


Commentary

Streptococcus pyogenes, the group A β-haemolytic streptococcus, remains a troublesome micro-organism. Good hygiene, antibiotics, and other attributes of a high standard of living have lessened the frequency of severe infection, but the bacterium has kept its virulence factors. Given a chance to spread among close contacts and to invade through broken skin, outbreaks with severe or fatal cases are possible. The paper by Engelgau and colleagues reminds us of this and shows how investigations should be done. Three aspects merit a comment: the organism, the route of infection, and the outbreak management.

The M type protein of S pyogenes is an important determinant of its pathogenesis and epidemiology. This is clearest in glomerulonephritis, which is the sequel of infections by a few M types (for example, M types 12, 49, 55, and 60). T type 1 is equivalent to M type 1 in this incident, but T typing is less specific for most other types. M type 1 is the type most often reported in overwhelming infection, but it is a common strain and typing is most likely to be done for the bacteraemic and other severe infections. The M type is associated with specific protective immunity, which should not be confused with the antibodies against exotoxins used in diagnostic tests, of which streptolysin O is the best known. This type specific immunity partly explains why streptococcal diseases fluctuate under the influence of herd immunity to common strains.

Chickenpox is one of several skin conditions that may become secondarily infected by S pyogenes. Burns, insect bites, and minor injuries are common portals of streptococcal skin infection, but seem to have less risk of bacteraemia with consequent shock, osteomyelitis, or arthritis. Children are the population in whom S pyogenes circulate most as they have least type specific immunity and closest contacts with other infected individuals.

Streptococcal infection is endemic, and virulent bacteria in susceptible children will cause the occasional death. Should we investigate the contacts of people who have severe streptococcal disease, when little is done in the numerous mild cases? Is this not shutting the stable door after the horse has bolted? The death of a child creates a feeling that ‘something must be done’. The paper by Engelgau et al shows that it is difficult to test everyone in a large group, and the time taken for laboratory tests diminishes the benefit of prophylactic treatment. The finding of lower carriage rates in children who had had antibiotics recently, suggests that good access to primary care is one reason why streptococcal infections have become less threatening in modern times.

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