant to imipenem, meropenem, or doripenem. Cultures collected on days 1–3 of hospitalization were considered community-onset (CO) and cultures from later were hospital-onset (HO). Duplicate isolates identified within 14 days of an incident culture and surveillance cultures were excluded. For each year, a raking procedure

generated weights to extrapolate the sample estimate to match the American Hospital Association distributions based on US census division, hospital bed capacity, teaching status, and urban designation. We compared estimated rates in 2017 vs. 2012 using weighted multivariable logistic regression adjusting for hospital characteristics and hospital-level clustering.

Results. In 2017, the estimated rates of HO and CO CNAB rates were 0.77 and 1.39/10,000 discharges, and HO and CO CRPA rates were 3.14 and 6.57, respectively. Compared with 2017, rates of HO CNAB decreased 49% (Odds Ratio (OR) 0.51; 95% CI: 0.34–0.75) and rates of CO CNAB decreased 29% (OR 0.71; 95% CI: 0.54–0.92). For CRPA, the incidence of HO decreased (OR 0.66; CI: 0.49–0.88) with no change in CO rates (OR 0.93; CI: 0.79–1.11). Assessment of cultures from sterile sites alone showed similar results, but they did not reach statistical significance, Figure 1.

Conclusion. We estimate significant national decreases in the rates of HO and CO CNAB, and HO CRPA. Risk factors and effective interventions to reduce CO CRPA might differ from CNAB and HO CRPA. Additional prevention strategies are needed to address CO CRPA.

Figure 1. Relative Change in the Estimated Incidence of Carbapenem Non-susceptible *Acinetobacter* spp. (CNAB) and carbapenem-resistant *Pseudomonas aeruginosa* (CRPA) Clinical Cultures among Patients in U.S. Acute Care Hospitals Between 2012 and 2017



Disclosures. All authors: No reported disclosures.

546. Seasonal Changes in the Prevalence of Antibiotic-Susceptible *Acinetobacter baumannii* Results in Increased Multidrug Resistance Rates During Winter Months

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Background. Understanding the seasonal behavior of infections (“seasonality”) caused by Gram-negative pathogens, including *Acinetobacter baumannii*, is essential for the design of effective healthcare policies; however, the factors mediating seasonality remain elusive.

Methods. Over 2,000 *A. baumannii* cases identified in the BJC Health System between 2007 and 2017, were retrospectively analyzed according to isolation month, hospital acquisition, anatomical source, and antibiotic susceptibility profile.

Results. Compared with quarter 1 (Q1, December–February), *A. baumannii* case incidence was similar in Q2 (March–May) but significantly higher in Q3 (June–August) and Q4 (September–November). This seasonality was exhibited by antibiotic-susceptible but not antibiotic-resistant isolates. This was independent of tested antibiotic, anatomic source, or hospital vs. community acquisition.

Conclusion. Seasonality is absent from antibiotic-resistant *A. baumannii* cases. Selective decrease of antibiotic-susceptible cases in Q1/Q2 results in 50–100% increase in resistance rates compared with Q3/Q4. *A. baumannii* seasonality is possibly linked to the increased use of antibiotics during winter. As resistance determinants tend to be genetically linked in *A. baumannii*, pressure from community antibiotics may have the inadvertent effect of selecting for multidrug resistance. This link must be further studied, as it may also explain seasonality in other, more antibiotic-susceptible Gram-negative pathogens.

Disclosures. All authors: No reported disclosures.

547. Multidrug-Resistant *Pseudomonas aeruginosa* in an Academic Regional Burn Intensive Care Unit

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Background. *Pseudomonas aeruginosa* infection can lead to morbidity, mortality and increased hospital length of stay especially in Burn Intensive Care Units (BICU) patients. Reports of multi-drug-resistant *Pseudomonas aeruginosa* outbreaks in the BICU are increasing. We investigated the epidemiology of Carbapenem-Resistant *Pseudomonas aeruginosa* (CRPA) in our BICU.

Methods. Clinical and laboratory characteristics of all CRPA isolates identified between 5/8/16 and 3/14/19, in an 11-bed BICU in an academic 870-bed public safety-net hospital were reviewed and defined as Meropenem MIC 4 or greater. Retained isolates were sent for pulse-field gel electrophoresis (PFGE). Infection prevention (IP) observations and interventions were intensified and environmental cultures were collected. Patient charts were reviewed.

Results. 27 patients between ages 5–61 years old were found to have CRPA (only 2 patients < 18 years). 21/27 (77.7%) were male. 21/27 (77.7%) had >40% total body surface area (TBSA) burns, 3/27 (11.1%) had 20–39% TBSA burn and 1/27 (3.7%) had < 20% TBSA burn. 19/27 (70.3%) patients had bacteremia, 6 had respiratory infections with 3 (11.1%) Infection-related Ventilator-Associated Complications (IVAC), 3 had urinary tract infection, and 1 had CRPA from a central venous catheter tip. There were very few co-morbidities. Twenty isolates from 11 different patients were typed and revealed 2 different clonal strains. 5/11 (45%) patients had strain A, and 2/11 (18%) patients had strain B. 3/11 (27.2%) patients had unique strains. CRPA was isolated from 5 different rooms. Water cultures did not reveal CRPA. Failure of hand hygiene, non-adherence to isolation/PPE protocols and clutter were found. Each failure was corrected. No new CRPA patient isolates have been identified.

Conclusion. Transmission was halted by reinforcement of IP measures. Importantly water was not a source of CRPA in this setting and the data suggest transmission due to environmental contamination.

Disclosures. Trish M. Perl, MD; MSc, 7–11: Advisory Board; medimmune: Research Grant

548. Carbapenem-Resistant *Acinetobacter baumannii* Antibiotic Susceptibility Testing and Antibiogram Formation, Connecticut 2017–2019

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Background. Carbapenem-resistant *Acinetobacter baumannii* (CRAB) is an infectious disease threat with limited treatment options. Statewide CRAB reporting and isolate submission has been mandated in Connecticut (CT) since 2017, which allowed the creation of a statewide CRAB antibiogram to assist with empiric treatment options for CRAB.

Methods. Clinical CRAB isolates from 2017 through the first quarter of 2019 underwent carbapenemase and expanded susceptibility testing at the CT State Public Health Laboratory or the Antibiotic Resistance Laboratory Network regional lab for carbapenemase and expanded susceptibility testing. Susceptibility testing was done by broth microdilution and disk diffusion, and interpreted using Clinical and Laboratory Standards Institute breakpoints. Carbapenemase producers were detected by the modified carbapenem inactivation method. Polymerase chain reaction testing identified carbapenemase genes.

Results. Of the 64 CRAB isolates submitted, 40 remained after confirmation of carbapenem resistance, i.e., resistance to at least one carbapenem, and deduplication of patients. Of these, 19 were carbapenemase producers (CP), and 21 were non-carbapenemase producers (Non-CP). All isolates were non-susceptible to cefepime, ceftazidime, levofloxacin and all carbapenems. Colistin susceptibilities were available for 33 isolates, 32 (97%) of which were susceptible. Tobramycin susceptibilities were available for 31 isolates, only 10 (32%) of which were susceptible. Of the CP, all 15 were susceptible to colistin, but only 2 (14%) were susceptible to tobramycin. Of the Non-CP, 16 (89%) were susceptible to colistin, and 8 (47%) were susceptible to tobramycin. Most CRABs had a tigecycline minimum inhibitory concentration (MIC) of $\leq 2 \mu\text{g/mL}$, with a higher proportion of Non-CP with lower MIC values than CP.

Conclusion. CRAB shows resistance to all carbapenems, and most non-carbapenem antibiotics except colistin and in rare circumstances tobramycin. Most CRAB isolates had tigecycline MICs of $\leq 2 \mu\text{g/mL}$. The statewide antibiogram illustrates the lack of approved antibiotics for the treatment of CRAB, underscoring the importance of further antibiotic development for CRAB treatment.

	# of isolates	% Susceptible							
		Carbapenems			Other antibiotics				
		Doripenem	Imipenem	Meropenem	Cefepime	Ceftazidime	Colistin	Levofloxacin	Tobramycin
<i>Acinetobacter baumannii</i>	40	0	0	0	0	0	97	0	32
CP-CRAB	19	0	0	0	0	0	100	0	14
Non-CP-CRAB	21	0	0	0	0	0	89	0	47

CRAB Minimum Inhibitory Concentration Values for Antibiotics without Interpretive Values

Antibiotic	Total (31) MIC (mcg/mL) (#)	CP (14) MIC (mcg/mL) (#)	Non-CP (17) MIC (mcg/mL) (#)
Ceftazidime-avibactam	≥ 32 (24) 16 (7)	≥ 32 (14) 16 (0)	≥ 32 (10) 16 (7)
Ceftolazone-tazobactam	≥ 16 (26) 8 (2) 4 (2) 2 (0) ≤ 1 (1)	≥ 16 (14) 8 (0) 4 (0) 2 (0) ≤ 1 (0)	≥ 16 (12) 8 (2) 4 (2) 2 (0) ≤ 1 (1)
Tigecycline	4 (1) 2 (17) ≤ 1 (13)	4 (1) 2 (10) ≤ 1 (3)	4 (0) 2 (7) ≤ 1 (10)
Moxifloxacin	≥ 16 (20) 8 (7) 4 (3)	≥ 16 (10) 8 (4) 4 (0)	≥ 16 (10) 8 (3) 4 (3)
Colistin	≥ 4 (1) 2 (1) 1 (2) 0.5 (3) ≤ 0.25 (21)	≥ 4 (0) 2 (1) 1 (0) 0.5 (1) ≤ 0.25 (12)	≥ 4 (1) 2 (0) 1 (2) 0.5 (2) ≤ 0.25 (9)

Disclosures. All authors: No reported disclosures.

549. First Report for Emergence of Chromosomal Borne Colistin Resistance Gene *mcr-1* in a Clinical *Acinetobacter baumannii* Isolates from India

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Background. Efficacy of Colistin the last line agent against infections due to multidrug-resistant (MDR) gram-negative pathogens, has been challenged when Liu et al. reported a plasmid-mediated gene, *mcr-1*, in 2015. Thereby this plasmid-borne *mcr* has been reported in bacterial isolates worldwide taken from humans, animals, farms, foods, and the environment. The present work investigate the *mcr* gene among clinical isolates of *Acinetobacter baumannii* at our tertiary referral hospital of India.

Methods. The study was conducted at Sanjay Gandhi Post Graduate Institute of Medical Sciences, Lucknow, India. MIC values for 100 consecutive non-duplicate MDR isolates of *Acinetobacter* were checked for Colistin. PCR amplification of *mcr* gene was performed followed by sequencing of the amplicons. Clinical features of patients infected with *mcr* positive isolates were unveiled. Clonal relatedness of these isolates was investigated by Pulsed-field gel electrophoresis (PFGE). The *mcr-1* localization was checked by conjugation followed by PFGE southern hybridization.

Results. 20/100 (20%) isolates were colistin resistant with having MIC values of more than $8 \mu\text{g/mL}$. The 20 colistin resistances isolates were PCR positive for *mcr-1* and had been assigned EMBL/GeneBank nucleotide accession numbers MH730099-MH730118. Other antibiotic resistance gene like ESBL, NDM-1, VIM, and 16s rRNA methyl transferases like Arm A, rmtC, rmt F were also found in these isolates. Majority of these patients recovered from the infection (65%) after proper antibiotic therapy. The IS*Apl1* transposable elements were not detected in these isolates. These isolates were found clonally unrelated when analyze by pulsed-field gel electrophoresis. The conjugation attempt to transfer *mcr-1* to recipient's *E. coli* J53 failed, Southern hybridization showed that *mcr-1* was found located on chromosome in multiple copies.

Conclusion. This is the first case of *mcr-1* in a human clinical isolate in *Acinetobacter baumannii* from India. These findings highlight the vertical transferability of colistin resistance by *mcr-1* gene in *Acinetobacter baumannii* with the association of known some unknown insertion sequence located on chromosome. Strategies required to contain their spread and evolution of such genes.

Disclosures. All authors: No reported disclosures.