

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).

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INTRODUCTION

Degenerative myelopathy, also known as chronic degenerative radiculomyelopathy⁸, is a chronic progressive spinal cord disease that frequently occurs in large dogs, particularly German shepherds^{2-4,8,15}. Small dogs and cats have also been reported to develop the disease^{11,12}. 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^{2-5,8,15}. Many symptomatic dogs have been reported to present with upper motor neuron signs (UMN signs) in the pelvic limbs^{3,4,8,15}. Degenerative myelopathy is diagnosed by clinical signs, signalment, cerebrospinal fluid (CSF) findings and imaging, after excluding other potential causes^{3-5,8,9}. 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.

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 recurred 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 unremarkable. No pain was evident on back pressure from the thoracolumbar to the lumbosacral spinal cord. On neurological examination, no abnormal postural reactions were observed in the thoracic limbs, but postural reactions such as proprioception, hopping and placing tests all yielded positive results for the pelvic limbs. Spinal reflexes were normal in the thoracic limbs and the right pelvic limb. In the left pelvic limb, the patellar reflex was normal, but flexor reflex and deep pain were depressed. The urinary bladder was distended, with no muscle tone. Survey radiography showed no abnormalities.

As disease of the lumbosacral spinal cord was suspected, MRI was performed at Nihon University Animal Medical Center. MRI was performed using a 0.5-T Flex-Art system (Toshiba, Tokyo, Japan). T1-weighted imaging (T1WI) was performed using a spin echo sequence (repetition time (TR), 350 ms; echo time (TE), 15 ms) and T2-weighted imaging (T2WI) was performed using a fast 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 isoflurane (Isoflurane 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, Amipenix; Kawasakimitaka, 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.

DISCUSSION

Clinically, degenerative myelopathy is characterised by progressive ataxia of the pelvic limbs, beginning with loss of proprioception^{2-5,8,15}. The course of this disease has been reported as 6–12 months from onset of clinical signs^{2-5,8,15}. However, patients maintained beyond the loss of rear leg function eventually develop foreleg dysfunction, finally succumbing to brainstem involvement³. After anaesthesia for MRI, this dog developed worsening respiratory status and died, but the forelegs did not show dysfunction. Cause of

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death may have been respiratory disease such as pneumonia or pulmonary oedema, as hyper-leukocytosis ($28\,000/\mu\text{l}$) 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^{6,13}. 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 neuropathies with MRI abnormalities^{2-5,8,10}. 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 pain¹⁰. 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^{3,4,15}. LMN signs have been attributed to demyelination and axonal swelling in dorsal spinal nerve roots, as in the thoracolumbar spinal cord^{3,4,8}. However, the reported LMN signs of degenerative myelopathy have mainly involved decreased patellar reflex^{2-5,15}. 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 MRI⁵. 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)^{3,4}. 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^{14,16}. On

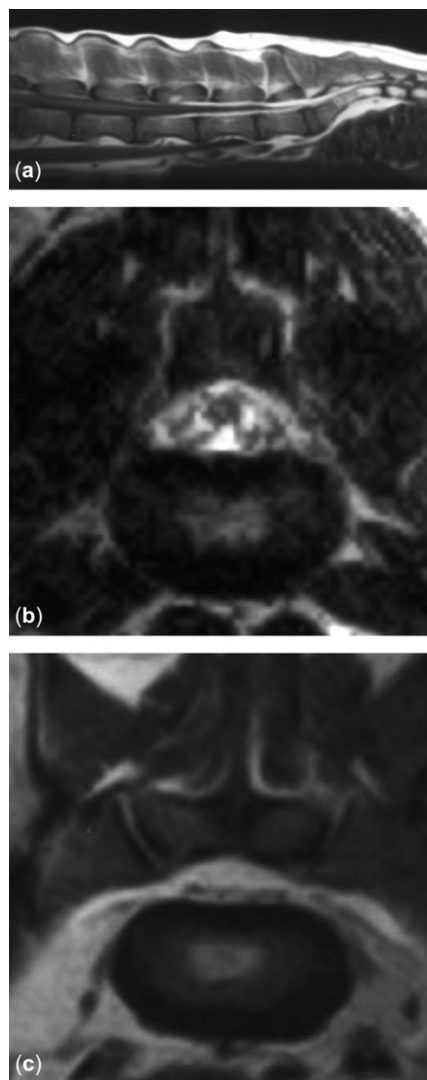


Fig. 1: a, Sagittal T2-weighted imaging; slight protrusion is apparent at the disc between L7 to S1; no abnormalities are observed in the lumbar spinal cord. b, Transverse T2-weighted imaging; no abnormalities are present in L4/5. c, Transverse T2-weighted imaging; slight protrusion is apparent at the disk between L7 and S1; no abnormalities are present in the lumbar spinal cord.

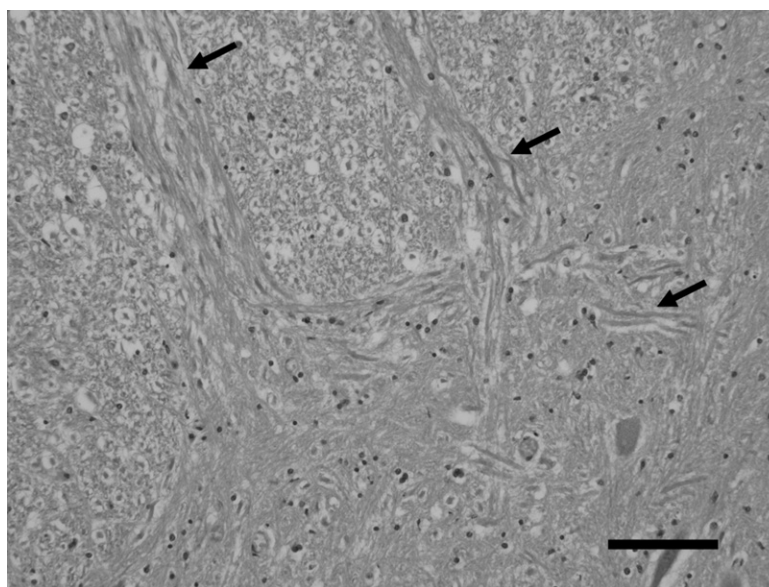


Fig. 2: Histopathological examination shows scattered axonal swellings (arrows), macrophage infiltration, vacuolation, demyelination and phagocytosis of degenerated axons in the white matter of the spinal cord. Haematoxylin and eosin; scale bar = 100 μm .

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^{7,16}. The detection rate for spinal cord lesions on T2WI using a conventional spin echo sequence in MS patients is reportedly 47–65%¹. 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 MS¹. 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 corgis⁵.

REFERENCES

1. Agosta F, Filippi M 2007 MRI of spinal cord in multiple sclerosis. *Journal of Neuroimaging* 17: 46S–49S
2. Averill D R Jr 1973 Degenerative myelopathy in the aging German shepherd dog: clinical and pathologic findings. *Journal of the American Veterinary Medical Association* 162: 1045–1051
3. Clemmons R M 1989 Degenerative myelopathy. In Kirk R W (eds) *Current veterinary therapy small animal practice* (10th edn). W B Saunders Company, Philadelphia: 830–833
4. Clemmons R M 1992 Degenerative myelopathy. *Veterinary Clinics of North America: Small Animal Practice* 22: 965–971
5. Coates J R, March P A, Oglesbee M, Ruaux C G, Olby N J, Berghaus R D, O'Brien D P, Keating J H, Johnson G S, Williams D A 2007 Clinical characterization of a familial degenerative myelopathy in Pembroke Welsh corgi dogs. *Journal of Veterinary Internal Medicine* 21: 1323–1331
6. Dowdle S M, Joubert K E, Lambrechts N E, Lobetti R G, Pardini A D 2003 The prevalence of subclinical gastroduodenal ulceration in dachshunds with intervertebral disc prolapse. *Journal of the South African Veterinary Association* 74: 77–81
7. Ge Y 2006 Multiple sclerosis: the role of MR imaging. *American Journal of Neuroradiology* 27: 1165–1176
8. Johnston P E J, Barrie J A, McCulloch M C, Anderson T J, Griffiths I R 2000 Central nervous system pathology in 25 dogs with chronic degenerative radiculomyelopathy. *Veterinary Record* 27: 629–633
9. Jones J C, Inzana K D, Rossmeisl J H, Bergman R L, Wells T, Butler K. 2005 CT myelography of the thoraco-lumbar spine in 8 dogs with degenerative myelopathy. *Journal of Veterinary Science* 6: 341–348
10. Lorenz M D, Kornegay J N 2004 *Handbook of veterinary neurology* (4th edn). W B Saunders Company, Philadelphia, USA
11. Matthews N S, de Lahunta A 1985 Degenerative myelopathy in an adult miniature poodle. *Journal of the American Veterinary Medical Association* 186: 1213–1215
12. Mesfin G M, Kesewitt D, Parker A 1980 Degenerative myelopathy in a cat. *Journal of the American Veterinary Medical Association* 176: 62–64
13. Neiger R, Gaschen F, Jaggy A 2000 Gastric mucosal lesions in dogs with acute intervertebral disc disease: characterization and effects of omeprazole or misoprostol. *Journal of Veterinary Internal Medicine* 14: 33–36
14. Nielsen J M, Korteweg T, Pokman C H 2007 Diagnosing MS: recent guidelines and future goals focusing on magnetic resonance imaging. *International MS Journal* 14: 29–34
15. Summers B A, Cummings J F, de Lahunta A 1995 Degenerative disease of the central nervous system. *Veterinary neuropathology*. Mosby, St Louis, Missouri
16. Tartaglino L M, Friedman D P, Flanders A E, Lublin F D, Knobler R L, Liem M 1995 Multiple sclerosis in the spinal cord: MR appearance and correlation with clinical parameters. *Radiology* 195: 725–732
17. Uldry P A, Regli F, Uske A 1993 Magnetic resonance imaging in patients with multiple sclerosis and spinal cord involvement: 28 cases. *Journal of Neurology* 240: 41–44