Non-traumatic Causes of Paresis

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The causes of acute paresis without trauma are variable. Paraparesis/paraplegia is a bilateral motor dysfunction of the pelvic limbs. Paraparesis is a deficit of voluntary movements involving both pelvic limbs. Paraplegia is a complete loss of voluntary movements in both pelvic limbs. Diagnosis of the cause for paresis is variable and depends on history, signalment, and the physical/neurological examination. A methodical approach to the canine patient with suspected spinal disease can improve the chances of successful diagnosis and treatment. Important factors to consider include the history in combination with the signalment of the progression of clinical signs. The complete physical exam, including the neurological exam also are essential for identification of problem. An important portion of the patient evaluation is evaluation of the animal’s gait. Much can be determined from gait evaluation. With acute paresis, the presence and location of hyperesthesia also has particular importance. This information then allows the clinician to consider appropriate diagnostic tests. Once all of the information is combined, appropriate therapeutic recommendations can be made.

**Signalment**

Breed: Certain breeds tend to have a higher incidence of a particular disease. An example in the thoracolumbar spine is Degenerative Myelopathy in the Corgi. It is also important to avoid narrowing differentials, for instance, an ataxia German Shepherd Dog does not automatically have degenerative myelopathy.

Age: Age is an important consideration in dogs with neck pain or ataxia. In a young dog, (less than 2 years of age), intervertebral disk disease is very unlikely but an inflammatory disease or congenital anomaly would be much more likely. In older dogs disk disease and neoplasia are more likely.

History and clinical signs: The history may provide the clinical course of disease and may help determine appropriate differentials. The client’s complaint is important for formulating differentials. Are the clinical signs progressive in nature or static? Did they develop suddenly or has their development been insidious (degenerative myelopathy)? Does the animal seem to have pain associated with the condition (IVDD, Neoplasia)? Are the signs of dysfunction symmetrical (asymmetry suggestive of FCE)?

Response to corticosteroid therapy can help determine the potential disease process present. A dog with a compressive myelopathy (IVDD, Neoplasia) may have a reduced degree of ataxia with steroid administration. A dog with an inflammatory condition (meningitis) may have a marked reduction in pain after receiving steroids. In general, spinal cord compression causes demyelination and axonal swelling that then progress to vasogenic edema of white matter, which further exacerbates compression. Steroids reduce edema within the spinal cord and
therefore reduce neurological deficits. Their anti-inflammatory properties also decrease pain. For most cervical spinal diseases an anti-inflammatory dose of prednisone (1 mg/kg day for 4-7 days then every 48 hours for 7 days) is recommended.

*Diagnosis*
Radiographs of the thoracolumbar spine may not provide the definitive diagnosis in most cases, however they can help to rule out other conditions. In a dog with suspected intervertebral disk disease, they not identify the lesion, they have been shown to have only a about 50-60% accuracy for the localization of the herniated disk. However, radiographs help to rule out other conditions such as diskospondylitis. Signs of disk disease include a narrowed disk space, mineralized disk material, collapse of an articular facet, change in the shape of intervertebral foramen, a wedged disk space or the presence of mineralized disk material in the spinal canal or intervertebral foramen. In general, spondylosis deformans, is not a significant abnormality, and in the vast majority of dogs, is not of clinical significance.

There are multiple causes for paraparesis in dogs. The history, physical exam, and diagnostic testing are all used to determine the possible etiology for the underlying problem. An exhaustive review for the causes of paraparesis is beyond the scope of this lecture, however we will discuss conditions for which new information is available or that have been recently described.
INTERVERTEBRAL DISC DISEASE

Intervertebral disc disease (IVDD) is a very common and important neurological problem in dogs. It is the most common cause of paraparesis in dogs. Up to 85% of dogs with acute IVDD are considered chondrodystrophic. About 20% of dachshunds will have an episode related to IVDD at some time in their lives.

Disc degeneration is a multifactorial process. The disease differs depending on the breed of dog affected: chondrodystrophic compared to non-chondrodystrophic dogs. The causes of disc disease are not completely understood. There is probably a combination of factors at work including genetic influences, mechanical forces on the spine, and biochemical alterations associated with aging.

Thoracolumbar (TL) disc disease accounts for between 75% of the overall case population. Cervical disc disease represents about 25% of dogs with disc disease. The proportion of dachshunds affected by this disease is much higher than in other dogs. Of large breed dogs, German Shepherds have been reported to represent 33% of TL cases. Age ranges in our group of dogs were from 1.5 to 17 with a mean age of 6.8 years. Cervical IVDD cases had an average age of 8.4, while TL cases averaged 6.5 years. In the thoracolumbar spine, the most common site of disk herniation is T12-13 (26%), with T13-L1 the second most common. In the cervical spine, the C2-3 location is most common for IVDD.

Diagnostic evaluation of a dog with suspected IVDD includes a complete neurological examination and survey spinal radiographs. Other diagnostics may include cerebrospinal fluid analysis, myelography, CT or MRI.

Survey radiographs may indicate disc disease, but they are generally inaccurate for exact localization of the lesion. Radiographs are necessary to rule out other diseases such as discospondylitis, trauma, and neoplasia. Narrowing of the disc space is considered to be the most useful sign of disc disease, but is only 65% accurate. The presence of multiple radiographic signs makes localization more accurate, but a myelogram or other advanced imaging is still needed. In dogs with thoracolumbar disc disease, there was a 51-61% accuracy of determining the site of disc extrusion while this value drops to 35% for radiographs of cervical disc disease. By 6-18 months of age, chondrodystrophic dogs may begin to have signs of disc mineralization that are evident on radiographs, with a maximum number of mineralized discs present by about 2 years. Some mineralized discs may eventually resolve radiographically. The presence of spondylosis deformans on radiographs is not associated with Type I IVDD, however there may be an association with Type II IVDD. CT or MRI is generally necessary to confirm intervertebral disk herniation. CT is very helpful in many dogs. However, it has recently been shown to be less helpful for chondrodystrophic, female, older and smaller (<7 kg) dogs. CT is less expensive than MRI and is faster.

Treatment of disc disease can be divided into conservative management (medical management) and surgical decompression. Corticosteroids and rest are commonly used as a
form of medical management. About 50% of non-ambulatory dogs will be able to walk with conservative management. About 90-98% regain the ability to walk with decompressive surgery as long as they retain deep pain perception. Medical management of suspected IVDD in the thoracolumbar and cervical spine can be successful with strict confinement and appropriate pain management. The use of anti-inflammatory drugs continues to be a controversial topic.

Corticosteroids have two potential functions in dogs with IVDD. First, they act as anti-inflammatory agents, which decrease pain and edema around the site of the disc extrusion. With chronic compression, they reduce edema within the spinal cord and may reduce the amount of neurological deficits present. High doses (neurotrauma) of methylprednisolone sodium succinate (Solu-Medrol® or MPSS) were thought to have an anti-oxidant effect, and therefore decrease lipid peroxidation. In general, this drug protocol is often inappropriately used to treat IVDD in dogs. Corticosteroids are of limited benefit for severe neurological dysfunction and when possible should not be used instead of decompression. However, anti-inflammatory doses of corticosteroids are very effective for pain management. These drugs should be used for short periods (1-2 weeks) for severe pain (prednisone 0.5 mg/kg q 12-24 hrs). With chronic disc disease the dose of prednisone may be adjusted based on clinical response.

Corticosteroids have never been proven to be effective for IVDD related spinal trauma. No canine studies have demonstrated benefits from these drugs.

**Medical management of pain associated with IVDD**

Use an anti-inflammatory drug (either NSAID or steroid). I typically use prednisone (1 mg/kg/day for 5-7 days then every other day for 6 days), in combination with another form of analgesia (tramadol 2-4 mg/kg every 8-12 hours). If muscle fasciculations are present (more likely in the cervical spine), I generally use diazepam 0.5 mg/kg PO every 8 hours. Methocarbamol may also be used (in my opinion, it does not seem to be quite as effective when compared to diazepam). For dogs with chronic pain, gabapentin (Neurontin) may be used (50-100 mg per dog every 12 hours). Severe pain may require surgery.

Success rates of surgery for dogs with the presence of neurological deficits are generally very good, with 90-98% of dogs that are non-ambulatory (with deep pain perception intact) making a significant recovery after surgery. However, not all dogs recover completely. There are many variables that factor into each case, including the degree of spinal cord compression, duration of signs, the speed at which the compression occurred, the concussive forces that were initially applied to the spinal cord, and the location and extent of the compression. After surgery residual paresis or incontinence may persist. The number of dogs that present with no deep pain has not been clearly reported. With a loss of deep pain, the success rate for surgery is 50%. About 1/3 had residual urinary and or fecal incontinence, and some walk but have no deep pain perception. The period of time an animal can be without deep pain and still regain the ability to
walk is unclear. Generally, 24-48 hours is considered to be the time that has the greatest effect on prognosis. However, some animals that have surgery after this time period may go on to walk again. Myelomalacia is an autodestructive degeneration of the spinal cord. It can be focal or generalized and spread in an ascending/descending manner. The pathophysiology is incompletely understood, and the dog is at risk for at least the first week following the injury. The total percentage of dogs that are affected by this process is not clear, but it is probably less than 3-6% of dogs with acute IVDD.

Post-operative care is important for recovery and avoidance of other problems. Pain management is essential. Physiotherapy, while not proven to improve the chances of walking, is still considered important. Also, it has been shown that after spinal surgery dogs are more likely to develop UTIs, with female, non-ambulatory dogs at the highest risk.
Suggested Reading:


Meningomyelitis

Inflammatory diseases tend to be much more common in the brain of the dog. However with what seems to be growing frequency, we are identifying dogs with inflammatory diseases of the spinal cord. This inflammation is thought to be a variant of granulomatous meningoencephalitis, or meningitis of unknown etiology. This condition may be seen in small or large breed dogs. It is generally associated with a relatively rapid progression of pelvic limb weakness/ataxia. This condition may easily be mistaken for a herniated intervertebral disk. Diagnosis of this condition is done with MRI of the spine. Changes present on MRI consist of inflammation within the spinal cord. Cerebrospinal fluid analysis is also done to identify the type of inflammation. Treatment is similar to that of intracranial GME. Generally, this consists of prednisone in combination with other immunosuppressive drugs (Cytarabine, Leflunomide, Azathioprine). The prognosis for this condition is variable.
Exercise Associated Disk Herniation:

Type III disk herniation is an acute event in which the nucleus pulposus herniates with a high velocity into the spinal cord. This has been increasingly recognized with the availability of MRI. This is also called acute noncompressive nucleus pulposus extrusion, traumatic disk herniation, exercise-associated peracute thoracolumbar disc extrusion, or Hansen type III disk herniation.

A non-degenerate nucleus pulposus rapidly herniates through the annulus fibrosus. The disk is about 85% water and can diffuse through the epidural fat, dura mater, or spinal cord, therefore not resulting in spinal cord compression. The injury to the spinal cord is a contusion. In some dogs, there may be some spinal cord compression associated with a previous disk herniation at the site of injury in about 29% of cases. This is more common in older and chondrodystrophic dogs. The location of the lesion is likely to be a static-dynamic junction (T12-13, T13-L1, C2-3).

The onset of signs is acute. In many dogs, there is physical activity associated with the event. These patients may be painful at first, but this resolves. The signs progress rapidly up to 24 hours. The main diagnosis of this condition is with MRI. Radiographs can be helpful to rule out other diseases (such as neoplasia or a fracture). Therapy of this condition is supportive care. Corticosteroids have not been evaluated but are unlikely to be effective. Fluid therapy is recommended to maintain spinal cord perfusion. Pain control may be necessary. Surgery is not generally recommended as there is no compression present, or the compression is unrelated to the problem. A recent study showed no difference in outcome following surgery. Rehabilitation may be necessary. The prognosis is dependent on the severity of the initial injury. In a recent report, 2/3 of dogs with Type III IVDD recovered significant function, however there may continue to be fecal or urinary incontinence. In a more recent report, the prognosis was considered good for dogs that retain pain perception.

Suggested reading:


Aortic Thromboembolism (Ischemic Neuromyopathy)

An aortic thromboembolism (AT) causes an ischemic neuromyopathy. Typically, this is a disease that is easily recognized in cats. However, this condition will occur in dogs as well. Aortic thromboembolism in dogs may be misdiagnosed as a primary spinal cord disease. The condition may be acute or chronic in presentation with the most common site of thrombus formation being the distal portion of the aorta and external iliac arteries. More widespread formation of a thrombus may be found in the distal aorta, internal and external iliac arteries, and the femoral arteries. Partial flow past the thrombus is common. The median age at presentation is 10 years, and it tends to be more common in larger dogs. The signs are lower motor neuron and may be easily confused with a primary spinal cord disease. Client complaints include gait abnormalities (82%), exercise intolerance, and non-specific pain. Acute cases may have unilateral limb involvement, but chronically affected dogs have bilateral paresis. The median duration of clinical signs is about 8 weeks in all dogs and 11 weeks in the chronically affected dog. Most dogs will be ambulatory at the time of presentation. Physical examination findings that are suggestive of this condition include reduced pulses in the femoral arteries, cool pelvic limbs, and cyanosis of the nails. About ½ of the dogs with this condition have a predisposing condition, including hyperadrenocorticism, protein-losing glomerulonephropathy, or neoplasia. Generally, creatinine phosphokinase (CPK) will be elevated with this condition. Diagnosis is generally done by abdominal ultrasound. However, in some cases the thrombosis is evident on CT or MRI. The prognosis for this condition is generally very guarded, regardless of the underlying etiology. The median survival time with the chronic form of this disease is 30 days. The median survival time for the acute form of the disease is 1.5 days.

Treatment of this condition has been relatively unsuccessful. There are limited cases reporting of the use of tPA, surgical removal of the thrombus, antiplatelet therapy (Clopidogrel, aspirin), and heparin. These treatments have been largely unsuccessful. Oral warfarin therapy appears to offer a significant improvement in survival times in dogs treated for AT. Improvement may take about 2 weeks. The median survival time with this drug has been about 2 years. An International Normalized Ration (INR) of 2-3 is recommended for warfarin therapy. Dosing of warfarin can be found elsewhere. Antiplatelet therapy has been used in combination with this therapy.

Suggested Reading:


Fibrocartilaginous Embolism

Fibrocartilaginous Embolism (FCE) is a common cause for an acute, nonprogressive disease. The etiology is unknown, but multiple theories exist. There is a sudden disruption of blood flow to the spinal cord secondary to a small fragment of fibrocartilage (disk material). The disruption in blood flow causes ischemia in the spinal cord with secondary edema. This causes secondary compression of the spinal cord. Loss of function results as a result of these events. Necrosis is the end result of this process. FCE is most common in large breed, active dogs and miniature schnauzers. It is rare in cats, but is reported. The problem is associated with activity in most instances. These animals have no history of trauma and may initially have pain. Signs progress rapidly over 24 hours. The signs should not progress longer than 24 hours. In most instances, there is a marked asymmetrical paresis. There is generally no spinal pain. Diagnosis of this condition begins with survey spinal radiographs. This helps to rule out other causes for acute paresis. MRI of the spine is recommended for this condition. Therapy of this condition consists of supportive care, as well as fluid therapy to maintain spinal cord perfusion. Corticosteroids have not been shown to be effective, particularly since the type of edema (cytotoxic) does not respond to corticosteroids. Physical therapy is helpful and has been shown to improve recovery, particularly when other problems, such as disk disease, have been ruled out. The prognosis for this condition is dependent on the severity of the initial injury. In general, lesions of the intumescence and loss of pain perception tend to have a more guarded prognosis.

Suggested Reading:


Upper Thoracic Intervertebral Disk Disease

Intervertebral disk disease is a well-described cause for paraparesis and back pain in dogs. While typically small breed dogs seem to be more affected by this problem, some large breed dogs have a relatively high incidence of upper thoracic IVDD. In particular, the German shepherd dog has a high incidence of this disk disease. This is of particular importance, because the disease is commonly missed due to the assumption that the dog should be painful with disk disease. This is not always the case. Recent reports have described a number of large breed dogs, the German shepherd in particular, with significant spinal cord compression in the upper thoracic spine. Golden retrievers were also over-represented. The affected dogs were between 7 and 9 years of age in an early report, but a larger study reported a median of 9.5 years. The disks were located at T2-3 and T4-5 most commonly. This condition in GSDs is an important consideration, because this can be misdiagnosed as DM. Many of these dogs will have multiple disk herniations. These dogs do not tend to have spinal pain. They may have a sudden or chronic, asymmetric paraparesis. Diagnosis of this condition requires MRI or myelography. Surgical decompression is recommended in many cases.

Suggested Reading:


Extrusion of Hydrated Nucleus Pulposus in the Cervical Spine

A relatively uncommon cause of acute tetraparesis in dogs is the sudden herniation or extrusion of hydrated or non-degenerate disk material into the spinal canal. This phenomenon is also described in humans and has been called a discal cyst or a hydrated nucleus pulposus extrusion (HNPE). This condition has fairly unique features, which include a peracute onset of severe tetraparesis or tetraplegia with a lack of significant neck pain. Dogs may have some resistance to neck manipulation. This condition may be confused with FCE or exercise associated disk herniation. The signs tend to be symmetrical and in some dogs the front limbs are more severely affected. This condition differs from the more common form of disk disease in the cervical spine, which is generally associated with significant neck pain and relatively less frequent occurrence of tetraparesis (9-17% of dogs). Classically, IVDD in the cervical spine is most frequently encountered at C2-3. With HNPE, the location of the lesion is almost always C4-5 or C3-4. The diagnosis of this condition is with MRI of the cervical spine. In general, this is considered a surgical condition. A ventral slot is recommended, and the prognosis is generally very good to excellent.

Suggested reading:

