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MODERN APPROACHES TO THE DIAGNOSIS, TREATMENT, AND PREVENTION OF MENINGOCOCCAL INFECTION: A LITERATURE REVIEW

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Abstract

Background: Meningococcal infection, caused by Neisseria meningitidis, remains a significant global public health challenge characterised by rapid clinical progression, high case-fatality rates of 5–15%, and severe sequelae in survivors. Despite advances in vaccination and antimicrobial therapy, outbreaks continue to occur across all age groups and geographic regions, with a particularly high burden among infants and adolescents.

Aim: This review aims to synthesise recent evidence on modern diagnostic methods, treatment strategies, vaccination programmes, chemoprophylaxis, and public health prevention measures for meningococcal disease, with a focus on publications from 2021 to 2025.

Methods: A narrative literature review was conducted using PubMed, Scopus, Web of Science, and authoritative guidelines from the WHO, CDC, and ECDC.

Key Findings: Molecular diagnostics, particularly real-time PCR and multiplex PCR platforms, have substantially improved sensitivity and reduced the time to diagnosis, especially in antibiotic-pre-treated patients. Ceftriaxone-based empirical therapy remains the cornerstone of management, with intensive care support critical in fulminant meningococcaemia. Conjugate vaccines targeting serogroups A, C, W, and Y have significantly reduced disease incidence, while MenB protein-based vaccines (Bexsero, Trumenba) address the remaining diagnostic and preventive gap.

Conclusions: Early clinical recognition, rapid molecular diagnostics, evidence-based antibiotic protocols, and high vaccination coverage are mutually reinforcing pillars of effective meningococcal disease control. Ongoing genomic surveillance and next-generation vaccine development are critical priorities.

Keywords

meningococcal infection, Neisseria meningitidis, bacterial meningitis, meningococcaemia, sepsis, PCR diagnosis, antimicrobial therapy, meningococcal vaccination, chemoprophylaxis, epidemiological surveillance.

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INTRODUCTION

Meningococcal disease, caused by the Gram-negative diplococcus Neisseria meningitidis, constitutes one of the most alarming medical emergencies encountered in both paediatric and adult clinical practice. The disease manifests primarily as bacterial meningitis, septicaemia (meningococcaemia), or both simultaneously, and is characterised by an explosive clinical course that can progress from initial symptoms to death within 24 hours [1]. Even with optimal management, the case-fatality rate ranges from 5% to 15% for meningitis and may exceed 40% in fulminant septic shock, while 10–20% of survivors experience permanent sequelae including sensorineural hearing loss, limb amputation, and neurocognitive impairment [2].

Globally, meningococcal disease affects approximately 1.2 million individuals annually, with the highest incidence in sub-Saharan Africa — the so-called 'meningitis belt' — and sporadic outbreaks in Europe, the Americas, and the Asia-Pacific region [3]. Serogroup distribution varies geographically and temporally: serogroups A, C, W, and Y have historically predominated in Africa and certain European countries, while serogroup B accounts for the majority of cases in Western Europe, Australia, and North America [4].

The clinical urgency of meningococcal disease demands that clinicians and public health systems maintain a high index of suspicion, act on the earliest clinical signs — including the characteristic non-blanching purpuric rash, fever, photophobia, and neck stiffness — and initiate treatment without awaiting laboratory confirmation [5]. Simultaneously, robust epidemiological surveillance, contact tracing, and vaccination programmes represent the principal instruments for reducing community transmission and outbreak impact.

Over the past five years, major advances have occurred in molecular diagnostics, vaccine technology, and genomic surveillance that have transformed the clinical and public health approach to meningococcal disease. This review consolidates current evidence to support clinicians, microbiologists, and public health practitioners in applying best practice.

AIM OF THE REVIEW

To analyse recent scientific literature on modern diagnostic methods, treatment strategies, vaccination programmes, chemoprophylaxis, and prevention of meningococcal infection, with the purpose of providing a comprehensive, evidence-based reference for clinical and public health practice.

MATERIALS AND METHODS

This article is a narrative literature review. The literature search was conducted between January 2025 and May 2025 using the following electronic

databases and resources: PubMed/MEDLINE, Scopus, Web of Science, the WHO Global Health Library, the US Centers for Disease Control and Prevention (CDC) website, and the European Centre for Disease Prevention and Control (ECDC) database. Search terms included 'meningococcal infection', 'Neisseria meningitidis', 'bacterial meningitis', 'meningococcal vaccine', 'PCR meningococcus', 'chemoprophylaxis meningococcal', 'meningococcal epidemiology', and their combinations using Boolean operators.

Inclusion criteria: (1) original research articles, systematic reviews, meta-analyses, clinical guidelines, and epidemiological reports; (2) publication date 2021–2025; (3) indexed in Scopus or Web of Science, or published by WHO, CDC, ECDC, or equivalent national health authority. Exclusion criteria: pre-2021 publications (except landmark guidelines cited for context), non-peer-reviewed sources, grey literature without institutional endorsement, and duplicate publications. A total of 17 primary references were selected for citation in this review.

MAIN BODY

Etiology and Epidemiology

Neisseria meningitidis is an obligate human pathogen that colonises the nasopharyngeal mucosa asymptomatically in 5–15% of the general population, rising to 25% in adolescents and young adults [1]. Thirteen capsular serogroups have been described, of which serogroups A, B, C, W, X, and Y are clinically and epidemiologically most significant. The polysaccharide capsule is the primary virulence determinant, facilitating resistance to complement-mediated killing and phagocytosis.

Serogroup A has historically caused large-scale epidemics in the African meningitis belt, though the MenAfriVac conjugate vaccination campaign launched in 2010 has dramatically reduced its incidence [3]. Serogroup B now accounts for the largest proportion of cases in high-income countries, including the United Kingdom (>50%), Norway, and Australia. Serogroup C has been controlled in countries with routine conjugate vaccination, while serogroup W (particularly the hypervirulent cc11 clonal complex) has exhibited an alarming global expansion since 2000, causing outbreaks in South America, the UK, and the Hajj pilgrimage context [4].

High-risk populations include infants under 12 months, adolescents aged 15–19 years (reflecting both increased carriage acquisition and high-risk social mixing behaviours), immunocompromised individuals including those with complement deficiencies or asplenia, military recruits, and university students in residential settings [5]. Transmission occurs via respiratory droplets and close mucosal contact, with an incubation period of 2–10 days.

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Clinical Manifestations

The clinical spectrum of invasive meningococcal disease encompasses meningitis, meningococcaemia, and combined disease, presenting across a spectrum of severity from self-limiting meningitis to rapidly fatal septic shock [2]. Meningococcal meningitis presents with the classical triad of fever, neck stiffness, and altered consciousness, accompanied by photophobia, phonophobia, and Kernig's or Brudzinski's signs. In infants, the clinical presentation is frequently atypical, with bulging fontanelle, paradoxical irritability, and high-pitched crying rather than classical meningeal signs.

Meningococcaemia, or meningococcal septicaemia, is clinically distinguished by the rapid appearance of a petechial or purpuric rash — the pathognomonic non-blanching haemorrhagic rash — resulting from disseminated intravascular coagulation and microvascular thrombosis due to endotoxin-induced endothelial activation [2]. The rash may be absent or sparse in early disease; its identification requires systematic full-body skin inspection. Waterhouse-Friderichsen syndrome, characterised by bilateral adrenocortical haemorrhage leading to acute adrenal insufficiency, represents the most severe end of the meningococcaemic spectrum.

Septic shock develops when circulatory failure is compounded by myocardial dysfunction, vasodilatory collapse, and multi-organ failure. Paediatric presentations frequently involve a compensated shock phase characterised by tachycardia and prolonged capillary refill time before overt hypotension develops, emphasising the importance of clinical vigilance in children with non-specific febrile illness [6].

Modern Diagnostic Methods

The diagnosis of meningococcal disease integrates clinical assessment, microbiological culture, and molecular diagnostics. No single test is infallible, and the diagnostic strategy must account for the clinical acuity, the timing of antibiotic pre-treatment, and local laboratory capacity.

Blood cultures, obtained prior to antibiotic administration, remain the gold standard for definitive microbiological diagnosis and susceptibility testing, with sensitivity of 40–60% in untreated patients [7]. Cerebrospinal fluid (CSF) examination, including opening pressure measurement, cytochemical analysis, Gram staining, and culture, provides definitive evidence of bacterial meningitis; characteristic CSF findings include neutrophilic pleocytosis (>1000 cells/mm^3), elevated protein (>1 g/L), and reduced glucose (<40% of concurrent blood glucose).

Real-time PCR targeting the meningococcal-specific ctrA, porA, and sodC genes has revolutionised meningococcal diagnostics, achieving sensitivity

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exceeding 90% even in antibiotic-pre-treated patients and providing serogroup-specific results within 2–4 hours [7,8]. Multiplex PCR platforms, such as the BioFire FilmArray Meningitis/Encephalitis panel and the Unyvero system, enable simultaneous detection of the most common bacterial, viral, and fungal CNS pathogens from a single CSF sample in approximately 1 hour, substantially accelerating empirical therapy rationalisation [9].

Whole-genome sequencing (WGS) of N. meningitidis isolates has become the standard approach for genomic surveillance in reference laboratories, enabling precise clonal complex typing, identification of hypervirulent lineages, antimicrobial resistance profiling, and outbreak investigation [10]. While not a point-of-care tool, WGS is indispensable for informing vaccination policy and tracking serogroup shifts. Table 1 summarises the principal diagnostic modalities and their clinical characteristics.

Table 1. Modern diagnostic methods for meningococcal infection and their clinical significance.

Diagnostic Method	Sample Type	Advantages	Limitations	Clinical Relevance
Blood culture	Venous blood (10 mL)	Gold standard; allows antimicrobial susceptibility testing; serogroup confirmation	Low sensitivity (40–60%) after antibiotic pre-treatment; results in 24–72 h	Essential for definitive diagnosis and guiding targeted therapy
CSF microscopy and culture	Cerebrospinal fluid (lumbar puncture)	High specificity; enables Gram staining and cytochemical analysis	Requires experienced personnel; contraindicated if raised ICP; sensitivity drops post-antibiotics	Critical in suspected bacterial meningitis; guides initial antibiotic choice
Real-time PCR (monoplex)	Blood, CSF, nasopharyngeal swab	High sensitivity (>90%) and specificity; rapid result (2–4 h); positive after antibiotic initiation	Requires molecular laboratory; expensive; does not provide susceptibility data	Method of choice when blood cultures are negative; recommended by WHO and ECDC
Multiplex PCR (e.g., BioFire	Blood, CSF	Simultaneous detection of multiple pathogens	High cost; limited serogroup	Especially valuable in critically ill

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FilmArray)		within 1–2 h; reduces time to targeted therapy	discrimination; not universally available	patients with undifferentiated sepsis or meningitis
Latex agglutination antigen detection	CSF, urine, serum	Rapid bedside test; no specialized equipment required	Low sensitivity (50–70%); poor performance for serogroup B; largely superseded by PCR	Limited role in modern diagnostics; may be useful in resource-limited settings
Whole-genome sequencing (WGS)	Bacterial isolate	Comprehensive genomic characterisation; tracks outbreak strains; identifies virulence and resistance genes	Available only in reference laboratories; not a point-of-care tool; costly	Public health and epidemiological surveillance; hypervirulent lineage monitoring (cc11, cc32, cc41/44)

Treatment Strategies

The management of suspected meningococcal disease constitutes a medical emergency requiring immediate action. International guidelines — including those from NICE (UK), the Infectious Diseases Society of America (IDSA), and the WHO — consistently recommend that empirical antibiotic therapy must not be delayed pending microbiological confirmation [11]. In patients presenting to primary care or pre-hospital settings with a classical purpuric rash, intramuscular or intravenous benzylpenicillin should be administered immediately prior to hospital transfer, provided no documented penicillin allergy exists.

On hospital admission, third-generation cephalosporins — ceftriaxone (2 g IV every 12 hours in adults; 100 mg/kg/day in children) or cefotaxime (2 g IV every 4–6 hours) — constitute the empirical regimen of choice, providing broad coverage against meningococcus and the other principal causes of bacterial meningitis [11]. Once N. meningitidis is confirmed and penicillin susceptibility is established (MIC ≤0.06 mg/L), de-escalation to benzylpenicillin (2.4 g IV every 4 hours in adults) is recommended. The standard treatment duration is 5–7 days for uncomplicated meningococcal meningitis.

Management of meningococcaemic septic shock requires concurrent resuscitation using the Surviving Sepsis Campaign principles: early fluid resuscitation (10–20 mL/kg boluses of isotonic crystalloid, titrated to

haemodynamic response in children), vasopressor support with noradrenaline as first-line agent, and corticosteroid supplementation in refractory shock or suspected Waterhouse-Friderichsen syndrome [12]. Adjunctive dexamethasone (0.15 mg/kg every 6 hours for 4 days) is recommended in bacterial meningitis to reduce the inflammatory cascade, with greatest benefit demonstrated in pneumococcal meningitis but a favourable safety profile in meningococcal disease.

Antimicrobial resistance in N. meningitidis, while currently less prevalent than in other bacterial pathogens, requires ongoing vigilance. Reduced susceptibility to penicillin (MIC 0.12–0.5 mg/L) has been reported in up to 30% of isolates from some European countries, mediated by alterations in the penA gene encoding penicillin-binding protein 2 [10]. Ciprofloxacin-resistant isolates have emerged in France and Spain. Systematic antimicrobial susceptibility testing of all isolates and integration with WGS-based resistance profiling is strongly recommended.

Prevention and Control

Upon clinical or laboratory confirmation of invasive meningococcal disease, immediate public health notification is mandatory in most jurisdictions (Category A notifiable disease). The patient must be placed in droplet precautions for the first 24 hours of effective antibiotic therapy, after which transmission risk is considered negligible.

Close contacts — defined as individuals who have had prolonged close contact (>8 hours face-to-face or household contact within 7 days prior to symptom onset) with the index case — must be identified and offered chemoprophylaxis within 24 hours of the index case diagnosis [11]. Chemoprophylaxis regimens include:

• Rifampicin 600 mg orally twice daily for 2 days (adults); 10 mg/kg twice daily (children >1 month); 5 mg/kg twice daily (infants <1 month).

• Ciprofloxacin 500 mg as a single oral dose (adults only; avoid in pregnancy and children).

• Ceftriaxone 250 mg IM single dose (adults); 125 mg IM (children <15 years); 250 mg IM (pregnant women).

Contacts should also be screened for early signs of disease and advised to seek emergency care if symptoms develop. In outbreak settings (two or more confirmed cases within 4 weeks in a defined population), reactive vaccination of the at-risk community, guided by the circulating serogroup, is recommended [3].

Vaccination

Vaccination is the most effective long-term strategy for reducing meningococcal disease burden. Two categories of licensed meningococcal vaccines

are currently available: conjugate vaccines targeting polysaccharide capsule antigens of serogroups A, C, W, and/or Y (MenACWY conjugates), and protein-based vaccines targeting serogroup B (MenB) [13].

MenACWY conjugate vaccines (Menveo, Nimenrix, MenQuadfi) are immunogenic from 6 weeks of age and confer T-cell-dependent immunity leading to immunological memory and reduction of nasopharyngeal carriage. National immunisation programmes in the UK, USA, Australia, and numerous European countries include MenACWY in the routine childhood schedule at 12–15 months with an adolescent booster at 11–16 years. The meningitis belt immunisation programme using MenAfriVac has reduced serogroup A disease by over 99% in sub-Saharan Africa since 2010 [3].

MenB protein-based vaccines address the major unmet need left by ACWY vaccines. Bexsero (4CMenB, GlaxoSmithKline) contains four antigens — fHbp, NHBA, NadA, and PorA (P1.4) from OMV — and has been incorporated into the UK infant schedule since 2015, contributing to a >70% reduction in serogroup B infant disease [14]. Trumenba (bivalent rLP2086, Pfizer) has been approved by the FDA for use in adolescents. A key challenge is the antigenic diversity of serogroup B strains, which may not all express vaccine-targeted antigens at sufficient levels; the meningococcal antigen typing system (MATS) assay was developed to predict strain coverage.

Special vaccination recommendations apply to individuals at increased risk: those with complement deficiencies (C5–C9, properdin deficiency), functional or anatomical asplenia, HIV infection, and laboratory workers routinely handling N. meningitidis cultures, who should receive both MenACWY and MenB vaccines regardless of age [15].

Current Challenges and Future Perspectives

Several unresolved challenges continue to hamper optimal control of meningococcal disease. First, the dynamic evolution of circulating serogroups — most notably the global expansion of serogroup W cc11 and the re-emergence of serogroup X in Africa — underscores the need for continuous, genomics-informed surveillance to guide vaccine policy updates [4,10].

Second, the polysaccharide nature of serogroup A, C, W, and Y conjugate vaccines means they do not protect against serogroup B, which remains the predominant cause of endemic disease in Europe and North America. While MenB vaccines are increasingly incorporated into national schedules, cost and multi-dose regimen requirements limit global coverage, particularly in middle- and low-income countries.

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Third, emerging antimicrobial resistance — including reduced penicillin susceptibility and sporadic fluoroquinolone resistance — necessitates systematic susceptibility monitoring and readiness to adjust empirical and prophylactic treatment guidelines. The integration of WGS into national reference laboratory workflows provides an unprecedented opportunity to simultaneously characterise serogroup, clonal complex, and resistance determinants [10].

Future priorities include the development of pentavalent vaccines incorporating serogroup B antigens alongside ACWY coverage, the refinement of MATS-equivalent assays to improve vaccine coverage prediction, and the expansion of WGS-based surveillance networks to include low-income countries disproportionately affected by meningococcal outbreaks [13,16].

Table 2. Prevention strategies for meningococcal infection.

Prevention Method	Target Group	Advantages	Limitations	Practical Significance
MenACWY conjugate vaccine (Menveo, Nimenrix)	Infants ≥6 weeks, adolescents 11–18 yrs, immunocompromised, travellers to endemic regions	High immunogenicity; induces herd immunity; long-lasting protection; reduces nasopharyngeal carriage	No coverage for serogroup B; waning immunity; limited uptake in low-income countries	Core of national immunisation programmes; recommended by WHO and ACIP for adolescent catch-up
MenB vaccine (Bexsero, Trumenba)	Infants, adolescents, students entering university, asplenic patients, laboratory workers	Effective against serogroup B strains; cross-protective against divergent strains via NHBA/fHbp antigens	Multiple-dose schedule; variable cross-reactivity due to antigenic diversity; higher cost	Increasingly incorporated in national schedules (UK, Italy, Australia); addresses major gap in ACWY vaccines
Chemoprophylaxis with rifampicin	Close household contacts, healthcare workers with unprotected exposure	Highly effective in eradicating nasopharyngeal carriage; oral administration	2-day regimen; drug interactions (oral contraceptives, anticoagulants); rising resistance in some regions	First-line prophylaxis in most national guidelines; must be initiated within 24 h of diagnosis

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Single-dose ciprofloxacin (500 mg oral)	Adults who are close contacts; alternative when rifampicin is contraindicated	Single-dose convenience; high compliance; effective carriage eradication (>90%)	Not recommended in pregnancy or children; fluoroquinolone resistance emerging in some serogroup C strains	Preferred in adults due to ease of administration; widely used by public health authorities
Single-dose ceftriaxone (250 mg IM)	Pregnant women, children, contacts where oral agents are unsuitable	No significant resistance reported; safe in pregnancy and young children	Requires injection; parenteral route less convenient; not available in all primary care settings	Drug of choice for prophylaxis in pregnancy; also used in outbreak settings for rapid mass prophylaxis
Epidemiological surveillance and contact tracing	General population; close contacts; outbreak settings	Enables rapid containment; identifies secondary cases; guides vaccine deployment	Resource-intensive; dependent on timely notification and laboratory confirmation	Essential public health function; integrated with molecular typing (WGS) for outbreak investigation

DISCUSSION

The diagnostic landscape for meningococcal disease has undergone a paradigm shift over the past decade. Traditional culture-based methods, while essential for antimicrobial susceptibility data, are increasingly superseded in clinical urgency by molecular techniques. The ECDC's 2023 surveillance report confirmed that PCR now contributes to over 50% of confirmed meningococcal disease diagnoses in EU/EEA countries, particularly in settings where antibiotic pre-treatment is common [16]. The advent of multiplex PCR platforms capable of simultaneously identifying the full range of bacterial, viral, and fungal CNS pathogens within 1–2 hours represents a watershed advance in reducing the diagnostic uncertainty that historically drove broad-spectrum empirical antibiotic prescribing.

The imperative for early diagnosis cannot be overstated from a mortality-reduction perspective. A systematic review by Cohn et al. (2021) demonstrated that a delay of more than two hours from first clinical contact to antibiotic

administration was independently associated with a threefold increase in case-fatality rate [2]. This evidence reinforces international guidelines mandating pre-hospital penicillin in community settings and immediate ceftriaxone upon hospital admission, without awaiting CSF or culture results.

Vaccination effectiveness data from national programmes provide compelling evidence for the preventive paradigm. The UK's experience with the 4CMenB (Bexsero) infant programme — the first national programme globally — demonstrated 71.4% vaccine effectiveness against serogroup B disease in the first three years, with no significant safety signals beyond the expected reactogenicity profile [14]. The MenAfriVac experience in sub-Saharan Africa, with virtually complete elimination of epidemic serogroup A disease, represents one of the most successful public health vaccination campaigns in recent history [3].

A critical but frequently underemphasised dimension of meningococcal control is the quality of epidemiological surveillance and its integration with genomic data. WGS has enabled the prospective identification of emerging hypervirulent clonal complexes — notably cc11, which drives the global serogroup W expansion — and can detect importation chains and inter-continental spread in near real-time [10]. The convergence of genomic epidemiology with machine learning approaches for outbreak prediction represents a frontier that will increasingly inform proactive, rather than reactive, public health responses.

The principal limitation of this narrative review is inherent to the methodology: it does not involve a systematic search with pre-specified inclusion/exclusion criteria or quantitative meta-analysis. Consequently, conclusions regarding treatment efficacy or vaccine effectiveness are drawn from the existing evidence base rather than de novo synthesis. Future systematic reviews and network meta-analyses of emerging vaccine candidates and diagnostic modalities are warranted.

CONCLUSION

1. Meningococcal disease remains a life-threatening emergency characterised by rapid clinical progression and high mortality; early clinical recognition — particularly of the non-blanching purpuric rash — followed by immediate empirical antibiotic therapy with a third-generation cephalosporin constitutes the single most impactful clinical intervention for reducing case-fatality.

2. Real-time PCR and multiplex PCR platforms have substantially enhanced the sensitivity, speed, and serogroup specificity of meningococcal diagnosis, particularly in antibiotic-pre-treated patients in whom culture sensitivity is severely compromised; their integration into standard hospital diagnostic pathways is strongly recommended.

3. Conjugate MenACWY and protein-based MenB vaccines are highly effective in their respective target populations; achieving and sustaining high coverage in infants and adolescents, combined with targeted vaccination of immunocompromised and high-risk individuals, is the cornerstone of long-term disease burden reduction.

4. Chemoprophylaxis of close contacts with rifampicin, ciprofloxacin, or ceftriaxone must be initiated within 24 hours of index case diagnosis; the choice of agent should be guided by local resistance patterns, patient characteristics, and national guidelines to maximise carriage eradication.

5. Antimicrobial resistance in N. meningitidis, while currently limited, requires proactive monitoring through systematic susceptibility testing and WGS-based resistance profiling; integration with national surveillance databases is essential to detect and respond to emerging resistant lineages.

6. Whole-genome sequencing has become an indispensable tool for genomic surveillance, enabling real-time tracking of hypervirulent clonal complex expansions, serogroup shifts, and vaccine escape mutations; investment in WGS capacity across all income settings and international data-sharing is a global health priority.

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