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SHORT PAPER

Diffuse Intracranial Oligodendroglioma in a Cow

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Summary

This report describes the clinical, morphological and immunohistochemical findings in an adult cow with cerebral oligodendroglioma. The tumour extended from the metencephalon to the telencephalon, with infiltration of the meninges. Immunohistochemically, the tumour cells lacked expression of a specific tumour antigen, were partially positive for S-100 and tau protein, and were negative for a range of antigens including glial fibrillary acid protein, neuron-specific enolase, myelin basic protein and synaptophysin. This is the first report describing a diffuse, cerebral oligodendroglioma in a cow.

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Tumours of neuroepithelial origin are most often found in dogs and cats. They are mainly composed of a mixture of glial cells, and the diagnosis is based on the predominant cell type present (Koestner *et al.*, 1999). Oligodendrogliomas are common tumours of the central nervous system of dogs, but only two cases, both spinal tumours, have been reported in cattle (Baker and Kippax, 1980; Uchida *et al.*, 1999). Comparable to human patients, oligodendroglioma in dogs and cats arise predominantly in the cerebral hemispheres and subcortical white matter, but may also occur in the basal nuclei (Koestner *et al.*, 1999; Dickinson *et al.*, 2000; Snyder *et al.*, 2006; Reifengerger *et al.*, 2007). Patients with cerebellar, brain stem, spinal cord and primary leptomeningeal oligodendrogliomas have also been reported (Mamom *et al.*, 2004; Snyder *et al.*, 2006; Reifengerger *et al.*, 2007). Oligodendrogliomas tend to infiltrate the overlying leptomeninges. Macroscopically they appear as red, pink-red or grey-pink, circumscribed or diffuse masses with either a solid or soft gelatinous cut surface (Koestner *et al.*, 1999; Reifengerger *et al.*, 2007). Additionally, calcification, cystic degeneration

or intratumoural haemorrhages may occur (Koestner *et al.*, 1999; Koestner and Higgins, 2002; Reifengerger *et al.*, 2007). In humans, neurological signs include epileptic seizures and headache, whereas in dogs seizures and mental changes are reported as predominant clinical findings (Snyder *et al.*, 2006; Reifengerger *et al.*, 2007). The nature of the neurological signs depends upon the location of the tumour and the two reported cases of spinal oligodendrogliomas in cattle were characterized by fore and hind limb paralyses or dysfunction, or difficulty in raising the head (Baker and Kippax, 1980; Uchida *et al.*, 1999). This report describes the clinical, morphological and immunohistochemical findings in an adult cow with cerebral oligodendroglioma.

A Simmenthaler crossbred cow, aged 6 years and 8 months, developed mild behavioural changes (altered locomotion, stumbling) accompanied by loss of body condition over a two-week period. The cow had normal feed and water intake, but general ataxia associated with temporary paraparesis was present. Neurological examination demonstrated a decreased menace reflex and a flaccid tail. The tone of the tongue and lips and the functions of the trigeminal and facial nerves appeared normal. Haematological abnormalities were not observed, but serum biochemical examination revealed mild hypocalcaemia

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(2.01 mmol/l; reference range, 2.1–3.0 mmol/l) and mild selenium deficiency (56 µg/l; normal, >70 µg/l). Cerebrospinal fluid collected by lumbosacral puncture was colourless and clear. The cell count, total protein concentration and glucose concentration were within physiological ranges.

The animal was initially treated with glucocorticoid (0.4 mg/kg Dexamethason®; CP-Pharma, Burgdorf, Germany) and antibiotics twice daily (15 mg/kg Ampicillin®; CP-Pharma) together with daily administration of thiamine (1.5 g Thiasel®; Selectavet, Weyarn-Holzolling, Germany). As there was no improvement of clinical signs after five days of intensive therapy, the animal was humanely killed and submitted for necropsy examination.

Gross examination revealed diffuse thickening of the meninges (up to 0.3 cm) with ochre to yellow discolouration of the pons (metencephalon), crus cerebri (mesencephalon), corpora geniculata (diencephalon), trigonum olfactorium and lobus piriformis (telencephalon) (Fig. 1). Up to 0.4 cm of the adjacent parenchyma displayed the same discolouration. A severe multifocal to coalescing, ulcerative dermatitis was present in the pastern region of all limbs, but the remaining tissues and organs were normal.

Tissue samples were collected from the major parenchymatous organs, central nervous system, musculature and joints, fixed in 10% neutral buffered formalin and embedded in paraffin wax. Tissue sections (4 µm) were stained with haematoxylin and eosin (HE). Microscopical examination of the affected areas of the brain revealed the presence of a diffuse, non-encapsulated, highly cellular neoplasm with infiltration of the meninges (Fig. 2). Cells were arranged in closely-packed sheets with sparse vascularization. The neoplastic cells were round to oval in shape, 8–12 µm in diameter, with indistinct cell borders and a moderate quantity of pale eosinophilic homogenous cytoplasm. Occasionally, there was

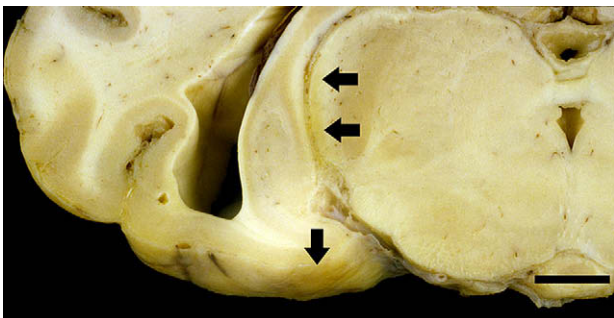


Fig. 1. Coronal-section of the ventrolateral part of the hippocampus region (telencephalon) and corpora geniculata (diencephalon). There is ochre to yellow discolouration of the meninges and adjacent neuroparenchyma (arrows). Bar, 2 cm.

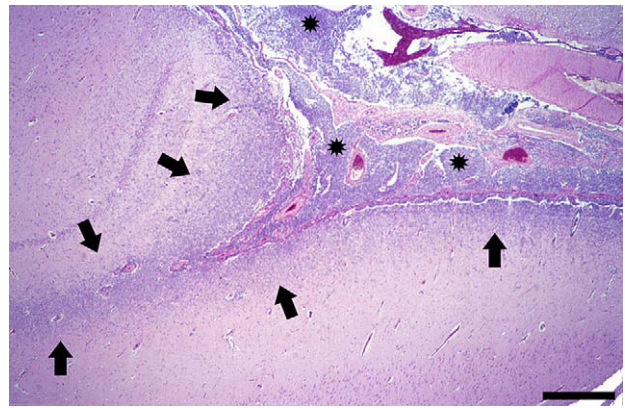


Fig. 2. A highly cellular oligodendroglioma diffusely infiltrates the brain and leptomeninges (asterisks). Arrows indicate the margin between normal parenchyma and the tumour. HE. Bar, 175 µm.

a small clear perinuclear halo (Fig. 3). The nuclei were centrally located and had a small quantity of coarse heterochromatin and a basophilic nucleolus. Mild anisokaryosis was present and up to two mitoses per high-power field (HPF; ×40 microscope objective) were detected. Microcystic areas containing pale, eosinophilic material were also noted. Small amounts of blue pericellular material were observed in these areas.

Serial sections from selected tissues were subject to immunohistochemistry (IHC). IHC was performed with a panel of murine monoclonal or rabbit polyclonal antibodies (all from Dako, Glostrup, Denmark unless otherwise stated) specific for the following molecules: vimentin (1 in 100, clone V9), CD3 (1 in 1000, polyclonal), CD79a (1 in 60, clone HM57), lysozyme (1 in 250, polyclonal), myelohistiocytic antigen (1 in 200, clone MAC387), melan-A (1 in 600, clone

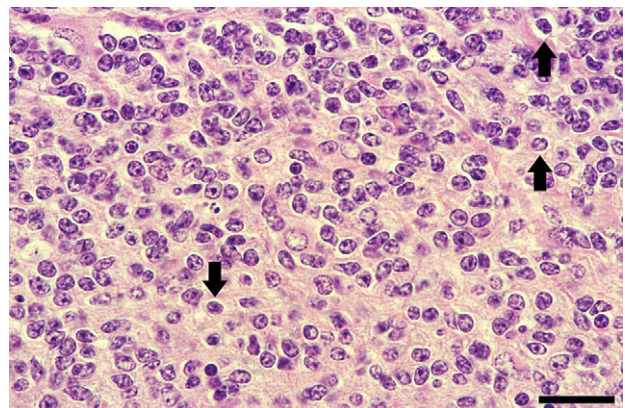


Fig. 3. Neoplastic cells are round to oval in shape with indistinct cell borders and a moderate amount of pale eosinophilic homogenous cytoplasm. Occasionally, a small clear perinuclear halo is present (arrows). HE. Bar, 28 µm.

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