

Meningococcal Disease: A truly frightening disease

Many a doctor will relate of a well first year college student joking with his friends in the dormitory in the morning but reportedly being slightly unwell just before lunch. Four hours the youngster is dead; a victim of the *Neisseria meningitidis* bacterium (meningococcus) that can cause meningitis or disseminate throughout the bloodstream causing septicaemia. It was baffling to earlier clinician that the same organism could present with such different manifestations. In fact the prestigious British Medical Journal in 1906 noted that: 'Many have doubted that 2 diseases, so different in their clinical course and in their distribution, could be due to one and the same organism.' Horder in 1918 referred to it as: 'With a disease so protean in its manifestations, it is not surprising that frequent errors should arise in diagnosis. The chief difficulty lies in not suspecting the presence of the disease.'

In July 2015 more than 30 000 youngsters from about 150 countries attended the World Scout Jamboree in Japan. At least 8 cases of meningitis in returning scouts were reported from Scotland and Sweden and an international alert was circulated. So what exactly is this devastating disease? Meningococcus is a uniquely human pathogen and its ecological niche is our nasopharyngeal passages. The bacterium has a capsule which contains different polysaccharides and this is used to classify them into about 13 serogroups of which 6 (A,B,C,W,Y and X) are associated with disease. Spread is via respiratory droplets when a carrier coughs or sneezes or via intimate contact such as

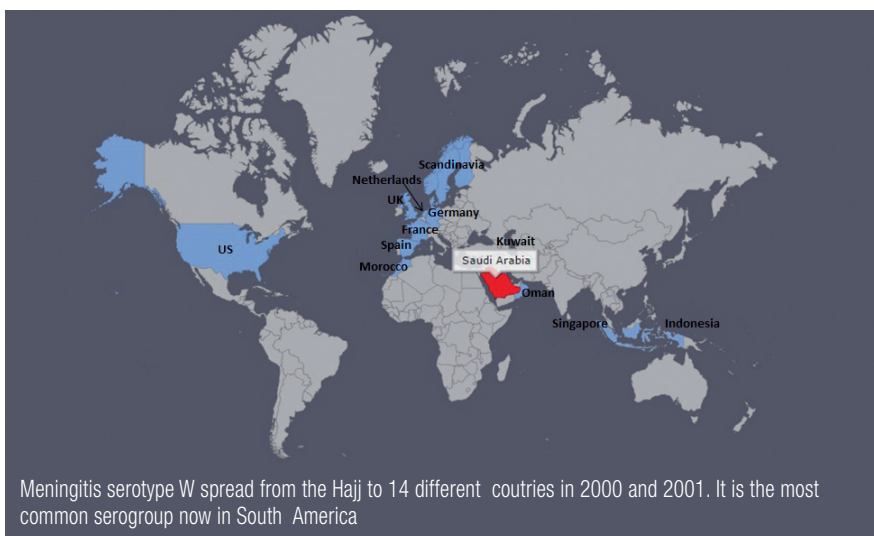
kissing. Crowded conditions, such as those found in dormitories, military barracks and mass gatherings, such as the annual Hajj in Saudi Arabia where up to 5 million pilgrims are crowded in relatively confined spaces, increases the risk of spread.

The burden of global meningococcal disease is well known to be in children under one year of age with a secondary peak in adolescents and young adults. Nasopharyngeal carriage is less than 1% in infants and peaks in young adults (up to 35%) and declines with age. Asymptomatic carriage has been documented in up to

86% of Hajj pilgrims. Carriage is normally transient and does not result in disease in the vast majority of cases and in fact leads to a degree of immunity. Immune deficiency, a preceding upper respiratory tract infection and smoking have been associated with an increased risk of carriage leading to invasive disease. A household contact of someone with symptomatic meningococcal disease has up to an 800 fold increase risk of contracting the disease compared to the general population.

The incidence of meningococcal disease varies markedly worldwide. Serogroups A, B, C and lately W account for up to 90% of the burden of the disease but again with tremendous variability across the globe. The Meningitis Belt of sub-Saharan Africa which stretches from Senegal in the west to Ethiopia in the east has the highest prevalence with a baseline endemic incidence of 10 to 20 cases per 100 000 of the population. Annual epidemics during the dry season from December to June and cyclical explosive

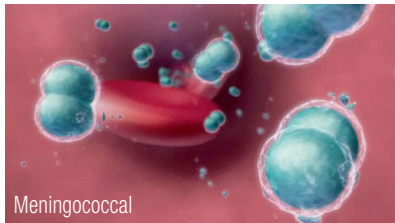
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Horder 1918



TRAVEL MEDICINE

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epidemics can increase this rate to 1000 per 100 000 population. These cycles are variable and can peak every 8-12 years. The epidemic of 1996-1997 infected a quarter million people and had a 10% mortality. A significant percentage suffered permanent neurological damage. During endemic periods the highest attack rates are observed in young children. During epidemics, older children, teenagers and young adults are also affected. In South Africa the pattern of meningococcal disease is characterised by sporadic cases throughout the year with occasional small clusters and a definite seasonal increase in winter and early spring. In 2014 most cases in the country were caused by serogroup W, except for the Western Cape where serogroup B predominates. The number of cases in South Africa has been low lately and it is not known whether this represents the bottom of a current cycle.

Asymptomatic carriage has been documented in up to 86% of Hajj pilgrims. In the general population carriage is normally transient and does not result in disease in the vast majority of cases and in fact leads to a degree of immunity. While



the incidence of infection in travellers to developing countries is about 0.5 per 100 000, it has been documented to be much higher in Hajj pilgrims (640 per 100 000) and their contacts upon returning home (up to 28 per 100 000) and peaks during meningitis belt epidemics (close to 1000 per 100 000).

A major outbreak of meningococcal disease, caused by serogroup A, occurred during the Hajj of 1987.

This could be traced to an epidemic that originated in China in the mid-eighties. From there it spread to Nepal and it was Nepalese pilgrims travelling to the Hajj who introduced it to Saudi Arabia. The disease was spread to other pilgrims and Saudi indigenous people. Returning pilgrims took it to their home countries in the African meningitis belt resulting in epidemics there. In Qatar, 15 cases were seen in a 21-day period following the Hajj. In 2000 and 2001 there were 1,300 and 1,109 cases of meningococcal disease respectively amongst pilgrims in Saudi Arabia, with the

Petechiae are caused by blood leaking out of the capillaries due to septicaemia



origin traced to pilgrims from the Meningitis Belt. In addition, in 2000, over 300 secondary cases were recorded in 14 different countries including the United Kingdom and France, which were associated with close contact with returning pilgrims. These outbreaks were predominantly caused by the serogroup W. Since then, the quadrivalent A,C,W,Y vaccine has become a visa requirement for all pilgrims, as it protects them against four strains of the bacterium.

Outbreaks of the disease are however still occurring. Between January and April of 2012 there have been 11,647 meningitis cases including 960 deaths in 10 countries in the meningitis belt; most of them being caused by W serogroup. In 2015 an outbreak of serogroup C was recorded.

An effective conjugate ACWY vaccine is used locally with a serogroup B vaccine not yet available in South Africa. The conjugate vaccine has several advantages over the older polysaccharide vaccines in that it is effective in children under two (who bear the brunt of the disease), can be boosted, lasts longer, eliminated carriage (and so prevents spread) and induces herd immunity. There have been massive gains in the meningitis belt against serogroup A after the introduction of a very affordable conjugate vaccine and even talks of eliminating disease caused by it. Whilst the incidence of the disease is currently low in South Africa, targeting infants, adolescents, young adults attending institutions and travellers to specific destination will ensure adequate control of this very frightening disease.

