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Paper Poster Session
The complexity of antibacterial resistance mechanisms

Antimicrobial susceptibility and molecular mechanisms of acquired resistance in Actinotignum (Actinobaculum) schaalii isolated in patients with hidradenitis suppurativa

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Background: *Actinotignum* (*Actinobaculum*) *schaalii* is an emerging uropathogen in elderly and patients with predisposing urological conditions, but it has recently been recovered from chronic lesions of patients suffering from hidradenitis suppurativa (HS). Since the combination clindamycin-rifampicin (CLI-RIF) is empirically recommended as first-line therapy in HS patients, there is a potential risk of emerging resistance. The aim of the study was then to assess the in vitro antimicrobial susceptibility of *A. schaalii* isolated in this context as well as to dissect the genetic basis of acquired macrolide-lincosamide-streptogramin (MLS) and RIF resistance.

Material/methods: A total of 20 clinical isolates of *A. schaalii* collected from skin lesions from 14 HS patients were studied. Identification was carried out by MALDI-TOF mass spectrometry. MICs of 18 antibiotics were determined using the agar dilution method on Mueller-Hinton agar plate supplemented with 5% lysed horse blood. Screening for *erm*(A), *erm*(B), *erm*(C), *erm*(F), *erm*(G) and *erm*(X) class genes was performed by PCR. The rifampicin-resistance-determining region (RRDR) of the *rpoB* gene was also identified and sequenced.

Results: All 20 isolates exhibited low MICs for amoxicillin, piperacillin, ceftriaxone, imipenem, vancomycin, teicoplanin, quinupristin-dalfopristin, tetracycline and tigecycline ($MIC_{50/90}$ at 0.12/0.12, 0.06/0.06, 0.01/0.01, 0.03/0.03, 0.12/0.25, 0.25/0.25, 0.25/0.5, 0.5/0.5 and 0.12/0.12 mg/L, respectively). MICs of ciprofloxacin, levofloxacin, moxifloxacin, linezolid, gentamicin and cotrimoxazole were slightly higher with $MIC_{50/90}$ at 2/2, 1/1, 0.5/1, 1/1, 1/1 and 4/4 mg/L, respectively. Sixteen isolates (80%) were highly resistant to both erythromycin (MICs >256 mg/L) and CLI (MICs from 32 to >256 mg/L), including three isolates (15%) also resistant to RIF (MICs at 128 mg/L). All MLS-resistant isolates only harboured the *erm*(X) resistance gene. All RIF-resistant isolates possessed mutations in the RRDR of *rpoB* (*Escherichia coli* numbering): a double mutation (Ser509Phe + Arg529His) for two isolates and a unique mutation (Ser531Leu) for the last isolate.

Conclusions: This study shows a high prevalence (80%) of MLS resistance among *A. schaalii* recovered from HS patients for which CLI is commonly used. As previously described, it is due to Erm(X), suggesting that *A. schaalii* could be an important reservoir for this resistance determinant. Also, this is the first report of RIF resistance in this species with characterization of the corresponding molecular mechanism. Taken together, this confirms the risk of emerging resistance to both CLI and RIF in HS patients, which may be due, at least partially, to the antagonistic effect between these two molecules.