Central Nervous System Infections: A Brief Review on Epidemiological Study in group of Population

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Abstract
Glycemic control is an important aspect of patient care in the surgical. Infections of the nervous system are among the most difficult infections in terms of morbidity and mortality posed to patients, and thereby require urgent and accurate diagnosis. Although viral meningitides are more common, it is the bacterial meningitides that have the potential to cause a rapidly deteriorating condition that the physician should be familiar with. Viral encephalitis frequently accompanies viral meningitis, and can produce focal neurologic findings and cognitive difficulties that can mimic other neurologic disorders. Brain abscesses also have the potential to mimic and present like other neurologic disorders, and cause more focal deficits. Finally, other infectious diseases of the central nervous system, such as prion disease and cavernous sinus thrombosis, are explored in this review.

Keywords
Brain, encephalitis, infectious, meningitis, nervous

Introduction
Infections of the central nervous system (CNS) pose a unique challenge to physicians, due to both the potential morbidity and mortality that they cause as well as the inherent difficulties involved in their treatment. These infections mainly involve meningitis, encephalitis, and brain abscesses, and tend to cause more morbidity and mortality on average than infections involving other organ systems. Due to their potential for adverse consequences, it is important for the physician to be well versed in the presentations and care of the more common CNS infections, as these represent a not infrequent etiology of complaints seen from the primary care clinic to the surgeon’s bed to even the psychiatric ward.

Meningitis
Streptococcus pneumoniae
Streptococcal meningitis from the causative organism S. pneumoniae is the leading cause of meningitis in the elderly, and one of the leading causes of bacterial meningitis in all adults and children older than 2 months. The illness is usually not as rapid and fulminant as that caused by meningococcal meningitis, but nonetheless can present with similar symptoms. Workup is similar to those patients suspected of having meningococcal meningitis, with LP preceding empiric treatment with a third-generation cephalosporin. LP should return gram-positive diplococci. In cases where resistance to cephalosporin is suspected, vancomycin should be added for additional coverage. Out of the several species of bacterial meningitis, clinical trials to date have provided the greatest benefits for adjuvant dexamethasone therapy for pneumococcal meningitis. An estimated 1 in 12 cases of streptococcal meningitis is fatal, with 1 in 3–4 survivors suffering neurologic sequelae, including deafness, persistent seizures, and mental retardation in children. Since the advent of the heptavalent pneumococcal vaccine in 2000, pneumococcal meningitis has decreased significantly in children, estimated by the Center for Disease Control (CDC) to be around 77% lower than immediately before vaccine introduction, while diminishing in adults as well due to herd immunity.

Haemophilus influenzae
Haemophilus influenzae, particularly serotype B (HiB), is a frequent cause of meningitis in children under the age of 5 years. This organism is a gram-negative rod and a frequent inhabitant of the sinuses, inner and middle ear, respiratory tract, and bloodstream; it commonly causes meningitis via a combination of both direct sinus and hematogenous spread. The frequency of HiB strain in the United States has decreased dramatically, however, in the past couple of years since the advent of the HiB vaccine, and is now a distant third behind streptococcus and meningococcus in childhood meningitis.

Fungal Meningitis
Common fungal agents of meningitis include Cryptococcus neoformans, Candida albicans, Histoplasma capsulatum, Blastomyces, and Coccidioides immitis Susceptible patients are usually either immunocompromised or have undergone direct neurosurgical interventions, such as shunt placement. The primary method of spread in most cases involves respiratory infection with subsequent hematogenous dissemination. Symptoms are typical of those of meningitis, including fever, headache, AMS, nausea, vomiting, and neck stiffness, in addition to complications of abscess, papilledema, seizures, and focal neurologic deficits.

Lyme Disease
Lyme disease, classically obtained from bites from the Ixodes scapularis tick carrying the causative agent Borrelia burgdorferi, is primarily seen in the eastern United States, particularly in the northeastern regions.
Neurologic symptoms of Lyme disease are preceded by an annular skin rash termed erythema migrans and nonspecific symptoms of a low-grade fever, malaise, and fatigue. The neurologic symptoms of Lyme disease begin to occur roughly about a month after initial tick bite, and frequently include focal neurologic findings as well as signs and symptoms of meningismus. The pathogenesis of CNS disease includes direct invasion by the bacterium itself in addition to vascular invasion. Focal neurologic findings seen typically include a sixth nerve palsy, but the organism can also affect cranial nerves III, IV, V, VII, and VIII. Sensory deficits, especially in the face, are frequently seen. Papillitis and posterior uveitis have been causally linked to Lyme disease as well; optic neuritis, however, has not.

**Neurosyphilis**

One of the world’s oldest infectious diseases, syphilis is caused by the spirochete *Treponema pallidum*. Although initially spread often as a sexually transmitted infection, disseminated, chronic untreated syphilis can affect all aspects of the nervous system, from peripheral nerves to the brain and spinal cord. Although most chronic forms of syphilis tend to be asymptomatic, a variety of focal neurologic findings, including cranial nerve abnormalities as well as meningeal signs of fever, headache, and stiff neck, are often seen. Less common than these classical symptoms, neurosyphilis also has the capability of presenting with psychiatric disturbances, movement disorders, hearing loss, dementia, stroke-like syndrome, seizures, or even mimicking amyotrophic lateral sclerosis.

**Equine encephalitis**

The equine encephalitides, including EEE, western equine encephalitis (WEE), and Venezuelan encephalitis (VEE) are caused by alphaviruses, single-stranded positive-sense RNA viruses belonging to the Togaviridae family. Although relatively uncommon, these infections, in particular EEE, have the potential to cause much morbidity and possess a high rate of mortality, demanding their urgent and accurate diagnosis by the emergency physician.

EEE has its major incidence along the southeastern United States found in hotter swampy locations inhabited by the mosquito *Culex tarsalis*, its primary vector; birds are the major reservoir of disease. Clinical presentation is nonspecific, and can involve a typical viral prodrome of flu-like symptoms, including fever, chills, malaise, and myalgias lasting for up to a couple of weeks followed by encephalitis involving severe headache, nausea, vomiting, AMS, and focal neurologic deficits with complications of seizures, nerve palsies, coma, and death.

**Flavivirus Encephalitis**

The flaviviruses encompass a large group of viruses causing a widespread range of diseases with high global morbidity and mortality, including the causative agents of West Nile encephalitis, St. Louis encephalitis, Tick-borne encephalitis, Japanese encephalitis, and others.

**Rabies**

The rabies virus is part of the Rhabdoviridae family of viruses, which comprises a linear, single-stranded negative sense RNA virus found within a bullet-shaped envelop. It is a zoonotic infection, spreading from animals to humans mainly by way of bite, with the primary culprits in the United States being bats, raccoons, and skunks, whereas the primary culprits worldwide are dogs. Rodents and hares on the other hand have not been known to carry or transmit the virus to humans. Although animal bite is the primary mode of transmission, infected aerosolized bat urine from entering heavily infested caves is a significant source of disease. Incubation of the virus typically takes place over 1–3 months, and CNS infection takes place through the process of retrograde spread of the virus from peripheral nerves unto the central nerves and finally into the brain parenchyma. Infection with the rabies virus is always fatal once encephalitis occurs, and therefore once a suspected exposure occurs, immediate treatment is of utmost importance.

**Cerebral abscess**

Cerebral abscesses can frequently present with a new-onset headache that can evolve over several hours to several weeks, accompanied by focal neurologic deficits. Lethargy, nuchal rigidity, nausea, vomiting, and new-onset seizures often accompany the development of abscesses. The two major causes of brain abscesses include hematogenous spread of pathogens across the blood–brain barrier and via direct contiguous spread from the sinuses after a sinus infection, with the latter accounting for over 70% of all brain abscesses.

**Toxoplasmosis**

Toxoplasmosis, from the causative agent *Toxoplasma gondii*, frequently occurs in immunocompromised individuals, such as end-stage HIV patients, those on systemic chemotherapy, in patients with hereditary immunologic disorders, or in the infants of pregnant women exposed to cat litter. Congenital toxoplasmosis presents with mental retardation, seizures due to calcification of basal ganglia, blindness, and death in infants.

**Conclusion**

Infections of the CNS represent a significant source of morbidity and mortality throughout the world, and demand that physicians in a variety of specialties are familiar enough with their presenting signs and symptoms and can formulate a diagnosis in time before further damage arises. Despite advances in vaccinations, meningitis arising from *N. meningitides* and *S. pneumoniae* species in adults and children and from Group B Streptococcus, *E. coli*, and *Listeria* species in neonates remain common reasons for presentation to the emergency room.

**References**