BMJ Best Practice

Meningococcal disease

Straight to the point of care

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Summary

Meningococcal disease is an acute contagious illness, characterized by fever, petechial or purpuric rash, and signs of sepsis and/or meningitis.

May progress rapidly to septic shock, with hypotension, acidosis, and disseminated intravascular coagulation.

Highest rates of invasive infection are in children under age 5 years, especially under age 1 year, with a second peak occurring in 11- to 24-year-olds and a third peak in people age >65 years.

Diagnosis confirmed by isolation of *Neisseria meningitidis* from a normally sterile body site.

Confirmed meningococcal infection is treated with a third-generation cephalosporin. Where a cephalosporin is not appropriate, the choice of agent is based on the individual patient circumstances, antibiotic susceptibilities, and local availability.

Overall mortality rate is 10% to 15%. Between 10% and 20% of survivors of meningococcal meningitis have permanent neurologic sequelae, including sensorineural hearing loss, seizure disorders, blindness, motor disorders, and intellectual impairment.

Definition

Meningococcal infections are caused by *N meningitidis* , a gram-negative diplococcus that colonizes the nasopharynx. Bacteria invade the bloodstream or spread within the respiratory tract. A case is confirmed by detection of *N meningitidis* -specific nucleic acid (using a validated polymerase chain reaction assay) in a specimen obtained from a normally sterile site (e.g., blood or cerebrospinal fluid), or by isolation of *N meningitidis* from a normally sterile site or from purpuric lesions.[1] Probable cases include those where *N meningitidis* antigen is detected by immunohistochemical staining on formalin-fixed tissue, or in cerebrospinal fluid by latex agglutination.[1]

THEORY

Epidemiology

In the US, rates of invasive meningococcal infection declined since the late 1990s to a low of 235 (0.07 per 100,000 people) in 2020.[7] Reduced incidence of meningococcal disease has been linked to the introduction of meningococcal conjugate vaccines.[8] However, cases in the US have increased sharply since 2021. In 2024, 503 confirmed and probable cases were reported. This is the largest number of meningococcal disease cases reported in the US since 2013, with *Neisseria meningitidis* serogroup Y responsible for much of this recent increase.[7]

Most infections are sporadic, but about 5% of infections occur as part of outbreaks, caused predominantly by serogroups B, C, and Y.[1] The increasing incidence of serogroup W infections in the US, Europe, and Australia has been associated with a hypervirulent clonal strain.[9]

The highest rates of invasive infection are in children under age 5 years, especially those under age 1 year, with a second peak occurring in 11- to 24-year-olds and a third peak in people age >65 years.[10] Infections in infants and children ages 1-5 years are predominantly caused by serogroup B.[10]

Risk factors

Strong

young age

Bactericidal serum and mucosal antibodies elicited by nasopharyngeal colonization with both *Neisseria meningitidis* and other *Neisseria* species increase over the first decade of life. In the third trimester of pregnancy these protective antibodies are transmitted transplacentally from mother to fetus. The highest rates of infection occur in children under age 1 year, following the physiologic waning of this maternally derived antibody.[4] [19] Increased exposure to meningococcal carriers is probably responsible for a second peak in invasive infections in older adolescents.[20] [21]

complement deficiency

Hereditary or acquired deficiencies of the common complement pathway components C3, properdin, Factor D, Factor H, or C5-C9 are associated with high rates of invasive meningococcal infection and chronic meningococcemia.[4] [19] Serum from patients with these deficiencies kills bacteria poorly, implying that serum complement-dependent bactericidal activity is a critical host defense.

The prevalence of complement deficiency in patients with invasive meningococcal infections ranges from 0% to 25%, and is higher in patients with recurrent meningococcal infections and those with infections caused by unusual serogroups.[22]

use of eculizumab or ravulizumab

There is an estimated 1000- to 2000-fold increase in the risk of meningococcal infection among patients taking eculizumab, even if vaccinated.[23] Experts believe that the use of ravulizumab also confers an increased risk of invasive infection.

Eculizumab and ravulizumab are complement inhibitors that may be used in the treatment of paroxysmal nocturnal hemoglobinuria, atypical hemolytic uremic syndrome, and generalized myasthenia gravis.

immunoglobulin deficiency

Opsonophagocytic antibodies directed against the meningococcal capsular polysaccharide and against other bacterial antigens contribute to bacterial killing. Patients with congenital or acquired hypogammaglobulinemia, IgG subclass deficiencies, or functional immunoglobulin deficiencies are more susceptible to meningococcal infections, although rates of meningococcal infection in these patients are not as high as those of *Streptococcus pneumoniae* or *Haemophilus influenzae*.

HIV infection

People with HIV infection, in particular those with a low CD4 count or high viral load, are at increased risk of meningococcal disease.[24] [25] [26] [27] In the US, the meningococcal serogroup ACWY vaccination is recommended in all patients with HIV over the age of 2 months, regardless of CD4 count.[20]

asplenia or hyposplenia

People with anatomic or functional hyposplenia are at increased risk of severe meningococcal infections, although the risk is not as great as that for *Streptococcus pneumoniae* infections.[28]

college attendance

College students in the US and UK are at significantly higher risk for meningococcal infection than similarly aged peers who do not attend college.[27] [29] [30] Rates in first-year students, and particularly those living in dormitories, are 2- to 5-fold higher than in other students.[4] [31] This is presumably related to a rapid increase in rates of meningococcal colonization during the first year of college.[21]

close contact with invasive meningococcal infection

Over 95% of meningococcal infections in the US are sporadic. However, secondary cases may occur in contacts of patients with meningococcal infections.[32] Household contacts of people with meningococcal infection have infection rates ranging from 0.25% to 3%.[12] Contacts in schools and the workplace have a lower risk of infection (0.04% to 2.5%), with the highest rates in adolescents and people living or working in crowded conditions. Most secondary cases are diagnosed within 2 weeks of the index case. Outbreaks do occur within certain communities: for example, among gay and bisexual men in Melbourne, Australia in 2017, and in Florida in 2022.[33] [34] People who are in a community affected by an outbreak are recommended to get vaccinated or receive booster vaccinations, depending on their individual risk factors and vaccination status.[20] [35]

household crowding

Household crowding is a risk factor for invasive meningococcal infection in children and adolescents, likely because living close to others facilitates the transmission of bacteria between household members.[20]

travel to a hyperendemic or epidemic area

Serogroups A and C meningococcal infections are hyperendemic in the "sub-Saharan meningitis belt" extending from Senegal to Ethiopia, especially in the December-to-June dry season. During epidemics, attack rates may be as high as 1000 in 100,000 people.[36] Epidemics caused by W-135 and Y strains have occurred in several African countries, and serogroup A strain epidemics have occurred in India.[37] Travelers to these regions have increased rates of meningococcal disease.

Infections have also been reported in participants in the Hajj pilgrimage to Saudi Arabia, and their household contacts.

laboratory workers

Clinical microbiologists who have occupational exposure to *Neisseria meningitidis* are at increased risk of infection.[20] [38] In many cases, these people have reported activities that were likely to have exposed them to infectious droplets or aerosols, or had confirmed exposure in the form of handling a confirmed *N meningitidis* isolate or specimen in the 14 days prior to symptom onset.[38]

tobacco smoke exposure

Both smoking and passive exposure to tobacco smoke are risk factors for meningococcal carriage and invasive meningococcal disease.[20] [27] [39] Tobacco smoke impairs physical barriers to bacterial colonization, such as ciliary function and local cellular immune responses. It is toxic to respiratory epithelial cells, and bacteria may be better able to colonize and invade damaged respiratory epithelium.

Weak

recent move into a new community

People moving to a new living environment, particularly into a closed or semiclosed community such as a residential school, college dormitory, or military facility, have increased rates of meningococcal infection. Meningococcal carriage rates are high or increase rapidly in the first weeks of residence and are frequently sustained at elevated levels. The elevated risk of disease in these environments is likely related to higher rates of colonization in susceptible people.

respiratory infection

Up to 50% of people with invasive meningococcal infections have had recent symptoms of an upper respiratory infection, and many have identifiable respiratory copathogens.[40] [41] Respiratory pathogens may promote bacterial colonization by impairing local immunity or facilitating bacterial invasion. Coughing and sneezing may promote the transmission of *Neisseria meningitidis* to close contacts.

visiting bars/clubs

In adolescents and young adults, risk factors for meningococcal disease include visiting bars and clubs.[27] [42] Smoking is a strong confounder in many studies. It is likely that these crowded settings and close personal contact facilitate the spread of meningococci to susceptible people.

kissing

In adolescents and young adults, a risk factor for meningococcal disease is intimate kissing.[42] Smoking is a strong confounder in many studies.

Etiology

Meningococcal infections are caused by *Neisseria meningitidis* , an aerobic gram-negative diplococcus found exclusively in the human nasopharynx.

Meningococci colonize the human nasopharynx by adhering to nonciliated columnar epithelial cells.[11] Transmission occurs by inhalation of respiratory droplets or by direct contact with infected secretions.[12]

Approximately 10% of people are colonized at any given time, with peak rates in adolescents and young adults (10% to 35%) and the lowest prevalence in young children.[13] [14] Carriage may be transient or persist for months. Many colonizing strains of *N meningitidis* are not pathogenic. Rarely, however, pathogenic strains may invade the bloodstream, causing systemic illness and hematogenous infections, or spread to the lower respiratory tract.[1] Disease typically occurs within 10 days after colonization of a susceptible host by pathogenic strains.[4]

Virulence factors expressed by pathogenic strains include the capsular polysaccharide, lipooligosaccharide, pili, and other outer membrane proteins.[4]

Whereas *N meningitidis* strains that colonize the nasopharynx are diverse, most invasive disease is caused by a relatively small group of genetically related bacteria, suggesting these organisms have increased pathogenicity.[15]

Pathophysiology

Sepsis caused by meningococci is multifactorial.[16] Bacterial factors, chiefly lipooligosaccharide, stimulate a proinflammatory cytokine response.[17] [18]

The signs and symptoms of meningitis are a result of local inflammatory responses leading to cerebral edema, elevated intracranial pressure, and vascular thrombosis.

Hypotension results from increased vascular permeability and, in later stages of the illness, dysregulation of vascular tone. Both myocarditis and myocardial depression may contribute to poor tissue perfusion.

Bacteria release endotoxin, which triggers the inflammatory response; this in turn leads to activation of the coagulation cascade and downregulation of anticoagulant and fibrinolytic pathways. Disseminated intravascular coagulation is caused by these acquired deficiencies of protein C, protein S, and antithrombin III; increases in plasminogen activator inhibitor and thrombin-activatable fibrinolysis inhibitor; and reduced activation of protein C on endothelial cells. Resulting small-vessel thrombosis and skin necrosis cause purpura fulminans.

More rarely, thrombosis of larger blood vessels results in ischemia or infarction of digits or extremities.

Waterhouse-Friderichsen syndrome is caused by bilateral adrenal hemorrhage and necrosis with acute adrenal insufficiency.

Classification

Serologic classification[2]

Phenotypic classifications of the bacteria are based on the surface structures they elaborate. The most clinically relevant classification is serogrouping. Most pathogenic strains of *N meningitidis* possess 1 of 12 structurally and serologically distinct polysaccharide capsules:[2]

- Serogroups A, B, C, Y, and W-135 cause >95% of invasive infections.
- Strains can be further distinguished by serotyping of major and minor outer membrane proteins and by immunotyping of lipooligosaccharides.

Genomic typing[3]

Multilocus or whole-genome sequencing can classify distinct strains of *N meningitidis* and is useful for epidemiologic studies.

Case history

Case history #1

A 20-year-old college student presents to the emergency department with fever and confusion. The previous night he felt ill and complained of a headache. This morning he was difficult to rouse, seemed confused, and felt warm to touch. On physical exam he is acutely ill with fever, tachycardia, and mild hypotension. He opens his eyes and withdraws in response to painful stimuli. Nuchal rigidity and a few truncal petechiae are present.

Case history #2

A 9-month-old girl is brought to the emergency department with a history of fever and a rash. She was in good health until this morning, when she developed a fever, irritability, and poor feeding. In the afternoon her parents noticed purple bruises on her legs and trunk. On examination she is alert but appears acutely ill with fever, tachycardia, cool extremities, delayed capillary refill time of 5 seconds, and multiple ecchymoses on her legs and trunk.

Other presentations

Occasionally *Neisseria meningitidis* causes focal infections such as pneumonia, conjunctivitis, pericarditis, myocarditis, septic arthritis, endophthalmitis, peritonitis, and salpingitis.[4] Although most patients with meningococcemia present with overt signs of serious illness, approximately 5% of young febrile children with occult bacteremia are found to have meningococcal bacteremia (meningococcemia).[5] The majority of patients present with meningitis without bacteremia, or with concurrent bacteremia and central nervous system infection. Chronic meningococcemia is an uncommon syndrome characterized by several weeks to months of recurrent or continuous fever, headache, migratory arthritis or arthralgia, and a maculopapular or petechial rash.[6]

Recommendations

Key Recommendations

Meningococcal infections classically present with abrupt onset of fever and malaise, progressing rapidly (within 24 hours) to signs and symptoms of sepsis and/or meningitis. Typically, this is in infants under age 1 year, adolescents, or young adults.[10] [46]

A thorough history is important, focusing on place of employment, recent travel, place of residence, and a history of comorbidities such as immunoglobulin deficiency or asplenia, which are factors that indicate an increased risk of meningococcal infection.

Clinical evaluation

Many patients have a history of a recent mild respiratory illness. Early symptoms are nonspecific, including:

- Fever
- Irritability
- Poor feeding or anorexia
- Nausea and vomiting
- Lethargy
- Headache
- Leg pain or generalized aches
- Pallor, sore throat, and coryza[41] [47]

These are followed rapidly by more obvious symptoms and signs of serious illness such as:

- Thirst
- Respiratory distress
- Rash
- Cold hands and feet
- Altered consciousness
- Photophobia
- Hypotonia
- Neck pain and stiffness
- Seizures
- Tachycardia
- Hypotension
- Shock

Healthcare professionals should be aware that some of these classical signs of meningitis may not be present, and combinations of symptoms and signs may be more useful than individual clinical signs to identify serious disease.

In infants, a bulging fontanel and a characteristic high-pitched cry are characteristic signs of meningitis, particularly bacterial meningitis, though these are often absent.[41] [46] [47] Older adult patients with bacterial meningitis are more likely to present with altered consciousness, and less likely to develop headache and neck stiffness, than younger adult patients.[48]

Signs and features

A positive Kernig or Brudzinski sign indicates meningeal inflammation and is suggestive of meningitis but is present in a minority of patients.[48]

Distinguishing between meningococcal infections and less serious conditions is difficult in the early phases of infection, and it may be necessary to begin empiric antibiotic therapy while awaiting results of diagnostic tests. One study of meningococcemia in children identified signs of sepsis (cold hands and feet, leg pain, pallor or mottled skin, and, in young children, drowsiness and respiratory distress) as the most common indicators of serious illness.[41] Specific attention to these features, which typically develop in the first 12 hours of illness, may aid in the prompt recognition of meningococcal disease.

A rash is noted in 42% to 83% of patients, most commonly 4-18 hours after the initial symptoms of illness.[41] [49] Typically, the rash is a nonblanching petechial or purpuric exanthem, but a minority of patients may initially have nonspecific erythematous macular or maculopapular lesions. The rash may be less visible in patients with darker skin tones; soles of feet, palms of hands, and conjunctivae should be checked.

Although only a minority of patients with fever and a petechial rash will ultimately be found to have meningococcal infections, these findings should prompt the institution of empiric antibacterial therapy and investigations to exclude meningococcemia, unless an alternative diagnosis is likely.

Symptoms of meningitis typically develop 13-16 hours after the onset of illness and ultimately develop in 50% to 89% of patients.[41] [49] Clinical symptoms may progress even after the institution of effective antibiotic therapy.[4] [19] [46]

Routine laboratory tests

Complete blood count and differential, electrolytes, glucose, calcium, magnesium, phosphate, and coagulation profile should be obtained.

All patients should have blood cultures. Isolation of *Neisseria meningitidis* from a normally sterile body site (blood, cerebrospinal fluid [CSF], joint, pleural fluid, pericardial fluid, or aspiration or biopsy of a purpuric lesion) is the definitive test for diagnosis of invasive meningococcal infections.[1] [50]

Tests for rapid diagnosis

Rapid tests are most useful when it is desirable to individually tailor antibiotic therapy or if identification of a meningococcal infection has immediate public health implications, such as the need to provide antibacterial prophylaxis to close contacts.

A Gram stain of CSF or skin biopsy demonstrating gram-negative diplococci is suggestive of meningococcal infection in people with a compatible clinical illness. Biopsies of skin lesions typically demonstrate hemorrhagic vasculitis with a polymorphonuclear infiltrate. Gram-negative diplococci may be visible within vessel walls or intravascular thrombi. Detection of *N meningitidis* antigen in tissue specimens by immunochemical staining increases the sensitivity and specificity and should be ordered routinely if a biopsy has been taken.

Cerebrospinal fluid testing

CSF cultures should be considered in patients with signs and symptoms of meningitis. A lumbar puncture (LP) is contraindicated in patients with cardiovascular or respiratory instability, coagulopathy, or infection (including petechial or purpuric lesions) overlying the puncture site. Some experts recommend that LP be deferred in patients with classical presentations of meningococcal disease, as results of this test are unlikely to influence patient management.[51] However, clinical judgment is involved. Deferring an LP is prudent in a critically ill patient with a coagulopathy, but the information obtained by LP in a patient with milder disease may be very helpful in management and outweighs potential risks.

N meningitidis is isolated in up to 80% of people with clinically suspected meningococcal meningitis; therefore, a negative culture does not exclude infection.[52] [53] CSF should also be sent for glucose

and protein, cell count, and differential. The majority of patients with bacterial meningitis have a CSF glucose concentration of <45 mg/dL, or an absolute ratio of CSF to serum glucose concentrations of <0.4.[52] CSF protein is typically elevated. In rapidly progressive infections, values may be only slightly abnormal or normal. In bacterial meningitis, CSF cell counts are typically >100 cells/microliter, with a polymorphonuclear predominance.[48] [52] In fulminant infections and in young infants, CSF cell counts may be only mildly elevated or normal. In early bacterial meningitis, there may be a transient pleocytosis.[54]

Testing in patients who have been treated empirically

Gram stain of normally sterile body fluids and tissues and antigen detection may still be useful in patients who are treated with antibiotics before cultures have been obtained. Positive blood cultures are reported in up to 86%, and positive CSF cultures in up to 80%, of cases of clinically suspected meningococcal infection.[52] [53] Diagnostic yields are much lower in patients who have received antibiotics before cultures are obtained. Failure to isolate *N meningitidis* , therefore, does not reliably rule out meningococcal infection in people with a clinically compatible illness. Gram stains of CSF should be ordered if CSF is obtained.

The presence of group A, B, C, Y, and W-135 *N meningitidis* capsular polysaccharide antigen in CSF or serum may be detected by latex agglutination. These tests are useful in patients who receive antibiotic treatment before cultures are obtained, because antigen may persist in CSF for several days. The sensitivity of antigen detection ranges from 40% to 95%, but testing of serum or urine specimens is not recommended because of poor sensitivity and specificity.[50] The capsular polysaccharide of serogroup B *N meningitidis* and serotype K1 *Escherichia coli* is identical; therefore, neonatal meningitis caused by the latter organism may also produce a positive antigen detection test.

Polymerase chain reaction (PCR) amplification of *N meningitidis* DNA from blood and CSF is of particular value in patients who have received antibiotics before diagnostic samples are obtained. It is more rapid, sensitive, and specific than conventional microbiologic techniques.[55] [56] Real-time PCR assay can identify specific serogroups of *N meningitidis* from clinical isolates (typically blood or CSF).[57] Multiplex PCR (such as the QIAstat-Dx Meningitis/Encephalitis [ME] Panel or the BioFire FilmArray ME Panel) is used to rapidly screen for multiple causative pathogens in a single reaction.[58] [59] [60]

Focal infection tests

Cultures of non-CSF normally sterile body fluids (pericardial, pleural, synovial fluid) are indicated if focal meningococcal infection involving these areas is suspected: for example, when pericardial, pleural, or joint effusion is clinically apparent.

Nasopharyngeal cultures

Nasopharyngeal cultures are of limited usefulness in routine patient management. The isolation of *N meningitidis* from the nasopharynx of a patient with sepsis or meningitis is suggestive, but because nasopharyngeal colonization is common, it does not prove causality. Nasopharyngeal cultures may be helpful in identifying the serogroup of *N meningitidis* circulating in a community and whether immunization may be helpful in the prevention of secondary cases.

Diagnostic imaging

The role of imaging in the diagnosis of meningococcal infections is limited.

Computed tomography (CT)

CT of the head is commonly obtained prior to performing LP in patients with suspected bacterial meningitis (of any etiology) to exclude the presence of a focal intracranial lesion, although there is no conclusive evidence that LP increases the risk of cerebral herniation in this setting.[61] Although head CT is also frequently performed to exclude significantly elevated intracranial pressure, it is not a sensitive test for this purpose.[62] Most authorities feel that pre-LP head CT is indicated in patients with significant

alterations of mental status, focal abnormalities on neurologic exam, papilledema, antecedent focal central nervous system disease, or immunocompromise.[54] [61]

If head CT is requested prior to LP, antibiotics should be given immediately and not be delayed pending test results.

Other diagnostic imaging tests

Chest radiographs, joint films, or echocardiography are helpful in the diagnosis of meningococcal pneumonia and empyema, or hematogenous complications of meningococcemia such as septic arthritis and pericarditis.

A chest radiograph is indicated when pneumonia is clinically suspected (cough, dyspnea, tachypnea, chest pain, increased work of breathing, cyanosis, nasal flaring, intercostal or subcostal indrawing, grunting, decreased breath sounds, auscultatory crackles, pleural rub).

Bone and joint radiography is indicated when septic arthritis is suspected (joint erythema, swelling, warmth, pain, decreased range of motion, joint effusion).

Echocardiography is indicated when pericarditis is suspected (tachycardia, chest pain, muffled heart sounds, pericardial friction rub, poor peripheral perfusion, reduced arterial pulse pressure, pulsus paradoxus, increased cardiac size on chest radiography).

History and exam

Key diagnostic factors

rapid onset of illness and rapid deterioration (common)

Meningococcal infections typically evolve rapidly. A febrile illness persisting >24 hours without progression is unlikely to be caused by *Neisseria meningitidis* .[41]

fever (common)

Sustained fever occurs in most meningococcal infections. However, fever is not always present, especially in neonates.[47]

leg pain (common)

Up to 72% of children and adults with meningococcemia complain of leg pain, an early sign of sepsis.[41]

seizures (common)

Reported in 5% to 20% of patients with meningococcal meningitis.[41] One or more neurologic complications (impairment of consciousness, seizures, or focal neurologic abnormalities) are seen in 30% to 40% of patients.[49]

neck pain and stiffness (common)

Neck pain and stiffness are caused by meningeal inflammation. Neck stiffness is a common sign of meningitis but is not reliably present in young infants.

paresis (common)

May be caused by meningeal irritation.

headache (common)

An early nonspecific symptom.

photophobia (common)

May be caused by meningeal irritation.

altered mental status (common)

Altered mental status (including confusion or delirium) is often observed in patients with meningococcemia or meningitis.[4] One or more neurologic complications (impairment of consciousness, seizures, or focal neurologic abnormalities) are seen in 30% to 40% of patients.[49]

altered consciousness (common)

Common sign of severe infection, including meningitis and sepsis, and is observed in up to 50% of patients with meningococcal infections.[41] One or more neurologic complications (impairment of consciousness, seizures, or focal neurologic abnormalities) are seen in 30% to 40% of patients.[49] Older adult patients with bacterial meningitis are more likely to present with altered consciousness, and less likely to develop headache and neck stiffness, than younger adult patients.[48]

focal neurologic deficit including cranial nerve involvement and abnormal pupils (common)

May be caused by meningeal irritation and raised intracranial pressure, and exudates encasing the nerve roots. One or more neurologic complications (impairment of consciousness, seizures, or focal neurologic abnormalities) are seen in 30% to 40% of patients.[49]

hypotension (common)

Typically occurs late in septic shock and is a risk factor for death in meningococcal infections.[51]

shock (common)

Early manifestation of sepsis.

toxic/moribund state (common)

A sign of serious illness.

pallor or mottled skin (common)

Early sign of meningococcal sepsis.[41]

rash (common)

A hemorrhagic (non-blanching petechial or purpuric) rash is noted in 42% to 83% of patients with meningococcemia.

In a minority of patients, an erythematous, blanching, maculopapular rash may be initially observed, becoming hemorrhagic later in the course of disease.[41] A rash most commonly develops 4-18 hours after the initial symptoms of illness.[41] [49] The rash may be less visible in patients with darker skin tones - check soles of feet, palms of hands, and conjunctivae.[47]

cold hands and feet (common)

Early manifestation of sepsis.[41]

hypotonia (common)

May be a sign of severe systemic illness, particularly in young infants.

high-pitched cry (uncommon)

In infants, a bulging fontanel and a characteristic high-pitched cry are characteristic of meningitis, particularly bacterial meningitis, though these are often absent.[41] [46] [47]

Kernig sign (uncommon)

Severe stiffness of the hamstrings causing inability to straighten the leg when the hip is flexed to 90 degrees. Uncommon, but indicates meningeal inflammation and is suggestive of meningitis. Should not be relied on for diagnosis as sensitivity can be low.[48]

It is elicited by having the patient lie supine and flexing the thigh so that it is at a right angle to the trunk, and extending the leg at the knee joint. If the leg cannot be completely extended due to pain, this is considered positive.

Brudzinski sign (uncommon)

Severe neck stiffness causing the patient's hips and knees to flex when the neck is passively flexed. Uncommon, but indicates meningeal inflammation and is suggestive of meningitis. Should not be relied on for diagnosis as sensitivity can be low.[48]

bulging fontanel (uncommon)

In infants, a bulging fontanel and a characteristic high-pitched cry are characteristic of meningitis, particularly bacterial meningitis.[41] [46] [47]

Other diagnostic factors

irritability (common)

An early nonspecific symptom.

lethargy (common)

An early nonspecific symptom.

muscle ache/joint pain (common)

An early nonspecific symptom.

poor appetite or feeding (common)

An early nonspecific symptom.

nausea or vomiting (common)

Vomiting may be a nonspecific indication of illness or a symptom of elevated intracranial pressure.

thirst (common)

Common early symptom of sepsis.

coryza, sore throat, or cough (common)

Recent upper respiratory tract infection is a risk factor for meningococcal infection and is described in up to 50% of patients.

respiratory distress (common)

A sign of serious illness.

tachycardia (common)

A sign of serious illness.

Tests

1st test to order

Test	Result
blood cultures Traditional definitive test for the diagnosis of meningococcemia and should be obtained from all people with suspected meningococcal infections. Positive blood cultures are reported in up to 86%, and positive cerebrospinal fluid cultures in up to 80%, of cases of clinically suspected meningococcal infection.[52] [53] To optimize sensitivity, appropriate volumes of blood should be obtained (at least 1-20 mL in children, 20 mL in adults).	positive for *Neisseria meningitidis*
CBC and differential Patients with meningococcal infections may have an elevated WBC with a polymorphonuclear predominance. Patients with rapidly progressive infections, however, may initially have normal WBC. Neutropenia is not uncommon in severe infections. Thrombocytopenia and mild anemia are common.	leukocytosis, anemia, thrombocytopenia
electrolytes, calcium, magnesium, phosphate, glucose Patients with severe meningococcal infections often have metabolic abnormalities, especially acidosis, hypokalemia, hypoglycemia, and hypocalcemia.	acidosis, low Ca/Mg/PO_4, or hyper/hypoglycemia
coagulation profile (prothrombin time, INR, activated PTT, fibrinogen, fibrin degradation products) Coagulopathy is common in severe meningococcal infections. Disseminated intravascular coagulation (DIC) is caused by acquired deficiencies of protein C, protein S, and antithrombin III; increases in plasminogen activator inhibitor and thrombin-activatable fibrinolysis inhibitor; and reduced activation of protein C on endothelial cells.	evidence of DIC (prolonged thrombin time, elevated fibrin degradation products or D-dimer, low fibrinogen or antithrombin levels)

Other tests to consider

Test	Result
cerebrospinal fluid (CSF) Gram stain Gram-negative diplococci suggest meningococcal infection in people with a compatible clinical illness and may provide a rapid presumptive diagnosis. Gram stains are positive in 30% to 80% of patients with culture-confirmed meningococcal meningitis.[52] [53]	gram-negative diplococci
CSF cell count and differential In bacterial meningitis, CSF cell counts are typically >100 cells/microliter, with a polymorphonuclear predominance. In fulminant infections and in young infants, CSF cell counts may be only mildly elevated or normal. In early bacterial meningitis, there may be a transient pleocytosis.[54]	polymorphonuclear pleocytosis
CSF glucose, protein The majority of patients with bacterial meningitis have a CSF glucose concentration of <45 mg/dL or an absolute ratio of CSF to serum glucose concentrations of <0.4.[63] CSF protein is typically elevated. In rapidly progressive infections, values may be only slightly abnormal or normal.	low glucose and elevated protein
CSF culture *Neisseria meningitidis* is isolated from up to 80% of people with clinically suspected meningococcal meningitis; therefore, a negative culture does not exclude infection.[52] [53] Treatment with antibiotics before cultures rapidly reduces the yield of testing, but administration of antibiotics should not be delayed if diagnostic evaluation cannot be completed promptly.	*N meningitidis*
antigen detection in CSF Serogroup A, B, C, Y, and W-135 polysaccharide antigen can be detected by latex agglutination in 40% to 95% of patients with meningococcal meningitis.[50] Antigen may persist in CSF for several days, making this test useful in patients treated with antibiotics before diagnostic specimens have been obtained and for the rapid presumptive diagnosis of meningococcal infection. Serogroup B *Neisseria meningitidis* and serotype K1 *Escherichia coli* polysaccharides cross-react, so test results should be interpreted cautiously in neonates. Antigen detection testing on body fluids other than CSF, including serum or urine, is not recommended because of poor sensitivity and specificity.[50]	*N meningitidis* capsular polysaccharide antigen
chest x-ray Diagnostic imaging should be obtained when meningococcal pneumonia or a focal hematogenous complication of meningococcemia is suspected. Chest x-rays in meningococcal	may show lobar consolidation

Test	Result
pneumonia typically demonstrate lobar consolidation, with or without pleural effusion.	
CT head Commonly obtained prior to performing a lumbar puncture (LP) in patients with suspected bacterial meningitis to exclude the presence of a focal intracranial lesion. However, there is no conclusive evidence that an LP increases the risk of cerebral herniation in this setting.[61] Although head CT is also frequently performed to exclude significantly elevated intracranial pressure, it is not a sensitive test for this purpose.[62] Most authorities feel that pre-LP head CT is indicated in patients with significant alterations of mental status, focal abnormalities on neurologic exam, papilledema, antecedent focal central nervous system disease, or immunocompromise.[54] [61] If head CT is requested prior to an LP, antibiotics should be given immediately and not be delayed pending test results.	normal; elevated intracranial pressure or intracranial lesion if other pathologies
Gram stain of non-CSF body fluid A Gram stain of pleural, pericardial, or joint fluid or material from a skin lesion aspirate or biopsy demonstrating gram-negative diplococci is suggestive of meningococcal infection in people with a compatible clinical illness, and may provide a rapid presumptive diagnosis.	gram-negative diplococci
culture of non-CSF body fluid Positive culture of a normally sterile body fluid is indicative of focal meningococcal infections such as septic arthritis, pericarditis, endophthalmitis, peritonitis, and salpingitis. The sensitivity of cultures obtained from these sites is not well described. Because *Neisseria meningitidis* is part of the normal flora of the nasopharynx, its isolation from this site does not confirm an illness is due to this organism.	*N meningitidis*
immunohistochemical staining of skin lesion biopsy Biopsies of skin lesions in meningococcemia typically demonstrate hemorrhagic vasculitis with a polymorphonuclear infiltrate. Gram-negative diplococci may be visible within vessel walls or in thrombosed vessels. Immunostaining of *Neisseria meningitidis* antigens in tissue specimens increases test sensitivity and specificity.	positive for *N meningitidis*
echocardiography In purulent pericarditis, echocardiography demonstrates pericardial effusion, with or without cardiac tamponade.	possible pericardial effusion
joint x-ray In septic arthritis, radiographs may show typical findings.	may show joint space widening or soft-tissue swelling
polymerase chain reaction Polymerase chain reaction (PCR) amplification of *Neisseria meningitidis* DNA from blood and CSF is more sensitive and specific than traditional microbiologic techniques.	*N meningitidis* DNA

Test	Result
PCR may be helpful in diagnosing bacterial meningitis in patients who have been pretreated with antibiotics.[55] [56] Real-time PCR assay can identify specific serogroups of *N meningitidis* from clinical isolates (typically blood or CSF).[57] Multiplex PCR (such as the QIAstat-Dx Meningitis/Encephalitis [ME] Panel or the BioFire FilmArray ME Panel) is used to rapidly screen for multiple causative pathogens in a single reaction.[58] [59] [60]	

Differentials

Condition	Differentiating signs / symptoms	Differentiating tests
Streptococcus pneumoniae sepsis	• Purpura fulminans is more characteristic in meningococcemia than in sepsis caused by other bacterial pathogens.	• Blood or other body fluid cultures diagnostic.
Staphylococcus aureus sepsis	• Purpura fulminans is more characteristic in meningococcemia than in sepsis caused by other bacterial pathogens.	• Blood or other body fluid cultures diagnostic.
Streptococcus pyogenes sepsis	• Purpura fulminans is more characteristic in meningococcemia than in sepsis caused by other bacterial pathogens.	• Blood or other body fluid cultures diagnostic.
Coronavirus disease 2019 (COVID-19)	• Residence in or travel history to an area with local transmission of COVID-19, or close contact with a suspected or confirmed case in the 14 days prior to symptom onset. • May be difficult to distinguish clinically from bacterial pneumonia. In addition to fever, cough, and dyspnea, other common presenting symptoms include sore throat, myalgia, fatigue, and altered sense of taste and/or smell. • Patients with respiratory distress may have tachycardia, tachypnea, or cyanosis accompanying hypoxia.	• Real-time reverse transcription polymerase chain reaction (RT-PCR): positive for SARS-CoV-2 RNA. • It is not possible to differentiate COVID-19 from other causes of pneumonia on chest imaging.
Streptococcal pharyngitis	• Pharyngeal erythema and, frequently, tonsillar exudates and tender cervical adenopathy. Although the onset is abrupt, the infection does not progress rapidly.	• Throat culture or antigen detection is positive for beta-hemolytic *S pyogenes* .
Gonococcemia	• Typically presents with septic arthritis, tenosynovitis, and tender pustular skin lesions. Infections are most common in women and often	• Urethral, cervical, rectal, and oropharyngeal cultures, nucleic acid amplification tests, or blood cultures

Condition	Differentiating signs / symptoms	Differentiating tests
	begin within a week of the start of a menstrual period. Unlike meningococcemia, disseminated gonococcemia rarely progresses rapidly.	indicate gonococcal infection.
Leptospirosis	• The disease course may be bimodal, with fever, meningitis, and a rash, which may be hemorrhagic, developing several days after improvement in initial symptoms. Hepatitis, jaundice, interstitial nephritis, and myocarditis are common in severe *Leptospira* infections but are rare in meningococcal infections.	• Serologic testing confirms leptospirosis.
Rocky Mountain spotted fever	• Typically progresses more slowly than meningococcemia. The rash of Rocky Mountain spotted fever (RMSF) begins on the distal extremities and spreads proximally.	• Hyponatremia, hypoalbuminemia, and mild hepatitis are common laboratory abnormalities. Serologic testing confirms RMSF.
Ehrlichiosis	• Typically progresses more slowly than meningococcemia. Rash is uncommon, especially in adults.	• Serologic testing confirms ehrlichiosis. Inclusion bodies (morulae) may be seen in peripheral blood leukocytes.
Anaplasmosis	• Typically progresses more slowly than meningococcemia. Rash is uncommon.	• Serologic testing confirms anaplasmosis. Inclusion bodies (morulae) may be seen in peripheral blood leukocytes.
Infective endocarditis	• Bacterial endocarditis infrequently progresses rapidly to septic shock or meningitis, and patients typically have a longer duration of fever prior to presentation. A new or changed heart murmur, septic emboli, immunologic sequelae such as glomerulonephritis, and splenomegaly are common in endocarditis and not observed with meningococcal infections.	• Echocardiography typically demonstrates a valvular or intracardiac abnormality.

Condition	Differentiating signs / symptoms	Differentiating tests
Toxic shock syndrome	• Nonpurulent conjunctivitis, pharyngitis, and erythroderma or a scarlatiniform rash that later desquamates are characteristic of toxic shock syndrome (TSS). Gastrointestinal complaints, hepatitis, severe muscle pain, elevated serum creatine kinase (CK), and renal abnormalities are more common in TSS than in meningococcal infections. Focal pyogenic infections may be observed in patients with TSS, particularly those caused by staphylococci.	• Blood cultures are positive in about half of patients with streptococcal TSS and 5% with staphylococcal TSS.
Enteroviral infection	• Rashes are most commonly erythematous and maculopapular but may be petechial. Stomatitis is characteristic of group A Coxsackie viruses.	• Cerebrospinal fluid (CSF) from patients with enteroviral meningoencephalitis typically reveals mild lymphocytic pleocytosis, mildly elevated or normal glucose, and normal protein concentration. Enterovirus may be isolated from blood, CSF, stool, throat, or urine. Enteroviral nucleic acid may be detected in CSF.
Epstein-Barr virus	• Cervical and generalized adenopathy, hepatosplenomegaly, and hepatitis are common. Severe illness is rare.	• Epstein-Barr virus (EBV) infection is diagnosed serologically. Nucleic acid detection may confirm EBV in immunocompromised patients.
Cytomegalovirus	• Beyond the neonatal period, a hemorrhagic rash is unusual. Pharyngitis, cervical adenopathy, and hepatosplenomegaly are common. Severe illness, with pneumonitis, chorioretinitis, enteritis, hepatitis, meningoencephalitis, coagulopathy, and pancytopenia, occurs in neonates and immunocompromised patients.	• Cytomegalovirus infection is confirmed by serology, viral culture, antigen detection, or nucleic acid amplification.

Condition	Differentiating signs / symptoms	Differentiating tests
Human parainfluenza virus	• Respiratory symptoms, including pharyngitis, rhinitis, and cough, are prominent. Rash is uncommon, but petechiae may be present.	• Parainfluenza virus infection may be confirmed by viral culture, antigen detection, or nucleic acid amplification.
Respiratory syncytial virus	• There may be an ongoing community outbreak. Respiratory symptoms, including pharyngitis, rhinitis, and cough, are prominent. Rash is uncommon, but petechiae may be present.	• Respiratory syncytial virus infection may be confirmed by viral culture, antigen detection, or nucleic acid amplification.
Influenza	• There may be an ongoing community outbreak. Respiratory symptoms, including pharyngitis, rhinitis, and cough, are prominent. Rash is uncommon, but petechiae may be present.	• Influenza may be confirmed by viral culture, antigen detection, or nucleic acid amplification.
Dengue/yellow fever	• There may be a history of travel to an endemic area 1 to 12 days prior to symptoms. Hepatitis and jaundice are more common in viral hemorrhagic fevers than in meningococcal infections.	• Serologic testing confirms a viral hemorrhagic fever.
Immune thrombocytopenia	• Typically, lack of fever or other signs of infection.	• Bone marrow aspirates or biopsy, assays for the presence of antiplatelet antibodies or evaluation for other autoimmune disorders, and tests for coagulation factor deficiencies confirm immune thrombocytopenia.
Henoch-Schonlein purpura	• Palpable purpuric rash, most commonly on the lower extremities; abdominal pain and vomiting; joint pain; and swelling and edema of the distal extremities, scalp, and scrotum.	• Hematuria and proteinuria are common. Skin biopsy demonstrates a leukocytoclastic vasculitis.
Thrombotic thrombocytopenic purpura	• Fever less common than in meningococcemia.	• Microangiopathic hemolytic anemia, thrombocytopenia (platelets <50 × 10^9/L), elevated serum LDH concentration, and hyperbilirubinemia are typical.

Condition	Differentiating signs / symptoms	Differentiating tests
Aplastic/myelodysplastic syndromes	• Fever less common than in meningococcemia but may be present in patients with secondary infections. Most patients have physical and laboratory findings suggestive of their primary disorder, such as bleeding from mucosal sites and pancytopenia.	• Typical laboratory findings of bone marrow failure syndromes include pancytopenia and evidence of abnormal hematopoiesis on bone marrow exam.
Bone marrow infiltration by malignancy	• Most patients have physical and laboratory findings suggestive of their primary disorder, such as weight loss, splenomegaly, adenopathy, and pancytopenia.	• Examination of peripheral blood and/or bone marrow confirms malignancy.

Criteria

Centers for Disease Control and Prevention case classification[1]

A case is confirmed by:

- Detection of *Neisseria meningitidis* -specific nucleic acid (using a validated polymerase chain reaction assay) in a specimen obtained from a normally sterile site (e.g., blood or cerebrospinal fluid); or
- Isolation of *N meningitidis* from a normally sterile site or from purpuric lesions.

Probable cases include those where *N meningitidis* antigen is detected by:

- Immunohistochemical staining on formalin-fixed tissue, or
- In cerebrospinal fluid by latex agglutination.

Suspected cases include those with:

- Clinical purpura fulminans in the absence of an *N meningitidis* positive blood culture, or
- Gram-negative diplococci, not yet identified, isolated from a normally sterile site.

Screening

Screening of carriers

Because nasopharyngeal carriage of pathogenic and nonpathogenic *Neisseria meningitidis* strains is very common and rarely progresses to invasive infection, routine screening for carriers is not recommended.

Recommendations

Key Recommendations

All patients with presumed meningococcal infections should be isolated in private rooms and droplet precautions taken. Antibiotics should be administered as soon as possible. This may reduce the yield of lumbar puncture, but administration of antibiotics should not be delayed if diagnostic evaluation cannot be completed promptly.

Meningococcal disease is a notifiable disease in many countries, including the US; cases should be reported immediately to local and national health departments.[19]

Because meningococcal infections are not readily differentiated from serious infections caused by other bacterial pathogens, empiric antibiotic therapy should include broad-spectrum agents that encompass coverage of *Streptococcus pneumoniae* and *Staphylococcus aureus* .[54] [64]

Empiric antibiotic therapy for suspected bacterial meningitis

Initial antibiotic choice is dependent on age:[54] [67] [68]

- Age ≤1 month and immunocompetent: ampicillin plus cefotaxime. If a cephalosporin cannot be administered (e.g., patients with an allergy), an alternative regimen is ampicillin (to cover *Listeria monocytogenes*) plus an aminoglycoside (e.g., gentamicin). Specialist advice should be sought for newborns.
- Age >1 month to <50 years and immunocompetent: ceftriaxone or cefotaxime plus vancomycin. If a cephalosporin cannot be administered (e.g., patients with an allergy), a carbapenem (e.g., meropenem) plus vancomycin can be considered.
- Age ≥50 years or immunocompromised (any age): ampicillin plus ceftriaxone or cefotaxime plus vancomycin. If ampicillin cannot be administered (e.g., patients with an allergy), trimethoprim/sulfamethoxazole could be considered as an alternative in place of ampicillin for some patient groups to ensure cover for *L monocytogenes* . Specialist advice should be sought for immunocompromised newborns.

Adjunctive corticosteroid therapy for suspected bacterial meningitis

- Some studies have shown that high-dose corticosteroids reduce the likelihood of neurologic sequelae, particularly in meningitis secondary to *Haemophilus influenzae* or *S pneumoniae* . However, the role of adjunctive corticosteroids in meningococcal meningitis remains controversial.[54] [69] [70]
- For suspected bacterial meningitis, most experts recommend that patients age >6 weeks receive dexamethasone for 2-4 days, with the first dose given prior to or concurrently with the first dose of antibiotics.[48] [68] [71] [72]
- Corticosteroids should be discontinued if the diagnosis of bacterial meningitis is disproved. Most experts advise discontinuing corticosteroids for bacterial meningitis if the causative organism is proven to be *Neisseria meningitidis* , although some advise that adjunctive treatment should continue irrespective of the pathogen.[54]

Empiric antibiotic therapy for suspected meningococcal bacteremia

The choice of empiric therapy for patients with suspected meningococcal bacteremia should be based on local susceptibility patterns, but in general includes:[73]

- Children age <1 month: cefotaxime or ceftriaxone or ceftazidime or an aminoglycoside (e.g., gentamicin) plus ampicillin. Acyclovir is indicated in infants with clinical features of herpes simplex virus (HSV) infection, including an ill appearance, mucocutaneous vesicles, seizures, or cerebrospinal fluid pleocytosis.
- Children age ≥1 month: ceftriaxone or cefotaxime or cefepime plus vancomycin.
- Adults: vancomycin plus ceftriaxone or cefotaxime or cefepime or imipenem/cilastatin or meropenem with or without an aminoglycoside (e.g., gentamicin).

The management of suspected bacteremia is becoming increasingly complex and should be based on local microbiology, risk factors (e.g., immunocompromised state, focal infection), and the severity of illness.

Supportive therapy

The major goal of supportive therapy is to restore and maintain normal respiratory, cardiac, and neurologic function.

Meningococcal infections may progress rapidly, and clinical deterioration may continue despite the prompt institution of antibiotic therapy. Initial assessment should follow the principles of pediatric and adult advanced life support, with evaluation of the patient's airway, breathing, and circulatory status, and the establishment of secure large-caliber intravenous catheters for administration of fluids.[51] [74]

Patients with shock (pulse rate may be persistently elevated, the skin mottled, the extremities cool due to increased systemic vascular resistance, the capillary refilling prolonged, and the urinary output decreased) or respiratory distress should receive appropriate respiratory support. This might include supplemental oxygen, or intubation and mechanical ventilation for those with severe respiratory distress, altered consciousness, or evidence of elevated intracranial pressure.[74] [75]

Adequate oxygenation, prevention of hypoglycemia and hyponatremia, anticonvulsant therapy if needed for early control of seizures, and measures to decrease intracranial pressure and to prevent fluctuating cerebral blood flow are important in managing patients with bacterial meningitis and meningococcal disease.[48] [74] [75] [76]

Vasopressors should be administered to patients with hypotension or poor perfusion who do not respond promptly to fluid resuscitation.[74] [75] If necessary, consult a specialist for guidance on suitable vasopressor/inotrope regimens.

Fluids should be administered cautiously in patients with evidence of elevated intracranial pressure, myocardial dysfunction, or acute respiratory distress syndrome (ARDS).

Treatment for confirmed meningitis

Once the diagnosis of a meningococcal infection is confirmed (generally within 12-48 hours of hospitalization), the patient's antibiotic therapy is changed, if necessary, to suitable definitive therapy. Most experts advise discontinuing corticosteroids for bacterial meningitis if the causative organism is

proven to be *N meningitidis* , although some advise that adjunctive treatment should be continued irrespective of the pathogen, to a total of 2-4 days' treatment.[54] [69]

The treatment for confirmed *N meningitidis* (duration of therapy 7 days) is based on susceptibilities:[54] [67] [77]

- Penicillin-susceptible (minimum inhibitory concentration [MIC] <0.1 micrograms/mL): ampicillin or penicillin-G. Alternatives include a third-generation cephalosporin (e.g., ceftriaxone).
- Penicillin-intermediate (MIC 0.1 to 1.0 micrograms/mL): ceftriaxone or cefotaxime. Alternatives include a fluoroquinolone or meropenem.

Systemic fluoroquinolone antibiotics may cause serious, disabling, and potentially long-lasting or irreversible adverse events. This includes, but is not limited to: tendinopathy/tendon rupture; peripheral neuropathy; arthropathy/arthralgia; aortic aneurysm and dissection; heart valve regurgitation; dysglycemia; and central nervous system effects including seizures, depression, psychosis, and suicidal thoughts and behavior.[78]

- Prescribing restrictions apply to the use of fluoroquinolones, and these restrictions may vary between countries. In general, fluoroquinolones should be restricted for use in serious, life-threatening bacterial infections only. Some regulatory agencies may also recommend that they must only be used in situations where other antibiotics that are commonly recommended for the infection are inappropriate (e.g., resistance, contraindications, treatment failure, unavailability).
- Consult your local guidelines and drug information source for more information on suitability, contraindications, and precautions.

The choice of agent is based on individual patient circumstances, antibiotic susceptibilities, and local availability.

Patients not treated with third-generation cephalosporins should receive therapy with rifampin, ceftriaxone, or ciprofloxacin to eradicate nasopharyngeal colonization prior to hospital discharge.

Resistant strains

In a national population-based survey of antimicrobial susceptibility of US meningococcal isolates, 25% of strains were penicillin or ampicillin intermediate.[79] Less than 1% were resistant to penicillin and ampicillin, ciprofloxacin, or levofloxacin, and all strains were susceptible to cefotaxime, ceftriaxone, meropenem, rifampin, minocycline, and azithromycin.[79] Chloramphenicol and fluoroquinolone resistance is increasingly reported in Africa and Asia, and among serotype Y strains in the US.[80]

Treatment of infections caused by these resistant strains should be based on results of antibiotic susceptibility testing. Resistant isolates reported to date have remained susceptible to cefotaxime and ceftriaxone.

Treatment for confirmed meningococcal bacteremia

Once the diagnosis of meningococcal bacteremia without meningitis is confirmed, the patient's antibiotic therapy should be changed to an intravenous third-generation cephalosporin or other definitive therapy. Treatment is usually for a duration of 5-7 days depending on the patient's age, severity of infection, and response to initial therapy.[50]

Most meningococcal isolates in the US are susceptible to penicillin, and this may be used for fully susceptible strains. Alternative agents include ampicillin, meropenem, or chloramphenicol. The choice of agent is based on individual patient circumstances, antibiotic susceptibilities, and local availability. If the patient is receiving dexamethasone for suspected meningitis, this should be discontinued.[47] [54]

Patients not treated with third-generation cephalosporins should receive therapy with rifampin, ceftriaxone, or ciprofloxacin to eradicate nasopharyngeal colonization prior to hospital discharge.

Treatment algorithm overview

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer

Initial (summary)			
suspected meningitis			
	immunocompetent: ≤1 month of age	**1st**	**empiric antibiotic therapy**
		plus	**supportive care**
	immunocompetent: >1 month to <50 years of age	**1st**	**empiric antibiotic therapy**
		plus	**supportive care**
		consider	**intravenous corticosteroid**
	≥50 years of age or immunocompromised	**1st**	**empiric antibiotic therapy**
		plus	**supportive care**
		consider	**intravenous corticosteroid**
suspected meningococcal bacteremia			
	<1 month of age	**1st**	**empiric antibiotic therapy**
		consider	**acyclovir**
		plus	**supportive care**
	children ≥1 month of age	**1st**	**empiric antibiotic therapy**
		plus	**supportive care**
	adults	**1st**	**empiric antibiotic therapy**
		plus	**supportive care**

Acute		(summary)
confirmed meningococcal meningitis: penicillin-susceptible		
	1st	targeted antibiotic therapy
	plus	supportive care
	plus	nasopharyngeal eradication predischarge
confirmed meningococcal meningitis: penicillin-intermediate sensitivity		
	1st	targeted antibiotic therapy
	plus	supportive care
	plus	nasopharyngeal eradication predischarge
confirmed meningococcal bacteremia		
	1st	targeted antibiotic therapy
	plus	supportive care
	2nd	alternative targeted antibiotic therapy
	plus	supportive care
	plus	nasopharyngeal eradication predischarge

Treatment algorithm

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer

Initial

suspected meningitis

immunocompetent: ≤1 month of age

1st **empiric antibiotic therapy**

Primary options

» ampicillin: consult specialist for guidance on neonatal dose
-and-
» cefotaxime: consult specialist for guidance on neonatal dose

Secondary options

» ampicillin: consult specialist for guidance on neonatal dose
-and-
» gentamicin: consult specialist for guidance on neonatal dose

» Presumptive treatment for neonatal meningitis should be active against meningococci and other causes of serious bacterial infection, including *Listeria* .[54] Antibiotic therapy should be administered as soon as feasible.

» The recommended regimen is ampicillin plus cefotaxime.[54] [67]

» If a cephalosporin cannot be administered (e.g., patients with an allergy), an alternative regimen is ampicillin plus an aminoglycoside (e.g., gentamicin).[54]

» Specialist advice should be sought for newborns.

plus **supportive care**

Treatment recommended for ALL patients in selected patient group

» Patients with meningococcal infections must be monitored closely for complications such as shock, elevated intracranial pressure, seizures, and coagulopathy.

» Severe meningococcal infections are frequently complicated by hypoglycemia or hyperglycemia, acidosis, and other biochemical abnormalities that require attention.

Initial

» Patients with symptoms of shock (pulse rate may be persistently elevated, the skin mottled, the extremities cool due to increased systemic vascular resistance, the capillary refilling prolonged, and the urinary output decreased) or respiratory distress should receive appropriate respiratory support. This might include supplemental oxygen, or intubation and mechanical ventilation for those with severe respiratory distress, altered consciousness, or evidence of elevated intracranial pressure.[75]

» Adequate oxygenation, prevention of hypoglycemia and hyponatremia, anticonvulsant therapy if needed for early control of seizures, and measures to decrease intracranial pressure and to prevent fluctuating cerebral blood flow are important in management.[75] [76]

» Vasopressors should be given to patients with hypotension or poor perfusion who do not respond promptly to fluid resuscitation.[75] Consult a specialist for guidance on suitable vasopressor/inotrope regimens.

» If the patient is hypovolemic or in shock, additional intravenous fluids must be given. Fluids should be given cautiously to patients with evidence of elevated intracranial pressure, myocardial dysfunction, or acute respiratory distress syndrome.

» Meningococcal disease is a notifiable disease in many countries, including the US; cases should be reported immediately to local and national health departments.[19]

immunocompetent: >1 month to <50 years of age

1st

empiric antibiotic therapy

Primary options

» vancomycin: children: 15 mg/kg intravenously every 6 hours; adults: 15-20 mg/kg intravenously every 8-12 hours
Adjust dose according to serum vancomycin level.

--AND--

» ceftriaxone: children: 80-100 mg/kg/day intravenously given in divided doses every 12-24 hours, maximum 4 g/day; adults: 2 g intravenously every 12 hours
-or-
» cefotaxime: children: 200-300 mg/kg/day intravenously given in divided doses every 6-8 hours, maximum 12 g/day; adults: 2 g intravenously every 4-6 hours

MANAGEMENT

Initial

Secondary options

» vancomycin: children: 15 mg/kg intravenously every 6 hours; adults: 15-20 mg/kg intravenously every 8-12 hours
Adjust dose according to serum vancomycin level.

-and-
» meropenem: children ≥3 months of age and <50 kg body weight: 40 mg/kg intravenously every 8 hours, maximum 6 g/day; children ≥3 months of age and ≥50 kg body weight and adults: 2 g intravenously every 8 hours

» Presumptive treatment for bacterial meningitis should be active against meningococci and other causes of serious bacterial infection, including *Streptococcus pneumoniae* .[54] Antibiotic therapy should be administered as soon as feasible.

» The recommended regimen is ceftriaxone or cefotaxime plus vancomycin.[54] [67]

» If a cephalosporin cannot be administered (e.g., patients with an allergy), a carbapenem (e.g., meropenem) plus vancomycin can be considered.[81]

plus **supportive care**

Treatment recommended for ALL patients in selected patient group

» Patients with meningococcal infections must be monitored closely for complications such as shock, elevated intracranial pressure, seizures, and coagulopathy.

» Severe meningococcal infections are frequently complicated by hypoglycemia or hyperglycemia, acidosis, and other biochemical abnormalities that require attention.

» Patients with shock (pulse rate may be persistently elevated, the skin mottled, the extremities cool due to increased systemic vascular resistance, the capillary refilling prolonged, and the urinary output decreased) or respiratory distress should receive appropriate respiratory support. This might include supplemental oxygen, or intubation and mechanical ventilation for those with severe respiratory distress, altered consciousness, or evidence of elevated intracranial pressure.[74] [75]

Initial

» Adequate oxygenation, prevention of hypoglycemia and hyponatremia, anticonvulsant therapy if needed for early control of seizures, and measures to decrease intracranial pressure and to prevent fluctuating cerebral blood flow are important in management.[48] [74] [75] [76]

» Vasopressors should be given to patients with hypotension or poor perfusion who do not respond promptly to fluid resuscitation.[74] [75] Consult a specialist for guidance on suitable vasopressor/inotrope regimens.

» If the patient is hypovolemic or in shock, additional intravenous fluids must be given. Fluids should be given cautiously to patients with evidence of elevated intracranial pressure, myocardial dysfunction, or acute respiratory distress syndrome.

» Meningococcal disease is a notifiable disease in many countries, including the US; cases should be reported immediately to local and national health departments.[19]

consider **intravenous corticosteroid**

Treatment recommended for SOME patients in selected patient group

Primary options

» dexamethasone sodium phosphate: children >6 weeks of age: 0.15 mg/kg intravenously every 6 hours; adults: 10 mg intravenously every 6 hours

» For suspected bacterial meningitis, most experts recommend that patients older than age 6 weeks receive dexamethasone, with the first dose given prior to or concurrently with the first dose of antibiotics.[48] [68] [71] [72]

» Some studies have shown that high-dose corticosteroids reduce the likelihood of neurologic sequelae, particularly in meningitis secondary to *Haemophilus influenzae* or *Streptococcus pneumoniae* . The effectiveness of adjuvant corticosteroids in meningitis caused by *Neisseria meningitidis* is not yet clear.[54] [69] [70]

» Most experts advise discontinuing corticosteroids for bacterial meningitis if the causative organism is proven to be *N meningitidis* , although some advise that adjunctive treatment should be continued irrespective of the pathogen.[54] Corticosteroids

Initial

should be discontinued if the diagnosis of bacterial meningitis is disproved.

≥50 years of age or immunocompromised — **1st** — **empiric antibiotic therapy**

Primary options

» ampicillin: neonates: consult specialist for guidance on dose; children: 200-400 mg/kg/day intravenously given in divided doses every 4-6 hours, maximum 12 g/day; adults: 1-2 g intravenously every 3-4 hours

--AND--

» vancomycin: neonates: consult specialist for guidance on dose; children: 15 mg/kg intravenously every 6 hours; adults: 15-20 mg/kg intravenously every 8-12 hours

Adjust dose according to serum vancomycin level.

--AND--

» ceftriaxone: neonates: consult specialist for guidance on dose; children: 80-100 mg/kg/day intravenously given in divided doses every 12-24 hours, maximum 4 g/day; adults: 2 g intravenously every 12 hours

-or-

» cefotaxime: neonates: consult specialist for guidance on dose; children: 200-300 mg/kg/day intravenously given in divided doses every 6-8 hours, maximum 12 g/day; adults: 2 g intravenously every 4-6 hours

Secondary options

» sulfamethoxazole/trimethoprim: children ≥2 months of age and adults: 10-20 mg/kg/day intravenously given in divided doses every 6-12 hours

Dose refers to trimethoprim component.

--AND--

» vancomycin: neonates: consult specialist for guidance on dose; children: 15 mg/kg intravenously every 6 hours; adults:15-20 mg/kg intravenously every 8-12 hours

Adjust dose according to serum vancomycin level.

--AND--

» ceftriaxone: neonates: consult specialist for guidance on dose; children: 80-100 mg/kg/day intravenously given in divided doses every 12-24 hours, maximum 4 g/day; adults: 2 g intravenously every 12 hours

-or-

» cefotaxime: neonates: consult specialist for guidance on dose; children: 200-300 mg/

Initial

kg/day intravenously given in divided doses every 6-8 hours, maximum 12 g/day; adults: 2 g intravenously every 4-6 hours

» Presumptive treatment for bacterial meningitis should be active against meningococci and other causes of serious bacterial infection, including *Listeria* .[54] Antibiotic therapy should be administered as soon as feasible.

» The recommended regimen is ampicillin plus ceftriaxone or cefotaxime plus vancomycin.[54] [67] If ampicillin cannot be administered (e.g., patients with an allergy), trimethoprim/sulfamethoxazole could be considered as an alternative for some patient groups to ensure cover for *Listeria monocytogenes* in place of ampicillin.[67] [81] Specialist advice should be sought for immunocompromised newborns.

plus **supportive care**

Treatment recommended for ALL patients in selected patient group

» Patients with meningococcal infections must be monitored closely for complications such as shock, elevated intracranial pressure, seizures, and coagulopathy.

» Severe meningococcal infections are frequently complicated by hypoglycemia or hyperglycemia, acidosis, and other biochemical abnormalities that require attention.

» Patients with shock (pulse rate may be persistently elevated, the skin mottled, the extremities cool due to increased systemic vascular resistance, the capillary refilling prolonged, and the urinary output decreased) or respiratory distress should receive appropriate respiratory support. This might include supplemental oxygen, or intubation and mechanical ventilation for those with severe respiratory distress, altered consciousness, or evidence of elevated intracranial pressure.[74] [75]

» Adequate oxygenation, prevention of hypoglycemia and hyponatremia, anticonvulsant therapy if needed for early control of seizures, and measures to decrease intracranial pressure and to prevent fluctuating cerebral blood flow are important in management.[48] [74] [75][76]

» Vasopressors should be given to patients with hypotension or poor perfusion who do not respond promptly to fluid resuscitation.[74] [75]

Initial

Consult a specialist for guidance on suitable vasopressor/inotrope regimens.

» If the patient is hypovolemic or in shock, additional intravenous fluids must be given. Fluids should be given cautiously to patients with evidence of elevated intracranial pressure, myocardial dysfunction, or acute respiratory distress syndrome.

» Meningococcal disease is a notifiable disease in many countries, including the US; cases should be reported immediately to local and national health departments.[19]

consider **intravenous corticosteroid**

Treatment recommended for SOME patients in selected patient group

Primary options

» dexamethasone sodium phosphate: children >6 weeks of age: 0.15 mg/kg intravenously every 6 hours; adults: 10 mg intravenously every 6 hours

» For suspected bacterial meningitis, most experts recommend that patients older than age 6 weeks receive dexamethasone, with the first dose given prior to or concurrently with the first dose of antibiotics.[48] [68] [71] [72]

» Some studies have shown that high-dose corticosteroids reduce the likelihood of neurologic sequelae, particularly in meningitis secondary to *Haemophilus influenzae* or *Streptococcus pneumoniae* . The effectiveness of adjuvant corticosteroids in meningitis caused by *Neisseria meningitidis* is not yet clear.[54] [69] [70]

» Most experts advise discontinuing corticosteroids for bacterial meningitis if the causative organism is proven to be *N meningitidis* , although some advise that adjunctive treatment should be continued irrespective of the pathogen.[54] Corticosteroids should be discontinued if the diagnosis of bacterial meningitis is disproved.

suspected meningococcal bacteremia

<1 month of age **1st** **empiric antibiotic therapy**

Primary options

Initial

» ampicillin: consult specialist for guidance on neonatal dose

--AND--

» ceftriaxone: consult specialist for guidance on neonatal dose
-or-
» cefotaxime: consult specialist for guidance on neonatal dose
-or-
» ceftazidime sodium: consult specialist for guidance on neonatal dose
-or-
» gentamicin: consult specialist for guidance on neonatal dose

» Presumptive treatment for suspected bacteremia should be active against meningococci and other causes of serious bacterial infection, including group B *Streptococcus agalactiae* , *Escherichia coli* , and *Listeria* .[54] Antibiotic therapy should be administered as soon as feasible.

» Cefotaxime or ceftriaxone or ceftazidime or an aminoglycoside (e.g., gentamicin) plus ampicillin is a suitable option in these patients.[73]

» The management of suspected bacteremia is becoming increasingly complex and should be based on local susceptibility rates, risk factors (e.g., immunocompromised state, focal infection), and the severity of illness.

consider **acyclovir**

Treatment recommended for SOME patients in selected patient group

Primary options

» acyclovir: consult specialist for guidance on neonatal dose

» Acyclovir is indicated in infants with clinical features of herpes simplex virus (HSV) infection, including an ill appearance, mucocutaneous vesicles, seizures, or cerebrospinal fluid pleocytosis.

plus **supportive care**

Treatment recommended for ALL patients in selected patient group

» Patients with meningococcal infections must be monitored closely for complications such as shock, elevated intracranial pressure, seizures, and coagulopathy.

MANAGEMENT

Initial

» Severe meningococcal infections are frequently complicated by hypoglycemia or hyperglycemia, acidosis, and other biochemical abnormalities that require attention.

» Patients with symptoms of shock (pulse rate may be persistently elevated, the skin mottled, the extremities cool due to increased systemic vascular resistance, the capillary refilling prolonged, and the urinary output decreased) or respiratory distress should receive appropriate respiratory support. This might include supplemental oxygen, or intubation and mechanical ventilation for those with severe respiratory distress, altered consciousness, or evidence of elevated intracranial pressure.[75]

» Adequate oxygenation, prevention of hypoglycemia and hyponatremia, anticonvulsant therapy if needed for early control of seizures, and measures to decrease intracranial pressure and to prevent fluctuating cerebral blood flow are important in management.[75] [76]

» Vasopressors should be given to patients with hypotension or poor perfusion who do not respond promptly to fluid resuscitation.[75] Consult a specialist for guidance on suitable vasopressor/inotrope regimens.

» If the patient is hypovolemic or in shock, additional intravenous fluids must be given. Fluids should be given cautiously to patients with evidence of elevated intracranial pressure, myocardial dysfunction, or acute respiratory distress syndrome.

» Meningococcal disease is a notifiable disease in many countries, including the US; cases should be reported immediately to local and national health departments.[19]

children ≥1 month of age **1st** **empiric antibiotic therapy**

Primary options

» vancomycin: 15 mg/kg intravenously every 6 hours
Adjust dose according to serum vancomycin level.

--AND--

» ceftriaxone: 80-100 mg/kg/day intravenously given in divided doses every 12-24 hours, maximum 4 g/day
-or-
» cefotaxime: 200-300 mg/kg/day intravenously given in divided doses every 6-8 hours, maximum 12 g/day

Initial

-or-

» cefepime: children ≥2 months of age: 150 mg/kg/day intravenously given in divided doses every 8 hours

» Presumptive treatment for suspected bacteremia should be active against meningococci and other causes of serious bacterial infection, including *Streptococcus pneumoniae* and *Staphylococcus aureus* .[54] Antibiotic therapy should be administered as soon as feasible.

» Ceftriaxone or cefotaxime or ceftriaxone or cefepime plus vancomycin is a suitable option in these patients.[73]

» The management of suspected bacteremia is becoming increasingly complex and should be based on local susceptibility rates, risk factors (e.g., immunocompromised state, focal infection), and the severity of illness. Additional antibiotic cover may be required for those with relevant risk factors (e.g., for *Listeria*).

plus **supportive care**

Treatment recommended for ALL patients in selected patient group

» Patients with meningococcal infections must be monitored closely for complications such as shock, elevated intracranial pressure, seizures, and coagulopathy.

» Severe meningococcal infections are frequently complicated by hypoglycemia or hyperglycemia, acidosis, and other biochemical abnormalities that require attention.

» Patients with symptoms of shock (pulse rate may be persistently elevated, the skin mottled, the extremities cool due to increased systemic vascular resistance, the capillary refilling prolonged, and the urinary output decreased) or respiratory distress should receive appropriate respiratory support. This might include supplemental oxygen, or intubation and mechanical ventilation for those with severe respiratory distress, altered consciousness, or evidence of elevated intracranial pressure.[75]

» Adequate oxygenation, prevention of hypoglycemia and hyponatremia, anticonvulsant therapy if needed for early control of seizures, and measures to decrease intracranial pressure and to prevent fluctuating cerebral blood flow are important in management.[75] [76]

Initial

» Vasopressors should be given to patients with hypotension or poor perfusion who do not respond promptly to fluid resuscitation.[75] Consult a specialist for guidance on suitable vasopressor/inotrope regimens.

» If the patient is hypovolemic or in shock, additional intravenous fluids must be given. Fluids should be given cautiously to patients with evidence of elevated intracranial pressure, myocardial dysfunction, or acute respiratory distress syndrome.

» Meningococcal disease is a notifiable disease in many countries, including the US; cases should be reported immediately to local and national health departments.[19]

adults — **1st** — **empiric antibiotic therapy**

Primary options

» vancomycin: 15-20 mg/kg intravenously every 8-12 hours
Adjust dose according to serum vancomycin level.

--AND--

» ceftriaxone: 2 g intravenously every 12 hours
-or-
» cefotaxime: 2 g intravenously every 4-6 hours
-or-
» cefepime: 2 g intravenously every 8 hours
-or-
» imipenem/cilastatin: 500 mg intravenously every 6 hours
Dose refers to imipenem component.

-or-
» meropenem: 2 g intravenously every 8 hours

OR

» vancomycin: 15-20 mg/kg intravenously every 8-12 hours
Adjust dose according to serum vancomycin level.

--AND--

» ceftriaxone: 2 g intravenously every 12 hours
-or-
» cefotaxime: 2 g intravenously every 4 hours
-or-
» cefepime: 2 g intravenously every 8 hours

Initial

-or-

» imipenem/cilastatin: 500 mg intravenously every 6 hours

Dose refers to imipenem component.

-or-

» meropenem: 2 g intravenously every 8 hours

--AND--

» gentamicin: 5 mg/kg/day intravenously given in 1-3 divided doses

Adjust dose according to serum gentamicin level.

» Presumptive treatment for suspected bacteremia should be active against meningococci and other causes of serious bacterial infection, including *Streptococcus pneumoniae* and *Staphylococcus aureus* .[54] Antibiotic therapy should be administered as soon as feasible. Therapy should include one or more broad-spectrum antibiotics active against gram-positive and gram-negative organisms.

» Vancomycin plus ceftriaxone or cefotaxime or cefepime or imipenem/cilastatin or meropenem with or without an aminoglycoside (e.g., gentamicin) is a suitable option in these patients.[73]

» The management of suspected bacteremia is becoming increasingly complex and should be based on local microbiology, personal risk factors (e.g., immunocompromised state, focal infection), age, and the severity of illness. Additional antibiotic cover may be required for those with relevant risk factors (e.g., for *Listeria*).

plus **supportive care**

Treatment recommended for ALL patients in selected patient group

» Patients with meningococcal infections must be monitored closely for complications such as shock, elevated intracranial pressure, seizures, and coagulopathy.

» Severe meningococcal infections are frequently complicated by hypoglycemia or hyperglycemia, acidosis, and other biochemical abnormalities that require attention.

» Patients with shock (pulse rate may be persistently elevated, the skin mottled, the extremities cool due to increased systemic vascular resistance, the capillary refilling

Initial

prolonged, and the urinary output decreased) or respiratory distress should receive appropriate respiratory support. This might include supplemental oxygen, or intubation and mechanical ventilation for those with severe respiratory distress, altered consciousness, or evidence of elevated intracranial pressure.[74]

» Adequate oxygenation, prevention of hypoglycemia and hyponatremia, anticonvulsant therapy if needed for early control of seizures, and measures to decrease intracranial pressure and to prevent fluctuating cerebral blood flow are important in management.[48] [74]

» Vasopressors should be given to patients with hypotension or poor perfusion who do not respond promptly to fluid resuscitation.[74] Consult a specialist for guidance on suitable vasopressor/inotrope regimens.

» If the patient is hypovolemic or in shock, additional intravenous fluids must be given. Fluids should be given cautiously to patients with evidence of elevated intracranial pressure, myocardial dysfunction, or acute respiratory distress syndrome.

» Meningococcal disease is a notifiable disease in many countries, including the US; cases should be reported immediately to local and national health departments.[19]

Acute

confirmed meningococcal meningitis: penicillin-susceptible

1st **targeted antibiotic therapy**

Primary options

» penicillin G potassium: neonates: consult specialist for guidance on dose; children: 250,000 to 400,000 units/kg/day intravenously given in divided doses every 4-6 hours, maximum 24 million units/day; adults: 2 million units intravenously every 2 hours

OR

» ampicillin: neonates: consult specialist for guidance on dose; children: 200-400 mg/kg/day intravenously given in divided doses every 4-6 hours, maximum 12 g/day; adults: 1-2 g intravenously every 3-4 hours

Secondary options

» ceftriaxone: neonates: consult specialist for guidance on dose; children: 80-100 mg/kg/day intravenously given in divided doses every 12-24 hours, maximum 4 g/day; adults: 2 g intravenously every 12 hours

» After diagnosis is confirmed (generally within 12-48 hours of admission to the hospital), antimicrobial therapy can be modified according to the causative organism and antibiotic susceptibilities.[67] [76] Typically, the duration of antibacterial treatment depends on the clinical response and the cerebrospinal fluid microbiologic response after treatment has started. Most experts advise discontinuing corticosteroids for bacterial meningitis if the causative organism is proven to be *Neisseria meningitidis* , although some advise that adjunctive treatment should be continued irrespective of the pathogen.[54]

» *Neisseria meningitidis* that is penicillin-susceptible (minimum inhibitory concentration [MIC] <0.1 micrograms/mL) should be treated with ampicillin or penicillin-G. Alternatives include a third-generation cephalosporin (e.g., ceftriaxone).[67] [77]

» Treatment course: 7 days.

plus **supportive care**

Acute

Treatment recommended for ALL patients in selected patient group

» Patients with meningococcal infections must be monitored closely for complications such as shock, elevated intracranial pressure, seizures, and coagulopathy.

» Severe meningococcal infections are frequently complicated by hypoglycemia or hyperglycemia, acidosis, and other biochemical abnormalities that require attention.

» Patients with shock (pulse rate may be persistently elevated, the skin mottled, the extremities cool due to increased systemic vascular resistance, the capillary refilling prolonged, and the urinary output decreased) or respiratory distress should receive appropriate respiratory support. This might include supplemental oxygen, or intubation and mechanical ventilation for those with severe respiratory distress, altered consciousness, or evidence of elevated intracranial pressure.[74] [75]

» Adequate oxygenation, prevention of hypoglycemia and hyponatremia, anticonvulsant therapy if needed for early control of seizures, and measures to decrease intracranial pressure and to prevent fluctuating cerebral blood flow are important in management.[48] [74] [75] [76]

» Vasopressors should be given to patients with hypotension or poor perfusion who do not respond promptly to fluid resuscitation.[74] [75] Consult a specialist for guidance on suitable vasopressor/inotrope regimens.

» If the patient is hypovolemic or in shock, additional intravenous fluids must be given. Fluids should be given cautiously to patients with evidence of elevated intracranial pressure, myocardial dysfunction, or acute respiratory distress syndrome.

plus **nasopharyngeal eradication predischarge**

Treatment recommended for ALL patients in selected patient group

Primary options

» rifampin: children: 20 mg/kg/day orally given in 2 divided doses for 2 days, maximum 600 mg/dose; adults: 600 mg orally twice daily for 2 days

OR

Acute

» ceftriaxone: children: 125 mg intramuscularly as a single dose; adults: 250 mg intramuscularly as a single dose

OR

» ciprofloxacin: adults: 500 mg orally as a single dose

» Patients with invasive meningococcal infections who are not treated with ceftriaxone or cefotaxime should also receive rifampin, ciprofloxacin, or ceftriaxone before discharge from the hospital, to eradicate nasopharyngeal colonization.[4] [50]

» These treatments are equally effective. Choice is based on the age of the patient and the presence of any contraindications to an individual drug. Specialist advice should be sought for newborns.

confirmed meningococcal meningitis: penicillin-intermediate sensitivity

1st **targeted antibiotic therapy**

Primary options

» ceftriaxone: neonates: consult specialist for guidance on dose; children: 80-100 mg/kg/day intravenously given in divided doses every 12-24 hours, maximum 4 g/day; adults: 2 g intravenously every 12 hours

OR

» cefotaxime: neonates: consult specialist for guidance on dose; children: 200-300 mg/kg/day intravenously given in divided doses every 6-8 hours, maximum 12 g/day; adults: 2 g intravenously every 4-6 hours

Secondary options

» meropenem: children ≥3 months of age and <50 kg body weight: 40 mg/kg intravenously every 8 hours, maximum 6 g/day; children ≥3 months of age and ≥50 kg body weight and adults: 2 g intravenously every 8 hours

OR

» levofloxacin: children: consult specialist for guidance on dose; adults: 750 mg intravenously every 24 hours

MANAGEMENT

Acute

OR

» ciprofloxacin: children: consult specialist for guidance on dose; adults: 400 mg intravenously every 8-12 hours

» After diagnosis is confirmed (generally within 12-48 hours of admission to the hospital), antimicrobial therapy can be modified according to the causative organism and antibiotic susceptibilities.[67] [76] Typically, the duration of antibacterial treatment depends on the clinical response and the cerebrospinal fluid microbiologic response after treatment has started. Most experts advise discontinuing corticosteroids for bacterial meningitis if the causative organism is proven to be *Neisseria meningitidis* , although some advise that adjunctive treatment should be continued irrespective of the pathogen.[54]

» *Neisseria meningitidis* that is penicillin-intermediate susceptible (minimum inhibitory concentration [MIC] 0.1 to 1.0 micrograms/mL) should be treated with ceftriaxone or cefotaxime. Alternatives include a fluoroquinolone (e.g., levofloxacin, ciprofloxacin) or meropenem.[67] [77]

» Systemic fluoroquinolone antibiotics may cause serious, disabling, and potentially long-lasting or irreversible adverse events. This includes, but is not limited to: tendinopathy/tendon rupture; peripheral neuropathy; arthropathy/arthralgia; aortic aneurysm and dissection; heart valve regurgitation; dysglycemia; and central nervous system effects including seizures, depression, psychosis, and suicidal thoughts and behavior.[78] Prescribing restrictions apply to the use of fluoroquinolones, and these restrictions may vary between countries. In general, fluoroquinolones should be restricted for use in serious, life-threatening bacterial infections only. Some regulatory agencies may also recommend that they must only be used in situations where other antibiotics that are commonly recommended for the infection are inappropriate (e.g., resistance, contraindications, treatment failure, unavailability). Consult your local guidelines and drug information source for more information on suitability, contraindications, and precautions.

» Treatment course: 7 days.

plus **supportive care**

Acute

Treatment recommended for ALL patients in selected patient group

» Patients with meningococcal infections must be monitored closely for complications such as shock, elevated intracranial pressure, seizures, and coagulopathy.

» Severe meningococcal infections are frequently complicated by hypoglycemia or hyperglycemia, acidosis, and other biochemical abnormalities that require attention.

» Patients with shock (pulse rate may be persistently elevated, the skin mottled, the extremities cool due to increased systemic vascular resistance, the capillary refilling prolonged, and the urinary output decreased) or respiratory distress should receive appropriate respiratory support. This might include supplemental oxygen, or intubation and mechanical ventilation for those with severe respiratory distress, altered consciousness, or evidence of elevated intracranial pressure.[74] [75]

» Adequate oxygenation, prevention of hypoglycemia and hyponatremia, anticonvulsant therapy if needed for early control of seizures, and measures to decrease intracranial pressure and to prevent fluctuating cerebral blood flow are important in management.[48] [74] [75] [76]

» Vasopressors should be given to patients with hypotension or poor perfusion who do not respond promptly to fluid resuscitation.[74] [75] Consult a specialist for guidance on suitable vasopressor/inotrope regimens.

» If the patient is hypovolemic or in shock, additional intravenous fluids must be given. Fluids should be given cautiously to patients with evidence of elevated intracranial pressure, myocardial dysfunction, or acute respiratory distress syndrome.

plus **nasopharyngeal eradication predischarge**

Treatment recommended for ALL patients in selected patient group

Primary options

» rifampin: children: 20 mg/kg/day orally given in 2 divided doses for 2 days, maximum 600 mg/dose; adults: 600 mg orally twice daily for 2 days

OR

MANAGEMENT

Acute

» ceftriaxone: children: 125 mg intramuscularly as a single dose; adults: 250 mg intramuscularly as a single dose

OR

» ciprofloxacin: adults: 500 mg orally as a single dose

» Patients with invasive meningococcal infections who are not treated with ceftriaxone or cefotaxime should also receive rifampin, ciprofloxacin, or ceftriaxone before discharge from the hospital, to eradicate nasopharyngeal colonization.[4] [50]

» These treatments are equally effective. Choice is based on the age of the patient and the presence of any contraindications to an individual drug. Specialist advice should be sought for newborns.

confirmed meningococcal bacteremia

1st **targeted antibiotic therapy**

Primary options

» ceftriaxone: neonates: consult specialist for guidance on dose; children: 80-100 mg/kg/day intravenously given in divided doses every 12-24 hours, maximum 4 g/day; adults: 2 g intravenously every 12 hours

OR

» cefotaxime: neonates: consult specialist for guidance on dose; children: 200-300 mg/kg/day intravenously given in divided doses every 6-8 hours, maximum 12 g/day; adults: 2 g intravenously every 4-6 hours

» Once the diagnosis of a meningococcal infection is confirmed, the patient should be treated with an intravenous cephalosporin (e.g., ceftriaxone or cefotaxime). Treatment is usually for a duration of 5-7 days depending on the patient's age, severity of infection, and response to initial therapy.[50]

» If the patient is receiving dexamethasone for suspected meningitis, this should be discontinued.[47] [54]

plus **supportive care**

Treatment recommended for ALL patients in selected patient group

Acute

» Patients with meningococcal infections must be monitored closely for complications such as shock, elevated intracranial pressure, seizures, and coagulopathy.

» Severe meningococcal infections are frequently complicated by hypoglycemia or hyperglycemia, acidosis, and other biochemical abnormalities that require attention.

» Patients with shock (pulse rate may be persistently elevated, the skin mottled, the extremities cool due to increased systemic vascular resistance, the capillary refilling prolonged, and the urinary output decreased) or respiratory distress should receive appropriate respiratory support. This might include supplemental oxygen, or intubation and mechanical ventilation for those with severe respiratory distress, altered consciousness, or evidence of elevated intracranial pressure.[74] [75]

» Adequate oxygenation, prevention of hypoglycemia and hyponatremia, anticonvulsant therapy if needed for early control of seizures, and measures to decrease intracranial pressure and to prevent fluctuating cerebral blood flow are important in management.[48] [74] [75] [76]

» Vasopressors should be given to patients with hypotension or poor perfusion who do not respond promptly to fluid resuscitation.[74] [75] Consult a specialist for guidance on suitable vasopressor/inotrope regimens.

» If the patient is hypovolemic or in shock, additional intravenous fluids must be given. Fluids should be given cautiously to patients with evidence of elevated intracranial pressure, myocardial dysfunction, or acute respiratory distress syndrome.

2nd **alternative targeted antibiotic therapy**

Primary options

» penicillin G potassium: neonates: consult specialist for guidance on dose; children: 250,000 to 400,000 units/kg/day intravenously given in divided doses every 4-6 hours, maximum 24 million units/day; adults: 2 million units intravenously every 2 hours

OR

MANAGEMENT

Acute

» ampicillin: neonates: consult specialist for guidance on dose; children: 200-400 mg/kg/day intravenously given in divided doses every 4-6 hours, maximum 12 g/day; adults: 1-2 g intravenously every 3-4 hours

OR

» meropenem: children ≥3 months of age and <50 kg body weight: 40 mg/kg intravenously every 8 hours, maximum 6 g/day; children ≥3 months of age and ≥50 kg body weight and adults: 2 g intravenously every 8 hours

OR

» chloramphenicol: children ≥1 month of age and adults: 75-100 mg/kg/day intravenously given in divided doses every 6 hours, maximum 4 g/day

» Where a cephalosporin is not appropriate, alternative agents include penicillin-G, ampicillin, meropenem, or chloramphenicol. Choice of agent is based on individual patient age, circumstances, and antibiotic susceptibilities, and on local availability.

» Treatment of infections caused by resistant strains should be based on results of antibiotic susceptibility testing.

» If the patient is receiving dexamethasone for suspected meningitis, this should be discontinued.[47] [54]

» Specialist advice should be sought for newborns.

plus **supportive care**

Treatment recommended for ALL patients in selected patient group

» Patients with meningococcal infections must be monitored closely for complications such as shock, elevated intracranial pressure, seizures, and coagulopathy.

» Severe meningococcal infections are frequently complicated by hypoglycemia or hyperglycemia, acidosis, and other biochemical abnormalities that require attention.

» Patients with shock (pulse rate may be persistently elevated, the skin mottled, the extremities cool due to increased systemic vascular resistance, the capillary refilling prolonged, and the urinary output decreased) or

Acute

respiratory distress should receive appropriate respiratory support. This might include supplemental oxygen, or intubation and mechanical ventilation for those with severe respiratory distress, altered consciousness, or evidence of elevated intracranial pressure.[74] [75]

» Adequate oxygenation, prevention of hypoglycemia and hyponatremia, anticonvulsant therapy if needed for early control of seizures, and measures to decrease intracranial pressure and to prevent fluctuating cerebral blood flow are important in management.[48] [74] [75] [76]

» Vasopressors should be given to patients with hypotension or poor perfusion who do not respond promptly to fluid resuscitation.[74] [75] Consult a specialist for guidance on suitable vasopressor/inotrope regimens.

» If the patient is hypovolemic or in shock, additional intravenous fluids must be given. Fluids should be given cautiously to patients with evidence of elevated intracranial pressure, myocardial dysfunction, or acute respiratory distress syndrome.

plus **nasopharyngeal eradication predischarge**

Treatment recommended for ALL patients in selected patient group

Primary options

» rifampin: children: 20 mg/kg/day orally given in 2 divided for 2 days, maximum 600 mg/dose; adults: 600 mg orally twice daily for 2 days

OR

» ceftriaxone: children: 125 mg intramuscularly as a single dose; adults: 250 mg intramuscularly as a single dose

OR

» ciprofloxacin: adults: 500 mg orally as a single dose

» Patients with invasive meningococcal infections who are not treated with ceftriaxone or cefotaxime should also receive rifampin, ciprofloxacin, or ceftriaxone before discharge from the hospital, to eradicate nasopharyngeal colonization.[4] [50]

Acute

» These treatments are equally effective. Choice is based on the age of the patient and the presence of any contraindications to an individual drug. Specialist advice should be sought for newborns.

Emerging

Conjugate vaccines targeting Neisseria meningitidis serogroup X

N meningitidis is a leading cause of bacterial meningitis globally, with six serogroups (A, B, C, W, X, and Y) responsible for over 95% of invasive meningococcal disease cases worldwide.[65] In the African meningitis belt, following the success of vaccination programs with serogroup A meningococcal conjugate vaccine, *N meningitidis* serogroups C, W, and X, and *Streptococcus pneumoniae* have been identified as the main causative agents of more recent epidemics.[82] The Men5CV (NmCV-5) vaccine is a pentavalent vaccine targeted at meningococcal serotypes A, C, W, Y, and X. This vaccine is directed toward global control of meningococcal disease in the African meningitis belt and beyond, with demonstrated safety and effectiveness against a wider variety of serogroups including serogroup X.[83] The vaccine has demonstrated comparable immune response in phase 1 and 2 trials.[83] [84] It has been recommended by the World Health Organization (WHO) for incorporation into routine immunization programs in countries in the African meningitis belt, and Nigeria was the first country in the world to introduce the vaccine.[65] [85] [WHO: defeating meningitis by 2030 - a global road map] While the vaccine is available in Africa, it is not currently available in other countries as yet.

Invasive intracranial pressure (ICP) monitoring

One systematic review looked at the role of invasive ICP monitoring and ICP-based management in the treatment of acute bacterial meningitis. Overall, the studies demonstrated enhanced patient outcomes in acute bacterial meningitis with the use of treatment strategies aiming to normalize ICP using continuous invasive monitoring and cerebrospinal fluid diversion techniques.[86]

Primary prevention

Many developed countries offer routine childhood vaccination for prevention of meningococcal disease. Since the introduction of routine quadrivalent meningococcal conjugate vaccination in the US, the national incidence of meningococcal infection in adolescents and young adults has fallen 2- to 3-fold.[43] The World Health Organization (WHO) and the Advisory Committee on Immunization Practices (ACIP) also recommend meningococcal vaccination for selected high-risk populations.[20] [44]

For full details of US immunization schedules, including indications for booster doses, the ACIP guidelines should be consulted.[20] [CDC: adult immunization schedule by age - recommendations for ages 19 years or older] [CDC: child and adolescent immunization schedule by age - recommendations for ages 18 years or younger]

Children

Tetravalent meningococcal conjugate vaccine (MenACWY) is recommended by the ACIP for routine vaccination of all children, preferably at age 11 or 12 years, with a booster dose at age 16 years.[20] [45] MenACWY vaccination may also be recommended for younger children (from age 2 months) with high-risk conditions, or at increased risk of disease (anatomic or functional asplenia, HIV infection, receiving eculizumab or ravulizumab, or with persistent complement component deficiency), and for children traveling to countries with hyperendemic or epidemic meningococcal disease.[20] [23] Serogroup B vaccination is recommended by the ACIP for children and adolescents ages over 10 years with high-risk conditions or at increased risk of disease (anatomic or functional asplenia, HIV infection, receiving complement inhibitor [e.g., eculizumab, ravulizumab], or persistent complement component deficiency), and may also be considered routinely in adolescents age 16 years or older who are not at increased risk based on shared clinical decision-making.[20]

Adults

Tetravalent meningococcal conjugate vaccine (MenACWY) and serogroup B vaccine (MenB) are recommended in adults with high-risk conditions or at increased risk of disease (anatomic or functional asplenia, HIV infection, persistent complement component deficiency, or receiving complement inhibitor [e.g., eculizumab, ravulizumab]).[20] First-year college students who live in residential housing (if not previously

vaccinated at age 16 years or older) and military recruits should receive a single dose of MenACWY. MenACWY vaccination is also recommended for travel in countries with hyperendemic or epidemic meningococcal disease.[20]

Complement inhibition

Patients who will be treated with eculizumab or ravulizumab should receive meningococcal vaccines at least 2 weeks prior to commencing therapy, unless the risks of delaying this therapy outweigh the risks of developing meningococcal infection. Healthcare providers should also consider the use of antibacterial prophylaxis for patients receiving eculizumab or ravulizumab to reduce the risk of meningococcal disease.[20] [23]

Secondary prevention

All patients with presumed meningococcal infections should be cared for using droplet precautions until 24 hours of effective therapy has been completed. Patients should be hospitalized in private rooms. In addition to standard precautions, people should wear surgical masks when within 3 feet of the patient or within the patient's room, and the patient should wear a mask when transport outside their room is necessary. Masks and eye protection or face shields should be worn during procedures that are likely to result in exposure to patient secretions, such as endotracheal intubation.

Reportability

Any case of invasive meningococcal disease should be reported to local and state health departments.

Antibiotic prophylaxis

Close contacts of patients with meningococcal infections should receive chemoprophylaxis as soon as feasible, ideally within 24 hours of identification of the index case.[1] [32] Chemoprophylaxis is probably of little or no benefit when administered more than 14 days after the onset of disease in the index case.[32]

Most meningococcal infections in the US are sporadic; however, secondary cases may occur in contacts of patients with meningococcal infections.[32] Most secondary cases are diagnosed within 2 weeks of the index case. Close contacts include:

- Household members
- People with other close social contact (those who frequented the patient's residence or were directly exposed to patient's secretions by kissing or sharing of utensils within 7 days of the index case's illness)
- Air travelers seated directly next to patients on flights of over 8 hours' duration
- Healthcare providers having unprotected contact with patients' respiratory secretions

Give exposed healthcare providers antimicrobial prophylaxis, irrespective of their vaccination status. Exclude potentially infectious healthcare providers from work until 24 hours after the start of treatment.[32] Prior to discharge, administer a course of antimicrobial prophylaxis to patients with invasive meningococcal infections who are not treated with ceftriaxone or cefotaxime.[4] [50]

Although rifampin, ceftriaxone, and ciprofloxacin are all effective in eradicating meningococcal carriage, the emergence of resistance to rifampin has been noted following prophylactic use.[32] [88]

Immunoprophylaxis

In ongoing outbreaks of meningococcal infection caused by vaccine-preventable serogroup A, B, C, Y, and W-135 organisms, immunization of contacts may prevent secondary cases.[20] [32] The preferred vaccine varies according to the individual's age and the serotype of the outbreak strain.[4] [20] [89]

Screening for complement deficiency

Some authorities advise that all patients with invasive meningococcal infections should be screened for complement deficiency.[90] Immunization of patients with complement deficiencies with meningococcal polysaccharide vaccine reduces the risk of invasive infection, but rates remain significantly higher than in the general population.[91] Routine immunization with quadrivalent conjugate vaccine is recommended.[20]

Nasopharyngeal culture

May be helpful in identifying the serogroup of *Neisseria meningitidis* circulating in a community and whether immunization may be helpful in the prevention of secondary cases.

Patient discussions

After meningococcal meningitis, children, and adults who experience decreased hearing, should have a test as soon as possible (within 4-6 weeks).

Patients may need educational, occupational, or physical rehabilitation after infection.

Monitoring

Monitoring

As with all patients who survive bacterial meningitis, patients with meningococcal meningitis are at risk for long-term neurologic sequelae. Early detection of hearing loss and referral for rehabilitation is particularly important to ensure optimal speech development in young children.

FOLLOW UP

Complications

Complications	Timeframe	Likelihood
shock	**short term**	**medium**
Hypotension results from increased vascular permeability and, in the later stages of the illness, dysregulation of vascular tone. Both myocarditis and myocardial depression may contribute to poor tissue perfusion. Resuscitation should follow the principles of pediatric and adult advanced life support, with evaluation of the patient's airway, breathing, and circulatory status, and establishment of secure large-caliber intravenous catheters for administration of fluids and antibiotics.[51] [74] Inotropic medications should be administered to patients with hypotension or poor perfusion who do not respond promptly to fluid resuscitation.[74] The possibility of adrenal hemorrhage should be considered.		
elevated intracranial pressure (ICP)	**short term**	**medium**
Local inflammatory responses to bacteria, altered cerebral blood flow, and vasculitis lead to cerebral edema. Lumbar puncture is contraindicated in patients suspected of having elevated ICP. Patients with evidence of elevated ICP should be intubated and ventilated to maintain adequate oxygenation and normocapnia. Patients should be positioned with heads elevated 30° and in a midline position, and stimuli reduced by sedation and minimal handling. Seizures should be treated aggressively. Mannitol, furosemide, dexamethasone, and short periods of hyperventilation may be indicated for the acute treatment of severely elevated ICP.		
seizures	**short term**	**medium**
Seizures should be treated aggressively with benzodiazepines. Patients with seizure disorders generally require long-term anticonvulsant therapy.		
coagulopathy	**short term**	**medium**
Disseminated intravascular coagulation (DIC) is caused by acquired deficiencies of protein C, protein S, and antithrombin III; increases in plasminogen activator inhibitor and thrombin-activatable fibrinolysis inhibitor; and reduced activation of protein C on endothelial cells. Coagulation defects are corrected by fresh frozen plasma, coagulation factor concentrates, and platelet or cryoprecipitate infusion, to reduce the likelihood of hemorrhagic complications of infection.		
sensorineural hearing loss	**short term**	**medium**
Moderate to severe hearing loss occurs in 2% to 10% of survivors of meningococcal meningitis. Local inflammatory responses damage cochlear nerves, resulting in sensorineural hearing loss. The severity may vary from mild to profound, and hearing loss may be unilateral or bilateral.		

Complications	Timeframe	Likelihood
cognitive impairment, blindness, and motor and speech abnormalities	**short term**	**medium**
Vasculitis, intravascular thrombosis, cerebral edema, and direct toxicity to neurons may cause these neurologic sequelae. Early detection and referral for rehabilitation is important to ensure optimal outcomes. Survivors of severe meningococcal infections may have emotional, learning, and behavioral disorders that require multidisciplinary assessment and treatment.		
necrosis of skin and extremities	**short term**	**low**
Vasculitis, intravascular thrombosis, and tissue edema may lead to ischemic necrosis of skin, compartment syndrome, or other ischemic injury to extremities. These injuries may require surgical debridement or skin grafting and should be managed in collaboration with experienced plastic and orthopedic surgeons.		
adrenal insufficiency	**short term**	**low**
Acute adrenal insufficiency may be caused by adrenal hemorrhage, and functional adrenal insufficiency may occur in patients with sepsis. Patients with refractory symptoms of shock should receive replacement doses of corticosteroids until adrenal insufficiency can be excluded.		

Prognosis

The overall mortality rate of meningococcal infections is 10% to 15%.[19]

Patients with meningitis have a lower mortality rate (5%) than those with meningococcal sepsis (5% to 40%). Most deaths occur in the first 24 hours of illness. Mortality rates are higher in adolescents than in younger children, and higher during outbreaks than in sporadic disease.[87]

Clinical and demographic risk factors for adverse outcomes include age (infants, young adults, and those age >60 years), association with an outbreak, coma, hypotension, a rapidly progressive rash, absence of nuchal rigidity, focal neurologic signs, leukopenia/neutropenia, acidosis, thrombocytopenia, coagulopathy, and low serum C-reactive protein concentration.

Between 10% and 20% of survivors of meningococcal meningitis have permanent sequelae, including sensorineural hearing loss, seizure disorders, blindness, motor disorders, and intellectual impairment.[19] Thrombosis and tissue edema may result in skin necrosis, compartment syndrome, or other ischemic injury to extremities, and require skin grafts or amputation of digits or extremities.

Diagnostic guidelines

International

Guidelines for the management of suspected and confirmed bacterial meningitis in Canadian children older than 2 months of age [64]

Published by: Canadian Paediatric Society **Last published:** 2020

WHO guidelines on meningitis diagnosis, treatment and care [65]

Published by: World Health Organization **Last published:** 2025

WHO recommendations for management of serious bacterial infections in infants aged 0-59 days [66]

Published by: World Health Organization **Last published:** 2024

ESCMID guideline: diagnosis and treatment of acute bacterial meningitis [54]

Published by: European Society of Clinical Microbiology and Infectious Diseases **Last published:** 2016

Meningitis (bacterial) and meningococcal disease: recognition, diagnosis and management [47]

Published by: National Institute for Health and Care Excellence (UK) **Last published:** 2024

The UK joint specialist societies guideline on the diagnosis and management of acute meningitis and meningococcal sepsis in immunocompetent adults [48]

Published by: UK Joint Specialist Societies **Last published:** 2016

Treatment guidelines

International

Guidelines for the management of suspected and confirmed bacterial meningitis in Canadian children older than 2 months of age [64]

Published by: Canadian Paediatric Society **Last published:** 2020

WHO guidelines on meningitis diagnosis, treatment and care [65]

Published by: World Health Organization **Last published:** 2025

ESCMID guideline: diagnosis and treatment of acute bacterial meningitis [54]

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Published by: UK Joint Specialist Societies **Last published:** 2016

Online resources

1. CDC: adult immunization schedule by age - recommendations for ages 19 years or older *(external link)*
2. CDC: child and adolescent immunization schedule by age - recommendations for ages 18 years or younger *(external link)*
3. WHO: defeating meningitis by 2030 - a global road map *(external link)*

Key articles

- Centers for Disease Control and Prevention. Meningococcal disease. Feb 2024 [internet publication]. Full text
- Mbaeyi SA, Bozio CH, Duffy J, et al; Centers for Disease Control and Prevention. Meningococcal vaccination: recommendations of the Advisory Committee on Immunization Practices, United States, 2020. MMWR Recomm Rep. 2020 Sep 25;69(9):1-41. Full text Abstract
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