Case 3.1 – Bovine 8/12 caudal brainstem **Review** – with 2 supplementary images with more detail 1) Abnormalities evident are: Image 1 – focal area of pallor; randomly scattered mild perivascular, parenchymal and meningeal hypercellularity. Image 2 – perivenular and pericapillary lymphohistiocytic infiltrates; locally extensive parenchymal rarefaction with scattered degenerate neutrophils; solitary axonal spheroid. **Image 3 – parenchymal** rarefaction with degenerate neutrophils and acute capillary and venular thrombosis. 2) Pathologic processes

evident are – subacute perivascular inflammation, both lympho-plasmohistiocytic and neutrophilic; acute infarction; acute parenchymal necrosis with neutrophilia and acute small vessel thrombosis.

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3) MDx –

Meningoencephalitis and thrombo-vasculitis, subacute, moderate, lympho-histiocytic, plasmacytic and neutrophilic, with acute infarction and locally extensive necrosis. 4) Changes are consistent with SBE (Chlamydophila percorum). The age of the animal is fairly typical, with changes widespread in brain and cord, in both white and grey matter, but seldom grossly visible. Also expected would be fibrinous polyserositis. Clinical course is subacute, and herd morbidity generally low. 5) Vasculitis and infarction occur in Histophilus somni or fungal infections, but certain pathological and epidemiological features would be expected to be different (consult appropriate literature)





Review –

1) Image 1 – dense multifocal and confluent hypercellularity symmetrically distributed in the dorsal funiculi; Image 2 – ditto, with apparent normality in adjacent grey matter; Image 3 – large histiocytoid cells cuffing vessels and crowding adjacent parenchyma 2) Process evident - extensive angiocentric histiocytosis with associated loss of normal tissue elements. Possibilities might inlcude neoplasia, or "lepromatous" type of granulomatous reaction (continued next slide)



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3) The principal characteristics are the monomorphism of the cellular reaction, and the restriction and remarkable bilateral symmetry of the lesion, suggesting the possibility of a metabolic disturbance of the tissue, rather than those mentioned above.

4) A general MDx might be "leukomyelopathy, subacute symmetrical histiocytic, proprioceptive tracts"

5) A specific disease suggested is Globoid Cell Leukodystrophy ("Krabbe's" Disease; Galactosyl-ceramidase deficiency) – the Dx in this case. Lesions typically are widespread in the brain and also in peripheral nerves. (Consulte literature for details)



Case 3.3. – Midbrain (unspecified initially) Review –

There is a non-suppurative, predominantly lymphocytic inflammatory reaction with a distinct peri-aqueductal/ependymal orientation
 An acceptable MDx might be – Encephalitis, periventricular with ependymitis, lymphocytic and histiocytic, subacute, moderate (continued next slide)



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3) A specific disease suggested by this pattern of lesion a) in an ovine would be Visna, b)in an equine would be EIA.
5) Given that the subject in this case was a caprine, a probable diagnosis would be CAEV (which was the case). However this "Visna-like" reaction is NOT TYPICAL of this infection and the classical lesions have a very different character (see the following slide).
Comment – periventricular inflammation is also a prominent feature of Feline Coronavirus (FIP), but the reaction usually features many neutrophils and plasma cells, and a fibrinous effusion.



Case3.3 – with supplementary images to show the more typical type of lesion with CAEV infection. Lesions are localised areas of inflammatory demyelination, often in the spinal cord (as shown here). There is dense lympho-histiocytic infiltration, reactive astrogliosis and frequently eventual cavitation.

Case 3.4 – Thalamus of a cat – Hx of acute seizures and death **Review – with supplementary** image - adjacent thalamus 1) The general features are perivascular lymphoid cuffing and localised parenchymal hypercellularity, probably due to a combination of microgliosis and cellular infiltration. In addition, some neurons contain large, eosinophilic, intracytoplasmic inclusion bodies (arrows) 2) MDx – Encephalitis, Subacute non-suppurative, thalamus, with intracytoplasmic neuronal inclusion bodies. The disease which should be excluded is Rabies (or other Lyssavirus infection?)

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3) Cautionary note – in several species "pseudo-Negri bodies" can occur in certain regions of the brain. In the cat, this includes the lateral geniculate body (as in this case) and the hippocampus. The third image here shows numerous such bodies in the LGN of the cat in this case. Note also that Negri bodies without inflammation does not exlude rabies. In this case rabies was exluded by immunocytochemistry.

cortex - unspecified **Review** – 1) The significant features illustrated are: Palor, vascular prominence and and rarefaction of the neuropil in outer cortical grey laminae; Numerous eosinophilic neurons; perivascular protein globules; Venules with hypertrophied endothelium and perivascular infiltration by numerous eosinophils and mononuclear cells 2) Processes that can be identified are: extensive acute laminar ischemic neuronal necrosis and vasogenic oedema of the neuropil, with moderate inflammatory response, predominantly eosinophilic

Case 3.5 – Cerebral

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3) An acceptable MDx could be: Cerebrocortical neuronal necrosis, acute, laminar, with moderate perivascular eosinophilic meningoencephalitis. These changes are consistent with acute salt poisoning/water deprivation in the pig, and this was the diagnosis in this case.

Comment: The combination of cortical necrosis and eosinophilic inflammation is virtually pathognomonic for this porcine disease. The pathogenesis of the eosinophilia is unknown, but acute inflammatory infiltrates in pigs often contain many eosinophils.

