O554

Abstract (oral session)

Capsular switching in a group B Streptococcus ST-17 hypervirulent clone

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Objectives: The capsular polysaccharide (CPS) is a virulence factor of Group B Streptococcus (GBS), a major neonatal pathogen. Ten CPS serotypes are now recognized: Ia, Ib, II to IX. GBS population studies by Multi Locus Sequence Typing (MLST) and genome sequencing showed that five main clonal complexes, CC1, CC10, CC23, CC19, and CC17 account for most strains in humans. However, there is no strong correlation between CPS serotype and MLST, and whole genome sequence comparison demonstrated "en bloc" horizontal gene transfer of CPS locus genes between GBS strains. Up to now, the worldwide spread of the hypervirulent GBS clone ST-17 responsible for neonatal meningitis (> 80%) was restricted to CPS type III. Here, we described a CPS switching that have occurred in three unrelated GBS ST-17 clinical isolates. Methods: All GBS strains received at the National Reference Centre for Streptococci were serotyped using a multiplex PCR assay CPS type determination and presence of the ST-17 specific surface protein encoding gene hvgA a gene was investigated by a real-time PCR assay. Three out of the 1,281 GBS ST-17 strains studied were CPS type IV and further characterized by sequencing of the entire cps locus, conventional MLST typing and a cluster analysis including additional 7 housekeeping genes, and pulse field gel electrophoresis. Results: The 3 GBS strains selected were responsible for invasive infections (neonatal n=1 and adult n=2). PFGE analysis of SmaI restricted total DNAs revealed that they were epidemiologically unrelated. PCR sequencing of the entire cps locus confirmed that they were actually CPS type IV although their genome backbones was indistinguishable from that of an ST-17 clone, as demonstrated by using an extended MLSTyping scheme. We also showed that besides hvgA, these strains possess the genes srr2 and spb1 encoding other specific ST-17 surface-anchored proteins. Conclusion: Several studies have suggested a role of recombinational replacements in genome evolution of GBS. Until this report, capsular switching was described within non-ST17 GBS strains and was thought to contribute to the rise of new-serotype-genotype combination, allowing escape of immune pressure. Here, we characterized ST-17 hypervirulent strains that have switched their CPS type III to the less frequent, but emerging CPS type IV. Our results indicate that CPS switching must be taken into account in the design of conjugated GBS CPS multivalent vaccines development.