Ischemia and infarction of the spinal cord is a known cause of acute spinal injury in dogs. Currently, the diagnosis of spinal cord infarction in small animals is based on history, clinical signs, and the exclusion of other differentials with radiography and myelography. It is a diagnosis only confirmed through necropsy examination of the spinal cord. The aim of this paper is to describe the Magnetic resonance imaging (MRI) findings of the spinal cord of dogs with suspected spinal cord infarcts to utilize this technology for antemortem support of this diagnosis. This retrospective study evaluated the spinal MR examinations of 11 dogs with acute onset of asymmetric nonpainful myelopathies. All patients except one (imaged at 2 months) were imaged within 1 week of clinical signs and managed conservatively with minimal medical and no surgical intervention. They were followed clinically for a minimum of 4 months after discharge. MR findings in all dogs were characterized by focal, intramedullary, hyperintense lesions on T2-weighted images with variable contrast enhancement similar to what is reported in humans. Though it could not be used to diagnose spinal cord infarction definitively, MRI was useful in excluding extramedullary spinal lesions and supporting intramedullary infarction as a cause of the acute neurologic signs. Together with the history and clinical examination findings, MRI is supportive of a diagnosis of spinal cord infarction. Veterinary Radiology & Ultrasound, Vol. 46, No. 3, 2005, pp 225–229.

Key words: canine, infarct, ischemia, MRI, spinal cord.

**Introduction**

Infarction of the spinal cord has been documented in several species including man, dog, cat, pig, horse, turkeys, and tayra. Fibrocartilage believed to originate from the nucleus pulposus of the intervertebral disc is the most commonly recognized cause of spinal cord infarction in these species, though vascular occlusion secondary to atherosclerosis, thrombi, bacterial emboli, parasitic emboli, and neoplastic emboli are also potential causes. Typically, affected animals have acute asymmetric spinal cord disease that is nonpainful and nonprogressive after the initial 24 h. The degree of recovery depends on the severity of the initial injury to the spinal cord. The diagnosis of spinal cord infarction is presently one of exclusion. It relies on the signalment, history, neurologic findings, and an absence of specific pathology on survey radiographs, myelography, and computed tomography (CT) to distinguish it from intervertebral disc disease, neoplasia, and trauma.

Magnetic resonance (MR) imaging has been used in humans since 1989 to support the suspicion of spinal cord infarctions. MR imaging (MRI) in human spinal cord infarction is most commonly characterized by a hyperintensity within the gray matter of the spinal cord on T2-weighted images. Contrast enhancement on T1-weighted images is commonly seen, developing most clearly 1 week after an acute infarct, and usually resolving within a month’s time. A literature search failed to produce any publication describing MRI of suspected infarcts in veterinary patients.

**Methods**

Medical records were searched retrospectively to identify dogs presented to the Animal Health Trust from April 2000 to July 2002 with a presumptive diagnosis of spinal cord infarction and MR images evaluation of the spine. Dogs with evidence of intervertebral disc protrusion at the intervertebral disc spaces directly beneath or immediately cranial or caudal to the spinal cord lesion on the MRI were excluded. Additionally, any patients that had been given prolonged courses of antibiotics or steroids at the time of diagnosis were excluded due to the possibility that their recoveries were due to treatment of inflammatory or infectious diseases of the spinal cord. All patients had acute...
focal neurological signs localized to the cervical or thoracolumbar spinal cord. Dogs that received intravenous steroids prior to referral without further steroid administration were not excluded from the study as it was felt this initial dose would not be enough to completely resolve a primary inflammatory disease of the spinal cord without relapse over the (minimal) 4-month follow-up period. Two dogs were euthanized and had spinal cord infarction confirmed at necropsy. Hematology and serum biochemistry results were available for three patients (4,8,10), and titers (paired cerebrospinal fluid (CSF) and serum) for Canine Distemper Virus were evaluated in two patients (11). Titers for Toxoplasma and Neospora were evaluated in three patients (4,10,11), and titers (paired cerebrospinal fluid (CSF) and serum) for Canine Distemper Virus were evaluated in patient 11 as well.

For MRI, all patients were premedicated with diazepam* (0.23–0.32 mg/kg) or immediately induced with intravenous propofol† (to effect, up to 8 mg/kg). All patients were intubated and maintained on inhalant isoflurane anesthesia throughout the MR scanning, which was performed using a 1.5 Tesla GE Signa echospeed MRI scanner.‡ T1-weighted spin echo (SE), T2-weighted FSE, and contrast-enhanced§ T1-weighted SE images were obtained in the sagittal plane. T2-weighted FSE dorsal and transverse images of the affected area of the spinal cord were also obtained. Additional imaging sequences were performed at the discretion of the radiologist. MR images were evaluated for evidence of spinal cord compression, degenerate intervertebral discs, and parenchymal lesions. The animals were grouped into early (imaged within 48 h of clinical onset; seven dogs), middle (imaged 1 week after clinical onset; three dogs), and late (imaged 3 months after clinical onset; one dog) groups to try to correlate the lesion appearance between these three stages of infarct aging (see Table 1). While under general anesthesia for MRI, CSF was collected via lumbar puncture for protein and cell content analysis.

### Results

Eleven dogs were identified that met the selection criteria. The mean age at presentation was 5.5 years (median 4 years, range, 3.5–10 years). Breeds included Labrador Retriever (five), and one each of Staffordshire Bull Terrier, Bearded Collie, Basset Hound, Golden Retriever, Yorkshire Terrier, and Cross-breed dog. The majority of dogs were male (six intact, three neutered). Two of the dogs were neutered females. Five of the patients had been given steroids by the referring veterinarians at the onset of clinical signs. None of the patients were continued on steroid medication or antibiotics once admitted to the referral institution with the exception of one dog that had been receiving oral antibiotics (Amoxicillin with clavulanic acid, 18 mg/kg b.i.d.) for an unrelated tail injury for 2 weeks prior to presentation. The course of antibiotics in this patient was continued until the tail wound healed 2 weeks after presentation.

Five of the dogs were referred the same day that the neurologic signs developed and were imaged within 24 h of clinical onset. Two additional dogs were imaged within 48 h of clinical onset. Three dogs were not referred until 1 week after onset of clinical signs, and one dog was referred 3 months after an acute onset paraparesis because the neurological deficits had not completely resolved. Nine dogs had clinical improvement beginning as early as 48 h from the initial trauma, and there was continued improvement over several weeks. This is consistent with the described clinical course of spinal infarction where improvement, if noted, is recognized within 10–14 days.2,7,9

Dog 4, a 3.5-year-old Labrador Retriever that presented with paraplegia and absent deep pain in the left pelvic limb, was euthanized 4 days after admission due to poor prognosis resulting from persistent urinary incontinence and failure to recover deep pain sensation in the left pelvic limb. Spinal cord infarction secondary to fibrocartilaginous embolism as well as cord swelling and areas of liquefaction necrosis were confirmed in the lumbar spinal cord postmortem. Dog 5, a 4-year-old Labrador Retriever that presented with nonambulatory tetraparesis, was euthanized 1 week after presentation due to failure to recover adequate function and poor overall prognosis. Myelomalacia of the caudal cervical and cranial thoracic segments with the most severe changes seen at the level of C6 was confirmed postmortem. Microscopically, there was fibrocartilaginous material in the vessels around the area of necrosis.

In the early group imaged (within 48 h of clinical onset), there was some variability in the appearance in T1-weighted images with one lesion having areas of hypointensity and hyperintensity (dog 4). A hypointense lesion was noted

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**Table 1. Magnetic Resonance Imaging Appearance Grouped Into Age of Suspected Infarct**

<table>
<thead>
<tr>
<th>Time</th>
<th>T2-weighted images</th>
<th>T1-weighted images</th>
<th>Contrast-enhanced T1-weighted images</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early &lt;48 hours (7 dogs)</td>
<td>Hyperintense</td>
<td>Isointense (4)</td>
<td>Mild enhancement (2)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hyperintense(2)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Mixed</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Intensities (1)</td>
<td></td>
</tr>
<tr>
<td>Middle 1 week (3 dogs)</td>
<td>Hyperintense</td>
<td>Isointense</td>
<td>Enhancement (2)</td>
</tr>
<tr>
<td>Late 3 months (1 dog)</td>
<td>Hyperintense</td>
<td>Hyperintense</td>
<td>No enhancement</td>
</tr>
</tbody>
</table>

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*Diazepam: Diazemuls® Dumex, Barnstaple, UK.
†Propofol: Rapinovet™ Schering-Plough Animal Health, Welwyn Garden City, UK.
‡1.5 Tesla Signa echospeed MRI, General Electric Medical Systems, Milwaulk, WI.
§Gadolinium: Omniscan® (gadodiamide), Nycomed, Oslo, Norway.
in dog 3 dorsal to the central canal and medial to a lesion appearing hyperintense on T2-weighted FSE images in left dorsal gray matter, which may be due to edema next to the area of direct injury to the cord. There was mild contrast enhancement of the lesion in two of these dogs. The middle-aged infarct suspects (1 week) had focal hyperintensity on T2-weighted FSE scans, and two of these dogs had homogenous contrast enhancement of the lesion. T1-weighted images were isointense in all middle-aged lesions. The late-imaged suspected infarct (3 months) had regions of hyperintensity on T2-weighted FSE images as well as a region that was hyperintense to normal spinal cord on T1-weighted images. Though T1-weighted images had some variability in the lesion appearance, all lesions imaged were hyperintense on T2-weighting at all time periods. Contrast enhancement was most consistent in the middle-aged lesions (2/3), though more dogs are needed to verify this trend.

Lesions in the parenchyma of the cord mostly affected the gray matter, though some of the lesions extended to the border of the white matter. There was asymmetry in all the lesions that directly correlated with the asymmetry seen on clinical neurologic examination. Four of the dogs had clinical signs that were worse on the right side at presentation, and the lesion seen on MR images was also more pronounced on the right. Five dogs that had left-sided localization on examination also had more pronounced MR abnormalities on the left. There were two dogs that had symmetric pelvic limb neurologic deficits on presentation at 1 week and 2 months postonset. In one, the spinal cord lesion was centrally located, but extended slightly to the left. The other had no specific asymmetry on MR images. Three dogs had involvement of C6-T2 spinal cord segments, seven dogs had T3-L3 lesions, and one case had involvement of the spinal cord segments L4-caudal.

CSF analysis was abnormal in several dogs. Five of the seven dogs (71%) evaluated within 48 h had elevation in cerebrospinal protein content (mean, 1.22 g/l; reference range, 0–0.45 g/l). These dogs also had elevated cell counts ranging from 8 to 12 cells/ul (reference <5). One dog evaluated 1 week after onset of clinical signs also had an elevated protein of 1.04 g/l.

Hematology and serum biochemistry were available for three of the patients and there were no abnormalities. Ser um titers to Toxoplasma and Neospora were negative in three dogs tested. Canine Distemper Virus analysis in dog 10 was compatible with vaccination for the disease.

Discussion

Infarction of the spinal cord is an area of necrosis resulting from decreased blood supply (ischemia). MRI has been used to support the diagnosis of spinal cord infarction in man since 1989. Since that time, over 100 patients with MRI of suspected or confirmed spinal cord infarction have been described. Though MRI has been available for some time in veterinary medicine, and spinal cord infarction has been a recognized pathology since 1973, there have been no reports published in the veterinary literature regarding the MR appearance of suspected spinal cord infarction in dogs.

In all dogs presented herein, MRI was useful in excluding extradural compression as the cause of the transverse myelopathy. The MR images were characterized by asymmetric lesions within the gray matter of the spinal cord, which correlated with the clinical neurolocalization and lateralization in each dog. All the dogs, regardless of time lapsed since clinical signs began, had focal, intramedullary hyperintensities on T2-weighted FSE images (Fig. 1). Further evaluation of the lesions using inversion recovery to eliminate pure fluid confirmed the intramedullary nature of the hyperintensities (Fig. 2). This finding is believed to be related initially to cytotoxic or vasogenic edema and later to gliosis. Humans suspected of having spinal cord infarcts consistently have T2-weighted hyperintensity as early as 9 h and as late as 2 months after clinical presentation. In a review by Fortuna et al., 93% of lesions were hyperintense on T2-weighting (the other 7% were isointense). Seventy percent were isointense on T1-weighting (19% were hypointense and 11% were hyperintense). Forty percent had contrast enhancement (Fig. 3), with the majority occurring in lesions imaged 5–6 days after clinical onset. However, the imaging data were not consistently separated into early, middle, and late imaged lesions.
Many other reports in human medical literature document a lack of contrast enhancement in the first week of spinal cord injury, with enhancement developing around week after the injury, and absence of enhancement by month.\textsuperscript{18,19,22–24} This is consistent with documented changes from cerebral infarcts, where enhancement increases in infarcts aged 8–14 days and decreases in infarcts older than 15 days.\textsuperscript{25,26} Experimentally, the blood–brain barrier has been shown to break down within 6 h of ischemia. However, the complete lack of blood supply to the area normally prevents contrast medium delivery to the tissue until it is revascularized, which experimentally takes 5–7 days.\textsuperscript{25} Some variability in the timing of contrast enhancement may therefore be expected and depends upon the degree of interruption to the blood supply to the nervous tissue and the rate of revascularization. Contrast enhancement in two of the early infarcts in this report may be due to early revascularization or leaking of contrast agents through damaged capillaries in regions where there is less than complete occlusion of blood supply.\textsuperscript{26}

In a review of 161 cases of confirmed and suspected fibrocartilaginous embolism (FCE) published in the animal literature, 19 (11.8%) localized to C1-C5, 35 (21.7%) to C6-T2, 54 (33.5%) to T3-L3, and 53 (32.9%) to L4-caudal.\textsuperscript{3–8,10,11} There is a greater proportion of thoracolumbar and lumbosacral distribution in series that include unconfirmed infarction than in reports which include only pathologically confirmed infarction. It is likely that the data in reports with pathology as an inclusion criteria have skewed data because euthanasia is more likely in animals with more extensive disability, such as tetraparesis from a cervical infarct compared with paraparesis from a thoracolumbar infarct. In this series, three (27%) localized to C6-T2, seven (63%) to T3-L3, and one (9%) to L4-caudal. There are not enough dogs in this report to make conclusions regarding the true distribution of lesions. However, there may be a greater percentage of thoracolumbar lesions in this series as a pathologic diagnosis was not part of the inclusion criteria.

The majority of lesions (9/11) had an asymmetrical appearance, which corresponded to the clinical presentation of the dog. The spinal cord vascular anatomy is likely responsible for the asymmetry seen in ischemic myelopathy. The arterial supply to the canine spinal cord consists of a ventral spinal artery with a central branch that extends through the ventral fissure then branches to supply the intermediate and ventral gray matter of the spinal cord and surrounding white matter unilaterally or bilaterally through discrete sections of cord. There are also two paired dorsolateral spinal arteries that supply the dorsal gray matter and dorsolateral white matter, as well as radicular arteries around the spinal cord that supply discrete regions of the white matter. All arteries below the surface
of the spinal cord are considered functional end arteries, and occlusion of any of these branches leads to neuronal death in its supplied territory. The resulting spinal cord damage leads to asymmetrical clinical signs reflecting the region of the spinal cord supplied by the individual vessel. Venous occlusion in the peripheral internal plexi that feed into an arcuate vein around the spinal cord may also lead to asymmetrical injury. Both venous and arterial occlusions from embolic material have been reported in the dog. In human literature, the anterior (ventral) spinal artery is often the source of infarction leading to predominantly gray matter injury, as was seen in this case series.22,23,27 The higher metabolic demand of gray matter compared with white matter may also contribute to the findings of predominantly gray matter injury.

MRI is helpful in supporting a suspicion of spinal cord infarction based on history and clinical examination findings. In addition to excluding extradural causes of myelopathy, findings of asymmetric T2-hypointensity in the gray matter are supportive of spinal cord infarction. Though a final diagnosis can only be made based on histopathologic assessment of the spinal cord, this result is often unavailable in patients with spinal cord infarction due to the favorable prognosis for recovery.

REFERENCES