

A Case of an Acute Non-compressive Nucleus Pulposus Extrusion

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Class of 2019

Clinicopathologic Conference

January 11, 2019

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Introduction

Acute spinal cord injury is commonly reported in dogs, most frequently due to Hansen type 1 intervertebral disk disease (IVDD). Hansen type I IVDD occurs when a degenerative disk has extruded into the spinal canal, causing compression of the spinal cord. However, with the increasing use of magnetic resonance imaging (MRI), we are becoming aware of newer causes of acute spinal cord injuries with similar initial presentations. Small amounts of non-degenerative nucleus pulposus can extrude into the spinal canal with such force as to cause contusions to the spinal cord without causing concurrent compression. While the initial presentation of acute spinal cord injuries may be similar, it is important to reach a definitive diagnosis due to the different treatments and prognosis.

Several terms have been used to describe this acute non-degenerative disk rupture. Historically, terms such as Hansen type III IVDD and high-velocity low-volume disk extrusion have been used, however acute non-compressive nucleus pulposus extrusion (ANNPE) has become the most widely accepted term. The term ANNPE accurately describes this condition in that peracutely, non-degenerative nucleus pulposus ruptures into the spinal canal causing spinal cord contusions without spinal cord compression. The resulting neurologic deficiencies differ depending on the segment of spinal cord affected, but signs generally stabilize within 24 hours and improve over the course of a few days to weeks depending on the severity of the injury¹.

History and Presentation

Crush is a 6 year-old male intact Keeshound who presented to the Mississippi State University College of Veterinary Medicine Emergency Service early on 5/3/18 from the Animal Emergency and Referral Center (AERC) after he became acutely 'down' in his pelvic limbs. He was playing in the yard with his canine housemate when his owner noticed that he could not move his back legs. She took him to AERC where he was evaluated and referred to the MSU-CVM Neurology service. On presentation to the MSU-CVM, Crush was anxious and "dog sitting" with his hindlimbs extended forward. His temperature was 102.2F, his heart rate was 112 bpm, and he was panting. He weighed 20.4kg and his mucous membranes were pink with a capillary refill time (CRT) of less than 2 seconds. His SPO₂ was 100% and there were no significant findings on his ECG. His heart and lungs auscultated normally and there were no abnormalities on abdominal palpation. On neurologic examination, Crush displayed normal behavior, mentation and cranial nerves. There were normal postural reactions and reflexes in the forelimbs. He was assessed as non-ambulatory paraparetic, displaying decreased motor function of the hindlimbs with absent postural reactions bilaterally and normal reflexes in the hindlimbs and perineum. Cutaneous trunci was absent caudal to L2 with no obvious hyperpathia on spinal palpation. Due to the severity of his neurologic status, an ultrasound was used every 6 hours to measure his bladder size and he was catheterized as needed. Crush was then transferred to the MSU CVM Neurology Service with a neurologic localization of a T3-L3 myelopathy.

Diagnostic Approach/ Considerations

Upon presentation to the Neurology service, his neurological exam was unchanged from presentation thus several diagnostics were performed. A pre-anesthetic diagnostic approach was taken which included a complete blood count revealing a mild lymphopenia (678K/ul 1200-6500), a mild monocytopenia (113K/ul 175-1700), and a mild eosinopenia (113K/ul 120-1300).

A serum chemistry was performed revealing a low CO₂ (13.4mEq/L 20-28), a mild hypocalcemia (8.5 mg/dL 8.8-11.2), and a severely elevated CK (2410 U/L 50-300). A urinalysis was performed revealing green colored urine with a specific gravity of 1.032, with an elevated protein (2+) and SSA (2+), with a large amount of blood (50-100 RBC), and 1-3 coarse granular casts. However, a urine culture revealed no growth. Thoracic radiographs and a full body metal scan were performed revealing no significant abnormalities.

Given Crush's history and neurologic examination findings, the two most likely differentials were an acute non-compressive nucleus pulposus extrusion or a fibrocartilaginous embolism (FCE). However, IVDD could not be ruled out. An MRI was then performed revealing a mild decreased T2 signal intensity of the nuclei pulposus at T13-L1, L2-3, and L7-S1. There was also a mild dorsal protrusion of the annulus fibrosus at T13-L1. Within the dorsal aspect of the vertebral canal at the mid body of T13, there was a poorly defined mildly contrast enhancing region. There was a poorly defined T2 and T2 FS hyperintense region within the spinal cord at this site as well that was mainly oriented in a dorsoventral direction, and the dorsal margin of the spinal cord was irregular. These findings were indicative of an extradural non-compressive myelopathy with diffuse spinal cord T2 weighted hyperintensity at T13-L1 with consideration given to an acute non-compressive nucleus pulposus extrusion with likely concurrent hemorrhage in the vertebral canal.

The key MRI findings that suggested a diagnosis of an ANNPE rather than an FCE include a decrease of the T2 signal intensity of the nucleus pulposus with a corresponding T2 hyperintense region of the spinal cord directly over it. Other criteria that can be used for diagnosis include a narrowing of the associated disk space, none to very mild signs of spinal cord compression, and a small volume of extradural material dorsal to the affected disk space¹. These

findings differ from that of a FCE which will have a sharply demarcated intramedullary lesion usually of the gray matter that is hyperintense to normal gray matter on T2 and FLAIR images and longer than one vertebral body length⁵.

Pathophysiology

In order to understand the pathophysiology of an ANNPE, anatomy must be taken into consideration. An intervertebral disk, found between each vertebral body, is composed of an inner nucleus pulposus and an outer annulus fibrosis with a transition zone between the two portions. The nucleus pulposus is normally composed of greater than 80% water and is commonly referred to as the “jelly” portion of the disk. The annulus fibrosis surrounds the nucleus and is composed of collagen layers. It is also important to recognize that the annulus fibrosis is thinner dorsally than ventrally¹. The function of the intervertebral disk is to provide stability as well as motion in order to transmit compressive forces across the spinal column². Due to the osmotic nature of the nucleus pulposus, it consistently has a higher pressure than the surrounding annulus fibrosis².

It has been proposed that an alteration in the forces applied to the vertebrae causes a tear in the annulus fibrosis, allowing the high-pressured nucleus pulposus to rapidly enter the spinal canal³. Commonly, these events of altered forces occur during intense exercise or trauma⁴. When the nucleus pulposus extrudes under this high pressure, necropsy evidence shows contusions to and hemorrhage within the spinal cord¹. Some of these effects can also be seen on MRI as described above in Crush’s MRI findings.

Clinical signs associated with an ANNPE depend on the location of the expulsion, however, the most commonly affected region is T3-L3⁴. Also, almost 90% of cases will present with lateralized neurological deficits³. Usually, neither an FCE or an ANNPE will present with spinal hyperpathia, unlike IVDD. However, there are several reports of dogs crying out or initially presenting responsive to stimuli⁴. Within 24 hours these cases were no longer responsive to spinal palpation⁵. Also, maybe the most important criteria for suspecting an ANNPE or FCE, the clinical signs are non-progressive after 24 hours³.

Treatment and Management

As of now, there is no specific treatment for ANNPE; thus, supportive care and physical therapy is the mainstay of treatment. However, since there are some dogs that in early presentation will have spinal hyperesthesia, analgesia is commonly prescribed for the at least the first 24 hours¹. The location of the lesion and severity of neurologic deficits will determine the type of supportive care needed, such as manual bladder expression, caloric supplementation, prevention of pressure sores and cubital ulcers, etc. Strict activity restriction is usually recommended with controlled leash walks for 4-6 weeks after the incident, in order to prevent any further disk material extrusion¹. As with any spinal cord injury, physical therapy is the best long- term therapy, the severity of the injury dictating the amount and length of therapy needed. In hospital, therapy is indicated for assisting with patient comfort, and to reduce the amount of muscle atrophy that occurs.

In this case, Crush was initially prescribed methadone as an analgesic, then switched to gabapentin and acetaminophen with codeine. A urinary catheter was placed and maintained for three days. Crush was also prescribed diazepam as a muscle relaxant and to aid with bladder expression. Bethanechol was administered to assist with manual bladder expression. Physical

therapy was initiated which included passive range of motion exercises, underwater treadmill therapy, and sit to stand exercises.

Case Outcome

The prognosis for an acute non-compressive nucleus pulposus extrusion is excellent with some publications reporting 100% recovery¹. Most patients regain continence and ambulation. Recovery has been associated with the severity of the spinal cord insult, with less severe insults reaching more complete recoveries⁴. Those injuries with loss of nociception having higher incidence of not regaining continence¹. One must also consider that most of these studies are reviews of cases and inclusion criteria, presumptive diagnosis, and recovery definitions all vary. It is also important to consider the range of recovery times associated with presumptive diagnosis of ANNPE, some articles reporting recovery of ambulation as early as one day, others reporting that some dogs still show improvement after months from time of diagnosis⁵.

Crush was hospitalized for two weeks during which time he regained urinary and fecal continence (within 5 days) as well as stability and ambulation (within 14 days). Crush is considered to have achieved a full recovery from his spinal cord injury with minimal lasting gait abnormalities.

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