

# Fibrocartilaginous Embolic Myelopathy in Small Animals

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## KEYWORDS

- Fibrocartilaginous embolic myelopathy • Ischemic myelopathy
- Dog • Cat

Fibrocartilaginous embolic myelopathy (FCEM) has been reported commonly in dogs<sup>1–9</sup> and sporadically in several other species, including humans,<sup>10</sup> cats,<sup>11,12</sup> horses,<sup>13</sup> pigs,<sup>14</sup> turkeys,<sup>15</sup> a lamb,<sup>16</sup> a calf,<sup>17</sup> a tiger,<sup>18</sup> a tayra,<sup>19</sup> and a pigtail macaque.<sup>20</sup> FCEM occurs when fibrocartilaginous material histologically and histochemically identical to the nucleus pulposus of the intervertebral disc occludes the spinal vasculature, causing ischemic necrosis of dependent regions of spinal cord parenchyma. Neurologic signs are peracute in onset and have distribution and severity referable to the site and extent of the spinal cord infarction. Neurologic signs usually stabilize within 24 hours, and subsequently remain static or improve depending on the severity and extent of the ischemic insult.

## PATHOPHYSIOLOGY

The various hypotheses on the pathogenesis of FCEM are best understood after reviewing the vascular anatomy of the spinal cord.

### *Vascular Anatomy of the Spinal Cord*

The arterial supply of the spinal cord originates from spinal branches of the paired vertebral arteries in the cervical spine, intercostal arteries in the thoracic spine, and lumbar and sacral arteries in the lumbar and sacral spine, respectively.<sup>21,22</sup> The paired spinal branches enter the vertebral canal through the intervertebral foramina, penetrate the dura mater, and divide into dorsal and ventral radicular arteries. Some of these supply only the nerve roots, whereas others also contribute to the anastomotic

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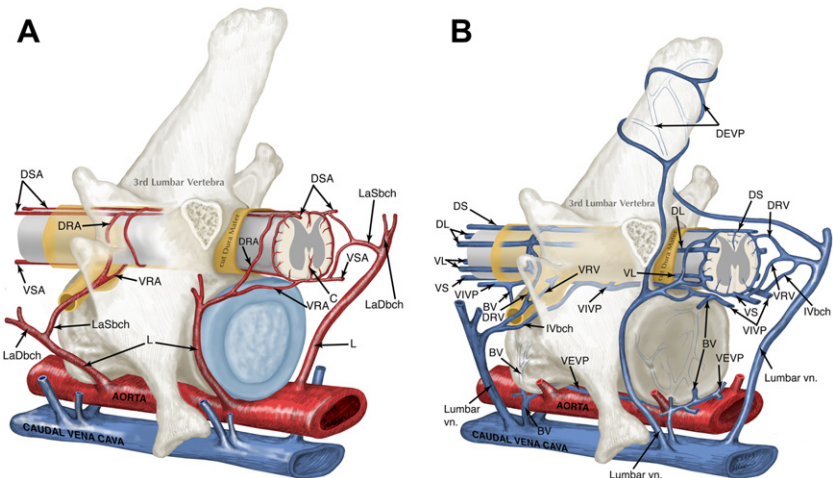
plexus on the surface of the spinal cord and to the dorsal and ventral spinal arteries (**Fig. 1A**).

The percentage of ventral radicular arteries contributing to the unpaired ventral spinal artery has been reported to be 88.8% in the cervical region, 31.2% in the thoracic region, and 45.0% in the lumbar region of the spinal cord.<sup>23</sup> The ventral spinal artery extends the entire length of the spinal cord along the ventral median fissure, is larger in diameter at the cervical and lumbar regions than in the thoracic region, and gives rise to the central arteries.<sup>23</sup>

The central arteries supply most of the gray matter and part of the lateral and ventral white matter of the spinal cord, sending branches to each side, or alternatively and irregularly to the left or right side of the spinal cord. The mean percentage of central arteries that send branches bilaterally has been reported to be 21% in the cervical region, 42% in the thoracic region, and 63% in the lumbar region of the spinal cord.<sup>23</sup> The asymmetric distribution of the central artery at several spinal cord segments explains why its embolization can result in unilateral ischemia/infarction and lateralized neurologic deficits.

The percentage of dorsal radicular arteries contributing to the paired dorsal spinal arteries has been reported to be 88.1% in the cervical region, 49.6% in the thoracic region, and 60.0% in the lumbar region of the spinal cord.<sup>23</sup> The dorsal spinal arteries are continuous throughout the spinal cord, have a larger diameter in the cervical and lumbar regions than in the thoracic region, and supply the dorsal white and gray matter.<sup>23</sup> The anastomotic plexus on the surface of the spinal cord gives rise to radial arteries that enter the spinal cord and supply the lateral and ventral white matter.<sup>22</sup>

The venous drainage of the spinal cord consists of several intraparenchymal little veins distributed in a radial pattern to a network of veins on the surface of the spinal



**Fig. 1.** (A) Arterial supply to the lumbar spinal cord. C, central artery; DRA, dorsal radicular artery; DSA, dorsal spinal artery (paired); L, lumbar artery; LaDbch, lumbar artery dorsal branch; LaSbch, lumbar artery spinal branch; VSA, ventral spinal artery; VR, ventral radicular artery. (B) Venous drainage of the lumbar spinal cord. BV, basivertebral vein; DEVP, dorsal external vertebral venous plexus; DL, dorsolateral vein (paired); DRV, dorsal radicular vein; DS, dorsal spinal vein; IVbch, intervertebral vein branch; VEVP, ventral external vertebral venous plexus; VIVP, ventral intervertebral venous plexus; VL, ventrolateral vein (paired); VRV, ventral radicular vein; VS, ventral spinal vein.

cord (see **Fig. 1B**).<sup>22,24</sup> These veins drain into the ventral internal vertebral venous plexus, which mainly consists of two valveless large veins on the floor of the vertebral canal. These veins converge at mid-vertebral body level, occasionally anastomose, and diverge over the intervertebral disc. They are connected with the basivertebral veins that drain the vertebral bodies. The ventral internal vertebral venous plexus drains at the level of the intervertebral foramina via branches of the intervertebral veins into the major veins of each region of the spine (vertebral, azygos, and caudal vena cava in the cervical, thoracic, and lumbar region, respectively).<sup>24</sup>

### ***Pathophysiology Hypotheses***

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The intraparenchymal (intrinsic) spinal cord arteries are functional end arteries and their occlusion results in ischemia of the territory supplied. In patients with FCEM, the embolizing material has been identified as fibrocartilage histologically and histochemically identical to the nucleus pulposus of the intervertebral disc. Several hypotheses have been proposed to explain how the fibrocartilaginous material, originating from the intervertebral disc nucleus pulposus, enters the spinal vascular system.

#### ***Hypothesis 1***

Direct penetration of nucleus pulposus fragments into spinal cord or vertebral vessels. Direct penetration into the venous system, which is more likely than into the arterial system because arteries have relatively thick muscular walls. However, an injection type of entrance through the arterial wall has been proposed.<sup>3</sup> Arteriovenous anastomoses have been shown in the epidural and periradicular space in dogs and humans and could explain the presence of emboli on either side of the circulation, regardless of whether the entry point is arterial or venous. In dogs with histologically confirmed FCEM, extruded degenerated disc material has been identified into the internal vertebral venous plexus adjacent to the affected spinal cord segment.<sup>1,25,26</sup> Increased intrathoracic and intraabdominal pressure during coughing, straining, exercise, or trauma (Valsalva's maneuver) could generate retrograde venous propulsion of the fibrocartilage into the intrinsic spinal arteries and veins.

#### ***Hypothesis 2***

Chronic inflammatory neovascularization (arterial and venous) of the degenerated intervertebral disc. In-growth of blood vessels within the degenerated annulus fibrosus has been documented in humans and in nonchondrodystrophic dogs with disc degeneration. A sudden rise in intervertebral disc pressure exceeding arterial blood pressure may result in penetration of nucleus pulposus fibrocartilage into the newly formed intervertebral disc blood vessels and progression into the intrinsic spinal cord vasculature. Fibrocartilage, histochemically identical to the nucleus pulposus, has been identified within newly formed blood vessels in the degenerated intervertebral disc of two dogs with histologically confirmed FCEM.<sup>26</sup>

#### ***Hypothesis 3***

Presence of embryonic remnant vessels within the nucleus pulposus (which is normally avascular in adults). The hypothesized mechanism of entrance of the fibrocartilage into the intrinsic spinal cord vasculature is similar to the one described earlier.

#### ***Hypothesis 4***

Mechanical herniation of nucleus pulposus into the vertebral bone marrow sinusoidal venous channels, with subsequent retrograde entrance into the basivertebral vein

and internal vertebral venous plexus. This hypothesis also may apply to human beings because Schmorl's nodules (focal masses of fibrocartilage within the vertebral body cancellous bone) are not uncommon. However, Schmorl's nodules are extremely rare in dogs and have never been reported in dogs with histologically confirmed FCEM.

In addition, it has been hypothesized that fibrocartilage may arise from vertebral growth-plate cartilage in immature dogs or metaplasia of the vascular endothelium, which later ruptures into the lumen and embolizes within the intrinsic spinal cord vasculature.

The ischemic injury caused by the arterial obstruction initiates a series of biochemical and metabolic changes that result in neuronal and glial cell death (secondary spinal cord injury).<sup>27</sup> The gray matter is affected more severely than the white matter because of its greater metabolic demand.

### CLINICAL PRESENTATION

FCEM has been reported most commonly in large and giant breed dogs. However, it has been described also in small breed dogs (particularly miniature schnauzers)<sup>1–8</sup> and has been confirmed histologically in two chondrodystrophic breed dogs.<sup>6,28</sup> Approximately 80% of dogs with FCEM diagnosed antemortem or confirmed histologically had a body weight greater than 20 kg.<sup>1,2,5,8</sup> The reported male-to-female ratio has ranged from 1:1<sup>2</sup> to approximately 2.5:1<sup>4,8</sup> in different studies. The age at diagnosis in dogs has ranged from 2 months<sup>29</sup> to 11 years and 11 months,<sup>8</sup> with a median of 5 or 6 years in most studies.<sup>1,2,5,8</sup>

The typical clinical presentation is characterized by peracute (<6 hours) onset of nonprogressive and nonpainful (after the first 24 hours), and often asymmetric myelopathy. In 29%<sup>8</sup> to 80%<sup>2</sup> of dogs with an antemortem diagnosis of FCEM, and in 43%<sup>5</sup> to 61%<sup>2</sup> of dogs with histologic diagnosis of FCEM, the owner reported that the dog was performing some type of physical activity, such as walking, running, or playing, at onset of neurologic signs. Signs of sudden and transient hyperalgesia (eg, yelping as in pain) were observed by the owners at the onset of neurologic signs in approximately 50% of dogs in one study<sup>2</sup> and in only 12% of dogs in another study.<sup>5</sup> Most commonly, maximal neurologic deterioration occurs within the first 6 to 24 hours and is followed by gradual improvement or stabilization of signs, depending on the extent and severity of the ischemic injury.<sup>2,5,8</sup> Rarely, neurologic dysfunction can progress for longer than 24 hours, possibly from additional embolizations or secondary spinal cord injury.

Neurologic deficits vary depending on the location and severity of the spinal cord ischemic injury, and are asymmetric in 53% to 86% of dogs.<sup>1,2,5,8</sup> The most commonly affected spinal cord segments have been L4-S3 (43%–47%) and C6-T2 (30%–33%) in dogs with a histologic diagnosis of FCEM,<sup>2,5</sup> and L4-S3 (44%–50%) and T3-L3 (27%–42%) in dogs with an antemortem diagnosis of FCEM.<sup>2,5,8</sup> Commonly, neurologic deficits refer to the affected spinal cord segment (eg, C1-5, C6-T2, T3-L3, or L4-S3). However, in the acute stages of the disease, a decreased withdrawal reflex has been observed in severely paraparetic and paraplegic dogs, with MRI changes consistent with FCEM within the T3-L3 spinal cord segment.<sup>8</sup> Most of these dogs had an interruption of the cutaneous trunci reflex consistent with the site of FCEM on MRI.<sup>8</sup>

Experts have suggested that the transient depression or abolition of pelvic limb spinal reflexes in dogs with acute thoracolumbar spinal cord injury results from sudden interruption of descending supraspinal input on motor neurons and interneurons,

fusimotor depression, and increased segmental inhibition.<sup>30</sup> Focal mild to moderate hyperesthesia can sometimes be elicited on palpation of the affected spinal segments in dogs examined within the first few hours after onset of neurologic signs, although this rapidly resolves.

Most cats reported with FCEM have been domestic short hairs. Males and females were nearly equally represented. The age at onset of FCEM ranged from 4 to 12 years and most affected cats were older than 7 years.<sup>11,12</sup> Onset was acute in all reported cats, and nonprogressive after the first 24 hours of disease in most of them. However, clinical deterioration was observed over 2 to 5 days in a few cats with histologically confirmed FCEM. None of the reported cats had a history of trauma or exercise at the onset of disease and none had discomfort or hyperesthesia on spinal palpation. The most commonly affected spinal cord segments have been C6-T2; however, any spinal cord segment can be affected. All reported cats had asymmetric neurologic signs.

## DIFFERENTIAL DIAGNOSES

Material other than fibrocartilage, such as thrombi or bacterial, parasitic, neoplastic, or fat emboli, can obstruct the intrinsic spinal blood vessels and result in spinal cord ischemia and necrosis. The clinical presentation and MRI findings are very similar or identical to the ones that characterize FCEM. Underlying medical conditions that may predispose to embolization or thrombosis, including cardiomyopathy, hypothyroidism, hyperthyroidism, hyperadrenocorticism, chronic renal failure, and hypertension, should be considered and investigated, particularly in cats.

Another condition that results in peracute onset of nonprogressive (after the first 24 hours), and often asymmetric, myelopathy is the so-called acute noncompressive nucleus pulposus extrusion<sup>31</sup> or traumatic intervertebral disc extrusion.<sup>32</sup> This condition occurs when nondegenerated nucleus pulposus extrudes during strenuous exercise or trauma, contuses the spinal cord, and dissipates within the epidural space, causing minimal to no spinal cord compression. Discomfort or hyperesthesia during palpation of the affected spinal segments has been reported in 57% of dogs with acute noncompressive nucleus pulposus extrusion,<sup>31</sup> and represents the main clinical finding to differentiate this condition from FCEM because it generally persists for more than 24 hours. High-field MRI and experience in neuroimaging help differentiate these two diseases.<sup>7,8,31,32</sup> The MRI features of acute noncompressive nucleus pulposus extrusion include the presence of a focal intramedullary hyperintensity overlying a narrowed intervertebral disc, with reduced volume and signal intensity of the nucleus pulposus on T2-weighted fast spin echo (FSE) images, and extraneous material or signal change within the epidural space dorsal to the affected disc, with absent or minimal spinal cord compression.<sup>31,32</sup>

Other differential diagnoses include compressive intervertebral disc extrusion, infectious and immune-mediated focal myelitis, neoplasia, and intra- and extramedullary hemorrhage (eg, secondary to coagulopathy). History, clinical signs, disease progression, and diagnostic findings (particularly MRI and cerebrospinal fluid [CSF] analysis) allow these disorders to be differentiated from FCEM.

For patients with unknown or incomplete history, exogenous traumatic spinal injury (resulting in vertebral fracture, subluxation/luxation, spinal cord contusion, or hemorrhage) should be considered in the differential diagnoses list. Patients with exogenous traumatic spinal injury generally present with severe spinal hyperesthesia and should be manipulated minimally and carefully until survey spinal radiographs rule out an unstable lesion.

## DIAGNOSIS

The definitive diagnosis of FCEM can be reached only through histologic examination of the affected spinal cord segments. The antemortem diagnosis of FCEM is based on the typical clinical presentation (peracute nonprogressive after 24 hours, nonpainful, usually asymmetric myelopathy) and exclusion of other causes of peracute/acute focal myelopathy (through diagnostic imaging and CSF analysis). In addition, complete blood cell count, serum biochemistry, coagulation profile, and echocardiography can help rule out diseases that can predispose to thromboembolism, such as vasculitis and endocarditis.

### *Plain Radiographs*

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Plain radiographs of the spine help rule out vertebral fracture, subluxation/luxation, neoplasia, and osteomyelitis/discospondylitis.

### *Myelography*

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The main value of myelography in the antemortem diagnosis of FCEM is the exclusion of other causes of peracute/acute focal myelopathy, particularly those resulting in spinal cord compression, such as intervertebral disc extrusion. In dogs and cats with FCEM, myelography may be normal or may show an intramedullary pattern suggesting focal spinal cord swelling in the acute stage of the disease. This pattern has been observed in 39% to 47% of dogs with a histologic diagnosis of FCEM and in approximately 26% of dogs with an antemortem diagnosis of FCEM.<sup>2,5</sup> However, an intramedullary pattern may also be observed with other causes of myelopathy, including focal myelitis, intramedullary neoplasia, intraparenchymal hemorrhage, and acute noncompressive nucleus pulposus extrusion. Acute noncompressive nucleus pulposus extrusion should be suspected when the area of spinal cord swelling is above a collapsed intervertebral disc space (on good-quality radiographs with optimal patient positioning) and focal spinal hyperesthesia persists for more than 24 hours.

### *CT*

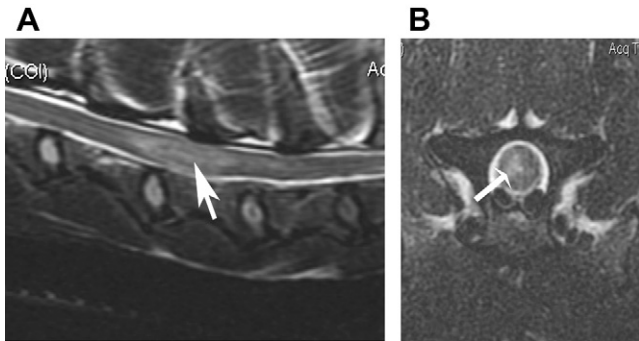
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CT can also help to rule out other causes of peracute/acute myelopathy. CT–myelogram may show an intramedullary pattern of spinal cord swelling in the acute stage of FCEM.

### *MRI*

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MRI of the affected spinal region is the preferred diagnostic imaging modality for the antemortem diagnosis of FCEM. In addition to excluding other causes of myelopathy, it allows visualization of signal intensity changes suggestive of ischemic infarction of the spinal cord. These changes include a focal, relatively sharply demarcated, and often asymmetric intramedullary lesion (oedematous infarcted tissue), predominantly involving the gray matter that appears hyperintense to normal spinal cord gray matter on T2-weighted FSE images, and iso- or hypointense to normal spinal cord gray matter on T1-weighted FSE images (**Fig. 2**).<sup>7–11</sup> Focal spinal cord swelling is a common finding.<sup>8</sup> Postcontrast T1-weighted FSE images may show various degrees of enhancement of the affected area, generally on the fifth to seventh day of disease.<sup>7–10</sup> Sometimes no intraparenchymal signal intensity changes are observed on MRI performed within 24 to 72 hours after onset of FCEM.<sup>8,33</sup> The degree and extent of the ischemic injury and the availability of high contrast resolution MRI influence the ability to detect signal intensity changes in the early stage of FCEM.<sup>8,34</sup> Dogs that are ambulatory on presentation are more likely to have a normal MRI than nonambulatory dogs.<sup>8</sup>



**Fig. 2.** (A) Mid-sagittal T2-weighted FSE image of a 9-year-old golden retriever with peracute onset of nonambulatory tetraparesis. An extensive hyperintense lesion (*arrow*) is seen within the spinal cord parenchyma over the vertebral body of C5. Histopathology confirmed a fibrocartilaginous embolus within the vasculature of the cord. (B) Transverse T2-weighted FSE image through the intramedullary hyperintensity depicted in A. The focal and relatively sharply demarcated lesion (*arrow*) involves the white and gray matter and is lateralized to the left.

The severity of neurologic dysfunction at initial examination has been associated with the longitudinal and transverse extent of the intramedullary ischemic lesions on MRI.<sup>8</sup> Repeated MRI studies have been used in humans to detect an ischemic lesion that was not apparent in the acute stage of the disease,<sup>34,35</sup> and may also be helpful in evaluating certain veterinary patients with suspected FCEM.

Diffusion-weighted (DW) MRI has been used in humans to increase the sensitivity and specificity for diagnosing spinal cord infarction in the early stage of the disorder.<sup>35</sup> DW MRI presents a technical challenge because of the relatively small size of the spinal cord (particularly in veterinary patients), low spatial resolution, and the magnetic susceptibility to artifacts caused by vascular and CSF pulsation.<sup>8,34,35</sup> Some of these technical problems may be overcome by using a multishot technique, which improves signal-to-noise ratio and is less sensitive to off-resonance effects.<sup>36,37</sup> DW MRI has shown increasing intramedullary hyperintensity from 1 to 24 hours postembolization in experimental dogs with spinal cord infarction induced by embolization of the spinal branches of T9-11 intercostal arteries bilaterally.<sup>37</sup>

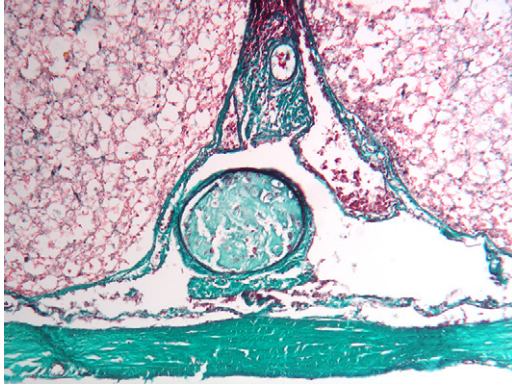
### **CSF Analysis**

CSF analysis may be normal or may reveal nonspecific abnormalities, including xanthochromia, mild to moderate pleocytosis (7–84 white blood cells per microliter) and elevated protein concentration. CSF abnormalities (mainly elevated protein concentration) have been reported in up to 46% of dogs with a histologic diagnosis of FCEM and in 44% to 75% of dogs with an antemortem diagnosis of FCEM.<sup>2,8</sup> Polymerase chain reaction for different infectious agents on CSF may help rule out specific causes of meningomyelitis.

### **Histologic Examination**

Histologic examination of the affected spinal cord segments shows fibrocartilaginous material in spinal vessels (arteries or veins) within or near an area of focal myelomalacia (**Fig. 3**). The distribution of the lesion reflects the territory of the embolized vessels and therefore is frequently asymmetric. The gray matter is generally more severely affected than the white matter. The lesion margins tend to be well delineated from





**Fig. 3.** Histopathologic transverse section of the midcervical spinal cord of a 13-year-old West Highland White Terrier with peracute nonambulatory tetraparesis. Intravascular fibrocartilaginous material is present within the ventral spinal artery and ischemic necrosis of dependent spinal cord parenchyma has occurred (Goldner stain,  $\times 320$ ). (Courtesy of Dr Carlo Cantile, University of Pisa, Italy.)

normal tissue. Infarcted areas are usually ischemic but sometimes may be accompanied by hemorrhage.

### TREATMENT

Treatment of FCEM involves reducing secondary spinal cord injury (maintenance of spinal cord perfusion, neuroprotection) in the acute stage of the disease, nursing care, and physiotherapy. In patients with neurologic impairment of ventilation (eg, severe cervical spinal cord lesions, impaired phrenic nerve function) or with concurrent cardiovascular or respiratory disease, systemic blood pressure, ventilation, and oxygenation should be monitored and maintained within normal limits to ensure adequate spinal cord perfusion.

Neuroprotective agents such as methylprednisolone sodium succinate (MPSS) and polyethylene glycol could help minimize secondary spinal cord injury. However, the clinical benefits of these drugs require further investigation in dogs and cats with spinal cord injury and there are increasing concerns about the adverse effects of MPSS. Nursing care and physiotherapy play an essential role in the management of patients with FCEM (particularly those with severe neurologic impairment), because they promote recovery and help prevent complications. Nursing care includes providing adequate bedding, regular turning, skin care to prevent decubital ulcers and urine scalding, care of the respiratory system (prevention/treatment of hypoventilation, aspiration pneumonia, pulmonary atelectasis), bladder and bowel management, and adequate nutrition. Physiotherapy stimulates neuronal plasticity and therefore maximizes functional recovery mediated by unaffected neural tissue, and minimizes disuse and immobilization changes such as muscle atrophy and muscle and joint contractures.

### PROGNOSIS

The prognosis for recovery in patients with FCEM depends on the severity and extent of the ischemic injury. Recovery rates have ranged from 58%<sup>1</sup> to 84%<sup>8</sup> in various studies, probably because of differences in inclusion criteria, severity and distribution



of ischemic lesions, definition of outcome, and owner's commitment.<sup>1,2,5,8</sup> In a recent study, 42 of 50 (84%) dogs had either a complete clinical recovery or a partial recovery compatible with life as a functional pet (eg, urinary and fecally continent, being able to perform daily activities without extra care from the owner).<sup>8</sup>

Negative clinical prognostic factors reported include loss of nociception,<sup>1,2,5</sup> lower motor neuron signs,<sup>1,2</sup> symmetric neurologic deficits,<sup>2,5</sup> severity of neurologic signs at initial examination quantified by a neurologic score,<sup>8</sup> owner's reluctance to pursue nursing care and physiotherapy (particularly in large or giant breed dogs),<sup>2</sup> and lack of improvement within the first 14 days.<sup>1</sup>

Outcome has been associated with the extent of the ischemic intramedullary lesion on MRI, defined as the ratio between the length of the intramedullary hyperintensity on mid-sagittal T2-weighted images and the length of the vertebral body (referred to as *lesion length–vertebral length ratio*) of C6 (in dogs with cervical lesions) or L2 (in dogs with thoracolumbar lesions), and as the cross-sectional area of the largest intramedullary hyperintensity on transverse T2-weighted images expressed as a percentage of the cross-sectional area of the spinal cord at the same level (referred to as *percent cross-sectional area of the lesion*). Dogs with a lesion length–vertebral length ratio greater than 2.0 or a percent cross-sectional area of the lesion of 67% or greater were significantly more likely to have an unsuccessful outcome than those with lower values for these parameters.<sup>8</sup>

In one study, the presence of CSF abnormalities (increased protein concentration  $\pm$  pleocytosis) was associated with a poorer outcome in dogs with FCEM.<sup>5</sup> However, a more recent study found no association between the presence of CSF abnormalities and outcome, or the extent of the ischemic intramedullary lesion on MRI.<sup>8</sup>

Intervals between onset of neurologic signs and recovery of voluntary motor activity, unassisted ambulation, and maximal recovery have been reported to be 6 days (range, 2.5–15 days), 11 days (range, 4–136 days), and 3.75 months (range, 1–12 months), respectively.<sup>8</sup> No statistically significant association has been identified between clinical or MRI variables and recovery times.<sup>5,8</sup>

Although reported cases are limited, prognosis for cats undergoing adequate nursing care for at least 2 weeks after onset of FCEM is likely to be as good as for dogs.<sup>11</sup>

## SUMMARY

Prognosis for dogs and cats with FCEM is generally favorable. Severity of neurologic signs at initial examination and extent of the lesions seen on MRI can help predict outcome in dogs with FCEM.

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