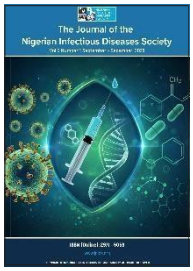
https://www.jnids.org ISSN: 2971-5059

Journal of the Nigerian Infectious Diseases Society



Review Article

Epidemic Meningococcal Meningitis

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Received: 20 Sep 2024
Revised: 20 Dec 2024
Accepted: 14 Jan 2025
Published: 24 Oct 2025

DOI
10.58539/JNIDS.2025

ABSTRACT

Neisseria meningitides, the major causative agent of bacterial meningitis was first identified and described by Vieusseux in 1805, before which time little was known about the disease. The disease was often fatal as there was no effective therapy at the time. Little was known about its pathophysiology, and severe disease was almost uniformly fatal, particularly when complications such as meningococcaemia developed. Endemic disease occurs nearly all the time, often causing only mild to moderate symptoms, but epidemic disease, identified later, has been a major public health concern, particularly in resource-poor countries. Once the causative organism was identified, a number of interventions were tried with variable degrees of success, among them chemotherapy and chemoprophylaxis with sulphonamides and other sulpha drugs, and penicillin. The introduction of sulphonamides for treatment of meningococcal meningitis (otherwise known as cerebrospinal meningitis, CSM) was a defining moment, as described by Kirk in 1950. The drug came in tablet form known popularly as M and B (made by May and Baker) so administration was problematic; initially, the tablets had to be crushed and administered parenterally in divided doses, which was a problem in Africa. However, sulphonamides reduced mortality from about 80% to 10%. Despite their success in reducing morbidity and mortality, these chemotherapeutic agents did not prevent epidemics from occurring periodically. Epidemics used to occur infrequently, but became more regular, occurring every five to ten years in the African meningitis belt, due largely to increasing resistance of meningococci to sulphonamides. Other causative agents of meningitis include Streptococcus Pneumoniae, Haemophilus influenza type b and Mycobacterium tuberculosis. This review will concentrate, in the main, on epidemic meningococcal disease with particular emphasis on Nigeria. Sporadic cases of meningitis are seen throughout the year, and in every continent, but severity of the infection varies from region to region. The factors that influence severity of disease are complex, but as a general guide these include nutritional status and competence of the immune system. The symptoms of meningitis are generally well known: fever (often high), backache and neck-ache both accompanied by stiffness, photophobia and generalized malaise. The onset is sometimes insidious but may be abrupt, beginning as a 'flu-like' illness. Vomiting may be a feature in some patients and a characteristic skin rash may be the first symptom, the latter believed by some scientists as evidence of immune complex deposition. Diarrhoea may occur and is sometimes so profuse as to be confused with cholera. The symptoms and complications of the disease are mostly self-limiting, excepting the most severe ones such as stroke, and disappear on successful treatment.

Key Words: Meningococcal Meningitis, Africa, Conjugate vaccine

INTRODUCTION

Sporadic cases of meningococcal meningitis (cerebrospinal meningitis, CSM) blend with other infections prevalent in sub-Saharan Africa, so do not require special attention. Epidemics of the disease are however a serious public health problem because they upset almost the entire healthcare effort, as most attention is diverted towards controlling them. These epidemics are deadly, disruptive and emotionally overwhelming among the affected community. Initially, sulphonamides were introduced as a treatment option, significantly reducing mortality from about 80% to 10% [1]. However, the causative agent of meningococcal meningitis, Neisseria meningitidis, became increasingly resistant to these agents, as well as to the Penicillins, rendering chemotherapy ineffective in preventing or curtailing outbreaks [2,3]. Given the failure of chemotherapy, it became imperative to look for other means of prevention and control. Vaccination was the obvious alternative, so vaccines were developed against the capsular polysaccharides (PS) of the meningococcus. The African meningitis belt was first described by Lapeyssonnie in 1963, shown below is the area in the world where epidemics of meningococcal meningitis mostly occur [4].

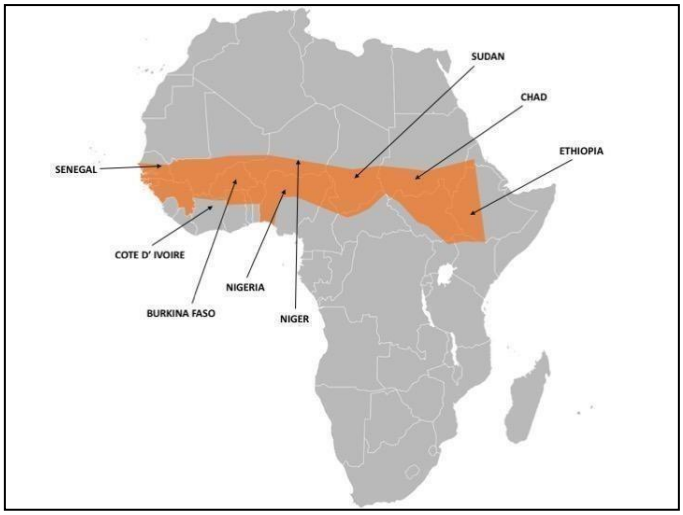

Figure 1. The African Meningitis Belt – after Lapeyssonnie 1963

The main serogroup responsible for epidemics in Africa was group A [5], and the early available vaccine was bivalent polysaccharide A and C (Institut Mérieux). Several workers conducted vaccination with the available polysaccharide (PS) vaccines, and although no large-scale trials of these vaccines were conducted, it was observed that they conferred some protection against epidemic outbreaks, though this was not sufficiently protective, nor did protection last long enough [4,6,7]. Several other workers had used group A vaccines to prevent epidemics but they also similarly showed that although antibodies to the polysaccharide coat of the meningococcus were produced as a result, they were not sufficiently protective. Polysaccharide antigens are not immunogenic enough and do not confer memory in the host, so are of limited use in young infants.

Pathogenesis

Neisseria meningitidis, the causative agent of meningococcal meningitis, primarily colonises the nasopharyngeal mucosa of human hosts. While nasopharyngeal carriage is generally asymptomatic, invasive disease occurs when the bacteria cross the mucosal barrier, enter the bloodstream, and eventually breach the blood-brain barrier, leading to meningitis [8].

N. meningitides has developed survival strategies to evade the host immune system. These virulence factors include polysaccharide capsules, lipopolysaccharides, type IV pili, and surface proteins [9]. The organism's polysaccharide capsule, a key virulence factor, helps evade host immune responses by inhibiting phagocytosis [10]. Several genetic and immunologic factors influence disease progression, including the host's genetic susceptibility and complement system deficiencies. In meningococcal meningitis, endotoxins like lipopolysaccharide or lipooligosaccharide (LOS) trigger an overwhelming inflammatory response, which leads to cerebral oedema, increased intracranial pressure, and neuronal damage. The endotoxin binds to CD14 on the cell surface in association with toll-like receptor R (TLR4) leading to the release of tumour necrosis factor-alpha (TNF-α), interleukins (IL-1, IL-6, IL-8, and IL-10), IL-1 receptor antagonist, plasminogen-activator inhibitor 1 (PAI-1), leukaemia inhibitory factor, and nitric oxide [11]. The concentration of the endotoxin and the extent of the inflammatory response determine the severity of the infection.

The mechanisms that enable meningococci to invade the meninges and cross the blood-brain barrier are not completely understood. Due to the absence of humoral and cellular immunity, once in the subarachnoid space, the organism continues to multiply uninterrupted [12,13]. The inflammatory response results in an increased permeability of the blood-brain barrier and an influx of neutrophils. Subsequently, the inflammatory products released result in clinically overt meningitis. Cerebral oedema also ensues, and since the brain cannot expand beyond the rigid skull, raised intracranial pressure from cerebral oedema may cause life-threatening brain stem herniation if it is not promptly addressed.

Invasive meningococcal disease (IMD) may also cause meningococcaemia which essentially results from endothelial injury with increased vascular permeability, thrombosis, and purpura [8]. The capillaries leak fluid and electrolytes into tissues leading to hypovolaemia, and peripheral and pulmonary oedema. Meningococcaemia may therefore, lead to septic shock, disseminated intravascular coagulation (DIC), and multi-organ failure.

Clinical presentation

Infectivity in meningococcal meningitis depends on a number of factors among them nasopharyngeal carriage [3], high temperature, low absolute or relative humidity, poor nutrition, low immunity due to lack of, or no, vaccination with the appropriate vaccines, poor environmental conditions, overcrowding, low, poor or no education (illiteracy or semi-literacy) and poverty [14,15]. The clinical features of the disease are fairly well-known to physicians, and during epidemic outbreaks even the unlettered are well aware of the signs and symptoms. This helps because there should be no delay in rushing suspected patients to a healthcare facility. Other than Streptococcus pneumoniae, the other main organisms responsible for the disease are Neisseria meningitides and Haemophilus inflenzae. Patients may have the classic triad of neck stiffness, fever, and altered mental status. Other symptoms include headache, backache, nausea, vomiting and an intense dislike for sunlight (photophobia). On examination, patients may have signs of meningeal irritation like the Kernig and Brudzinki's signs, other signs include fever, tachycardia, hypotension, petechia and ecchymotic rashes.

Severe disease leads to meningococcaemia with confusion, delirium, and loss of consciousness. Some of the severe complications of meningitis are attributable to immune complex phenomena, with complement activation leading to anaemia, splinter and petechial haemorrhages and kidney damage arising either from disseminated intravascular coagulation or release of pharmacological activators of hypersensitivity [16]. Some patients present with acute dehydration and cardiovascular collapse sequel to sudden fall in blood pressure. Such patients require resuscitation in addition to specific antimicrobial therapy.

Epidemiology

Sporadic cases of meningitis occur throughout the year irrespective of the factors that determine infectivity. These cases are usually mild although a few of them can be serious. Twelve serotypes of meningitis have been identified, but only six (A, B, C, W, X, Y) have epidemic potential [17]. The disease can affect any age group including adults, but infants and young children are mostly affected particularly during epidemics. Epidemics have a cyclical and geographic pattern of occurrence. Epidemics used to occur cyclically every 5 years, but have become more common lately. In terms of geographic distribution, the region affected most is the meningitis belt of sub-Saharan Africa, due mostly to the hot dusty and low humid conditions, in addition to low immunity, dirty environmental conditions and poor nutrition [14]. It is important to have a strong functional epidemic preparedness and response mechanism in order to detect epidemics early and initiate control action in a timely fashion. Once an epidemic has taken hold, it is difficult to achieve full control, and it usually runs its course until the onset of the rans when it suddenly terminates, as explained by Greenwood [18].

A severe epidemic of meningitis occurred in Nigeria in 1996 due to Neisseria meningitidis serogroup A, in which there were 109,580 confirmed cases and 11,717 deaths, giving a case fatality rate of 10.7% [19]. Later epidemics were recorded in 2003, 2008, and 2009 from Nigeria. From 2013 onward, Neisseria meningitidis serogroup C became the predominant cause of epidemics, with major outbreaks recorded in 2013, 2014, 2016, and 2017[20]. Between October 2022 and April 2023, Nigeria recorded and 1,686 suspected cases, 532 confirmed cases of meningococcal meningitis, with up to 124 deaths (case fatality rate of 7%) [21].

Meningococcal meningitis in Africa

Of all the main causes of meningococcal infection, the meningococcus serotype A is by far the major cause of epidemics in Africa, the incidence being as high as 70% in some areas [5]. A review of the intercontinental spread of epidemic meningitis shows that the disease arrived Africa relatively later than it did in Asia and Europe.

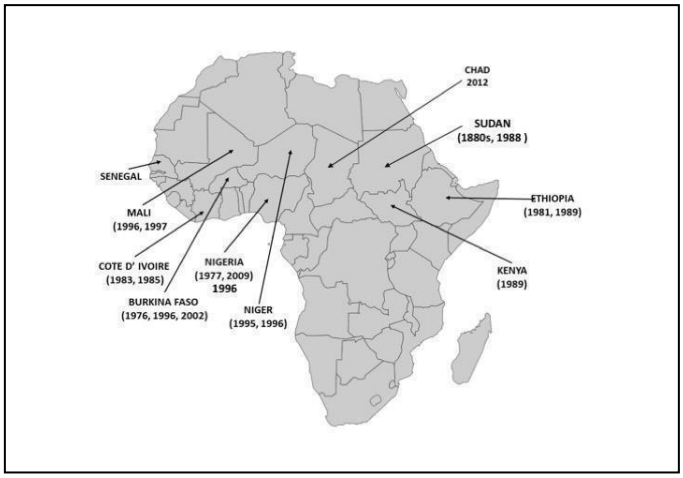

Figure 2. *Epidemic meningitis in Africa*

The first reported epidemic occurred in Geneva in 1805[1], whereas the first African epidemic occurred in French soldiers in Algiers in 1841[22]. West Africa recorded its first epidemic in the Cape Coast in 1900[23]. Those early West African epidemics were relatively mild but disruptive all the same. However, subsequent ones were more severe, for example, the one that occurred in northern Nigeria in 1921 christened "Dan Kanoma" after the town where it supposedly occurred [24], although there is now some uncertainty as to the authenticity of the location. Sporadic disease is seen all year round in the "African meningitis belt", in adults as well as in children, though the disease exerts a heavier toll in children aged 5 and below. The meningococcus resides in the nasopharynx and in polymorphonuclear lymphocytes (polymorphs).

Epidemics of meningococcal meningitis arrived in Africa nearly 100 years after they occurred in Europe. However, although the early epidemics were relatively mild, the later ones were particularly severe, deadly and disruptive. The mild ones were less severe and carried much lower mortality but were disruptive all the same.

It is well recognized that African epidemics of meningococcal meningitis are always disruptive, often leading health authorities to devote nearly all attention to its control, bringing the system to a standstill with respect to other healthcare interventions. These epidemics are therefore a major public health problem in sub-Saharan Africa, hence the urgent need for timely control measures. Early and timely detection of cases is necessary to determine when the epidemic threshold has been reached, so as to enable prompt intervention to curtail or stop its spread. For this to happen an efficient epidemiological surveillance must be in place, as well as epidemic preparedness and response mechanism for action. This is no easy matter, as these essential prerequisites for successful control require adequately trained public health personnel, timely action at the local level and constant and reliable two-way communication, and – of course – adequate funds.

Control or Eradication?

At some stage and for a long time, the debate became topical as to whether the best strategy for preventing epidemics was to control or eradicate the disease. It eventually dawned on researchers and public health officials that eradication may be ideal but that this utopian goal was unlikely to be achieved given the characteristics of the causative organism. Sporadic cases are impossible to prevent due to the fact that Neisseria meningitides reside in the nasopharynx of asymptomatic carriers in the community, thus serving as a constant source of infection. Attention therefore turned to control of epidemics by vaccination, now recognised as the best means of control. Another question then arose as to whether routine mass vaccination or selective vaccination should be employed because the countries most affected by epidemics of meningitis are middle- or low-income countries whose economic circumstances make it virtually impracticable for them to embark on routine mass vaccination with the available vaccines which were costly. These countries were also thought incapable of conducting the campaigns seamlessly due to executive incapacity [25].

Results from early vaccinations

Until the mid-twentieth century no policy existed as to the best means of preventing epidemics of meningococcal disease.

Researchers at the US National Institute of Child Health and Human Development in Bethesda had been conducting research on human immunity to the meningococcus. In the course of this research, Gotschlich, Goldschneider and Artenstein showed that susceptibility to meningococcal disease was closely related to a selective deficiency of humoral antibodies [26]. This was in the mid-1960s and since then not much was done to vaccinate against the disease. Greenwood and co-workers had conducted several investigations during outbreaks of the disease in Zaria, northern Nigeria in the course of which they showed that selective vaccination of affected communities led to termination of the epidemic [27]. Bosman et al. followed up persons vaccinated with the PS vaccine for 15 months during which no case of the disease occurred in any vaccinated persons [28], arguing that this was evidence of the effectiveness of the vaccine, though they also postulated that this might imply the development of 'herd immunity' conferred by the exercise.

The first large-scale mass vaccination with the PS vaccines was conducted in North-Eastern Nigeria between 1978 and 1981 when over 2.5 million persons were vaccinated with the bivalent A and C PS vaccine, with fairly good results [29]. Subsequently, the campaign was extended to other states in North-Eastern Nigeria and Plateau states during which nearly 7.5 million persons were vaccinated, and antibody levels determined by passive haemagglutination. High antibody levels persisted up to 4 years after vaccination [30], indicating that routine mass vaccination may only need to be undertaken every 4 to 6 years, making it cost effective. However, the poor antibody response in young infants and children coupled with short duration of the protection offered ([29–31]), led to WHO not recommending the routine use of PS vaccines in mass vaccination campaigns. Despite this, several workers held the view that until a conjugate vaccine was developed, the PS vaccines should be used to prevent epidemics of meningitis in Africa [28,32,33]. As the dispute continued the Bill and Melinda Gates Foundation gave a grant that enabled the development, production and deployment of the group A conjugate MenAfriVac vaccine that has been used to virtually eliminate group A epidemics in the meningitis belt [34]. It took fully 10 years to develop the conjugate vaccine, so in the meantime several workers continued vaccinating the susceptible populations with the PS vaccine, with fairly good results.

Selective vaccination with the PS vaccine was carried out in Chad Republic where a severe disease occurred, so it was no surprise the exercise failed [26].

In an earlier study in the 1970s Greenwood and coworkers had used selective vaccination in a small community in Zaria where the epidemic was terminated in the vaccinated community [27]. This was a community that was properly mapped before the vaccination began. This strategy is obviously unsuitable for large community such as the one in Chad. Among their findings in Chad, Spiegel forum that mass vaccination was efficacious, and that the exercise was feasible. In a similar fashion, Chippaux et al, after studies during a massive epidemic in Niger commented as follows:

"A massive outbreak of meningitis in Niamey in 1994-1995 followed a six-year period during which the mean rate of vaccine coverage remained <25%. These data indicate that in the meningitis belt, preventive immunization should avoid a greater number of deaths and be less expensive than vaccination campaigns performed after the epidemics have begun" [28].

A similar study in Rwanda showed further efficacy of polysaccharide vaccines [35]. These and other findings led Robbins and colleagues from the US National Institute of Child Health and Human Development to conclude that "The time has come to recognize that WHO strategy as the only policy for meningitis in sub-Saharan Africa, is too late too little" [32,33]. All this is now history as the development of a serogroup A conjugate vaccine, MenAfriVac, has rendered PS vaccines irrelevant in prevention or control of epidemic meningitis in Africa.

Control with conjugate vaccines

Despite pressure from these scientific researchers urging the use of PS vaccines for control of epidemic meningitis in Africa, WHO stood its ground, with relatively good – though not infallible – reason. This necessitated a personal appeal by a group of concerned workers, among them several of those quoted above, to make a direct appeal to the Bill and Melinda Gates Foundation for funding to develop a conjugate meningococcal A vaccine for use in sub-Saharan Africa (Robbins, personal communication, 2000). The response was immediate and positive, leading to the establishment of the WHO/PATH Meningitis Vaccine Project (MVP) which moved swiftly to mandate Serum Institute of India to produce the conjugate Men A vaccine at a relatively cheap cost to Africa [34,36]. The vaccine in question, MenAfriVac, was rolled out in 2010 beginning with Burkina Faso and Niger [37].

There was a dramatic response to the vaccine, as cases of meningitis dropped in three of the earliest countries vaccinated within a year [38]. By the end of the rollout, over 250 million persons were vaccinated and since then, no major epidemic of group A meningitis has occurred in the African meningitis belt .

New challenges

As happened earlier in Europe, sequel to the successful vaccination of the population with conjugate meningitis B vaccine, a new problem arose: as cases of meningitis B disease dropped sharply, so cases of group C meningitis replaced group B as the major cause of epidemic meningococcal disease. Nature abhors a vacuum, so it has come as no surprise that as meningococcal A disease was virtually eliminated as an epidemic problem in the African meningitis belt, other serotypes have now emerged as replacement groups in the region. Epidemics due to group C meningococci were reported in northern Nigeria [39], as were others due to groups W135 [40,41]. This paradoxical phenomenon of success begetting problems is no less a challenge in Africa as it was in Europe, so we were back to square one!

Mercifully, perhaps as a result of the experience from Europe, forward looking action was taken to develop a new pentavalent A, C, W, X, Y conjugate vaccine, which Nigeria was the very first to introduce this April.

CONCLUSION

Epidemics of meningococcal meningitis had plagued sub-Saharan Africa for 100 years, causing massive disruption of public health services as, during those epidemics, nearly all attention turned towards controlling the scourge in order to save lives. After decades of indecision as to the feasibility and efficacy of mass vaccination with polysaccharide vaccines, several large epidemics occurred, including the 1996 group A epidemic in Nigeria, a conjugate vaccine was developed and deployed across the meningitis belt of Africa beginning in 2010. This has virtually eliminated group A meningococcus as an epidemic serotype. However, replacement serotypes such as C, W135 and Y have emerged, but can now be addressed by the newly developed 5-valent (MENFIVE, MENCV5) conjugate vaccine.

Declarations Competing interests: None Declared
Funding: None
Authors' contributions: All authors contributed equally.
Acknowledgements: None
Source(s) of support: None
Conflicting Interest: None Declared

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