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Diagnosis and therapeutic management of acute traumatic spinal cord injury-associated paraplegia in a six-year-old Pomeranian

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Abstract

Background: Acute spinal cord injury (SCI) in dogs is a neurological emergency most commonly resulting from road traffic accidents and blunt trauma. Secondary spinal cord injury mechanisms, including inflammation and oxidative damage, play a major role in neurological deterioration following trauma. Early diagnosis and timely therapeutic intervention, particularly during the acute phase, are critical for reversing neurological deficits and improving prognosis.

Case presentation: This case report describes the diagnosis and therapeutic management of acute spinal cord injury mediated paraplegia in a six-year-old male white Pomeranian following a bike accident. The dog was presented with a history of blunt trauma followed by acute onset hindlimb paresis progressing to paraplegia. Neurological examination localized the lesion to the thoracolumbar spinal cord.

Management and Outcome: High-dose methylprednisolone sodium succinate (MPSS) was administered during the acute phase, along with intensive supportive care and strict confinement. Following MPSS therapy, progressive neurological improvement was observed with reversal of paraplegia, and the dog gradually regained hindlimb motor function. The patient ultimately made a complete and uneventful recovery without residual neurological deficits.

Conclusion: This case demonstrates that early administration of MPSS during the acute phase of traumatic spinal cord injury can lead to progressive neurological recovery and favorable neurological outcomes. Prompt intervention aimed at minimizing secondary spinal cord injury remains a key determinant of successful recovery in canine acute SCI cases.

Keywords: Acute spinal cord injury, paraplegia, methylprednisolone sodium succinate, blunt trauma, road traffic accident

Introduction

Acute spinal cord injury (SCI) in dogs is a serious neurological condition that results from a traumatic insult to the spinal cord, often leading to significant motor, sensory, and autonomic dysfunction caudal to the site of injury (Sulla *et al.*, 2018) ^[6]. The most common causes of acute SCI are disc extrusion due to degenerative diseases of intervertebral disc, exogenous trauma and infarction due to ischemia (Sulla *et al.*, 2018) ^[6]. In veterinary practice, the most frequent causes of traumatic SCI in dogs include road traffic accidents, falls, and other forms of blunt trauma, with small-breed dogs being particularly vulnerable due to their size and anatomical predispositions (Sulla *et al.*, 2018) ^[6].

The pathophysiology of acute SCI involves an initial mechanical insult that disrupts neuronal axons, blood vessels, and supporting structures, followed by a complex cascade of secondary injury processes such as ischemia, oxidative stress, inflammation, and excitotoxicity, which contribute to progressive neurological deterioration (Dewey, 2008; Sulla *et al.*, 2018) ^[1, 6]. Most of the therapeutic efforts in the medical and veterinary field are focused on reducing the degree of this secondary damage (Eminaga *et al.*, 2011) ^[2]. Thorough neurological examination and lesion localization are essential for accurate diagnosis and prognostication, especially when advanced imaging modalities are not immediately accessible (Sulla *et al.*, 2018) ^[6].

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Clinically, affected dogs may present with a spectrum of neurological deficits, ranging from paresis to complete paraplegia, accompanied by loss of deep pain perception and altered spinal reflexes depending on the level and severity of the lesion (Šulla *et al.*, 2018) ^[6]. Clinical signs vary depending on the location, size, type and duration of spinal cord affections (Dewey, 2008) ^[1].

Diagnostic imaging, including radiography, myelography, computed tomography (CT), and magnetic resonance imaging (MRI), enhances lesion characterization and guides management decisions in traumatic SCI cases (Šulla *et al.*, 2018) ^[6].

Therapeutic strategies for acute SCI in dogs aim to reduce secondary injury, preserve viable neural tissue, and support functional recovery (Dewey, 2008; Šulla *et al.*, 2018; Young, 1993) ^[1, 6, 7]. High-dose methylprednisolone sodium succinate (MPSS), a potent glucocorticoid, has been used in both human and veterinary medicine due to its anti-inflammatory properties and potential to inhibit lipid peroxidation, a key contributor to secondary spinal cord damage. It was known to produce satisfactory results when given in early stages of injury. Although MPSS was historically considered a cornerstone of acute SCI management, its routine use remains controversial. Human clinical studies have shown modest motor improvement when administered within the first 8 hours after injury, but the evidence for long-term benefit and clear clinical efficacy is limited and associated with potential complications (Evaniew *et al.*, 2016) ^[3]. Despite controversies surrounding pharmacologic interventions, early recognition and prompt, comprehensive management; combining stabilization, neuroprotective strategies, and careful monitoring; remain central to improving outcomes in canine traumatic SCI. The present case report details the diagnostic approach and therapeutic management of acute spinal cord injury mediated paraplegia in a six-year-old Pomeranian, with particular emphasis on the use of MPSS and supportive care in the acute phase.

Case history & observations

A six-year-old intact male white Pomeranian was presented with a history of a road traffic accident involving a bike, resulting in blunt trauma a few hours prior to presentation. According to the owner, the dog was apparently normal before the incident and subsequently developed acute onset hindlimb weakness. The animal was able to bear weight to a very limited extent, but frequently collapsed while attempting to walk, indicating ambulatory paraplegia. No history of loss of consciousness, seizures, or prior neurological illness was reported (Fig. 1).



Fig 1: Paraplegia upon presentation

On general physical examination, the dog was alert and responsive, with normal mentation. Vital parameters including rectal temperature, heart rate, and respiratory rate were within normal physiological limits. No external injuries or skeletal deformities were observed. Pain was elicited on palpation of the thoracolumbar region, suggestive of localized spinal discomfort.

Neurological examination revealed severe ambulatory paraparesis characterized by marked hindlimb weakness, poor coordination, and frequent collapsing during attempts to ambulate. Postural reactions, including proprioceptive placing, were delayed in both pelvic limbs, indicating impaired conscious proprioception. Superficial pain sensation was reduced, while deep pain perception remained intact and robust, as evidenced by consistent conscious behavioral responses to noxious digital stimulation. Spinal reflexes of the pelvic limbs were within normal limits, consistent with an upper motor neuron lesion. Thoracic limb function was normal. Urinary bladder tone was mildly increased, further supporting localization of the lesion to the thoracolumbar spinal cord segment (T3-L3).

Based on the neurological deficits, the lesion was neuroanatomically localized to the thoracolumbar spinal cord segment (T3-L3). Routine laboratory investigations performed on Day 0, including complete blood count (CBC), liver function tests (LFT), and renal function tests (RFT), were within normal reference ranges, indicating overall systemic stability at presentation. However, 12 hours post-injury on Day 0, serum C-reactive protein (CRP) levels were found to be elevated (28 mg/L), suggestive of acute traumatic tissue stress and localized inflammatory response secondary to blunt trauma. Additionally, a mild elevation in alkaline phosphatase (ALP; 234 IU/L) was observed, suggestive of systemic inflammatory response secondary to trauma.

Survey radiographs of the thoracolumbar spine revealed normal vertebral alignment with no evidence of fractures, luxations, or vertebral instability. The absence of radiographically detectable skeletal abnormalities, in conjunction with acute neurological deficits following blunt trauma, was suggestive of spinal cord contusion. Advanced imaging was not pursued due to financial constraints.

Based on the preservation of deep pain perception, ambulatory status, and early clinical presentation, the prognosis was considered favorable with timely medical intervention, and the case was immediately initiated on intensive therapeutic management.

Treatment & Discussion

Following confirmation of acute traumatic spinal cord injury with ambulatory paraplegia, the dog was immediately initiated on aggressive medical management aimed at limiting secondary spinal cord damage and promoting neurological recovery. Early therapeutic intervention was prioritized, considering the short interval between injury and presentation and the preservation of deep pain perception.

Modified protocol with Methylprednisolone sodium succinate (MPSS) therapy was instituted within the acute phase of injury. The first dose of MPSS was administered 2 hours post-injury at a dose of 30 mg/kg, given as a slow intravenous infusion diluted in normal saline. For this 10 kg dog, a total dose of 300 mg was administered. A second dose of MPSS at 15 mg/kg IV slow in normal saline (150 mg total dose) was administered 8 hours after injury, both doses being given on Day 0. This dosing regimen was selected to suppress inflammatory cascades, reduce lipid peroxidation, and

minimize spinal cord edema associated with secondary injury mechanisms.

On Day 2, the dog received two additional doses of MPSS at 2.5 mg/kg, administered intravenously as slow infusions in normal saline, at appropriate intervals. These tapering doses were aimed at sustaining anti-inflammatory effects while minimizing potential adverse effects associated with prolonged high-dose corticosteroid therapy.

Concurrent analgesic management and supportive fluid therapy were provided as needed to maintain hydration, optimize spinal cord perfusion, and ensure patient comfort, while strict cage rest and activity restriction were enforced throughout the treatment period to prevent further mechanical insult to the spinal cord; adjunctive neuro-supportive therapy included oral Nurokind® Pet syrup (5 mL/day in two divided doses), multiminerals supplementation, mecobalamin (1500 µg orally once daily), and gabapentin (20 mg/kg orally) administered for 30 days, with regular neurological assessments performed to monitor therapeutic response.

Progressive neurological improvement was observed over the course of hospitalization. By Day 3, the dog exhibited improved hindlimb stability. By Day 7, the patient showed marked clinical improvement, characterized by restored weight-bearing on the pelvic limbs, a normal gait, and normalization of proprioceptive responses, with no observed treatment-related complications or adverse effects.

The favorable outcome in this case can be attributed to early initiation of MPSS within a critical therapeutic window, preservation of deep pain perception, absence of vertebral instability, and comprehensive supportive care. Although the use of MPSS in acute spinal cord injury remains controversial, this case supports its potential benefit when administered judiciously and promptly in carefully selected canine patients.

Acute traumatic spinal cord injury (SCI) initiates a cascade of primary mechanical damage followed by secondary pathophysiological processes, including ischemia, inflammation, free radical production, and excitotoxicity. These secondary mechanisms contribute significantly to progressive neuronal loss and worsened neurological outcomes, forming the rationale for early pharmacologic intervention aimed at attenuating secondary injury processes (Olby, 1999) [5].

In the present case, preserved deep pain perception and early presentation were favorable prognostic indicators, consistent with established findings that early neurological status strongly influences recovery potential. The absence of radiographically visible vertebral instability supported a diagnosis of suspected spinal cord contusion rather than structural disruption.

Methylprednisolone sodium succinate (MPSS) was administered in the acute phase with the aim of limiting secondary inflammatory damage via inhibition of lipid peroxidation and modulation of inflammatory mediators. The theoretical basis for this approach originated from early experimental and clinical studies suggesting a neuroprotective effect of high-dose steroids when administered soon after SCI (Young, 1993) [7]. However, despite its historical adoption, the clinical efficacy of MPSS in improving long-term neurological outcomes remains controversial.

Randomized clinical trials in humans, including the NASCIS (National Acute Spinal Cord Injury Studies), have provided mixed evidence. Some subgroup analyses suggested modest

motor score improvements when MPSS was initiated within 8 hours of injury, but the overall clinical benefit was small and not consistently reproducible across all outcome measures (Fehlings *et al.*, 2017) [4].

Subsequent systematic reviews and meta-analyses have noted that while MPSS may confer a small benefit under specific conditions, evidence for robust, long-term improvement is limited and should be interpreted cautiously (Evaniew *et al.*, 2016) [3].

In the present case, neurological improvement occurred progressively after therapy initiation; however, causal attribution to MPSS alone cannot be established. Recovery is more plausibly multifactorial, influenced by early presentation, preserved deep pain perception, absence of gross mechanical compression, rigorous activity restriction, and comprehensive supportive care, including appropriate analgesia. The contribution of MPSS, if any, should be interpreted within this broader clinical context rather than as the sole determinant of outcome.

Conclusion

In this case report, the successful diagnosis and treatment of acute traumatic spinal cord injury-related ambulatory paraplegia were documented in a male Pomeranian aged six years who was subjected to a road traffic accident. The acute neurological evaluation revealed a preserved sensitivity for deep pain, which rendered a favorable prognosis. The treatment was initiated using high doses of methylprednisolone sodium succinate as recommended, followed by a gradual dosage decrease. The dog demonstrated progressive neurological recovery with the achievement of full weight-bearing and normal ambulatory function without any issues by day five. These aspects highlight the significance of early treatment and a multidisciplinary approach in managing acute spinal cord injuries.

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