MRI findings in a young dog with gliomatosis cerebri

A 3-year-old male entire boxer presented with a 3-week history of lethargy, hyporexia, hypersalivation, low-head carriage, pelvic limb tremors and intermittent vestibular ataxia. Neurological examination revealed kyphosis, wide-based stance, an alternating head tilt, left leaning, bilateral positional ventral strabismus and intermittent vertical nystagmus. Exacerbation of neurological signs and increased extensor tone in all limbs were noticed after dorsal extension of the neck. Neuroanatomical localisation was cerebellum and/or brainstem. Haematology, biochemistry and blood pressure were within normal limits. Serology for common infectious diseases was negative. MRI demonstrated non-enhancing mild T2W and FLAIR hyperintensity and swelling of the whole cerebellum with more marked hyperintensity on the ventrolateral left hemisphere/flocculus (Fig. 1A,B,C). Increased intracranial pressure was suspected based on rostral transtentorial and foramen magnum herniation, cervical syringomyelia and lateral ventricular dilation, and therefore cerebrospinal fluid was not collected. Thoracic and abdominal CT was unremarkable. Differential diagnoses included inflammation (meningoencephalitis of unknown origin [MUO]), protozoal cerebellitis) or neoplasia (glioma/gliomatosis cerebri, lymphoma). MUO was considered likely; immunosuppressive prednisolone and mycophenolate and prophylactic clindamycin were started. After mild improvement, signs were static for 5 months. Mycophenolate was discontinued at 3 months due to gastrointestinal signs; owner declined another chemotherapeutic and prednisolone was continued. Two months later, the dog deteriorated (intention tremors, ataxia, intermittent collapses), with cerebellar changes being static on repeat MRI (Fig. 1D,E,F). Intravenous cytarabine was tried without response. Owners elected euthanasia. Histopathology revealed a disseminated proliferation of presumed glial origin infiltrating the subpial region, molecular layer, nuclei and white matter of the cerebellum bilaterally, the cerebrum, medulla, midbrain, hippocampus and medial geniculate bodies, consistent with gliomatosis cerebri (type 1).

This case demonstrates a rare pattern of gliomatosis cerebri predominantly affecting the cerebellum. There was little progression in the imaging changes over a 5-month period, with clinical stability upon treatment.

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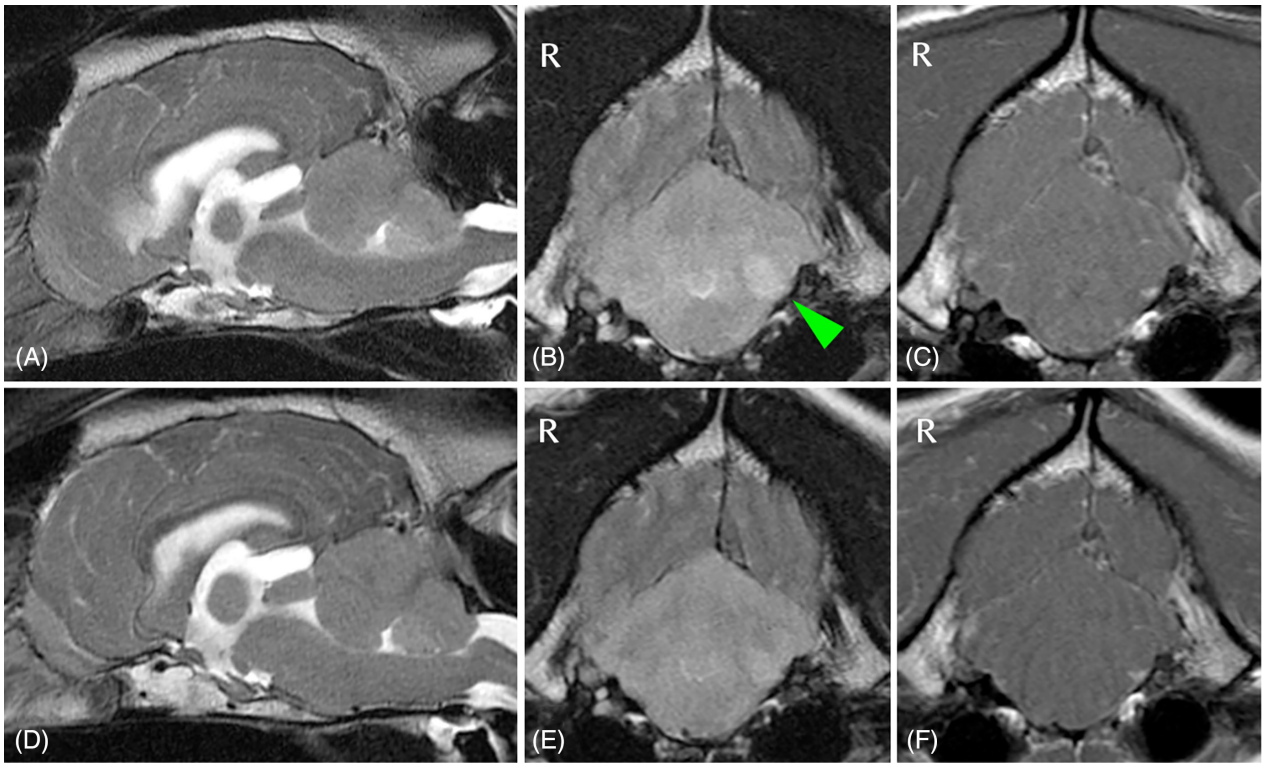

FIG 1. (A–C) MRI at the time of presentation of a dog diagnosed with gliomatosis cerebri: (A) T2-weighted sagittal sequence showing diffuse T2W hyperintensity at the cerebellum and swelling characterised by the absence of the cerebellar sulci and the loss of differentiation between grey and white matter. (B) T2-weighted transverse sequence showing diffuse T2W hyperintensity of the cerebellum more evident on the ventrolateral left hemisphere/flocculus (green arrowhead). (C) T1-weighted post-contrast transverse sequence showing no contrast uptake of the cerebellum. (D–F) T2-weighted sagittal (D), T2-weighted transverse (E) and T1-weighted post-contrast (F) sequences at a 5-month follow-up MRI showing no change

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