Radiographic and computed tomographic features of a subarachnoid diverticulum causing compressive cervical myelopathy in a yearling warmblood horse: a case report

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Abstract
A one-year-old Belgian warmblood with a previously known history of successfully repaired temporal and parietal bone fracture sustained at 14 days of age was presented for investigation of a recent, acute, and progressive four limbs ataxia. The owner reported a lack of coordination and difficulty getting up with progressing paresis two weeks prior to the presentation. Neurological examination revealed pronounced hypermetria/dysmetria in walk and trot in all four limbs. No evidence of cranial nerve deficit was observed. The horse was in good general condition with unremarkable clinical parameters. Survey lateral radiographs of the cervical spine showed moderate to severe signs of cervical malformation of the vertebral canal and articular process joints, indicating cervical stenotic myelopathy. Cervical computed tomographic (CT) myelography revealed the presence of a dorsal subarachnoid diverticulum causing significant spinal cord compression at multiple locations, with associated osteoarthrosis of the cervical articular process joints. Due to a poor prognosis and warranted surgical outcome, the owner declined further treatment, and the horse was discharged with conservative corticosteroid treatment. The patient was euthanised shortly after the initial presentation due to progressive worsening. To the author’s knowledge, CT myelography findings in a yearling with cervical subarachnoid diverticulum have not been previously published. This case illustrates the usefulness of advanced imaging techniques, such as CT myelography, in combination with static and dynamic radiography to provide a better and more accurate diagnosis.

SUMMARY
A one-year-old Belgian warmblood with a previously known history of successfully repaired temporal and parietal bone fracture sustained at 14 days of age was presented for investigation of a recent, acute, and progressive four limbs ataxia. The owner reported a lack of coordination and difficulty getting up with progressing paresis two weeks prior to the presentation. Neurological examination revealed pronounced hypermetria/dysmetria in walk and trot in all four limbs. No evidence of cranial nerve deficit was observed. The horse was in good general condition with unremarkable clinical parameters. Survey lateral radiographs of the cervical spine showed moderate to severe signs of cervical malformation of the vertebral canal and articular process joints, indicating cervical stenotic myelopathy. Cervical computed tomographic (CT) myelography...
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Keywords: equine, subarachnoid diverticulum, ataxia, stenotic myelopathy, cervical stenosis

INTRODUCTION

Intradural-extramedullary cyst-like conditions of the spinal cord are recognised diseases in equine literature but have been rarely reported and are poorly understood in this species (Allison et al., 2000). These are more commonly described in dogs and humans and rarely in cats as a cause of compressive myelopathy and subsequent progressive neurological dysfunction (Lowrie et al., 2005; Lowrie et al., 2009; Dyce et al., 1991; Mauler et al., 2014; Ness MG, 1998; Parked et al., 1983; Rohdin et al., 2014; Rylander et al., 2002; Skeen et al., 2003; Shamir et al., 1997; Cloward et al., 1968; Jurina et al., 2004). Subarachnoid diverticulum, also known as an intradural arachnoid diverticulum, is an abnormal focal dilation of the cerebrospinal fluid (CSF) within the subarachnoid space, lacking characteristic epithelial tissue lining (Hardie et al., 1996). Aetiology and pathogenesis remain poorly understood. Most cases seem to be congenital and associated with abnormal development of the arachnoid membrane (duplicating or splitting during embryologic development) (Lowrie et al., 2005). A single-way functional valve allowing one-direction flow causing focal CSF dilations and pressure disturbance within the subarachnoid space, leading to gradual spinal cord compression, was also speculated (Da Costa et al., 2016; Gage et al., 1968). Subarachnoid diverticula have been reported in association with other conditions such as trauma, arachnoiditis, intervertebral disc disease, or vertebral malformations in dogs (Bagley et al., 1997; Galloway et al., 1999; Aikawa et al., 2007; Mia, 2018). In humans, subarachnoid diverticula can develop from arachnoid adhesions following trauma, inflammation, infection or as a complication of lumbar myelography, laminectomy and vertebroplasty (Fan et al., 2018). Subarachnoid diverticula can arise at any level along the neural axis within the spinal canal and are associated with high-motion areas in dogs. Intracranial intra-arachnoid diverticula have been reported in men and dogs. They typically are closely associated with an intracranial arachnoid cistern, considered a primary malformation, though they may develop due to trauma or inflammation (Reed et al., 2009). They are most commonly found in the cervical region in large dog breeds and the thoracic region in small breed dogs (Bentley et al., 1991; Parker et al., 1974; Gnirs et al., 2003). In horses, a subarachnoid diverticulum has been reported in the cervical spine, with or without concurring vertebral malformation or degenerative osteoarthropathy (Allison et al., 2000; Fisher et al., 1981). To date, no radiographic nor advanced imaging studies have been reported; only diagnosis based on necropsy findings was confirmed as a cavity (diverticulum) located within the arachnoid space causing various spinal cord compression and consequently progressive ataxia in the juvenile horse (Allison et al., 2000; Fisher et al., 1981).

CASE HISTORY, CLINICAL EXAMINATION

A one-year-old Warmblood colt was presented for an acute onset of progressive and moderate to marked four limbs ataxia of 2 weeks. According to the owner, it started with low-degree front limb lameness and incoordination in the field, with occasional stumbling and struggling to rise. At 14 days of age, the patient sustained parietal and temporal bones fracture of traumatic origin associated with brain injury. The fracture was successfully repaired with an L-shaped reconstruction plate, and full recovery was achieved seven months post-surgery, with no persistent neurological deficits (Haardt et al., 2021). On admission, the patient was bright and alert, with an increased heart rate of 60 beats per minute and a respiratory rate of 28 breaths per minute. No arrhythmia or heart murmur was detected. The body condition score was moderate (5/9). Complete blood count and serum biochemistry profile were unremarkable. No central nervous system abnormality was detected during neurological examination, but moderate to marked ataxia and hypermetria
of all four limbs were observed during dynamic examination. Cervical compressive myelopathy was suspected.

**DIAGNOSIS**

**Radiographic findings and interpretation.**

Survey standard right lateral cervical spine radiographs were obtained with a Philips Optimum generator (Philips N.V., Eindhoven, The Netherlands) and AGFA DR detector (DR 40, AGFA-Gevaert N.V., Mortsel, Belgium) with the head and neck in a neutral position under sedation. Radiographic examination revealed an increased dorsal flaring of C2, C3, C4, C5, and C6 caudal vertebral endplates and mild caudal elongation of C3 and C4 dorsal laminae. The articular process joints between C5-C6 were mildly enlarged, whereas the articular process joints between C6-C7 and C7-T1 were moderate to severely enlarged, with ventral buttress formation completely superimposed to the intervertebral foramina (**Figure 1**). The neutral standing position demonstrated moderate ventroflexion of the C2-C3 vertebrae (163 degrees). The vertebral canal at C6 displayed a funnel shape with a narrower cranial aspect. The vertebral canal was subjectively narrowed within C4, C5 and C6 cervical vertebrae with intravertebral (C4: 0.462, C5: 0.424, C6: 0.425) and intervertebral (C3-C4:0.474, C4-C5: 0.458, C5-C6:0.461) sagittal diameter ratios lower than the reference range (Hahn CN et al., 2008) at these vertebrae. A small L-shaped surgical plate and four screws, compatible with the previous osteosynthesis, were superimposed on the dorsal aspect of the parietal bones (**Figure 5 C**). Radiographic findings were consistent with stenotic cervical myelopathy due to cervical vertebral malformation, including osteoarthropathy and hypertrophy of several vertebral articular process joints.

**CT and CT myelography**

CT scan of the cervical vertebral column, followed by IV contrast and CT myelography, were performed under general anaesthesia in dorsal (including brain and cranial cervical vertebrae) and right recumbency (caudal aspect of the cervical spine), using a 320-row scanner (Aquilion One, Canon Medical Systems, Tokyo, Japan) with a gantry opening of 78 cm, a scan field of view of 50 cm (slice thickness 0.5 mm, rotation time 0.5 s, pitch 0.638, exposure settings 250 mA and 135 kV, 512x512 matrix). Pre- and post-intravenous (IV) contrast injection CT scan from the head to C4 was performed in dorsal recumbency, highlighting the brain and cranial part of the cervical spine. For the IV contrast enhancing CT study, two power pressure injectors connected to extension sets attached to the jugular catheters were placed on both sides of the neck (400mL iodine/ml of 300mg iodine/mL, Iomeron, Bracco Imaging spa, Milano, Italy). Then, the poll area was clipped and aseptically prepared, and a spinal needle was inserted into the subarachnoid space at the level of the atlanto-occipital junction (Gough et al., 2019). Approximately 60 mL of CSF was aspirated, and the same volume of diluted (50:50 dilution with sterile saline) contrast medium (Iomeron 300 mg iodine/mL, Bracco Imaging spa, Milano, Italy) was injected. Following contrast injection, the head and neck were elevated for 5 minutes and repositioned in right lateral decubitus. This was followed by two consecutive CT scans (volume and helical) to include the entire cervical spine from C1 until C7. Bone and soft tissue reconstructions were acquired.

The cervical CT scan showed small hypoattenuating regions within the occipital condyles (left>right) compatible with incidental ongoing endochondral ossification (Sage SE et al., 2020).

Moderate to marked (right>left) enlargement of C3 and C4 cranial articular processes was present, with moderate narrowing of the intervertebral foramina. Moderate osteophyte of the right caudal articular process was appreciated. Moderate bilateral axial enlargement of C5, C6 and C7 cranial and caudal articular processes were present, resulting in moderate intervertebral foramina narrowing. Mild ventral enlargement of the right cranial articular process of T1.

Severe axial osteophytes with heterogeneous attenuation were present on the right side at C3. An ill-defined, cranio-caudally oriented hypoattenuating line compatible with a blood vessel or a fissure was seen at the axial aspect of the cranial articular process C4. Moderate axial osteophytes were noted between C3 and C4 articular process joints on the left side. On CT myelography, the contrast agent was present within the subarachnoid space until the level of C6. It demonstrated tear-drop-shaped dorsal and dorsolateral dilations of the subarachnoid space causing moderate to severe spinal cord compression from C3 until the cranial aspect of C7 (**Figure 2**). After the CT, radiographic cervical myelography (right lateral projections)
was acquired in flexion and extension. In all projections (flexed and extended), the dorsal contrast column was mild to moderately increased in dorsoventral height with an undulating ventral margin, progressively increasing in dorsoventral height caudally and most pronounced caudally from the level of C6 and C7. On flexed projections, there was greater than 50% (ref) narrowing of the dorsal contrast column at C2-C3 and C3-C4 with greater than 20% reduction in the total dural diameter (ref) at C2-C3, C3-C4, C4-5 and C5-C6, most severe at C2-C3. There was a subjectively increased angulation of the C2-C3 articulation (approx. 163 degrees).

On extended views, the undulating moderate widening of the dorsal contrast column was increased in height compared to the flexed views (Figure 3).

Diagnostic imaging findings were compatible with dorsal and dorsolateral subarachnoid diverticula causing moderate to severe spinal compression at C3, C4-C5, C5-C6, and caudal to C6 with concurrent multifocal, moderate to severe, vertebral articular process joints osteoarthrosis, most severe at C2-C3 and C3-C4. The radiographic myelogram confirmed subarachnoid diverticulum (diagnosed previously by CT) with severe dynamic compression of the spinal cord in multiple regions.

Additionally, the brain CT showed a moderately sizeable area of hypoattenuation at the lateral aspect of the left parietal lobe parenchyma with similar size and attenuation with the previous CT exam eight months before the last repeated scan, a month after initial traumatic brain osteosynthesis, with a mean 20 HU (Figure 4).

The left lateral ventricle was significantly enlarged, suggesting a left lateral ventriculomegaly. The left midbrain was decreased in size and had decreased attenuation, indicative of high fluid content, atrophy and loss of brain parenchyma.

There was complete bridging of the fractures at the level of the metallic plate, with a remaining cortical defect ventral to the plate (maximal dorsoventral gap 3mm). There were no signs of surgical implant failure (Figure 5A, B).

OUTCOME/FOLLOW UP

Due to limited surgical options, costs, and an unfavourable prognosis, the patient was discharged with conservative corticosteroid treatment. Shortly after discharge, the neurological deficits progressed, and the patient was euthanised at home with no post-mortem exam request.

DISCUSSION

Ataxia, paresis, and dysmetria are common complaints in horses and can be attributed to lesions of the spinal cord, cerebellum, or vestibular system (Oliver et al., 1997; Mayhew et al., 2009; Furr et al., 2015). Cervical vertebral stenotic myelopathy (CVSM), also known as cervical vertebral malformation or malarticulation, is the most reported cause of ataxia in juvenile and older horses (Levine et al., 2008; Dimock, 1938; Mayhew et al., 1978). CVSM has been divided into two categories: one that affects young horses (Type I, dynamic) and one that affects an older horse population (Type II, static) (Van Biervliet et al., 2007; Woodie et al., 2022). In our study, the presence of ataxia without central nervous system deficit combined with radiographic evidence of cervical vertebral malformation and hypertrophy of several articular process joints is highly indicative of compressive stenotic myelopathy than acute trauma or viral infection.

Plain radiographs are the first modality of choice to investigate ataxia in horses. However, it has limitations due to the superimposition of multiple osseous structures and demonstrates only narrowing in the sagittal plane/dorsoventral direction. In one study, sensitivity and specificity for degenerative changes of the vertebral articular joints in horses with suspected CVSM on radiographs were only 65% when compared to necropsy (Levine et al., 2010). A cross-sectional imaging modality such as CT can overcome these limitations and, thus, is more sensitive for detecting bone changes and axial location of abnormal bone formation (Fairburn et al., 2022). In our study, CT and CT myelograms were conducted to determine the clinical relevance of the radiographic findings. Clinical manifestation of the subarachnoid diverticulum is charac-
terised by proprioceptive ataxia with various degrees of tetraparesis or paraparesis, with spinal-associated hyperpathia and faecal/urinary incontinence depending on the location and size of the arachnoid lesion. They are believed to be not associated with pain in animals (dogs, horses) (Costa et al., 2016); in contrast, in men, neuropathic pain and headaches are commonly associated with clinical signs (Petridis et al., 2010).

Besides the cervical vertebral malformation with osteoarthrosis of several articular process joints, subarachnoid diverticula from C3 until C6 was present with stenotic myelopathy. Subarachnoid diverticula have been rarely described in equine literature. To date, two juvenile equine cases of the subarachnoid diverticulum (at the cervical and thoracic level) causing spinal cord compression of the cervical and cranial thoracic spine and subsequently marked ataxia, confirmed by necropsy, were found in veterinary literature (Allison et al., 2000; Fisher et al., 1981). In both cases, the age at the presentation was below two years. One was a Quarter-horse, and the other a Thoroughbred. Both were male. Similarly, our case was younger than two years of age and was also male. No breed is overrepresented, but a male prediction may be suggested; however, the number of cases is limited. Similarly, male overrepresentation was also described in dogs (Rohdin et al., 2014; Mauler et al., 2017).

Subarachnoid diverticula are becoming increasingly recognised in small animals due to more accessible advanced imaging techniques (Mauler et al., 2014; Jurina et al., 2004). Their aetiology remains unclear, although multifactorial causes have been hypothesised (Rohdin et al., 2014). In young dogs, subarachnoid diverticula are suspected to be of congenital origin, likely due to developmental malformation of the arachnoid membrane (Rylander et al., 2002). It is considered hereditary in Rottweilers (Rylander et al., 2002; Gnirs et al., 2003). Genetically inherited predisposition in Pugs is suspected, but pedigree or genetic analysis has not been performed (Rohdin et al., 2014). Acquired subarachnoid diverticulum has also been reported secondary to other underlying spinal diseases (Mauler et al., 2014). It seems a common trend in small breed dogs such as French bulldogs and Pugs with coexisting intervertebral disc extrusion/protrusion or previous vertebral surgery (Da Costa et al., 2016; Mauler et al., 2014). Other reported causes include spinal trauma, spinal dysraphism, and concurrent vertebral malformations (Chen AV et al., 2005; Skeeën et al., 2003; Mai W, 2018).

In our case, the patient sustained a skull fracture at 14 days of age, associated with traumatic brain injury. After surgery, neurological signs diminished with time, and the foal remained normal until two weeks before presentation. In humans, acquired subarachnoid diverticula can develop after significant trauma or following infection (Page et al., 1987). Reed et al. reported an association of arachnoid diverticula with neoplasms, postsurgical adhesions, leptomeningitis, and haemorrhage in cats and dogs (Reed et al., 2009). Another study by Matiassek et al. suggests that acquired subarachnoid diverticulum can result from post-inflammatory loculation of the subarachnoid space after a head injury, intracranial infection, and haemorrhage (Matiassek LA et al., 2007). It is impossible to determine whether the yearling subsequently developed subarachnoid diverticulum after the significant brain trauma, subsequent haematoma or due to post-surgical adhesions. It cannot be excluded that a subarachnoid diverticulum was present at birth and slowly resulted in gradual neurological impairment over time. Initial CT was only performed at the level of the skull and did not include the cervical region. However, a repeated CT scan identified persistent left lateral ventriculomegaly with mild atrophy and decreased attenuation in the left midbrain at the level of the previous calvarial fracture, which could indicate a potential acquired cause as a result of maintained traumatic brain injury.

Subarachnoid diverticula cannot be evaluated on plain radiographs. Advanced imaging techniques such as radiographic myelography, CT-myelography, or MRI are necessary. MRI is the gold standard modality in dogs and humans, but it is not feasible on live horses due to size-limiting factors. Our CT-myelography demonstrated the typical tear-drop-shaped dilatation of the subarachnoid space described in humans and small animals, leading to multifocal spinal cord compression (Rylander et al., 2002; Cambridge et al., 1997; Gnirs et al., 2003; Lowrie et al., 2014; Mauler et al., 2014; Smith et al., 2020).

A subarachnoid diverticulum can develop at any level along the spinal cord but is mainly found in the cervical (C2-C3 and C5-C6) and thoracolumbar (T9-T13, T13-L1) regions in dogs, at areas of high spinal mobility (Gnirs et al., 2003; Skeeën et al., 2003). Other studies suggested that large-breed dogs are more prone to
develop subarachnoid diverticulum in the cervical area. In contrast, the subarachnoid diverticulum in small and medium-breeds dogs (Pugs and French Bulldogs) more commonly involves the thoracolumbar region (Mauler et al., 2014). In our study, the cervical spine was affected. Although, due to the large patient’s size and gantry limitation, a CT scan of the thoracolumbar region could not be performed.

According to studies conducted in dogs, 90% of subarachnoid diverticulum are located dorsal or dorsolateral to the spinal cord (Frykman, 1999; Gnirs et al., 2003; Jurina et al., 2004). This is compatible with our findings.

An arachnoid diverticulum can also occur intracranially in close association with the intracranial arachnoid cisterns. They are a focal dilation of the subarachnoid space with increased separation between the pia mater and arachnoid. In dogs, intercranial diverticulum is a common incidental finding regarded as either congenital in origin or as a consequence of trauma, infection or inflammation (Mai, 2018; Vernau et al., 1997). A cranial infratentorial arachnoid cyst-like lesion has recently been reported in a foal with a concurrent complex congenital malformation. These cyst-like lesions are believed to cause no clinical signs unless they compress the central nervous system or are associated with haemorrhage (Luie et al., 2022).

The prognosis of subarachnoid diverticulum depends on available treatment options and case signment. A recent comparison study favoured surgical treatment over medical management in 96 treated dogs. Traditional medical treatment with anti-inflammatory drugs was associated with less clinical improvement, however, given the small number of treated cases and limited follow-up, effectiveness is debatable. Besides, the progressive nature of SAD makes the surgical approach treatment of the choice (Mauler et al., 2017). There are various described surgical procedures and diverse surgical approaches in small animals, including fenestration/and or aspiration of the diverticula with durotomy or durectomy and marsupialisation of the diverticulum or laminectomy/hemilaminectomy in combination with vertebral stabilisation (Smith et al., 2020; Frykman et al., 1999; Ness, 1998; Lowrie et al., 2005; Da Costa et al., 2016). Most of these procedures carry a high recurrent of clinical signs with rates of 25%, 66% and 64% in dogs, with signs reoccurring between 14-26 days after the surgery (Gnirs et al., 2003; Rylander et al., 2002; Lowrie et al., 2005). Better surgical outcomes were associated with ages below three years and less than four months of clinical signs (Skeen et al., 2003).

In contrast, surgical management in humans intends for complete cyst/diverticulum removal with consecutive closure of the subarachnoid space (Lee et al., 2012). Shunt placement is an alternative treatment option that permanently drains the intra-arachnoid diverticulum in a controlled manner (Alvisi et al., 1987; Hughes et al., 2018). Surgical treatment has not been implemented in horses to this date, considering their size, cost limitations, and likely excessive post-operative care.

CONCLUSION

In conclusion, the present report describes a rare case of cervical spinal cord compression secondary to the presence of a SAD in a one-year-old yearling with concurrent cervical stenosis. It highlights the usefulness of advanced imaging techniques to avoid misdiagnoses.

ABBREVIATIONS

CSF- cerebrospinal fluid  SAD- subarachnoid diverticulum  CT-M- computed tomography myelography  MRI – magnetic resonance imaging

References:


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