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Molecular bacterial typing methods

Infections by Streptococcus agalactiae among neonates in Portugal: 2005-2014

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Background: Group B streptococci (GBS) is a leading cause of neonatal invasive disease in most countries. We analysed 196 GBS isolates recovered from invasive infections in newborns in Portugal, between 2005 and 2014, with the aim of documenting changes in serotypes, antimicrobial resistance patterns and genetic lineages and evaluating their association with early-onset (EOD) or late-onset disease (LOD).

Material/methods: All GBS isolates were serotyped and assigned to sequence types (STs) according to the multilocus sequence typing scheme. Susceptibility to penicillin, erythromycin, clindamycin, vancomycin, chloramphenicol, tetracycline and levofloxacin was tested by disk diffusion according to the CLSI guidelines. The surface protein encoding genes *bca*, *alp2*, *alp3*, *alp4*, *eps* and *rib* and macrolide and tetracycline resistance determinants were assessed by PCR.

Results: Serotypes III (59%) and Ia (22%) were dominant in the population, together accounting for 158 isolates. Serotypes V, II, IV, VI, VIII and IX were also detected, the last two for the first time in Portugal. The isolates grouped into 33 STs and 7 clonal complexes (CC). CC17 included 50% of all isolates, highlighting the importance of the hypervirulent lineage represented by serotype III ST17/*rib*. Serotype Ia was found mainly in CC23, previously reported as dominant among invasive infections in non-pregnant adults in Portugal. Within CC23 we could distinguish two genetic lineages, ST23/*eps* (n=28) and ST24/*bca* (n=16), the latter at a higher frequency when compared to our previous studies. While ST23 and its SLVs were associated with EOD, ST24 and SLVs were dominant among LOD cases ($p<0.001$), indicating that within the same CC particular sublineages may be better adapted to specific age groups or disease presentations.

All isolates were susceptible to levofloxacin, penicillin, and vancomycin; and 3 were resistant to chloramphenicol. Most isolates were resistant to tetracycline (n=168; 86%) and carried *tetM* (n=161%). Macrolide resistance increased during the study period (Figure) ($P<0.001$), for an overall resistance rate of 15.3%. Most of the isolates presented the $cMLS_B$ phenotype associated with the *erm*(B) gene. Macrolide resistance was overrepresented among serotype Ib isolates ($P=0.004$) and particularly associated with CC1 ($P<0.001$) and CC19 ($P=0.003$) while most isolates of the hypervirulent CC17 lineage were susceptible ($P=0.003$).

Conclusions: The GBS recovered from 10 years of neonatal invasive infections in Portugal were dominated by small number of genetically distinct lineages that were present over a significant time span. The stability and dominance of a few lineages, namely of the hypervirulent serotype III ST17/*rib*, which remains responsible for the majority of infections in spite of continuous antibiotic and immune selective pressures, suggest that these are extremely well adapted to this niche. Still, we have also identified seemingly regionally successful clones, raising the possibility of an ongoing selection and expansion of specific virulent GBS clones.