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A SLAUGHTERHOUSE SURVEILLANCE OF BOVINE SPONGIFORM ENCEPHALOPATHY IN BANGLADESH

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Bovine spongiform encephalopathy (BSE) is a transmissible fatal neurodegenerative infectious disease first identified in the United Kingdom in 1987. This disease has a causal link with an old disease "Scrapie" which has been prevalent in sheep for over 200 years. The principal component of the infectious agent responsible for the disease appears to be an abnormal isoform of the host encoded prion protein (PrP), designated "PrPsc". The emergence of variant Creutzfeldt Jakob Disease (vCJD) in humans in 1996 in UK and its causal links with BSE has highlighted the need for comprehensive study on its pathogenesis, diagnosis, prevention and eradication approaches. To control BSE within a country or to prevent the entry of BSE into a country, EU, USDA and few other countries have taken emergency measures in prevention, education, surveillance, and exportation of bovine origin materials (feeds, tallow, gelatin, etc) into these countries need BSE surveillance certificate. Bangladesh has no BSE surveillance so far. In this study, preliminary slaughterhouse surveillance is conducting in two districts, Dhaka and Mymensingh, in Bangladesh. Around 1000 brain samples from Cattle older than 30 months of age, slaughtered for human consumption in the district slaughterhouses, were collected for this study. The brainstems (obex), pyriform lobe, pieces of cerebellum and cerebrum were subjected for Histopathology, immunohistochemistry and ELISA using an anti-PrP monoclonal antibodies 6H4 (prionic AG, Switzerland) specifically reacting with epitopes on ruminant PrPsc and commercial immunoperoxidase and ELISA kit. Although some of the brainstems showed mild gliosis and inflammation, but none of the brainstems exhibited characteristic histopathologic lesions as found for BSE. No PrPsc was detected on these brainstems using immuohistochemistry and ELISA. From this study it apparently seems that BSE is not present in the native cattle in Bangladesh. Still samples are being collected from different regions and are analysing using above-mentioned methods.

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MALIGNANT PERIPHERAL NERVE SHEATH TUMOUR IN A DOG

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Materials and Methods

The Siberian husky, male, 13 years old, started limping on left thoracic limb one year ego, and lameness increased during time. On MRI without contrast,

suspected diagnosis was neoplasia of brachial plexus peripheral nerves. Following euthanasia and postmortal examination tissue was taken for histological examination.

The tissue was routinely processed and stained with hematoxylin and eosin.

Immunocytochemical staining was done by use of a commercial avidin-biotin peroxidase complex kit. Antibodies for S-100 protein, glial fibrillary acidic protein(GFAP), cytokeratins AE1/AE3 and vimentin were tested. Diaminobenzidine (DAB) was used as the chromogen and Mayer's hematoxylin was used as the counterstain.

Results

Macroscopically, the tumour mass (6x8 cm) was located on the left axillar region closely connected with nerves of the brachial plexus, was nonencapsulated and infiltrated surrounding tissue. On section the tumour was solid with central core of haemorrhages and necrosis.

Histologically, neoplasm was infiltrative and poorly circumscribed. The cellularity and pattern of arrangement varied. In the more cellular areas, the spindle cells were arranged in fascicles, whorls, or sheets. The neoplastic cells had hyperchromatic oval to elipsoid nuclei with one or two prominent nucleoli, and fine, tapering, eosinophilic cytoplasm. The cell borders were indistinct. There were two to five mitotic figures per high-power field. Necrotic foci and hemorrhages comprised large areas of neoplastic tissue. In some nerves infiltrating neoplastic cells were seen in the endoneurium between apparently normal axons.

Immunohistochemically most spindle cells were positive for vimentin and negative for S-100, keratin and GFAP.

Conclusion

Based on the morphological and immunohistochemical features, the tumour was classified as a malignant peripheral nerve sheath tumour(MPNST) with dominant mesenchymal component.

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MORPHOLOGICAL ALTERATIONS IN OXYDATIVE MUSCLES ASSOCIATED WITH EQUINE ATYPICAL MYOPATHY

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Thirty-two 0.5- to 7-year-old horses kept on pasture were referred for medical and necropsic evaluation of a sudden ataxia/myoglobinuria syndrome. Clinical examination and plasma CPK, LDH and AST levels were consistent with