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MRI characteristics of suspected acute spinal cord infarction in two cats, and a review of the literature

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A 10-year-old neutered male Persian cat and a 4-year-old spayed female domestic shorthair (DSH) cat were evaluated for acute-onset severe lateralising tetraparesis and hemiplegia, respectively. Both cats also had left-sided Horner's syndrome. Neurological examination of the cats localised the lesion to cranial to C5 in the Persian and the left cervical intumescence (C6–T2) in the DSH. Physical examinations were otherwise generally unremarkable. Routine laboratory tests and spinal radiography were normal for the Persian cat and were not performed for the DSH cat. A cerebrospinal fluid (CSF) tap was attempted for the Persian cat but aborted because of gross blood contamination, and was not performed for the DSH cat. Magnetic resonance imaging (MRI) of the Persian cat revealed a lesion within the spinal parenchyma at segments C1 to C3 (slightly more left-sided) which was iso- to hypointense on T1-weighted scans and hyperintense on T2-weighted scans, and which enhanced slightly with gadolinium. MRI of the DSH cat revealed a lesion within the spinal parenchyma at segment C7 (predominantly left-sided) which was hypointense on T1-weighted scans and hyperintense on T2-weighted gradient echo scans. Contrast was not administered. The MRI findings in both cases were highly suggestive of acute spinal cord infarction, based upon comparison to human cases. Both cats made full neurological recoveries with supportive treatment only. This paper describes two cases of suspected acute spinal cord infarction in the cat, demonstrates the potential diagnostic value of MRI, and discusses the clinical syndrome of this condition with a brief review of published cases.

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Case reports

Case 1

A 10-year-old neutered male Persian cat developed acute tetraparesis. The cat, which lived exclusively indoors, did not appear to be in pain and was alert and responsive. Examination at a veterinary surgery revealed plegia of the left thoracic limb and both pelvic limbs, severe paresis of the right thoracic limb, and apparent urinary and faecal retention. Routine blood work and cervical spinal radiography were unremarkable. The cat was hospitalised and given a single dose of dexamethasone (0.5 mg/kg subcutaneously [SC]: Dexadreson; Intervet). No change was noted in the cat's condition during the following 8 h. The cat was subsequently referred to the University of Bristol Feline Centre.

On presentation, the cat was in lateral recumbency. He was able to move his head normally and his right thoracic limb very slightly, but was plegic in the other three limbs. Left-sided Horner's syndrome was present. Abdominal palpation revealed a distended urinary bladder and copious firm faeces in the colon. Mild dehydration and a grade II/VI heart murmur with point of maximum intensity at the left base were also noted on examination.

A thorough neurological examination was performed. The cat was alert with appropriate mentation. Cranial nerve examination was normal

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except for the Horner's syndrome. Proprioception was absent in the left thoracic limb and both pelvic limbs, and was markedly decreased in the right thoracic limb. Spinal reflexes were increased in all four limbs (upper motor neuron − UMN) and marked pelvic limb crossed extensors were also present (UMN). Spinal palpation did not elicit noticeable pain. The neurological findings suggested a somewhat lateralising (left-sided) lesion cranial to C5, with first order Horner's syndrome involving the lateral tectotegmentospinal tract. Differential diagnoses included spinal cord infarction, trauma (including intervertebral disc disease), focal myelitis/osteomyelitis, discospondylitis and neoplasia. Intracranial disease seemed less likely as no other cranial signs were present and the cat's behaviour and mentation were unaltered.

Routine blood work (haematology, biochemistry, FeLV/FIV screening) was normal. Echocardiography revealed a slight thickening of the aortic valve, which caused mild turbulence in blood flow and the resultant heart murmur. This finding was not thought to be significant. The heart was otherwise structurally normal. Thoracic radiography revealed hypostatic congestion and collapse of the right middle lung lobe attributed to prolonged recumbency. Abdominal radiography revealed a markedly distended urinary bladder and a large quantity of faeces in the colon, consistent with urinary and faecal retention. The spinal column on these radiographs and on an additional cervical spinal film appeared normal. A cisternal CSF tap procedure was attempted but aborted because of gross blood contamination.

Supportive treatment was initiated pending MRI of the brain and spinal cord. This included intravenous fluid administration (Hartmann's), manual bladder expression facilitated with diazepam (0.25 mg/kg orally [PO] sid-bid; Roche), cisapride (1.25 mg/kg PO bid-tid: Prepulsid; Janssen-Cilag) and enemas (Micralax; Medeva) to promote defecation, frequent turning, physiotherapy consisting of muscle massage and joint flexion/extension, and occasional carprofen (1−2 mg/kg SC sid; Zenecarp, C-Vet) as needed for analgesia. No corticosteroids were administered.

MRI scans were performed 4 days after clinical onset (Phillips NT, 0.5 T). Images of the brain were normal. T1- and T2-weighted images of the cervical spine revealed an extensive, poorly delineated parenchymal lesion over the C1 to C3 vertebral bodies which lateralised to the left (Figs 1 and 2). The lesion was iso- to hypointense on T1-weighted images and hyperintense on T2-weighted images. There was only mild irregular enhancement with gadolinium. The findings were suggestive of acute spinal cord infarction based on comparison to human studies (Elksnis et al 1991, Nagashima et al 1991, Fortuna et al 1995).

The cat remained hospitalised for a further 2 weeks. During this time he gradually regained

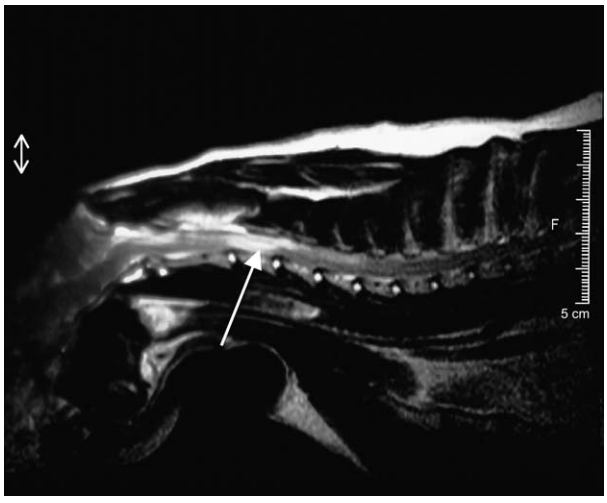

Fig 1. A sagittal T2-weighted MR image of the cervical spinal cord of case 1. There is a poorly delineated but extensive intraparenchymal hyperintensity noted in the cervical spinal cord over the level of vertebral bodies C1 to C3 (arrow). TR = 1806 ms, TE = 130 ms.

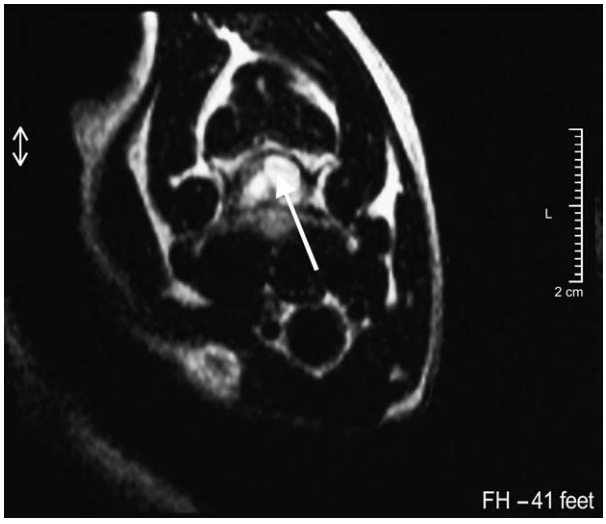

Fig 2. A transverse T2-weighted MR image of the cervical spinal cord of case 1 at the level of C2 vertebral body. There is a lateralised hyperintense lesion noted within the spinal cord (arrow). TR = 3000 ms, TE = 100 ms.

voluntary movement in all limbs and the ability to urinate. He was discharged on cisapride (0.625 mg/kg PO bid-tid) as needed to promote defecation. His condition continued to improve, and approximately 6 weeks following onset the owner reported apparent full neurological recovery. Subsequent examination by the referring veterinary surgeon did not reveal any neurological deficits. The cat had no known recurrence of clinical signs in the following 4 years and then was lost to follow-up.

Case 2

A 4-year-old spayed female DSH cat developed acute onset knuckling of the left thoracic limb. The cat's condition progressed over the next few hours to involve the left pelvic limb as well. Examination at a veterinary surgery revealed left-sided hemiplegia. Treatment consisted of dexamethasone (0.5 mg/kg SC: Dexadreson; Intervet) and amoxycillin/clavulanate (10 mg/kg SC: Synulox; Pfizer). The cat was referred to Stone Lion Veterinary Centre after 24 h, with slight improvement noted by the owners at that point.

On presentation, the cat was unsuccessfully attempting to stand. The left thoracic and pelvic limbs were plegic and the right thoracic and pelvic limbs were mildly paretic. Left-sided Horner's syndrome was present. The physical examination was otherwise unremarkable.

A thorough neurological examination was performed. The cat was alert with appropriate mentation. Cranial nerve examination was normal except for the Horner's syndrome. Proprioception was absent in the left thoracic and pelvic limbs and was mildly decreased in the right thoracic and pelvic limbs. Spinal reflexes were decreased in the left thoracic limb (lower motor neuron (LMN)), increased in the left pelvic limb (UMN) and normal in the right thoracic and pelvic limbs. Spinal palpation did not elicit noticeable pain. The neurological findings suggested a left-sided lesion at the cervical intumescence (C6–T2), with second order Horner's syndrome involving the intermediate grey matter and cell body of the second order neuron. Differential diagnoses included spinal cord infarction, trauma (including intervertebral disc disease), focal myelitis/osteomyelitis, discospondylitis and neoplasia.

No further diagnostic tests were undertaken except for an MRI scan (Siemans Impact Expert, 1 T) which was performed approximately 36 h after the clinical onset. T1-weighted scans of the cervicothoracic spinal cord revealed a small hypointense area in the central cord at the level of the C7 vertebra, sagittal view (not shown). Contrast was not administered. T2-weighted gradient echo scans revealed a hyperintense lesion extending from the vertebral bodies of C5–T1 and was most obvious over vertebral body C7, sagittal view (Fig 3). On transverse views, the lesion was predominantly left-sided and involved both white and grey matter (Fig 4). Again, the findings were suggestive of acute spinal cord infarction based on comparison to human studies (Elksnis et al 1991, Nagashima et al 1991, Fortuna et al 1995).

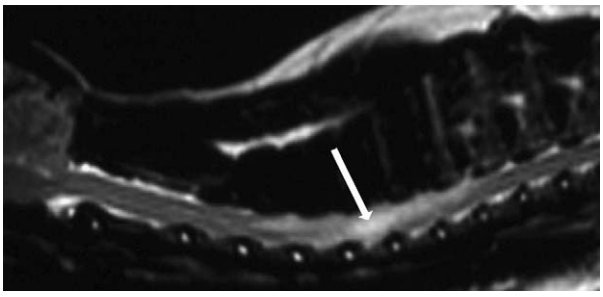

Fig 3. A sagittal T2-weighted MR image of the cervical spinal cord of case 2. There is extensive dorsal parenchymal hyperintensity noted over the seventh cervical to the second thoracic vertebrae (arrow). TR = 3500 ms, TE = 112 ms.

The cat was treated only with physiotherapy consisting of muscle massage and joint flexion/extension and made a complete recovery over 2 weeks. Neurological re-evaluation by the

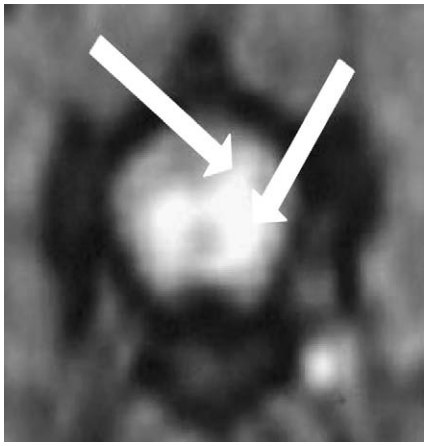

Fig 4. A transverse T2-weighted gradient echo MR image of the spinal cord of case 2 at the level of the seventh cervical vertebra. There is irregular asymmetrical hyperintensity within the parenchyma of the spinal cord (arrows). TR = 630 ms, TE = 22 ms.

referring veterinarian was normal. The cat has had no known recurrence of clinical signs.

Discussion

Acute spinal cord infarction (ASCI) is a well-recognised clinical entity in both human and veterinary medicine (Sandson and Friedman 1989, Neer 1992). Resultant neurological deficits are based upon the extent and location of the infarct(s). A typical presentation for ASCI in veterinary patients is a sudden onset, frequently asymmetric, mono-, hemi-, or tetraparesis or -plegia (Shell and Dyer 1997). Spinal hyperpathia is not usually present, although there may be some discomfort at the apparent instant of infarction (Neer 1992, Shell and Dyer 1997). The neurological deficits associated with ASCI are generally non-progressive unless ascending/descending myelomalacia develops (Neer 1992). Deficits frequently improve with time, especially those primarily associated with white matter lesions (Shell and Dyer 1997). Steroid administration may be helpful in reducing cord swelling in the early stage of infarction (Penwick 1989, Dyce and Houlton 1993) although the evidence for this remains largely circumstantial (Neer 1992). Supportive care during hospitalisation including physiotherapy, attention to urinary and faecal retention if necessary, and prevention of decubital ulcers is essential for patient recovery.

In human medicine, potential causes of ASCI are numerous and diverse, and include atherosclerosis, vasculitis, embolic events, infection and consequences of surgery or trauma (Sandson and Friedman 1989). In contrast, in the dog − the veterinary species in which ASCI is best described − fibrocartilaginous embolisation (FCE) is clearly the most common cause (Neer 1992). Therefore, the term FCE is often used synonymously with ASCI in this species, although this is not completely accurate (Cook 1988). In FCE, fibrocartilaginous material forms single or multiple emboli in the spinal vasculature, resulting in focal or multifocal infarction. The fibrocartilage is thought most likely to originate from the nucleus pulposus of an intervertebral disc. Several theories have been proposed to explain how fibrocartilaginous material could gain access to the spinal vasculature (Penwick 1989, Neer 1992, Dyce and Houlton 1993).

In addition to FCE, other potential causes of ASCI in veterinary patients include thromboembolism (eg, associated with cardiovascular disease), hypercoagulable states (eg, polycythaemia), vasculopathy, parasitic embolisation and septic embolisation (Neer 1992, Dyce and Houlton 1993). In the feline species, five case reports of FCE have been published: two from the UK (Scott and O'Leary 1996, Abramson et al 2002), one from Canada (Turner et al 1995), one from Germany (Bichsel et al 1984) and one from the US (Zaki et al 1976). To the authors' knowledge, there are no case reports documenting any other causes of ASCI in the cat.

The major differential diagnosis for ASCI is acute spinal trauma, including intervertebral disc herniation. Focal myelitis, osteomyelitis, discospondylitis and spinal neoplasia should also be considered, although typically the clinical presentation for these conditions is more subacute or chronic and the associated neurological deficits are usually progressive (Neer 1992). Spinal hyperpathia may or may not be present. Patient history, clinical examination and routine laboratory testing may help to further rule in or rule out some of these differential diagnoses. Spinal radiography and myelography may be particularly useful to identify trauma, disc disease, osteomyelitis, discospondylitis, and extramedullary neoplasia (Neer 1992). CSF analysis may help to diagnose infectious, inflammatory and some neoplastic processes (Sandson and Friedman 1989).

If ASCI is suspected, the clinician should attempt to identify the underlying cause. A patient with a cause of spinal cord infarction other than FCE (as listed above) may have some findings on examination or testing which suggest the definitive diagnosis. Conversely, patients with FCE generally have no clinical problems apart from their neurological signs, have no specific laboratory abnormalities and have normal radiography (Shell and Dyer 1997). Myelography may reveal cord swelling (Cauzinille and Kornegay 1996) especially if performed within hours of clinical onset (Shell and Dyer 1997). CSF analysis may be normal or may reveal nonspecific abnormalities such as elevated protein, hypercellularity, or both (Cauzinille and Kornegay 1996). Currently, a definitive diagnosis of FCE requires histopathological evaluation of tissue (Cauzinille and Kornegay 1996) obtained at post-mortem examination. ASCI due to other causes may also remain undetected or undiagnosed without post-mortem examination.

In human medicine, MRI is routinely performed on patients with clinical signs suggestive of spinal cord pathology (Sandson and Friedman

1989, Fortuna et al 1995). ASCI is well-documented in human patients and MRI findings characteristic of this condition have been described (Elksnis et al 1991, Nagashima et al 1991, Fortuna et al 1995). In 1995, Fortuna et al published a literature review of human cases of acute spinal cord infarction that had been diagnosed with MRI. Sixty-one patients with a total of 80 MRI scans were included in the review. T1-weighted images revealed spinal cord lesions which were isointense in 70% of cases, hypointense in 18% of cases and hyperintense in 11% of cases. T2-weighted images revealed spinal cord lesions which were hyperintense in 93% of cases and isointense in 7% of cases. Variance in intensity was attributed to a difference in the amount of oedema and/or haemorrhage present. Lesions enhanced with gadolinium in 70% of cases. This finding was difficult to interpret as previous studies (eg, Nagashima et al 1991) had shown the timing of infarct enhancement to be variable. In particular, 'early' infarcts (with MRI scans performed within hours) may not enhance. Gadolinium enhancement is likely to be most evident approximately 5–6 days after the onset of infarction (Nagashima et al 1991). Based upon these results, Fortuna et al (1995) concluded that the following MRI characteristics are suggestive of ASCI in human patients: iso- or hypointensity of spinal cord lesion on T1-weighted images; hyperintensity of lesion on T2-weighted images; and gadolinium enhancement of lesion, most apparent 5–6 days after clinical onset. While these authors caution that ASCI cannot be definitively diagnosed based upon MRI alone because other disease entities (eg, multiple sclerosis, intramedullary neoplasia) could produce similar results, certain MRI findings interpreted in conjunction with suggestive historical, clinical and laboratory data can strongly support an ante-mortem diagnosis of ASCI in human patients (Elksnis et al 1991).

In veterinary medicine, the application of MRI in the diagnosis of acute spinal cord infarction may also have value. Both cats in this report had typical presentations for ASCI – acute onset paresis/plegia, lack of progression and absence of spinal hyperpathia. No other significant clinical or laboratory abnormalities were present in either case. Localisations to specific spinal cord segments based upon neurological examination were confirmed with MRI. Spinal cord lesions in both cases had the same characteristics: iso- or hypointense lesions on T1-weighted images and hyperintense lesions on T2-weighted images. In addition, gadolinium (only administered to case 1) produced mild enhancement. These findings are identical to those documented in the Fortuna et al (1995) review of human patients with ASCI.

A presumptive diagnosis of ASCI was made in both cases. Given the previously published literature and a lack of other significant clinicopathological findings, fibrocartilaginous embolism was considered to be the most likely underlying aetiology for both cats.

A definitive diagnosis of fibrocartilaginous embolism could obviously not be obtained in either case, as the cats survived and, therefore, post-mortem examinations were not performed. However, previous publications (deLahunta and Alexander 1976, Gilmore and deLahunta 1987, Cauzinille and Kornegay 1996, Junker et al 2000) have discussed 'suspected' cases of FCE in dogs and, following the same principles, it would seem logical to categorise these two cats as suspected FCE cases. While FCE has previously been described as rare in the cat (Chrisman 1991), the authors' experience with these two cases, and other unpublished clinical cases, suggests that FCE may be more common in the cat than previously thought.

The specific neurological location of confirmed/suspected FCE lesions may be of some interest and importance to the clinician. A review of 62 canine FCE cases (Cauzinille and Kornegay 1996) revealed an unequal distribution of lesions for both confirmed and suspected cases (Table 1). The L4–S3 region was most frequently affected in both confirmed and suspected cases, although all spinal segments were represented. In the cat, the confirmed cases of FCE have all been located at the intumescences – either the cervical intumescence C6–T2 (Turner et al 1995, Abramson et al 2002) or the lumbar intumescence L4–S3 (Zaki et al 1976, Bichsel et al 1984, Scott and O'Leary 1996). The Persian cat presented here is the first published case of FCE (suspected) in the C1–C5 region of a cat. The neurological localisation for the five previously published cases plus the two presented here is shown in Table 1. Obviously, the small sample size precludes drawing any valid conclusions, but the results are presented for interest and for comparison to the canine results. While no significant conclusions can yet be drawn, it is important to consider that FCE could potentially occur in any area of the feline spinal cord, and therefore the clinician should not rule out a diagnosis of

Table 1. Neurological localisation of confirmed and suspected cases of FCE in the dog (Cauzinille and Kornegay 1996) and the cat (literature review and two cases presented here)

Neurological localisation	Canine			Feline
	Confirmed % (36 cases)	Suspected % (26 cases)	Total % (all 62 cases)	Total % (all 7 cases)
C1−C5	2.8	3.8	3.2	14.2
C6−T2	30.6	3.8	19.3	42.9
T3−L3	19.4	42.3	29.0	0.0
L4−S3	47.2	50.0	48.4	42.9

FCE based solely upon the area of the spine that is affected.

The prognosis for FCE in the cat, as in the dog, undoubtedly depends upon a number of factors. For example, canine patients with lower motor neuron involvement tend to have a poorer outcome than those with upper motor neuron involvement (Dyce and Houlton 1993). Additional factors that may influence prognosis include extent and severity of lesion, development of ascending/descending myelomalacia, and provision of appropriate supportive care (Neer 1992, Shell and Dyer 1997). In the review by Cauzinille and Kornegay (1996), the majority of dogs with suspected FCE improved clinically (74%). However, when both confirmed and suspected cases were considered together, this figure dropped to only 27%. It is extremely difficult to interpret these findings for a number of reasons (eg, variation in patient size, age, neurological localisation, treatment), but most significantly because all confirmed cases, by definition, were euthanased. The true prognosis is, therefore, unknown.

All previously reported cats with FCE were euthanased, and thus again true prognosis cannot be determined. Two cats (Bichsel et al 1984, Turner et al 1995) deteriorated − the former was euthanased 3 days after initial onset because of progression and severity of signs (including respiratory compromise), and the latter was euthanased after an unknown duration due to progression from paraparesis to paraplegia. Two cats (Zaki et al 1976, Abramson et al 2002) demonstrated minimal improvement and were euthanased after 6 and 7 days, respectively. One cat (Scott and O'Leary 1996) demonstrated gradual improvement over 12 days but was then euthanased because of a suspected poor prognosis for total recovery. In contrast, the cats presented here showed gradual improvement over 6 weeks (case 1) and 2 weeks (case 2) and made eventual complete recoveries with no treatment except for supportive care measures. Other suspected cases of feline FCE have been seen by the authors (eg, approximately one to two cases per year (MacKay); unpublished observations), with improvement occurring despite a lack of definitive diagnosis or treatment. While clearly these observations are impossible to interpret in a scientific manner, it is worthwhile to consider that the prognosis for feline FCE may not always be poor, especially if initial deterioration does not occur, if supportive care measures are instituted when necessary, and if enough time is allowed for recovery. Much remains to be learned about FCE in the cat, and further studies will be necessary to better determine the prognostic indicators in this species.

A clinician should consider a diagnosis of acute spinal cord infarction (fibrocartilaginous embolism or other aetiology) when presented with a cat exhibiting characteristic signs of acute-onset, non-progressive, possibly asymmetric limb paresis or plegia. Thorough physical examination, laboratory work-up, and survey radiography should be performed, along with CSF analysis and/or myelography if appropriate. MRI may be indicated in equivocal cases or in suspected cases to further support the diagnosis and to determine the location and extent of the lesion for prognostic value. Cats with suspected ASCI are probably most likely to have FCE and these cats may have a better prognosis than previously reported.

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