Multiple-Clones of Group-B Streptococci Clinical Isolates with an Unusual Erythromycin-Susceptible and Clindamycin-Resistant Phenotype

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Objective:
Group-B Streptococci (GBS) is a common cause of neonatal diseases such as sepsis and meningitis. Macrolides are the recommended second-line agents and therapeutic alternative for mothers with a penicillin allergy. The National Surveillance (WHO-ARG, 70 Hosp) reported increase in the resistance to erythromycin (ERY) and clindamycin (CLI) from 7.2% and 3% in 2005 to 12.8% and 5.7% in 2007. Resistance to lincosamides in GBS is most commonly mediated by Erm-type methylases, but ribosomal mutations and Lnu-type nucleotidyl-transferases were also described. LnuB enzyme was only described in 3 GBS worldwide, 2 from Canada and 1 from the US. During 2006-2008, six GBS expressing an unusual ERY susceptibility and CLI resistance (L-phenotype) by disc diffusion were submitted to the National Reference Laboratory (INEI) for molecular characterization. The objective of this work was to characterize the mechanism of resistance and to evaluate the relationship between these isolates.

Methods:
Six GBS from 3 hospitals from 3 different cities (PYR, RAW and FER) displaying an L-phenotype were recovered from recto-vaginal screening culture. MICs by agar dilution were performed according CLSI guidelines. Detection of mefA, ermA, ermB, lnuA and lnuB genes was carried out by PCR. DNA sequence was determined by standard methods. Molecular typing was assessed by ApaI-PFGE.

Results:
All GBS were susceptible to ERY (≤ 0.12 mg/L) and azithromycin (≤ 0.25 mg/L), and resistant to CLI (4 mg/L) and lincomycin (64-128 mg/L). All six isolates were positive only for lnuB gene, and DNA sequence was confirmed by sequencing. Four clones were discriminated: A to D. Clone A (3 strains) was detected in FER hospital, and the remaining were from PYR (clone B), RAW (clone C) and FER (clone D) hospitals.

Conclusions:
Here we are describing the polyclonal emergence of GBS harboring the lnuB gene in Argentina. The continuous surveillance of the antibiotic susceptibility of GBS is necessary; not only to detect known resistance phenotypes, but also to identify newly acquired resistance mechanisms.