

Group A Streptococcal infections

Farida Jamal, MBBS, MSc, FRCPath

Department of Medical Microbiology & Immunology, Faculty of Medicine, University Kebangsaan Malaysia, Kuala Lumpur.

INTRODUCTION

Group A Streptococcus (GAS) is a common and versatile pathogen, encountered worldwide. It causes a variety of infections, particularly in children. These include pharyngitis and impetigo, noted for their non-suppurative sequelae which are mediated by immunological reactions. Rheumatic fever/rheumatic heart disease (RF/RHD) is a sequelae of GAS pharyngitis, whereas acute post-streptococcal glomerulonephritis (APSGN) is a sequela of both pharyngitis and impetigo. These associations confer a very important and unique position to GAS among human bacterial pathogens. Other manifestations of GAS disease include pneumonia, peri-tonsillar and retropharyngeal abscesses, otitis, sinusitis, erysipelas, cellulitis, necrotizing fasciitis, gangrene, myositis, puerperal sepsis, vaginitis, perianal cellulitis, scarlet fever and toxic shock syndrome (TSS). Epidemics of scarlet fever associated with a high mortality rate were a major possible health problem in the Western Hemisphere early in this century. Outbreaks of RF/RHD were also regularly encountered in the past. However, APSGN was and still is generally encountered in warmer climates, as a sequela of skin infection.

RESURGENCE OF SEVERE GAS INFECTIONS IN THE WESTERN HEMISPHERE IN THE LATE 1980s

The incidence of RF/RHD and severe GAS disease declined in the West during the post-antibiotic era, probably due to improved standards of living and better health care. However, it is believed that changes in GAS virulence and host immunity played an important role. In the late 1980s a resurgence of RF and severe systemic illness, including TSS and necrotizing fasciitis, associated with GAS infections were noted in Europe and the USA. Most of these occurred in apparently healthy children and adults, without any well defined underlying conditions. During the same period, changes in the laboratory characteristic of the prevalent strains were also observed. The proportion of M types 1, 3 and 18 had increased in the USA. By contrast, M types 4 and 12 had

decreased. Similar changes in M type distribution were observed in England.² The proportion of M type 1 increased from 1% in 1980s to 30% in 1987. Genetic studies suggested a characteristic restriction fragment profile in strains isolated from severe infections and TSS.³ These strains expressed streptococcal pyogenic exotoxin (SPEA) which was a possible virulence factor. A role for another exotoxin (SPEB) was also suggested, as fatal cases had low antibody levels for that toxin in Swedish studies.⁴ These toxins could act as super antigens. A virulent clone seemed to have emerged, possibly in a non-immune population, resulting in TSS, severe GAS disease and outbreak of acute RF. Most of the strains were encapsulated and exhibited the characteristic "mucoid" colonial morphology, a well recognised feature of virulent strains. The resurgence was focal and seems to have subsided.⁵

GAS INFECTIONS IN INDUSTRIALIZING COUNTRIES

GAS infection and its sequelae remain endemic in many industrializing countries. However, no increase in the incidence and severity of GAS disease was reported during the recent resurgence in Europe and the USA. Although under-reporting cannot be completely ruled out, it is unlikely that a change has gone unnoticed. Several other factors may account for this difference in the epidemiology of GAS disease. These include herd immunity towards an emergent clone, rendering it less virulent, or preventing it from colonizing the population and different prevalent strains.

A study conducted in Thailand suggests that the M proteins of GAS prevalent in this region may be different from those implicated in the recent resurgence in the West.⁶ Similar studies were conducted on strains isolated from well defined cases during the past five years in Kuala Lumpur.⁷ About 42% strains could not be T typed with antisera obtained from commercial sources. M typing was carried out in collaboration with the WHO Collaborating Centre, Minneapolis and only 2% strains were typable. These data suggested that the Malaysian strains belonged to M types

which were not included in the standard panel of antisera in Minneapolis. Also, very few of these strains had exhibited mucoid colonial morphology (6.3%), suggesting that they were not exceptionally virulent.

It is possible that yet uncharacterized and perhaps new M types of GAS exist in this region. This is supported by other studies on M typing of GAS isolated in this part of the world.⁸ In another study on GAS isolated in Asia, the presence of M protein was demonstrated although the strains were not M typable.⁹ There is a need to expand this work and include strains from other parts of Malaysia and the ASEAN region, isolated from well defined cases in hospitals and general practice setting.

The recent resurgence of GAS disease in the Western Hemisphere has stimulated interest in this area of research. Current trends include the development of a vaccine, with M protein as the focus. Such a vaccine may soon become available with industrializing nations as its markets. These developments necessitate improved understanding of epidemiology of RF/RHD and characterization of the strains of GAS implicated in different types of infections and non-suppurative sequelae in such countries.

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