In Confidence

Scrapie-like spongiform encephalomyelopathy in a domestic cat: report of a confidential consultation with the Department of Pathology, School of Veterinary Science, University of Bristol, Langford, Bristol BS18 7DU

On May 1, 1990 I was consulted by Dr G R Pearson, Department of Pathology, School of Veterinary Science, University of Bristol regarding the central nervous system pathology observed in a 5 year old neutered male Siamese cat referred to the Department of Veterinary Medicine of the school (Clinic Accession No M27610) (Path Lab No 90-315).

As a result of an initial telephone consultation I visited Dr Pearson’s laboratory on May 1 and examined histological sections from the case. Preliminary findings suggested a vacuolar encephalomyelopathy closely resembling those attributable to unconventional viruses. The neuropathology was compared with that of an adult cat with an intrathecal spinal lymphosarcoma and a case of feline dysautonomia (Key-Gaskell Syndrome). Additional material was selected from the brain and spinal cord for processing in Pathology Department, Central Veterinary Laboratory, Weybridge.

Further neuropathological observations support a diagnosis of scrapie-like spongiform encephalomyelopathy comprising moderate to severe spongiform change (spongiosis) in grey matter neuropil, vacuolation of neuronal perikarya and astrocyte reaction throughout the neuroaxis of the central nervous system. In routine haematoxylin and eosin stained sections amyloid-like plaques were not apparent but further study of this aspect is anticipated. Mild to moderate axonal degeneration was present in some white matter tracts of the brain and spinal cord.

This is the first report of unequivocal light microscopic changes of a naturally occurring subacute spongiform encephalopathy resembling scrapie in the domestic cat (Felis domesticus).

The clinical history of this animal is as yet incomplete, requiring for completion disclosure of the histopathological diagnosis to the referring veterinary surgeon and the owners.

The cat presented first on February 20 with an ataxia which had a pelvic to pectoral limb progression with hypermetria particularly of the pectoral limb gait. The behaviour is reported to have been normal. There was postural difficulty with falling associated with defecation and micturition.

On admission to the School of Veterinary Science small animal clinic blood and cerebrospinal fluid (CSF) were sampled. No abnormalities were detected on haematological and blood biochemical examinations nor on examinations of CSF.

Euthanasia and necropsy were carried out on April 6, 1990. The general bodily condition was described as "fat". The bodyweight was 4.5Kg. The lungs were congested. There were no other significant gross pathological findings.

Histopathological examinations were made of selected viscera including
liver but no significant changes were found.

The pathomorphology in this case leaves little doubt regarding the nature of disease but in view of the far reaching consequences of the diagnosis additional work is required before it can be confirmed as a member of the transmissible spongiform encephalopathy group:-

1. Completion of clinical history including feeding practices and social contacts.

2. Immunocytochemical demonstration of protease resistant protein (PrP) in tissue sections (in hand).

3. Transmission attempts to small laboratory rodents, particularly mice and hamsters and possibly also to cats. From the present case only formalin fixed brain material is available but Dr Pearson is aware of the requirement for fresh CNS tissue (caudal medulla, cervical spinal cord and frontal and occipital cerebrum) also spleen, taken aseptically with disposable instruments from future cases presenting similarly. This material could be made available also for SAF examinations.

An appendix to this report giving a brief relevant literative review is in preparation.

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