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Perspective

Treatment and Management of Bacterial Meningitis

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INTRODUCTION

Bacterial meningitis is the most common severe infection of the central nervous system. Infection of the subarachnoid space leads to Cerebro Spinal Fluid (CSF) inflammation, meningeal irritation, and the clinical trial of headache, fever, and meningioma's. Few ailments have been affected more by the arrival of antimicrobial therapy than bacterial meningitis. From its recognition in 1805 to the early 20th century, bacterial meningitis was lethal. Although the introduction of antibiotics made it curable, morbidity and mortality from the ailment remain unacceptably high. Few years after the arrival of antibiotics for clinical use, bacterial meningitis remains a significant cause of morbidity and mortality. As such, it denotes a unique human infectious ailment, because the pathophysiologic effects of ailment progress and suboptimal outcomes occur despite bacteriologic therapy of the infection. Specifically, the mortality rate for adults who have Streptococcus pneumonia meningitis remains 20 to 30 percent, with neurologic morbidity affecting half of survivors. The occurrence of insistent sensor neural hearing loss in children who survive bacterial meningitis is 10 percent.

DESCRIPTION

Nearly 10,000 cases of bacterial meningitis occur each year in the United States, whereas other parts of the world have considerably higher occurrences. Even when cured with highly effective antibiotics, the ailment is fatal in 5% to 40% of the patients and causes neurologic consequences in up to 30% of the survivors. Neurologic consequences from bacterial meningitis can be grouped into three types: (1) Hearing impairment, which is most commonly the outcome of direct invasion of the inner ear by the subarachnoid space inflammation; (2) Obstructive hydrocephalus; and (3) Damage to the brain parenchyma, leading to neurologic consequences such as focal sensory-motor deficits, mental retardation, and seizure disorders.

The present 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 faults or local infections are potential entering routes. In the clinical setting, such faults should be recognized by CCT or MRI scans. The anatomical site of bacterial attack from the bloodstream remains unidentified. Experimental evidence recommends that the choroid plexus may be a site of invasion. Meningococci are found in the choroid plexus as well as in the meninges and pneumococci infiltrate the leptomeningeal blood vessels in meningitis. These data suggest that numerous highly vascularized sites are potential entry sites. 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. Streptococcal proteins such as CbpA interact with glycoconjugate receptors of phosphorylcholine with Platelet Activating Factor (PAF) on the eukaryotic cells and endorse endocytosis and crossing the blood brain barrier. Meningococci's PilC1 adhesin interacts with CD46 and the outer membrane protein connects to vitronectin and integrin's. Bacteria triggering meningitis in newborns, most importantly Group B Streptococcal (GBS) and *E. coli*, are also well equipped with adhesive proteins allowing them to invade the CNS.

Patients with bacterial meningitis are commonly treated by primary care and emergency medicine physicians at the time of primary presentation, frequently in consultation with infectious diseases specialists, neurologists, and neurosurgeons. In contrast to many other infectious diseases, the antimicrobial rehabilitation for bacterial meningitis is not continuously based on randomized, prospective, double-blind clinical trials, but rather on data primarily obtained from experimental animal models of infections. A model usually utilized is the experimental rabbit model, in which animals are anesthetized and placed in a stereotactic frame. In this procedure, the cisterna magna can be punctured for repeated sampling of CSF and injection of microorganisms. Frequent sampling of CSF permits measurement of leukocytes and chemical parameters and quantitation of the relative penetration of antimicrobial agents into CSF and the effects of meningitis on this entry parameter, the relative bactericidal efficacy (defined as the rate of bacterial eradication) within purulent CSF, and CSF pharmacodynamics. Results obtained from these and other animal models have led

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to clinical trials of particular agents in patients with bacterial meningitis.

CONCLUSION

Bacterial meningitis kills or harms about a fifth of people with the ailment. Early antibiotic therapy improves outcomes, but the effectiveness of widely existing antibiotics is threatened by global emergence of multidrug-resistant bacteria. New antibiotics, such as fluoroquinolone, could have a role in these conditions, but clinical data to support this notion are scarce. Additionally, whether or not adjunctive anti-inflammatory rehabilitations (e.g., dexamethasone) progress outcomes in patients with bacterial meningitis remains controversial; in resource-poor regions, where the ailment burden is highest, dexamethasone is ineffective. Other adjunctive therapeutic approaches, such as glycerol, paracetamol, and induction of hypothermia, are being tested further. Therefore, bacterial meningitis is a significant and evolving therapeutic challenge.