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Brief Communication

Update on Microbiological and Epidemiological characterstics of Neisseria meningitidis

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Microbiology of Neisseria meningitidis

Neisseria meningitidis (Meningococcus) is a gram-negative diplococcus, bean shaped, non-motile, non-spore forming, and oxidase positive bacteria. Like other gram-negative bacteria, meningococcus is surrounded by outer membrane lipids, outer membrane proteins (OMPs), and endotoxic lipopoly-saccharides (LPS). Moreover, pathogenic meningococci are enveloped by a polysac-charide capsule attached to the outer membrane. Meningococci can grow on most non-selective routine laboratory media such as blood and chocolate agar in microaerophilic condition (5% O2 and 10% CO 2). This microaerophilic condition can be achieved by using either candle jar or gas generating kits. For rapid diagnosis and in cases in which cultures are negative because of previous antimicrobial therapy, meningococcal capsular poly-saccharide antigen can be detected in CSF, blood, and urine by latex agglutination procedures.

Meningococci are exclusively human pathogens and reveal more genetic diversity than most other pathogenic human bacteria. This is partly explained by horizontal intraspecies recombination and incorporation from closely related Neisseria species (1). Traditionally, strains were typed by using group specific antisera that recognize surface exposed epitopes on the capsule or the outer membrane. By this method, 13 serogroups (based on capsular polysaccharide), 20 serotypes (based on outer membrane proteins) have been identified (2). Further additional typing is also possible by using the antigenic properties of immunoglobulin A1 (IgA1) proteases and pilli (3).

Serotyping is of great importance for the development of vaccination strategies. However, although phenotyping chara-cterization may reveal close genetic relatedness, serotyping is not suitable for modern epidemiological purposes (1). Typing schemes, based on variation of a few genes which are probably under selection pressure will not identify the overall relatedness of the chromosomal genome of N. meningitidis (1). By using genetic approaches, in particular multilocus enzyme electrophoresis (MEE), which identifies naturally occurring allelic variations in multiple chromosomal house-keeping genes, a better insight into the epidemiology and clonal expansion of disease causing N. meningitidis can be gained (4). Other techniques which can be used for phenotyping characterization of N. meningitidis are DNA finger printing and PCR (5,6).

Epidemiology

The only natural reservoir of Neisseria meningitidis is the human nasopharyngeal mucosa and approximately 10% of the human populations harbor meningococci in the nose. Meningococci are transferred from one person to another by close contact or via respiratory droplets for a distance up to one metere (7). However, invasive disease caused by meningococci is relatively rare, as it occurs only

the following conditions have to be fullfilled (8). These conditions are (i) exposure to a virulent strain, (ii) colonization of the naso-oropharyngeal mucosa by virulent strain, (iii) penetration of the bacterium through the mucosa, and (iv) survival and multiplication of the meningococcus in the bloodstream. These properties are influenced by bacterial properties, climatological and social conditions, preceding or concomitant viral infections, the immune status, and age of the patient.

Meningococcal disease occurs word-wide as endemic infection (1, 8-10). Serogroups B and C cause the majority of infections in developed countries, while serogroups A and, to a lesser extent, C dominate in developing countries (1,8,-11). The incidence of meningococcal disease during the last 30 years varied from 1-3/100,000 population in most developed countries to 10-25/100,000 populations in some developing countries. These different attack rates reflect the different pathogenic properties of N. memomgotidis strains and different socioeconomic, environmental, and climato-logical conditions.

Sub-Saharan African countries have a special epidemiological pattern. This region, designated as "meningitis belt", which extends from Mali across the semiarid Sahel zone south of the Sahara, was first described by Lapeyssonnie in 1963. The countries included in this belt are Burkina Faso, Ghana, Togo, Benin, Niger, Nigeria, Chad, Cameroon, Central African Republic, and the Sudan (12). Later, Ethiopia, Mali, Guinea, Senegal, and the Gambia were added to form what is presently denoted as "the expanded meningitis belt" (9,13). In this region, meningococcal disease caused by serogroup A occurs in periodic recurrent waves. The disease attack rate rises at the end of the dry season and declines rapidly after the beginning of the rainy season (8-10, 12). During epidemic peaks, the incidence rate of the diseases may approach 1,000/100,000 inhabitants (9). Initially, a cyclic pattern with epidemics every 8-12 years was reported, but this pattern has

not been confirmed in later studies for most of the countries (9, 12, 15).

Since the end of the 1960s, widespread epidemics due to genetically closely related strains of N. meningitidis belonging to clonal complexes have occurred (1).

The biggest epidemics, which originated in Northern China and spread to the south and later globally, were caused by clones of serogroup A (1, 10). These subgroups spread to the Indian subcontinent in 1983 to 1987. In 1987, this clone reached the Middle East and caused a massive epidemic among pilgrims during the Haji in Mecca (1, 8,9,10). From here the organism was transported with the Hajis (14), causing epidemics in 1988 in the Sudan and Chad and, in the following years, in Ethiopia, Kenya, and Uganda (15). In the 1990s, the epidemic moved to countries south of the traditional meningitis belt, reaching Nigeria and South Africa in 1996 (16). During this period, more than 150,000 cases and, at least, 16,000 deaths ware reported in Africa (1, 10, 17). Interestingly, transfer of strains from the same clonal complex by Hajis to the United States and Europe did not elicit epidemics in these parts of the world (14, 18).

In Ethiopia, meningococcal meningitis was reportd for the first time in 1901, followed by outbreaks reported in 1935, in the 1940s and 1950s, in 1964, 1977, and in 1981 - 1983 and 1988-89 (19). The earlier epidemics were thought to have spread from West Africa to Ethiopia, while the 1988-89 epidemic spread with pilgrims returning from Mecca (15). Bacteriologic studies during the 1981-83

epidemic period showed that serogroups A and C were dominating (20) while, the during 1988-89 epidemic, serogroup A (with one isolate of B) was the most prevalent serogroup (11,21-22). Serogroup A was the cause of the reported recent epidemic of meningitis in Ethiopia (personal commu-nication).

Conclusion

In conclusion, microbiological and epide-miological studies by modern molecular methods have disclosed a complex picture of a few pathogenic meningococcal clones spreading world-wide. However, the mechanism by which potential pathogenic strains cause large-scale epidemics only in some regions of the world is largely unknown (14). It appears that the occurrence of invasive menigococcal disease is not determined solely by the introduction of a new virulent bacterial strain but also by other factors that enhance transmission and by the susceptibility of the population (23). Having a knowledge of an update information related to the micro-biological and epidemiological characteristics of meningococci provide basic information for microbiologists, epidemiologists and clinicians to design appropriate diagnostic, preventive, and curative measures, respectively, to the populations which are at risk, particularly the young children which are primarily the victims of this disease.

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