O314
2-hour Oral Session
Updates in bloodstream infection epidemiology and management

Genomic tracks behind spread of bacteraemic group A Streptococcus type emm89 in Finland, 2004-2014

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Background: Invasive Group A streptococci (iGAS) cause severe infections as bacteremia, toxic shock syndrome and necrotising fasciitis. *Emm* gene typing is used to estimate genetic relationships among strains causing disease. During last decade increasing numbers of *emm*89 iGAS cases have been reported in many countries, including Finland. In this study, the epidemiological characteristics of *emm89* iGAS cases in Finland during 2004-2014 were investigated and linked to whole-genome sequencing (WGS) data obtained from corresponding strains. The aim was to assess pathogen genetic factors linked to this increase.

Material/methods: Cases of iGAS defined as bacteremia due to Group A *Streptococcus* were identified from National Infectious Disease Register (NIDR) notifications. Strains of type *emm89* from corresponding cases were available from National Reference Laboratory (NRL). NIDR notifications included data on age, gender, date of isolation, and place of treatment. To assess 30-day case fatality (CF), information about death within 30-day follow-up period after iGAS isolation was obtained from National Population Register. WGS was performed with Illumina technology. Fisher exact test and exact logistic regression were used to compare differences between iGAS *emm89* cases belonging to different genetic clades and subclades, respectively.

Results: A total of 1,928 iGAS cases were identified; 278 (range by year: 5-58) were *emm89* iGAS cases (median age: 55 years, range: 0-97; 50% male). WGS was performed on the 272 available *emm89* iGAS strains. We identified two genetically distinct clades arbitrarily designated clade 2 and clade 3. Six subclades (designated subclades A-F) were identified based on phylogenetic analysis of clade 3 core genomes. Both clades were present during 2004-2008, but clade 3 strains increased rapidly in frequency causing all *emm89* iGAS cases from 2009 onward. A total of 16 clade 2 cases (median age: 44 years, range: 2-66; 56% male) and 256 clade 3 cases (median age: 56 years, range: 0-97; 50% male) were identified. Cases with clade 3 strains were significantly older than those with clade 2 (p=0.04). 30-day CF was 0% for clade 2 and 8% for clade 3 (p=0.617). Subclade A included 74, subclade B 41, subclade C 14, subclade D 32, subclade E 46, and subclade F 49 cases. Subclade D had the highest estimated CF (19%), followed by subclade C (14%), subclade B (12%), subclade E (7%), subclade F (4%), and subclade A (3%). CF differed significantly between subclades (p=0.04). Subclade D had higher age-adjusted CF odds than subclade A (OR 7.2; 95%CI: 1.2-77.7; p=0.03).

Conclusions: A new *emm89* clone, clade 3, emerged in 2009 and spread rapidly in Finland. Patients infected with certain subclades of clade 3 were significantly more likely to die. A specific PCR-test based on WGS was developed in order to rapidly track the spread of subclade D in 2015 and beyond.