P1745
Paper Poster Session
Klebsiella pneumoniae: a special pathogen

Virulence profile evolution in Klebsiella pneumoniae isolates: from TEM-1 to KPC-3

Cátia Caneiras*[1], Luís Marques Lito[2], José Melo-Cristino[3], Aida Duarte[4]

[1]*Imed.UI - Research Institute for Medicines and Pharmaceutical Sciences, Faculty of Pharmacy, University of Lisbon, Lisboa, Portugal*

[2]*Serviço de Patologia Clínica, Laboratory of Microbiology, Centro Hospitalar Lisboa Norte E.P.E., Lisboa, Portugal*

[3]*Instituto de Medicina Molecular, Faculty of Medicine, University of Lisbon , Lisboa, Portugal*

[4]*Imed.UI - Research Institute for Medicines and Pharmaceutical Sciences, Faculty of Pharmacy, University of Lisbon , Lisboa, Portugal*

Background: The epidemiology of *Klebsiella pneumoniae* is complex, involving ecological persistence and carriage of this bacterium is frequently associated with colonization of the upper respiratory tract or gastrointestinal tract with potential for selection of antibiotic resistant strains of *K. pneumonia e*following antibiotic therapies. Since the first broad-spectrum β-lactamase (BSBL), TEM-1, the evolution of extended-spectrum β-lactamases (ESBL) and carbapenemases, are one of the most significant epidemiologic changes in infectious diseases. The aim of this study was to determinate the virulence profile of *K. pneumoniae* β-lactamase producers collected during 33 years in one single tertiary hospital centre.

Material/methods: This study included 100 representative *K. pneumoniae* isolates collected between 1980 and 2013, namely TEM-1 BSBL (n=15), TEM-10 (n=7) and -24 (n=5), CTX-M-15 (n=46) and KPC-3 carbapenemases (n=27).The virulence factors were assessed by PCR with specific primers for K2 serotype *(k2A),* fimbrial adhesins type 1 *(fimH)* and type 3 (*mrkDv*$_1$ and *mrKD*$_{v2\text{-}4}$), haemolysin (*khe*) and aerobactine *(iucC*). The virulence profiles according to the β-lactamase produced were also studied.

Results: The multidrug-resistant isolates showed fimH in 96%, mrkDv1 in 90% and khe in 63% of the isolates. The K2 serotype was found in 23% of the isolates and only 6% had mrkD$_{v2\text{-}4}$ and iucC. From 35% of the isolates have been detected 3 genes virulence profile fimH, khe, mrkD$_{V1}$ followed by 2 genes fimH, mrkD$_{V1}$ (27%) and 4 genes K2, fimH, khe, mrkD$_{V1}$ (11%). Different virulence profiles was found according to antibiotic resistance, namely 27% of TEM-1 producers had the VF2 (fimH, khe, mrkD$_{V2\text{-}4}$), followed by VF7 (khe, mrkD$_{V1}$) in 20% of the isolates. From TEM ESBL producers 50% of isolates has showed the VF1 (fimH, khe, mrkD$_{V1}$), followed by VF6 (fimH, khe) and VF3 (fimH, khe, and mrkD$_{V1}$, K2) in 25% and 17% of isolates, respectively. TEM-10 isolates were included frequently (57%) in profile VF1 (fimH, khe, mrkD$_{V1}$) and 60% of TEM-24 isolates had the profile VF5 (fimH, khe). By other hand the CTX-M-15 ESBL producers showed the VF1 (fimH, khe, mrkDV1) and VF4 (fimH, mrkD$_{V1}$) in 50% and 48% of isolates, respectively. Finally, the KPC-3 isolates predominantly showed the virulence profile VF3 (fimH, khe, mrkD$_{V1,}$ K2) and VF4 (fimH, mrkD$_{V1,}$ K2). Only the virulence profile VF1 (fimH, khe, mrkDV1) was shared by all β-lactamase producers, namely by TEM ESBL and CTX-M-15 (50%), KPC-3 (15%) and TEM-1 (13%).

Conclusions: The association of haemolysin and fimbrial adhesins type 1 and 3 (khe, fimH, mrkD$_{V1}$) was the predominant virulence profile in *K. pneumoniae* β-lactamase producers. The results indicate

an association among the different enzymes produced and virulence profile. Of concern, considering the 9 different virulence profiles presented, the KPC-3 producers isolates seems to successful adapt to the host environment and maintain virulence via several pathways.