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ePoster Session

Characterizing pathogens using molecular techniques

Group B streptococcal (GBS) colonization among non-pregnant adults in Portugal: prevalence, serotypes, genetic lineages and resistance

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Background: While GBS colonization is well studied among pregnant women, much less is known among non-pregnant adults. We have determined the prevalence of GBS colonization among non-pregnant adults in Portugal and identified the genetic lineages of the colonizing GBS that could uncover particular tropisms for certain age groups. By comparing this data with that of infection we wanted to evaluate if adult colonization could be a source of infections in both adults and neonates.

Material/methods: The study was approved by the institutional review board of Faculdade de Medicina. Non-pregnant adults (n=288) in the community were screened for GBS colonization by swabbing the mouth, vagina and rectum. Urine was also collected and cultured. GBS isolates were serotyped and characterized by multilocus sequence typing (MLST). Antimicrobial susceptibility testing was performed by disk diffusion according to the CLSI guidelines. The presence of macrolide resistance conferring genes, surface protein encoding genes *bca*, *alp2*, *alp3*, *alp4*, *eps* and *rib*, and the pilus islands (PI) 1, 2a and 2b were tested by PCR.

Results: 82 volunteers (28%) were colonized with GBS in one or more body sites. Colonization rates were similar in men (29%) and women (28%), and increased with age (P=0.03). Overall the serotypes were differentially distributed among age groups (Figure). Serotype V was the most frequent serotype in the older population (P<0.05), in agreement with its dominant role in invasive disease cases in older adults. We detected a surprisingly high proportion of serotype Ib in colonization (30.5%), in contrast to its modest representation in invasive disease cases in Portugal. Furthermore, this serotype was mostly represented by ST1/*alp3*/PI-1+PI-2a, a genetic background shared with serotype V and previously associated only with the latter. CC17 and CC23, the most frequent CCs causing invasive disease in newborns, were moderately represented in colonization (13% each).

Overall, macrolide resistance was 42%, significantly higher than that found in invasive infection in neonates (15%) and adults (13%). Macrolide resistance was associated with serotype V in multiple studies from diverse geographic locations. In our study it was overrepresented in the new serotype Ib ST1/*alp3*/PI-1+PI2a genetic lineage (P=0.002). Taken together, these data suggest that this new serotype/genotype combination may have arisen from a capsular switching event.

Conclusions: Overall, the genetic lineages colonizing asymptomatic non-pregnant adults appear to be the same as those causing invasive disease in multiple age groups and support the hypothesis that colonizing strains are likely to be the source of infection. An exception is the significant prevalence of a new highly resistant serotype Ib/ST1 genetic lineage, suggesting the recent emergence of serotype Ib within a genetic background that was previously associated with serotype V. Further studies will determine if this lineage will become established as significant cause of invasive infections.

