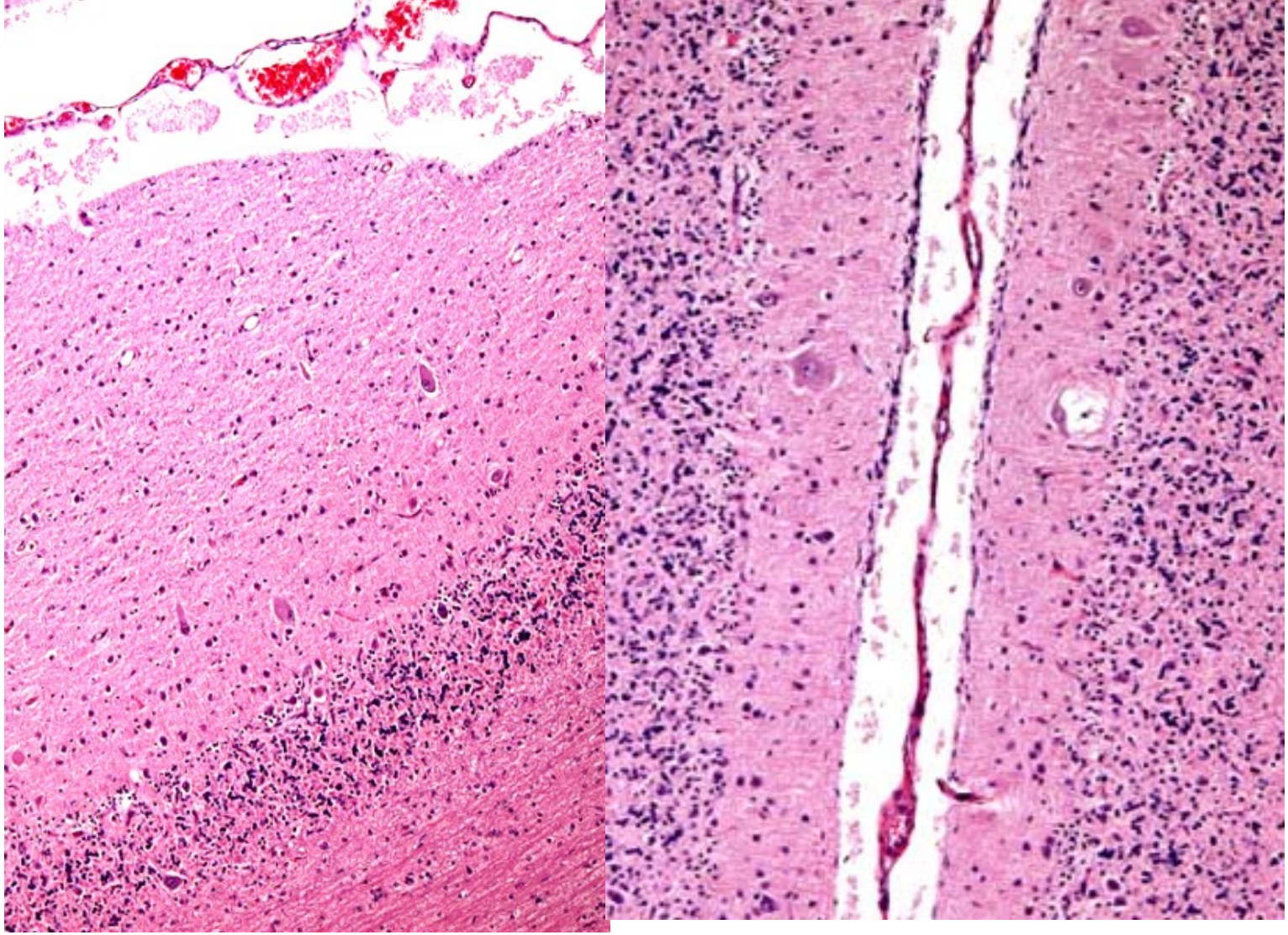
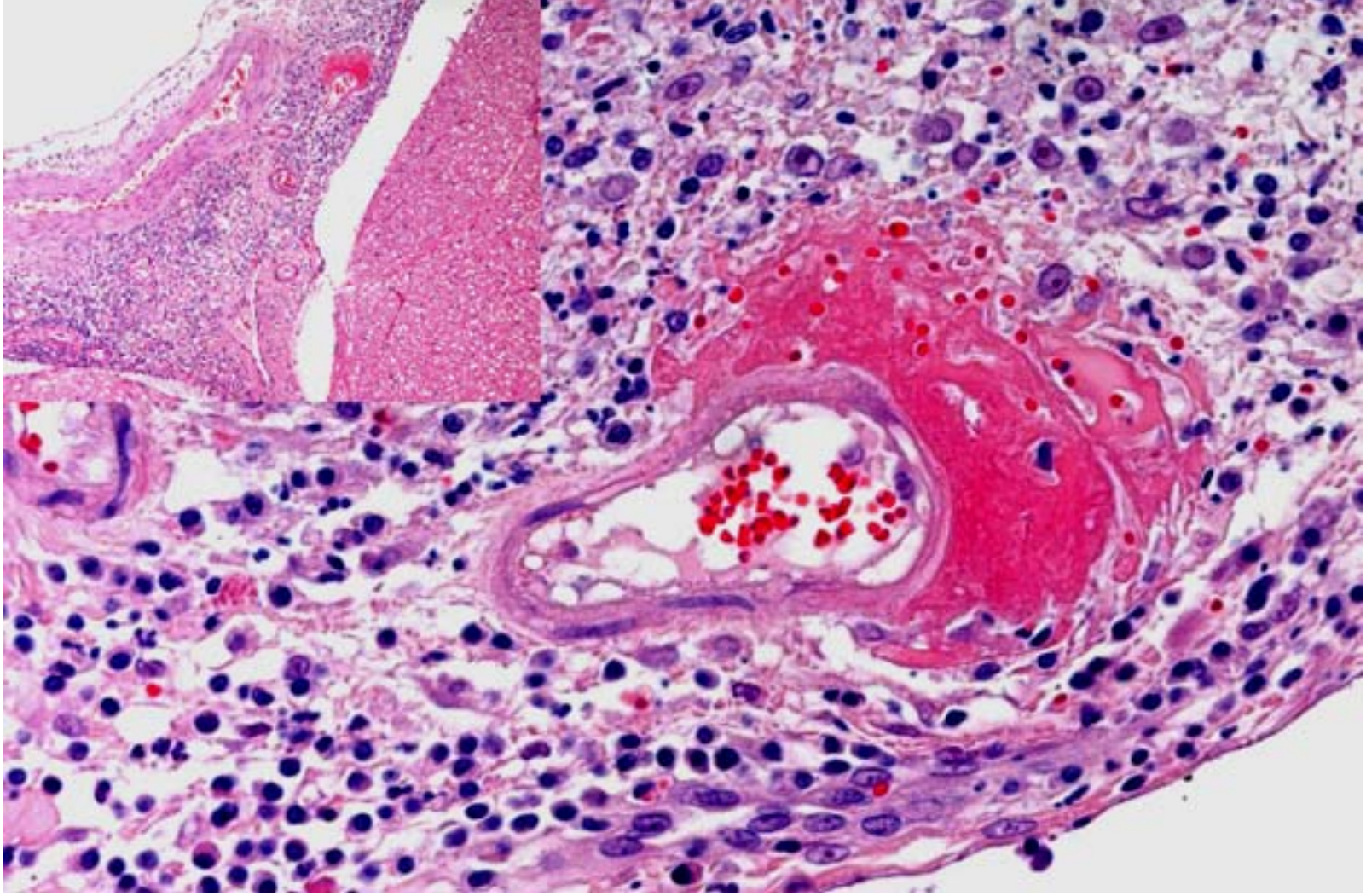


Case 1.1 Representative field from the cerebellar cotex of a young dog
Review – with supplementary image to show extra detail
1) - The two major abnormalities are 1) Paucity of Purkinje and Granule cells 2) Heterotopia of Purkinje cells. An additional feature is mild hypercellularity in the molecular layer.
2) - These features imply damage inflicted, or intrinsic programming failure, during the phase of organogenesis. We see here the end result, a deficit in neuronal numbers and tissue dysplasia.
The dysplastic features would distinguish this type of lesion from degeneration of the cerebellum after complete normal development (cerebellar abiotrophy). Remember the varying degrees of cerebellar development at birth between different species.



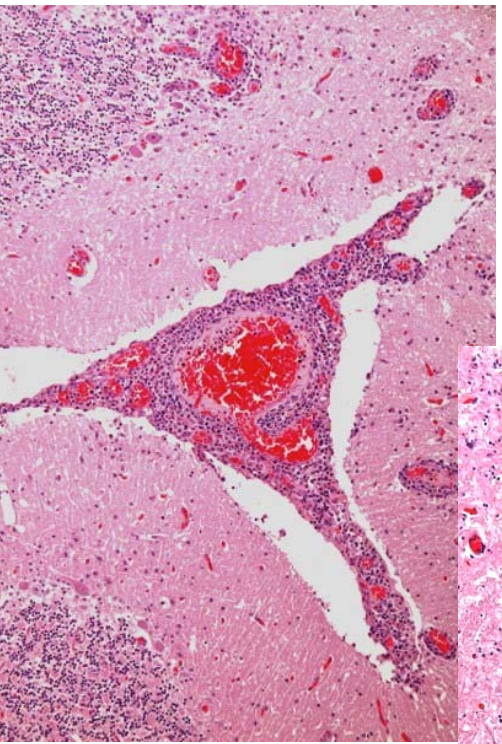
Case 1.2 - Basilar meninges of a cat, caudal brainstem

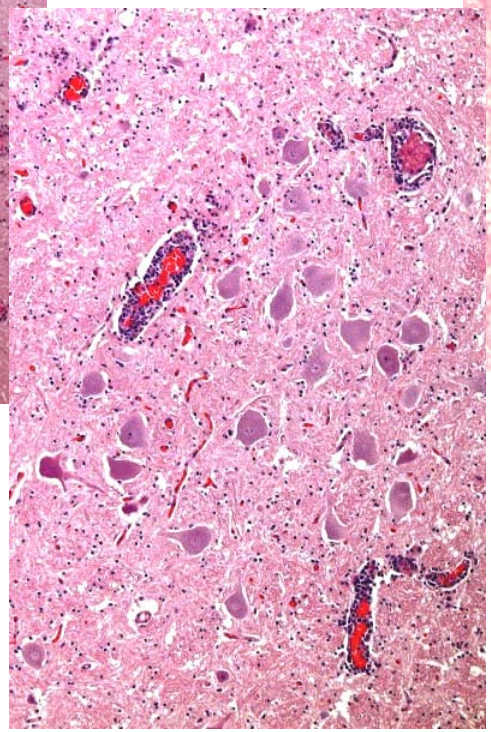
Review-

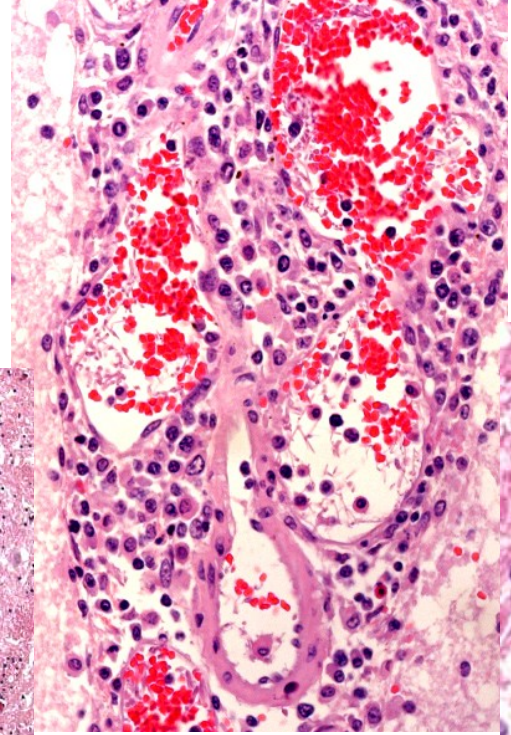
1) Two major processes evident are 1) dense histiocytic, lympho-plasmacytic and neutrophilic meningeal infiltration, and 2) florid protein and red cell leakage from a venule.

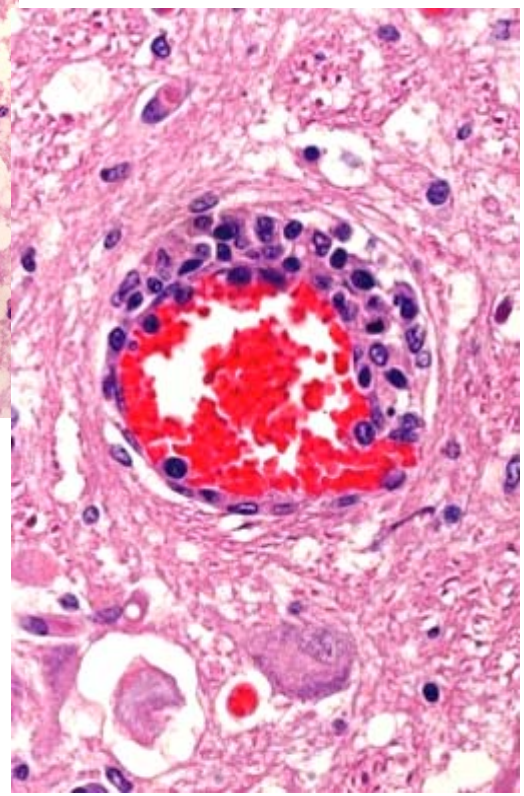
2) An acceptable MDX could be – leptomeningitis, histiocytic, lympho-plasmacytic and neutrophilic, subacute, with vascular protein leakage.

Comment: Such a pattern of meningitis/periventriculitis with vasculitis in a cat suggests FIP, which was the diagnosis in this case. Vasculitis in FIP may or may not be prominent, principally involves venules, but may involve arterioles. In this case there is more a manifestation of vascular injury rather than overt vasculitis. Aetiology could be confirmed by ICH for coronavirus antigen, which would be seen within the cyoplasm of lesional macrophages.









Case 1.3 - Samples from a pig
Review.

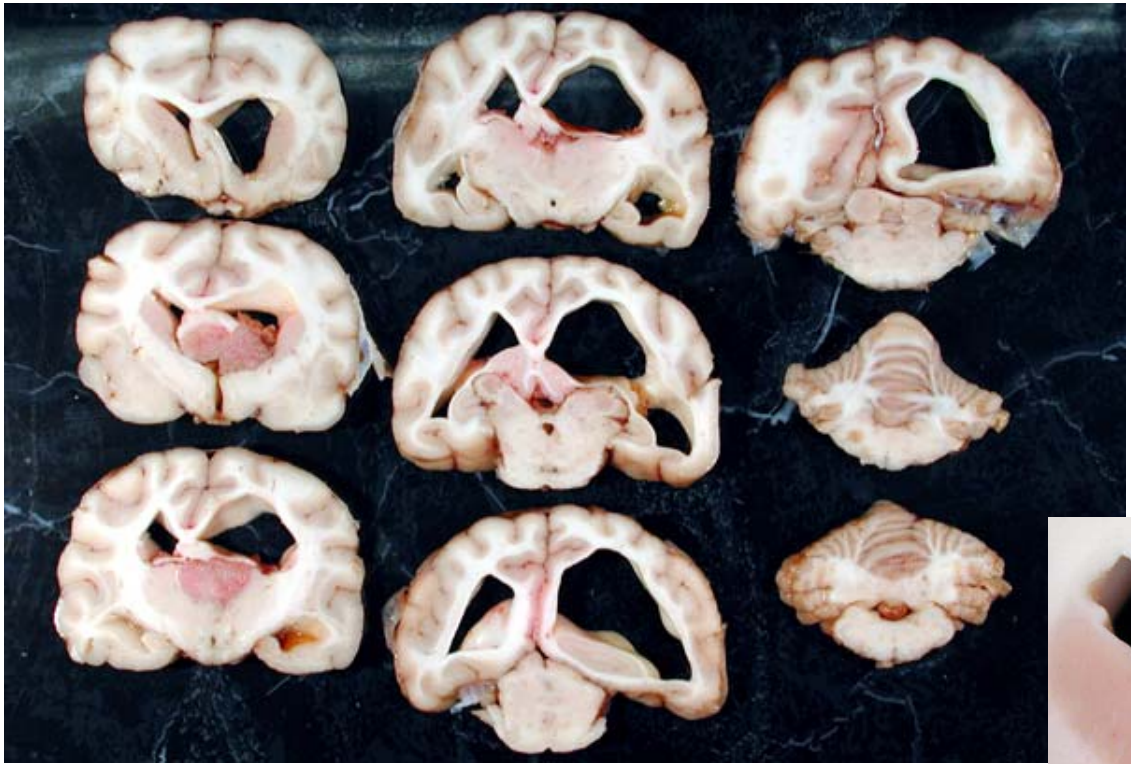
1) The major process evident is an angiocentric infiltrate of mixed mononuclear cells in the leptomeninges and brain. There is little tendency for these cells to extend into the neuroparenchyma. Degenerative changes and reactive gliosis in neuroparenchyma are MINIMAL and focal.

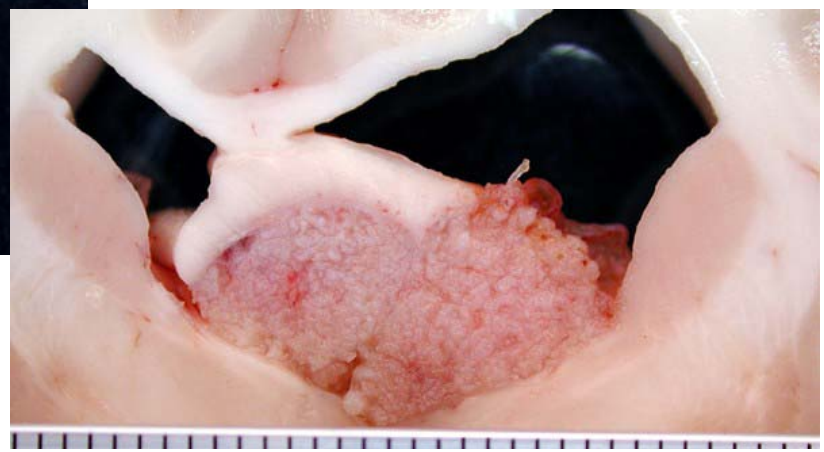
2) An acceptable MDX could be – leptomeningitis and encephalitis, perivascular, lymphoplasmacytic and histiocytic, subacute

3) Aetiologies to consider are – Classical Swine Fever, African Swine Fever, and Malignant Catarrhal Fever (the diagnosis in this case).

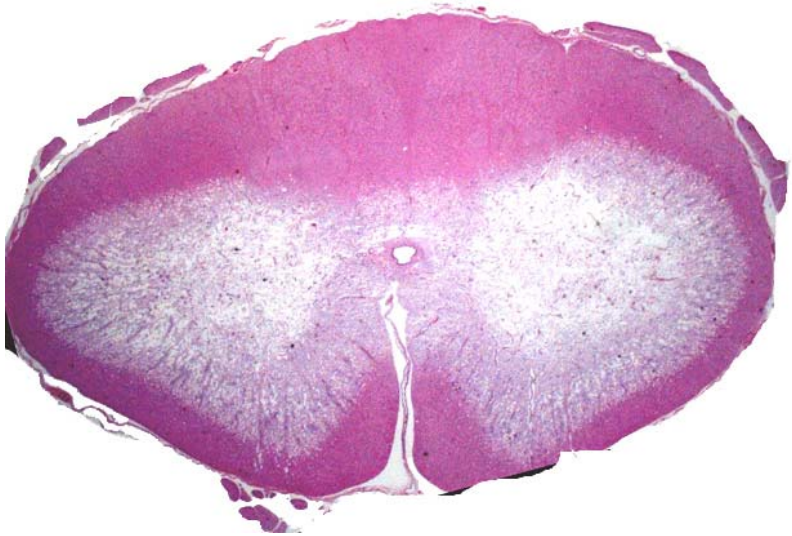
Comment: MCF associated with OHV-2 has recently emerged as an entity in pigs. Aids to diagnosis include lesions in other organs and PCR. As in cattle, overt vasculitis is NOT a regular feature in the CNS.

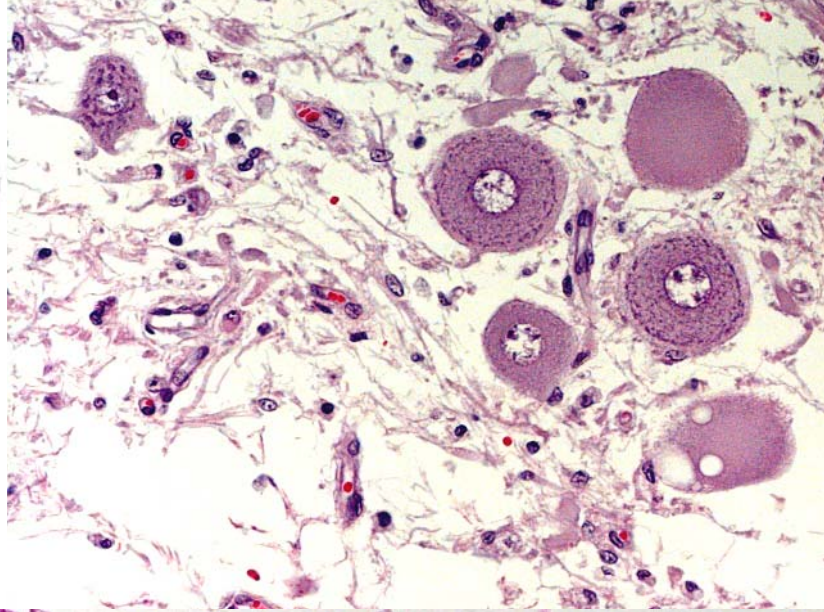
Inflammatory infiltrates in pigs often contain some EOSINOPHILS non-specifically

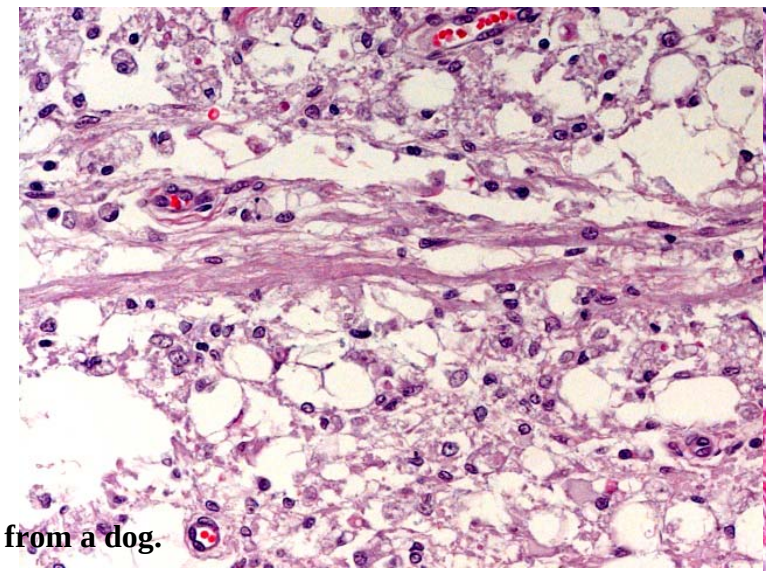


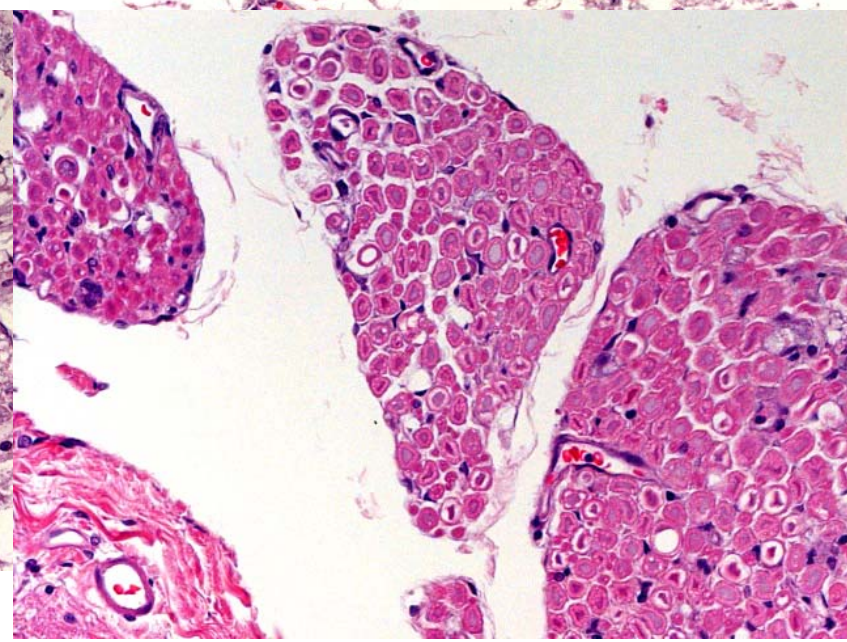


Case 1.4- brain of a cat.
Review.
1. Four major abnormalities include
- A granular, midline, pink/gray mass which projects into the lateral ventricles beneath the fornix and also compresses/?invades the thalamus
- Pronounced assymetric dilation of the lateral ventricles and dilation of the third ventricle
- Associated cerebral swelling and midline shift.
- Pronounced distortion of the midbrain
2. DDx for the primary lesion – Ependymal or Choriod plexus neoplasm, probably malignant.
3. Secondary lesions
- Hydrocephalus due to obstruction of CSF flow into the Aquecuct of Sylvius
- Dorsal compression of the midbrain due to cerebral swelling
The Dx in this case was CHOROID PLEXUS CARCINOMA









Case 1.5 - Spinal cord from a dog.

Review.

1) - Changes to be described are –

Image 1 – Bilateral, symmetrical pallor and tissue rarefaction involving gray matter and extending into adjacent funiculi but sparing the margins of the cord.

Image 2 – Motor neurons either morphologically normal or chromatolytic and vacuolated, within a rarefied neuropil containing presumptive glial cells, capillaries and demyelinated, swollen axonal segments

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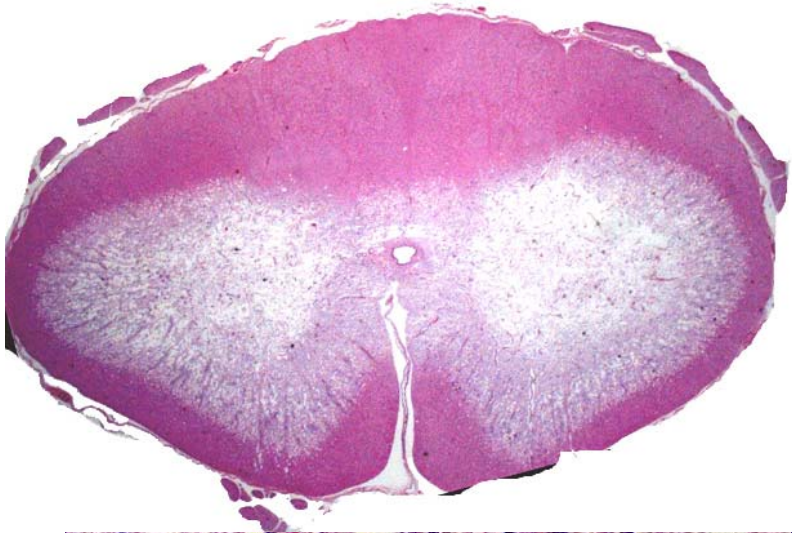

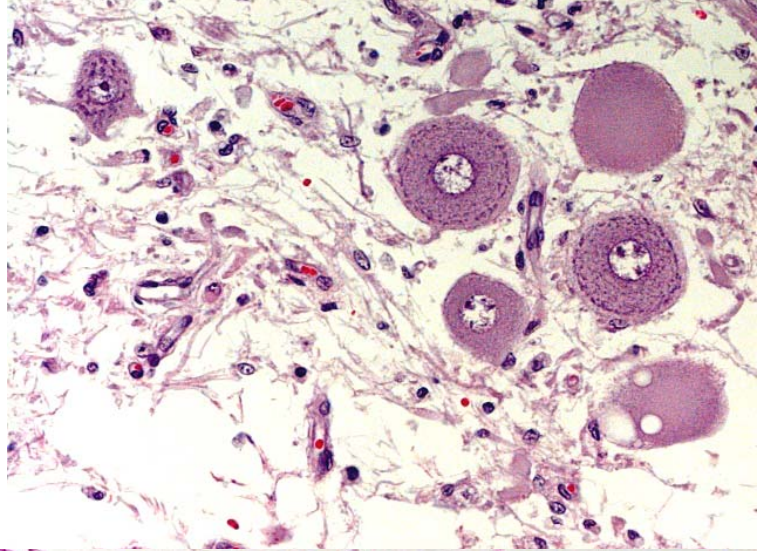

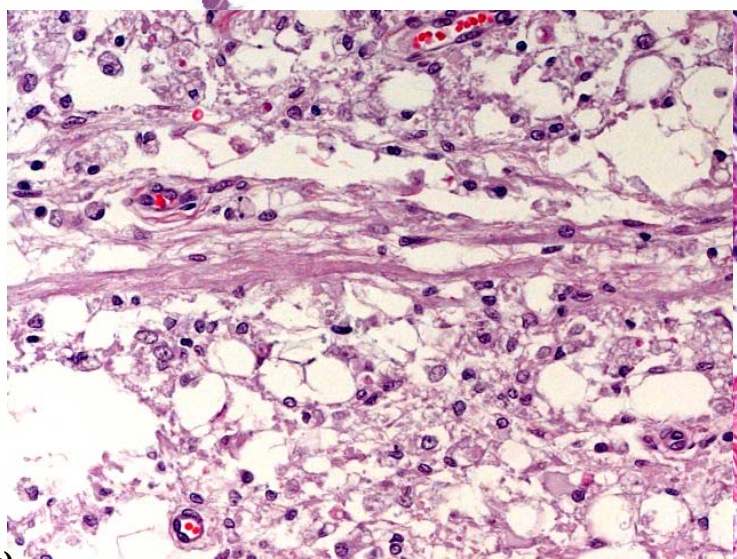

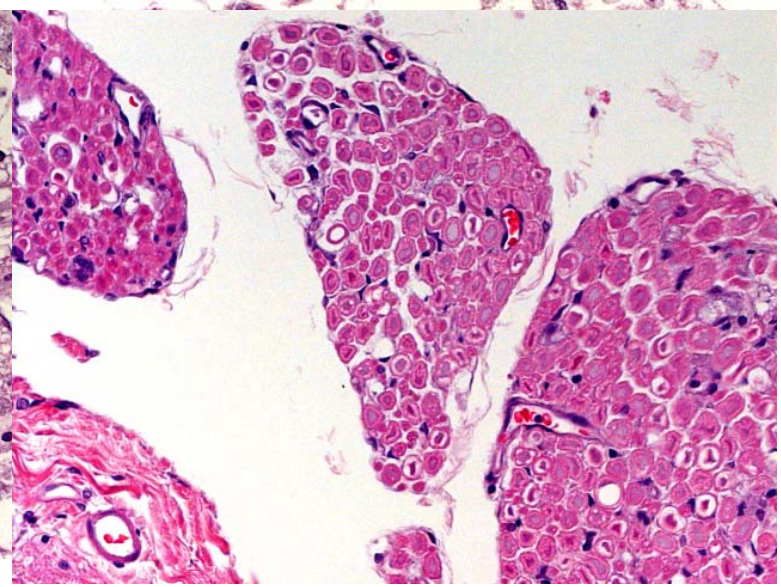

(continued from previous)

Image 3 – Demyelinated but intact motor axons traversing rarified neuropil which also contains some Gitter cells. These axons are on their way FROM the ventral horn neurons TO the motor rootlets

Image 4 – Motor nerve rootlets with normal myelinated axons

2) - Mechanisms NOT apparently operative would be –

ischaemia, inflammatory demyelination, and primary neuronal/axonal degeneration.

Apparently operative are – demyelination and severe oedema

The disease is the result of a gentically-based metabolic disturbance producing encephalomyelopathy in AUSTRALIAN CATTLE DOGS (consult the literature)