FAO: Mr Seamens

Rept no N-202940/ 1
Date 17/06/2003

NECROPSY REPORT - Hosp no 202940

Name XENA
Species DOG
Breed DEERHOUND

Owner Mr Seamens
Age 3.5 Y Sex F Dest N
Slide 03/2018,2483-94,2065
Date 12/06/2003 Cost 0.00

Report to ALEX LUJAN
NEUROLOGY

FIBROCARTILAGINOUS EMBOLISM
MYELOMALACIA
SPINAL CORD HAEMORRHAGE

Gross Pathology: The three and a half year old female Deerhound had a substantial increase in cerebrospinal fluid in the cisterna magna and the subarachnoid space around the medulla oblongata and first cervical segment of the spinal cord. Extradural haemorrhage, mainly on the left side, was evident at the level of the first cervical vertebra (C1) and the third lumbar vertebra (L3). A sagittal section of the L2-L3 vertebral junction revealed a 4 mm diameter gelatinous focus within the intervertebral disc. Hydrocephalus was evident in the right ventricle of the brain on sectioning. The liver was enlarged and had rounded borders. There was accentuation of hepatic lobules with a yellow reticulate pattern in portal areas. The spleen was congested and the ventral pole was irregularly enlarged to 10 cm x 10 cm x 8 cm. There were multiple 1 mm diameter, brown to yellow, coalescent plaques on the splenic capsule.

Histopathology: Longitudinal and cross-sectional segments of spinal cord were examined at cervical levels C1, C3, C5 and C7, thoracic levels T2, T6, T9 and T12, lumbar levels L2, L3 and L4 and sacral level S2. Multifocal, locally extensive haemorrhage and myelomalacia were evident in the grey and white matter of L3, L4 and S2. Some blood vessels in these segments contained degenerate fibrocartilaginous emboli or eosinophilic fibrillar material and had margination of neutrophils. Extradural haemorrhage and thrombosis were present in C1, T12, L2 and L3. Arterioles with subintimal haemorrhage, mural necrosis and fibrillar plaques were present in C3, C5 and C7.

Mild vacuolation, phagocytic chambers and spheroids, including a few targetoid spheroids, were evident throughout the spinal cord. C7 and L3 had a few foci of mononuclear cells. Some white matter areas of the brain had mild vacuolation. The lateral ventricles were expanded. There were no interpretable findings in a decalcified section of L2-L3 intervertebral disc. The splenic mass exhibited congestion, mild haemosiderosis and mineralised plaques on the capsule. The lung had congestion, alveolar oedema with a few macrophages and partial atelectasis. The liver was congested and there was vacuolation of perisinusoidal cells. Hepatocytes exhibited mild diffuse vacuolar change. The kidney had mineralisation of a tubule. Mild infiltrates of lymphocytes and plasma cells were present in the intestinal mucosa. The heart and stomach were unremarkable.

Comment: The haemorrhage and myelomalacia in spinal cord segments L3 to S2 are consistent with a vascular accident due to fibrocartilaginous embolism. Degenerate fibrocartilaginous material was present in some blood vessels in the affected segments. Although a gelatinous focus was visible grossly in the L2-L3 intervertebral disc, no degenerative changes could be confirmed at this site histologically. The extradural haemorrhages in the cervical portions of the spinal cord would be due to collection of cerebrospinal fluid. The splenic mass was consistent with benign nodular hyperplasia. The hepatomegaly and vacuolar change in hepatocytes would have been due to treatment with corticosteroids.

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