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IN CONFIDENCE 44

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Scrapie-like disorder in a Nyala (Tragelaphus angasi)

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~~Nyala (tragelaphus angasi) are African antelope belonging to the family Bovinae. We were unable to locate reports in the literature concerning nervous disease of this species and this communication describes neurological signs associated with a diffuse encephalomyelopathy in a captive nyala.~~

start here

(Tragelaphus angasi)

A two and a half year old female nyala, bred and maintained at a wildlife park in England, developed hind limb ataxia, followed several days later by head tilt, opisthotonus and brief episodic torticollis ~~ensued.~~ Persistent licking of the tail base and rump ~~was noted and~~ led to mutilation with ulceration of the skin. Frequent micturition was observed. Neurological signs and mutilation were progressive and after a three week illness the animal was killed and a necropsy ~~was~~ conducted.

There were no significant gross lesions. On histopathological examination of the brain and cervical spinal cord segments ~~showed bilateral~~ spongiform change and neuronal vacuolation were the predominent features. Small round optically empty vacuoles which were sometimes confluent ~~coalesced~~ were distributed bilaterally in neurophil of grey ~~matter~~ and white matter. ~~Such~~ This ~~spongiform~~ change was most severe at the level of the medulla oblongata and ~~lesions~~ ~~gradually~~ diminished rostrally and caudally. ~~Spongiform change~~ Vacuoles were sparse rostral to midbrain. ~~were mild and~~ In cerebral cortex a laminar pattern of vacuolation involved the deep layers. ~~of cortex was present. Neuronal vacuolation was also present.~~

laterally symmetrically or diffuse

Paragraph

(X) There are few reports in the literature describing diseases of the Nyala. Cardio myopathy usually associated with death and no preminitory signs has been reported in twenty one Nyala in a Zoological collection (Liu and others. 1982) but we were unable to find any reports of neurological conditions in this species.

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sharply defined, or multilocular

Neuronal vacuoles were single or multiple ~~sharply defined~~ and also optically empty. Single vacuoles were usually eccentric and displaced Nissl substance. Vacuolar content did not stain for ~~Vacuoles did not contain~~ neutral lipid or glycogen. The severity of neuronal vacuolation paralleled that of spongy change of the neurophil and was most frequent in anatomic nuclei ~~at the level~~ involving of the caudal medulla, particularly the dorsal vagal ~~nuclei~~ (Fig. 1), cuneate and ~~nuclei~~, pontine nuclei, neurons of the and reticular formation. ~~Neuronal vacuolation was~~ but though sparse in basal ganglia and thalamus ~~but~~ locally ~~moderate~~ frequent in the nucleus of the habenulopeduncular tract of the thalamus.

? —

There were focal perivascular mineralised plaques ~~were present~~ in lobule 1 of the cerebellar vermis. ~~Tissue~~ Sections of medulla, midbrain, thalamus, cerebellum and cerebrum immuno-stained for glial fibrillary acidic protein ~~did not~~ showed no evidence of astrocytosis ~~asymmetry of distribution or abnormal concentrations of astrocytes.~~ Selected sections of brain were stained for amyloid by the congo red method with negative results.

The nyala was from the larger of two captive groups of the species ~~located at the wildlife park.~~ ~~This group~~, comprising ~~of~~ five breeding females and their calves and six sub-adult males, ~~are~~ kept on hard standing with ~~and have~~ no direct contact with other ruminants ~~ungulates~~. ~~X~~ The smaller group which included an 18 month old full sibling of the index case ~~and is~~ was kept in an adjacent paddock and ~~which~~ had fence line contact with arabian oryx (Oryx damma). None of the remaining nyala showed neurological signs. P// The clinical signs

Insert (X) attached.

(A)

Scrapie of domestic sheep and goats is the exemplar of these slowly progressive or subacute transmissible spongiform encephalopathies which also were recorded ~~elsewhere~~ in mule deer (Latin name) (ref), elk (Latin name.) (ref), mink (Mustela vison) (ref) and man (refs).

and the distribution and frequency of neuropathological changes in this case resemble closely those of scrapie of sheep (Fraser 197

~~reported here are similar to some of those seen in scrapie~~ ?

~~of sheep (Dickinson, 1976).~~

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While neuronal vaculation and spongiform changes of the neuropil are essentially non-specific features and may be present ~~are found~~ in a wide range of infectious and metabolic conditions as well as ~~being found~~ clinically in normal sheep brain. ~~However, taking into consideration the absence of lipid or glycogen within vacuoles, the multiloculated~~ the nature, ~~of the neuronal vaculation and the degree and~~ distribution and frequency of ~~vaculation~~ these lesions are strongly suggestive of ~~Scrapie of sheep and goats~~ the spongiform encephalopathies (Dickinson, 1976, and ~~chronic wasting disease of mule deer (Williams and Young, 1980).~~

A →

~~Other components of~~ Scrapie and Chronic Wasting Disease of mule deer pathology (Hadlow 1961; and Williams & Young 19

~~includes~~ Astrocytosis (cytosis) (gliocytosis or sclerosis) a reported feature of ~~(Hadlow, 1961; Williams & Young, 1980) which~~ was not recognised in this case.

~~At least~~ According to some workers, however ~~sources~~ astrocytosis is an inconsistent feature of scrapie of sheep (McKenzie, 1983; Frazer, 1976) and studies of experimentally infected mouse brains shows only a very low incidence (3% of 10,000 brains) of "gliocytosis" (Frazer, 1979). Frazer ~~xxxx~~ suggests that the controversy surrounding astrogliosis is related to difficulties of interpretation of capricious metallic gold impregnation techniques and the difficulties of handling large enough samples for ~~meaningful~~ valid quantitative analysis. but one study (McKenzie 1983) that showed ~~It is perhaps worth noting that an immunocytochemical~~

~~study of the distribution~~ of ~~the~~ astrocyte specific protein glial fibrillary acidic protein ~~did not show~~ no significant astrocytosis in sheep affected with clinical Scrapie, employed minimum staining (~~McKenzie, 1983~~). P // Cerebral amyloidosis ~~Amyloid~~ is seen commonly in chronic wasting disease of mule deer (Bahmanyar, Williams, Johnson, Young and Gajdusek, 1985) but is infrequent in Scrapie of sheep (Gilmour, Bruce and Mackellar, 1986). In murine Scrapie amyloid ~~is found~~ features only in specific genotypes infected with selected strains of Scrapie agent (Bruce, Dickinson and Frazer, 1976) // ~~Thus~~, We conclude that the ~~clinical and histopathological changes found~~ findings in this case are sufficiently similar to ~~those of~~ the transmissible spongiform encephalopathies of phytogenetically related species. to warrant a provisional diagnosis of scrapie-like disease. Transmission experiments ~~and~~ or examination ~~of~~ of fresh tissues were not retained for ~~for~~ Scrapie associated fibrils ~~were indicated in future cases~~. (Merz et.al. 1981)

Experimental ~~A~~ transmissible spongiform encephalopathies have been confirmed in ~~five species~~ naturally occuring disorders of: man (Creutzfeldt-Jacob disease and Kuru) (Marsh, 1976), sheep and goats (Scrapie) Dickinson, 1976), mule deer (chronic wasting disease) Williams, Young and Marsh, 1982), and mink (transmissible mink encephalopathy) (Marsh, 1976). ~~A sixth~~ Another species, elk, has been reported to have a spongiform encephalopathy in geographic // areas where chronic wasting disease of mule deer is endemic (Williams and Young, 1982), but transmission experiments have not been reported. ~~Previous studies have shown that the infectious~~ to a variety of laboratory species (Scientific name) (Mustela vison) (name) (B)

order chronologically - quote signed ref. using transmission

Ref. from Mike Dawson on Table of transmission studies

There is some evidence that these disorders may also by chance ~~agent of the spongiform encephalopathies may~~ cross species barriers, ~~and may be transmitted in a variety of circumstances~~. For example the disease called transmissible mink encephalopathy may be acquired by mink as a result of ~~transmitted by~~ intradermal inoculation ~~injection~~ on the teeth of littermates fed scrapie infected tissues (Marsh and Hanson, 1979); ~~Kuru is acquired by ingestion of infected human tissues in cannabalistic rites (Alpers, 1979)~~.

(B) It is thought that under natural conditions sheep are infected orally (Hadlow, Kennedy and Race, 1982) and that both horizontal and vertical infection may occur (Hourrigan, Klingsporn, Clark and de Clamp, 1979). The mode of vertical transmission in Scrapie under natural circumstances has not been determined but infectious agent has been demonstrated in the nasal mucosa of the dam and in the placenta (Pattison and Millson, 1961).

~~The cause of the disease in this case, assuming it is infectious~~ is unclear. P // The nyala in this case ~~have~~ had no direct contact with sheep or goats and the paddocks to which they had access ~~have~~ not previously been used by these species. It ~~The affected animal~~ was bottle reared on domestic cow's ~~bovine~~ milk by an attendant with responsibility for a small number of sheep grazed at a different location but Scrapie has not been reported in these sheep. Neither mink nor North American deer are kept at the wildlife park.

→ (C) Spongiform encephalopathies have not previously been reported ~~This is the first report of a scrapie-like disease~~ in the sub-family bovinae ~~(or family) of the family~~ Bovidae. In view of

prevalences attained by
the ~~limit incidence of~~ Scrapie in some sheep flocks and chronic
wasting disease in some mule deer populations ~~there are~~ these observations may have possible serious related
implications for captive breeding programmes of endangered
bovinae.
~~antelope species~~.

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REFERENCES

1 ALPERS, M.P.: Epidemiology and etology in Kuru. In: Slow transmissible diseases of the Nervous System, ed. Prusiner and Hadlow, vol. 1, pp. 67-90, Academic Press, New York, 1979.

2 BAHMANYAR, S.; WILLIAMS, E.S.; JOHNSON, F.B.; YOUNG, S.; GAJDUSEK, D.C. Amyloid plaques in spongiform encephalopathy of mule deer. J Comp Pathol 95: 1-5, 1985.

3 BECK, E.; DANIEL, P.M.: Kuru and Creutzfeldt-Jakob disease; Neuropathological lesions and their significance. In: Slow transmissible diseases of the Nervous System, ed Prusiner and Hadlow, vol. 1, pp. 253-270, Academic Press, New York 1979.

4 DICKENSON, A.G.: Scrapie in sheep and goats. In: Slow diseases of animals and man, ed Kimberlin, R.H., pp. 239-242, North Holland publishing company, Amsterdam, 1976.

5 GILMOUR, J.S.; BRUCE, M.E.; MACKELLAR, A.: Cerebrovascular amyloidosis in scrapie affected sheep. Neuropathol Appl Neurobiol 12: 173-183, 1986.

6 HADLOW, W.J.; KENNEDY, R.C.; RACE, R.E.: Natural infection of suffolk sheep with scrapie virus. J Infect Dis 146: 657-664, 1982.

7 HOURRIGAN, J.; KLIGSPORN, A.; CLARK, W.W.; de CAMP, M.: Epidemiology of scrapie in the United States. In: Slow transmissible diseases of the Nervous System, ed. Prusiner and Hadlow, vol. 1, pp. 331-356, Academic Press, New York, 197[illegible]

8 MARSH, R.F.: The subacute spongiform encephalopathies. In: Slow Virus diseases of animals and man, ed Kimberlin, R.H., pp. 359-382, North Holland publishing company, Amsterdam, 1976.

9 MARSH, R.F.; HANSON, R.P.: On the origin of transmissible mink encephalopathy. In: Slow transmissible diseases of the Nervous System, ed Prusiner and Hadlow, vol. 1, pp. 451-460, Academic Press, New York, 1979.

10 MASTERS, C.L.; GAJDUSEK, D.C.; GIBBS, C.J.: ~~Cure~~ Creutzfeldt-Jakob disease virus isolations from Gerstamann-Straussler syndrome; with an analysis of the various forms of amyloid plaque deposition in the virus-induced spongiform encephalopathies. Brain 104: 559-588, 1981.

11 MASTERS, C.L.; GAJDUSEK, D.C.; GIBBS, C.J.; BERNOULLI, C.; ASHER, D.M.: Familial Creutzfeldt-Jakob disease and other familial dementias: an inquiry into possible modes of transmission of virus induced familial diseases. In: Slow diseases of the Nervous System,

ed Prusiner and Hadlow, vol. 1, pp. 143-194. Academic Press, New York, 1979.

12 MACKENZIE, A.: Immunohistochemical demonstration of glial fibrillary acidic protein in Scrapie. J Comp Pathol 93: 251-259, 1983.

13 PATTISON, I.H.; MILLSON, G.C.: Experimental transmission of scrapie to goats and sheep by the oral route. J Comp Path 71: 171-176, 1983.

14 WILLIAMS, E.S.; YOUNG, S.: Spongiform encephalopathy of rocky mountain elk. J Wildl Dis 18: 465-471, 1982.

15 WILLIAMS, E.S.; YOUNG, S.: Chronic wasting disease of captive mule deer: A spongiform encephalopathy. J Wildl Di 16: 89-98, 1980

16 WILLIAMS, E.S.; YOUNG, S.; MARSH, R.F.: Preliminary evidenc of transmissibility of chronic wasting disease of mule de Proceedings of the Wildlife Disease Association Annual conference, Madison, Wisconsin, Abstract no. 22, 1982.