NURS 821 Neurological Disorders

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Lecture 11

Part 3 Disorders of the Central Nervous System:Infections and Inflammation

Infections of the CNS

Result from:

- Bacteria
- Viruses
- Fungi
- Protozoans
- Rickettsiae

Meningitis

- Definition: primary infection of pia mater and arachnoid, the subarachnoid space, ventricular system, and the cerebrospinal fluid (CSF)
- May be acute, subacute, or chronic
- Causes:
  - Bacteria
  - Viruses
  - Fungi
  - Parasites
  - Toxins

Meningitis

- Pathophysiology and clinical manifestations vary with causative organism
- viral infection- more common
- bacteriological infection- more lethal

Bacterial Meningitis

- The most common causes are:
  - Meningococcus (Neisseria meningitidis)
  - Pneumococcus (Streptococcus pneumoniae)
  - Haemophilus influenza

Bacteriologic Infection of the CNS

- Pyogenic (bacteriological) infections reach CNS by:
  - Hematogenous spread-emboli of bacteria or infected thrombi
  - Extension from cranial structures-ears, paranasal sinuses, skull infection, penetrating cranial injuries, or congenital sinus tracts
  - Iatrogenic - being introduced in the course of cerebral or spinal surgery

Bacterial Meningitis

- Meningococcus meningitis
  - Worldwide
  - Predominantly in men and boys
  - More frequently in the fall, winter, and spring
  - Epidemics occur in about 10 year cycles, affecting mostly children and adolescents

Bacterial Meningitis

- Pneumococcal meningitis
  - Young and those over 40 are most affected
- Haemophilus influenzae
  - Exclusively in children 2 month to 7 years

Bacterial Meningitis

- Pathophysiology:
  - Bacteria are normal nasopharyngeal flora
  - Conditions such as a URI must be present before the bacteria become blood borne

Bacterial Meningitis

- Bacteria or toxins irritate, inducing an inflammatory response in the pia, arachnoid, CSF, and ventricles ⇨ increased permeability of meningeal vessels ⇨ neutrophils migrate into the subarachnoid space producing an exudate ⇨ thickening of the CSF ⇨ interfering with normal CSF flow around the brain and spinal cord ⇨…

Bacterial Meningitis

- …exudate can obstruct arachnoid villi ⇨ hydrocephalus ⇨ purulent exudate increases rapidly (especially around the base of the brain) ⇨meningeal cells become edematous ⇨ increased intracranial pressure (ICP) ⇨ small and medium sized subarachnoid vasculature engorge, disrupting flow ⇨ producing thrombosis…death may ensue

Bacterial Meningitis

Clinical features arise from meningeal inflammation:

- Generalized, throbbing, severe HA
- Forceful flexion of the neck causing flexion of the legs and thighs (Brudzinski reflex)
- Due to extreme pain, inability to extend leg with hip flexed at a right angle (Kernig sign)

Bacterial Meningitis Clinical Features

Irritation and damage affect cranial nerves, cervical spinal nerves, the vomiting center, and the cortex

Fever, petechial or purpuric rash covering skin and mucous membranes

- **With increased ICP:**
  - **Papilledema**
  - **Delirium**
  - **Unconsciousness, death**
- **CSF: increased WBC with predominance of neutrophils (PMNs) and low CSF glucose**

Viral Infection of the CNS

Enters the body by:

- Mumps, measles, and varicella enter via the respiratory passages
- Polioviruses and other enteroviruses enter by the oral-intestinal route

Viral Infection of the CNS

- Etiology: animal or mosquito bites (Arbovirus infection)
- Fetus infected transplacentally by rubella virus, CMV, and HIV
- Infection passed along peripheral nerves
  - Centripetal movement of virus is accomplished by retrograde axoplasmic transport system
  - Herpes simplex, Varicella Zoster, and rabies viruses utilize this peripheral nerve pathway

Aseptic Meningitis

- Common- annual incidence rate of 11 to 27 cases per 100,000 population
- Peaks- August and September
- Enterovirus and Coxsackie virus make up 80% of cases
  - Organisms grow in the intestinal tract, spread by fecal-oral route
  - Family outbreaks are usual and infections are most common among children

Aseptic Meningitis

Pathophysiology:

- Enters host ⇨ grows in extraneural tissues (fat, muscle, lymph nodes, resp. or GI tracts) ⇨ viremia - RBC absorbs, grows in WBC, decreased RES clearance ⇨ crosses blood-brain barrier ⇨ grows in choroid plexus ⇨ passes through small vessels to brain (transported by infected leukocytes) ⇨infects vascular endothelium ⇨diffuses across normal cells and membrane ⇨passes through areas of permeability

Aseptic Meningitis Clinical Features

Acute onset

Mild compared to bacterial meningitis

Generalized throbbing HA

Mild photophobia

Mild neck pain, stiffness fever, and malaise

Lethargy, irritability and drowsiness

CSF: pleocytosis (lymphocyte predominant) with normal CSF glucose

Encephalitis

Definition: Acute febrile illness, usually of viral origin, with nervous system involvement

Etiology:

1. The most common forms caused by mosquito-borne viruses and Herpes Simplex type I
2. Can occur as a complication of systemic viral diseases such as:

   poliomyelitis, rabies, mononucleosis, rubella, or rubeola

Encephalitis

- Incidence: 20,000 cases of acute viral encephalitis are reported annually in the U.S. (CDC, 2001)
- Mortality: Death occurs in 5 – 20% of these patients

Encephalitis

- Complications: Residual signs of mental deterioration, amnesic defect, personality change, recurrent seizures, and hemiparesis are seen in another 20% among all types of viral infections
- Herpes simplex encephalitis – 50% of patients are severely impaired or die

Encephalitis Pathophysiology

Meningeal involvement ⇨ widespread nerve cell degeneration due to edema, necrosis and/or hemorrhage ⇨ increases intracranial pressure ⇨ herniation ⇨ large degenerative injuries

Encephalitis

Clinical features:

Range from mild infectious disease to severe life-threatening disorder

- Difficult to differentiate from meningitis
- Generally, acute febrile illness, evidence of meningeal involvement (HA, stiff neck, photophobia) and various combinations of CNS symptoms

Encephalitis

CNS Symptoms:

- Convulsions, delirium, confusion, stupor or coma
- Aphasia or mutism
- Hemiparesis
- Involuntary movements, ataxia
- Nystagmus, ocular palsies, facial weakness

Encephalitis

- Diagnosis is made by history and clinical presentation
  - CSF: slightly elevated protein
  - WBC: elevated
  - CT, or MRI: often normal, can show diffuse edema, degeneration

Rabies

Differs from other acute viral infections because of the latent period following virus inoculation, and its distinctive clinical and pathological features.

Rabies

- Epidemiology:
  - Human examples are rare
  - Between 1980 and 1997 only 34 cases
  - Usually fatal once symptoms appear
  - Annually, 20,000 – 30,000 individuals are treated with rabies vaccine

Rabies

- Etiology:
  - All cases are a result of transdermal viral inoculation by an animal bite (raccoon, bat, squirrel, domestic animal)
  - Rabid animals may bite without provocation
  - Virus spreads along peripheral nerves to reach the nervous system

Rabies Pathophysiology

- Distinguished by presence of cytoplasmic eosinophilic inclusions, negri bodies, most prominently in hippocampus and Purkinje cells
  - Inflammatory necrosis is widespread, but most intense in the brainstem

Rabies Clinical Manifestations

Must be reported to public health!

- Incubation-usually 20 to 60 days (may be as short as 14 days)
- Tingling or numbness at bite site even after healing, may reflect an inflammatory response incited when the virus reaches the sensory ganglion

Rabies Manifestations

- Prodromal period – fever, HA, malaise
- 2-4 days later – severe apprehension, dysarthria, and psychomotor activity, followed by dysphagia (hence salivation "frothing at the mouth")
- Throat muscle spasms induced by attempts to swallow water, dysarthria, numbness of face, and spasms of facial muscles

Rabies Manifestations

- Generalized seizure, confusional psychosis, agitation may follow
- ↑ ICP leading to coma, ↑ release of ADH, diabetes insipidus, extremes of autonomic dysfunction especially hyper- and hypotension
- Death ensues within 4-10 days, or longer
- With modern intensive care techniques, there have been survivors!