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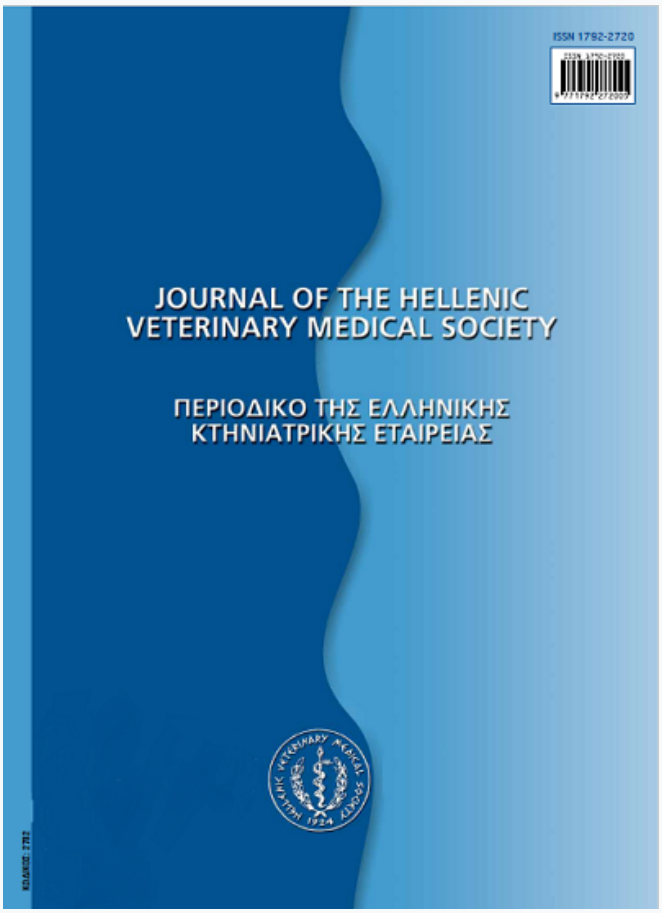

Endoscopic and histological findings in teats of dairy goats

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Research article
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Clinical, clinicopathological and diagnostic imaging findings in 14 dogs with suspected fibrocartilaginous embolic myelopathy

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Κλινικά, εργαστηριακά και απεικονιστικά ευρήματα σε 14 σκύλους με ενδεχόμενη ισχαιμική μυελοπάθεια από ινοχόνδρινα έμβολα

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ABSTRACT. Fibrocartilaginous embolic myelopathy was diagnosed in 14 dogs with acute neurological dysfunction, based on history, findings of clinical examination, diagnostic imaging evaluation, follow-up and outcome. The dogs were presented with signs of variable involvement of the spinal cord, which were lateralized in 4 cases. The initial clinicopathological evaluation was unremarkable, while cerebrospinal fluid analysis was abnormal in one dog. Diagnostic imaging investigation (plain radiographs of the spinal column and myelography) did not reveal any abnormalities in the vertebrae and adjacent tissues or compression of the spinal cord, with the exception of one case, where there was evidence of focal intramedullary oedema corresponding to the lesion location. Seven dogs improved significantly with supportive treatment; complete remission of clinical signs was evident in two. Moderate improvement was seen in three animals and minimal or no improvement in four dogs, which were euthanised due to persisting neurological incapacitation.

Keywords: dog, fibrocartilaginous embolic myelopathy

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ΠΕΡΙΛΗΨΗ. Η παρούσα μελέτη αφορά στην αναδρομική αξιολόγηση 14 σκύλων με ισχαιμική μυελοπάθεια από ινοχόνδρινα έμβολα. Η επιλογή των ζώων έγινε με βάση το ιστορικό, τα ευρήματα της κλινικής, εργαστηριακής και απεικονιστικής διερεύνησης, την εξέλιξη και την τελική έκβαση των περιστατικών. Όλοι οι σκύλοι προσκομίστηκαν στην Κλινική Ζώων Συντροφιάς της Κτηνιατρικής Σχολής του ΑΠΘ, επειδή εμφάνισαν απότομα (σε διάστημα 6-24 ωρών) διάφορης έκτασης πάρεση ή παράλυση. Η εκδήλωση των συμπτωμάτων ήταν ασύμμετρη σε τέσσερα ζώα και συμμετρική στα υπόλοιπα. Από την αιματολογική εξέταση, τη βιοχημική εξέταση του ορού, την ανάλυση του ούρου και του εγκεφαλονωτιαίου υγρού δεν προέκυψαν παθολογικά ευρήματα. Στην ακτινολογική εξέταση της σπονδυλικής στήλης δεν παρατηρήθηκαν αξιόλογα ευρήματα, με εξαίρεση ένα ζώο, στο οποίο διαπιστώθηκε η παρουσία διαμαρτιών διάπλασης σε ορισμένους θωρακικούς σπόνδυλους. Σε όλους τους σκύλους, πραγματοποιήθηκε μυελογραφία, από την οποία, σε καμία περίπτωση, δεν προέκυψαν ευρήματα συμβατά με συμπιεστική μυελοπάθεια, αλλά σε ένα περιστατικό υπήρξαν ενδείξεις εντοπισμένου ενδομυελικού οιδήματος στην οσφυϊκή μοίρα του νωτιαίου μυελού. Η θεραπευτική αντιμετώπιση ήταν μόνο συμπτωματική και υποστηρικτική σε όλα τα ζώα. Επτά σκύλοι παρουσίασαν σημαντική βελτίωση της κλινικής εικόνας ή και πλήρη ίαση, ενώ μερική ή καμία βελτίωση των συμπτωμάτων διαπιστώθηκε σε τρία και τέσσερα ζώα, αντίστοιχα. Στα τελευταία έλαβε χώρα ευθανασία, λόγω της βαρύτητας των νευρολογικών τους διαταραχών.

Λέξεις ευρετηρίασης: ινοχόνδρινα έμβολα, ισχαιμική μυελοπάθεια, σκύλος

INTRODUCTION

Fibrocartilaginous embolic myelopathy (FCEM) has been described in several animal species (dogs, cats, ruminants, pigs, horses), as well as in humans (De Risio and Platt, 2010). It is a vascular myelopathy, attributed to the occlusion of arterial and/or venous supply to a specific region of the spinal cord by fibrocartilaginous material histologically and histochemically identical to the *nucleus pulposus* of intervertebral discs (Sharp and Wheeler, 2005). Several theories regarding the potential pathophysiology of fibrocartilaginous embolic myelopathy have been proposed, although the mechanism by which fibrocartilaginous material reaches the spinal cord vasculature still remains unknown. Hypotheses proposed include the direct penetration of *nucleus pulposus* fragments into the spinal cord vessels (possibly associated with increased intrathoracic and intraabdominal pressure) (Gilmore and de Lahunta, 1986), chronic inflammatory neovascularisation of degenerated intervertebral disks (Hayes et al., 1978), presence of embryonic remnant vessels within the nucleus pulposus (Hayes et al., 1978) and mechanical herniation of *nucleus pulposus* into the vertebral bone marrow (Olby and Jeffery, 2003). Initially fibrocartilaginous embolic myelopathy was thought to affect animals of large and giant breeds, however there have been several reports of cases in small as well as in chondrodystrophic breeds (Gandini et al., 2003; Grunenfelder et al., 2005).

Affected dogs are typically presented with a history of peracute to acute onset of clinical signs, progressing within a period of two to four hours. The degree of neurological dysfunction depends on localization and severity of the spinal cord ischemic injury. The majority of patients present signs of a non-progressive, non-painful and often asymmetric myelopathy, most commonly affecting the cervicothoracic (C8-T2) or lumbosacral (L4-S3) spinal cord segments (Gilmore and de Lahunta, 1986; Gandini et al., 2003). The differential diagnosis includes other causes of embolic myelopathy (bacterial and parasitic diseases, neoplasia), underlying medical conditions predisposing to embolisation or thrombosis (hypo- or hyperthyroidism, hyperadrenocorticism, cardiomyopathy, hypertension, chronic renal failure), traumatic and compressive intervertebral disk extrusion, focal myelitis and intra- or extramedullary haemorrhage. *Ante-mortem* diagnosis is supported by history and exclusion of the various aforementioned conditions, with the aid of clinical and clinicopathological assessment and advanced diagnostic imaging (Dewey, 2008; De Risio and Platt, 2010).

The aim of this retrospective case series is to present the clinical, clinicopathological and diagnostic imaging findings, as well as the follow-up and final outcome of 14 dogs with suspected fibrocartilaginous embolic myelopathy.

MATERIALS AND METHODS

A total of 14 dogs presented with the main complaint of acute, non-progressive ataxia, paresis or paralysis were included in the study. Data recorded

Table 1. Details, clinical and diagnostic imaging findings in 14 dogs with suspected fibrocartilaginous embolic myelopathy.

Case no.	Age (years)	Breed	Gender	Duration of signs (hours)	Location of lesions	Prior exercise	Lateralization of signs	Findings in spinal radiography	Findings in myelography
1	3.5	Gekas	M	6	Thoraco-lumbar (T3-L3)	Yes	No	WNL	WNL
2	2	German shepherd	M	24	Cervico-thoracic (C6-T2)	No	Yes (R)	WNL	WNL
3	2.5	Poodle	F	6	Thoraco-lumbar (T3-L3)	Yes	No	WNL	Spinal cord swelling (L1-L2)
4	8.5	Rottweiler	M	12	Thoraco-lumbar (T3-L3)	No	No	WNL	WNL
5	5.5	German shepherd	M	24	Thoraco-lumbar (T3-L3)	No	No	WNL	WNL
6	9	Siberian husky	F	6	Thoraco-lumbar (T3-L3), spinal hyperaesthesia	Yes	No	WNL	WNL
7	10	Rottweiler	F	24	Cervico-thoracic (C6-T2)	No	Yes (R)	Spondylosis deformans of the lumbosacral joint	WNL
8	5	Labrador retriever	F	24	Cervico-thoracic (C6-T2)	Yes	Yes (R)	WNL	WNL
9	3	French bulldog	M	6	Thoraco-lumbar (T3-L3)	No	No	Multiple congenital vertebral abnormalities	WNL
10	11	Mixed breed	M	24	Lumbosacral (L4-S3)	No	No	WNL	WNL
11	3.5	French bulldog	M	24	Lumbosacral (L4-S3)	No	No	WNL	WNL
12	6.5	English setter	M	6	Cervicothoracic (C6-T2)	Yes	No	WNL	WNL
13	8	Gekas	F	24	Cervicothoracic (C6-T2)	Yes	No	WNL	WNL
14	3	Yorkshire terrier	F	6	Cervicothoracic (C6-T2)	Yes	Yes (R)	WNL	WNL

M: male, F: female, L: left, R: right, WNL: within normal limits.

in all dogs included information concerning the onset and progression of signs, previous medical treatments, presence or absence of spinal pain, severity of insult (paresis/plegia, presence/absence of deep pain sensation), symmetry of neurological signs, neuroanatomic lesion localization. The degree of spinal cord dysfunction was scored using a scale of 0 to 5 (Sharp and Wheeler, 2005). In all cases, spinal cord trauma had been excluded from the differential diagnosis based on history and initial clinical evaluation.

Clinicopathological tests carried out in all cases included a complete blood cell count, serum biochemistry profile (total proteins, albumin, glucose, cholesterol, triglycerides, urea nitrogen, creatinine, alkaline phosphatase, alanine aminotransferase, calcium, phosphorus, sodium and potassium) and urinalysis. Cerebrospinal fluid analysis, performed in all dogs in samples collected prior to myelography, included macroscopic evaluation, total and differential cell counts and measurement of albumin concentration.

Diagnostic imaging evaluation was performed in all dogs under general anesthesia and included survey radiographs of the spinal column and cisternal myelography. The latter procedure was performed within two days of the initial presentation and examination of patients.

Physical therapy procedures to be performed by the owners of the animals were initiated after establishment of diagnosis and was modified according to the daily routine programme and competence of the owners. It included passive manipulation and massage of the affected limbs, support of severely incapacitated animals in the standing position and frequent changes of posture (lateral to sternal recumbency). Provision of further standard supportive treatment for recumbent animals (assisted feeding and drinking, soft bedding, cleanliness) was also advised (Sharp and Wheeler, 2005).

Follow up of the cases was performed by re-examination of the patients.

Table 2. Follow-up and outcome in 14 dogs with suspected fibrocartilaginous embolic myelopathy

Case no.	Neurological signs	Duration of follow-up	Outcome
1	Acute paraplegia (grade IV)	2 months	Significant improvement, first noted after 1 month
2	Acute right thoracic limb monoparesis → tetraplegia (grade IV)	1 month	Significant improvement, first noted after 10 days
3	Acute paraparesis → paraplegia (grade V)	2 months	Significant improvement, first noted after 1 month
4	Acute paraparesis (grade III)	1.5 months	Moderate improvement, first noted after 20 days
5	Acute paraparesis (grade III)	1 month	Moderate improvement, first noted after 10 days
6	Acute paraplegia (grade IV)	2 weeks	No improvement, euthanasia
7	Acute right thoracic limb monoparesis → tetraparesis (grade III)	1 month	No improvement, euthanasia due to concurrent medical problems
8	Acute tetraraparesis (grade III)	1 month	Moderate improvement, first noted after 15 days
9	Acute paraplegia (grade IV)	2 months	Significant improvement, first noted after 15 days
10	Acute paraparesis (grade IV)	3 months	Significant improvement, first noted after 1.5 months
11	Acute paraplegia (grade V)	2 weeks	No improvement, euthanasia
12	Acute tetraparesis → tetraplegia (grade IV)	6 months	Significant improvement, first noted after 15 days; normal after 1 month
13	Acute tetraplegia (grade IV)	2 months	Minimal improvement, euthanasia
14	Acute right-sided hemiparesis → hemiplegia	2 months	Significant improvement, first noted after 15 days; normal after 2 months

RESULTS

All dogs were adults and their age ranged from 2 to 11 years. Regarding their sex and breed, 8 were male and 6 female, 13 were pure-breds and one was a mixed breed (Table 1). All dogs were presented for evaluation because of acute or peracute paresis or paralysis of one or more limbs. Physical exercise of variable intensity prior to the appearance of signs was reported in 7 dogs. Eight dogs were presented with acute paraparesis (3/8) or paraplegia (5/8), five with tetraparesis (2/5) or tetraplegia (3/5) and one with right sided hemiparesis that progressed to hemiplegia. Lateralization of neurological signs was observed in four dogs, two of which initially had been presented with paresis of the right thoracic limb and subsequently asymmetric tetraparesis or tetraplegia. The remaining two animals exhibited right sided hemiparesis or asymmetric tetraparesis (Table 2).

No abnormalities were detected in routine clinicopathological evaluation (complete blood count, serum biochemistry and urinalysis). Cerebrospinal fluid analysis revealed mild (25 nucleated cells/μl) pleocytosis in one dog. Radiographic examination of the spine revealed presence of congenital vertebral anomalies in one animal and mild *spondylosis deformans* of the lumbosacral joint in another (Table 1). Cisternal myelography did not show spinal cord compression at the sites corresponding to the neurological lesion localization. In one animals, there was evidence of focal spinal cord oedema involving the L1 and L2 segments.

Follow up of patients ranged from two weeks to six months. Significant improvement or complete remission of signs (regarding ambulation) was noted in seven dogs. In these patients, the first signs of improvement were noted within 10 to 20 days. In three animals, improvement was considered to be moderate and in the remaining four as minimal or absent (Table 2). In these four dogs, euthanasia was finally performed, due to their poor response to conservative treatment.

DISCUSSION

Initially reported principally in large sized dogs, fibrocartilaginous embolic myelopathy is now considered a disease that may affect all breeds. Although the large and giant breeds of dogs still represent a significant proportion of animals reported in many relevant publications (Gandini et al., 2003), smaller breeds, as well as mixed breed dogs, may also be affected (Nakamoto et al., 2008, De Risio and Platt, 2010), in some studies even outnumbering larger size animals (Nakamoto et al., 2008, Nakamoto et al., 2009). In the present study, six dogs were of large breed, whilst four were medium- and four small-sized dogs (Table 1). Two dogs belonged to a chondrodystrophic breed (French bulldog) and seven other to breeds reported to be predisposed to degenerative intervertebral disk

disease, either small (Poodle, Yorkshire terrier) or large-sized (German shepherd, Rottweiler, Labrador retriever) (Table 1) (Olby, 2004; Brisson, 2010).

The age range of the study population (2-11 years) is in agreement with that reported in the literature (Cauzinille and Kornegay, 1997; Gandini et al., 2003, De Risio et al., 2008). Nearly half of the animals included in the study (6/14) were young adults (less than 5 year-old). Similarly, male and female dogs were almost equally represented (Table 1). Reported male to female ratios vary from 1:1 to 2.5:1 (Cauzinille and Kornegay, 1996; Gandini et al., 2003; De Risio et al., 2008; De Risio and Platt, 2010).

Patients with fibrocartilaginous embolic myelopathy are typically presented with a history of peracute to acute onset and progression of clinical signs, which is valuable information for the differential diagnosis. Most dogs reach the peak severity of neurological dysfunction within 24 hours, many within 6 hours or less, as was observed in the present study (Table 1) (Olby, 2004; Dewey, 2008). Owners often observe the affected dog to cry out in apparent pain during exercise shortly before the onset of neurological dysfunction (Sharp and Wheeler, 2005; De Risio and Platt, 2010). These dogs are often not in any detectable pain by the time they are presented to the clinician, as had been observed in all but one of our cases. The incidence of spinal hyperesthesia varies from 12 to 50% (Cauzinille and Kornegay, 1996; Gandini et al., 2003). Pain was reported in only one dog, which had developed peracute paraparesis and paraplegia while attempting to defecate. Increased intra-abdominal pressure during straining or exercise (Valsalva's manoeuver), potentially causing retrograde propulsion of *nucleus pulposus* material into the spinal vasculature, has been proposed as one of the pathomechanisms for fibrocartilaginous embolic myelopathy (De Risio and Platt, 2010).

Clinical signs in fibrocartilaginous embolic myelopathy vary depending upon location and severity of the spinal cord ischemic injury. The cervicothoracic (C5-T2) and lumbosacral (L4-S3) spinal cord segments are most commonly affected, with reported frequencies of 30 to 33% and 43 to 47%, respectively (Cauzinille and Kornegay, 1996; Gandini et al., 2003; De Risio et al., 2007). In the study population of the series reported here, almost half of the animals involved (6/14) showed signs of cervicothoracic syndrome. Conversely, the lumbosacral syndrome was noted only in two dogs. The remaining dogs were presented with thoracolumbar (T3-L3) spinal cord dysfunction, which has been reported less frequently in some studies (De Risio and Platt, 2010). In contrast, thoracolumbar syndrome has been noted as the most frequent neurolocalization in fibrocartilaginous embolic myelopathy in other clinical studies (Nakano et al., 2008; Nakano et al., 2009). Asymmetry of clinical signs, an important and common feature of fibrocartilaginous embolic myelopathy, was observed in four of the animals with cervicothoracic syndrome, initially mimicking orthopaedic diseases causing lameness (Sharp and Wheeler, 2005; Dewey, 2008). These dogs were submitted for examination very soon after the onset of signs (within 6-24 hours), compared to the rest of the patients, in which the lack of lateralization symptoms could be attributed to their delayed referral or to the more extensive involvement of the spinal cord. With the exception of secondary spinal cord injury, neurological deterioration in fibrocartilaginous embolic myelopathy rarely progresses for over 24 hours (Dewey, 2008; De Risio and Platt, 2010).

Diagnosis of the disorder is based on history, clinical signs and by ruling out other causes of acute myelopathy. Other causes of acute and potentially asymmetrical spinal cord dysfunction include acute compressive (Olby, 2004; Dewey, 2008) and non-compressive intervertebral disk extrusion (Chang et al., 2007; De Risio et al., 2009; Brisson, 2010), acute focal myelitis, embolisation or thrombosis secondary to other systemic disorders (cardiomyopathy, coagulopathies, hypothyroidism, chronic renal failure) (De Risio and Platt, 2010). From a clinical point of view, spinal hyperaesthesia is common in acute intervertebral disk extrusion and the main clinical sign differentiating it from fibrocartilaginous embolic myelopathy, which is non-painful (De Risio and Platt, 2010). Most of the other diagnostic differentials, predisposing to embolization or thrombosis, are either identified or excluded during clinical, clinicopathologic (complete blood cell count, serum biochemical evaluation, urinalysis) and cerebrospinal fluid analysis, which had been performed in all the cases described, yielding unremarkable results. Cerebrospinal fluid analysis has been reported to offer low sensitivity in fibrocartilaginous embolic myelopathy, rather than excluding myelitis.

Plain radiographs of the spine aim to ruling out traumatic spinal injuries and diskospondylitis. In one animal, congenital anomalies (hemivertebrae) were

identified in the cranial thoracic (T1-T4) vertebrae; however, their presence was asymptomatic, as no spinal cord compression was observed during contrast studies. Congenital vertebral anomalies are a common finding in French bulldogs, although their clinical course varies considerably from asymptomatic disease to severe compressive myelopathy (Westworth and Sturges, 2010). Myelography is used for excluding compressive myelopathies and offers the additional advantage of cerebrospinal fluid collection for analysis. Patients with fibrocartilaginous embolic myelopathy may show an intramedullary pattern, associated with spinal cord swelling around the embolised region, or have normal findings in myelography (Nakamoto et al., 2008; Nakamoto et al., 2009). In this study, myelography was performed in all cases and indicated no spinal cord compression. However, in one animal there was evidence of focal intramedullary oedema, compatible with the neuroanatomical lesion localisation. This dog had been referred immediately after onset of clinical signs and myelography had been performed within 24 hours. In contrast, in another animal, also peracutely affected and immediately submitted for evaluation, no similar findings were noted (Table 1). Acute non-compressive intervertebral disk extrusion, which is a possible diagnosis in cases of peracute myelopathy, has been reported to be suspected when there was evidence of spinal cord oedema located over a collapsed disk space combined with persisting spinal pain for more than 24 hours (Brisson, 2010; De Risio and Platt, 2010). Moreover, accuracy of myelography in diagnosing peracute compressive myelopathy secondary to intervertebral disk extrusion is reported to range from 72 to 97% (Brisson, 2010).

The preferred diagnostic imaging procedure for fibrocartilaginous embolic myelopathy diagnosis is magnetic resonance imaging. Advantages include the exclusion of other differential diagnostic possibilities and the identification of signal intensity changes associated with vascular infarction (Nakamoto et al., 2008; Nakamoto et al., 2009). Unfortunately, although magnetic resonance imaging had been proposed as a diagnostic test in all dogs studied, it had been rejected by their owners for financial reasons.

The clinical value of glucocorticoid treatment in fibrocartilaginous embolic myelopathy is questionable, especially when administered in recumbent patients susceptible to a variety of secondary complications (Dewey, 2008; De Risio and Platt, 2010). Physical therapy and good nursing care are part of the preferred treatment regime, which should begin as soon as possible and be protracted, as duration has a positive impact on neurological recovery (Sharp and Wheeler, 2005; Dewey, 2008). In addition, the absence of surgical wounds and pain allow its application immediately and without restrictions in fibrocartilaginous embolic myelopathy patients. Reported rehabilitation rates vary in published studies, possibly reflecting the non-homogeneous study populations and handling of cases (Gandini et al., 2003; De Risio et al., 2008).

Prognosis of fibrocartilaginous embolic myelopathy is variable, depending on location of lesions and severity of ischaemia. Recovery rates ranging from 58 to 84% have been reported (De Risio et al., 2007; Dewey, 2008). The prognosis is good for dogs that regain functional status within the first two weeks of spinal cord injury. Lesions affecting the cervicothoracic (C6-T2) and the lumbosacral (L4-S3) spinal cord segments bear a worse prognosis due to the involvement of lower motor neurons (Dewey, 2008; De Risio and Platt, 2010). In the present study, of six dogs with lesion localization in the aforementioned areas, four eventually showed significant or moderate improvement rendering them functional as pets (Table 2). Other negative prognostic factors are loss of deep pain sensation, urine and/or faecal incontinence (De Risio and Platt, 2010). One of the two dogs of our study with grade V spinal cord dysfunction and evidence of intramedullary oedema improved significantly after one month, while the other remained severely incapacitated and was euthanised.

Concluding remarks

Despite its acute onset and severely debilitating clinical presentation, fibrocartilaginous embolic myelopathy is a disease with potentially favourable prognosis. Thorough clinical and clinicopathological diagnostic work, combined with application of myelography, is important for establishing aetiological diagnosis, prognosis and treatment outline.

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