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Abstract (poster session)

Characterisation of a cfr-positive methicillin-resistant Staphylococcus epidermidis strain of the lineage ST22 implicated in a life-threatening human infection

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Objectives. The gene cfr encodes a methyltransferase which produces the methylation of the 23S rRNA leading to a multiresistant phenotype. This gene was identified in Methicillin resistant Staphylococcus epidermidis (MRSE) isolates from a very serious human case. The aim was to characterize the cfr positive MRSE isolates and to determine the localization and the genetic environment of this gene. Methods. Three MRSE isolates were detected in samples from cerebrospinal fluid, pleural drainage and vascular catheter of the patient. Antibiotic susceptibility testing was performed by Wider system and for 9 agents also by the agar dilution method (chloramphenicol, clindamycin, erythromycin, lincomycin, linezolid, tetracycline, tiamulina, trimethoprim and virginiamycin). The 3 MRSE isolates were typed by MLST, PFGE and SCCmec-typing. The presence of resistance genes was studied by PCR. The presence of mutations in 23S rRNA, L3, L4, L22, grlA, gyrA and fusA was investigated by PCR and sequencing. The 3 MRSE isolates were tested by PCR for the genes lukF/lukS-PV, icaA, icaB, icaC and for the IS256 element. Plasmid or chromosomal gene location was determined by S1-PFGE and I-CeuI-PFGE hybridization. Genetic environment was studied by PCR-mapping and sequencing. Conjugative transfer of cfr gene was performed. Results. The 3 MRSE strains showed the same PFGE-pattern, belonged to ST22 and had SCCmec-type III. All of them were resistant to 10 antimicrobials groups. The presence of cfr, fexA, aac(6´)-aph(2"), dfrA, icaA, icaB and icaC genes was confirmed by PCR. Mutations mediating quinolone resistance revealed the S80F and D84N exchanges in GrlA and S84F in GyrA. MRSE isolates possessed L101V and A58T substitutions and 135QGRGPM136 insertion in L3 and N64K and N158S exchanges and 71G72 insertion in L4. The sequencing of 23S rRNA revealed the mutation C2534T. No mutations were identified in L22 and in fusA. Hybridization experiments revealed the presence of the cfr gene in a plasmid of 45-kb and in chromosomal DNA. One transconjugant was obtained and the genetic environment was similar to pSCFS7 (FR675942.1), including the gene fexA. PVL and the IS256 element were not detected. Conclusion. We describe a fatal human case in which cfr-positive MRSE ST22 isolates were detected. The spread of this resistance mechanism is especially worrisome due to this gene lead to simultaneous resistance for several antimicrobials.