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CASE REPORT

Canine vertebral venous thrombosis: confirmatory MRA before retreatment following symptom recurrence

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Venous thrombosis within the internal vertebral venous plexus is a rare occurrence in both human and veterinary medicine. This case report details the evaluation and management of an 18-month-old female spayed Siberian Husky Mix diagnosed with internal vertebral venous plexus thrombosis after presenting with cervical pain and mild tetraparesis. Initial imaging using magnetic resonance imaging (MRI) revealed asymmetric enlargement of the right internal vertebral venous plexus without evidence of spinal cord compression. Cerebrospinal fluid analysis was unremarkable. The dog was treated with clopidogrel and analgesics, leading to significant clinical improvement. However, clinical signs recurred after 20 weeks, prompting a repeat MRI that confirmed persistent thrombus. The treatment regimen was reinitiated, resulting in resolution of clinical signs. This case underscores the need for specific guidelines regarding the diagnosis and management of venous thrombosis in veterinary patients, as current literature predominantly references human medicine. The report also highlights the potential role of advanced imaging techniques, such as phase-contrast magnetic resonance angiography (MRA), in diagnosing vascular conditions in veterinary practice.

Keywords: small animal neurology, cervical pain, internal vertebral venous plexus thrombosis, anticoagulant therapy, magnetic resonance angiography

Introduction

Thrombosis within the internal vertebral venous plexus is exceedingly rare in both human and veterinary medicine. In human medicine, it has been documented in only a few cases, all associated with underlying conditions such as deep vein thrombosis and neoplasms of the pancreas, lungs, and gastrointestinal tract (Köcher & Hofmann 2018). Similarly, in veterinary literature, internal vertebral venous plexus thrombosis is exceptionally uncommon, with only a single published case report to date (Rhue et al. 2017)

Thrombotic diseases in dogs are often linked to various underlying causes, including protein-losing nephropathy, hyperadrenocorticism, neoplasms, and the concurrent administration of steroids (Winter & Budke 2017). In this previously reported case, the patient had a history of concurrent steroid administration, suggesting an association with the thrombotic event. There was also moderate spinal cord compression due to the thrombus. Notably, this case lacked follow-up MRIs to assess thrombus resolution, leaving a gap in our understanding of the long-term consequences of such events.

The case report presented below contributes to the growing body of literature by presenting a unique case involving an 18-month-old female spayed Siberian Husky Mix with thrombosis in the internal vertebral sinus plexus. In contrast to the previously reported case, a follow-up MRI with magnetic resonance angiography (MRA) was performed to confirm the presence of a thrombus and assess the resulting engorgement of the internal vertebral venous plexus.

Thrombus formation is a complex process driven by imbalances in the haemostatic system, primarily explained by Virchow's Triad: (1) decreased blood flow (stasis), which allows clotting factors to accumulate; (2) vessel wall injury, exposing prothrombotic subendothelial tissue factor and activating platelets; and (3) systemic changes in procoagulant and anticoagulant factors, such as deficiencies in natural anticoagulants (e.g. protein C, antithrombin III) or increased clotting factors (Rosenberg & Aird 1999). These elements disrupt the balance between clot formation and dissolution, leading to localised thrombotic lesions.

The venous supply to the spinal cord can be divided into the ventral and dorsal internal vertebral venous plexuses. The ventral plexus runs through the epidural space between the ventrolateral dura and adjacent vertebral bodies and pedicles, while the dorsal plexus is located between the dorsal dura mater and laminae. Both plexuses drain into the segmental intervertebral vein, which connects to the external vertebral venous plexus via the intervertebral and basivertebral veins (Vernon et al. 2017). Engorgement or thrombosis of the internal vertebral venous plexus increases pressure within the spinal canal, leading to compression of the spinal cord or nerve roots. This can cause localised inflammation, impaired blood flow, ischaemia, and ultimately, pain and discomfort in the affected area (Donmez 2015).

Case history

An 18-month-old female spayed Siberian Husky Mix was presented to the Veterinary Specialist for Anesthesia and Neurology (VSCAN) in Canada for evaluation of neck pain. The owners first noticed signs of discomfort when petting the dog approximately two months prior to presentation. The dog exhibited significant pain, particularly when turning the neck to the right. Empirical pain management was initiated by the primary veterinarian, but the response was only partial, with symptoms recurring upon discontinuation of medication.

On physical examination, the dog weighed 30.9 kg and was bright, alert, and responsive. A board-certified neurologist, along with a neurology resident, performed a neurological evaluation. The dog exhibited mild ambulatory tetraparesis and a floating gait in the thoracic limbs. The hopping response was mildly decreased in the right thoracic and pelvic limbs, while the patellar reflexes remained normal bilaterally. Withdrawal reflexes in the thoracic limbs were reduced bilaterally, but muscle tone and mass were within normal limits in all limbs. Moderate hyperesthesia was noted in the cervical region, with no significant orthopaedic abnormalities detected. Based on the neurological examination, a myelopathy affecting the C1–T2 region, with a focus on C6–T2, was suspected. Routine diagnostic tests, including a complete blood count and biochemistry panel, were unremarkable. A urinalysis was also performed, with normal results.

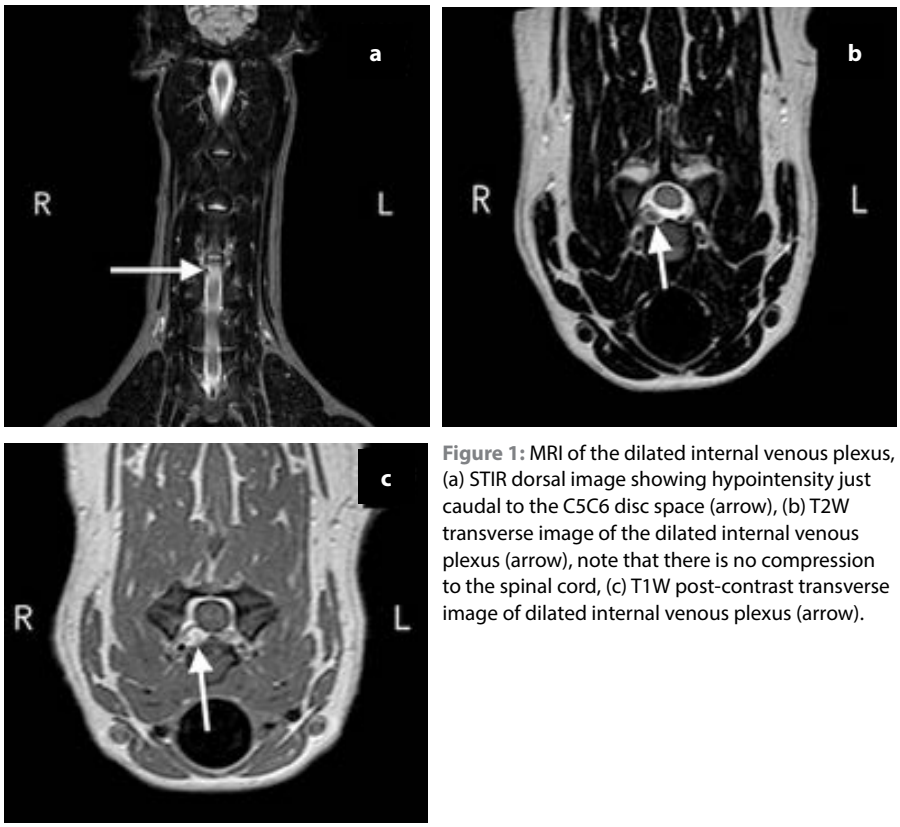

Figure 1: MRI of the dilated internal venous plexus, (a) STIR dorsal image showing hypointensity just caudal to the C5C6 disc space (arrow), (b) T2W transverse image of the dilated internal venous plexus (arrow), note that there is no compression to the spinal cord, (c) T1W post-contrast transverse image of dilated internal venous plexus (arrow).

Imaging findings – initial scan

The dog was premedicated with hydromorphone hydrochloride (0.1 mg/kg IV, Hydromorphone Hydrochloride Injection USP 10 mg/ml, Pfizer Canada ULC, Kirkland, Quebec, Canada) and dexmedetomidine (1 mcg/kg IV, Dexvetidine 1 mg/ml, Modern Veterinary Therapeutics LLC, Sunrise, Florida, United States), induced with propofol (2 mg/kg IV, Propoflo 28 10 mg/ml, Zoetis Canada Inc, Kirkland, Quebec, Canada) and ketamine (1 mg/kg IV, Narketane 100 mg/ml, Vetoquinol N A Inc, Lavaltrie, Quebec, Canada). The dog was intubated with a 57F cuffed ET tube and maintained on isoflurane (between 1.2%–1.5%, Isoflurane USP 99.9%, Fresenius Kabi Canada Ltd, Toronto, Ontario, Canada) during the procedure. Positioned in dorsal recumbency for cervical spine MRI, images were acquired using a 1.5 Tesla High-Field MRI unit (Intera MRI System, Philips, Eindhoven, the Netherlands) with a slice thickness of 3 mm. T2-weighted (T2W) images (TR = 5200 ms, TE = 120 ms), T1-weighted (T1W) images (TR = 508ms, TE = 8.2ms) pre- and post-contrast administration intravenous administration of gadolinium contrast (0.1 mmol/kg, ProHance; Bracco Imaging, Canada) were obtained in sagittal, transverse and dorsal planes. Also, a Half-Fourier Acquisition Single-shot Turbo Spin Echo (HASTE) sequence and a proton density image (PD) was obtained in sagittal plane and a short tau inversion recovery (STIR) (TR = 2500 ms, TE = 80 ms, TI = 150 ms) was obtained in the dorsal plane.

The spinal cord demonstrated normal signal intensity on both T1 and T2-weighted images, with an appropriate segmental diameter. No enlargement or abnormal signal intensity of the spinal nerves was identified. Additionally, no abnormal enhancement was observed in the cervical spine following contrast administration. All nuclei pulposi were well hydrated. There were no visible abnormalities in the paraspinal musculature. However, it was noted that there was asymmetric mild enlargement of the right internal venous plexus at C5–6, which was thought to be attributed to a thrombus or congenital malformation. There was no evidence of spinal cord compression (Figure 1). It was concluded that there were no definitive macroscopic abnormalities to explain the clinical signs besides the right-sided internal vertebral venous plexus dilation focally at C5–6.

Further laboratory findings: cerebrospinal fluid analysis

A cerebrospinal fluid sample was collected from the atlantooccipital cistern for further analysis and was considered cytologically unremarkable. Both protein concentration and total/differential WBC count were within established reference ranges. There was no evidence of inflammation or infectious disease. Specific infectious agents were tested, including *Bartonella spp*., *Blastomyces dermatitidis*, *Coccidioides spp*., *Cryptococcus spp*., *Histoplasma capsulatum*, Canine distemper virus, West Nile virus, *Borrelia burgdorferi*, *Neospora spp*., and *Toxoplasma gondii*. All tests were performed using PCR and yielded negative results.

Treatment and outcome

Given the absence of definitive macroscopic abnormalities to explain the clinical signs, the focal right-sided internal vertebral venous plexus dilation at C5–6 was considered the most plausible cause. Although a thrombus could not be confirmed at this stage, it was deemed the most likely explanation, warranting empirical antithrombotic therapy. Therefore, the dog was treated with clopidogrel (1.25 mg/kg SID, Apo-Clopidogrel

75 mg, Apotex Inc., Toronto, Ontario, Canada). Additionally, empirical pain management was provided with gabapentin (16.4 mg/kg TID, Apo-Gabapentin, Apotex Inc., Toronto, Ontario, Canada), methocarbamol (32.8 mg/kg TID, Robaxin 1 000 mg, Pfizer, Mississauga, Ontario, Canada). The dog was presented for a recheck three weeks later and had only very subtle tetraparesis. The hopping response on the right thoracic limb was markedly improved and that of the right pelvic limb was resolved. The withdrawal response on the right thoracic limb was still mildly decreased. There was only very mild hyperesthesia in the cervical region when the neck was turned to the right. Clopidogrel was discontinued eight weeks after the first presentation because of the complete resolution of the clinical sign. Due to the favourable outcome with antithrombotic medication, we are confident that the clinical signs were due to the suspected thrombus, supporting a presumptive diagnosis of internal vertebral venous plexus thrombosis.

Twenty weeks after the first presentation, the clinical signs relapsed. Empirical treatment with previous pain medication was started again without clopidogrel. The neurological deficits reverted to a severity similar to the initial presentation. There was moderate pain when the neck was flexed to both sides. MRI and CSF sampling were repeated to look for the underlying cause.

Imaging findings – follow-up MRI scan

The dog was anaesthetised and an MRI of the cervical spine (Figure 2) was repeated using the same anaesthetic protocol and imaging setting as described above. Also included in this study were 3D phase-contrast MR venograms (TR = 21 ms, TE = 4.1 ms) as well as a Time of Flight (TOF) Volume Rendered (VR) Maximum Intensity Pixel (MIP) image.

No areas of abnormal spinal cord parenchymal change or enhancement were seen. There was also no evidence of any extradural compression to the spinal cord. The previous dilation of the internal venous plexus was still evident in the same area (Figure 2a). Both the TOF MIP (Figures 2b and 2c) and the MR venogram (Figure 2d) revealed severe engorgement of the right internal venous plexus. The suspected thrombus was also visualised directly (Arrow, Figure 2b and 2c). The heterogenous signal intensity cranial and caudal to the thrombus signifies turbulent blood flow.

Treatment and outcome

Clopidogrel (1.25 mg/kg SID) was reintroduced alongside the previously mentioned analgesic medication. The dog was presented for a recheck one month later. There were no neurological deficits and only mild resistance in neck flexion upon examination. Because of the recurrent nature, a three months course of clopidogrel was prescribed. Complete resolution of the clinical signs was reported one month later via email communication.

Discussion

There is a notable gap in veterinary medical literature when it comes to guidelines for diagnosing and treating venous thrombosis, particularly in the internal vertebral venous plexus. In human medicine, conditions like deep vein thrombosis are well studied and typically managed with anticoagulants such as warfarin or rivaroxaban for three to six months, with extended treatment if an underlying cause remains (Ortel et al. 2020).

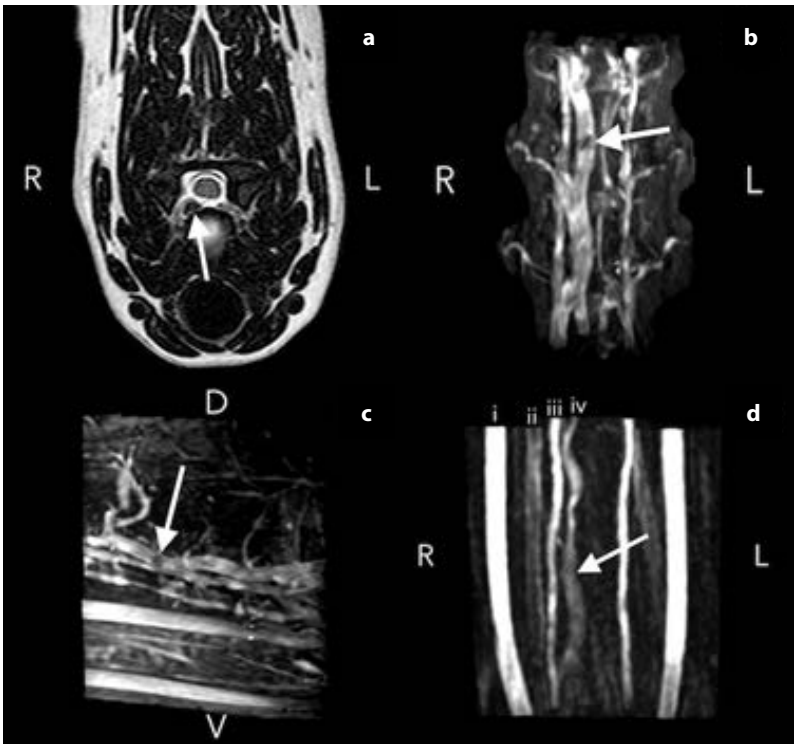

Figure 2: Follow up MRI, (a) T2W image of the dilated internal vertebral venous plexus (arrow), (b) 3D Phase-contrast MR venogram in dorsal plane, note the thrombus (arrow) within the dilated internal vertebral venous plexus, (c) 3D Phase-contrast MR venogram in sagittal plane, note the thrombus (arrow) (d) 2D TOF VR MIP image in the dorsal plane, i = external jugular vein, ii = carotid artery, iii = external vertebral venous plexus, iv = internal vertebral venous plexus, note the engorgement of the internal vertebral venous plexus (arrow).

In veterinary medicine, venous thromboembolism (VTE) is commonly associated with conditions like protein-losing nephropathy and immune-mediated haemolytic anaemia (IMHA) in dogs, where thrombi form under low-shear conditions and are fibrin-rich, making their formation less dependent on platelet function. In contrast, arterial thromboembolism (ATE), such as those seen in feline cardiomyopathies, develop under high-shear conditions and are platelet-rich, making antiplatelet drugs more effective for their prevention (Blais et al. 2019). In this case, clopidogrel was used as the sole antithrombotic therapy, despite its primary role as an antiplatelet agent. Clopidogrel works by irreversibly inhibiting the P2Y12 receptor on platelets, preventing ADP (adenosine diphosphate)-mediated platelet activation, thereby reducing platelet aggregation and thrombus formation. Although anticoagulants are generally recommended for VTE and antiplatelet drugs for ATE prophylaxis, there is evidence in human medicine that some crossover efficacy exists. Direct comparisons of anticoagulants and antiplatelet agents

for preventing venous thrombosis in veterinary patients are warranted.

In this case report, TOF and phase-contrast MRA were employed to assess venous thrombosis within the internal vertebral venous plexus. This imaging technique utilises the natural movement of blood through the magnetic field, taking advantage of differences in signal between flowing blood and stationary tissues to visualise blood vessels. The most common applications of MRA in human medicine are brain and cerebrovascular imaging for stroke, aneurysms, and stenosis; assessing peripheral artery disease by evaluating blood flow in the legs; and evaluating coronary artery disease by detecting blockages or stenosis in the coronary arteries. The clinical application of MRA imaging in veterinary medicine is still in its early stages of development, with limited studies documenting its use. For instance, MRA has been employed in diagnosing aortic thrombosis and portosystemic shunt in dogs (Brofman & Thrall 2006; Mai & Weisse 2011).

Our case report presents distinct differences from the previously reported case of internal vertebral venous plexus thrombosis. In the previously reported case, the patient had a history of steroid use, suggesting an association with the thrombotic event. In contrast, despite extensive investigation, no underlying cause could be identified in our case. Additionally, in the prior report, moderate spinal cord compression caused by the thrombus was readily apparent on sagittal T2-weighted MRI images, whereas in our case, no evidence of compression or abnormal signal intensity suggestive of inflammation or ischaemia was observed on conventional MRI sequences. It was not until MRA was performed that we discovered the extensive internal vertebral venous plexus engorgement, which had been previously undetected. This aligns with the findings of a previous study on vertebral venous system abnormalities in sighthounds, which showed that venous abnormalities, such as engorgement of the internal vertebral venous plexus, can be difficult to detect using only conventional MRI (Vernon et al. 2017). This case highlights the importance of considering additional MRA sequences when clinical signs and MRI findings do not align in severity or when further evaluation of vascular dilation is necessary. It also demonstrates that a thrombus alone can cause significant clinical signs without apparent compression on conventional imaging. Engorgement or thrombosis of the internal vertebral venous plexus can increase pressure within the spinal canal, potentially leading to spinal cord compression, localised inflammation, impaired blood flow, and ischaemia (Donmez 2015). While no overt MRI findings suggestive of these pathophysiological processes secondary to the thrombus were observed in our investigation, these changes may still occur at a microscopic level, undetectable by standard imaging techniques. The precise course of the thrombus throughout the treatment process remains unclear. It is uncertain whether the thrombus never dissolved, whether a new thrombus formed, or whether the original thrombus initially shrank but later regrew after discontinuing the clopidogrel. However, given that the clinical signs were well managed with the initial treatment regimen, recurrent thrombosis is considered the more likely explanation.

In retrospect, a reconsideration of the therapeutic approach in this case reveals potential refinements. One notable alternative is the exploration of rivaroxaban at a dosage of 1–2 mg/kg/day (Blais et al. 2019) as an antithrombotic agent instead of clopidogrel. Unlike clopidogrel, rivaroxaban directly inhibits activated factor Xa, a distinct mechanism of action that might be more effective in addressing venous thrombosis. Monitoring the drug's efficacy through a quantitative method, such as thromboelastography, enables a more precise adjustment of the treatment plan (Bae et al. 2019). Furthermore, extending the duration of rivaroxaban administration to a six-month period aligns with established human medicine guidelines for treating venous thrombosis. Subsequent to this extended treatment, a repeated MRI would serve as a crucial diagnostic tool to ascertain the persistence or resolution of the clot, providing valuable insights into the efficacy and long-term outcomes of the chosen therapeutic approach.

Conflict of interest

The authors declare they have no conflicts of interest that are directly or indirectly related to the research.

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Ethical approval

MRI and CSF sampling were performed after we gave informed consent and received the owner's agreement.

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