Intervertebral Disc Disease (IVDD)

Chelsea A. Meyer
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CPC Advisor:
Michaela J. Beasley, DVM, MS, DACVIM
Assistant Clinical Professor – Neurology

INTRODUCTION

Intervertebral discs are present throughout the entire canine spine. They connect each individual vertebrae with the exception of C1-C2, also known as the atlanto-axial joint, and the fused sacrum.[1] Intervertebral discs are composed of the annulus fibrosis, which is the outer layer, and nucleus pulposus, which is the inner layer.[1,2] Their main functions are to provide shock absorption and to allow flexibility to the vertebral column. Intervertebral disc disease, or IVDD, is a degenerative condition in which one or more of the intervertebral discs either herniate (extrude) or bulge (protrude) into the vertebral canal.[2] This disease process is fairly common in dogs, but is more rare in the feline patient.[3] Classification of disk degeneration is described as either Hansen Type I and Hansen Type II. Hansen Type I IVDD usually occurs in chondrodystrophic breeds, and Hansen Type II more commonly occurs in non-chondrodystrophic breeds. Treatment options for IVDD are divided into two main categories – medical management and surgical correction. Medical management is considered conservative, whereas surgical correction is accepted as the treatment of choice.[4,5]

HISTORY AND PRESENTATION

The most common areas of the spinal cord that are affected by IVDD include the cervical and thoracolumbar region. As such, this will be the focus of this paper. Clinical presentation of IVDD varies greatly depending on the following factors: location, progression (acute or chronic), the dynamic force at which the disc material compresses the spinal cord, the degree of hypoxia present in the spinal cord tissue, the mechanical displacement of the spinal cord and the duration of injury.[6] Cervical intervertebral disc disease accounts for 12.9%-25.4% of overall intervertebral disc herniation cases.[1] Cervical IVDD patients are typically chondrodystrophic small breed dogs, with dachshunds and beagles between the ages of 6-8 years of age being the

most commonly affected.[1] Other small breed dogs that are commonly affected are Shi Tzu's, Cocker Spaniels, and Basset Hounds. However, it has been recently reported that 24-50% of Hanson type 1 cervical IVDD patients have been large breed, non-chondrodystrophic dogs, with Labrador retrievers and Rottweilers most commonly represented.[1] The most common cervical vertebrae affected by IVDD in small breed dogs is C2 to C3, and in large breed dogs is C6 to C7.[7] Hansen Type I and Type II herniations occur in this region, however Hansen type I extrusions are more common in both small and large breed dogs.[1,7] Cervical IVDD patients present with guarding of their neck, muscle fasciculations, and cervical hyperpathia.[1,8] Compared to the thoraco-lumbar spine, cervical IVDD is associated with a lower rate of neurologic deficits due to the larger vertebral canal/spinal cord ratio.[1,3] In the cervical spine, larger disc extrusions can occur without severely compressing or compromising the spinal cord.[1] Other clinical signs that can occur are a unilateral or bilateral lameness, which can be attributed to cervical nerve root compression (root signature).[1,9] Ataxia with tetraparesis or tetraplegia can develop in a small percentage of cervical IVDD patients, but it is not common.[1,9]

Thoraco-lumbar intervertebral disc disease (T3-L3) accounts for 66%-87% of overall intervertebral disc herniation cases.[1] In chondrodystrophic dogs with thoracolumbar IVDD, the most commonly affected disc spaces are T12-T13 and T13-L1.[1] In large breed dogs, the most commonly affected disc spaces are T13-L1 and L1-L2 followed by L2-L3.[1] Clinical signs of thoracolumbar IVDD include the folllowing: spinal hyperesthesia, reluctance to walk and kyphosis, paraplegia with or without pain perception, and "spinal shock" or Schiff-Sherrington postures.[9,10] As with cervical IVDD, Hansen type I and type II can occur in the thoracolumbar region. However, Hansen type I IVDD most commonly occurs in this region of

chondrodystrophic breeds.[1] Hansen type II most commonly occurs in this region of non-chondrodystrophic large breed dogs, such as the German shepherd dog.[11]

PATHOPHYSIOLOGY

Intervertebral discs are composed of an outer fibrous layer, termed the annulus fibrosis, and an inner gelatinous layer, termed the nucleus pulposus.[2] The annulus fibrosis is made from concentric fibrous laminae, and it is two times thicker ventrally and laterally. It is composed primarily of type I collagen and is made of dehydrated tissue.[1,12] The nucleus pulposus' consistency is semi-fluid in young healthy discs, and it is under constant pressure by the annulus fibrosis that surrounds it.[1,12] Since the nucleus pulposus is negatively charged and hydrophilic, it draws in water and acts as the shock absorbing component of the disc.[1]

Intervertebral disc degenerative changes occur with age, and can be classified as chondroid metaplasia and/or fibroid metaplasia. Chondroid metaplasia has a loss of glycosaminoglycans (gag layer) as well as an increase in hyaline cartilage, and decrease in water content.[1,12] This process weakens fibers of the annulus fibrosis and reduces the hydrostatic pressure of the nucleus pulposus. This leads to mineralization of the nucleus pulposus.[1,12] The end result of these degenerative changes is a loss of the hydroelastic properties of the disc and its ability to withstand pressure. This ultimately leads to the nucleus pulposus extruding into the vertebral canal via a torn annulus fibrosus.[1] Chondroid metaplasia typically occurs in chondrodystrophic breeds, has an acute onset and is the cause of Hansen type I disc extrusions.[1] This disease process can occur due to compressive, concussive, or a combination of these forces. In 75-90% of chondrodystrophic breed dogs, their nucleus pulposus is converted from a gelatinous material to a more hyaline and cartilaginous tissue by 1 year of age.[1] This degenerative process can affect the entire vertebral column and could potentially lead to

calcification in all discs.[1] It was recently reported that in Dachshunds, there is a mean of 2.3 calcified discs per dog.[1] Non-chondrodystrophic breed dogs maintain high non-collagenous protein levels into old age, so they are less prone to Hansen type I disc extrusions.

Fibrous Metaplasia occurs in dogs 7 years of age or older, and is typically associated with non-chondrodystrophic breeds. Fibrous metaplasia causes the nucleus pulposus to bulge within the weakened, but intact, annulus fibrosis. This then leads to dorsal intervertebral disc protrusion and is termed Hansen type II disc protrusion.[1,12] These disc herniations are often asymptomatic due to their chronic progressive nature, and mostly caused by compressive forces.[12] As with chondroid metaplasia, this degenerative change can also occur at any location along the vertebral column.[1] Fibrous metaplasia affects a few number of discs, and mineralization often does not occur. A total of 40-60% of dogs aged 7 years or older show biochemical evidence of nucleus pulposus degeneration and 10-30% exhibit macroscopic intervertebral disc protrusion[1]

As previously mentioned, intervertebral disk disease can be classified as Hansen type I or Hansen type II. Chondrodystrophic breeds are predisposed to Hansen type I and non-chondrodystrophic breeds are predisposed to Hansen type II, but ultimately there can be overlap between these two classification.[12] There is a type III IVDD category, but it is not recognized in the Hansen classification. This disease process is usually the result of trauma, in which case a small amount of nucleus pulposus extrudes through the annular deficit under high pressure.[12] Unlike Hansen type II IVDD, which is mostly compressive, type III is classified as concussive secondary to trauma.

DIFFERENTIAL DIAGNOSES

As with clinical signs, differential diagnoses are dependent on which part of the spinal cord is affected by IVDD. Cervical IVDD (C1-C5) differentials include the following: fibrocartilaginous embolism, hemorrhage, fracture/luxation, diskospondylitis, meningomyelitis, granulomatous meningioencephalomyelitis, neoplasia, arachnoid cyst, spinal articular cyst, cervicospondylomyelopathy, syringohydromyelia, atlantoaxial subluxation, and steroid responsive meningitis-arteritis.[13,14,15,16] Thoracolumbar IVDD (T3-L3) differentials include the following: fibrocartilaginous embolism, hemorrhage, fracture/luxation, diskospondylitis, meningomyelitis, granulomatous meningoencephalomyelitis, neoplasia, arachnoid cyst, spinal articular cyst, and degenerative myelopathy.[13,14,15,16]

DIAGNOSTIC APPROACH/CONSIDERATIONS

The diagnostic approach to IVDD begins with a thorough physical and neurologic examination as well as a minimum database, including a complete blood count, serum chemistry, and urinalysis.[16] The neurologic examination evaluates the patient's mental state, posture, gait (paresis/paralysis, ataxia, circling and lameness), postural reactions (knuckling, hopping, wheelbarrowing and hemi-walking), muscle tone and size, spinal reflexes, perineal reflex/anal tone, sensory perception (nociception),cutaneous trunci, and cranial nerve status.[8,9,16] Lesion localization along with radiographs and advanced imaging (CT, MRI) will definitively diagnose an IVDD lesion.[1,9,16]

Survey radiographs of the affected region should be taken with the patient heavily sedated or anesthetized to allow proper positioning and to decrease motion.[1,15,16,17] A significant predictor of disc herniation and a risk factor for recurrent herniation post-surgical correction is disc calcification.[1] Survey radiographic findings indicative of IVDD herniation include narrowing of the intervertebral disc space, narrowing or increased opacity of the intervertebral

foramen, narrowing of the articular facets, and presence of mineralized disk material within the vertebral canal.[1,15,16] In one study, 19 chondrodystrophic breed dogs with acute thoracolumbar disc disease extrusion, radiographs had a reported accuracy of 51% to 94.7% for the correct identification of the herniated disc space for surgical decompression.[18] Despite the high reported sensitivity of radiography for localizing the lesion in some studies, this modality cannot be used alone for diagnosing IVDD herniation because it does not provide information on lateralization of the extrusion, extent, and degree of spinal cord compression and presence of other lesions.[18]

Myelography is sometimes used along with radiographs to diagnose IVDD. This requires an injection of dye into the subarachnoid space surrounding the spinal cord either in the cervical region (cisternal myelogram) or in the lumbar region (lumbar myelogram).[3] Myelography distinguishes regions of the spinal cord that may be swollen due to a contusive injury, as well as areas of deviation in the subarachnoid space, which could indicate an extruded nucleus or protruded annulus.[1,16] There are 4 myelographic patterns described – normal, extradural, intradural/extramedullary, and intramedullary.[19] A normal myelographic pattern is often associated with fibrocartilaginous embolism and degenerative myelopathy.[19] The most common cause of an extradural myelographic pattern is intervertebral disc disease where the disc is either extruding or protruding.[19] When there is a lesion within the subarachnoid space, but not invading the parenchyma of the cord it is a termed an intradural/extramedullary pattern.[19] This pattern is mostly associated with neoplasia, primarily meningiomas and nerve sheath tumors.[19] Intradural/extramedullary lesions may produce enough spinal cord swelling that contrast is excluded from the region, and this is termed intramedullary.[19] This pattern is usually associated with spinal cord edema, expansile parenchymal masses, or intraparenchymal hemorrhage.[19] In most cases, before the dye is injected for a myelogram, cerebrospinal fluid (CSF) is sampled

caudal to the disease site due to the changes the dye will cause to future analysis.[3] Changes will occur 90 minutes after the dye is injected, and these include a false-positive Pandy score, high specific gravity, and monocytoid pleocytosis.[4] A recent report on lumbar CSF analysis in dogs with Hansen type I IVDD revealed pleocytosis in 51% of dogs, including 23% with cervical lesions and 61% with thoracolumbar lesions.[20] In acute Hansen type I IVDD, CSF is classically described as normal or mildly inflammatory with a predominance of large mononuclear cells or neutrophils.[20] Increase in protein concentration was more common in dogs with cervical (60%) than thoracolumbar (16%) IVDD extrusion, and a predominance of lymphocytes was significantly more common in dogs examined more than 7 days from the onset of signs, which may indicate an immune-mediated response to chronically herniated disc material.[20]

Computed tomography (CT) and magnetic resonance imaging (MRI) are sensitive and noninvasive advanced imaging modalities that aid in definitively diagnosing IVDD. Typically, MRI can be used alone, whereas CT can be used alone or in addition to a myelogram study. CT is superior in detecting bony abnormalities of the vertebral bodies and skull.[16] CT with contrast can be used to highlight any lesions that disrupt the vascular endothelium.[16] A recent study comparing CT to myelography revealed similar diagnostic sensitivities (83.6% and 81.8%, respectively) for localizing the site of disc herniation; however, CT was more sensitive than myelography (80% vs 38%) in detecting chronic lesions because of disc mineralization, and myelography was more sensitive in dogs weighing less than 5 kg (100% vs 50%).[21] CT had the ability to guide surgical decision making due to its accuracy.[1] A study reported that the agreement of CT and helical CT with surgical findings were 100% and 94.7%.[18] One study performed surgical decompression on chondrodystrophic breed dogs with acute signs of IVDD based on their neurologic exam and CT findings.[13] Clinical follow-up exams were performed 1-

14 days post surgically and revealed that CT detected a lesion consistent with clinical findings in 63 of the 69 cases (91%).[13] All 63 dogs with Hansen type I IVDD lesions were identified on CT alone.[13] It was determined that CT imaging can be used as a single imaging modality in chondrodystrophic dogs presenting with acute paresis.[13]

MRI is the imaging modality of choice for the brain, spinal cord, and peripheral nerves due to its ability to detect very subtle differences in soft tissues, and can be used to guide surgical decisions due to its accuracy.[16] By identifying varying signal intensities, MRI sequences such as the gradient-echo sequence can enable the differentiation between disc material and hemorrhage.[1] In two studies performed in 2005 and 2008, there was 100% agreement between MRI and surgical findings reported with regard to the affected TL IVDD and lesion lateralization.[1]

TREATMENT AND MANAGEMENT OPTIONS

Cervical IVDD conservative treatment consists of exercise restriction, confinement to a small cage for 2-6 weeks, physical therapy, analgesics, muscle relaxants, and anti-inflammatory drugs.[1,8] Additionally, it is recommended that a chest harness is used rather than a neck collar. A recent study stated that of 88 dogs with cervical lesions, 48.9% of dogs were successfully managed conservatively, 33% had recurrence, and 18.1% failed conservative treatment.[1] Of the dogs included in that study, 97% were ambulatory at the time of presentation.[1] In a separate study, less severe neurologic status and administration of a non-steroidal anti-inflammatory drug (NSAID) were associated with a successful outcome, but steroid use and the duration of cage rest were not.[1] It was reported that most clinicians will take the medical management approach to cervical IVDD if the patient is only mildly affected, and if it has an acute history.[1]

Surgical decompression and removal of extruded disc material is indicated in cervical IVDD patients that display severe neck pain, neurologic deficits, recurrence or worsening of clinical signs after medical management, and/or dogs that have a chronic history at time of presentation.[1,16] The most common decompression surgery used for cervical IVDD patients is the ventral slot. A study of 111 ambulatory dogs with cervical IVDD herniation revealed that ventral decompression was significantly superior to cervical fenestration with regard to improved neurologic status (87% vs 73%) and speed of recovery.[1] If deemed appropriate, prophylactic fenestration of adjacent discs can be performed through the ventral slot approach.

Thoracolumbar IVDD conservative treatment consists of strict cage rest, anti-inflammatory drugs, muscle relaxants, analgesics, and physical therapy.[1,16] A retrospective study reported success rates of 50% with conservative treatment of thoracolumbar IVDD, with recurrence rates reaching 30%.[1] A study of 78 dogs suspected to have thoracolumbar IVDD extrusion revealed dogs treated with an NSAID or MPSS had lower recurrence rates than dogs treated with other corticosteroids.[1,22] As with corticosteroid use in cervical IVDD patients, administration has been associated with lower quality of life and decreased odds of successful outcome in conservatively managed patients.[1] There is experimental evidence that glucocorticoids may lead to excitotoxic neuronal death, worsening of oxidative injury, and lactate accumulation within the spinal cord parenchyma. Gastrointestinal ulceration, pneumonia, and increased duration of hospitalization have been seen with glucocorticoid administration in both veterinary and human patients with neurologic disease and may play a role in outcome measures.[22] Dogs that were administered NSAIDs were more likely to have a higher quality of life scores when compared with those not administered these medications.[22] This is likely due to the analgesic effects of NSAIDs and blunted tissue inflammatory response.[22] Improved analgesia

and reduced tissue inflammation have been speculated to improve mobilization and lessen disuse muscle atrophy in veterinary patients with neurologic and orthopedic disease.[22]

Decompression and removal of extruded disc material is the treatment of choice for patients with thoracolumbar IVDD. This can be achieved by a dorsal laminectomy or hemilaminectomy.[1,15,16] Hemilaminectomy is the most common approach to the thoracolumbar spinal cord because it is associated with a more satisfactory decompression by removal of disc material, significantly higher rate of postoperative neurologic improvement, and less postoperative biomechanical instability compared with dorsal laminectomy.[1,16] The hemilaminectomy provides direct access to the lateral and ventral aspects of the vertebral canal, which facilitates removal of the extruded disc material for complete spinal cord decompression, and it provides access to the disc space allowing fenestration to be performed.[1,15]

EXPECTED OUTCOME AND PROGNOSIS

Prognosis for patients affected with cervical IVDD with neck pain alone, mild neurologic deficits, and retained ambulatory status is good.[1,3,15,18] Patients that were ambulatory prior to surgical decompression typically remained ambulatory post-operatively.[1] Prognosis for non-ambulatory chondrodystrophic or small breed patients with thoracolumbar IVDD that retained deep pain prior to decompressive surgery varied between 86% and 96%.[1] Prognosis for non-chondrodystrophic large-breed dogs with thoracolumbar IVDD that had decompression surgery performed is 88% to 85%.[1]

CONCLUSION

In conclusion, intervertebral discs are present throughout the entire spine, with the exception of C1-C2 (atlanto-axial joint), and the fused sacrum. Intervertebral discs undergo a

degenerative change that ultimately leads to intervertebral disc disease. Intervertebral disc degeneration is classified as either Hansen Type I, which usually occurs in chondrodystrophic breeds, or Hansen Type II, which usually occurs in non-chondrodystrophic breeds, and there can be overlap between these classifications. The cervical and thoracolumbar regions are the most commonly affected by IVDD. A definitive diagnosis can be achieved through radiographs in combination with advanced imaging modalities, such as CT with or without a myelogram study and MRI. Both cervical and thoracolumbar IVDD can be conservatively managed or surgically treated, and both carry good outcomes as long as neurologic deficits are not too severe.

REFERENCES

1. Brisson, B.A. (2010). Intervertebral Disc Disease in Dogs. *Veterinary Clinics of North America: Small Animal Practice*. 2010; 40:829-858.
2. Evans, H.E. (1993). Miller's anatomy of the dog**.** 3rd edition. WB Saunders, Philadelphia; 1993
3. Pritchard, William, Dr. "Disc Disease | UC Davis School of Veterinary Medicine - Veterinary Medical Teaching Hospital." *Disc Disease | UC Davis School of Veterinary Medicine - Veterinary Medical Teaching Hospital*. N.p., n.d. Web. 7 Mar. 2017.
4. Hansen, H.J. (1951). A Pathologic-Anatomical Interpretation of Disc Degeneration in Dogs**.** *Acta Orthop Scand*. 1951; 20: 280–293
5. Hansen, H.J. (1952). A Pathologic-Anatomical Study on Disc degeneration in Dog. *Acta Orthop Scand Suppl*. 1952; 11: 1–117
6. University of Pennsylvania School of Veterinary Medicine website. Intervertebral Disk Disease. Available at: http://cal.vet.upenn.edu/projects/saortho/chapter_62/62mast.htm. Accessed March 7, 2017.
7. Cauzinelle, L. (2000). Fibrocartilaginous Embolism in Dogs. *Veterinary Clinics of North America: Small Animal Practice*. 2000;30: 155-167.
8. Richard W, Nelson and C.G. Couto. Disorders of the Spinal Cord. In: Small Animal Internal Medicine. 4th ed. St. Louis: Elsevier, 2009; 1066-1073.
9. Richard W, Nelson and C.G. Couto. Lesion Localization and the Neurologic Exam. In: Small Animal Internal Medicine. 4th ed. St. Louis: Elsevier, 2009; 989-1004.
10. Dewey, C. W. (2016). Kyphosis and Kyphoscoliosis Associated with Congenital Malformations of the Thoracic Vertebral Bodies in Dogs. *Veterinary Clinics of North America: Small Animal Practice*. 2016; 46:295-306.

11. Cudia, S.P. and Duval, J.M. Thoracolumbar Intervertebral Disc Disease in Large, Nonchondrodystrophic dogs: A Retrospective Study. *Journal of the American Animal Hospital Association*. 1997; 33: 456-460
12. Coates, J. R. (2000). Intervertebral Disk Disease. *Veterinary Clinics of North America: Small Animal Practice*. 2000; 30:77-110.
13. Bibevski J.D., Daye R.M., Henrickson, T.D., et al. A Prospective Evaluation of CT in Acutely Paraparetic Chondrodystrophic Dogs. Journal of the American Animal Hospital Association. 2013; 49:363-369.
14. Cook Jr, J. (1998). Fibrocartilaginous Embolism. *Veterinary Clinics of North America: Small Animal Practice*. 1998; 18:581-592.
15. Jeffery, N.D., Levine J.M., Olby, N.j., and Stein, V.M. (2013). Intervertebral Disk Degeneration in Dogs: Consequences, Diagnosis, Treatment and Future Directions. *Journal of Veterinary Internal Medicine*. 2013; 27: 1318-1333.
16. Richard W, Nelson and C. G. Couto. Diagnostic Tests for the Neuromuscular System. In: Small Animal Internal Medicine. 4th ed. St. Louis: Elsevier, 2009; 1012-1013.
17. Da Costa, R.C. (2010). Cervical Spondylomyelopathy (Wobbler Syndrome) in Dogs. *Veterinary Clinics of North America; Small Animal Practice*. 2010; 40:881-913.
18. Hecht, S., Thomas, W.B., Marioni-Henry, K. et al. Myelography vs. Computed Tomography in the Evaluation of Acute Thoracolumbar Intervertebral Disk Extrusion in Chondrodystrophic Dogs. *Veterinary Radiology and Ultrasound*. 2009; 50:353-359
19. Dewey, C.W., Da Costa, R.C., Myelography. In: Practical Guide to Canine and Feline Neurology. 3rd ed. 2015; 301-321
20. Windsor, R.C., Vernau, K.M., Sturges, B.K., et al. Lumbar Cerebrospinal Fluid in Dogs with Type I Intervertebral Disc Herniation. *Journal of Veterinary Internal Medicine.* 2008; 22: 954-960.
21. Israel, S.K., Levine, J.M., Kerwin, S.C. et al. The Relative Sensitivity of CT and Myelography for Identification of Thoracolumbar Intervertebral Disk Herniations in Dogs. *Veterinary Radiology and Ultrasound*. 2009; 50: 247-252.
22. Mann, F. A., Wagner-Mannm C.C., Dunphy, E.D. et al. Recurrence Rate of Presumed Thoracolumbar Intervertebral Disc Disease in Ambulatory Dogs with Spinal Hyperpathia Treated with Anti-inflammatory Drugs; 78 cases (1997-2000). *Journal of Veterinary Emergency and Critical Care*. 2007; 17: 53-60.
23. Cherrone, K.L., Dewey, C.W., Coates, J.R. et al. A Retrospective Comparison of Cervical Intervertebral Disc Disease in Nonchodrodystrophic Large Dogs vs Small Dogs. *Journal of the American Animal Hospital Association*. 2004; 40: 316-320.