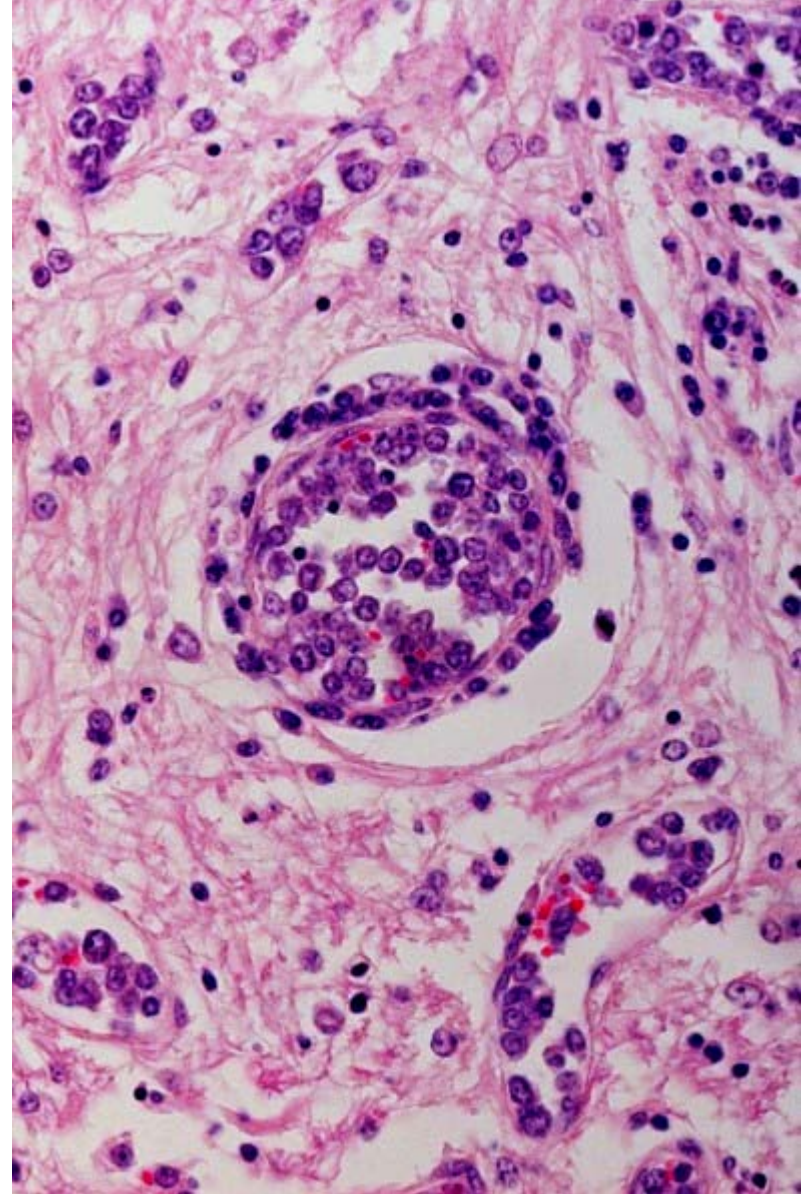
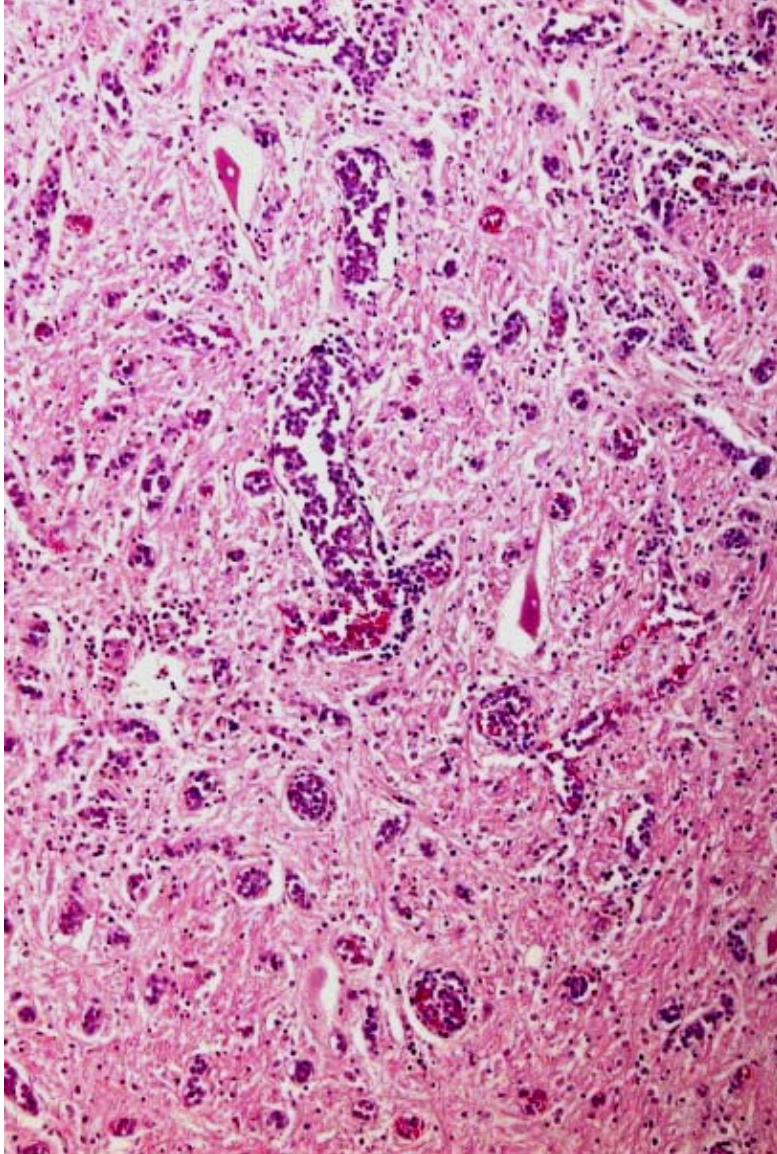


**Case 2. 1 Fields from the caudal brainstem - unspecified
Review**

- 1) The dominant feature is the crowding of dilated blood vessels with large monomorphic mononuclear cells, often with prominent nucleoli. The neuroparenchyma is loosened and mildly hypercellular, suggesting oedema with either gliosis or cellular infiltration or both.**
 - 2) The picture is highly suggestive of intravascular neoplasia, lymphoid or myeloid in nature**
- (Continued next slide)**



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3) a) In a dog likely diagnoses would include **LEUKAEMIA** or **INTRAVASCULAR LYMPHOMA**. b) in a bovine another possibility would be **THEILERIOSIS**, which was the diagnosis in this case.

Comment – The relevant species of *Theileria* are the only known non-viral infectious agents that can induce neoplastic transformation of cells. In intravascular (angiotrophic) lymphoma, neoplastic cells sequester in cerebral blood vessels. In both diseases, infarction is a common additional process.

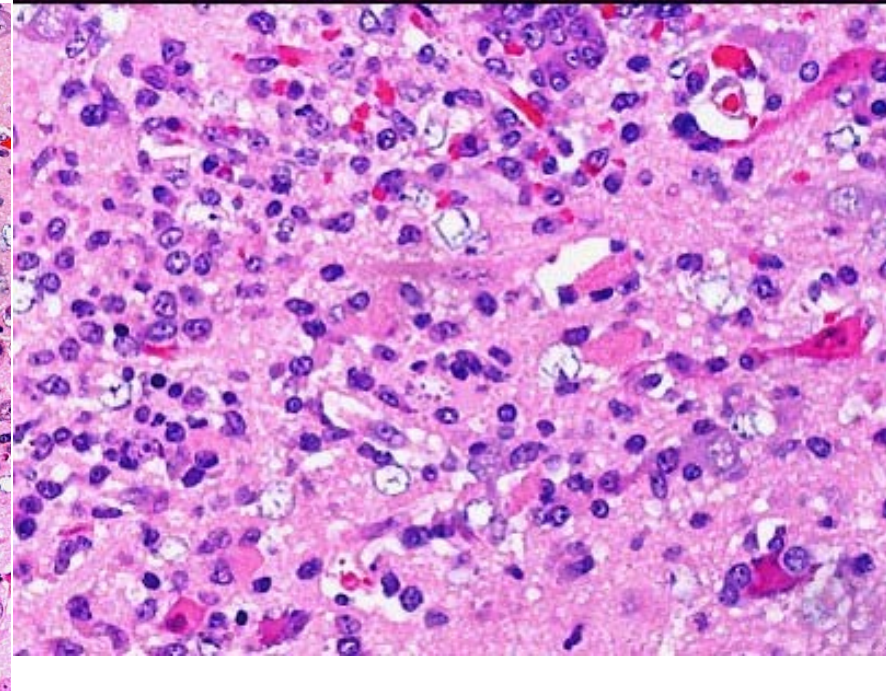
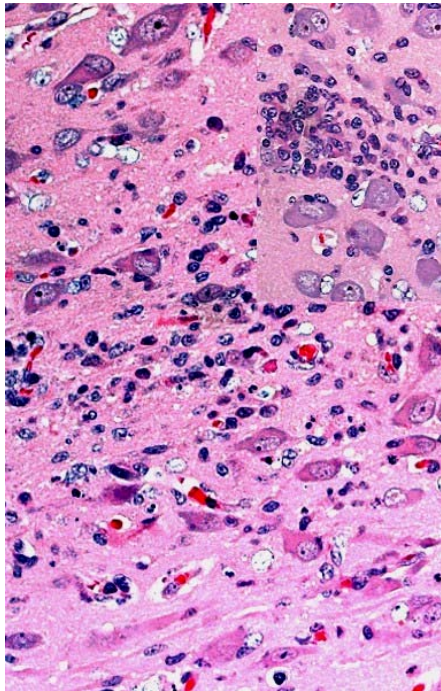
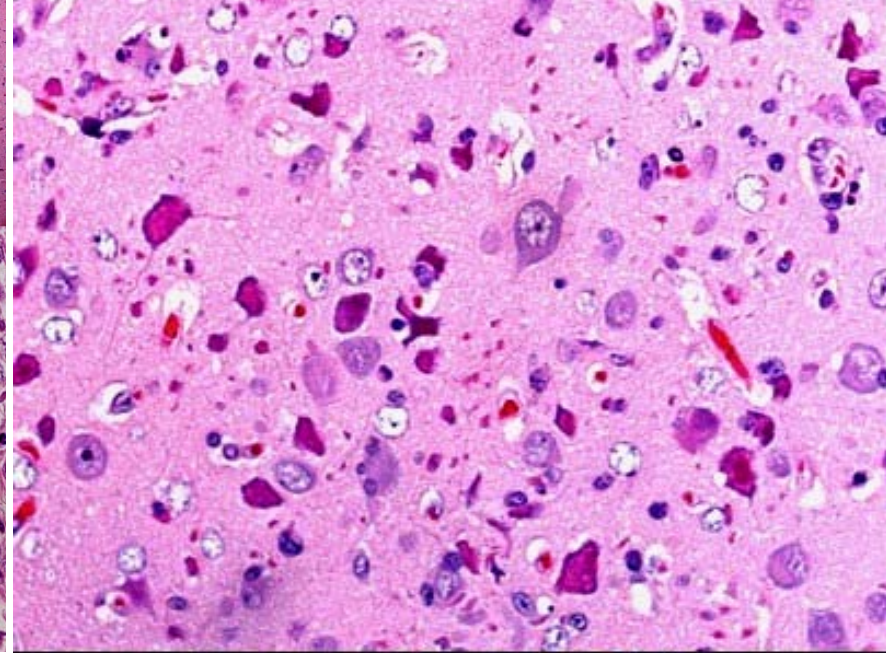
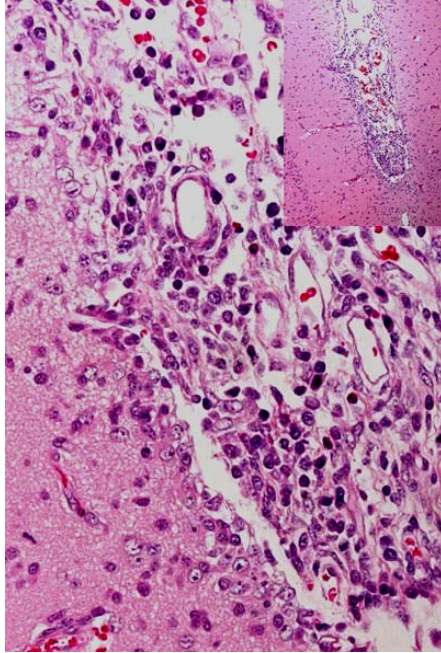
Case 2.2 – Cerebrum (cortex) from a neonatal pig
Review -

1) Significant abnormalities are – hypercellularity of the leptomeninges due to mononuclear inflammatory infiltration; focally variable hypercellularity of the parenchyma due to ditto, with also possible microgliosis; numerous eosinophilic degenerate neurons; pyknotic debris in the parenchyma – origin uncertain, possibly glial or inflammatory cells.

An acceptable MDx could be: Meningoencephalitis, acute, non-suppurative with numerous ischemic neurons, and occasional neuronophagia.

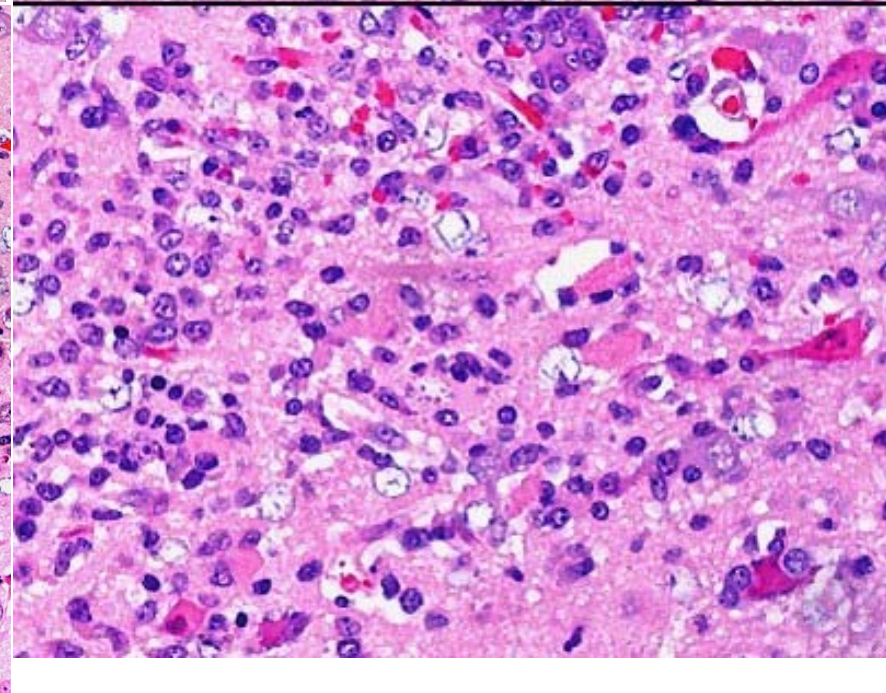
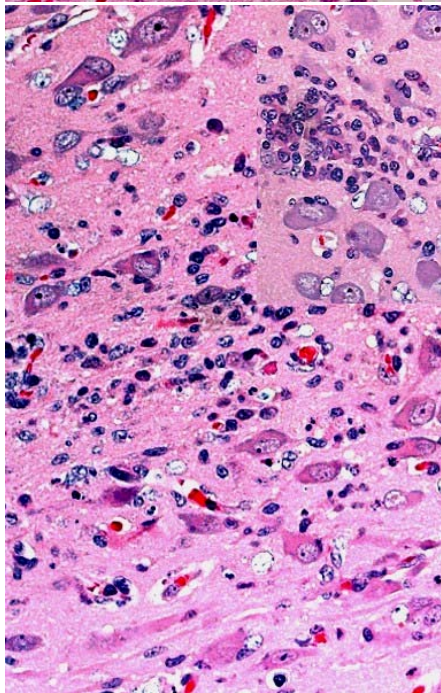
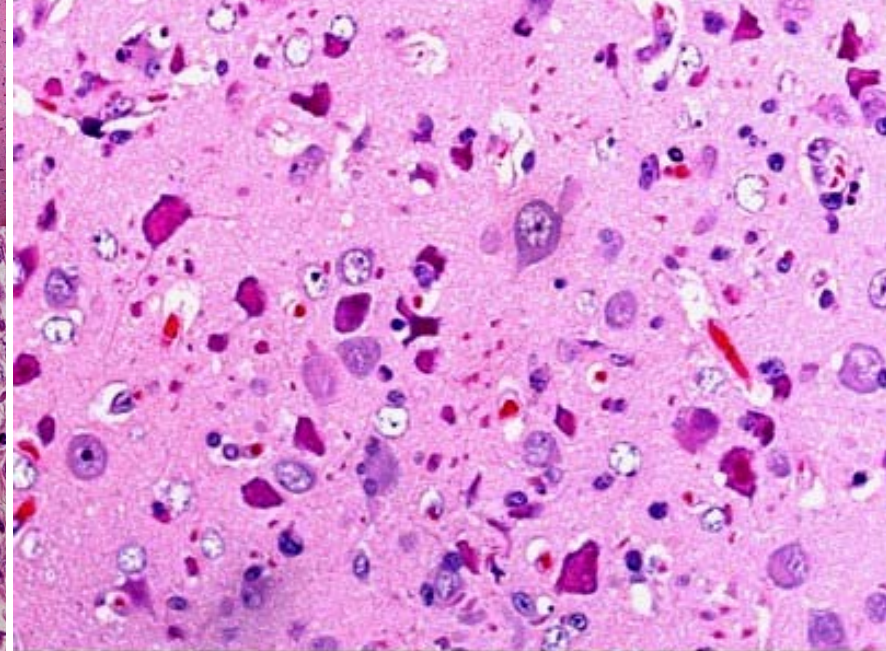
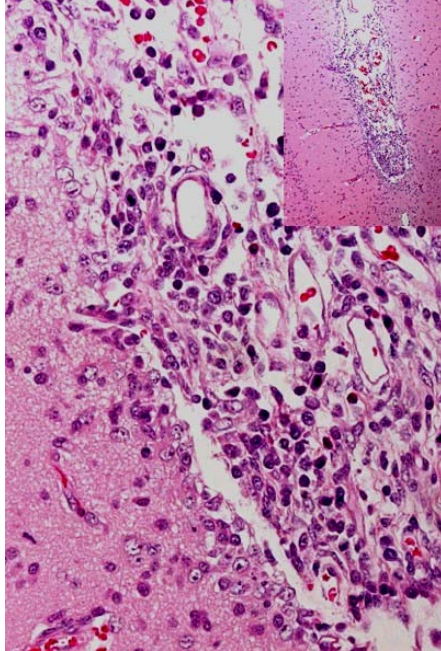
2) The likely class of agent is a neurotrophic virus

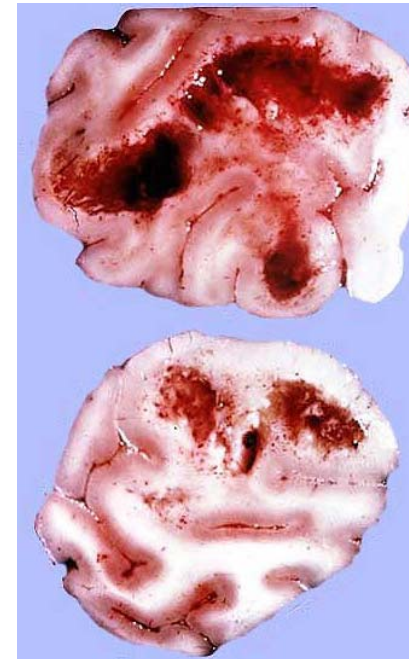
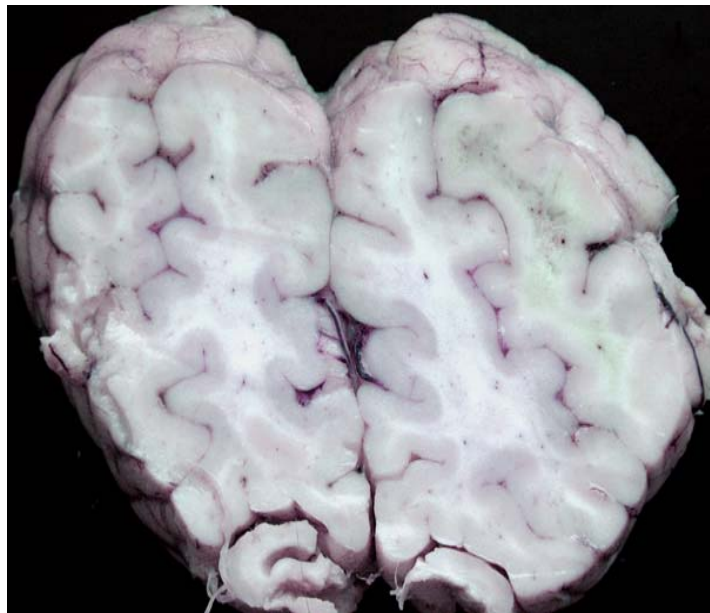
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3) The AGE of the animal and the NATURE and ANATOMIC LOCATION of the lesion suggest two possible aetiologic agents - Herpesvirus suis, and Japanese encephalitis virus – the cause in this case. These agents could be discriminated by immunodiagnostic methods, PCR, and viral culture. For HS inclusion bodies might also be present in neurons and glia, along with ganglionitis and focal necrosis in spleen, liver, tonsil and lymph nodes. Older animals might be affected clinically. In JEV large numbers of associated abortions, mummies and CNS terata would be expected. Older animals would NOT be affected clinically.



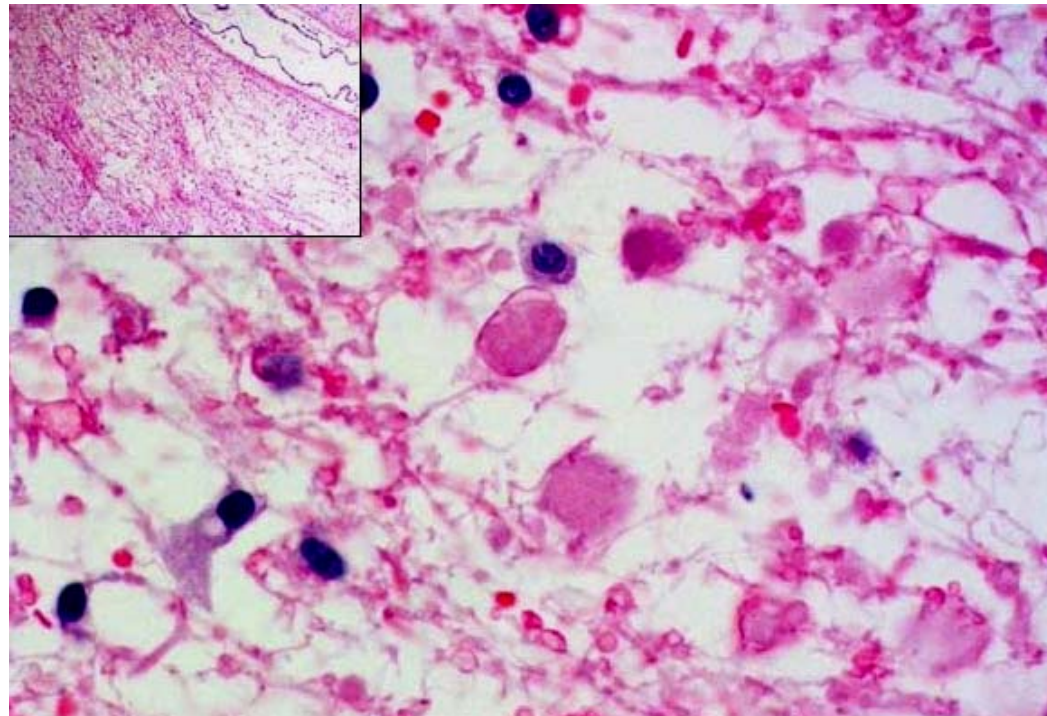


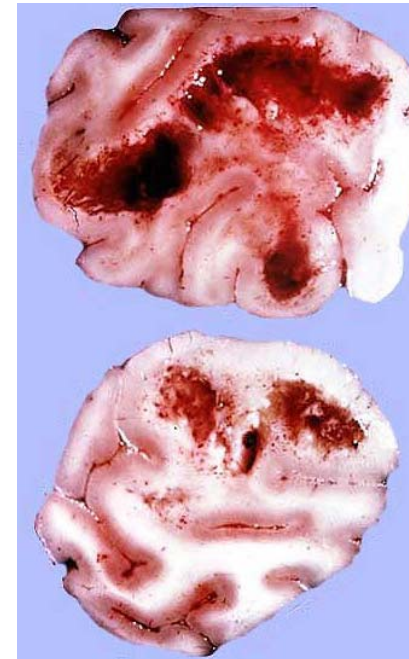
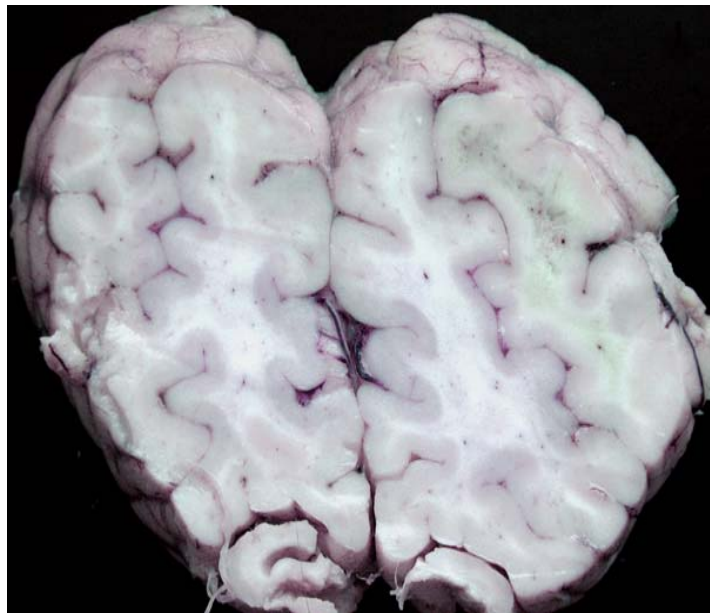
Case 2.3 – Occipital poles of the cerebrum

Review- with supplementary gross and histologic images

- 1) visible abnormalities reasonably assumed not to be artifact are a) asymmetry – diffuse enlargement (swelling) of the right hemisphere b) locally extensive expansion of the white matter on the swollen side c) locally extensive apparent early malacic change (presumptive necrosis) in white matter on the swollen side.
- 2) Likely underlying pathologic processes are VASOGENIC OEDEMA (shown histologically) with locally extensive acute leukomalacia reflecting progression to NECROSIS

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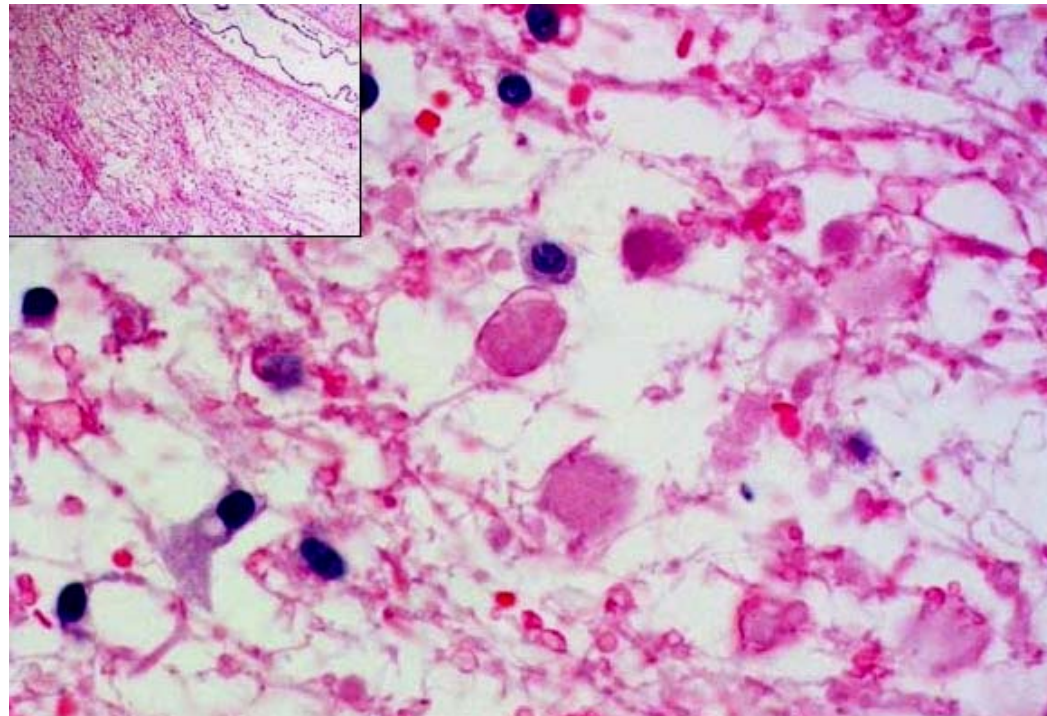


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3) The likely species of the patient is EQUINE and the disease is FUMONISIN B1 MYCOTOXICOSIS

4) In the CNS lesions may also occur in the brainstem, cerebellum and spinal cord. The LIVER may also be afflicted.

Comment – Brain lesions in this disease may be unilateral or at least assymetrical. The basis of the lesion appears to rest on vascular injury in the white matter, and haemorrhage is often significant (as illustrated) Initial vasogenic oedema progresses to necrosis and cavitation, which may focally involve the cerebral cortex as well as the white matter.



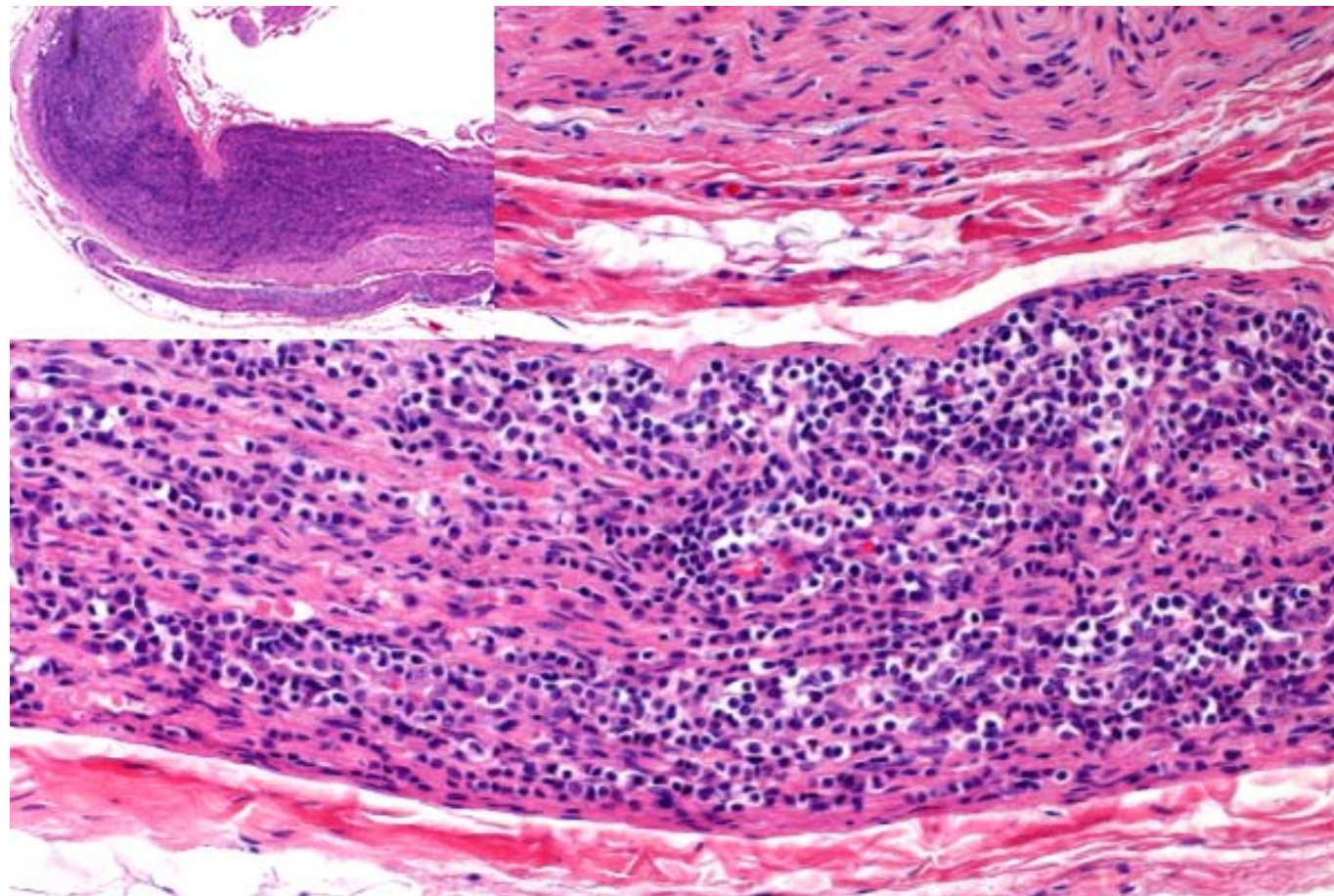
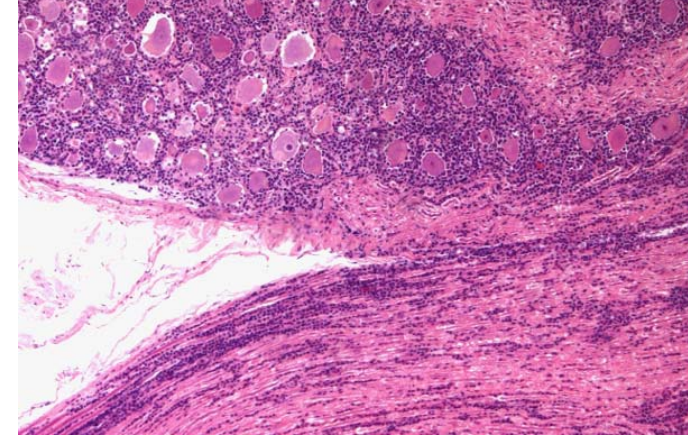
Case 2.4 – Paravertebral tissue from a cat with clinical megaloesophagus and constipation.

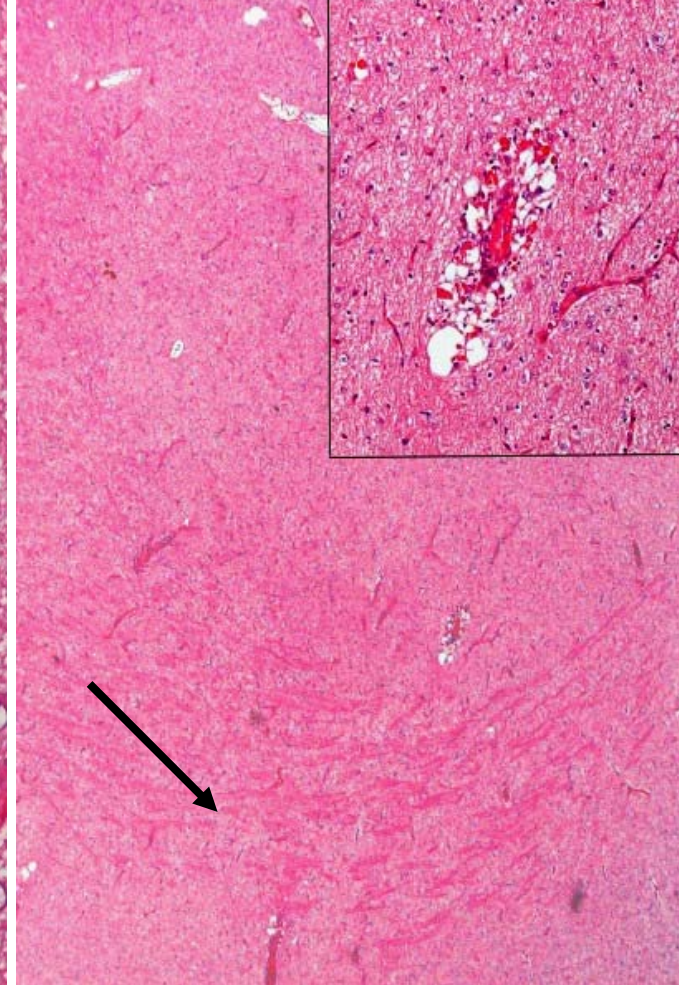
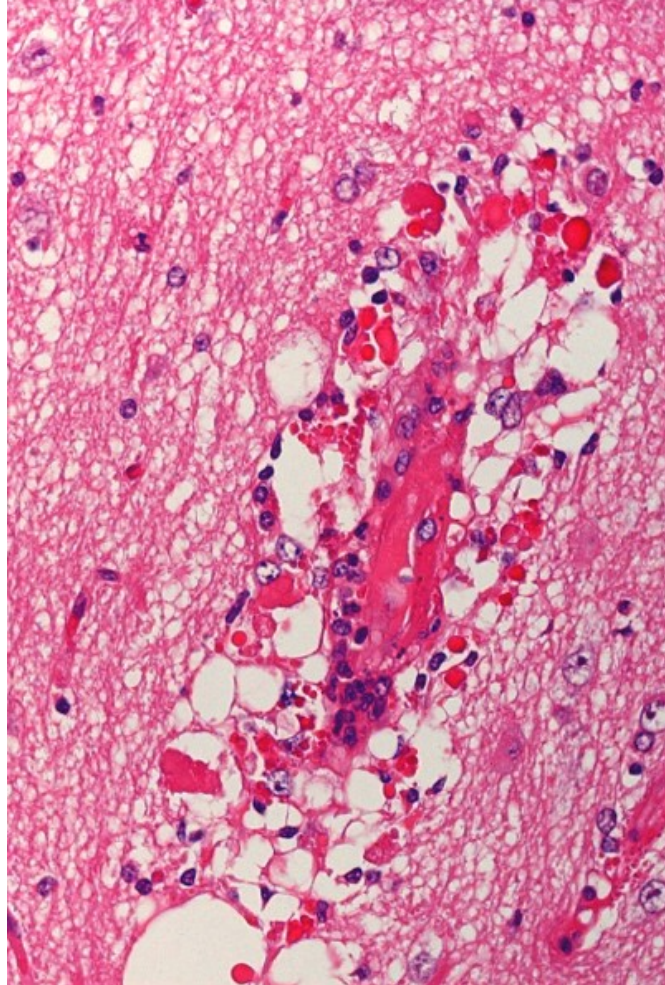
Review- with supplementary images to show more detail

- 1) The paravertebral structures evident are a dorsal root ganglion and adjacent ventral nerve root.**
- 2) Given the Hx, tissues to examine would be autonomic nerves and ganglia, since there were signs suggestive of dysautonomia. The lower image shows splanchnic nerves in the region of the coeliac ganglion.**
- 3) The major abnormality is dense, irregular “interstitial” infiltration of nerves and ganglia by a largely monomorphic cell population. No particular tissue element appears to be specifically targeted, but there is considerable effacement of normal structures.**

- 4) Possible processes are inflammatory or neoplastic infiltration. The monomorphism of the cells, and the “indian file” pattern of infiltration amongst axons in the ventral nerve root point towards round cell neoplasia**

- 5) The most likely Dx in a cat, given the above is lymphoma**
Comment – Tissue infiltration patterns in lymphoma reflect the specificity of the “targetting” of the neoplastic cells. In places, the lesions in this cat also had an inflammatory component and thus resembled Mareks Disease in some respects. The presumptive Dx was lymphoma.

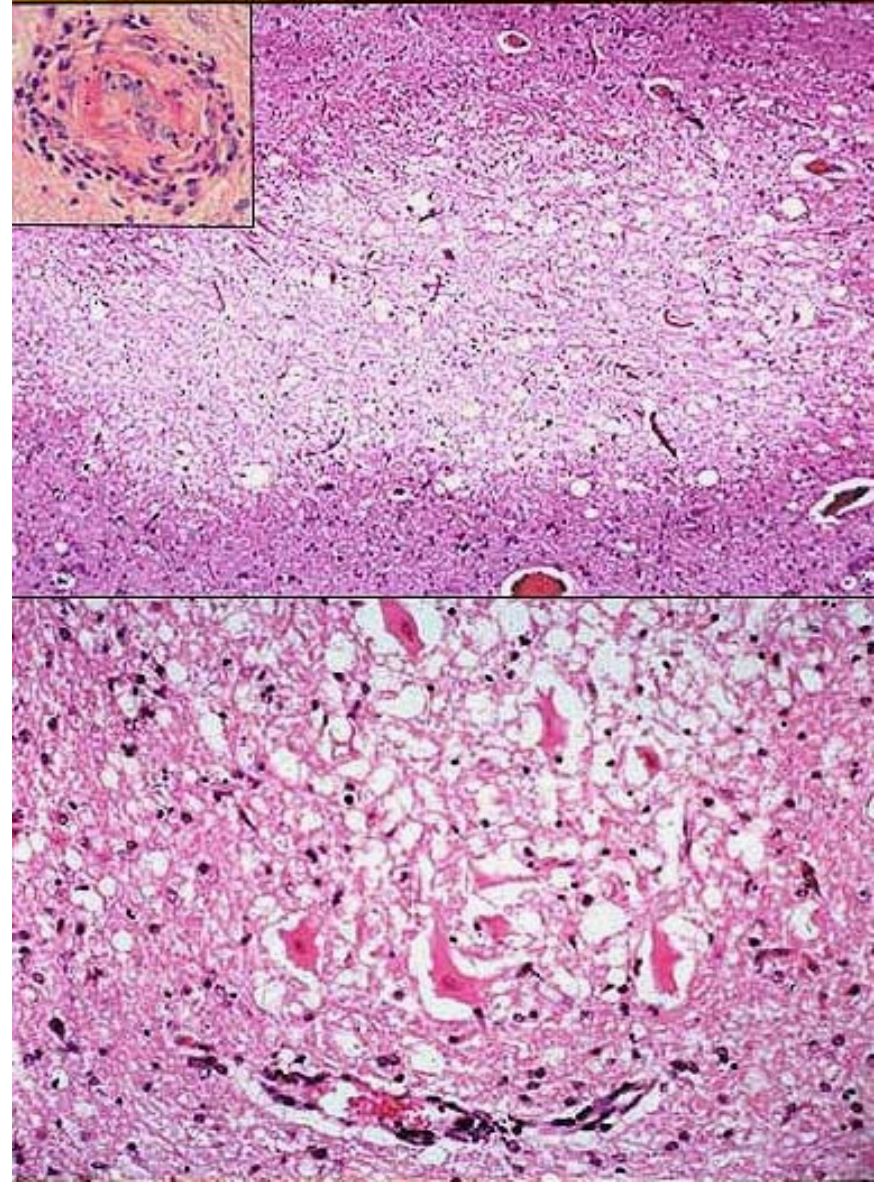
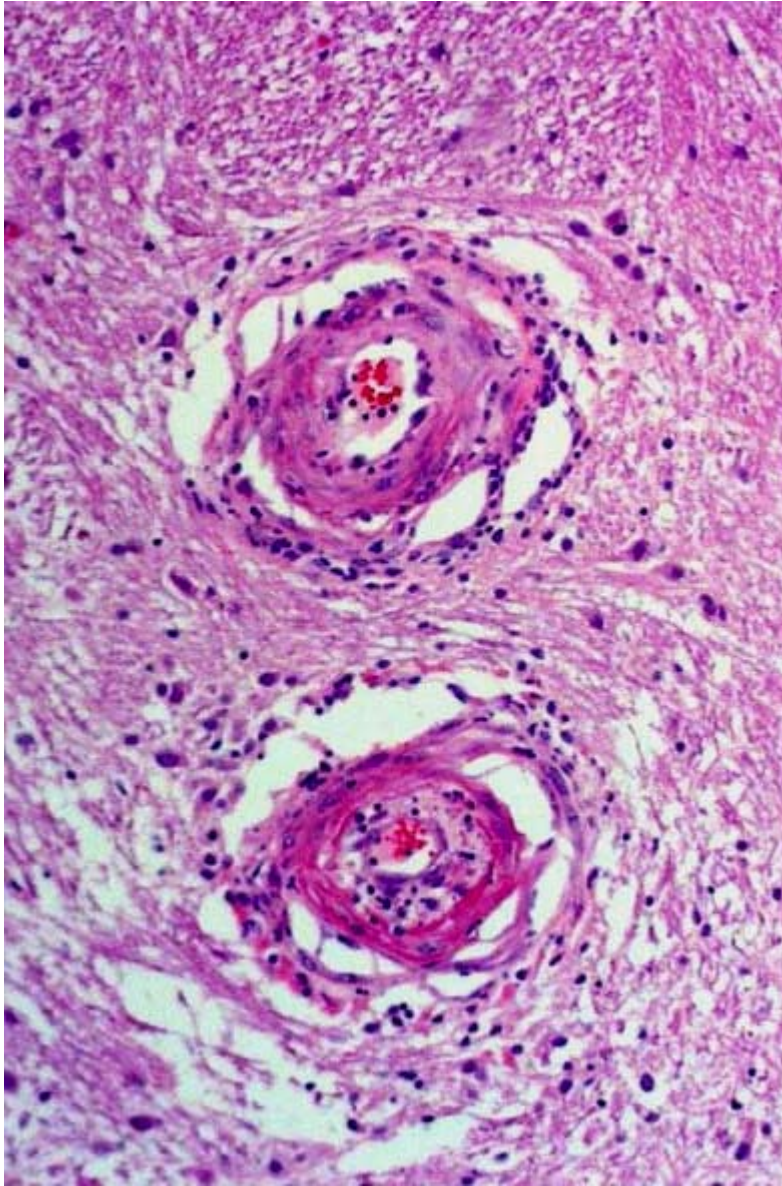




Case 2.5 – midbrain – representative fields of a widely distributed lesion - unspecified

Review

- 1) Three descriptive features of the lesion are: Expansion of the perivascular space with globular hyaline deposits; Hyalinization of the vascular wall; Endothelial swelling.**
- 2) The process appears very acute as there are no degenerative changes in the neuroparenchyma and minimal cellular response in and around the vascular compartment. (see the following figure for an older lesion)**
- 3) The major process operating is acute vascular injury with permeability changes.**
- 4) In a pig, diseases featuring such a lesion could include edema disease (the Dx in this case) and Mulberry Heart Disease. In a ruminant such changes might suggest Clostridium Perfringens epsilon toxemia, although more typically there is homogenous perivascular protein leakage, and lesions have a distinct topographic distribution in the striatum, thalamus and cerebellar white matter.**



Case 2.5 (figure b) – supplementary images to illustrate longer-standing lesions in edema disease. On the left inflammatory and reactive changes in and around vessel walls, on the right recent infarctions of the neuroparenchyma. Over a few weeks these areas would become cavities filled with Gitter cells.