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CASE REPORT

Complex central nervous system malformations in a Dutch Warmblood foal

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Abstract

A neonatal Dutch Warmblood colt was evaluated for inability to stand, incoordination and intention tremor. Despite partial improvement in clinical signs during the first 4 days of hospitalization, neurological deficits remained. Magnetic resonance imaging identified a unilateral infratentorial arachnoid cyst-like lesion with ipsilateral compression and displacement of the cerebellar hemisphere, absent corpus collosum, polymicrogyria, suspect leukoencephalopathy, and noncompressive occipitoatlantal malformation. Improvement in clinical signs during the first 6 months of life suggests that horses can survive with complex congenital neurological malformations, but prognosis for athletic performance is poor. The accessibility of magnetic resonance imaging should improve the diagnostic accuracy of central nervous system disorders in neonatal foals in which congenital malformations are suspected. Euthanasia often is elected in foals with suspected congenital central nervous system disorders because of poor prognosis for athletic performance, limiting understanding of clinical progression in these cases.

KEYWORDS
cerebellum, cerebrum, congenital neurological anomaly, craniocervical junction

1 | CASE DESCRIPTION

A 3-hour old approximately 50 kg Dutch Warmblood colt was presented for evaluation of inability to rise, incoordination and intention tremor. The colt was born to a multiparous mare at 352 days gestation after an uncomplicated pregnancy. A full sibling to the foal was born 1 year previously with no abnormalities. The dam had no history of illness during pregnancy and was vaccinated at 5, 7, 9 months (Pneumabort K1 + 1b, Zoetis), and 10 months gestation (Vetera EWT + WNV, Boehringer Ingeleim). The mare was treated for gastrointestinal parasites at 10 months gestation (Zimectrin, Merial). Parturition was unassisted, but observed, and second stage labor was noted to last <30 minutes. The placenta was normal. The colt was unable to stand and nurse on the farm, prompting referral. Upon arrival at the referral hospital, the colt was recumbent but rousable and had a consistent head tremor. Physical examination identified dull mentation, mild dehydration, a strong suckle and was suspicious for central blindness based on intact pupillary light reflexes (PLR), absent dazzle reflex and menace response, and inability to track objects or respond to changes in light, but vision was challenging to assess because the menace response is expected to be absent at this age. Moderate kyphosis of the thoracolumbar vertebral column was noted. Cranial nerves and spinal reflexes including cervicofacial and cutaneous

Abbreviations: MRI, magnetic resonance imaging; OAM, occipitoatlantal malformation.

trunci were within normal limits. Postural reflexes were not tested. No anatomical abnormalities of the cranium were apparent. The foal was unable to place himself or remain in sternal recumbency without assistance. Laboratory testing disclosed mild neutrophilia at 7430/μL (reference range, 2180-6960/μL), mild increase in serum creatinine concentration at 2.6 mg/dL (reference range, 0.7-1.8 mg/dL), and mildly increased creatine kinase activity at 511 U/L (reference range, 50-400 U/L). All other laboratory test results were within normal limits for this foal's age.

Treatment consisted of enteral feeding colostrum, urinary bladder catheterization, oxygen supplementation via nasal cannula, and IV crystalloid fluid and plasma therapy. Antibiotics were initiated consisting of ceftiofur (5 mg/kg IV q6h) and potassium penicillin (22 000 IU/kg IV q6h). At approximately 24 hours of age, mild seizures were noted consisting of bilateral rapid eye movement and rhythmic chewing motion. A single dose of diazepam (0.1 mg/kg) was administered with no change and a midazolam constant rate infusion (CRI; 0.04 mg/kg/hr) was initiated. The midazolam CRI was incrementally increased up to 0.1 mg/kg/hr over the course of 8 hours with no improvement in seizure activity. Concurrently, mannitol (0.5 g/kg IV q6h for 4 doses) was administered. The CRI was discontinued and a single dose of levetiracetam (32 mg/kg IV) was administered, which improved the signs of mild seizure activity. Orally administered levetiracetam (32 mg/kg PO q12h) was continued and controlled seizure activity completely. Intragastric feedings were continued at approximately 5% body weight and were well tolerated. When the feeding rate was increased, gastric reflux occurred and partial parenteral nutrition supplementation consisting of lipids, dextrose and amino acids was initiated at half of resting energy requirements (2500 kcal/d). Physical rehabilitation consisting of passive range of motion and assistance to sit sternal was initiated, and the colt showed improvement in mental status and ability to maintain sternal recumbency. Despite decreased seizure activity, fine head tremor persisted and worsened when the colt was in sternal recumbency. At 96 hours of age, the foal was able to remain in sternal recumbency without assistance and could stand with assistance. The colt was unable to remain standing without continued assistance and was noted to have a marked head tremor and truncal swaying when standing (Video S1). It also had an inconsistent left head tilt and leaned to the left while standing.

Because of persistent neurologic abnormalities despite intensive care and control of seizures, imaging of the central nervous system was performed. Radiographs of the skull, cervical, thoracic, and lumbar vertebral column identified mild kyphosis of the thoracolumbar vertebrae of unknown clinical relevance. Occipitoatlantal malformation (OAM) also was noted with partial fusion of the right occipitoatlantal bones and rounding of the right side of the atlas caudally. The axis appeared within normal limits (Figure 1).

High-field magnetic resonance imaging (MRI) of the head was performed (3T, Siemens Skyra, Malvern, Pennsylvania) and T2 total spin echo (TSE) sagittal and T2 TSE, T1 TSE and T2 fluid-attenuated inversion recovery (FLAIR) transverse images were obtained. While anesthetized for the MRI, the foal became hypercapnic, and the study was terminated before IV administration of contrast agent or further sequence acquisition, and the foal was recovered from anesthesia. Magnetic resonance

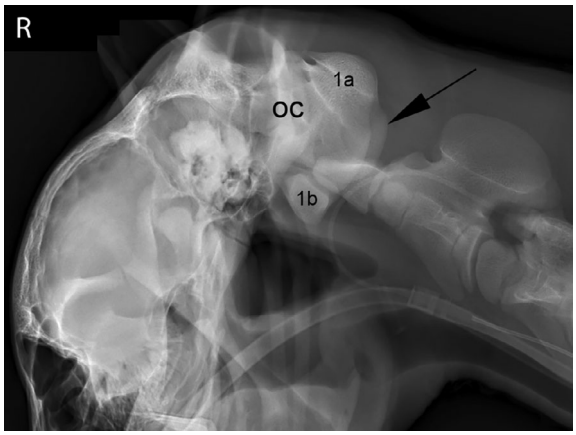

FIGURE 1 Right lateral radiograph showing occipitoatlantal malformation. The occipital (oc) bone is malformed (it does not form well-defined occipital condyles) and it is at least partially fused with the atlas (note that no occipitoatlantal joint space is seen clearly). The dorsal (1a) and ventral (1b) arches of the atlas are shown. The caudal margin of the atlas (arrow) is somewhat rounded resembling occipital condyles. The axis appears within normal limits. A nasogastric feeding tube and jugular IV catheter summate with the throat and neck

imaging identified a roughly spherical, medium-sized (3.1 cm × 2.0 cm), space-occupying lesion in the infratentorial space between the left cerebellar hemisphere and occipital bone. The lesion compressed the left cerebellar hemisphere and displaced it to the right decreasing the subarachnoid space between the vermis and left cerebellar hemisphere, and rostrally with ipsilateral rostral tentorial herniation. In addition, a ventral, ventrally convex membrane separated the cyst-like lesion from the subarachnoid space. The vermis was morphologically normal and not displaced. The portion of the occipital bone adjacent to the lesion was thin and deformed with the inner lamina conforming to the shape of the fluid-filled lesion. These findings are most consistent with a mass effect. The lesion had signal intensity characteristics identical to cerebrospinal fluid (CSF), including T2 hyperintensity. The lesion did not communicate with the ventricular system or extend through the bone, thus making a cyst or cyst-like structure most likely. Cerebellar atrophy, degeneration, or hypoplasia with secondary filling of the space with CSF were considered less likely because of the compression and displacement of the left cerebellar hemisphere, tentorial herniation and thinning of the adjacent portion of the occipital bone (Figure 2). Additional abnormalities were detected in the cerebrum. The corpus collosum was absent. In both cerebral hemispheres, the gyri were decreased in size and increased in number (polymicrogyri) and the cortical white matter was bilaterally symmetrical and T2 hyperintense (consistent with leukoencephalopathy or possibly within normal limits for the foal's age). At the craniocervical junction, OAM findings observed on radiographs were evident concurrently on MRI (Figure 2). The images obtained did not extend to the level of the axis.

Supportive care including parenteral antibiotics, IV fluid therapy, gastroprotectants, oxygen supplementation, and feeding via nasogastric tube was continued. Antiepileptic drugs were discontinued. Feeding was transitioned from nasogastric administration of milk to pan feeding and

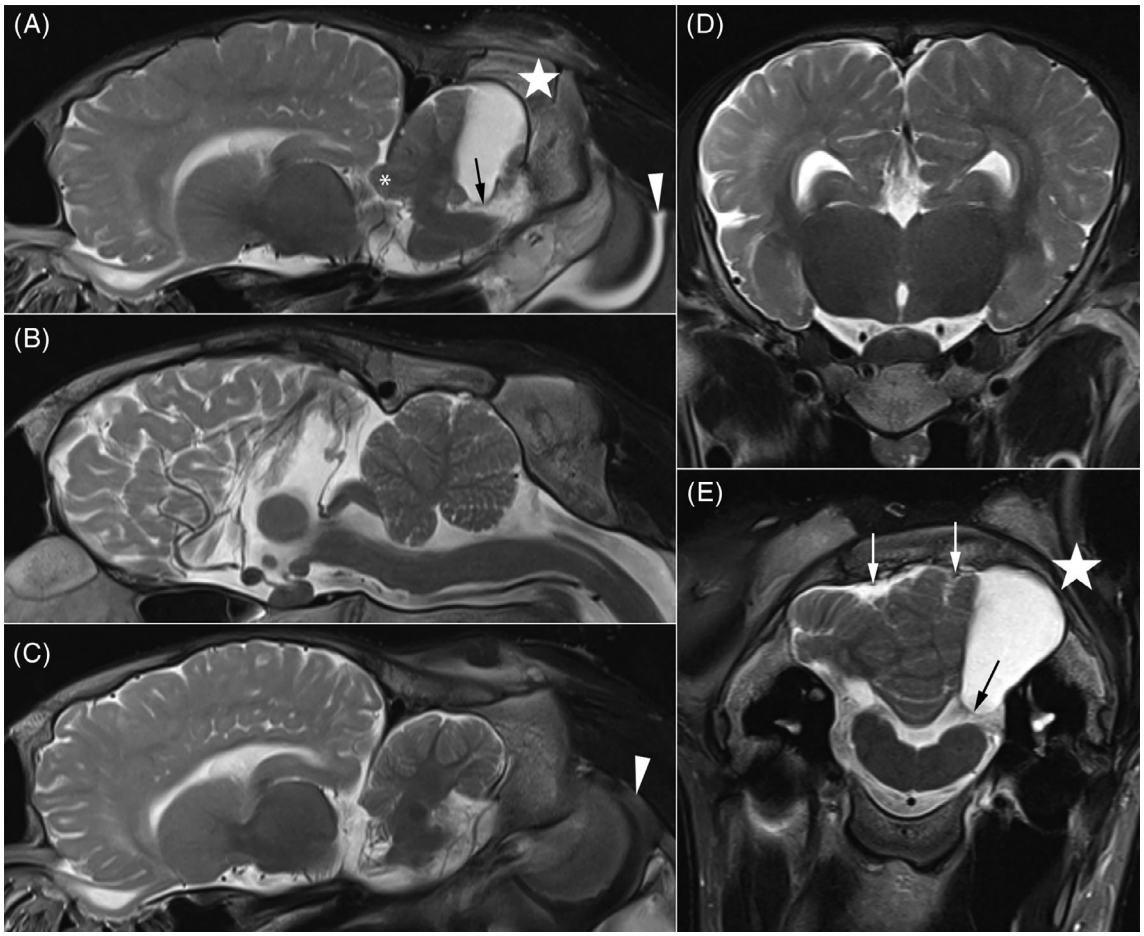

FIGURE 2 Sagittal (A and C), median (B), and transverse (D and E) T2-weighted MRI scans of the foal's brain. A is obtained left of midline and B is from right of midline. D is through the supratentorial space and E is through the infratentorial space. In the infratentorial space (A and E), there is a medium size, circumscribed, T2 hyperintense cyst-like lesion between the left cerebellar hemisphere and occipital bone. The left cerebellar hemisphere is small, and the vermis and right cerebellar hemisphere are normal. No communication between the abnormality and fourth ventricle is detected. In A and E, the extraaxial cyst-like lesion displaces the left cerebellar hemisphere rostrally and to the right. Note the ventrally convex hypointense membrane (black arrows) and the rostral tentorial herniation of the left cerebellar hemisphere (denoted by * in A) compared to normal on the opposite side (C). The bone adjacent to the abnormality is thin with outward expansion of the inner table (denoted by star in A and E). Also in E, note the left cerebellar hemisphere is displaced toward the vermis with loss of the subarachnoid space, compared to the normal on the right side (white arrows). In the supratentorial space (B and D) the corpus callosum and septum pellucidum are absent. Also, the gyri and sulci have increased number and smaller size (polymicrogyria), and the corona radiata are T2 hyperintense to gray matter which may be within normal limits of development or due to incomplete myelination or cerebral edema. A and C compare the left and right occipitoatlantal joints (white triangle). The left joint is normal with a normally formed occipital condyle and T2 hyperintense joint space. The right joint is abnormal with partial fusion of the occipital bone and atlas, and absence of the T2 hyperintense joint space. These latter findings are consistent with an occipitoatlantal malformation; neural compression due to the bony malformation was not detected

was well-tolerated without evidence of dysphagia. The colt was unable to suckle effectively from the dam because of intention tremor. Mild postanesthesia pneumonia was noted ultrasonographically and responded well to parenteral antibiotic treatment. Assistance to stand was continued, and the colt continued to show improvement in strength and coordination. On day 7, the colt was able to remain standing without assistance for 30 seconds and on day 13 was able to rise without assistance. The foal was assisted to walk, and physical rehabilitation exercises to build strength were performed daily. Supportive care was slowly discontinued, but the colt remained on omeprazole (4 mg/kg PO q24h) and antibiotics (trimethoprim sulfamethoxazole, 30 mg/kg PO q12h).

After 25 days of hospitalization, the mare and foal were discharged to the owner for continued management. Home care consisted of continued pan feeding, monitoring in a stall separated from the dam but with constant visibility, and daily controlled exercise initially with use of a harness for support. The colt continued to show improved balance and coordination, decreasing truncal sway and improved visual navigation of its environment. By the age of 6 months, the colt was able to rise without assistance, and walk, trot and canter with a hypermetric gait; the intention tremor and wide-base stance remained. At 1 year of age, the hypermetric gait, intention tremor and wide-base stance in all limbs were unchanged. Menace response was

present bilaterally. The colt also had marked kyphosis and symmetrical lack of epaxial musculature. Despite the ongoing neurological deficits, pasture turnout and routine care could be safely accomplished (Video S2).

2 | DISCUSSION

We describe the presentation, diagnosis, and long-term management of a neonatal foal with a complex neurological disorder. Although computed tomography (CT) is more commonly utilized because of its availability, MRI is the most accurate and sensitive imaging modality to evaluate neurologic conditions in horses.[1-4] The primary abnormalities identified on MRI were an infratentorial arachnoid cyst-like lesion with compression and displacement of the left cerebellar hemisphere, abnormal development of the cerebrum (absent corpus collosum and polymicrogyria) and cerebral leukoencephalopathy (T2 hyperintensity of the corona radiata). Noncompressive OAM is a condition that often is seen with or without many complex neurologic abnormalities and is not thought to be the cause of clinical signs in this foal.[1,5] Complex neurological disorders in foals are relatively uncommon and minimal information is published on their pathogenesis.[6-12] This condition is distinct from Dandy-Walker syndrome and from lesions known to be associated with genetic conditions and in utero viral infections, toxicities and nutritional deficiencies.

Dandy-Walker syndrome is a well-documented neurological disorder of humans and other mammals defined by cerebellar hypoplasia, dilatation of the fourth ventricle and often an absent corpus collosum.[10,11,13,14] Patients also can have concurrent hydrocephalus.[10,11,13,14] Although this foal had absence of the corpus collosum, the cerebellar vermis appeared within normal limits, no dilatation of the fourth ventricle was observed, and the cerebellar lesion was isolated to the left cerebellar hemisphere, making the condition distinct from Dandy-Walker syndrome.

Genetic conditions causing neurological abnormalities are rare but have been documented in several breeds of horses. Cerebellar abiotrophy is a well described condition of Arabian horses but also has been described in Oldenburgs, Gotland ponies and a single mule.[15,16] The abnormality is usually diffuse or anatomically bilaterally symmetrical, unlike the lesion in this foal.[17,18] Occipitoatlantoaxial malformation has been described to be heritable in Arabian foals and has been described in other breeds as well, but without known heritability.[1,15] Heritable genetic origin causes in our case are unlikely. To our knowledge, there is no known genetic cause in any species of the lesions described here. Additionally, a full sibling to the foal described here born 1 year before had no neurological abnormality.

In utero viral infections as well as maternal nutritional deficiencies and toxin exposures represent other known causes of loss of cerebellar tissue and hypomyelination in neonatal animals.[17-21] In our case, the dam was vaccinated appropriately throughout gestation, there were no known viral or toxin exposures or nutritional deficiencies during gestation, and several other healthy foals were born on the farm in the same year, making a viral etiology unlikely. Although the placenta and fetal fluids were not tested for viral agents, gross examination was within normal limits.

In another case report, in a 9-week-old Hanoverian filly was examined for progressive ataxia and hypermetria of the right forelimb and right hindlimb.[9] The filly had episodes of spontaneous falling to the right and menace response was absent in the right eye. The filly was euthanized, and necropsy disclosed a complex, unilateral malformation of the right cerebellum and left mesencephalon with hypoplasia of the corpus callosum. No viral agents were detected, and histological findings suggested possible in utero vascular injury during development resulting in localized hypoxia and ischemia.[9]

The bilaterally symmetrical nature of the neurological deficits noted in our case suggest that the left-sided suspected arachnoid cyst-like lesion and cerebellar loss were not the only contributors to the clinical signs because unilateral deficits would be expected if such were the case. The cerebellar and cerebral lesions as well as potential other abnormalities not identified on MRI such as increased intracranial pressure are thought to have contributed to the observed neurological signs. A concurrent spinal cord lesion contributing to the clinical signs could not be ruled out. Given that the foal responded partially to treatment for failure of transfer of passive immunity, sepsis, and neonatal encephalopathy, these conditions also may have played a role in the initial clinical picture.

Based on the findings in this foal, developmental anomalies of the cerebellum and cerebrum are the most likely pathogenesis. The pathogenesis of the thoracolumbar kyphosis is likely separate from the brain abnormalities and bony malformation associated with OAM and is perhaps a consequence of failure of somitogenesis, endochondral ossification, or fetal akinesia. Regardless of origin, no case of arachnoid cyst-like formation, absent corpus collosum and OAM has been described in the human medical literature without substantial neurological deficits.[13,22]

Intracranial arachnoid cysts may be true cysts (epithelial-lined, secretory), secondary to meningeal fibrosis causing regional disruption of CSF because of a valve-like abnormality, or inclusion cysts in which some of the surface ectoderm is trapped in deeper tissues during neurulation.[14,23] In our case, it is possible that the intracranial arachnoid cyst-like lesion developed first and subsequently caused compression and displacement of the left cerebellar hemisphere. Alternatively, the left cerebellar hemisphere may not have developed normally or may have been destroyed in utero (eg, hypoplasia) and the space normally occupied by the cerebellum could then have filled with CSF. The former pathogenesis is thought to be more likely in part because of deformation of the occipital bone at the same level and ipsilateral rostral tentorial herniation of the left cerebellar hemisphere. Cranial arachnoid cysts often do not produce clinical signs unless they compress the central nervous system or are associated with hemorrhage.[24]

Lesions identified in the forebrain included absence of the corpus collosum, polymicrogyria and altered white matter T2 MRI hyperintensity suggestive of leukoencephalopathy. Absent corpus callosum is a type of forebrain induction disorder that affects midline structures.[3,25] It has been described in combination with many other central nervous system

developmental anomalies. Polymicrogyria is associated with abnormal neuronal migration.[24] Polymicrogyria also has been described in association with muscle weakness.[24] Axonal disorders may result from delayed myelin development, vasogenic edema, or inflammation.[23,24] Polymicrogyria and vasogenic edema can be associated with seizures,[24] although the seizures observed when the foal was 24 hours of age and the inability to stand could have been related to neonatal encephalopathy rather than the congenital abnormalities based on their rapid resolution.[1,26]

Because MRI was terminated early because of patient instability under general anesthesia, it is possible that additional abnormalities would have been identified if a complete study could have been performed. Furthermore, laboratory analysis of placental tissues, fetal fluids and CSF may have provided information related to the pathogenesis of the lesions identified by MRI.

3 | SUMMARY

We characterized a novel, complex neurologic condition including an infratentorial arachnoid cyst-like lesion, absent corpus collosum, polymicrogyria, suspect leukoencephalopathy and OAM in a Dutch Warmblood foal. The case is distinct from Dandy-Walker Syndrome. The availability of advanced imaging modalities should improve characterization of congenital neurological disorders in foals. The foal's ability to partially compensate for the described lesions suggests that further study is needed to understand the extent to which each malformation contributes to the clinical signs. Careful monitoring and examination as the colt matures will allow better understanding of this condition.

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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.

HUMAN ETHICS APPROVAL DECLARATION

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

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