Groups C and G Streptococci - Friend or Foe ?

Non Group A Streptococci especially Group C Streptococci (GCS) and Group G Streptococci (GGS) which were long considered to be commensal organisms, are now recognized as etiological agents of exudative pharyngitis and pyoderma in many parts of the world(1). Post streptococcal sequelae which were once believed to be exclusive to infections caused by Group A Streptococci (GAS), are now known to occur following acute GGS and GCS infections. These organisms have also been reported to cause invasive infections similar to that caused by GAS. Due to the overlap in the disease spectrum of GCS/GGS with that caused by GAS, it is possible that the disease burden attributed to GGS is grossly underestimated. The taxonomy of these organisms is debatable; however, it has been proposed that human isolates of GGS/GCS be called *S. dysgalactiae subsp. equisimilis*(2). There is extensive homology between the gene sequences of the M protein of GGS/GCS and GAS, hence typing of these strains can be done by *emm* gene sequencing(3). Since these organisms are similar to GAS, share the same tissue niche and virulence genes, it may be important to identify chronic carriers who may be at risk of transmitting the infection to susceptible subjects.

We undertook a study to look for the carriage rate of GGS and GCS in children aged 5-15 years in Corporation schools in Chennai. We found that 65/595 (10.9%) specimens grew GGS/GCS while 41 of the 595 (6.9%) throat swabs collected grew GAS. Interestingly, though majority of the GGS GCS (82.6%) were *S. dysgalactiae subsp. equisimilis*, many of the children were found to carry strains of animal origin such as *S.equi subsp zooepedimicus* (17.4%). Molecular typing of these strains by *emm* gene sequencing indicated that the strains were extremely heterogenous and were represented by 17 different *emm* types; however, the type *stG6792* which has been previously reported in invasive strains was seen in all the biotypes of GGS and GCS.

Sergrouping and typing of beta hemolytic Streptococci is done only in a few laboratories; others report beta-hemolytic streptococcal isolates only as group A or non-group A, which would fail to identify these potentially pathogenic organisms. Hence clinical laboratories should be encouraged to perform group identification of all beta-hemolytic streptococci to evaluate the role of these organisms in invasive and non invasive infections.

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