“Sam Caught the Ball but had an Unfortunate Fall”

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Introduction:

The following case report will highlight a subcategory of acute intervertebral disk (IVD) herniation, one of the most common spinal emergencies seen in dogs.[2] Acute Non-compressive Nucleus Pulposus Extrusion (ANNPE) was historically termed Hansen type 3 IVD disease, but the current terminology has been changed as it describes the key characteristics of a sudden extrusion of nondegenerate nucleus pulposus, which causes spinal cord contusion without significant compression.[2] Dogs of any age or breed may develop this disease, although older large-breed dogs, especially Border Collies and sighthounds seem to have a predisposition to this condition. [2, 5, 6, 7] Patient's characteristically present with a peracute onset of moderate to severe neurologic deficits, where the clinical signs are often lateralized and remain nonprogressive after the first 24 hours; 90% of patients present this way.[2] Clinical signs vary and are distributed according to the neuroanatomical location and severity of the lesion, but patients usually stabilize within 24 hours and then their condition improves or eventually remains static. [2] Although patients frequently vocalize at the onset of neurological signs, such as yelp as if in pain, this condition is not typically associated with severe or sustained spinal pain.[2, 3] In previous studies, patients have generally received a fair prognosis associated with ANNPE for recovery of motor function. [2, 6] Overall recovery rates are variable, with successful outcomes ranging based on severity of spinal cord injury (SCI), hospitalization time, and severity of neurologic dysfunction.[2] This case emphasizes the importance of addressing and reaching a presumptive diagnosis in a timely manner and implementing physical therapy to improve chances of recovery in patients with ANNPE. This case report will review the clinical signs, diagnostics, pathophysiology, and treatment of ANNPE, as well as the outcome of the patient, Sam, who was seen by the Neurology Department at Mississippi State University, College of Veterinary Medicine.

History and Presentation:

Sam is an approximately 7-year-old, male neutered Labrador Retriever dog. He presented to Mississippi State University College of Veterinary Medicine's Emergency Department on June 12, 2021 around 5am, for becoming acutely down in the hind. His owners reported Sam became acutely symptomatic while chasing a ball that evening in their yard. His owners noted that while chasing the ball, Sam slid in the wet grass and was unable to get up after the event. They report the event happened several hours earlier and that his condition has not improved since his fall, but he did not appear to be in pain. They did not report any history of previous medical conditions, with regards to Sam.

On presentation, Sam was bright, alert, and responsive. He weighed approximately 42.0 kilograms with a body condition score of 7/9. His mucous membranes were pink and moist with a capillary refill time of less than 2 seconds, indicating adequate hydration status. Vital parameters included a temperature of 103.2° F, a heart rate of 112 beats per minute, and a panting respiratory rate. Cardiopulmonary auscultation was within normal limits with no murmurs, arrythmias, crackles, or wheezes appreciated. Auscultation of his lungs revealed normal bronchovesicular sounds. His abdomen was soft and non-painful on palpation. All palpable lymph nodes were smooth, soft, and symmetrical. Neurologic examination revealed Sam was unable to stand on his pelvic limbs, even with assistance. When walked with the assistance of a sling there appeared to be minimal motor function in the pelvic limbs. Conscious proprioception was not present in either pelvic limb but was present bilaterally in the thoracic limbs. When Sam was placed in lateral recumbency, he demonstrated a Schiff Sherrington posture. Withdrawal reflexes were decreased bilaterally in the pelvic limbs and the Gastrocnemius reflex was decreased-absent bilaterally in the pelvic limbs. Nociception was

present bilaterally in the pelvic limbs. His Cutaneous trunci reflex was present cranial to L1-L2. No pain was elicited on palpation of the spinal column and Sam had good range of motion of the neck. All cranial nerves appeared normal. Sam had an intact menace OU, and palpebral and PLR's (direct and indirect) were normal. No strabismus nor positional nystagmus were noted, and physiological nystagmus was normal. Nasal stimulation was normal bilaterally. He was classified as non-ambulatory paraparetic with an T3-L3 myelopathy neurolocalization with spinal shock accounting for the depressed reflexes in the pelvic limbs Sam was started on Gabapentin 10 mg/kg PO q8 hours and Trazodone 3.5 mg/kg PO q8 hours in ICU and was scheduled to be transferred to MSU-CVM Neurology department the next day with plans for a full workup.

Diagnostic Approach:

Following initial physical examination, neurolocalization was determined to be T3-caudal. Differentials included Acute Non-compressive Nucleus Pulposus Extrusion, Fibrocartilaginous Embolism (FCE), Intervertebral disc herniation, trauma, and neoplasia. ANNPE and FCE were at the top of the differential list, however definitive diagnosis can only be made post-mortem by histopathology of the affected spinal cord segment. [2, 3, 4, 5, 6] Antemortem diagnosis of ischemic myelopathy is therefore based on history, clinical findings, and exclusion of other causes often by utilizing computed tomography and magnetic resonance imaging. [4, 8] Computed tomography (CT) as well as magnetic resonance imaging (MRI) are often used to assess damage to the spinal canal and associated structures by obtaining detailed images of the complex and overlapping structures. [1, 4] MRI is considered the gold standard for diagnostic imaging in ischemic myelopathy because it may reveal signal-intensity changes compatible with spinal cord infarction.[1, 4, 8]

The first step in our diagnostic plan, due to weekend availability of imaging modalities was to sedate Sam for a CT study with contrast to assess the vertebral column and associated structures. Sam was sedated with Methadone 0.1 mg/kg IV and Dexmedatomidine 5mcg/kg IV. This assessment was necessary to determine if a diagnosis of an osseous neoplasia or vertebral fracture/subluxation could be ruled out. Although CT with contrast is often diagnostic for the localization of IVDE in chondrodystrophic dogs, it had to be mentioned that a further diagnostic work up utilizing MRI may be required if no lesion was seen or if a lesion was identified that did not match the neurolocalization.[1] It's also worth mentioning that while the spinal cord is visualized on CT, changes to the spinal cord parenchyma cannot be visualized. [1, 4, 8] The CT scan reported mild osteophyte formation of the sacroiliac joints bilaterally as well as a mild midline shift of the spinal cord, but no cord compression was visible, therefore, surgery was not indicated at this time. However, due to the patient's clinical status, an MRI was indicated to obtain further information. At this point our top differentials were FCE vs. ANNPE, and the patient was treated as such while awaiting MRI. While awaiting further imaging during his hospitalization, it was noted that Sam's urine had a foul smell and he was frequently voiding his bladder. Urine was submitted for urinalysis and culture, which revealed too numerous to count white blood cells and bacteria indicating a urinary tract infection.

On day 5 of hospitalization, Sam was placed under general anesthesia for an MRI. The findings were as follows: the T12-T13 intervertebral disc space was narrowed with decreased T2 hyperintensity. A moderate amount of T1 FS, T2, and T2 FS hyperintense, T2* isointense material was within the spinal cord occupying up to 80% of the spinal cord diameter at the level of T12-T13. The material extended into the dorsal subarachnoid space and penetrated through the dorsal arcuate ligament. The spinal cord in this section was moderately enlarged and

heterogeneously T2 and T2 FS hyperintense. The lesion was determined to be non-compressive. These findings allowed for the diagnosis of an Acute Non-compressive Nucleated Pulposus Extrusion at T12-T13 with spinal cord and arcuate ligament dissection/penetration, spinal cord swelling/edema and acute epidural hemorrhage.

The combined results of the diagnostics performed on Sam lead to a tentative diagnosis of Acute Non-compressive Nucleus Pulposus Extrusion at the level of T12-T13. He was referred to MSU-CVM's Rehabilitation department in hopes that physical therapy would improve his mobility.

Pathophysiology:

Acute Non-compressive Nucleus Pulposus Extrusion refers to the spinal cord being contused by acute extrusion of the hydrated nucleus pulposus from an underlying intervertebral disc due to a sudden increase in intradiscal pressure, often during or following vigorous exercise such as running and jumping. [2, 3, 6] In order to understand the pathogenesis of ANNPE, we must first have an appreciation for the normal canine IVD anatomy. There is a strong osmotic gradient in the normal, nondegenerate nucleus pulposus, this gradient allows for water to be drawn into the nucleus pulposus which creates a naturally high intradiscal pressure. [2] The normal IVD is able to withstand significant variations of physiologic loading and biomechanical stress without suffering structural compromise due to the combination of healthy hydrated nucleus pulposus, which is surrounded by a dense and fibrous annulus fibrosus.[2, 3] However, in situations where the vertebral segment and IVD are exposed to supraphysiologic forces, for example during strenuous exercise or trauma, the structural integrity of the vertebral segment may fail.[2, 3] Under these circumstances, a small tear may happen in the complex lamellar structure of the annulus fibrosus,

which can then lead to a sudden extrusion of nondegenerate nucleus pulposus material upward dorsally into the vertebral canal.[2, 3] It's hypothesized that in ANNPEs the nuclear material extrudes with such force that is can cause a focal contusive injury to the adjacent spinal cord.[2, 3] Extradural material is nondegenerate and highly hydrated, therefore it typically dissipates rapidly or is resorbed, leaving minimal or no spinal cord compression.[2, 3] This hypothesis has been supported by postmortem findings in affected dogs with small tears in the dorsal annulus and the presence of extradural nondegenerate nucleus pulposus material in the vertebral canal.[2, 3] The region of the spinal cord adjacent to the site of extrusion may further demonstrate evidence of focal contusive injury, hemorrhage, and necrosis.[2]

Treatment:

At this time, there are no neuroprotective treatments available with proven efficacy to directly treat the contusive primary SCI.[2, 6] Successful treatment of ANNPE consists of supportive medical management, including restricted activity, supportive care, and physical rehabilitation.[2, 3, 6] Fourty eight to fifty seven percent of dogs with ANNPE present with evidence of spinal hyperesthesia, therefore treatment with analgesics may be indicated initially.[2] To minimize the risk of further extrusion of nuclear material, restricted activity and short lead walks have been recommended for a period of 4-6 weeks.[2, 3, 6] In order to prevent secondary complications, supportive care requirements are essential and vary depending on the severity of neurologic dysfunction.[2] Nursing care requirements to aid in recovery may involve manual bladder expression or urinary catheter maintenance in cases of urinary incontinence, prevention of dermatologic consequences of prolonged recumbency, such as urine scald or pressure sores, nutritional support to maintain the patient's body condition in order to support their physical rehabilitation, and monitoring and managing respiratory dysfunction which includes turning

recumbent patients every 4-6 hours to avoid atelectasis.[2, 3, 6, 7] Physical rehabilitation is being increasingly recognized in both human and veterinary medicine in supporting the recovery of patients with SCIs.[2] The goals and requirements of physical therapy are dictated by the severity of neurologic dysfunction, but the typical goal is to maintain range of motion in the joints, minimize muscle atrophy, and maintain the patient's comfort level during the recovery time.[2]

Case Outcome:

When Sam presented to MSU-CVM Neurology service he had positive nociception, however on day 6 in ICU it was noted that Sam had absent nociception in the right pelvic limb and severely blunted to absent in the left pelvic limb. This combined with the severity of the spinal cord lesion on MRI gave Sam a grave prognosis for return of function to the pelvic limbs. Sam received nursing care consisting of pain and bladder medical management along with 4 days of physical therapy but continued to decline. Due to Sam's guarded prognosis for return to function, his declining neurologic status and lack of response to physical therapy and the fact that his owners felt his quality of life was diminished they elected humane euthanasia. The owners consented to a necropsy which revealed extrusion of disc material and hemorrhage extending from T10-L1, which led to mild compression and severe necrosis of the adjacent neuropil. Based on the patient's clinical history, radiographic findings, and postmortem exam findings, the lesion was found to be most consistent with an Acute Non-compressive Nucleus Pulposus Extrusion. There were also findings of other cord lesions such as myelin degeneration and Wallerian degeneration with spheroids and axonal loss, all of which are secondary lesion to nucleus pulposus extrusion.

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