Encephalitis

Tuesday, November 4, 2025 6:47 PM

Definition

Encephalitis is an infection and inflammation of the brain parenchyma, usually caused by viruses.

When both brain and meninges are involved, the condition is termed meningoencephalitis.

Common Causative Viruses & Predisposing Factors

Predisposing Factor	Common Viruses	Key Comments
Neonate	*Herpes simplex virus type 2 (HSV-2)*	Acquired during birth from infected mother.
Child (>1 year) & Adult	*Herpes simplex virus type 1 (HSV-1)*	Affects temporal lobe; virus reactivates from trigeminal ganglion.
Animal bite (dog, cat, bat, skunk, raccoon)	*Rabies virus*	In the U.S., bats are the most common reservoir 🦇.

Mosquito bite	*West Nile virus (WNV), Eastern & Western equine encephalitis viruses, St. Louis encephalitis virus*	WNV is the most common arboviral cause of encephalitis in the U.S.
Immuno compromised host	*Cytomegalovirus (CMV)*	Seen in AIDS or transplant patients.
Post-infectious / Post-vaccination	*Various viruses* (e.g., Measles, VZV)	Immune-mediated post-infectious encephalomyelitis.

⚙ Pathophysiology

1. Routes of Infection

- HSV-2 (Neonates): Acquired at birth → enters bloodstream → hematogenous spread to brain
- HSV-1 (Adults): Reactivation from trigeminal ganglion → travels along sensory neurons → infects temporal lobe
- Rabies virus: Inoculated at bite site → enters peripheral nerves → travels retrograde via axons → CNS infection

- Arboviruses (e.g., WNV): Acquired via mosquito bite → bloodstream → brain parenchyma

- Varicella-Zoster Virus (VZV):
  ↳ *Primary infection* (Chickenpox) → can cause encephalitis
  ↳ *Reactivation* (Shingles) → can cause encephalitis or postinfectious encephalomyelitis

✿ *Encephalitis lesions are inflammatory (contain WBCs, especially lymphocytes), whereas encephalopathy lesions show degenerative changes without inflammation.*

---

2. Special Viral Patterns

Virus	Mechanism / Spread	Remarks
HSV-2	Bloodstream (neonates)	Severe neonatal disease; high mortality without treatment
HSV-1	Axonal spread from trigeminal ganglion	Causes necrotizing temporal lobe lesions
Rabies	Retrograde axonal Transport	Long incubation; almost always fatal once symptoms appear ⚠
Arboviruses (WNV, EEE, WEE, SLE)	Hematogenous spread via mosquitoes	Seasonal (summer–early fall) 🌞

VZV	Direct or immune-mediated	Can cause postinfectious demyelination
CMV	Hematogenous; opportunistic	Seen in AIDS/transplant patients
EBV	Rare complication of mononucleosis	Mild self-limited encephalitis
Postinfectious encephalitis	Immune-mediated demyelination	Occurs weeks after infection or vaccination

---

Post-Infectious Encephalitis

- Occurs 1-3 weeks after a viral infection or immunization.
- Immune-mediated demyelination of neurons in white matter (not direct viral damage).
- Common after measles, varicella, or vaccination.
- Presents with ataxia, altered sensorium, seizures, and rapid deterioration.

Distinction: Encephalitis vs Encephalopathy

Feature	Encephalitis	Encephalopathy
Definition	Inflammation of brain parenchyma	Non-inflammatory brain dysfunction
Cause	Usually viral infection	Metabolic, toxic, or hypoxic
CSF Findings	Lymphocytic pleocytosis	Normal
Histology	Inflammatory infiltrate (lymphocytes)	Neuronal degeneration, no WBCs
Example	HSV encephalitis	Hepatic or hypoxic encephalopathy

🩺 Clinical Manifestations

1. General Symptoms

- Fever 🌡️
- Headache 🤕
- Altered mental status / confusion
- Seizures ⚡
- Focal neurological deficits (e.g., aphasia, hemiparesis)

🧠 *These reflect inflammation and neuronal dysfunction within the brain parenchyma.*

---

2. Rabies Encephalitis — Two Clinical Forms

Form	Clinical Features	Frequency
Furious Rabies	Hyperactivity, agitation, delirium, hydrophobia, seizures	~80% of cases
Dumb (Paralytic) Rabies	Ascending flaccid paralysis without agitation	~20% of cases

Both forms progress to:

→ Coma → Respiratory failure → Death ☠️

🧭 Summary — Mechanisms of Viral Spread to CNS

Route	Examples	Description
Hematogenous	HSV-2 (neonates), CMV, Arboviruses	Virus enters bloodstream → crosses BBB

Neuronal (Axonal transport)	HSV-1, Rabies	Virus travels along peripheral nerves to brain
Immune-mediated (Postinfectious)	VZV, Measles	Immune attack on myelin, not direct infection

---

Pathogens (Causative Agents)

→ Main Cause:

Encephalitis is *predominantly viral* in origin. However, in about half the cases, the exact cause remains unknown.

→ Major Causative Viruses:

Virus	% of Cases / Frequency	Key Features	Remarks
HSV-1	~15%	Most common treatable cause of encephalitis	Affects temporal lobe; responds to acyclovir
HSV-2	Rare in adults; seen in neonates	Acquired during birth	Also treatable with acyclovir
Arboviruses (esp. WNV)	~5%	Mosquito-borne; summer/fall season	Most common arboviral cause in USA
Rabies virus	Rare in USA	From animal bites	Common in countries with

		(bats, raccoons, dogs)	poor dog vaccination
VZV, CMV, EBV	Variable	Often in immunocompromised individuals	Can cause primary or post-infectious encephalitis

West Nile Virus (WNV)

- 80% → Asymptomatic
- 20% → Mild *flu-like illness* 🤒
- <1% → Develop CNS disease
  ↳ 50% of these have encephalitis
- Transmitted by *Culex* or *Aedes* mosquitoes

Other Arboviruses Causing Encephalitis:

- St. Louis encephalitis virus
- La Crosse (California) virus
- Eastern equine (EEE) and Western equine (WEE) viruses

Postinfectious (Immune-mediated) Encephalitis:

Occurs after infection or vaccination due to autoimmune demyelination.

- Common triggers: Measles, Influenza, VZV

🔬 Diagnosis of Encephalitis

🧫 CSF Findings

Parameter	Typical Finding in Encephalitis
WBC count	Mild lymphocytic elevation
Protein	Slightly elevated
Glucose	Usually normal
Special note	CSF may even appear normal in some cases! 😮

🔍 Flowchart: Diagnostic Approach to Encephalitis

→ Suspected Encephalitis

↳ Perform Lumbar Puncture (CSF analysis) → Lymphocytic predominance + ↑ protein + normal

glucose

↳ PCR test of CSF → HSV-1 / HSV-2 / VZV confirmation

↳ WNV IgM in CSF → confirms West Nile encephalitis

↳ Rabies testing:

→ Direct fluorescent antibody (skin biopsy, nape of neck)
→ PCR from CSF / saliva / tissue
↳ *Can identify viral strain & geographic origin*

↳ MRI/CT findings:

→ Temporal lobe abnormalities → strongly suggest HSV-1

Treatment

Etiologic Agent	Treatment	Notes
HSV-1, HSV-2, VZV	IV Acyclovir	Early initiation greatly improves prognosis ⚡
Arboviruses (WNV, etc.)	*No specific antiviral*	Supportive care only
Rabies virus	*No treatment once symptoms start*	Prevention is key 🔑

Prevention

🐕 Rabies

1. Pre-exposure prophylaxis:

- Killed rabies vaccine for veterinarians, animal handlers, laboratory staff.

2. Post-exposure prophylaxis:

- Killed rabies vaccine + Rabies immunoglobulin (RIG)
  ↳ *Given at separate sites* to avoid neutralization.

💮 *Example of passive-active immunization.*

👶 HSV-2 in Neonates

- Pregnant women with active genital HSV infection:

  → Acyclovir therapy
  → Consider cesarean section to prevent neonatal transmission.

WNV and Arboviruses

- No vaccine available yet
- Prevent mosquito bites (repellents, nets, drainage of stagnant water).

Summary Table: Overview of Viral Encephalitis

Virus	Transmission	Key Diagnostic Clue	Treatment	Prevention
HSV-1	Reactivation from trigeminal ganglion	Temporal lobe lesions (MRI)	Acyclovir	None
HSV-2	Birth canal (neonates)	Neonatal seizures, CSF PCR+	Acyclovir	Cesarean + Acyclovir
WNV	Mosquito bite	WNV IgM in CSF	Supportive	Mosquito control
Rabies	Animal bite	DFA of neck skin biopsy	None (after onset)	Vaccine + RIG
VZV	Reactivation or primary infection	PCR in CSF	Acyclovir	None
Post infectious	After measles, influenza, VZV	Autoimmune demyelination	Supportive	—