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GROUP A BETA HEMOLYTIC STREPTOCOCCI ASYMPTOMATIC CARRIER STAGE - IS SURVEILLANCE REQUIRED?

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Group A beta hemolytic streptococci (*Streptococcus pyogenes*, GABHS) is a bacteria which causes a wide range of infections ranging from acute pharyngitis and tonsillitis to chronic sequelae like rheumatic fever and glomerulonephritis.[1] The throat and skin of the human host are the principal reservoirs for GABHS. GABHS is transmitted primarily person to person by either contact or droplet transmission. [2] It is listed as one of the cause for epidemic outbreaks of bacterial infections along with plague and cholera. [3]

The virulence factors and unique antigens on its surface has been elucidated in great detail and is well described. *Streptococcus pyogenes* or GABHS contain the Lancefield group A antigen on their cell surface and the major virulence factor of the organism is the M protein. The M protein is a fibrillar protein located at the cell wall surface and is encoded by the *emm* gene. Functionally, in the absence of opsonizing antibodies, the M protein is anti-phagocytic, inhibits deposition of complements, interacts with a large number of host proteins, possesses pro-inflammatory activities and contribute to mucosal adhesion.[4] Molecular mimicry of M proteins is also implicated in the pathogenesis of post-streptococcal glomerulonephritis and acute rheumatic fever. The traditional Lancefield classification system, which is based on

serotyping, has been replaced by *emm* typing, which has been used to characterize and measure the genetic diversity among isolates of *S pyogenes*. The targeted region of *emm* displays the highest level of sequence polymorphism known for an *S pyogenes* gene; more than 150 *emm* types have been described to date. [5]

The epidemiology of acute infections by GABHS and its long term effects as clinical syndromes has seen dramatic changes over the years. A low prevalence of rheumatogenic M types of *Streptococcus pyogenes* had contributed to a decline in the incidence of acute rheumatic fever in most developing countries in early twentieth century. [5] However, a resurgence of acute rheumatic fever was reported in the United states in the mid-1980s which was related to reappearance of mucoid strains of *S.pyogenes* type *emm* 18.1 [7,8]. From the late 1980s to 1990s, the epidemiology of *S. pyogenes* was dominated by a surge of invasive infections with high case-fatality rates, including necrotizing skin and soft tissue infections (such as necrotizing fasciitis and myositis) and streptococcal toxic shock syndrome in Europe and North America.[9] This was attributed to changes in the predominant *emm* clones as well as widespread transmission, strain displacement geographically and high prevalence in the community. [10,11] The

increased number and severity of group A streptococcal infections present special challenges to both the general practitioner and the infectious disease specialist, and the treatment of group A streptococcal infections has taken on greater importance. In addition, the acquisition of GABHS in the family environment poses problems for individuals in that environment who may have previously acquired rheumatic fever. Close contacts of primary cases of severe invasive GAS infections are at greater risk than the general population for development of colonization or superficial infection.

In the hospital environment, group A streptococcus can spread rapidly to patients with surgical wounds, burns or chicken pox or post-partum patients. Performing M-typing or comparing RFLP patterns is extremely important to determine if these cases originated from a common source such as an employee who is a carrier of GABHS. [12] Carriage rate is higher among children than adults as is the prevalence of infection. *S. pyogenes* carriage in the oropharynx may vary from 3% to 15%, the frequency of which will depend on the endemicity of acute infections [13]

Studies have reported an increase in the prevalence of specific GAS *emm* types (including *emm6* and *emm1*) in noninvasive disease or asymptomatic carriage in civilian and military populations[14,15] The duration of GAS carriage in the respiratory tract also has been identified as an important parameter that influences the probability that a strain will spread.

Investigations into hospital outbreaks have identified a range of transmission routes: colonised healthcare workers to patients, environmental sources to patients, and patient-to-patient transmission.

In the healthcare workers (HCW), throat colonisation is the most common source, although skin, vaginal and rectal colonisation has also been linked to outbreaks. [16, 17]

A study was initiated in our medical school to look into throat and nasal carriage of GABHS as a preliminary screening among medical students and dental students. Among a total of 200 volunteers, no isolate of Group A beta hemolytic streptococci was isolated. We attribute this to the age group under study (young adults, rather than children or hospital personnel) who are in the preclinical phase of their studies. A larger population based screening is required to effectively conclude the actual prevalence of GABHS in the population. Larger studies from various parts of the world would throw more light on the prevalence of carriage of beta hemolytic streptococcus among healthy individuals. With the recognition of increase incidence and severity of invasive group A streptococcal infections and changes in the epidemiology of its acute and chronic presentations, this microbe has returned to the forefront of infectious diseases. Surveillance for the carriage state has hence assumed greater importance in patient health care.

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