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Review Article

Acute Bacterial Meningitis: A Systemic Review of Diagnosis and Treatment

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Abstract

Bacterial meningitis is a medical emergency requiring immediate diagnosis and immediate treatment. Streptococcus pneumoniae and Neisseria meningitidis are the most common and most aggressive pathogens of meningitis. Emerging antibiotic resistance is an upcoming challenge. Clinical and experimental studies have established a more detailed understanding of the mechanisms resulting in brain damage, sequelae and neuropsychological deficits. We summarize the current pathophysiological concept of acute bacterial meningitis and present current treatment strategies.

Keywords

Bacterial meningitis, meningoencephalitis, pneumococci, meningococci, dexamethasone

Introduction

Bacterial meningitis is an inflammation of the meninges, in particular the arachnoid and the pia mater, associated with the invasion of bacteria into the subarachnoid space, principles known for more than 100 years . The pathogens take advantage of the specific features of the immune system in the CNS, replicate and induce inflammation. A hallmark of bacterial meningitis is the recruitment of highly activated leukocytes into the CSF. Beside bacteria, viruses, fungi and non-infectious causes as in systemic and neoplastic disease as well as certain drugs can induce meningeal inflammation. Usually the inflammatory process is not limited to the meninges surrounding the brain but also affects the brain parenchyma (meningoencephalitis) , the ventricles (ventriculitis) and spreads along the spinal cord . In recent years the damage of neurons, particularly in hippocampal structures, has been identified as a potential cause of persistent neuropsychological deficits in survivors.Bacterial meningitis is a medical emergency requiring immediate diagnosis and subsequent treatment.

Epidemiology

The risk of disease is highest in individuals younger than 5 years and older than 60 years. Some predisposing factors such as a former splenectomy, malnutrition or sickle cell disease are known The use of conjugate pneumococcal vaccines has led to a significant decline in invasive pneumococcal disease, including meningitis, in those regions promoting this approach An emerging problem is the growing prevalence of pneumococci resistant to beta-lactam antibiotics Prolonged persistence of pneumococci in the cerebrospinal fluid (CSF) may result in higher mortality as well as in pronounced neurological damage in survivors [These effects of living bacteria urge us to understand in detail the effects of bacterial toxins and released cell wall and surface components and their contribution to neuronal damage.

With *Haemophilus* on the decline, *Neisseria meningitides* has become the leading meningitis pathogen in developing countries, but it continues to pose a major health problem in the US and Europe. In addition to classical meningitis, meningococci frequently cause systemic disease including fulminant gram-negative sepsis and disseminated intravascular coagulopathy. WHO estimates at least 500 000 newly symptomatic infections per year worldwide, leading to at least 50 000 deaths.

The highest incidence is observed in the sub-Sahara meningitis belt where cyclic epidemics occur at least once per decade.

Pathogenesis

Bacterial invasion

The current assumption is that high-grade bacteremia precedes meningitis and that bacteria invade from the blood stream to the central nervous system (CNS). Alternatively, direct accesses to the CNS through dural defects or local infections are potential entrance routes. In the clinical setting, such defects should be identified by CCT or MRI scans.

The anatomical site of bacterial invasion from the bloodstream remains unidentified. Experimental evidence suggests that the choroid plexus may be a site of invasion [Daum *et al.* 1978]. Meningococci are found in the choroid plexus as well as in the meninges [Pron *et al.* 1997] and pneumococci infiltrate the leptomeningeal blood vessels

Rodriguez *et al.* 1991] in meningitis. These data suggest that several highly vascularized sites are potential entry locations. In order to cross the blood—brain or the blood—CSF barrier and to overcome sophisticated structures such as tight junctions, meningeal pathogens must carry effective molecular tools.

Inflammatory response

Inflammatory activation of endothelial cells seems to be a prerequisite for bacterial invasion but also results in the regulation of adhesion molecules as ICAM-1. Subsequently, these molecules promote the multistep process of leukocyte invasion. Leukocytes, in particular the presence of granulocytes in the CSF, are the diagnostic hallmark of meningitis. Early inflammatory response and bacterial invasion seem to progress in parallel and products of activated leukocytes such as MMPs and NO and others contribute to early damage of the blood—brain and blood—CSF barrier. Once bacteria have entered the subarachnoidal space, they replicate, undergo autolysis and cause further inflammation.

Neuronal damage

Up to 50% of survivors of bacterial meningitis suffer from disabling neuropsychological deficits clinically as well as experimentally, the hippocampus seems to be the most vulnerable area of the brain Neuronal loss translates into hippocampal atrophy and has been reported on MRI scans in survivors of bacterial meningitis .

The predisposition of the hippocampus for neuronal damage remains unclear. The extracellular fluid around brain cells is contiguous with the CSF and the proximity to the ventricular system allows diffusion between these compartments that could deliver soluble bacterial and inflammatory toxic mediators.

Treatment

Immediate antibiotic therapy is imperative and must not be postponed by diagnostic delays; for example, waiting for a CT scan. Prehospital antibiotic treatment is advised in cases of suspected meningococcal disease but depends on local resistance situation and the medical environment Prior to treatment, a blood culture should be obtained. Since microbiological identification of the pathogen is not immediately available, the initial choice of antibiotics is usually empirical.

Corticosteroids

Corticosteroids reduce brain edema, intracranial hypertension and meningeal inflammation in experimental models of bacterial meningitis. Subsequent clinical studies have led to conflicting results concerning potential benefits of steroid use in patients with meningitis. Currently available evidence supports a reduced incidence of severe hearing loss in children with *H. influenzae* meningitis while information on other pediatric pathogens is incomplete. In adults, a single double-blind RCT of 301 adult patients reported reduced mortality and lower frequency of hearing loss and neuropsychological sequelae Subgroup analysis suggested that protective effects of dexamethasone are limited to pneumococcal meningitis (death: 34% *versus* 14%; unfavorable outcome: 52% *versus* 26%) Expert opinion and several societal guidelines recommend routine treatment with dexamethasone for community-acquired meningitis of children (0.15mg/kg every 6 hours for 2–4 days) and adults (10 mg every 6 hours for 4 days). Discontinuation of this therapy is advisable if *H. influenzae* (children) and S. *pneumoniae* (adults and children) can be ruled out as the underlying pathogen.

Complications

Mortality from bacterial meningitis may reach 34% and is highest with *S. pneumoniae* and *L. meningitidis.* Long-term neurological sequelae are found in up to 50% of survivors Both intracranial and systemic complications contribute to this negative outcome. Complications are most likely to occur during the first few days of therapy. Sensorineural hearing loss or vestibular dysfunction are the most frequent problems. They are most frequent with *H. influenzae* and *S. pneumoniae.* As outlined above, the incidence of these complications is reduced by adjunctive dexamethasone therapy. The most threatening intracranial complications are brain edema, vascular alterations and hydrocephalus, which all contribute to increased intracranial pressure and parenchymal damage.

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