CASE REPORT

Suspected fibrocartilaginous embolism in a cat

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A 12-year-old cat was presented to the University of Queensland's Small Animal Teaching Hospital with a 1-day history of left hemiparesis of acute onset, with no evidence of trauma or toxin exposure. Neurological examination findings were consistent with a lesion in the caudal left cervical spinal cord (C6 to C8), which was non-painful and had not progressed since the onset of clinical signs. No other abnormalities were found, although myelography showed a mild swelling involving the caudal cervical and cranial thoracic spinal segments. A diagnosis of suspected fibrocartilaginous embolism was made on the basis of the history, clinical presentation and diagnostic tests results, making this case the first report of a suspected fibrocartilaginous embolism in a cat that returned to normal function.

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FCE Fibrocartilaginous embolism

Emboli to the nervous system can develop from a variety of sources such as endocarditis, sepsis, fat and fibrocartilaginous material, the latter being the most common form of spinal cord infarction in veterinary medicine.[1] FCE causes ischaemic necrotising myelopathy and it is generally agreed that it originates from the intervertebral discs,[2] although other sources such as vertebral growth plate cartilage[3] and a metaplastic vascular endothelium[4] have been suggested. Since the first description of FCE,[2] several theories have been proposed for its pathophysiology (Table 1).

FCE has been well documented in dogs,[8-12] occasionally reported in pigs,[3,13-15] horses,[16,17] turkeys,[18] and rarely in sheep,[19] mustelids[20] and humans.[5,21,22] In 36 confirmed canine cases, 47% occurred in the lumbosacral intumescence (L4 to S3), 30% in the cervical intumescence (C6 to T2) and spinal cord swelling was seen on myelography in 47% of those cases.[8] In 24 confirmed human cases, FCE occurred in the brain stem (25%), cervical (87%), thoracic (71%) and lumbar (16%) segments of the spinal cord, although most patients had lesions in more than one region.[22]

Cerebrospinal fluid analysis was normal in 54% of 36 dogs[8] and in 82% of 11 humans[23] with confirmed FCE. The abnormal findings in these studies were mild and consisted of increased protein, especially in chronic cases, and hypercellularity.

Of the four reports of confirmed FCE diagnosed in cats, two occurred in the lumbosacral intumescence[24,25] and the others were in the cervical intumescence.[26,27]

Acute onset of a nonprogressive (except for the first 48 hours), nonpainful asymetric paresis with no specific diagnostic findings are characteristic of FCE.[1,22,28] Magnetic resonance imaging has been useful in human cases and is likely to become the diagnostic procedure of choice for animals, although a definitive answer can only be obtained by histological examination of the spinal cord.[11,21]

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The case of this report is a suspected FCE of the caudal cervical region in a cat, with almost complete recovery after a period of 2 weeks.

Case report

A 12-year-old, desexed male, domestic shorthair cat was referred to The University of Queensland's Small Animal Teaching Hospital after an acute onset of left-sided hemiparesis. According to the owner's description, a very loud meowing was heard and the cat was found conscious, lying on its side with rigidly extended limbs. It soon recovered and was able to weight bear and ambulate, even though weakness was evident on the left side of the body. Annual vaccinations, deworming and heartworm preventive medication were current. The cat lived in a single-cat household with no outdoor access and no history of trauma or toxin exposure. On clinical examination, the patient was mentally alert and active, with normal cranial nerve and ophthalmic function. The cat had severe proprioceptive ataxia in the hind legs, especially on the left side, and the patellar reflexes were brisk in both legs. The left front limb had proprioceptive deficit with reduced weight bearing capacity, decreased reflexes and decreased deep pain sensation. Cutaneous trunci and perineal reflexes were normal and there was no pain elicited on palpation of the spinal column. Other physical examination findings were unremarkable. A complete blood health profile was normal and no abnormalities were seen in plain radiographs. Myelogram revealed a mild, diffuse cord swelling over the caudal cervical and cranial thoracic spine, findings suggestive of FCE.

A tentative diagnosis of FCE was made, and the patient was discharged after 2 days of hospitalisation. The owner reported that there was progressive improvement over the following days and the cat was acting normally 2 weeks after discharge. The patient was re-evaluated 7 weeks after the onset of clinical signs, and the only abnormality found was a slight limp and mild proprioceptive deficit in the left front leg. Cerebrospinal fluid

Table 1. Theories proposed for the pathophysiology of fibrocartilaginous embolism.

1. Herniation of nucleus pulposus material and exposure to the vertebral body vasculature. This results from direct manipulation during surgical procedures, trauma, or, in the case of humans, via the formation of islands of prolapsed fibrocartilage within the cancellous portion of the vertebra called Schmorl's nodes.[2,5]
2. Extrusion of disc material directly into the spinal vasculature.[6]
3. Herniation of disc material into persistent embryonal arteries of the annulus fibrosus.[2]
4. Disc herniation into anomalous vasculature adjacent to or in one of the intervertebral discs.[6]
5. Direct access of ruptured disc material into vessels of nucleus pulposus formed as a result of chronic inflammation.[7]

analysis was done 9 weeks after initial presentation , for a different reason, and it was normal.

Discussion

The differential diagnoses in this case included trauma, spinal cord or vertebral neoplasia, meningomyelitis, discospondylitis or intervertebral disc disease with disc herniation, aortic thromboembolism secondary to hyperthrophic cardiomyopaty and FCE.[4,29]

The patient was kept indoors and had no contact with other cats, making an infectious cause unlikely. Except for the neurological deficits, physical, laboratory and radiographic findings were normal. These, and the fact that there was no evidence of pain or progression of neurological signs within 48 hours following the initial onset, excluded trauma, neoplasia, infection and aortic thromboembolism. Intramedullary swelling at the site of the myelopathy is attributed to parenchymal haemorrhage and oedema at the lesion site,[8] which is likely to have happened in our case. The myelogram also excluded intervertebral disc herniation as the primary cause of the problem. Although CSF analysis was done long after initial presentation, it was still valuable to confirm the absence of infectious or neoplastic causes. Neurological findings in our cat were consistent with lower motor neuron involvement in the left fore leg, producing decreased reflexes and weight bearing capacity in that limb. Although worse on the left side, upper motor neuron signs of brisk patellar reflexes were seen bilaterally, which may have been a result of spinal cord oedema in the infarcted area.[4] Clinically, the lesion was localised in the left caudal cervical cord at the level of the brachial plexus, corresponding to spinal cord segments C6 to C8. The fact that cutaneous trunci reflexes were normal and there was no evidence of Horner's Syndrome excluded involvement of spinal cord segments caudal to C8.[30]

The acute screaming episode can be compared to the typical signalment of FCE in dogs[10, 11] and it may have been caused by the extrusion of fibrocartilaginous material, stimulating nociceptive receptors in bone, periosteum, ligaments or meninges.[1,4] Pain was absent after the onset of the supposed ischaemia because the spinal cord parenchyma does not contain pain receptors.[4]

The history and clinical findings of acute, nonprogressive, asymmetric, nonpainful focal myelopathy with no specific abnormalities found in the screening tests, except for a mild swelling observed on myelogram at the lesion site predicted by the neurological examination, were suggestive of FCE.

Treatment for this condition consists of supportive care. The cat of this report did not receive medication, as it was presented approximately 24 hours after the onset of the injury. Although the effectiveness of pharmacological intervention later than 8 hours following acute spinal injury still needs to be investigated with controlled clinical studies, it is currently not recommended.[31,32]

In all publications of confirmed FCE in cats, the patients were between 8 and 12 years of age, there was no sex predilection and all animals were euthanased after a period of 4 to 12 days from the onset of the clinical signs.

This is the first published report of a suspected FCE in a cat where marked improvement was seen over 2 weeks following initial diagnosis. The lower motor neuron deficits seen in the left front leg are associated with a poorer prognosis for full recovery of function of the limb, due to grey matter damage and motor neuron cell bodies destruction in the affected area.[1, 4] This cat was fortunate in its recovery with barely perceptible neurological deficits present 7 weeks after the onset of clinical signs.

FCE should be considered in the differential diagnosis of spinal disease in cats, especially if it is of acute onset, nonpainful and asymmetric. Since FCE can currently only be confirmed by histological examination, this may have contributed to the fact that only a few cases have been published so far, and they tended to have been the ones that were severely affected, carrying a worse prognosis.

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