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Abstract (poster session)

ISPa46, a novel insertion sequence in OprD porin of imipenem-resistant *Pseudomonas aeruginosa* isolate from a cystic fibrosis patient in Marseille, France

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Objective: Clinical isolates of *Pseudomonas aeruginosa* exhibiting high-level resistance to carbapenem were recovered from a French patient with Cystic Fibrosis (CF) who did not receive carbapenem therapy. This work was conducted to investigate the mechanism that conferred the resistance phenotype to carbapenems. **Methods:** Antibiotic susceptibility testing was performed using Etest method. Carbapenemase activity was tested using the modified imipenem-EDTA double disc synergy test. Molecular support for carbapenem resistance was investigated by PCR amplification of putative carbapenemase encoding genes as well as PCR amplification and sequencing of OprD porin gene. **Results:** Six clinical isolates recovered from the same CF patient between 2008 to 2011 were retrospectively analyzed (one imipenem-susceptible isolate and five imipenem-resistant isolates). Double disk synergy test and PCR targeting carbapenemase encoding genes were negative for all strains. However, PCR amplification and sequencing of OprD gene reveal the disruption of oprD gene by an insertion sequence (IS) element of 1,337-bp, ISPa46, that contained a novel transposase of 1,227-bp inserted the oprD gene, bordered by two terminal imperfect inverted repeats (IR) of 28-bp that was associated with carbapenem resistance for the five imipenem-resistant isolates. Interestingly, this ISPa46 was also present in the imipenem-susceptible isolate but was not transposed in OprD likely suggesting that intrinsic movement of this IS was responsible for resistance to carbapenems. **Conclusion:** In conclusion, we report a novel IS element, ISPa46, in *P. aeruginosa* clinical isolates of a CF patient in Marseille, France, that was associated with carbapenem resistance and selected in the absence of carbapenem treatment.