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## **SYNOPSIS:**

- Definition
- Epidemiology
- Etiology
- Types of Encephalitis
- Pathophysiology
- Risk factors
- Clinical Manifestation
- Diagnosis
- Differential Diagnosis

Medical Management

# DEFINITION

Encephalitis is defined as **an inflammation of the brain parenchyma** which is associated with the neurological dysfunction.

The infectious or inflammatory disorder of the brain manifested by fever and headache and associated with a depressed level of consciousness, an altered mental status (confusion, behavioural abnormalities), focal neurologic deficits, or new onset seizure activity.

# EPIDEMIOLOGY

- Overall, viruses are the most common cause of encephalitis.
- Epidemiologic studies estimate the incidence of viral encephalitis at 3.5 7.4 per 100,000 persons per year.
- Children and young adults are typically the groups that are most often affected. However, severity is usually more pronounced in infants and elderly patients.
- Encephalitis affects men more often than women.



The exact causes of the encephalitis is unknown. But it is most commonly occurs due to the viral infection.

Bacterial infection and non-infectious inflammatory agents also cause to produce encephalitis.

### Viral Cause:

- Herpes simplex virus (HSV)
- Varicella-zoster virus (VZV)
- Influenza virus
- Enteroviruses

- Rabies virus
- Epstein-Barr virus (EBV)
- Arthropod-borne viruses (arboviruses spread by mosquitoes, ticks and other insects)
- Flavivirus (Japanese encephalitis virus)
- Childhood virus including mumps and measles.

Bacterial Cause: H. Influenza, N. Meningitidis, M. Tuberculosis, Treponema pallidium, Mycoplasm,

Pneumococcus.

Other microorganism – Protozoa, fungus, aspergillus, cryptococcus, histoplasma, rickettsia.

Non Infective: Autoimmune encephalitis

# TYPES OF ENCEPHALITIS

There are two types of encephalitis:

- 1. Primary Encephalitis
- 2. Secondary Encephalitis

### **\* PRIMARY ENCEPHALITIS:**

This occurs mainly due to the entry of virus or other causative organism which may directly infects the brain parenchyma or nerve cells of the brain. The infection may spread to the areas of brain.

#### **\* SECONDARY ENCEPHALITIS:**

This occurs as a result of failure of the immune system reaction to the infective agents.

Instead of acting against to the infectious agent, the immune system attacks the healthy cells of the brain. This types of encephalitis may occurs two to three weeks after the initial infection.

# PATHOPHYSIOLOGY

Entry of virus into the blood

The virus may affects the neuronal cells and reaches the brain parenchyma, cerebral cortex, grey & white matters of brain, basal ganglia and brainstem.

It can leads to disruption in nerve cell functioning and damage to the neurons & glial cells.

It can cause to produce vascular congestion, hemorrhage or thrombus formation in small arteries of brain, inflammatory responses that may affects the grey and white matter of the brain.

Diffuse proliferation of glial cell and neurons of brain may leads to increased intracranial pressure.

## Increased Intracranial pressure leads to cerebral oedema

and vascular damage.

#### Transtentorial herniation of brain strucrure.

# **RISK FACTORS**

Anyone can develop encephalitis but the factors that may increase the risks of encephalitis is as follows:

- Age: Some types of encephalitis are more common in certain age groups. In general, young children and older adults are greater risk of most types of viral encephalitis.
- Weakened immune system: People will HIV/AIDS, intake of immunosuppression drugs, other diseases affecting immune system may predispose to encephalitis.

- **Geographical region:** Mosquitos and tick-borne viruses are common in slum areas. The peoples living in this particular geographical area may in risk of the infection.
- Season of the year: Mosquitos and tick-borne are most commonly seen in spring and rainy season.

# CLINICAL MANIFESTATIONS

Signs and symptoms may develops within a hours of weeks after the enter of virus into the blood streams. The clinical manifestation of the encephalitis are as follows:

Classic symptoms including fever, headache, disorientation, loss of consciousness, seizures and personality changes. Increased ICP may leads to nausea, vomiting and some times alteration in the conscious level.

Motor weakness like hemiparesis (depends upon the area of the lesion)

Exaggerated DTR, Extensor plantar response.

Tremor

Neurological symptoms including visual field defect, aphasia, dysphagia, ataxia and paresthesia. Brainstem lesion may present with Nystagmus, difficulty in ocular movement, hearing loss, dysarthria, loss of facial expression.

□ Limbic system involvement may leads to mood and personality change that progress to severe memory loss and delirium.





- Brain edema in the orbitofrontal and temporal areas, which HSV typically infects.
- Demyelination in progressive multifocal leukoencephalopathy
- Basal ganglia and thalamic abnormalities

### & Lumbar Puncture:

o Lymphocytic pleocytosis,

o Normal glucose,

o Mildly elevated protein,

o Absence of pathogens

**\* EEG:** 

• Sharp wave in temporal lobs

## DIFFERENTIAL DIAGNOSIS

The condition should be differentiated from the following disease:

- Meningitis
- Brain abscess
- SLE
- Status Epilepticus

# MEDICAL MANAGEMENT

The goals of pharmacotherapy are to reduce morbidity and prevent complications.

Antivirals are used to manage treatable viral encephalitides. Corticosteroids may be considered for postinfectious or noninfectious encephalitis.

- Anticonvulsants:
  - Benzodiazepines (General & localised seixure)
- Antiviral Drugs:
  - Acyclovir (Inhibitory activity against HSV)
  - Foscarnet
- Corticosteroids:
  - Dexamethasone (Reduce Inflammation)
- Diuretics:
  - Furosemide (Decrease ICP)
  - Mannitol (Decrease ICP)