carbapenem inactivation method was used for phenotypic detection of carbapenemase production. The presence of a variety of carbapenemase genes was evaluated by PCR with specific primers.

**Results.** Of 134 patients with monomicrobial CRE bacteremia, 48 (35.8%) were infected with CP-CRE, and 86 (64.1%) were infected with non-CP-CRE. The most common carbapenemase in non-CP-CRE isolates was KPC (42.6%), followed by NDM-1 (18.8%), OXA-48-like (10.4%), and VIM (4.1%). Baseline characteristics were similar between the two groups (Table 1). However, the CP-CRE group was significantly more likely to undergo removal of eradicable foci and to have meropenem MIC >8 µg/mL. A total of 33 (24.6%) patients died within 14 days, including 9 (18.8%) in the CP-CRE group and 24 (27.9%) in the non-CP-CRE group. Deceased patients were more likely to have a higher Pitt bacteremia score, nosocomial acquisition, ineradicable or not-eradicable foci, immunosuppressant use, inappropriate definitive treatment (Table 2). Combination therapy for definitive treatment was associated with decreased mortality. In a multivariate analysis including carbapenemase production, a higher Pitt bacteremia score (aOR, 5.15), ineradicable or not-eradicated foci (aOR, 4.05) and combination therapy for definitive treatment (aOR, 0.35) were independent risk factors for mortality.

**Conclusion.** Our study suggests that carbapenem production is not a mortality risk factor in CRE bacteremia and provides additional evidence for early source control and combination therapy.

Table 1. Baseline and clinical characteristics of patients with carbapenem-resistant Enterobacteriaceae (CP-CRE) and non-CRE bacteremia

| Characteristic/outcome | CP-CRE (n=48) | Non-CRE (n=86) | P value |
|-------------------------|--------------|---------------|---------|
| Age (yr), median (IQR)  | 62.5 (53.6-80) | 59 (46-64.3) | 0.01 |
| Male                    | 37 (77.1%)   | 63 (73.3%)    | 0.63   |
| Previous hospitalization|              |               |         |
| Healthcare-associated   | 34 (70.8%)   | 61 (71.4%)    | 0.75   |
| Nosocomial              | 26 (54.2%)   | 30 (35.3%)    | 0.02   |
| Me/Cab and Jackson classification | 46 (95.8%) | 83 (96.5%) | 0.64 |
| Ineradicable or not-eradicable foci (aOR, 4.05) and combination therapy for definitive treatment (aOR, 0.35) were independent risk factors for mortality.

Table 2. Results of analyses of risk factors for 14-day mortality in CRE bacteremia

| Risk factor | Univariate analysis | Multivariate analysis |
|-------------|---------------------|----------------------|
| Weight (kg) | 0.04 (0.01-0.08) | 0.05 (0.01-0.20) |
| Age (yr)    | 0.04 (0.01-0.08) | 0.05 (0.01-0.20) |
| Pitt bacteremia score ≥4 | 0.04 (0.01-0.08) | 0.05 (0.01-0.20) |
| Combination therapy for definitive treatment | 0.04 (0.01-0.08) | 0.05 (0.01-0.20) |
| Immunosuppressant use | 0.04 (0.01-0.08) | 0.05 (0.01-0.20) |

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Results. We included 250 patients during the study period. The median age was 55 (19–91) years, 55.6% (139/250) were male, and 49.2% (123/250) were admitted in the intensive care unit (ICU). 77.2% (193/250) had at least one comorbidity, with solid-organ malignancy (27.6%, 69/250) being the most common. Most patients had at least one invasive device, commonly a nasogastric tube (64.4%, 161/250) or mechanical ventilator (63.2%, 150/250). There was an increase in Acute Physiologic Assessment and Chronic Health Evaluation (APACHE) II score, qSOFA, and septic shock from baseline to 24 hours prior to colistin use (Table 1). The most common site of infection was the respiratory tract (90.8%, 227/250). Majority of patients (78.8%, 197/250) had Acinetobacter baumannii infection of which 79.9% were extensively drug-resistant (XDR), susceptible only to colistin and amikacin or minocycline. Colistin was given for a mean of 12 (2–43) days, concomitant with meropenem in 96.4% (241/250). Most patients received piperacillin–tazobactam (59%, 142/250) and/or meropenem (58%, 138/250) within the same admission. Nephrotoxicity (30.8%, 77/250) was the most frequent adverse effect (Table 2). Renal replacement therapy was needed in 6% (15/250) patients. Clinical success was seen in 61.2% (153/250) patients and overall mortality was 41.6% (104/250).

Conclusion. Colistin was frequently used in combination with a carbapenem for treatment of XDR-related respiratory infection or septic shock. Nephrotoxicity was a common adverse effect. The clinical success rate was modest and overall mortality was high.

Table 1. Septic shock, Acute Physiologic Assessment and Chronic Health Evaluation (APACHE) II, and quick Sequential Organ Failure Assessment (qSOFA) score

| Scoring Criteria | n=250 | Frequency (%) | Mean ± SD |
|------------------|-------|---------------|-----------|
| Septic shock     |       |               |           |
| Baseline         | 13 (5.2) |
| 24 hours prior to colistin | 40 (16) |
| APACHE II        |       |               |           |
| Baseline         | 11.3 ± 4.9 |
| 24 hours prior to colistin | 16.02 ± 4.47 |
| qSOFA            |       |               |           |
| Baseline         | 1.00 ± 0.87 |
| 0                | 82 (32.8) |
| 1                | 99 (39.6) |
| 2                | 56 (22.4) |
| 3                | 13 (5.20) |
| 24 hours prior to colistin | 1.40 ± 0.97 |
| 0                | 51 (20.4) |
| 1                | 83 (33.2) |
| 2                | 80 (32.0) |
| 3                | 36 (14.4) |
| Day 7 of colistin |        |               |           |
| 0                | 111 (44.4) |
| 1                | 68 (27.2) |
| 2                | 39 (15.6) |
| 3                | 32 (12.8) |

Table 2. Adverse Events

| Toxidities         | n=250 | Frequency (%) | Mean ± SD |
|--------------------|-------|---------------|-----------|
| Nephrotoxicity     | 77 (30.8) |
| AKI Stage 1        | 27 (10.8) |
| AKI Stage 2        | 20 (8.0)  |
| AKI Stage 3        | 30 (12.0) |
| CRT                | 15 (6.0)  |
| Neurotoxicity      | 10.4    |
| Hypersensitivity   | 11 (4.4)  |

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522. In Vitro Antimicrobial Activity of Ceftazidime/Avibactam Compared with Ceftolozane/Tazobactam Against Real-world Clinical Isolates of Pseudomonas aeruginosa at a Large Academic Tertiary Care Hospital

Twisha S. Patel, PharmD, BCPS, BCIDP1; Keith S. Kaye, MD, MPH2; Jay Krishnan, MD3; Vince Marshall4; John Mills5; Owen Albin, MD3; Aaron Smith1; Paul Lephart1 and Jason M. Pogue, PharmD, BCPS, BCIDP3. 1Michigan Medicine, Ann Arbor, Michigan; 2University of Michigan College of Pharmacy, Ann Arbor, Michigan; 3University of Michigan; 4Ann Arbor, Michigan; 5Michigan Medicine, Ann Arbor, Michigan; 6University of Michigan Medicine. Among all isolates including those displaying resistance to various β-lactams. Among all isolates of PSA, CZA (96.2% susceptible) was slightly more active than CT (94.2%) and both agents were ~10% more active than the closest comparator (ceftazidime, 86.6%). In in vitro cetepine, piperacillin/tazobactam, and meropenem were 84.8%, 78%, and 80.3%, respectively. The activity of both CZA and CT dropped significantly among isolates with pan-β-lactam resistance (i.e., resistance to all conventional anti-pseudomonal β-lactams, PBR) but CZA remained more active than CT (59.4% vs. 41.5%, P < 0.001). Of isolates displaying resistance to CT, 84 (48.6%) were susceptible to CZA. However, of those with resistance to CZA, only 21 (21.2%) were susceptible to CT (Table 2).

Conclusion. CZA was the most active β-lactam against PSA isolates at Michigan Medicine. Among PSA with PBR, CZA demonstrated superior activity compared with CT. Additionally, a significant number of isolates with resistance to CT were susceptible to CZA.