BG Pulse Journal USA
ISSN:1947-2536 http://www.bgpulseusa.com/index.php/bg
BG PULSE JOURNAL

BACTERIAL MENINGITIS IN CHILDREN: A REVIEW

Rashidova Guljahon O‘ktam qizi
Department of Preclinical Medical Sciences, Asian International University, Bukhara, Uzbekistan

https://orcid.org/0009-0006-1936-6821

Abstract: Bacterial meningitis remains one of the most severe infectious emergencies in pediatric medicine and continues to contribute substantially to global childhood morbidity and mortality despite major advances in vaccination and antimicrobial therapy. The disease is characterized by acute inflammation of the meninges caused by invasive bacterial pathogens, resulting in rapid neurological deterioration, systemic inflammatory response, and potentially irreversible complications. Neonates and young children are particularly vulnerable because of immature immune defense mechanisms and increased susceptibility to hematogenous dissemination of pathogens into the central nervous system.

The principal causative organisms vary according to age and geographic region and include Group B Streptococcus, Escherichia coli, Listeria monocytogenes, Streptococcus pneumoniae, Neisseria meningitidis, and Haemophilus influenzae type b. Clinical manifestations range from nonspecific symptoms in neonates, such as poor feeding, lethargy, irritability, and temperature instability, to classical meningeal signs in older children, including fever, severe headache, neck stiffness, vomiting, photophobia, and altered consciousness. Rapid progression to septic shock, cerebral edema, hydrocephalus, seizures, hearing loss, and long-term neurodevelopmental impairment is common in severe disease.

Diagnosis relies on cerebrospinal fluid analysis obtained through lumbar puncture, supported by microbiological cultures, polymerase chain reaction assays, inflammatory biomarkers, and neuroimaging techniques. Early empirical antimicrobial therapy combined with supportive intensive care and adjunctive corticosteroids significantly improves outcomes and reduces neurological sequelae. Widespread implementation of conjugate vaccines against major bacterial pathogens has substantially reduced disease incidence worldwide; however, bacterial meningitis continues to represent a serious public health challenge, particularly in low-resource settings.

This review summarizes the epidemiology, pathogenesis, clinical manifestations, laboratory diagnosis, differential diagnosis, treatment principles, complications, and preventive strategies of bacterial meningitis in children, emphasizing the importance of rapid diagnosis and evidence-based management to improve survival and neurological outcomes.

Keywords: Bacterial meningitis; Children; Pediatric infections; Streptococcus pneumoniae; Neisseria meningitidis; Cerebrospinal fluid; Neuroinflammation; Vaccination; Pediatric neurology; Central nervous system infections

Introduction

Bacterial meningitis is an acute inflammatory disease involving the meninges and cerebrospinal fluid surrounding the brain and spinal cord. Despite significant advances in antimicrobial therapy, vaccination strategies, and critical care medicine, bacterial meningitis remains one of the leading causes of infectious neurological morbidity and mortality among children worldwide. The disease progresses rapidly and constitutes a true pediatric medical emergency because delayed diagnosis and treatment are strongly associated with poor neurological outcomes and death.

The global burden of bacterial meningitis remains substantial, particularly in developing countries where healthcare access, vaccination coverage, and early diagnostic facilities are limited. According to the World Health Organization, hundreds of thousands of cases continue to occur annually, with the highest incidence observed in neonates and children under five years of age. Mortality rates vary depending on the causative pathogen, age, nutritional status, and quality of medical care, ranging from 5–10% in developed countries to more than 30% in resource-limited regions.

The introduction of conjugate vaccines against Haemophilus influenzae type b, Streptococcus pneumoniae, and Neisseria meningitidis has dramatically reduced the incidence of pediatric bacterial meningitis in many countries. Nevertheless, emerging antimicrobial resistance, incomplete vaccine coverage, delayed recognition, and severe neurological sequelae continue to present major clinical challenges.

This review discusses current knowledge regarding the epidemiology, etiopathogenesis, clinical presentation, laboratory diagnosis, differential diagnosis, therapeutic strategies, complications, and prevention of bacterial meningitis in pediatric patients.

Epidemiology

The epidemiological characteristics of bacterial meningitis vary according to age, geographic distribution, socioeconomic conditions, and vaccination coverage. Neonates and infants remain the most vulnerable populations due to immature immune responses and increased permeability of the blood-brain barrier.

In neonatal meningitis, Group B Streptococcus and Escherichia coli are the predominant causative organisms, commonly acquired vertically during childbirth. Listeria monocytogenes remains an important pathogen in neonates and immunocompromised individuals. In older infants and children, Streptococcus pneumoniae and Neisseria meningitidis represent the most common etiological agents worldwide.

Before widespread immunization programs, Haemophilus influenzae type b was a major cause of bacterial meningitis in young children. However, introduction of Hib conjugate vaccines has resulted in a dramatic decline in incidence and mortality. Similar reductions have been observed following implementation of pneumococcal and meningococcal vaccination programs.

Outbreaks of meningococcal meningitis continue to occur, particularly in the “meningitis belt” of sub-Saharan Africa, where seasonal epidemics contribute significantly to childhood mortality. Risk factors associated with bacterial meningitis include overcrowding, malnutrition, immunodeficiency, lack of vaccination, prematurity, and chronic medical conditions.

Etiopathogenesis

The pathogenesis of bacterial meningitis involves complex interactions between invading pathogens and host immune responses. Infection usually begins with colonization of the nasopharyngeal mucosa, followed by bacterial invasion into the bloodstream and subsequent penetration of the blood-brain barrier.

Several bacterial virulence factors facilitate central nervous system invasion. Polysaccharide capsules protect bacteria from phagocytosis, while adhesins and pili promote attachment to epithelial and endothelial surfaces. After entering the subarachnoid space, bacteria multiply rapidly because host immune defenses within cerebrospinal fluid are limited.

Bacterial cell wall components stimulate activation of macrophages, neutrophils, endothelial cells, and microglia, resulting in the release of proinflammatory cytokines including tumor necrosis factor-alpha, interleukin-1β, and interleukin-6. This inflammatory cascade increases blood-brain barrier permeability and promotes leukocyte migration into the cerebrospinal fluid.

The resulting inflammatory response contributes significantly to neuronal injury. Vasogenic and cytotoxic cerebral edema, impaired cerebral perfusion, increased intracranial pressure, oxidative stress, and excitotoxic neuronal damage may ultimately lead to cerebral ischemia, infarction, and brain herniation. In severe cases, systemic inflammatory response syndrome, septic shock, disseminated intravascular coagulation, and multiorgan failure may develop.

Children are particularly susceptible to rapid neurological deterioration because of their immature immune systems and limited physiological reserves.

Clinical Manifestations

Clinical manifestations of bacterial meningitis vary considerably according to patient age, causative organism, and disease severity.

Neonatal bacterial meningitis frequently presents with nonspecific symptoms, making early recognition difficult. Common manifestations include poor feeding, lethargy, irritability, weak cry, apnea, vomiting, hypotonia, temperature instability, seizures, and bulging anterior fontanelle. Classical meningeal signs are frequently absent in this age group.

In infants and young children, symptoms commonly include fever, persistent vomiting, excessive crying, irritability, photophobia, refusal to eat, lethargy, and altered consciousness. Seizures occur relatively frequently because the immature pediatric brain is highly sensitive to inflammatory and metabolic disturbances.

Older children and adolescents typically develop classical meningeal symptoms including severe headache, high fever, neck stiffness, photophobia, nausea, vomiting, and confusion. Kernig and Brudzinski signs may be present during physical examination. Meningococcal disease may additionally produce petechial or purpuric rash secondary to meningococcemia, indicating severe systemic involvement and poor prognosis.

The disease may progress rapidly within several hours, leading to septic shock, cerebral edema, coma, respiratory failure, and death if appropriate treatment is delayed.

Differential Diagnosis

Bacterial meningitis should be differentiated from numerous infectious and noninfectious neurological conditions presenting with fever, altered mental status, and meningeal irritation.

Viral meningitis is generally characterized by a milder clinical course and cerebrospinal fluid lymphocytic predominance with normal glucose levels. Enteroviruses, herpes simplex virus, and arboviruses represent common etiological agents.

Viral encephalitis may present with seizures, altered consciousness, focal neurological deficits, and behavioral changes resembling bacterial meningitis. Herpes simplex encephalitis is particularly important because delayed antiviral treatment may result in severe neurological injury.

Tuberculous meningitis usually develops subacutely with prolonged fever, weight loss, cranial nerve palsies, and progressive neurological deterioration. Cerebrospinal fluid findings include lymphocytic pleocytosis, elevated protein, and markedly decreased glucose concentration.

Febrile seizures in young children may initially mimic meningitis; however, persistent neurological abnormalities and meningeal signs are typically absent. Brain abscess, intracranial hemorrhage, sepsis, toxic-metabolic encephalopathy, and autoimmune neurological disorders should also be considered in differential diagnosis.

Accurate differentiation requires careful clinical evaluation combined with laboratory investigation and neuroimaging studies.

Laboratory Diagnosis

Lumbar puncture with cerebrospinal fluid analysis remains the gold standard for diagnosis of bacterial meningitis. Typical cerebrospinal fluid findings include elevated opening pressure, turbid appearance, neutrophilic pleocytosis, increased protein concentration, and decreased glucose levels. Cerebrospinal fluid glucose concentrations are commonly below 40 mg/dL or less than 50% of simultaneous serum glucose values.

Gram staining and cerebrospinal fluid culture provide direct microbiological confirmation and remain essential diagnostic methods despite reduced sensitivity following prior antibiotic administration. Blood cultures should also be obtained before initiation of antimicrobial therapy whenever possible.

Peripheral blood analysis frequently demonstrates leukocytosis with neutrophilic predominance, elevated erythrocyte sedimentation rate, increased C-reactive protein concentration, and elevated procalcitonin levels. Severe systemic infection may additionally result in thrombocytopenia and coagulation abnormalities.

Polymerase chain reaction assays have significantly improved diagnostic sensitivity and specificity, particularly in partially treated patients. Molecular diagnostic techniques enable rapid identification of bacterial pathogens and antimicrobial resistance genes, facilitating early targeted therapy.

Computed tomography and magnetic resonance imaging are useful for identifying complications such as cerebral edema, hydrocephalus, infarction, abscess formation, and subdural effusions. Neuroimaging is particularly important in patients presenting with focal neurological deficits, papilledema, prolonged seizures, or suspected intracranial complications.

Treatment Principles

Bacterial meningitis requires immediate empirical antimicrobial therapy because delays in treatment are strongly associated with increased mortality and neurological sequelae.

Empirical antibiotic selection depends on patient age and likely pathogens. In neonates, treatment commonly includes ampicillin combined with gentamicin or cefotaxime to provide coverage against Group B Streptococcus, Escherichia coli, and Listeria monocytogenes. In older infants and children, third-generation cephalosporins such as ceftriaxone or cefotaxime combined with vancomycin are commonly recommended.

After microbiological identification and antimicrobial susceptibility testing, therapy should be adjusted accordingly. Duration of treatment varies depending on the causative organism and clinical response.

Adjunctive dexamethasone therapy administered before or together with the first antibiotic dose may reduce inflammatory-mediated neurological complications, particularly hearing loss associated with Haemophilus influenzae type b meningitis.

Supportive management remains essential and includes adequate hydration, oxygen therapy, antipyretics, seizure control, electrolyte correction, intensive neurological monitoring, and management of increased intracranial pressure. Critically ill patients may require mechanical ventilation, vasopressor support, and admission to intensive care units.

Complications

Despite modern therapeutic advances, bacterial meningitis remains associated with significant short-term and long-term complications.

Neurological sequelae include sensorineural hearing loss, hydrocephalus, epilepsy, cerebral palsy, visual impairment, cognitive dysfunction, learning disabilities, and behavioral abnormalities. Hearing impairment represents one of the most common permanent complications in pediatric survivors.

Acute complications may include cerebral edema, septic shock, disseminated intravascular coagulation, subdural empyema, cerebral infarction, and respiratory failure. Mortality rates remain highest among neonates, immunocompromised children, and patients with delayed initiation of therapy.

Long-term neurodevelopmental impairment significantly affects quality of life and emphasizes the importance of early diagnosis, rapid treatment, and prolonged neurological follow-up.

Prevention

Vaccination remains the cornerstone of prevention of bacterial meningitis in children. Introduction of conjugate vaccines against Haemophilus influenzae type b, Streptococcus pneumoniae, and Neisseria meningitidis has substantially reduced disease incidence and mortality worldwide.

Maternal screening and intrapartum antibiotic prophylaxis for Group B Streptococcus significantly decrease neonatal infections. Additional preventive measures include improved

sanitation, infection control practices, chemoprophylaxis for close meningococcal contacts, and public health education.

Global public health initiatives aimed at improving vaccination coverage and access to early medical care remain essential for reducing the burden of pediatric bacterial meningitis.

Conclusion

Bacterial meningitis continues to represent one of the most severe pediatric infectious diseases worldwide despite considerable progress in vaccination programs, antimicrobial therapy, and intensive care medicine. The disease is characterized by rapid progression, severe neuroinflammatory responses, and high risk of mortality and long-term neurological sequelae, particularly among neonates and young children.

Clinical manifestations vary according to age, often complicating early diagnosis in neonates because of nonspecific presentations. Cerebrospinal fluid analysis remains the cornerstone of diagnosis, while modern molecular diagnostic techniques have improved rapid pathogen detection and therapeutic decision-making.

Early empirical antimicrobial therapy, adjunctive corticosteroids, intensive supportive care, and careful neurological monitoring are essential for improving survival and reducing complications. Continued expansion of vaccination programs against major bacterial pathogens has significantly decreased global incidence; however, important challenges remain, including antimicrobial resistance, unequal vaccine access, and limited healthcare resources in developing countries.

Future research should focus on rapid molecular diagnostics, neuroprotective therapies, optimization of antimicrobial strategies, and strengthening global immunization programs to further reduce the burden of pediatric bacterial meningitis and improve long-term neurological outcomes.

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