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Surgical Conditions of the Spine Mini Series

Session One: Disc Disease

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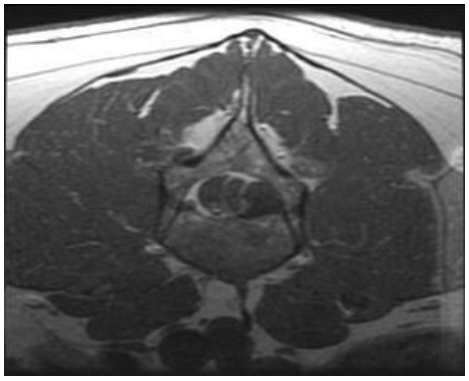

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INTERVERTEBRAL DISC DISEASE

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THE INTERVERTEBRAL DISC

The intervertebral discs are interposed in every intervertebral space (except between C1 and C2), uniting the bodies of the adjacent vertebrae forming amphiarthrodial joints. The thickness of the discs is greatest in the cervical and lumbar regions, the thickest ones being between the last few cervical vertebrae; the widest cervical intervertebral spaces are C4-5 and C5-6; the narrowest is C2-3.

Each intervertebral disc consists of an outer laminated fibrous ring (annulus fibrosus) and a central, amorphous, gelatinous center (nucleus pulposus).

The nucleus pulposus is highly hydrated gelatinous mesodermal remnant of the notochord. Its consistency is semi-fluid, and so bulges into the annulus when it is put under pressure by any movements of the vertebral bodies. Chondrodystrophic breeds show progressive collagenation and calcification in the nucleus pulposus and inner annulus fibrosus at an early age.
The annulus consists of bands of parallel fibers that run obliquely from one vertebral body to the next. They provide a means for the transmission of stresses and strains that are required by all lateral and upward movements. These bands of fibers cross each other in a lattice-like pattern and are more than eight layers thick ventrally.
Near the nucleus pulposus, the annulus fibrosus loses its distinctive structure and form and becomes more cartilaginous and less fibrous. The fibrous ring is one and a half to three times thicker ventrally than dorsally.

The cranial and caudal borders of the intervertebral disc are formed by hyaline cartilaginous end-plates, which cover the epiphyses of the vertebral bodies and are attached to the inner annular lamellae; the outer lamellae are a continuation of the Sharpey fibers of the vertebral epiphyses.

Intervertebral disc disease (IVDD) is often divided into two distinct categories, referred to as Hansen type I and Hansen type II. Intervertebral disc degeneration or disease leads to extrusion or protrusion (both termed herniation) of disc material into the spinal canal resulting in clinical signs resulting from neural tissue compression and or concussion.

HANSEN TYPE I IVDD

Hansen type I IVDD is seen in chondrodystrophic breeds of dogs which have chondroid metaplasia of the nucleus pulposus beginning early in life (usually first 2 years of life - can begin as early as 2 month of age). This metaplasia is characterised by a loss of glycosaminoglycans, an increase in collagen content, and a decrease in water content, resulting in loss of the hydroelastic properties of the disc and its ability to withstand pressure.

Chondroid metaplasia occurs along the entire vertebral column. The abnormal forces generated by the degenerate and mineralised nucleus pulposus cause tears to develop within the annulus fibrosus through which the disc extrude. Intervertebral disc extrusion is typically associated with chondroid degeneration and involves the herniation of nuclear material through all layers of the ruptured annulus fibrosus into the vertebral canal. The extrusion causes dorsal, dorsolateral or circumferential compression of the spinal cord.

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Acute disc extrusion is characterized by extradural hemorrhage and soft disc material.

Thoracolumbar area accounts for most cases causing upper motor neuron lesions in the pelvic limbs.

HANSEN TYPE II IVDD

In non-chondrodystrophoid dogs, degeneration of the disc occurs later in life, involving a different metaplastic process. Fibroid metaplasia is a slow and insidious increase of glycosaminoglycans concentrations and decrease of collagen when compared to chondrodystrophoid dogs. The nucleus maintains more of its gelatinous nature with less dehydration and mineralization. The changes within the discs occur at a much later age than that seen in chondrodystrophoid dogs resulting in partial rupture of annular bands and a domelike bulging of the dorsal annulus termed Hansen type II disc protrusion.

Hansen type II IVDD is annular protrusion caused by shifting of central nuclear material and is commonly associated with fibroid disc degeneration. The annulus fibrosus slowly protrudes into the vertebral canal to cause spinal cord compression.
The chronic compression can lead to focal ischaemic and other microvascular derangements of the spinal cord.

Type II IVDD usually occurs at the mobile points of the spinal column and is more common in older, non-chondrodystrophic breeds of dog. It is not uncommon to identify multiple affected disc spaces. Chronic spinal instability may be an underlying predisposition to type II IVDD.

Although the descriptions for type I and II disc degenerations are classically assigned to chondrodystrophoid and non-chondrodystrophoid dogs respectively, nuclear extrusions can occur in non-chondrodystrophoid dogs and vice versa.

CERVICAL DISC HERNIATION

Cervical spinal pain is the most common clinical sign of acute onset cervical IVDD. Low head and neck carriage, neck guarding, stilted gait, radicular pain, and spasms of the cervical spinal muscles are common clinical manifestations of cervical spinal pain.
Due to the greater ratio of the vertebral canal diameter to spinal cord diameter in the cervical spine, Hansen type I cervical disk disease commonly presents with pain only even if a large amount of nucleus is extruded. Pressure from disc material on the nerve root can cause nerve root ischemia and severe pain. Pain is often intermittent and manifests as thoracic limb lameness (root signature or radicular pain). Radicular pain also can be elicited by manipulation of the affected limb.
Gait evaluation is assessed as normal, general proprioceptive ataxia (more severe in the pelvic limbs), and hemi- or tetraparesis/plegia. Neurologic dysfunction can be asymmetric based on lateralization of the extruded disc. In general, thoracic limb spinal reflexes are normal to hyperreflexic with a C1 to C5 spinal cord lesion and normal to hyporeflexic with a C6 to T2 lesion. However, spinal reflex evaluations may not be reliable for neuroanatomic localization within the cervical spinal cord region following acute disc disease. A decreased withdrawal reflex does not always indicate a lesion from C6 to T2 and can also occur with lesions at the C1 to C5 spinal cord level.
Nonambulatory tetraplegia is an infrequent clinical manifestation of cervical IVDD. A study in 32 dogs summarized that fewer than one-third of dogs that are nonambulatory secondary to cervical disc herniation experience complete loss of voluntary motor function; sensory deficits are encountered even less frequently. Horner's syndrome can be a clinical manifestation caused by disruption of the tectotegmental spinal tract with severe cervical spinal cord injury. Loss of nociception is rare in dogs with acute cervical IVDD but is associated with severe spinal cord damage, myelomalacia, cardiac arrhythmia and respiratory dysfunction.

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Survey radiographs should be taken to identify degenerative changes typical of a disc herniation and to rule out other causes of the signs. Changes indicative of a disc herniation include narrowing of the intervertebral disc space, narrowing of the intervertebral foramen and the presence of mineralized material within the vertebral canal and disc space. A definitive diagnosis cannot be reached with survey radiographs alone with adequate accuracy for surgery to be undertaken and either computed tomography, myelography or MRI are used to identify the site of spinal cord compression.

Conservative therapy can be attempted while the dog is monitored for reduction of pain and improvement in neurologic status.

In dogs with cervical IVDD, conservative therapy alone is often ineffective. A possible reason for lack of response to strict cage rest is that total immobility of the cervical spine is difficult to maintain. Additionally, dogs with cervical spinal pain as the primary presenting sign may have a significant amount of extruded disc present within the spinal canal.

There is limited information available on the success of medical therapy for cervical IVDD in dogs but the recurrence rate has been reported to be as high as 36% in dogs treated medically for cervical IVDD. More recently Levine et al reported a success rate of nearly 50% with 33% having recurrence of clinical signs and 18% having therapeutic failure. This study also suggested that duration of cage rest was not significantly associated with success or 'quality of life' scores. The authors go on to state that an initial period of strict cage confinement may be beneficial but that prolonged (> 2 weeks) strict rest at the expense of physical rehabilitation may not have any benefit.
Surgical management is recommended when pain is refractory to standard pain management for longer than 1 to 2 weeks or if there is progression of neurologic deficits. Indications for surgical treatment of cervical intervertebral disc extrusion include pain refractory to medical management and / or severe and progressive neurologic deficits. In cervical IVDD, decompressive procedures via a ventral, lateral or dorsal approaches are the techniques of choice for removal of extruded disc. The selection of the decompressive procedure is usually determined by the location of the disc material in relation to the cord. The ventral slot technique is commonly performed for disc displaced ventral to the spinal cord. Using the identifiable landmarks of the ventral prominence of C1 and the transverse processes of C6 to identify the disc interspace of interest, a slot is cut into the ventral aspect of the cervical vertebrae using a high speed surgical drill.
Advantages of the ventral decompressive technique include minimal muscle dissection and exposure for prophylactic fenestration of adjacent cervical discs.
Disadvantages may include excessive venous sinus haemorrhage, lack of spinal cord decompression and inadequate exposure for lateral or intraforaminal disc extrusion. If the width of the slot is >30% of the size of the vertebral body, dogs may suffer from instability or subluxation at the surgery site.
Dorsal laminectomy involves dissection of the epaxial musculature and removal of the dorsal spinous processes and the laminae. Dorsal procedures provide spinal cord decompression and access for laterally extruded disc material, but access is limited to extruded disc located beneath the spinal cord.

A lateral approach has been described for lateral or intraforaminal disc extrusions at the C4-5 and C5-6 interspaces. Excessive haemorrhage from muscle dissection and damage to the internal vertebral venous plexus or vertebral artery and incomplete removal of disc material can be complications of dorsal decompression. Risk for spinal instability is considered less when compared to the ventral slot technique.
Prediction of recovery outcomes in dogs with cervical IVDD based on site of herniation, ambulatory status and breed size is controversial.

In a study of 190 dogs with cervical intervertebral disc disease treated surgically, outcomes were no different for the ambulatory versus nonambulatory dogs with intact nociception. After surgery, 99% of those dogs had resolution of cervical spinal hyperesthesia and were able to ambulate unassisted. Other previous studies have reported that dogs with caudal cervical disc extrusions respond less favourably and are more severely affected than dogs with cranial

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cervical disc extrusions. A study that also included dogs with Hansen type II IVDD associated with caudal cervical spondylomyelopathy reported only a 66% success rate following surgery in large breed dogs. Recently, a study in 32 dogs with nonambulatory tetraparesis reported that 62% had a complete recovery. Small dogs were 6 times more likely to recover than large breed dogs and dogs that regained ability to walk within 96 hours after surgery were 7 times more likely to recover than those not ambulating within 96 hours. If given more time, however, recovery of ambulatory function can occur but residual deficits are likely. In contrast to the same previous studies, site of disc herniation was not a significant predictor of complete recovery. Dissimilar to thoracolumbar IVDD, the severity of neurologic deficit was not a robust predictor of outcome.
Recurrence of clinical signs after surgery in dogs with cervical IVDD has been reported to range from 10% to 33%. The most common clinical sign during recurrence was cervical spinal hyperesthesia. A second disc extrusion at a site distinct from the initial lesion was the most common reason for recurrence.

THORACO-LUMBAR DISC HERNIATION

Clinical signs of thoracolumbar IVDD vary from spinal hyperesthesia only to paraplegia with or without nociception. Dogs with back pain only are reluctant to walk and may show kyphosis. Dogs with back pain alone or minimal neurologic deficits actually can have imaging evidence of substantial spinal cord compression. Lateralization of the extruded disc and spinal cord compression often will cause asymmetry of neurologic deficits.
Neuroanatomic localization of thoracolumbar spinal lesions is determined by normal to hyperreflexic (T3 to L3) or hyporeflexic (L4 to S3) spinal reflexes and by site of paraspinal hyperesthesia.

Dogs presented with peracute or acute thoracolumbar disc extrusions often manifest initial clinical signs of 'spinal shock' and / or Schiff-Sherrington postures. Spinal shock is usually manifested as flaccidity distal to the lesion. Likewise, the spinal reflexes are depressed to absent and the bladder may be flaccid with urine retention and sphincter hypotonia. The cause of spinal shock is unclear. A transient decrease in limb tone may be due to loss of descending supraspinal input on the alpha-motor neurons and interneurons, along with an increase in segmental inhibition. Spinal shock is important to recognize to prevent erroneous lesion localization. These transient phenomena indicate acute and severe spinal cord injury but do not determine prognosis.
A classification scheme adapted from Griffiths describing hyperesthesia, sensory and motor dysfunction, and loss of micturition has been used to as a functional grading system to provide treatment guidelines for thoracolumbar IVDD:

- Grade 0 = normal
- Grade 1 = Paraspinal hyperaesthesia only
- Grade 2 = General proprioceptive ataxia and/or ambulatory paraparesis
- Grade 3 = Non-ambulatory paraparesis
- Grade 4 = Paraplegia (intact nociception)
- Grade 5 = Paraplegia with loss of nociception

Loss of nociception is the most important prognostic indicator for acute spinal cord injury. Nociception by definition is perception of a noxious stimulus. Classically, 'superficial' pain response is elicited upon pinching a skin fold and 'deep' pain response is elicited upon use of a noxious stimulus, most commonly a bone of a digit. Distinguishing between superficial and deep 'pain' perception may not be reliable because of individual animal differences in perception of a 'painful' stimulus. Moreover, differences between these two nociceptive pathways are poorly defined.

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Ascending and descending hemorrhagic myelomalacia should be suspected in dogs with thoracolumbar IVDD that have an ascending loss of the cutaneous trunci reflex. Other neurologic signs of myelomalacia include loss of nociception caudal to the lesion, ascending and descending flaccidity and areflexia, tetraplegia, hyperthermia and respiratory distress.
Death results from asphyxia associated with intercostal and diaphragmatic muscle paralysis. Clinical signs of ascending and descending myelomalacia may manifest in hours to several days from onset of paraplegia.

Radiographic characteristics of acute intervertebral disc herniations include : uniform narrowing or asymmetrical narrowing (wedging) of the disc space, mineralised disc material either within the vertebral canal or displaced dorsally within the disc space, change in shape and opacity of the intervertebral foramen, narrowing of the articular process joint space and very rarely vacuum phenomenon. Patient should be anaesthetised to prevent movement and enable proper positioning. Higher milliamperage and moderate kilovolt peak should be used.

The absence of these signs does not rule-out the presence of intervertebral disc herniation. There is little to be gained from plain radiography as long as the patient is going to be managed medically. There is little value in knowing that the disc hernation is at T13/L1 if that information does not alter the treatment plan.

Survey radiographs have been reported to accurately identify the site of disc extrusion in around 70% of cases with surgically confirmed IVDD. Mineralisation of the nucleus pulposus, especially in the chondrodystrophoid dog, can be widespread without the presence of any clinical signs. Most calcified discs seen on radiographs are not responsible for the development of pain or paresis. Only when the calcified material is located within the vertebral canal or it is in the intervertebral disc space but is incomplete or fragmented should it be incriminated as the possible cause of clinical signs.

Contrast radiography (myelography) is necessary to confirm the presence of disc herniation and localise the site of spinal cord compression. It is also indicated when there is absence of a spinal lesion on survey radiographs, a lesion on survey radiographs is not compatible with the anatomic diagnosis, or survey radiographs indicate multiple lesions. Iohexol (Omnipaque) 180, 240 or 300 mg of iodine per ml and iopamidol are the preferred non-ionic radiographic contrast media. Myelographic contrast injection at the caudal lumbar region is preferred over the cerebellomedullary cistern for demonstrating thoraco-lumbar disc extrusion as it allows injection of contrast medium with some force, thus outlining the lesion. The recommended doses are between 0,25 and 0,5 ml/kg. Injection of the contrast medium should be slow and resistance to flow must be extremely weak and preferably absent altogether.

Lateral and ventrodorsal radiographs should be taken. If there are doubts about the side of the vertebral canal the disc material lies, oblique views should be taken. If the contrast flow is blocked by a lesion at a specific site and does not allow documentation of the entire spine or a precise definition of the lesion, it is recommended to repeat the myelogram using the other injection site.

An abnormal myelogram is characterised by changes in contrast columns and in spinal cord width and opacity. Abnormal myelographic patterns associated with disc extrusion include extradural and intramedullary swelling and opacification.
An extradural spinal compression centered over or around the disc space is characteristic.

In case the side cannot be determined, other imaging modalities such as CT-myelogram or MRI are indicated if available and time allows it. Alternatively, the surgeon can relies on the neurological signs findings such as difference in the cutaneous trunci reflex and/or degree of paresis between sides (agreement between side of the extrusion and clinical findings in 2/3 of dogs) or owner's report on which limb may have been first affected at the time of the onset.

In dogs with undetermined lateralisation, most right-handed surgeons elect to perform a left-sided hemilaminectomy for ease of surgical approach and instrument handling.

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Indications of non-surgical treatment of thoraco-lumbar IVDD include a first-time episode of spinal pain or mild paraparesis and financial constraint. The aim of non-surgical management is to prevent additional extrusion of disc material via healing of fissures in the dorsal annulus fibrosus. Strict cage rest is essential for a minimum of four weeks. Lead walk is only permitted for toilet purpose. Rest can be combined with pain relief using anti-inflammatory drugs, opioids and muscle relaxants. Dogs should be monitored very closely for deterioration of neurological status. If pain persists or neurological status deteriorates then surgical treatment is strongly indicated. It is impossible to predict which cases will improve and which will remain static or progress.

Success rates depends on the severity of the neurological signs with pain only or mild paresis range from 80 to 100% and non-ambulatory dogs range from 40 to 50%. It is ineffective for the vast majority of dogs with grade V paraplegia. One third of dogs which recover from the initial disc extrusion will experience relapse as the ruptured annulus fibrosus may fail to heal completely and further extrusion of nuclear material can subsequently occur at any time in the future (average interval from initial treatment to recurrence of 1.7 years).

Great deal of controversy exists regarding the use of corticosteroids in IVDD with most recommendation based on clinical experience rather that controlled studies. Used at high dosage, glucocorticoids appear to counter the secondary injury phenomenon of self-perpetuating necrosis by inhibition of lipid peroxidation by free radicals, normalization of extracellular calcium, preservation of neurofilament proteins... Studies on human and experimental animals with spinal cord injury have shown a significant improvement in the outcome by using methylprednisolone sodium succinate (Solumedrone) at 30 mg/kg IV followed by 5.4 mg/kg/hr CRI IV for 23 hours. Furthermore, patients treated with this protocol after 8 hours showed a decreased recovery of motor function compared to those treated with placebo. These positive results should however be interpreted with caution as methylprednisolone did not restore the ability to walk in paraplegic patients and the improvement in motor function observed in human may not be relevant to dogs.

Indications for surgical management of thoracolumbar IVDD include spinal pain or paresis refractory to medical therapy, recurrence or progression of neurologic deficits, paraplegia with intact nociception, and paraplegia with loss of nociception for less than 24-48 hours. Prolonged loss of nociception (> 48 hours) carries a poorer prognosis and owners should be made aware of this prior to surgery. Decompressive procedures for thoracolumbar IVDD include dorsal laminectomy, hemilaminectomy, and pediculectomy.
Hemilaminectomy significantly improves retrieval of extruded disc material with minimal spinal cord manipulation.

Pediculectomy is the least invasive and least destabilizing technique and can be used as an adjunct technique in cases requiring a bilateral approach to the vertebral canal; an accurate 3 dimensional depiction of the disc location is necessary prior to pursuing this surgical technique due to the small surgical field that it provides. Biomechanical studies have shown that unilateral facetectomy and fenestration do not significantly destabilize the spine during lateral bending.

Post-operatively, patient should be kept on thick mattress and turn every 4 to 6 hours to prevent decubital ulcers. Opioids and NSAIDS can be used 48 to 72 hours after surgery if pain persists.
Bladder management should be considered immediately after surgery. This includes the use of intermittent bladder catheterisation (two to three times daily) or expression, to prevent over-distension of the bladder and the development of a flaccid bladder, and medical management of excessive sphincter tone (if present). Regular manual bladder expresssion should be performed until the patient has regained the ability to urinate voluntarily. Intermittent or indwelling catheterization may be necessary in patients that are difficult to express manually. Therapeutic agents such as phenoxybenzamine (sympathetic alpha antagonist - 0.5 mg/kg BID) and diazepam (0.2 to 0.5 mg/kg BID) can be used to reduce urethral sphincter

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hypertonicity. Drug to enhance contractibility of the detrusor muscle such as betanechol (2.5 to 25 mg TID) should only be considered in the absence of urethral sphincter hypertonicity.
Management of long-term recumbency requires further physical therapy (e.g., massage, passive and active exercise), hydrotherapy and the use of walking aids such as hoist or slings.
Following the initial four weeks confinement period, dogs should be gradually returned to normal activity while avoiding running, jumping and climbing stairs.

Differences in recovery rates of nonambulatory dogs with thoracolumbar IVDD vary with the severity of neurologic dysfunction (neurologic grade), time interval from initial clinical signs to surgery, and speed of onset of signs.

Overall success rates after decompressive surgery range from 58.8% to 95%. However, the success of a surgical approach may depend on what criteria are used to define it, how long after the surgery the patient is assessed, as well as the outcome of which the owners are willing to accept. In other words, surgical success may consist of improvement in the patient's neurological grade but still may not translate to complete normality. In general, dogs with more severe motor dysfunction tend to have longer recovery times for regaining ambulatory function. Reported mean time from surgery to a pain-free ambulatory status varies from 10 days for dogs presenting with spinal pain only to 52 days for paraplegic dogs. Other long-term studies reported the recovery times as 2 to 14 days for dogs that were either ambulatory or nonambulatory paraparetic and up to 4 weeks for paraplegic dogs. Nociception is considered the most important prognostic indicator for functional recovery. In general, the majority of dogs with intact nociception, whether paraplegic or paraparetic, have an excellent prognosis, particularly if treated surgically. Without surgery or with delayed surgery, dogs with absent nociception have an extremely guarded prognosis although duration of absence of nociception prior to surgery as a prognostic indicator is controversial.

In general, dogs with loss of nociception for longer than 24 to 48 hours prior to surgery have a poorer prognosis for return of function. Recovery rates for dogs with thoracolumbar IVDD and absent nociception range from 0 to 76%. Recovery of nociception within 2 weeks after surgery has been associated with a successful outcome to ambulatory status. In a study of 87 dogs with loss of nociception, 58% regained nociception and the ability to walk.

In summary, dogs with absent nociception that have surgery within 12 hours have a better chance of more rapid and complete recovery than those with delay of surgery. Prognosis is considered poor if nociception does not return within 2-4 weeks after surgery.

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