

## Encephalitis

| Encephalitis   | Meningitis   |
|--|--|
| inflammation of the brain parenchyma that arises from penetration of the blood–brain barrier or overlying meninges.<br><br><b>Note:</b> viruses are the most common causative agents | inflammatory response is limited largely to the meninges<br><br>more common<br><b>note:</b> meninges appear to play a protective role in limiting pathogen spread to the CNS |

### Pathways for pathogen entry to the CNS:

- 1-Invasion of Sensory and motor Nerve Endings.(by infecting muscles and skin)
- 2-Infection of Brain Microvascular Endothelium(hematogenous)
- 3-Invasion by Infected Circulating Leukocytes

**Note:** Herpes simplex virus (HSV) is one of the most common pathogens that cause encephalitis. While encephalitis due to HSV infection is relatively rare, it can occur, especially in individuals with weakened immune systems or certain risk factors.

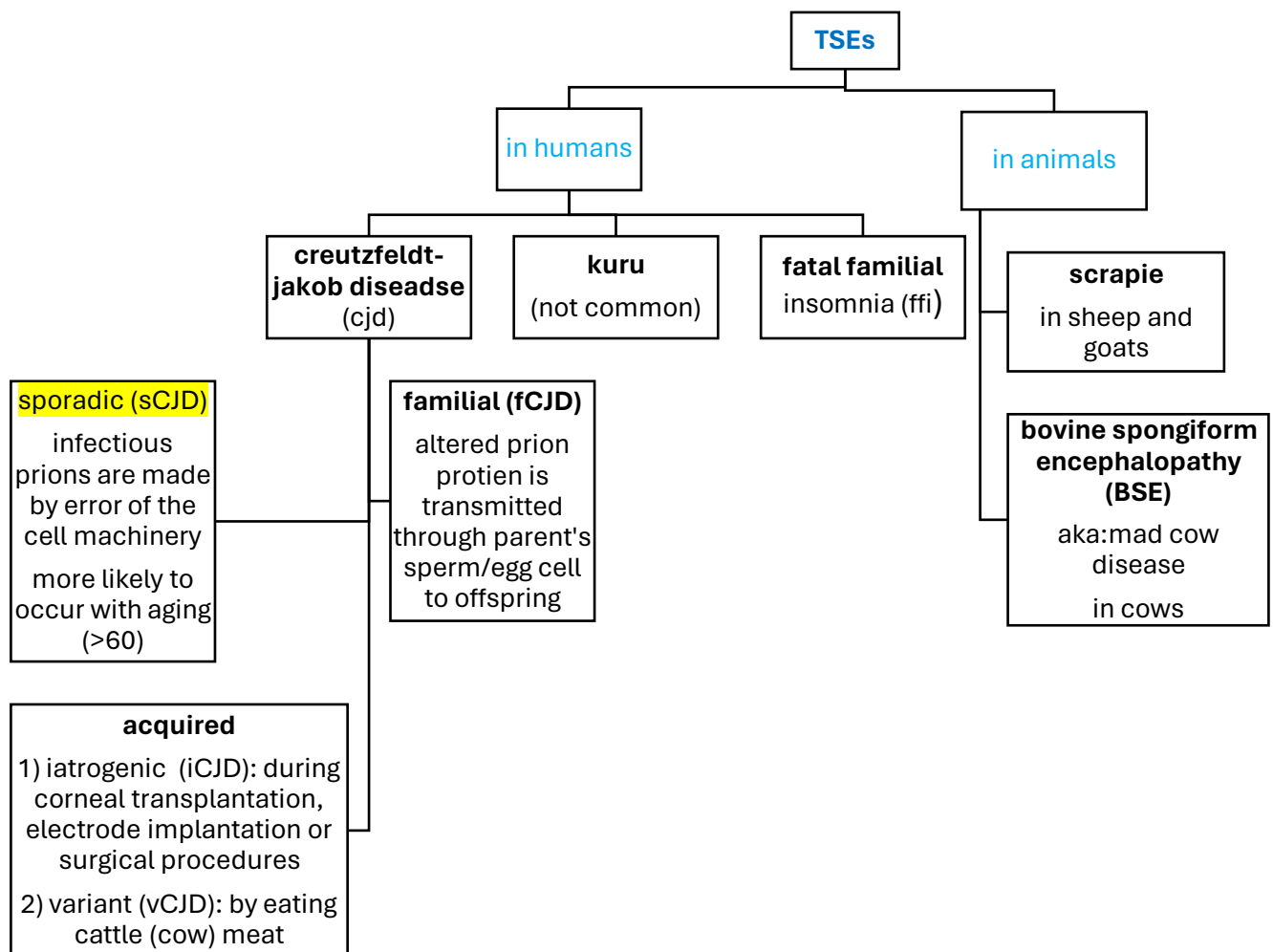
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|--|---|--|
| <p><b>Symptoms</b></p> <ol style="list-style-type: none"> <li>1-Fever &amp; headache</li> <li>2-symptoms of meningitis (if present)</li> <li>3- altered level of consciousness (hallucinations, agitation, personality change, behavioural abnormalities)</li> <li>4- Depressed level of consciousness ranging from mild lethargy to coma</li> <li>5- either focal or diffuse neurologic signs and symptoms (aphasia, ataxia, upper or lower motor neuron patterns o weakness).</li> <li>6- Focal or generalized seizures</li> </ol> | <p><b>Rabies as a cause</b></p> <ol style="list-style-type: none"> <li>1-zoonotic infection, transmitted by bites.</li> <li>2-Incubation period ranges from days to less than a year</li> <li>3- In addition to encephalitis symptoms, episodes of hyperexcitability are typically followed by periods of complete lucidity that become shorter as the disease progresses, with time causes brain dysfunction that causes death in (0-14 days).</li> <li>4- treated by post exposure prophylaxis (wound care) and passive immunization with rabies immune globulin</li> </ol> | <p><b>Diagnosis</b></p> <ol style="list-style-type: none"> <li><b>1-lumbar puncture:</b> csf consists of lymphocytic pleocytosis, a mildly elevated protein concentration, and a normal glucose concentration</li> <li><b>2- CSF PCR:</b> for viral CNS infections.</li> <li><b>3- Serology:</b> Anti WNV IgM antibodies in the CSF for WNV encephalitis.</li> <li><b>4- Neuroimaging:</b> helps identify or exclude alternative diagnoses</li> <li><b>5-brain biopsy</b> (last option)</li> </ol> |
| <p><b>Pathogenesis</b></p> <ol style="list-style-type: none"> <li>1-synchronous neutrophil extravasation during VSV encephalitis</li> <li>2-microglial activation by CNS viral infection</li> </ol>  | <p><b>Causes</b></p> <ol style="list-style-type: none"> <li>1-Mainly unknown causes</li> <li>2-m/c virus for sporadic cases= herpes virus</li> <li>3-epidemics by arbovirus</li> <li>4-rabies virus</li> </ol>  | <p><b>Management</b></p> <ol style="list-style-type: none"> <li>1-ICU: careful monitoring of vital signs and ICP.</li> <li>2- Acyclovir is of benefit in the treatment of HSV</li> </ol>   |

**Note:** Many patients with WNV infection have sequelae, including cognitive impairment; weakness; and hyper- or hypokinetic movement disorders, including tremor, myoclonus, and parkinsonism.

## transmissible spongiform encephalopathies (TSEs).

are a group of diseases that affect the brain and nervous system of humans and animals. The diseases are characterized by a degeneration of cerebral cortex & cerebellum tissue giving it a sponge-like appearance

**caused by prions:** pathogenic agents that are transmissible and induce abnormal folding of specific cellular proteins called prion proteins (PrP) that are in the brain. Prions composed of the prion protein (PrP) are hypothesized as the cause of transmissible spongiform encephalopathies

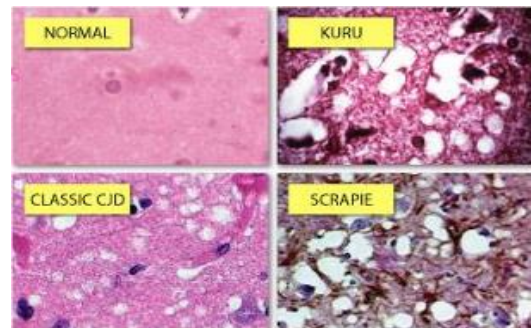


## Signs and symptoms of CJD

- 1) Rapidly progressive dementia (confusion, disorientation, and problems with memory, thinking, planning and judgment).
- 2) Rigidity.
- 3) Agitation, apathy and mood swings.
- 4) Myoclonus. brief, involuntary twitching of a muscle caused by sudden muscle contractions (positive myoclonus) OR brief lapses of contraction (negative myoclonus) (persists during sleep)
- 5) In late stages physical manifestations such as: Ataxia. speech impairment & changes in gait

## Diagnosis of CJD:

- 1) Electroencephalography (EEG): not for all types
- 2) Magnetic resonance imaging (MRI): more accurate
- 3) to **confirm** a diagnosis of CJD is by brain biopsy or autopsy done by a neuropathologist



## Treatment of CJD:

- There is no known cure & patient survival median is short
- Palliative care can be helpful
- Opiate drugs can help relieve pain if it occurs, and the drugs clonazepam and sodium valproate may help relieve myoclonus

## infectious myelopathies (myelitis)

when spinal is involved in infections ,Clinical manifestations depend on the exact level and location within the cord.

### Most common causes:

- 1) herpesviruses
- 2) enteroviruses

**notes:** 1) Pyogenic epidural abscess, a cause of extrinsic cord compression, requires immediate recognition, because permanent neurologic deficits may develop within 36 hours of symptom onset

- 2) Treponema pallidum, the causative agent of syphilis, is a rare etiology of myelopathy