VIRAL INFECTIONS OF THE NERVOUS SYSTEM

HISTOLOGICAL FEATURES OF NONPURULENT ENSEPHALITIS

- Viral infections of the CNS typically induce **nonsuppurative inflammation**

Vacsular changes

✓ Perivascular cuffing (*Infiltrating cells are predominantly lymphocytes*)
✓ Hyalinizing or fibrinoid change of the vessel wall
✓ Endothelial swelling and proliferation in small blood vessels
✓ Pressure created by cell infiltration

✓ **Glial reactions: D**iffuse or focal gliosis
✓ **Neuronal degeneration and necrosis**
✓ **Demyelination**
✓ **Meningitis**
✓ **Inclusion bodies**

RABIES

- Caused by the species ***Rabies virus*** (RABV), genus ***Lyssavirus***, family ***Rhabdoviridae***.

- Rabies virus has 2 biotypes: “fixed” virus and “street” virus:

✓ Fixed RABV, which is the basis of vaccine strains, is a laboratory biotype, is not secreted in saliva, and does not produce Negri bodies.

✓ Street RABV is the feral biotype, it is tropic for salivary glands and produces Negri bodies

Pathogenesis of Rabies

After a bite wound,
1, the rabies virus initially replicates in muscle (can enter peripheral nerves directly),
2, enters, *3*, and ascends (retrograde axonal transport) the peripheral nerve,
4, to the dorsal root ganglion,
5, enters the spinal cord,
6, and ascends, *7*, to the brain via ascending and descending nerve fiber tracts, infects brain cells, spreads to salivary glands,
8, and the eye and is excreted in saliva.

TYPICAL PATHOLOGICAL FINDINGS

- ✓ Nonsuppurative encephalomyelitis
- ✓ Ganglioneuritis
- ✓ Sialoadenitis (parotid adenitis)

- The **clinical course** of rabies is usually acute, from 1-2 days, but can be as long as 10 days.
- The clinical disease in the dog has been divided into three phases: **prodromal, excitatory, and paralytic**.

Furious rabies = the excitatory phase is predominant,

Dumb rabies =excitatory phase is short or absent and the disease progresses quickly to the paralytic phase.

Specific gross lesions are not present at autopsy,

but self-inflicted wounds and foreign bodies in the stomach of a carnivore should raise suspicion.

Histologic lesions of rabies:

- ✓ Perivascular cuffing (composed solely of lymphocytes)
- ✓ Neuronal degeneration
- ✓ Babès' nodules
- ✓ Focal and diffuse gliosis
- ✓ Negri bodies (intracytoplasmic inclusion bodies present in the hippocampus of carnivores and in the Purkinje cells of herbivores.

Diagnosis of rabies

- Fluorescent antibody labelling
- Immunohistochemistry performed on paraffin embedded formalin-fixed tissues
- In situ hybridization,
- Electron microscopy,
- PCR-based testing

AUJESZKY'S DISEASE

- Pseudorabies/"Mad itch"
- Not zoonotic
- Pseudorabies is not related to rabies but was named because its clinical signs sometimes resemble those seen with rabies.
- *Suid herpesvirus 1* (SuHV-1)
- Causes **encephalitis** primarily in **pigs**; the disease is sporadic in cattle, dogs, cats, sheep rats and minkes.
- Young, suckling piglets—can die from infection,

Mature pigs — remain persistently infected and act as latent carriers.

- **The signs and course** of pseudorabies in pigs are very variable.
- The mortality rate in nursing pigs and young weaners may be very high.
- In slightly older piglets, incoordination progresses rapidly to paralysis with muscular twitchings, tremors, and convulsion.
- The disease in older pigs is often characterized by fever, rhinitis, and coughing.
- Fetal resorption, mummification, stillbirths, and abortions are reported frequently.

- The CNS is free of **gross lesions.**

✓ Gross lesions nonneural tissues, include organs of the respiratory system, lymphoid system, digestive tract, and reproductive tract.

✓ Focal tissue necrosis also occurs in the liver, spleen, and adrenal glands, particularly in young suckling pigs.

✓ Evidence of facial pruritus is a common sequela to infection.

Microscopic lesions in pigs :

- Nonsuppurative meningoencephalomyelitis with
  Trigeminal ganglioneuritis.

- Intranuclear amphophilic inclusion bodies can be present in neurons and astrocytes.

➢ In cattle, sheep, dogs, and cats, the pathogenesis and lesions are comparable to that of pigs.

LOUPING ILL (OVINE ENCEPHALOMYELITIS)

- Tick-borne viral polioencephalomyelitis of sheep caused by species ***Louping ill virus*** (LIV).
- Cattle, horses, goats, deer and human can contract the disease.
- Genus *Flavivirus* of the family *Flaviviridae*.
- *Infection___regional lymph nodes___ viremia____CNS via the hematogenous route.*

Alternatively, the virus may localize indirectly from the blood in nasal structures and enter the brain via the ***olfactory nerves****.*

- *Louping ill is a systemic infection*
- Neurologic signs develop at about day 5 and are characterized by incoordination, tremors, cerebellar ataxia, and terminal paralysis.
- There are no gross lesions
- Histopathological lesions: **Acute polioencephalomyelitis**

Mild leptomeningitis, neuronal degeneration specially in Purkinje cells, cuffing and focal gliosis in the white matter, poliomyelitis affecting particularly the ventral horns, neutrophils very severe cases.

LENTIVIRAL ENCEPHALOMYELITIS OF SHEEP AND GOATS

Genus ***Lentivirus***, family ***Retroviridae***

- Caprine arthritisencephalitis of goats___***Caprine arthritis encephalitis virus*** (CAEV)
- Visna-maedi disease complex of sheep__***Visna-maedi virus*** (VISNA)

In both natural hosts, *4 clinical and pathologic syndromes are recognized:*

- ✓ **Mastitis**
- ✓ **Arthritis**
- ✓ **Interstitial pneumonia** (maedi, or ovine progressive pneumonia)
- ✓ **Encephalomyelitis** (visna of sheep).

VISNA

- Main clinical signs: caudal ataxia and fine trembling of the lips.
- Chronic multifocal *demyelinating leucoencephalomyelitis*.

Caprine arthritis-encephalitis (CAE)

- Arthritis and encephalitis in kids/ synovitis and periarthritis in adults.
- Hindlimb lameness and ataxia with paresis that progresses to paralysis.
- Leucoencephalomyelitis, mastitis, interstitial pneumonia.

CANINE DISTEMPER

- ***Morbillivirus*** (family ***Paramyxoviridae***)
- The infection is systemic, and clinical signs are often referable to the **respiratory, gastrointestinal,** and **nervous systems.**
- Ocular disease, **pustular and/or hyperkeratotic cutaneous lesions, dental defects,** and **abortion** are other manifestations

- Neurologic signs in all affected species include convulsions, myoclonus, tremor, disturbances in voluntary movement, circling, hyperesthesia, paralysis, and blindness.

- Lesions characteristically occur in the cerebellum, rostral medullary velum, optic tracts, spinal cord, and surrounding the fourth ventricle, and probably arises from distribution of virus through the cerebrospinal fluid.

- **Microscopically;**

✓ Demyelination,

✓ Gitter cells,

✓ Meningitis,

✓ Neuronal degeneration,

✓ Inclusion bodies (cytoplasmic, nuclear, or both) particularly in astrocytes, but also in ependymal cells and occasionally in neurons,

✓ Astrocytic hypertrophy,

✓ Microglial proliferation

✓ Endothelial proliferation, Perivascular cuffing, vasculitis

- Coryza Gangrenosa Bovum
- Infectious canine hepatitis (HCC Hepatitis contagiosa canis)
- *Sporadic Bovine Encephalomyelitis*
- Old dog encephalitis
- Necrotizing meningoencephalitis and necrotizing leukoencephalitis of Pugs and other small-breed dogs
- Granulomatous encephalitis
- Postinfectious encephalomyelitis (*perivenous mononuclear cell infiltration, demyelination*)