Negative MRI findings in a case of degenerative myelopathy in a dog

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ABSTRACT
An 11-year-old male Rough collie was submitted with paraparesis, but did not respond to medical treatment. Clinical signs worsened and the dog displayed paralysis, inability to stand and loss of voluntary bladder control, whereupon magnetic resonance imaging (MRI) was performed. No significant abnormalities were identified from MRI, blood tests, cerebrospinal fluid tests or radiography. After MRI, the dog developed dyspnoea and died. Autopsy and subsequent histopathological examination led to a diagnosis of degenerative myelopathy.

Keywords: degenerative myelopathy, dog, magnetic resonance imaging (MRI).

CASE HISTORY
An 11-year-old male Rough collie was brought to the animal hospital with paraparesis. About 1 year earlier, the owner had reported abnormal gait and falling when turning while walking. This worsened to total paraparesis and the dog was admitted at that time. Administration of prednisolone provided symptomatic relief. However, when similar signs reoccurred 6 months later, readministration of prednisolone (0.5 mg/kg, every other day) had no effect.

Disease of the thoracolumbar spinal cord was initially suspected. The dog had a temperature of 40.1 °C, heart rate of 100 beats/min, respiratory rate of 20 breaths/min and dysuria. Blood biochemistry was performed using a spin echo sequence (TR, 4000 ms; TE, 120 ms). Premedication administered intravenously included butorphanol tartrate (0.2 mg/kg, Stadol; Bristol-Myers Squibb Company, Tokyo) and midazolam (0.2 mg/kg, Dormicum; Astellas Pharma, Tokyo). Anaesthesia was maintained with isofluorane (Isofluorane for Animals; Schering-Plough Animal Health, Tokyo). No clear compressive lesions were found in the thoracolumbar spinal cord. No other abnormalities were observed in segments L4-S1 of the spinal cord, although a slightly protruded disk was apparent at L7-S1 (Fig. 1a-c).

Immediately after MRI, the dog developed pallor of the mucous membranes, tachypnoea, pyrexia of 40–41 °C, frequent vomiting and melaena, while chest radiography showed an alveolar, interstitial mixed pattern in the lower lobes of the lungs. Inhaled oxygen was provided. Despite the intravenous administration of sodium methylprednisolone succinate (30 mg/kg, Solu-medrol; Pfizer, Tokyo), ampicillin (20 mg/kg, Amiphenix; Kawanakimitaka, Kanagawa), furosemide (1 mg/kg, Lasix; Sanofi-Aventis Pharma, Tokyo) and neophylline (10 mg/kg, Neophylline; Eisai, Tokyo), the dog died 4 days after MRI.

 Necropsy was performed and histopathological examination showed scattered axonal swellings, macrophage infiltration, vacuolisation, demyelination and macrophage phagocytosis of degenerated axons in the white matter of the spinal cord, with no organisation in cells of the grey matter (Fig. 2). These pathological findings suggested a diagnosis of degenerative myelopathy.

INTRODUCTION
Degenerative myelopathy, also known as chronic degenerative radiculomyelopathy, is a chronic progressive spinal cord disease that frequently occurs in large dogs, particularly German shepherds\textsuperscript{a–c,2,8,15}. Small dogs and cats have also been reported to develop the disease\textsuperscript{1,2,12}. This peculiar disease is characterised by an extensive loss of myelin and axons beginning in the thoracolumbar spinal cord and progressive paraparesis beginning with the loss of proprioception in the pelvic limbs\textsuperscript{2–5,8,15}. Many symptomatic dogs have been reported to present with upper motor neuron signs (UMN signs) in the pelvic limbs\textsuperscript{a,b,c,13}. Degenerative myelopathy is diagnosed by clinical signs, signalment, cerebrospinal fluid (CSF) findings and imaging, after excluding other potential causes\textsuperscript{2,3,4,8,9}. A Rough collie was brought in for treatment, but was only ultimately diagnosed with degenerative myelopathy on post mortem and histopathological examination. In this paper, the clinical signs and findings on magnetic resonance imaging (MRI) are described.
death may have been respiratory disease such as pneumonia or pulmonary oedema, as hyper-leukocytosis (28 000/µl) was evident and radiography of the chest after MRI showed an alveolar, interstitial mixed pattern in the lower lobes of the lungs. Moreover, both midazolam and butorphanol were used as premedication and are respiratory depressants, which might have contributed to splanchnic and pulmonary ischaemia/hypoxia. In addition, vomiting and diarrhoea might have been complications of gastric ulceration. The dog displayed gastric ulceration at necropsy.

Neurological examination revealed depressed flexor reflexes in the pelvic limbs, indicating lower motor neuron (LMN) deficits. Differential diagnoses for chronic progressive disorders with LMN signs includes type II disc disease with spinal cord compression, degenerative lumbosacral stenosis, spondylosis deformans and neuronopathies with MRI abnormalities. The dog did not present abnormalities in the lumbar spinal cord, although slight protrusion was apparent at the disc between L7 and S1. A clinical diagnosis of ‘degenerative lumbosacral stenosis’ is reportedly characterised by localisation of hyperaesthesia in the lumbosacral region and about 90% of affected dogs are in pain. In spite of the slight disc protrusion, clinical diagnosis was difficult because the dog displayed no evidence of pain.

In general, this condition presents with UMN signs in the pelvic limbs, although LMN signs are seen in 10–15% of patients. LMN signs have been attributed to demyelination and axonal swelling in dorsal spinal nerve roots, as in the thoracolumbar spinal cord. However, the reported LMN signs of degenerative myelopathy have mainly involved decreased patellar reflex. Again, clinical diagnosis was difficult because this dog only exhibited decreased flexor reflex in the left pelvic limb.

Degenerative myelopathy in Pembroke Welsh corgis has been reported to show negative findings on MRI. It is likewise speculated that degenerative myelopathy in Rough collies shows no significant MRI abnormality, although these breeds differ substantially.

The aetiology of degenerative myelopathy has not been elucidated, despite an hypothesis that the condition represents an immunologically mediated neurodegenerative disease similar to multiple sclerosis (MS). MS is a chronic, relapsing human disorder that affects the central nervous system and is characterised by multiple demyelinating lesions. The lesions may be visualised by MRI. On Fig. 2: a. Histopathological examination shows scattered axonal swellings (arrows), macrophage infiltration, vacuolation, demyelination and phagocytosis of degenerated axons in the white matter of the spinal cord. Haematoxylin and eosin; scale bar = 100 µm.
T2WI of the spine, most plaques are located peripherally (commonly dorso-laterally) and less than 2 vertebral body segments in length. Lesions tend to be multifocal and present as well-circumscribed foci with increased signal intensity on T2WI. The detection rate for spinal cord lesions on T2WI using a conventional spin echo sequence in MS patients is reportedly 47–65%. However, in recent years, thanks to the development of sophisticated MRI techniques, conventional MRI (cMRI) has become capable of detecting cord lesions in up to 90% of patients with a diagnosis of MS. Signs in this dog involved chronic relapsing-remitting syndromes similar to MS. Inability to detect this lesion on MRI may have been due to the lesion being in remission at the time of imaging. Lesions of the demyelinated spinal cord may be detectable by different MRI techniques in degenerative myelopathy, but it is speculated that these lesions may also be more difficult to detect in rough collies based on the findings from Pembroke Welsh corgis.

REFERENCES