Intervertebral Disk Disease

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Class of 2018
Clinicopathological Conference
November 10, 2017

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Introduction:

In human medicine, the recognition of spinal disorders dates back to Egyptian, Greek, Roman, and Arabic texts. Hippocrates was likely the first to acknowledge the connection between back pain and injured vertebrae. He was also the first to recognize that signs, like back pain, were observed on the same side of the body as the lesion on the cord.[2,6] In 1881, the first report described in canine species was by Janson. He described the condition as a chondroma mass compressing the spinal cord. The first correct report was done by Tillman in 1939 and described extrusion of the nucleus pulposus. In 1952, Hansen documented the first accurate description of intervertebral disk disease (IVDD), which categorized the condition into type I and type II.[12,13] These classifications are still used in current medicine.

History and Presentation:

A 5-year-old neutered male German Shepherd dog (27.9 kg) presented to the Veterinary Specialty Center (VSC) for an acute onset of pain in the lower spine after falling onto a bedpost. A magnetic resonance imaging (MRI) scan was performed that revealed an intervertebral disk protrusion at L7-S1 with the left side being more affected. An epidural steroid injection was administered immediately following the MRI. The referring veterinarian performed a second epidural steroid injection a week later. Neither epidural injection's seemed to result in significant improvement. The dog was maintained on gabapentin and cannibidiol oil. About 5-weeks after original presentation to VSC, the owner brought the dog back for further work-up and treatment due to the continued presence of lower back pain and discomfort climbing stairs. The dog's respiratory rate was 40 breaths per minute, heart rate was 116 beats per minute, and temperature was 102.1°F. The heart and lungs auscultated within normal limits. There were no other significant findings.

Neurologic examination revealed that the dog was ambulatory with normal mentation and no cranial nerve deficits. There was a moderate amount of lumbosacral and iliopsoas pain elicited on palpation. Rectal examination further supported lumbosacral pain. Conscious proprioception was slightly delayed in the left hind limb. The remainder of the neurologic examination was within normal limits.

Pathophysiology:

The canine vertebral column has seven cervical vertebrae, thirteen thoracic vertebrae, seven lumbar vertebrae, and three fused sacral vertebrae. Each vertebra is composed of a body, vertebral arch, and various processes. The vertebral arch consists of two pedicles that form the walls of the vertebral foramen, while the laminae form the roof. Interarcuate ligaments connect each lamina. Cranial to the eleventh thoracic vertebra the intercapital ligaments lie dorsal to the annulus. It is suggested that disk herniation is less common in these areas due to the anatomical location of the ligament providing more strength to the T1-T10 vertebrae. The spinal cord is housed in the vertebral canal, which is formed by the bodies and vertebral arches. Between each body are the intervertebral disks. These disks provide stability and flexibility to the vertebral column and the gelatinous nucleus pulposus function as a shock absorber.[3,4,6,7,10] According to Griffin, these disks account for about 16% of the vertebral column length in the thoracic and lumbar regions.[4]

There are three anatomic regions in each disk: annulus fibrosis, nucleus pulposus, and cartilaginous end plate. The annulus is composed of type I collagen that forms the lamellae and have the ability to glide over one another during biomechanical loading. In the canine, the annulus is thickest ventrally and sparsely innervated peripherally, which explains why most herniation occurs dorsally.[4,10] The nucleus pulposus forms the center of the disk and contains an

extracellular matrix composed of water and proteoglycans. The proteoglycans are produced and assembled by notochordal cells that contribute to the regulation of intervertebral disk chondrocyte proteoglycan production and cell proliferation. The proteoglycan content changes and the water-binding capacity of the nucleus decreases primarily because of inherited factors in the young chondrodystrophic dogs and as an aging factor in other breeds.[4,8,10,11] The cartilaginous end plate is composed of hyaline cartilage and is the site of attachment between the intervertebral disk and the vertebral body. Nutrients diffuse across the end plates to the intervertebral disks. Occlusion of the end plates may contribute to disk degeneration due to insufficient nutrient availability.[8]

As defined by Hansen, there are two types of canine IVD disease: Type 1 involves extrusion of the disk and type II describes intervertebral disk protrusion. There has since been another form of disk disease, often referred to as type III, although it was not originally included in the Hansen classification. The type III has more recently been termed acute noncompressive nucleus pulposus extrusion (ANNPE).[3, 11] The two patterns of disk degeneration are fibroid and chondroid metaplasia. Various predisposing factors make an individual more prone to developing a certain type of disk herniation. Nonchondrodystrophic dogs maintain their intervertebral disk notochordal cells into adulthood, whereas chondrodystrophic breeds do not. Degenerative disk disease may be hindered by the preservation of notochordal cells. [4,11]

With chondrodystrophic breeds, such as the Basset Hound, Dachshund, Pekingese, French Bulldog, Welsh Corgi, Beagle, and American Cocker Spaniel, the hyaline cartilage replaces the nucleus and further degeneration leads to calcification of the nucleus.[7,8,10,11] Dachshunds have been found to be the breed affected the most. Further study of the breed suggests that IVDD is inherited in an autosomal polygenic manner and may be related to hair

coat type.[4] Due to the degenerative changes, the intervertebral disk loses the ability to absorb compressive pressures. The nucleus rigidity causes the annulus to tear and the degenerate nuclear material to be extruded into the vertebral canal. Chondroid metaplasia and degeneration commences at a few months of age but is compromised to the point that clinical signs associated with acute extrusions occur on average by 3 to 6 years of age.[4,5] It is proposed that by one year of age, 75% to 100% of chondrodystrophic dogs with IVDD undergo disk degeneration.[11] Due to the notochord being an immunopriviledged structure, when the nucleus pulposus is exposed to the immune system, inflammation develops. This is considered Hansen type I disk disease and is characterized by complete rupture of the annulus fibrosis with translocation of the nucleus pulposus into the vertebral canal.[5] This type of herniation is more common in young adult chondrodystrophic dogs, commonly occurs at the T12-T13 or T13-L1 disk spaces, and is typically a more acute process complicated by mineralization of the intervertebral disk.[4,7]

The fibroid form of metaplasia is a pathologic response to chronic torsion. This typically occurs in nonchondrodystrophic breeds around 8 to 10 years of age and involves the replacement of the nucleus with fibrocartilage. This type of disk protrusion usually leads to chronic compression of the spinal cord. This is considered a Hansen type II disk disease and is characterized by protrusion of the intervertebral disk. The disk protrusion is caused by rupture of the inner layers of the annulus fibrosis, partial displacement of the nucleus into the disrupted annulus, and annular hypertrophy.[5] This occurs more commonly in older, large-breed dogs and is a more chronic condition rarely seen with mineralization of the intervertebral disk.[4] About 80% of disk extrusions occur between the T13 and L3 vertebrae.[8]

The most common signs associated with thoracolumbar IVDD are paraspinal hyperesthesia and pelvic limb proprioceptive ataxia, followed by ambulatory paraparesis.

Clinical signs with type I protrusions often involve severe pain following an episode of trauma, which leads to sudden hind limb paresis or paralysis. Type II protrusions most often present as discomfort climbing stairs or jumping onto surfaces, or a reluctance to move. With lesions in the lumbosacral region, the cauda equina can become compressed within the vertebral canal causing signs sometimes associated more with pain and lameness than neurologic deficits. This area is susceptible due to the transfer of locomotive forces from the pelvis to the vertebral column.[7] The neurologic dysfunctions associated with disk herniation are associated with many factors. The dynamic force of the herniated disk material causes injury to the spinal cord or nerve roots. Physical displacement of the spinal cord by the disk material in the epidural space causes secondary pathologic effects. Hypoxic changes can occur with pressure on the vascular system within the cord, which progresses to ischemia and edema.[4,7,11]

Diagnostic Approach/ Considerations:

The diagnostic approach begins with the history and signalment, including all pertinent medical and neurologic information. This is followed by a thorough physical and neurologic examination. Loss of the cutaneous trunci reflex is suggestive of a lesion located one or two vertebrae cranial to the cutoff point.[5] Based on these initial steps, it is possible to localize the level of the spinal cord lesion in the thoracic and lumbar spine.

A more thorough evaluation should include a complete blood cell count, serum chemistry panel, urinalysis, and thoracic radiography. These are not only helpful to determine the current health status of the patient, but also to evaluate the cardiovascular structures and to rule out many possible comorbidities. Vertebral column radiography is also suggested to rule out diskospondylosis, trauma, or vertebral neoplasia. There are many radiographic signs that are suggestive of intervertebral disk herniation: narrowing of the disk space, decreased size of the

intervertebral foramen, reduced space between articular facets, and mineralized disk material in the vertebral canal or overlying intervertebral foramen.[12] Radiography alone rarely provides a definitive diagnosis of IVDD but may provide direction for additional imaging. Myelography, computed tomography (CT), or MRI is used to further characterize and localize the lesion before prior to surgery or additional procedures.

Myelography correctly identified the site of disk herniation in 85.7% to 98% of cases.[5] On the lateral view, spinal cord compression is viewed as dorsal deviation of the ventral subarachnoid contrast column and thinning of the dorsal contrast column dorsal to a disk. The major advantage of myelography is the ability to determine the lateralization of disk extrusions. It is also fairly inexpensive and does not require specialized equipment. The major disadvantages are the adverse side effects and artifacts caused by the epidural contrast injection. Commonly used contrast media are non-ionic iodinated agents such as iohexol or iopamadol. Seizures, especially in large breed dogs with cisternomedullary injections, are the most common side effect, but tend to be a short-term issue and not life-threatening .[3,5,9,11]

Computed tomography (CT) alone is less invasive than myelography and more accurately demonstrates gross morphologic changes in the disk; however, is not very effective at differentiating structures within the dura or assessing subtle changes suggestive of disk degeneration. Often, especially in non-chondrodystrophic dogs without disk mineralization, CT is combined with myelography. Herniated disk material appears as a heterogeneous, isoattenuating-to-hyperattenuating extradural mass. With increasing mineralization, the herniated disk material will become more homogenous and hyperattenuating.[5,7,9]

Magnetic resonance imaging is quite helpful in identifying IVDD and allows for early classification of disk herniation as extrusion, protrusion, or bulge. MRI was found to be 100%

sensitive and 79% specific for detecting disk degeneration in nonchondrodystrophic dogs.[5] The T2-weighted images directly correlate with the signal intensity of the nucleus pulposus proteoglycan concentration. When visualizing anatomic detail, T1-and proton-density-weighted images are most useful.[3,5,7] One study looked at the correlation between MRI features of canine disk extrusion accompanied by epidural hemorrhage or inflammation to compare those features with clinical signs and pathologic findings. The conclusion was that the prognosis of dogs with the mentioned disk extrusion did not appear to be different than for dogs with disk extrusion without imaging signs of epidural hemorrhage or inflammation.[9] There are several advantages when using MRI: demonstrates subtle degenerative changes in the intervertebral disk, provides more detailed localization of extruded disk material, allows for assessment of soft tissue structures, and less complications than when using myelography contrast. The main disadvantages are limited availability, the necessity of general anesthesia, and increased expense.[1,5,7,9] CT scans alone in chondrodystrophic dogs using only sedation are often diagnostic.

While often not needed, cerebral spinal fluid (CSF) analysis can be performed. This is used to rule out infectious or inflammatory causes of neurologic disease. With a study of 400 dogs that had concurrent CSF analysis and MRI performed, the most common clinical diagnosis was IVDD, followed by inflammatory disease, idiopathic disease, and brain and spinal neoplasia. The sensitivity of CSF analysis was 75% and the sensitivity of MRI was 89% in detecting an abnormality. MR images were abnormal 100% of the time in dogs with vertebral malformation/ instability and with IVDD.[1] CSF analysis is most helpful in ruling out meningomyelitis before surgery is performed.[5]

Treatment and Management:

There is a grading system for spinal cord injury that helps assess whether medical management or surgical intervention is most advantageous. Grade 1 simply includes dogs exhibiting pain along the spinal column. Grade 2 patients have pain and paraparesis, but are still capable of ambulatory paraparesis. Grade 3 involves pain, non-ambulatory paraparesis or paraplegia. Grade 4 has pain, paraplegia and urinary incontinence. Grade 5 has paraplegia, urinary incontinence and loss of deep pain perception. Grade 5 has a poorer prognosis and is a surgical emergency. The most important prognostic indicator is the presence or absence of nociception in the hind limbs. A very poor prognosis is given to a patient with loss of deep pain sensation for more than 24 hours.[11]

Medical management is indicated when there is ataxia, paresis with or without pain, no previous history of disk disease, or when there are financial restrictions. Surgery should be strongly considered with recurrent episodes of disk disease. Medical management revolves around strict cage rest for 4 to 6 weeks, opioid analgesics with concomitant use of gabapentin, and when indicated, muscle relaxants. Due to muscle spasms being the main source of pain, muscle relaxants are often indicated and diazepam is the drug of choice. Client education is very important for medical management to be successful. The owners must be willing to appropriately confine the animal and watch for signs of dysuria or worsening neurologic condition. If the owners are willing to have the patient hospitalized during medical management, then injectable analgesics and anti-inflammatory doses of prednisone will make the dog more comfortable and minimize disk inflammation. [5,7,11,12] Recent studies have explored the use of bee venom as an anti-inflammatory and analgesic. A single-blind control study was performed using 40 canines with neurologic deficits related to IVDD. Compared to the control treatment, the myelopathy scoring system grade and functional numeric scale scores were improved in the dogs injected

with bee venom. The overall result was that bee venom injections used at acupoints were found to be more protective in dogs with IVDD-induced neurologic dysfunction and pain than treatment alone.[13] While medical management is an option, surgical therapy is often indicated and will allow for a more complete and rapid recovery.

The goal of surgery is to decompress the spinal cord and remove herniated disk material. Surgery is indicated when there is pain or paresis unresponsive to medical therapy, recurrent or progressive signs of neurologic disease, paraplegia without sensory paralysis, or sensory and motor paralysis for less than 24 hours. The surgical procedure is based on the patient and the experience of the surgeon. Every patient that is undergoing surgery must have radiographic or advanced imaging evidence of an intervertebral disk protrusion that corresponds with the neurologic signs. The options for surgical procedures for thoracolumbar IVDD are hemilaminectomy, pediculectomy, or lateral corpectomy.[5,7,11]

Fenestration is most often performed on dogs with Hansen type II intervertebral disk protrusion presenting with only back pain or mild paresis. This procedure is used as a prophylaxis in combination with decompressive surgery or following successful medical management. Fenestration is the removal of the nucleus pulposus via perforation and curettage of the intervertebral disk space. Fenestration is routinely used when an intervertebral disk of T11-T12 through L3-L4 is affected.[5,6,11]

Decompressive surgery involves the removal of the dorsal or lateral components of the vertebral arch to relieve pressure on the spinal cord. With hemilaminectomy, the articular facets, laminae, and pedicle are removed. This surgical technique allows for better visualization of the herniated disk material. Dorsal laminectomy involves the removal of the spinous process, laminae and the articular processes or pedicle and is more often performed at L6-L7 or L7-

S1.[5,11,12] Dorsal laminectomy is performed using the Funquist B method or the modified dorsal laminectomy method. These procedures differ in the width of dorsal laminae removed. The patient is placed in sternal recumbency and a skin incision is made on the dorsal midline the length of two vertebrae cranial and caudal to the affected disk space. Using bone rongeurs, the spinous processes of one vertebra cranial and one vertebra caudal to the disk space is removed. With the Funquist B dorsal laminectomy, the dorsal laminae are removed with the width stopping short of the articular processes. This procedure provides good visualization and decompression of the spinal cord; however, due to the increased muscle dissection, there may be more cosmetic defects.[11]

Case Outcome:

The patient underwent dorsal laminectomy surgery. He recovered from anesthesia well and had no complications. He stayed in the intensive care unit at MSU for 3 days. He was given trazodone (3.5 mg/kg, PO, q 8 h) and gabapentin (10 mg/kg, PO, q 8 h) to help control his anxiety while in hospital. The patient was still very anxious the second day after recovering from surgery, so he was started on midazolam (0.2 mg/kg, IV, q 8 h). He was given cerenia (2 mg/kg, PO, q 24 h) as an anti-emetic. Famotidine (0.5 mg/kg, IV, q 12 h) was started to reduce gastric acid production as a prophylactic for gastrointestinal upset. The surgical site was iced every 6 hours for 3 days, followed by a warm compress every 6 hours for 2 days. The patient was sent home with gabapentin, trazodone, Tylenol 4, and diazepam. The owner was advised to restrict the dog's activity for 4 weeks.

Following surgery, conscious proprioception returned to the left hind limb area. The hyperpathia elicited in the lumbosacral region and iliopsoas was no longer present. The patient also had easily elicited nociception in all limbs. Laser therapy was performed for two days to

help decrease the healing time. The owner has stated in a follow-up phone conversation that Duke is very much improved. He is continuing with physical therapy at home and has been evaluated at a surgery specialty center. All indications are Duke is progressing very favorably and the owner states Duke is “totally much better.”

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