29th ECCMID Amsterdam, Netherlands 13 – 16 April 2019

The congress of ESCMID

P1295 Genomic epidemiology of carbapenemase-producing *Enterobacteriaceae* in hospitalised patients at Siriraj hospital, Thailand

Adhiratha Boonyasiri*[1,2], Kanokorn Lerdlamyong[1], Lauren M Brinkac[3], Xavier Didelot[4], Teerawit Tangkoskul[1], Chris Greco[3], Thomas H Clarke[3], Richard White[3], Kevin Nguyen[3], Indresh Singh[3], Granger Sutton[3], Elita Jauneikaite[4], Derrick Fouts[3], Visanu Thamlikitkul[1]

[1] Mahidol University, Faculty of Medicine, Siriraj Hospital, Bangkok, Thailand, [2] Imperial College London, National Institute of Health Research Health Protection Research Unit in Healthcare Associated Infection and Antimicrobial Resistance, London, United Kingdom, [3] J. Craig Venter Institute, Maryland, United States, [4] Imperial College London, Department of Infectious Disease Epidemiology, London, United Kingdom

Background: Carbapenemase-producing Enterobacteriaceae (CPE) has been a compelling cause of hospital-acquired infections with a high mortality rate. The recent rise in CPE infections has left clinicians with limited antimicrobial treatment options. Bacterial genomic studies provide useful data on pathogenicity, transmission pathways and can be applied to track CPE clones. This study sought to describe the genomic epidemiology of CPE in hospitalised patients in Bangkok between 2015-2017.

Materials/methods: A prospective cohort study was conducted to examine the natural history and factors associated with the development of CPE infections in hospitalised patients. Faecal isolates from the patients were collected and 45 CPE isolates were sent for whole genome sequencing (WGS) on the NextSeq 500 platform. Genomic analysis was conducted including determination of multi-locus sequence type, detection of antimicrobial resistance genes and phylogenetic relationship between isolates.

Results: 45 CPE isolates from 39 patients were included in this study. 88.9% of the CPE isolates were *Klebsiella pneumoniae* (n= 40), followed by 8.9% *Escherichia coli* (n=4) and one *Enterobacter hormaechei* isolate. Most patients were elderly males who received antibiotics prior to CPE detection. Approximately 11% of CPE isolates were resistant to colistin (n=5). All of them were *K.pneumoniae*. The most dominant carbapenemase gene family, OXA-48-like, was found in *K.pneumoniae* (n = 33) and *E.coli* (n = 2) and followed by NDM-1 identified in *K.pneumoniae* (n = 19), while IMP-14 was only identified in *E. hormaechei*. In addition, we identified 16 *K.pneumoniae* isolates harbouring both OXA-232 and NDM-1. The core genome single nucleotide polymorphism (SNP) phylogeny did not demonstrate clonal spread in our hospital. There were two major subclades of *K.pneumoniae* that were dominant, ST16 (n = 15) and ST231 (n = 14). 87% of ST16 isolates (n = 13) carried OXA-232 and NDM-1, whereas nearly all of the ST231 isolates (n = 13) had only OXA-232.

Conclusions: The majority of CPE in this study was *K.pneumoniae* with OXA-232. Multiple clones were responsible for the spread of carbapenemase-producing *K.pneumoniae* in our hospital.