Magnetic resonance imaging and marsupialization of a hemorrhagic intramedullary vascular anomaly in the cervical portion of the spinal cord of a dog

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Case Description—A 1-year-old female spayed Labrador Retriever was admitted for evaluation of a progressive gait disturbance characterized by tetraparesis and general proprioceptive ataxia in all limbs.

Clinical Findings—Neurologic examination suggested a dysfunction of the C6-T2 spinal cord segments, which was slightly worse on the right side. Discomfort was suspected upon lateral flexion of the neck. Two magnetic resonance imaging (MRI) examinations at a 3-week interval revealed an intramedullary fluid-filled cavity lesion adjacent to C7, containing a blood clot.

Treatment and Outcome—Following unsuccessful initial conservative management, surgical marsupialization of the lesion was performed through a dorsal laminectomy, durotomy, and myelotomy at C6 and C7. Histologic evaluation including immunohistochemistry was diagnostic for a vascular anomaly. Initially, the dog was nonambulatory with tetraparesis and became tetraplegic after surgery; movement was regained 6 days later. Four weeks after the procedure, the dog was able to walk unassisted. One year after surgery, the dog was actively running and jumping, with mild residual ataxia in the pelvic limbs.

Clinical Relevance—The intramedullary vascular anomaly in this dog was successfully treated with a surgical marsupialization technique. The combination of MRI, histologic evaluation, and immunohistochemistry enabled lesion localization, evaluation of cavity content, and final diagnosis. (J Am Vet Med Assoc 2008;232:399–404)

Abbreviations

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<th>Abbreviation</th>
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<th>Description</th>
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<tr>
<td>T2w</td>
<td>STIR</td>
<td>T2-weighted Short-tau inversion recovery</td>
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<td>MRI</td>
<td>FLAIR</td>
<td>Magnetic resonance imaging Fluid-attenuated inversion recovery</td>
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<tr>
<td>FVIIIRa</td>
<td>Factor VIII–related antigen</td>
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A 1-year-old 32-kg (70-lb) female spayed Labrador Retriever was referred to the Centre Hospitalier Universitaire Vétérinaire at the Université de Montréal for evaluation of a progressive gait disturbance characterized by tetraparesis and general proprioceptive ataxia in all limbs. Three weeks previously, the owners noticed a sudden onset of right-sided neurologic deficits. The dog’s previous medical history was unremarkable with no history of trauma. Initial assessment by the referring veterinarian revealed that the dog was bright, alert, and responsive, with abnormalities confined to the neurologic system. Proprioceptive ataxia and paresis of the right thoracic and pelvic limbs were observed. Deficits in postural reactions were observed in all limbs. Discomfort was thought to be present on lateral flexion of the neck, but the patient’s reaction was equivocal. The dog was treated with meloxicam* (0.1 mg/kg [0.05 mg/lb], PO, q 24 h) for 10 days. The dog seemed more comfortable and active under this regimen, but its neurologic status slightly deteriorated until referral.

At the time of admission, physical examination findings were largely unremarkable. A complete orthopedic examination did not reveal any clinically important findings, and muscle mass appeared normal. Neurologic examination revealed that cranial nerve function and mentation were considered normal. The dog was ambulatory with evidence of tetraparesis and general proprioceptive ataxia in all limbs. The thoracic limbs had a short-strided gait, compared with the pelvic limbs. Knuckling was observed in the right thoracic and pelvic limbs. Decreased postural reactions suggested that proprioceptive and motor deficits were present in all limbs. The deficits seen on gait analysis and postural reaction testing were more noteworthy on the right side of the body, compared with the left. Carriage of the head and neck was considered normal, but discomfort was suspected upon lateral flexion of the neck, similar to the findings by the referring veterinarian. Patellar and withdrawal reflexes were normal in the pelvic limbs, and withdrawal reflexes were slightly and moderately decreased in the left and right thoracic limbs, respectively. Perineal reflexes, anal tone, and continence were preserved. The neuroanatomic localization of the lesion was between the T6 and T2 spinal cord segments.

Magnetic resonance imaging of the cervical portion of the spinal cord was performed by use of a 1.5-T unit, with the dog under general anesthesia and in dorsal re-
cumbency. Sagittal and transverse T2w fast spin–echo images, sagittal STIR, sagittal and transverse pre- and postcontrast T1w fast spin–echo images, and sagittal and dorsal T2w 3-dimensional fast imaging employing steady state acquisition (FIESTA) gradient-echo images were obtained. Slice thickness was 3 mm with no interslice gap. Adjacent to the C7 vertebral body, a 2.5-cm-long by 1.1-cm-wide and -high, ovoid, well-defined, smoothly marginated structure was seen in the center of the spinal cord. This structure caused circumferential widening of the spinal cord and occupied approximately 90% of the spinal cord cross-sectional area. Marked circumferential thinning of the spinal cord tissue was present, as well as attenuation of the subarachnoid space and epidural fat. The thickest area of identifiable spinal cord was ventral and left-sided, and the central canal deviated ventrally, possibly indicating a right dorsal origin of the lesion. On T2w and STIR images, the intramedullary structure was divided into 2 distinct regions of differing signal intensity, associated with a well-defined, linear, and horizontal interface (Figure 1). The larger nondependent region was relatively isointense relative to CSF, and the dependent region was hypointense relative to CSF, but hyperintense relative to the spinal cord. A 0.8-cm-long well-defined, irregular, hypointense focus was also present in the nondependent portion of the intramedullary structure. This focus was also detected on T1w images, although it was hyperintense relative to the spinal cord (Figure 2). No evidence of peripheral cord signal alterations was found, and no contrast enhancement occurred.

The presence of an intraleSIONAL fluid line and signal characteristics consistent with fluid indicated that the lesion was cavitary and cystic in nature. The thinning of the spinal cord tissue suggested a chronically expansile lesion. The hypointense-dependent material within this cystic lesion was considered to represent cellular, necrotic, or hemorrhagic material. The nondependent region, although relatively intense on T2w images, was isointense relative to the spinal cord on T1w images, which suggested a protein content higher than the CSF. The smaller focus was evaluated as being a subacute blood clot or hematoma. Differential diagnoses on the basis of MRI findings were congenital cyst (epidermoid or dermoid) or a cystic neoplastic process. Focal congenital or acquired syringohydromyelia was also considered. Because of the absence of contrast enhancement and no evidence of signal void, active blood flow to the lesion was not suspected.

Conservative medical management with meloxicam (0.1 mg/kg, PO, q 24 h) and restricted exercise were recommended for another 21 days, allowing further observation of the progression of clinical signs. It also allowed the owners to consider the possibility of surgical treatment. Clinical signs worsened over this period, and the dog was readmitted to the hospital a little over 3 weeks later. Neurologic examination findings were similar to previous findings, but with increased severity of all reported deficits. The short-strided gait of the thoracic limbs was particularly severe, and the dog was barely ambulatory. Because of the possibility of acute worsening of the lesion or further intraleSIONAL hemorrhage, it was decided to repeat the MRI and to proceed with surgery.

Results of hematologic and serum biochemical analyses were within reference limits, as were arterial blood gas tensions determined before MRI and surgery.

Magnetic resonance imaging was repeated 26 days after the original study. Transverse and sagittal T2w fast spin–echo, sagittal before and after contrast T1w fast spin–echo, transverse intermediate-weighted proton-density fast spin–echo, and sagittal FLAIR images were obtained. The intramedullary structure was slightly longer (2.6 cm) than detected previously but was otherwise unchanged in appearance, and no contrast enhancement was present. Examination of FLAIR images revealed that the fluid in the nondependent portion of the lesion remained hyperintense, indicating a proteinaceous composition (Figure 3). On intermediate-weighted proton-density images, the fluid was moderately hyperintense relative to the spinal cord (Figure 4). The list of differential diagnoses remained the same.
A dorsal laminectomy was performed over C6 and C7, preserving the articular processes. Particular attention was paid to minimize lateral elevation of the multifidus muscular bellies, in an attempt to preserve anchorage for future dura mater marsupialization. The spinal cord was enlarged and darker than normal over the site of the cystic lesion. A 3-cm-long sagittal durotomy was then performed and readily transformed into a H-shaped durotomy to create a bipedicled flap. Only normal-appearing CSF was aspirated on penetrating the dura. The marsupialization was created by suturing both dura mater flaps to their adjacent musculature with 5-0 polypropylene in a loose simple continuous pattern. A small midline myelotomy was then made with a No. 11 blade until the middle of the cystic lesion was reached. A large amount of blood-tinged fluid exuded from the cavity, but no active bleeding was observed. The myelotomy was continued sagittally by blunt dissection and separation of the axons over 2.5 cm by use of 2 Freer periosteal elevators. A 0.8-cm-diameter organized blood clot was removed from the cavity, and further debridement allowed the removal of roughly a third of a poorly distinguishable cavity lining. No clear plane could be identified between the possible lining and neural tissue. The rest of the cavity wall was then left intact because excessive trauma to the cord would have been necessary for more complete resection. Multiple samples were sent for histologic analysis. The sample was not cultured, and CSF was not submitted for analysis. The spinal cord was collapsed and severely atrophied along the length of the cavitory lesion. The epaxial musculature, subcutaneous tissue, and skin were closed routinely, and the dog’s recovery from anesthesia was unremarkable. Special attention was paid to the dog’s breathing pattern and blood gas tensions because of the presence of cervical myelopathy. Respiratory patterns were normal, and the arterial partial pressure of oxygen was within reference limits during the recovery, but arterial oxygen saturation was 88%. Consequently, oxygen supplementation was provided through a nasal tube (at a fraction of inspired oxygen of 50%) for 12 hours and then discontinued successfully.

The biopsy specimen was in several small fragments. Fragments were processed, sectioned, and then stained with H&E (Figure 3). The largest piece was a coagulum of blood that was attached to the inner aspect of a cavitory structure. The blood clot was not laminated. Hemosiderin-laden macrophages were at the periphery. A small amount of white matter of the spinal cord was present in another fragment, and it was attached to the wall of the collapsed cavity. This wall was approximately 15 µm thick, amorphous, and hyaline with occasional flattened elongated nuclei spread at irregular intervals along the inner surface. There did not appear to be a distinct endothelial lining, and on the basis of MRI, surgical, and histologic findings, a hematoma within an intramedullary arachnoid cyst seemed most likely.

Immunohistochemistry was performed in an attempt to confirm this diagnosis and to exclude other possibilities (Figure 5). Examination for FVIIIRa was performed. Examination for pancytokeratin, cytokeratin 18, vimentin, and CD31 was also performed. The wall of the cavitory structure was strongly positive for FVIIIRa. Cells lining the structure were positive for CD31, and the wall was positive for vimentin. No staining for pancytokeratin and cytokeratin 18 was observed. These findings indicate the presence of endothelium, thus ruling out arachnoid cyst and making the cavitory lesion a vascular structure. Also observed in the FVIIIIRα- and CD31-positive specimens were cells forming occasional irregularly sized and thin-walled structures in the superficial portion of the blood clot.

During the first 2 days after surgery, neurologic examination was complicated by the heavy sedation provided by continuous rate infusions of fentanyl (5 µg/kg/min [2.5 µg/lb/min], IV) and lidocaine (25 µg/kg/min [12.5 µg/lb/min], IV). Passive range of motion to the 4 limbs was started the day after surgery and consisted of 4 daily sessions lasting 20 minutes each. After weaning from the fentanyl and lidocaine continuous rate infu

Figure 3—Sagittal FLAIR MRI view of the cervical portion of the spinal cord. The image has been rotated, as the dog was in dorsal recumbency at the time of imaging. The fluid in the nondependent portion of the lesion (arrows) remained hyperintense, indicating a proteinaceous composition.

Figure 4—Transverse follow-up MRI views of the spinal cord at the cranial aspect of C7, 26 days after initial images. The image has been rotated, as the dog was in dorsal recumbency at the time of imaging. On the T2w image (A), the cavitory, expansile intramedullary structure is seen (white arrows). The smaller dependent region (*) is still mildly hypointense relative to the spinal cord. On the intermediated weighted proton-density image (B), thinning of the spinal cord tissue dorsally and on the right (white arrows) are seen.
sions, the dog was nonambulatory, with the absence of purposeful movements and intact deep nociception in all 4 limbs, severely decreased withdrawal reflexes to the thoracic limbs, and normal patellar reflexes. Fecal continence was present, and a urinary catheter precluded the evaluation of micturition. Purposeful movements were seen for the first time in both pelvic limbs 6 days after surgery, so the urinary catheter was removed. For the next 3 weeks, passive- and active-assisted range of motion was performed 4 to 5 times daily, with sessions lasting approximately 30 minutes each. A cart and support slings were used to facilitate the active sessions. Four weeks after surgery, the dog regained the ability to walk unassisted. The dog was released with instructions to follow the same regimen of passive and active physical rehabilitation at home until reevaluation. Reevaluation was first performed 10 weeks after the surgery; the patient had clinical signs that were similar to those at the time of referral to the hospital, but without noticeable neck discomfort or knuckling on the right pelvic limb. One year after surgery, the dog was actively running and jumping, with normal thoracic limb function and a mild residual ataxia observed in the pelvic limbs.

Discussion

Focal intramedullary cavitary spinal cord lesions in dogs are rare and include focal congenital (primary) syringohydromyelia, acquired (secondary) syrinx-cavitation formation, epidermoid or dermoid cyst, abscess, vascular malformation, and cystic neoplastic process.\(^1\)\(^-\)\(^6\) Differential diagnoses in humans additionally include neurocysticercosis, ependymal cysts, neuroenteric cysts, and arachnoid cysts.\(^7\)\(^-\)\(^11\) In the dog presented here, although the histopathologic findings initially suggested an intramedullary arachnoid cyst, immunohistochemistry confirmed a vascular malformation.

Our preliminary diagnosis of intramedullary arachnoid cyst has not been described for dogs and is exceedingly rare in humans, with only 4 instances reported.\(^10\)\(^-\)\(^13\) Independent of its origin, the diagnosis of an arachnoid cyst is usually confirmed through histologic evaluation, where a cystic or multicystic cavity lined by mesothelial-like cells is observed with an absence of either an epithelial (ependymal) or endothelial lining. Cells lining an arachnoid cyst stain positive for vimentin and epithelial membrane antigen and variably stain positive for cytokeratin.\(^14\)\(^,\)\(^f\) Immunohistochemical staining is useful in differentiating CNS cysts in humans\(^15\) and has been described for dogs to characterize an intramedullary hemangioblastoma\(^15\) and intramedullary\(^16\) and cerebral hamartomas.\(^17\) The final diagnosis of a cavitary vascular anomaly in our dog was heavily based on the cells lining this lesion staining positively for FVIII:Ra (also called platelet endothelial cell adhesion molecule),\(^18\) a molecule found only on endothelial cells, neutrophils, and macrophages. Staining for FVIII:Ra is also supportive of a vascular structure; however, FVIII:Ra staining is known to occur in hematoma walls, when the antigen is absorbed from blood by the surrounding tissue. Epithelial membrane antigen staining was not performed in the dog of this report, as it is technically difficult and inconsistent. Distention of the cavity is presumed to have resulted in flattening and stretching of the endothelium, making the endothelial cells inapparent on routinely stained sections. The serosanguinous fluid seen at surgery may have been the result of retraction of a clot in the cavity and no further inflow of blood. The presence of a wall with endothelial cells excludes syringohydromyelia. The lack of staining with pancytokeratin and cytokeratin 18 further reduces the likelihood of the lesion being an arachnoid cyst.

Magnetic resonance imaging allows for more complete noninvasive evaluation of spinal cord lesions than
forms in humans commonly feature a T1w and contrast enhancement. In addition, these malformations are not separated by neural tissue and because further manipulations could not be identified between the possible lining and neural tissue and because further manipulations were considered overly traumatic. The surgical technique was moderately demanding, and magnification was mandatory. In conclusion, an intramedullary vascular anomaly was detected by use of MRI and contrast examination. The absence of a signal void on both T1w and T2w images, in addition to the lack of contrast enhancement, ruled out high- and low-velocity blood flow. The small structure diagnosed as a blood clot was hypointense on T2w and STIR images and hyperintense on T1w images, indicating early subacute hemorrhage. The important imaging, histologic, and immunohistochemical features of this vascular lesion were the intramedullary location, solitary cavity, apparent lack of active blood flow, apparent absence of epithelial lining, and positive staining results for endothelial cells. However, these characteristics cannot be strictly correlated with any of the vascular lesions described in dogs or humans. In humans, a simplified classification scheme proposed for vascular lesions of the spinal cord includes neoplasms (hemangioblastomas and cavernous malformations), aneurysms, and arteriovenous lesions (arteriovenous malformations). In dogs, hemangioblastoma, cavernous malformations (classified hamartoma and cavernous angioma), arteriovenous malformations, and aneurysms have been recognized. Of these, hemangioblastoma, unclassified hamartoma, cavernous angioma, and arteriovenous malformation* can be intramedullary. In the dog of this report, the lack of multiple dilated vessels with active blood flow and the lack of substantial hemorrhage from the lesion at surgery exclude patent arteriovenous malformation. Hamartoma is also excluded on the basis of the lack of multiple vessels and the lack of contrast enhancement. Hemangioblastoma is excluded by the absence of neoplastic cellular features and contrast enhancement. Cavernous angioma (cavernous hemangioma and cavernous malformation, cavernoma) is an entity in which many vascular spaces formed by variably sized, thick-walled vessels that are not separated by neural tissue are found and is found in dogs in the cerebrum and spinal cord. Although the histological appearance of this lesion could be part of such a malformation, the presence of a large single cavernous space is not typical. In addition, these malformations in humans commonly feature a T1w and T2w low-intensity rim, representing hemosiderin, and contrast enhancement. A final possibility is that thrombosis within the vascular lesion altered the MRI appearance of this lesion and could explain the lack of contrast enhancement and absence of blood flow.

Pertinent surgical options to treat cystic and cavernous lesions (intradural, extramedullary, or intramedullary) in humans and dogs include total excision, subtotal excision, multiple fenestrations, and marsupialization. Although total excision of a cystic lining is recommended when possible to minimize risks of recurrence, a subtotal excision or multiple fenestrations with or without dura mater marsupialization are often elected by surgeons to avoid excessive trauma to the spinal cord. Attempts to entirely remove a cyst wall adhered to the spinal cord parenchyma can lead to permanent neurologic deficits. Furthermore, although recurrence after complete surgical removal is thought to be less frequent, compared with subtotal removal, because of the rarity of disease, exact recurrence rates for each technique are unknown. Also, in 17 dogs with intradural or extramedullary subarachnoid cysts, factors associated with good outcome included marsupialization as the surgical technique, age < 3 years old at surgery, and < 4 months’ duration of clinical signs. With increasing age at surgery and a longer duration of clinical signs, one can assume that progressive atrophy of the spinal cord precludes a full recovery. In the dog described herein, it is interesting to see that only a mild residual ataxia was present in the pelvic limbs at a year after surgery, and only 10% of spinal cord cross-sectional area was occupied by axons on the preoperative MRI images. It was previously reported that as few as 5% to 10% of axons surviving within a lesion could allow functional recovery. The reason may be that the CNS is able to undergo plastic-type alterations in its circuitry to improve its efficacy in impulse transmission. Marsupialization was chosen for the dog of this report because of its potential link with a better prognosis. We recommend that the suture pattern in the dura mater not be too tight to minimize the risks of tearing and pull out during movement of the head. Subtotal cyst resection was performed because no clear plane could be identified between the possible lining and neural tissue and because further manipulations were considered overly traumatic. The surgical technique was moderately demanding, and magnification was mandatory. In conclusion, an intramedullary vascular anomaly was detected by use of MRI and confirmed on the basis of histologic findings and immunohistochemical results and was successfully treated by use of a surgical marsupialization technique in the dog of this report.

a. Metacam, Boehringer Ingelheim Canada Ltd, Burlington, ON, Canada.
b. GE EchoSpeed LX, Milwaukee, Wis.
c. Prolene, Ethicon, Somerville, NJ.
d. Prairie Diagnostic Services, Saskatoon, SK, Canada.
f. Fentanyl, Hospira Healthcare Corp, Vaughan, ON, Canada.
g. Lidocaine, Denis Giroux, Saint-Hyacinthe, QC, Canada.


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References