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Session: 60. HAI: MDRO – GNR Epidemiology, Acinetobacter
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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.

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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 ≤ 2 μ 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 ≤ 2 μ 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)

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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 $8 \leq \mu$ 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*AplI* 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.

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