

Viral CNS Infections

Bug	Virulence	Etiology/Pathogenesis	Clinical ID
HERPESVIRUSES			
- HSV Encephalitis		Most common fatal enceph in ADULTS Temporal lobe → permanent brain damage or death	Treatment: Acyclovir
- Herpes B Virus		OLD WORLD MONKEYS → death frequently Transmission: Saliva/bite	Treatment: Acyclovir
- Varicella Zoster Virus - CNS complications rare		- Transient cerebellar ataxia - Cerebral encephalitis - Aseptic meningitis - Transverse myelitis (paralysis) Congenital VZV → mental retardation	
- Congenital Cytomegalovirus	Transmitted to fetus when pregnant mom gets 1° VZV infection	1-2% born w/ CMV (usually asymptomatic) - Cytomegalic Inclusion Disease (10%) - Inclusion bodies of viral proteins → Deafness, MR, seizures, blind, death - Hearing deficits, ↓ intellect in others	
HUMAN POLYOMAVIRUSES			
	Nonenveloped, icosahedral dsDNA		
- BK Virus		Mild URT infection	
- JC Virus		IMMUNOSUPPRESSED 1° infection is asymp./Reactivation → dis. Progressive Multifocal Leukoencephalopathy → Repr. in oligodendrocytes → loss of myelin in cerebrum white matter only → progressive higher function loss → rapid progression- death 2-12 mo. - No inflammation (due to IC)	Dx by progressive neuro. loss in IC MRI/CT for lesions in subcortical and deep white matter PCR Brain biopsy

TRANSMISSIBLE SPONGIFORM ENCEPHALOPATHIES	Prion – infectious protein - Protein and lipid → normal PrP protein (α helices) to become abnormal (β sheets) and infectious	No inflammation or immune response (endogenous protein) → Vacuolar lesions in brain → astrocytosis (recruited b/c of lesions) → amyloid plaques PrP gene - Mutations → familial TSE's	
- Creutzfeldt-Jacob Disease (CJD)		Classic - onset 50-65 yrs. New Variant - younger onset (15-55 yrs) → Higher cortical defects (memory, judgement..) → Myoclonic movements (jerky) Rapid progression once symptomatic Incubation: years	NV-CJD- like BSE (transmission from cattle)
- Bovine Spongiform Encephalitis		Origin from Scrapie (sheep) - Infected sheep parts in animal-based cattle feed → cross “species barrier” OR Origin from spontaneous PrP mutation in cows	
- Gerstmann-Straussler Sheinker Syndrome (rare)		Spinocerebellar ataxia Dementia Plaque-like deposits Familial - PrP-c gene mutation	
- Kuru		Fore Tribe- Papua New Guinea Spread by ritual cannibalism Incubation- 20-30 yrs.	