NeuroNews

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September 2013

Fibrocartilaginous Embolic Myelopathy

NeuroNews is an informal, but hopefully informative, newsletter covering a range of clinically relevant neurological topics. All cases detailed in these articles are patients kindly referred to me by veterinarians in South Australia or western Victoria/NSW.

Fibrocartilaginous Embolic Myelopathy (FCEM) is a relatively common cause of acute onset paresis and ataxia in dogs. Although FCEM is occasionally seen in cats, feline ischaemic myelopathy is thought to more commonly occur secondary to thromboembolism (usually related to conditions such as cardiomyopathy, thyroid disease and renal failure).

Aetiopathogenesis

FCEM arises as a result of obstruction of blood flow to a region of the spinal cord by microscopic fragments of fibrocartilage. Although the exact pathogenesis is still unclear, histochemical studies indicate that the collagen contained in the fibrocartilaginous emboli is identical to that of the nucleus pulposus of intervertebral discs.

It is currently postulated that the fibrocartilage fragments may enter spinal cord blood vessels from an intervertebral disc in a variety of ways: by direct penetration into local vessels; following herniation into the bone marrow of a vertebral body and thence into the venous plexus; via persistent remnants of embryonic blood vessels in the nucleus of a disc; or as a result of neovascularisation of a degenerate disc.

FCEM lesions are typically solitary; however multifocal disease has been documented. Although almost invariably a myelopathic syndrome, focal brainstem ischaemia following fibrocartilaginous embolism has been recorded.

It is believed that embolism may be precipitated by a sudden increase in intra-discal, intra-thoracic or intra-abdominal pressure. This correlates well with the frequently observed onset of signs, consistent with FCEM, following a burst of vigorous exercise or exercise-related fall.

Clinical presentation

The classical picture is one of peracute onset of hindlimb monoparesis, hemiparesis, paraparesis or quadriparesis, with associated ataxia; plegia may be evident in severe cases. Evidence of spinal pain is uncommon, however it is occasionally defined in the first 24 hours.

Neurological deficits are commonly at least partially lateralised. The severity of signs typically peaks within 24 hours.

Specific neurological findings are dependent upon the location, extent and severity of the lesion. Diligent clinical neurological assessment will usually enable a firm neuroanatomic diagnosis.

Although classically considered to be a large-breed problem, FCEM has been recorded in all sizes and ages of dog and also cats (more commonly DSH). Miniature Schnauzers appear to have a propensity for the condition.

Diagnosis

Definitive ante-mortem diagnosis of FCEM is not possible, however a typical history and clinical presentation are strongly suggestive of FCEM. Appropriate diagnostic investigation includes magnetic resonance imaging and CSF evaluation. These procedures not only assist in the exclusion of other possible causes of peracute/acute onset myelopathy (especially disc-related disease and primary/secondary haemorrhage), but also frequently provide specific information which allows a relatively high degree of diagnostic confidence.

MRI: although abnormal spinal cord signal may not be evident in the first 24 to 48 hours, FCEM patients imaged after that time usually show evidence of T2 hyperintensity within the cord parenchyma (see images below). Lesions are typically iso- or mildly hypo-intense on T1-weighted sequences; occasionally mild post-contrast enhancement is noted.

CSF evaluation often reveals mild mixed cell pleocytosis and/or elevated protein level.

Management

While there is an empirical belief that the administration of methylprednisolone sodium succinate, ideally within 12 hours of onset, may provide a degree of neuroprotection and reduce the risk of progression of signs, to date there is no firm evidence that this therapy improves outcomes in cases of FCEM. No other medications have proven benefit. Treatment focuses on provision of high quality nursing care, physiotherapy and bladder management if necessary.

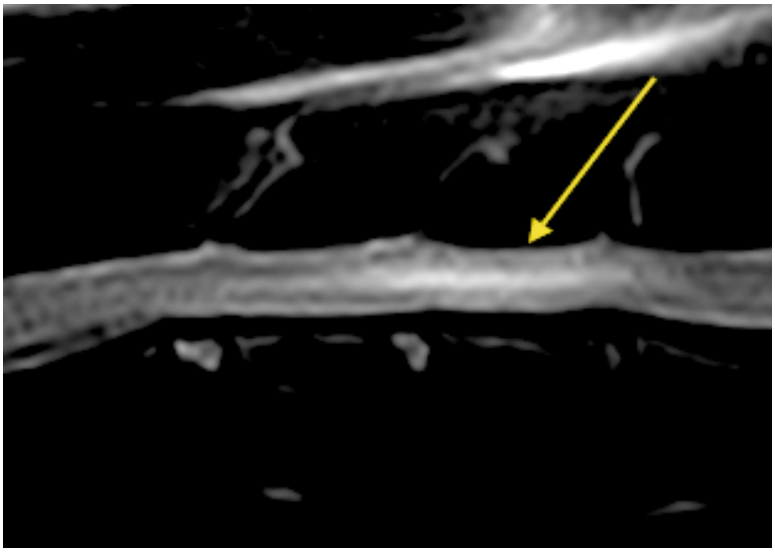
Prognosis

Recent data on FCEM suggests that the prognosis for functional recovery is between 65 and 84%. There is a suggestion that the length and cross sectional area of lesions defined on MRI has a correlation with outcomes. Clinical findings which suggest a poorer prognosis include absence of nociception and possibly the presence of LMN signs. Owner commitment to providing intensive nursing care and physiotherapy has a significant influence on recovery.

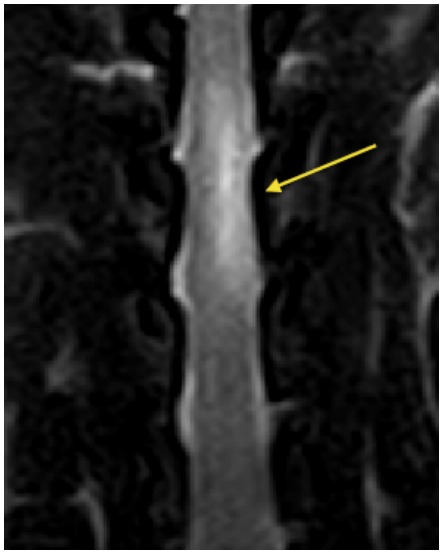
CASE 1: 2 y.o. FS French Bulldog presented to *NeuroVet* with peracute, left-sided, spastic hemiparesis and ataxia, with plegia of the left forelimb

MRI, performed 48 hours post-onset, revealed T2 hyperintensity predominantly on the left side of the spinal cord between C3 and C5. CSF obtained via lumbar centesis had a protein level of 0.75 g/L (normal < 0.45 g/L).

This case is of interest as FCEM is rarely documented in chondrodystrophoid breeds.



Sagittal T2-weighted image (Case 1)

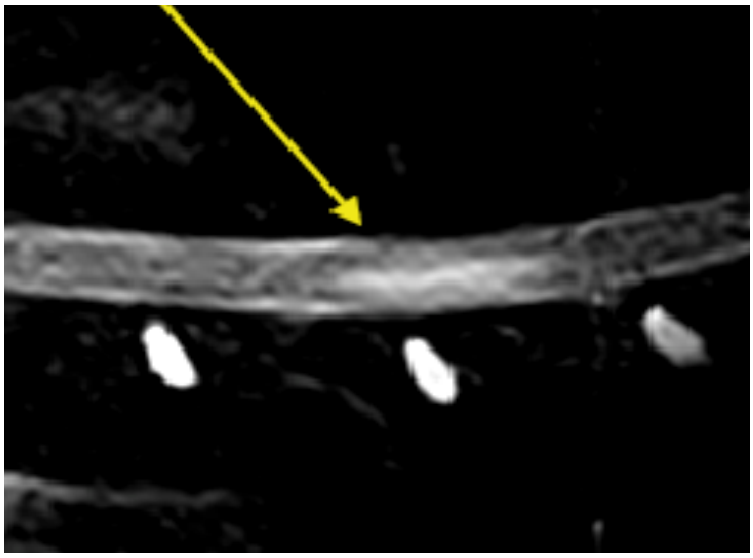


Dorsal T2-weighted image (Case 1)

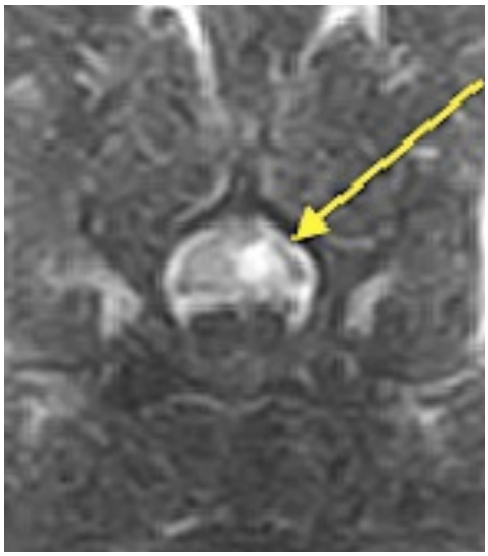
The dog made an excellent recovery and was strongly ambulatory on all limbs three weeks later.

CASE 2: 9 y.o. FS Staffordshire terrier crossbred presented to *NeuroVet* with acute, profound and predominantly left-sided, non-ambulatory quadriparesis

MRI was performed 72 hours after onset of signs, revealing typical T2/STIR hyperintensity on the left side of the cord, between caudal C5 and caudal C6. Lumbar CSF protein level was 0.76 g/L.



Sagittal T2/STIR image (Case 2)



Transverse T2-weighted image (Case 2)

Weakness was exacerbated by patient obesity and pre-existing orthopaedic problems. Improvement over the subsequent eight months was slow and incomplete, despite diligent nursing, physiotherapy and hydrotherapy.

NeuroVet is fully equipped to investigate suspect FCEM cases. I am delighted to perform the necessary diagnostics and assist with appropriate management of any patients which you feel may be affected with this condition.

Best wishes,

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