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Ceftazidime-avibactam and ceftolozane-tazobactam activity against Escherichia coli producing CTX-M beta-lactamases

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Background: Escherichia coli (ECOL) is one of the most common Gram negative bacteria that cause a variety of infection types. This organism has been related to produce resistance various types of resistance including extended-spectrum beta-lactamases (ESBL). There have been limited options for the treatment of ESBL producing pathogens and there have been recent approval of new cephalosporin beta-lactamase inhibitor combination for ESBL pathogens. The purpose of this study was to identify the ESBL producing enzyme from multidrug resistant ECOL and evaluate the activity of ceftazidime/avibactam and ceftolozane/tazobactam.

Methods: A retrospective analysis of ECOL clinical isolates collected at the centralized laboratory (Affiliated Laboratory, Inc., Bangor, ME) during January to December 2015. Antimicrobial susceptibility were collected and screening for ESBL was performed with Thermo Fischer Scientific Sensititre using cefuroxime and ceftriaxone resistance interpreted by Clinical & Laboratory Standards Institute. A random selection of one-third of screened isolates were selected identification of ESBL molecular detection. DNA template preparation and PCR amplification for beta-lactamases genes such as blaSHV, blaTEM, and blaCTX-M were carried out on a Thermal Cycler instrument. Selected isolates were grown on Mueller-Hinton blood agar media and ceftazidime/avibactam and ceftolozane/tazobactam Etest MICs were recorded.

Results: Total of 1818 ECOL unique isolates were collected. A total of 1370 (75.4%) were collected from an outpatient setting and 1357 (74.6%) were collected from urinary culture. A total of 119 (6.3%) isolates were identified to have cefuroxime resistance and 68 (3.7%) were screened and had a phenotypic resistance panel of cefuroxime and ceftriaxone concurrently. Of these screened

pathogens all of them were meropenem susceptible however 9 (13.2%) were resistant to ciprofloxacin and gentamicin concurrently. Twenty-three ECOL isolates were identified to produce CTX-M enzyme specifically 12 (52.2%) were identified as CTX-M-15 and 11 (47.8%) were identified as CTX-M-14. The ceftolozane/tazobactam MIC ranged from 0.094 to 4mg/L with MIC50 of 0.25mg/L and MIC90 of 0.5mg/L. The ceftazidime/avibactam MIC ranged from 0.064 to 1mg/L with MIC50 of 0.25mg/L and MIC90 of 0.38mg/L.

Conclusions: ESBL incidence was very low and that CTX-M was the common enzyme with subtype 14 and 15 in ECOL clinical isolates. One isolate was found to be intermediate with ceftolozane/tazobactam the remaining isolates were susceptible to ceftolozane/tazobactam. All of the isolates were susceptible to ceftazidime/avibactam. Of interest, our ECOL MIC90 values for both ceftazidime/avibactam and ceftolozane/tazobactam were two-fold lower than the upper susceptible breakpoint. These two new antimicrobials may be a good option for ECOL including isolates producing CTX-M-14/15; however follow up susceptibilities are still warranted.