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CASE REPORT

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Intracranial medulloblastoma as the cause of progressive ataxia in a 6-month-old draft horse cross gelding

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Abstract

We describe the unique clinical presentation of a central nervous system neoplasm in a 6-month-old draft horse cross gelding. Based on the neurologic examination at admission, neurolocalization was most consistent with a mildly asymmetric cervical, multifocal, or diffuse myelopathy. Mild vestibular involvement also was considered, but no cranial nerve deficits were observed. The gelding was negative for *Sarcocystis neurona* or *Neospora hughesi* based on paired serum and cerebrospinal fluid (CSF) samples analyzed, with no evidence of cervical compression based on contrast myelography. The horse was euthanized because of progression of clinical signs. At necropsy, a mass was identified associated with the cerebellum, and histopathology was consistent with medulloblastoma, which has not been reported previously in the horse.

KEYWORDS
ataxia, brainstem, cerebellum, equine, intracranial, neoplasia

1 | CASE DESCRIPTION

A 6-month-old draft horse cross gelding weighing approximately 375 kg was presented for evaluation of worsening ataxia after being unable to rise without assistance the morning of presentation. Neurologic clinical signs began 1 week before presentation. A standing castration had been attempted under IV sedation with 5 mg of detomidine and 3 mg of butorphanol, which caused the patient to sit and lie down in left lateral recumbency. Castration was completed and the horse remained down for 1 hour before rising without difficulty. The gelding subsequently was noted to be ataxic with difficulty rising from left lateral recumbency. Before referral, the horse had received 500 mg of flunixin meglumine PO.

On presentation, the gelding was quiet, alert, responsive and standing. Heart rate was 44 beats per minute, respiratory rate 32 breaths per minute, and temperature 100.4°F. Point-of-care hematology including venous blood gases, packed cell volume, total solids, and peripheral blood lactate concentration indicated no abnormalities. The patient had a normal serum amyloid A concentration of 0 μg/mL, mildly increased plasma fibrinogen concentration of 462 mg/dL (likely secondary to recent castration), and a normal white blood cell count.

A complete neurologic examination was performed. Behavior was considered normal with a mildly quiet mentation for a young horse. No muscle atrophy or asymmetry was noted. No sores were present from prolonged recumbency. No cranial nerve deficits were noted with appropriate tongue muscle tone. Both cervicofacial and cutaneous trunci reflexes were intact and normal. The patient exhibited appropriate lateral cervical flexion to the left with inconsistent lateral cervical flexion to the right and decreased dorsoventral cervical flexion. All 4 toes were worn with scuff marks present. At a walk in a straight line on hard ground, the patient exhibited a wide-based

Abbreviations: ARAS, ascending reticular activating system; CNS, central nervous system; CSF, cerebrospinal fluid; CT, computed tomography; CVSM, cervical vertebral stenotic myelopathy; EDM, equine degenerative myeloencephalopathy; eNAD, equine neuroaxonal dystrophy; EPM, equine protozoal myeloencephalitis; MRI, magnetic resonance imaging; UMN, upper motor neuron.

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stance in the pelvic limbs with a narrow-based stance in the thoracic limbs. Proprioceptive deficits were observed, characterized by delayed protraction, frequent dragging or scuffing of all 4 feet, forelimb spasticity with a prolonged searching phase, wide-based irregular pelvic limb foot placement, and frequent pelvic limb interference. The gelding consistently drifted to the left throughout the examination with inappropriate crossover and stumbling in the pelvic limbs. Marked asymmetric pelvic limb paresis was present and the gelding was easily pulled to the side with a light tail pull to the left. Neurologic deficits were exacerbated when the gelding was walked with an elevated head, circled in either direction, or walked backwards. Examination on a hill was not performed because of the severity of clinical signs. Based on the neurologic examination, the gelding was diagnosed with grade 4 of 5 proprioceptive ataxia using the modified Mayhew scale[1] with left pelvic limb paresis most consistent with a mildly asymmetric cervical, multifocal, or diffuse myelopathy. Mild vestibular involvement also was considered based on the tendency to drift to the left, but no other signs of vestibular disease were detected.

The gelding appeared comfortable in recumbency on either side, but persistently displayed an inability to rise from left sternal recumbency. When assisted to rotate into right sternal recumbency, the gelding was able to stand without assistance. Muscle and liver enzyme activities were evaluated to assess for possible myopathy or metabolic myelopathy or encephalopathy contributing to clinical signs, with no abnormalities noted. Differential diagnoses to explain the gelding's clinical signs at this time included cervical vertebral stenotic myelopathy (CVSM), cervical trauma, equine protozoal myeloencephalitis (EPM), equine degenerative myeloencephalopathy (EDM)/equine neuroaxonal dystrophy (eNAD), spinal abscess, neoplasia, or less likely viral etiologies. Flunixin meglumine (Prevail, VetOne, Boise, Idaho; 1.1 mg/kg PO q12h) was continued as an anti-inflammatory and analgesic agent.

On the 2nd day of hospitalization, survey right-to-left lateral cervical vertebral radiographs were obtained. No abnormalities were detected, and cervical myelography was recommended. Pretreatment with dexamethasone (Phoenix, Burlingame, California; 0.05 mg/kg IV once) was administered. The gelding underwent general anesthesia and contrast cervical myelography was performed by atlanto-occipital approach to allow cerebrospinal fluid (CSF) centesis from the cisterna magna and injection of 40 mL of iohexol (Omnipaque, GE Healthcare, Chicago, Illinois; 300 mgI/mL) into the subarachnoid space. Left-to-right lateral radiographic views from the occiput to the 2nd thoracic vertebra were obtained in neutral, flexed, and extended neck positions. The study was considered of good diagnostic quality with no evidence of dorsoventral spinal cord compression or suspicion of lateral cord compression. The CSF obtained was xanthochromic with a normal white blood cell count of 1 cell/μL (reference range, 0-5 cells/μL), increased total protein concentration of 120 mg/dL (reference range, 20-80 mg/dL), and an increased red blood cell count of 4250 cells/μL (reference range, 0 cells/μL), which could be consistent with trauma or possible blood contamination. Cerebrospinal fluid cytology indicated no abnormalities beyond albuminocytologic dissociation. The gelding did not have evidence of intrathecal antibody production against *Sarcocystis neurona* or *Neospora hughesi* based on paired serum and CSF samples analyzed using SnSAG 2,3/4 ELISA and NhSAG 1 ELISA (Equine Diagnostic Solutions LLC, Lexington, Kentucky). Serum vitamin E concentration was 2.9 ppm (reference range, 2-10 ppm). Whole blood and nasal swabs were negative for Equine Herpesvirus 1 by PCR.

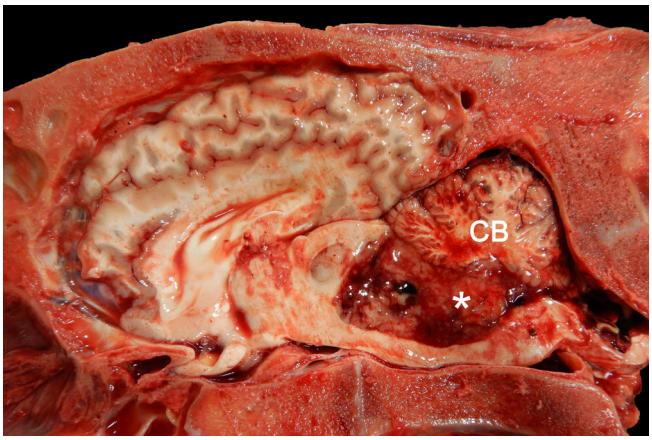

FIGURE 1 Parasagittal section of the head. A pink-gray mass (asterisk) extends from the ventral aspect of the cerebellum (CB) into the subjacent 4th ventricle and pons

Because of the degree of ataxia, an assisted sling recovery was performed after prolonged recovery from anesthesia without attempts to stand. The patient was able to stand with assistance and subsequently ambulate unassisted, but stumbled and fell while walking back to the stall, necessitating a 2nd induction under general anesthesia to allow safe transport to the stall on a rescue glide. After recovering from anesthesia in the stall, the gelding rose without complication or assistance from the right side. Empirical treatment for EPM was started using ponazuril (Marquis, Boehringer Ingelheim, Ridgefield, CT) with a loading dose of 15 mg/kg PO followed by 5 mg/kg PO q24h. Minocycline (Zydus Pharmaceuticals, Pennington, New Jersey; 4 mg/kg PO q12h) was started as an antimicrobial to treat potential bacterial causes of neurologic disease. Dexamethasone was continued at a higher dosage of 0.1 mg/kg IV q24h. Vitamin E (Elevate W. S. Vitamin E, Kentucky performance products, Versailles, Kentucky; 10 IU/kg PO q24h) supplementation also was initiated.

The gelding began to show increasing weakness and difficulty rising throughout the 3rd day of hospitalization, requiring multiple attempts to rise from right lateral recumbency, and was noted to stand for shorter amounts of time. By the 4th day of hospitalization, the patient was unable to stand without assistance, with a new inability to rise from right sternal recumbency, similar to the left side. A sling was used to support the gelding because of marked pelvic limb paresis and inability to bear weight. The EPM results returned negative, decreasing the likelihood of a treatable neurologic condition. Because of the patient's continued clinical decline and poor prognosis for recovery, euthanasia was elected and necropsy performed. A soft pink-gray mass extended from the ventral cerebellum into the pons and 4th ventricle (Figure 1) with accompanying ventricular dilatation and foci of hemorrhage. Microscopically, the cerebellar mass

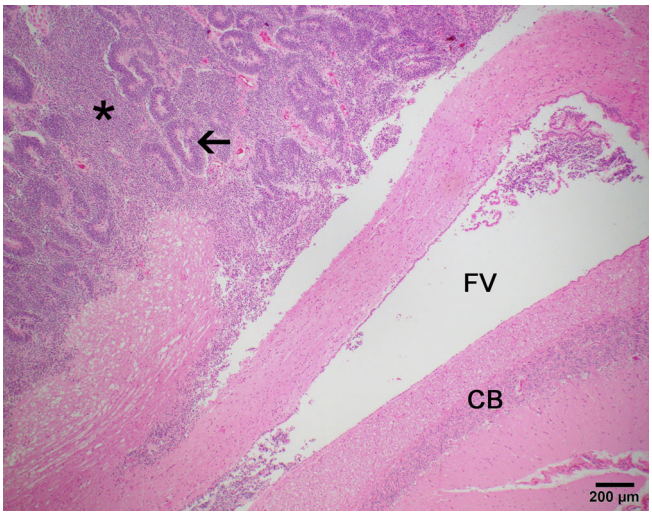

FIGURE 2 Photomicrograph of the mass with adjacent 4th ventricle (FV) and cerebellum (CB). The mass comprises small round to polygonal cells arranged in sheets (asterisk) and palisading streams/rosettes (arrow). Hematoxylin and eosin staining. Scale bar = 200 µm

comprised a proliferation of small round to polygonal cells arranged in sheets, palisading streams, and rosettes (Figure 2), features consistent with a diagnosis of medulloblastoma. No clinically relevant abnormalities were observed in the spinal cord or remainder of the body.

2 | DISCUSSION

To our knowledge, this report is the 1st of an intracranial medulloblastoma in a horse. Medulloblastoma is an embryonal primary central nervous system (CNS) tumor, previously classified as a primitive neuroectodermal tumor.[2,3] Medulloblastomas are the most common brain tumor and most reported posterior fossa neoplasm in children, making up approximately 15% to 20% of all pediatric tumors and nearly 40% of posterior fossa tumors across all age groups.[2] In children, medulloblastomas are diagnosed at an average age of 6 years.[2,4] Medulloblastomas have been reported rarely in veterinary medicine with bovine, canine, and feline cases recounted. Onset of clinical signs often occurs early in life, but age at onset varies. In calves, diagnosis has been described between 1 and 6 months of age.[5] In small animals, affected animals are older, with 2-year-old feline and canine patients reported[6,7] and a few reports in middle-aged dogs ranging from 4 to 7 years of age at diagnosis.[8-10]

In the documented cases in cattle, dogs, and cats, ataxia (vestibular and cerebellar) and cranial nerve deficits are common clinical findings, but signs vary among species and individuals.[5-11] Clinical signs in calves with medulloblastomas were most consistent with cerebellar ataxia.[5] More diverse clinical presentations are reported in small animals including vestibular[6-10] and cerebellar ataxia,[9,10] loss of balance,[8,9] decreased mentation,[7,11] collapse,[7] head tilt,[7,8,11] torticollis,[7] nystagmus,[9] intention tremors,[10] variation in facial sensation,[9] absent menace response,[10] intermittent opisthotonos,[8] and anorexia.[6] Clinical signs associated with an increase in intracranial pressure and subsequent Cushing's reflex were reported in an affected dog.[8] Neurolocalization in each of these cases was most consistent with central vestibular, cerebellar, or brainstem dysfunction.[2,4,7-11] Children with medulloblastomas often show nonspecific symptoms such as vomiting and headaches before onset of neurologic deficits, leading to a delay in diagnosis of 2 to 5 months.[4,12-15] Neurologic clinical signs including cerebellar ataxia, changes in consciousness or cranial nerve deficits were found to occur late in comparison to vague, nonspecific symptoms in over half of the cases (57%) or not at all in 24% of cases. Ataxia was reported as the 1st symptom in only 10% of cases, often presenting later in the clinical progression.[4]

In cats and dogs, definitive diagnosis of a brain tumor has been made using magnetic resonance imaging (MRI) and subsequent histopathology at the time of surgical removal,[6,8] with computed tomography (CT) being performed as an adjunctive diagnostic test to rule out metastasis.[8] In other cases, a presumptive diagnosis of neoplasia has been made using MRI with necropsy providing a definitive diagnosis.[7,9] In some cases that utilized CT and MRI, brainstem compression and infiltration into the 4th ventricle by the mass were identified, findings similar to those observed at necropsy in this gelding.[7,8] Because of the frequent rate of occurrence in humans, a presumptive diagnosis often is made based on clinical signs and imaging, with MRI being the modality of choice. Cerebrospinal fluid analysis has assisted in staging of the neoplasm in children, with recent studies demonstrating that approximately 45% of CSF cytologic examinations show characteristics of malignancy such as enlarged cells, high nuclear-to-cytoplasmic ratio, and pleomorphism.[2,16,17] Histopathology remains crucial to definitive diagnosis, as in our case.[2,18]

Treatment is often multimodal in approach but generally yields poor outcomes in veterinary species. Complete resection of the mass was performed in the affected cat but did not substantially affect prognosis, providing only a few additional weeks of survival.[6] In a dog, incomplete margins were obtained because ventral deviation of the mass compressed the brainstem ultimately resulting in euthanasia 5.5 months postoperatively because of patient decompensation.[8] In human medicine a combination of surgical intervention and chemotherapy using lomustine, vincristine, and procarbazine in addition to prednisolone has been used with better outcomes in cases diagnosed in children older than 3 years, in the absence of disseminated disease, with approximately 80% surviving at least 4 years after treatment.[17]

Brain tumors in horses remain rarely reported and poorly understood. Both primary and secondary neoplasms of the CNS are documented in case reports, with the incidence in the horse unknown. Primary nervous system neoplasms reported in the horse include ependymomas,[19-21] glioblastoma,[22] ganglioglioma,[23] astrocytoma,[24] meningioma,[24] pituitary adenomas,[24] pineoblastoma,[25] medulloepithelioma,[26,27] neuroendocrine tumors,[28] oligodendroglioma,[29] and Schwannoma[30] with various secondary neoplasms including adenocarcinoma,[24,29] hemangiosarcoma,[31] melanoma,[32] carcinoma,[33] and lymphoma.[34,35]

Our case is unique not only in the type of brain tumor, but in the clinical presentation. The gelding's mass was located within the caudal fossa, dorsal to the brainstem and ventral to the cerebellum. Space-occupying masses in this location typically would cause a constellation of clinical signs referable to the brainstem or cerebellum or both. Clinical signs from brainstem disease include cranial nerve deficits (e.g., anisocoria, facial asymmetry, dysphagia) and, with cranial nerve VIII involvement, vestibular ataxia.[36,37] The caudal brainstem (myelencephalon) is important to both pelvic and thoracic limb movement, including generation of movement and proprioceptive control, and dysfunction can cause loss of proprioception as well as upper motor neuron (UMN) paresis. Finally, dysfunction of the ascending reticular activating system (ARAS) within the brainstem can cause dullness, obtundation, or stupor. The hallmarks of cerebellar dysfunction include loss of coordination of rate, range and force of motion, which leads to characteristic cerebellar ataxia including a dysmetric gait, truncal sway, and tremors that might worsen with intention.[36,37] Interestingly, this gelding displayed clinical signs most consistent with a cervical, multifocal, or diffuse myelopathy without caudal fossa involvement. This discrepancy is likely associated with the precise location of the mass, which appeared to predominantly affect brainstem nuclei and tracts involved in gait generation and proprioception while sparing the cranial nerves, nuclei, and cerebellum. This type of presentation (i.e., solely ataxia, without cranial nerve deficits) is rare in humans with medulloblastoma.[2,12-17] In retrospect, the drifting to the left in this gelding was likely secondary to mild vestibular disease, although severe paresis as a contributing factor could not be ruled out during clinical examination, particularly in the absence of other compelling signs of vestibular disease such as head tilt, strabismus, or abnormal nystagmus.

In summary, we describe a unique clinical presentation of a CNS neoplasm not previously reported in horses. The case also highlights the challenges of reaching an accurate antemortem diagnosis in this species. Advanced imaging options are limited compared to what is available for small animal species, and maneuvers performed in small animals to induce signs of vestibular dysfunction, such as rolling the animal onto its back and holding it there to induce abnormal nystagmus, are not possible. Without compelling clinical signs of intracranial disease, the proprioceptive ataxia and paresis displayed by this gelding were assumed to represent spinal cord dysfunction. The most common causes of quadrupedal ataxia and paresis in young horses include CVSM, cervical trauma, EDM/eNAD, and infectious diseases such as EPM, bacterial discospondylitis, or spinal abscesses. Therefore, cervical imaging studies including radiography, cervical myelography, as well as CSF analysis were pursued. When these tests failed to provide a diagnosis or even cytological abnormalities, empirical treatment for remaining differential diagnoses was pursued rather than reconsideration of potential neurolocalization and additional imaging of the brain. In part, the absence of an MRI system capable of imaging the equine brain at our facility contributed to this decision. After diagnostic testing excluded CVSM and other structural lesions within the cervical region as well as EPM, the remaining differential diagnoses in this case remained EDM/eNAD, spinal cord trauma, or neoplasia. The only differential diagnosis with a possible favorable prognosis was previous trauma, which was not likely given that the patient's clinical status worsened over time. Therefore, euthanasia was elected, with necropsy yielding a definitive diagnosis. Although intracranial CNS tumors are uncommonly reported in the horse, they should remain a differential diagnosis in cases of progressive or persistent neurologic disease, even when myelopathies initially are suspected. Continued improvements in imaging equipment and availability eventually will allow improved antemortem diagnosis in affected horses.

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CONFLICT OF INTEREST DECLARATION

Authors declare no conflict of interest.

OFF-LABEL ANTIMICROBIAL DECLARATION

Authors declare no off-label use of antimicrobials.

INSTITUTIONAL ANIMAL CARE AND USE COMMITTEE (IACUC) OR OTHER APPROVAL DECLARATION

Authors declare no IACUC or other approval was needed. Owner approved writing of the report.

HUMAN ETHICS APPROVAL DECLARATION

Authors declare human ethics approval was not needed for this study.

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