

Fibrocartilagenous embolic myelopathy in a cat

Patricia V. Turner, Dean H. Percy, Kay Allyson

Fibrocartilagenous embolic myelopathy is an uncommon syndrome of acute spinal cord ischemia of uncertain etiology. The clinical presentation may be similar to other acute conditions of the spinal cord, such as trauma or intervertebral disk prolapse. The disorder has been almost exclusively described in the dog, with a total of 64 cases reported in the literature between 1973 and 1992 (1); only 2 cases in cats have been reported (2,3).

A 12-year-old, male domestic shorthair was presented to the Ontario Veterinary College (OVC) with a 1-day history of acute left-sided hemiparesis.

The cat had been found recumbent that morning, on his left side, meowing. Upon initial examination by the referring veterinarian, he appeared to be anxious but not pained. Vital signs and a cranial nerve examination were normal. There was reduced nociception and weak withdrawal of the left forelimb. The cat had appeared well and active prior to this incident, vaccinations were current, and there was no history of recent trauma. Previous history included recurrent lower urinary tract infections, the last episode occurring 4 mo previously.

Thromboembolism and intervertebral disk prolapse were the initial clinical conditions considered, and treatment was initiated with the administration of 2 mg of dexamethasone sodium phosphate (Dexamethasone 2, Vetoquinol Canada, Joliette, Quebec), SC. Survey radiographs indicated no evidence of thoracic or spinal lesions, and an electrocardiogram was normal. Anisocoria of the left pupil was observed periodically throughout the day. The cat was referred to the OVC later that same evening.

On presentation, the cat was hypothermic and recumbent. The bladder was distended, and when expressed, contained light red discolored urine. Neurological examination revealed an alert animal with miosis and partial prolapse of the 3rd eyelid on the left side. He was unable to bear weight and purposeful movement was limited to the hind limbs, the left hind limb being weaker. Flexor reflexes were intact to the hind limbs, but patellar reflexes were difficult to elicit. There was no flexor response in the left forelimb and minimal movement of the right forelimb. Proprioception was normal in both pelvic limbs and reduced in the right forelimb, it was not assessed in the left forelimb due to catheter placement. Nociception was normal in the pelvic limbs, reduced in the right forelimb, and absent in the left forelimb.

In summary, there were upper motor neuron signs in the hind limbs and marked lower motor neuron signs in the forelimbs, consistent with lesions in the white and

grey matter of the spinal cord, respectively. It was concluded that there was a severe asymmetric spinal cord lesion at the level of the cervical intumescence. The presence of a left-sided Horner's syndrome also suggested involvement of the spinal cord segments or ventral roots of the 1st to 3rd thoracic spinal nerves (T₁-T₃). Differential diagnoses for clinical neurological signs exhibited by the patient at this point included neoplasia, myelitis, vascular accident (stroke), trauma, and intervertebral disk disease.

Diagnostic tests included a complete blood count, serum biochemical analysis, and urinalysis.

Hematologic analysis revealed a mild nonregenerative, normochromic, normocytic anemia (hematocrit 0.204; reference range 0.24 to 0.45) and moderate lymphopenia (lymphocytes $0.32 \times 10^9/L$; reference range, 1.5 to $7.0 \times 10^9/L$). Biochemical abnormalities included mild hypokalemia (3.2 mmol/L; reference range 3.7 to 5.8 mmol/L), mild hypoproteinemia (54 g/L; reference range 60 to 82 g/L), and a mild increase in muscle enzyme activity (creatinase kinase 1039 U/L; reference range 0 to 580 U/L). Urine analysis revealed mild proteinuria (0.3 g/L), marked hematuria (200 to 300 red blood cells per high power field (HPF)), and a mild pyuria (0 to 3 white blood cells/HPF). Large numbers of hemolytic *Escherichia coli* were later cultured from the urine.

The mild changes in the hemogram and serum biochemistry were ascribed to the corticosteroid treatment and, possibly, an iron-limited anemia resulting from a chronic lower urinary tract infection. The mild increase in muscle enzyme activity was attributed to any of the following: recumbency, intramuscular injection, catheter placement, or restraint. Collection of cerebrospinal fluid (CSF) was considered; however, because the cat later began to exhibit signs of intermittent respiratory depression, there were concerns that general anesthesia might lead to further decompensation and respiratory embarrassment.

Treatment with a balanced electrolyte maintenance solution (Plasmalyte 148, Baxter Canada, Toronto, Ontario) was initiated at a rate of 2.8 mL/kg/h, IV. In addition, 50 mg of clindamycin (Dalacin-C, Upjohn, Don Mills, Ontario) was administered IV, q8h, as well as dexamethasone sodium phosphate (Dexamethasone 5, Vetoquinol Canada), 1.25 mg, IV, q8h.

The cat continued to deteriorate neurologically over the next 24 h, with progression to severe tetraparesis. He was placed in an oxygen tent after observation of a marked abdominal component to his respiratory pattern. This was attributed to involvement of innervation to the diaphragm at the level of the 5th and 6th cervical spinal nerves (C₅-C₆) and/or the thoracic spinal cord segments innervating intercostal muscles. Additionally, there could have been progressive loss of descending upper motor neuron tracts necessary for respiration. Trimethoprim-sulphadiazine (Tribrissen 24% Injection, Janssen Pharmaceutica, Mississauga,

Can Vet J 1995; 36: 712-713

Department of Pathology (Turner, Percy) and Department of Clinical Studies (Allyson), Ontario Veterinary College, University of Guelph, Guelph, Ontario N1G 2W1.

Address correspondence to Dr. D.H. Percy.

Ontario) (75 mg, SC, q12h), potassium chloride (Potassium Chloride, Astra Pharma, Mississauga, Ontario), (20 mEq/L), and B vitamins (Vitamin B Complex, Sanofi Sante Animale, Canada, Victoriaville, Quebec) (1 mL/L) were added to the treatment regimen.

Three days after the initial onset of hemiparesis, the cat became anorectic. There was no improvement in his condition and the abnormal respiratory pattern had become more pronounced. The owners elected to euthanize the cat and gave permission for postmortem examination.

At necropsy, there were no significant findings either internally or externally. Cross sections of fixed spinal cord revealed a locally extensive area of malacia with several petechial hemorrhages at the level of the cervical intumescence. Representative sections of the spinal cord were fixed in 10% neutral buffered formalin, processed, sectioned, and stained with hematoxylin and eosin, toluidine blue, and Masson's trichrome stains. Histologically, in the cervical region, there was moderate to marked asymmetric necrosis of the dorsal and lateral funiculi, with moderate involvement of the adjacent grey matter. In several consecutive sections, a prominent irregular area of malacia, bordered by swollen astrocytes and necrotic debris, occupied most of the dorsal and lateral funiculi and dorsal and intermediate columns of grey matter. There was a mild degree of associated hemorrhage, congestion, and microgliosis and a moderate infiltration of neutrophils. Occlusive fibrocartilaginous emboli were present within many of the blood vessels in these sections (Figure 1). Other levels of the spinal cord were histologically normal. On the basis of these findings, we diagnosed fibrocartilaginous embolism with infarction and ischemic necrosis of affected areas of the spinal cord.

Fibrocartilaginous embolic myelopathy (FCE) is an uncommon condition reported primarily in the dog. Two cases of cats with this condition have been reported previously, 1 in the United States (2) and 1 in Germany (3). To our knowledge, this is the first report of the syndrome in this species in Canada. The cats reported earlier were mixed breeds aged 10- and 12-years-old. With FCE, the anatomical site and extent of the infarction of the spinal cord defines the nature of the neurological deficits observed and determines the overall prognosis (4). In the other reports, both cats exhibited an acute onset of nonpainful, bilateral pelvic limb paresis, and urinary and fecal incontinence, with the spinal cord infarction limited to the lumbosacral plexus. In one case, the pelvic limb deficits progressed to paraplegia. In dogs with FCE, clinical signs are rarely reported to progress beyond the first 12 to 24 h after initial recognition (5). Rapid progression of neurological deterioration

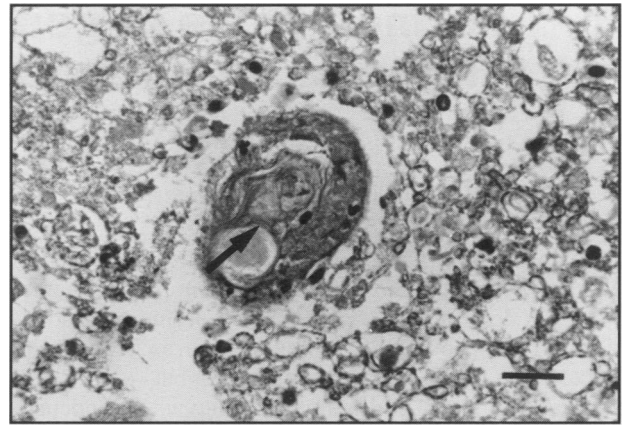


Figure 1. Photomicrograph of an occlusive fibrocartilaginous embolus (arrow) in the dorsal funiculus of the cervical spinal cord at the level of the 7th cervical vertebra. Toluidine blue. Bar = 10 μ m.

was evident in the cat reported herein, likely a result of the extensive spinal cord infarction.

There is no diagnostic test to confirm the occurrence of FCE; the antemortem diagnosis is one of exclusion. The diagnostic plan should include a CBC, complete biochemical profile, survey radiographs, and CSF collection and myelography, if possible. Other compressive or infarctive etiologies of acute onset that should be considered in the cat include trauma, thrombotic emboli associated with cardiomyopathy, neoplasia, myelitis, and intervertebral disk herniation. Definitive diagnosis requires postmortem examination and histopathology. Histochemically, the fibrocartilage embolus is reported to be identical to the content of the nucleus pulposus of the intervertebral disk (1). The mechanism by which the embolus later dislodges and reaches the spinal cord is unknown.

This case demonstrates that cats with an acute onset of paresis or paralysis of unknown origin must also be considered as candidates for the condition of fibrocartilaginous embolic myelopathy.

CJV

References

1. Neer, TM. Fibrocartilaginous emboli. *Vet Clin North Am Small Anim Pract* 1992; 22: 1017-1026.
2. Zaki FA, Prata RG, Werner LL. Necrotizing myelopathy in a cat. *J Am Vet Assoc* 1976; 169: 228-229.
3. Bichsel P, Vandeveld M, Lang J. L'infarctus de la moelle épinière à la suite d'embolies fibrocartilagineuses chez le chien et le chat. *Schweiz Arch Tierheilkd* 1984; 126: 387-397.
4. Little PB. Central nervous system rendezvous — Canine acute posterior paresis. *Can Vet J* 1994; 35: 517-518.
5. de Lahunta A. Fibrocartilaginous embolic ischemic myelopathy. In: Kirk RW, ed. *Current Veterinary Therapy VI*. Philadelphia: WB Saunders, 1977: 908-909.