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Abstract (poster session)

A swordless knight: the epidemiology and molecular characteristics of the blaKPC-negative sequence-type 258 *Klebsiella pneumoniae* clone

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Objectives: The KPC-producing *Klebsiella pneumoniae* sequence type (ST) 258 shows formidable epidemiological success. This clone has spread rapidly across all Israeli hospital in 2006. In July 2009, a blaKPC-negative, ertapenem-resistant ST-258 *K. pneumoniae* strain was isolated from a patient in the Laniado hospital. Our aims were 1) to assess weather the blaKPC-negative ST-258 *K. pneumoniae* clone will gain similar spread to that seen by its KPC producing isogenic strain and 2) to describe its molecular characteristics and resistance mechanisms. **Methods:** A prospective surveillance of all ertapenem non-susceptible, carbapenemase-negative *K. pneumoniae* (ENSCNKP) isolates was conducted at the Laniado hospital (314 beds) from July 2010 to June 2011, and at the Tel-Aviv Sourasky Medical Center (TASMC) (1200 beds) it was done retrospectively from July 2008 to December 2010. In addition, 50 ertapenem-susceptible and 20 KPC-producing *K. pneumoniae* isolates, collected during 2010-2011 were studied. Molecular typing was done by PFGE and multi-locus sequence typing. Mechanisms of resistance to ertapenem were studied by PCR of beta-lactamase genes and by sequencing of the ompK genes. Plasmid composition was studied by S1-nuclease analysis. The fate of the blaKPC-carrying plasmid, pKpQIL, was determined by PCR of the Tn4401 transposon and its unique genes, repA and the truncated blaOXA-9 gene. **Results:** During the study periods the ENSCNKP ST-258 clone was found in 6 of 44 ENSCNKP isolates in Laniado hospital (4 of them from a single ward during a 2-months period) and in 1 of 38 ENSCNKP in TASMC. ENSCNKP ST-258 strains were not associated with invasive infections. In contrast, none of the 50 ertapenem-susceptible *K. pneumoniae* and 17/20 of KPC-producing *K. pneumoniae* isolates belonged to ST-258. The MIC for ertapenem in the 7 ENSCNKP ST-258 isolates ranged from 4 to >32 mg/L. The isolates carried either the blaCTX-M-2 or the blaCTX-M-25 genes, and all possessed a frameshift mutation at the ompK35 gene, similar to that identified in the KPC-producing ST-258 strains. Plasmid analysis showed variability in plasmid composition and absence of the pKpQIL plasmid in the ENSCNKP ST-258 isolates. **Conclusion:** Our results suggest that ENSCNKP ST-258 evolved by loss of the blaKPC carrying plasmid pKpQIL and retained high ertapenem MIC due to porin loss. In contrast with the behavior of the parent strain, ENSCNKP ST-258 appears to have low epidemic and virulence potential.