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CASE REPORT

Central Cord Syndrome of Thoracic Limb Paraparesis in Presumptive Meningomyelitis in a Dog

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A B S T R A C T

A 6-year-old female Schnauzer dog that weighed 7.72 kg was presented with acute thoracic limb ataxia, normal pelvic limb function and no history of trauma. A magnetic resonance imaging examination revealed the absence of extraparenchymal compression and high signal intensity on T2-weighted lesion in the center of the parenchyma of the cervical spinal cord. The MRI along with clinical signs and cerebrospinal fluid (CSF) analysis were utilized to diagnose presumptive meningomyelitis. Treatment was initiated with prednisolone for immune suppression. Although mild neurological deficits remained in the thoracic limbs, the dog improved in the neurologic assessments and began walking after 45 days of treatment. This report describes the unusual symptoms referred to as central cord syndrome, which has not previously been described in the veterinary literature with a demonstration of MRI features.

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INTRODUCTION

Central cord syndrome (CCS) has been defined in human medicine as a symptom in which the thoracic limbs are highly dysfunctional with minimal to no effective in the pelvic limbs (Epstein and Hollingsworth, 2015). The spinal cords that travel to the pelvic limbs are minimally affected because the lesion is centralized in the cervical region, which only affects the thoracic limbs. In general, CCS has a good prognosis for functional recovery and its common etiology is traumatic disease in human medicine (McKinley *et al*., 2007). CCS treatments with nonsurgical management include cervical spine restriction with a neck collar, rehabilitation followed by physical therapy and occupational therapy. Surgical management is provided for patients who cannot be treated by conservative management alone (Nowak *et al*., 2009; Yoshihara and Yoneoka, 2013).

History and clinical examination: A 6-year-old female Schnauzer dog that weighed 7.72 kg was referred because of an acute onset of abnormal gait in the bilateral thoracic limbs. A physical examination revealed depression and thoracic limb ataxia without remarkable dysfunction of the pelvic limbs. A neurological examination revealed bilateral thoracic limb spasticity with reduction in postural reactions, which include hopping, hemistanding, wheel-barrowing and proprioception. Mentation, cranial nerve reflexes and bilateral thoracic and pelvic limb spinal reflexes were normal while pain perception of the bilateral thoracic limb and panniculus reflex was absent during the examination. From the clinical evaluations, myelopathy of the cervical to cervicothoracic regions was suspected.

The complete blood count revealed mild leukocytosis (18,340/µl; reference interval 4.9-15.4 x 10^3/µl). Other serum chemistry values and the blood gas analysis were within respective normal limits. Cervical vertebral radiography revealed no remarkable findings of the vertebrae or vertebral alignment. Differential diagnoses included infectious and noninfectious inflammatory myelitis, intervertebral disk disease, fibrocartilaginous embolism and traumatic spinal cord injury.

To further clarify the etiology of the neurological deficit, MRI was performed using a 0.2 T machine with T1 and T2-weighted spin echo sequences. The MRI examination revealed a region of hyperintense signals on the T2-weighted sagittal images of the spinal cord parenchyma ranged from the sixth cervical (C6) to second thoracic (T2) segments (Fig 1). The affected lesion on the transverse T2-weighted images was remarkably in the central part of the spinal cord parenchyma (Fig 2). After the MRI, CSF was collected by alanto-occipital puncture and the analysis of CSF revealed mild neutrophilic pleocytosis (12 nucleated cells/µl) (Fig 3). However,

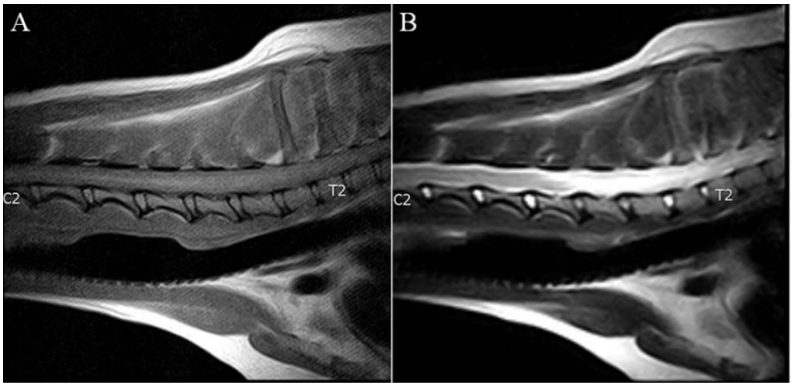

Fig. 1: Magnetic resonance images (sagittal). On magnetic resonance images, there was an iso-signal lesion on T1- (A) and a high-signal intramedullary lesion (arrowheads) on T2- (B) weighted images. The lesion had the equivocal margin from regions C5 to T2. There was no remarkable compression in the affected spinal cord.

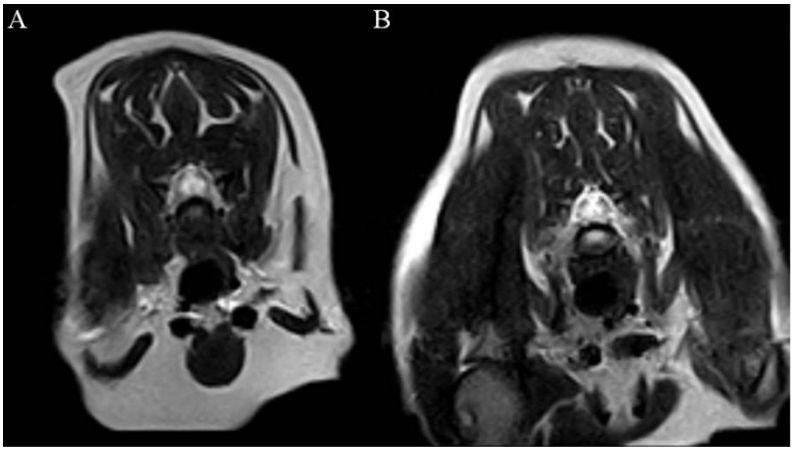

Fig. 2: Magnetic resonance images (transverse). On magnetic resonance images, high-signal lesions were detected on the T2-weighted images. The central parts of the spinal parenchyma with high signal intensity were noted at C7 (A) and T1 (B) regions. No abnormal findings were noted in the muscle surrounding the C7 or T1 vertebrae.

anaerobic and aerobic bacterial cultures were unremarkable. The virus antigen polymerase chain reaction (PCR) from the CSF for canine distemper virus was also negative.

Based on the clinical signs, MRI findings and CSF analysis results, the dog was tentatively diagnosed with non-infectious meningomyelitis, which includes steroid responsive meningomyelitis and meningomyelitis of unknown etiology.

Treatment and prognosis: Treatment consisted of medication for non-infectious meningomyelitis. Methylprednisolone sodium succinate 15 mg/kg IV was administered with cervical spine restriction and concurrent physical rehabilitation. Prednisone 1 mg/kg PO BID and cyclosporine 5 mg/kg PO BID was prescribed to reduce CNS inflammation and suppress immune-mediated process.

The neurological deficits improved 1 week after the initial presentation, which include remarkable improvement in the thoracic withdrawal and panniculus reflex. Forty-five days after the initial presentation, a serial neurological assessment revealed that the absent wheelbarrowing of the thoracic limbs was improved. Three months later, the dog exhibited a normal gait of the thoracic limbs. A neurological examination also demonstrated normal postural reactions and spinal reflexes, with the exception of reduced wheelbarrowing of the thoracic limbs.

Four months after the onset of clinical signs, the dog remained neurologically normal and had no clinical signs. A reevaluation of the MRI was not performed because of the positive clinical response and the client's concerns regarding financial burden and morbidity associated with the anesthesia procedure. The uncomfortable gait remained, but the general clinical signs were alleviated, thereby improving the patient's quality of life.

DISCUSSION

This case report demonstrates unusual symptoms that were characterized by a neurological deficit in the thoracic but not in the pelvic limbs. In general, cervical spinal cord diseases exhibit both thoracic and pelvic neurologic dysfunction but it has greater effect in the pelvic region, such as paraparesis and bladder dysfunction. In human literature, the symptom in which the thoracic limb is in a dysfunctional state with minimal to no deficit in the pelvic limbs has been referred to as CCS (Schneider *et al*., 1954). The lateral corticospinal and lateral spinothalamic tracts conduct each motor and sensory innervation. Partial damage of the spinal cord could impair nerve innervation in these regions because the central part of the tracts controls, in part, the cervical and thoracic regions. This finding would explain the greater deficits in the thoracic limbs compared with the pelvic limbs (Fig. 4).

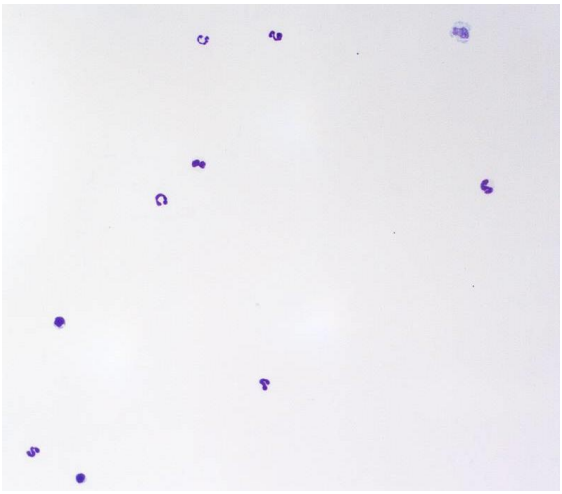

Fig. 3: Cerebrospinal fluid cytology sample shows that the neutrophil count was more than half of all nucleated cells. The cerebrospinal fluid analysis indicated mild neutrophilic pleocytosis (Diff-Quik stain, X 200).

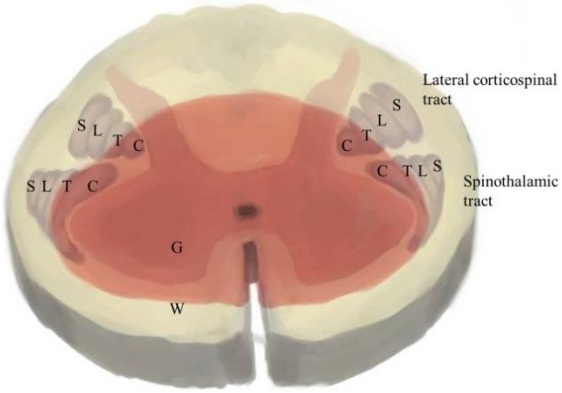

Fig. 4: Neuroanatomical concepts of the cervical spinal cord in central cord syndrome (CCS). The colored area is affected in CCS cases. According to the illustration, the sacral and lumbar structures are more peripheral in the lateral corticospinal and spinothalamic tracts; thus, these structures are preferentially spared in patients with CCS (C=Cervical, T=Thoracic, L=Lumbar, S=Sacral, G=Grey matter, W=White matter).

The spinothalamic tracts, which are located at the ventral funiculus, control the pain, temperature, and simple touch sensations of the trunk. The fasciculus cuneatus transmits proprioception and fine touch sensations of the thoracic limb (Li and Dai, 2010). The dog exhibited thoracic limb weakness and insensibility of pain and touch in the trunk and the thoracic limbs. These clinical signs most likely implied that the corticospinal tract and the motor neurons in the ventral horn and spinothalamic tracts of ventral funiculus were affected.

In general, CCS has been associated with more severe impairments in motor function than sensory function. Injury to the lateral corticospinal tract, which primarily conducts motor neurons, appears to be the main cause of the deficits associated with CCS (Nowak *et al*., 2009). In this case, the dog recovered from sensory reflexes, including withdrawal and panniculus reflexes, earlier than stiffness of the thoracic limbs.

A definitive diagnosis of most CNS diseases requires histopathology (Tipold and Stein, 2010). Although necropsy was not performed in this case, clinical signs and neurologic examination indicated cervicothoracic spinal cord parenchymal involvement, which was confirmed with MRI. MRI also ruled out an extradural compression lesion. Additionally, non-infectious meningomyelitis diagnosis was supported by CSF neutrophilic pleocytosis. (Griffin *et al*., 2008).

In general, CCS in human has a favorable prognosis for functional recovery (Dahdaleh *et al*., 2013). One study reported that the median rehabilitation length of stay was 30 days and 81% of patients were discharged (McKinley *et al*., 2007). CCS in human medicine is typically regarded as a traumatic disease (Molliqaj *et al*., 2014). To our knowledge, there is no case report of CCS in veterinary medicine; there are several reports that have examined spinal cord injuries in dogs (Webb *et al*., 2010). The dog in this report had neither identified trauma history nor imaging evidence of trauma. In meningomyelitis, canine distemper virus was the most common infectious agent, besides steroid responsive meningitis-arteritis was the most commonly recognized as non-infectious inflammatory disease process (Tipold and Stein, 2010). In this case, infectious etiology was ruled out by PCA examination. The dog was responsive to steroid and cyclosporine combination therapy.

Unfortunately, lack of MRI follow-up providing consistent imaging evidence of improved clinical signs was the limitation in this study.

Conclusions: This case report demonstrates that a non-infectious immune-mediated presumptive meningo-myelitis with partial spinal cord injury is similar to CCS in human medicine.

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