Volume 44 | Issue 2 Article 1

1982

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Phil Olson
Iowa State University

Robert W. Carithers
Iowa State University

Differential Diagnosis of Conditions Mimicking Intervertebral Disc Disease In the Canine

by
Phil Olson, DVM*
Robert W. Carithers, DVM, MS, PhD**

Many abnormal conditions of the canine can produce clinical signs that in some way mimic those associated with intervertebral disc disease. This oftentimes leads to unnecessary confusion and frustration in the diagnosis and subsequent treatment of these cases by practicing veterinarians. The purpose of this paper is to summarize the signs associated with those seen most commonly and thereby to clarify the manner in which these can be most readily differentiated in a clinical situation. This paper is not intended to be an in-depth analysis of all canine spinal cord problems, but only to provide a review of clinical signs, pathogenesis, and diagnosis of certain conditions which present in a similar manner.

CERVICAL VERTEBRAL INSTABILITY

Atlanto-axial subluxation occurs in miniature and toy breeds, resulting from fracture, degeneration, or malformation of the dens. The pathogenesis of absence of the dens is unknown, although some speculation exists that the mechanism is similar to femoral head necrosis of Legg-Perthes Disease.[5,6]

Luxation most commonly occurs at six to eight months of age. Absence of the dens or fracture allows the cranial aspect of the body of the axis to rotate dorsally into the vertebral canal, with subluxation sometimes a result. Clinical signs include severe neck pain, spastic tetraparesis, and recumbency. Thoracic limb paresis often is the most profound sign. The animal walks with a short stiff stride.[5,6,7]

Handling should be done with extreme care, with no manipulation of the atlantoaxial region. Ataxia and paresis along with forced exercise is likely to cause falling and greater subluxation. Anesthesia eliminates support of muscle tone, increasing the danger further.[5,7]

"Wobbler Syndrome" is another common cervical problem. The condition most commonly occurs in Great Danes and Doberman Pinschers, and clinical signs are due to compression of the cervical spinal cord caused by vertebral malformation-malarticulation of C_5, C_6, and C_7.[2,5,6]

Clinical signs are usually seen at less than one year of age, often by two, and occasionally later. Owners generally recognize ataxia of the pelvic limbs. The onset is usually insidious but sometimes acute, with signs normally progressive. The pelvic limbs are seen to cross each other, abduct or collapse on walking and especially on turning. The hind quarters sway awkwardly. The dog may knuckle over. The impression given is that the animal doesn't know where the limbs are because of a proprioceptive deficit. Thoracic limb signs, if present, are similar but less marked. Occasionally, limb crossing and knuckling over occur.[2,5,6]

Neurological exam reveals abnormal postural reactions present, especially hopping and proprioceptive positioning. Manipulation of the neck usually does not elicit pain. Blood, urine and CSF parameters are normal (possibly slightly elevated protein in the CSF). There is involvement of ascending proprioceptive and descending motor tracts.[2,5,6]

Intervertebral disc protrusion may be associated with the condition. Unrelated cervical disc extrusion occurs in older dogs and may be readily identified on plain radiographs. Cervical pain in this case is prominent. The primary neurological disease to differentiate from in young dogs is canine distemper myelitis. Neurological exam reveals other ab-

*Dr. Olson is a 1982 graduate of the College of Veterinary Medicine at Iowa State University.
**Dr. Carithers is a Professor in Veterinary Clinical Sciences at Iowa State University.

normalities not explained by a localized lesion, as distemper lesions are usually disseminated. For example, the examiner may see paraplegia with mild thoracic limb deficit, suggesting separate lesions, one thoraco-lumbar, and one cervical. Head tilt, tremor, and abnormal nystagmus may be present, suggesting a cerebello-vestibular lesion. Also, with distemper, the CSF is often abnormal.[5,6]

NEOPLASIA

Neoplasia may occur as extramedullary expansive masses, compressing the spinal cord to produce an ischemic myelopathy.

Classification is either of vertebral or spinal cord neoplasia. Vertebral neoplasia characteristically shows compression myelitis of the spinal cord due to invasive bony growth. It may be primary or metastic. Osteosarcoma, chondrosarcoma, and fibrosarcoma are most common. Spinal cord or meningeal neoplasms are seen in the dog, most often in the thoracic cord. Meningiomas are most common. Medullary neoplasms are rare and when present, are usually neurofibromas or neurofibrosarcomas.[6,7]

Typical signs of neoplastic spinal cord compression are slow in onset and progression. However, they often show acute signs and can progress rapidly. Necropsies have shown a mass may grow slowly a considerable time, with the spinal cord adapting. Suddenly a critical point is reached, where spinal cord circulation is compromised, resulting in lesions and clinical signs. Adaptation can be phenomenal, showing an amazing decrease in cord diameter on necropsy. Often the neoplasm is lateral, therefore initial signs are likely to be asymmetrical, with paresis and ataxia more pronounced ipsilaterally. Signs later progress to bilateral.[2,5,6]

Plain radiographs are normal unless vertebral invasion or origination has occurred. A myelogram usually demonstrates the lesion. A thorough physical exam and thoracic radiographs are important to diagnose metastasis.[2,5]

Neoplasia may be confused with vertebral infection as a slowly progressive lesion. If signs appear acutely, then an intervertebral disc problem, vascular accident and others must be included in the initial ruleouts. The fact that neoplasia generally occurs in older animals may also aid in the initial analysis of the case.

CANINE DISTEMPER

Signs of this disease may present due to predominant action of the virus on the spinal cord, with no history of systemic illness. Dogs less than one year old are especially suspect of having the disease. The history should be of signs of segmental myelopathy, with progressive worsening.[5,6,16]

Information helping to differentiate this etiology from a focal thoraco-lumbar spinal cord lesion, such as intervertebral disc disease, often is neurological deficits at multifocal areas. An example is mild thoracic limb deficit in a paraplegic dog. This suggests a mild cervical spinal cord lesion and a severe thoraco-lumbar lesion.[5,6]

Most often additional signs are present which make diagnosis much easier. Head tilt, abnormal nystagmus and head tremor suggest cerebellovestibular dysfunction. Nasal or ocular discharge, harsh lung sounds, hyperkeratosis of the nose and foot pads and chorioretinopathy may be seen. Myoclonus is a nearly pathognomonic sign. Diffuse lesion distribution and progressive nature suggest inflammatory disease. The CSF is many times normal, but may show a mild increase in mononuclear cells, protein or both.[5,6,16]

TOXOPLASMOSIS

Toxoplasmosis can produce unlimited neurological clinical signs. It is much less common than canine distemper. As with distemper, signs may manifest as segmental myelopathy.[5]

Possible clinical signs include seizures, blindness, tremor, hemi- or paraparesis and hindquarter paralysis. Spinal reflexes may be absent and extensor rigidity present in one or both hindlimbs. The history is usually of a dog less than one year of age, showing gradual progressive paralysis. Some common signs that when present with CNS signs are suggestive of toxoplasmosis include abortions, stunted young, iritis, retinitis, recurrent fever, lymphadenopathy, diarrhea, pneumonia, myocarditis, and icterus.[5,6]

Positive diagnosis is difficult. Serum titers can be taken but interpretation is uncertain, although rising titers are suggestive. Systemic mycoses may also produce CNS signs and retinitis.[6]

POLYRADICULONEUROPATHY—COONHOUND PARALYSIS

The etiology of Coonhound Paralysis is

unknown. The functional lesion is apparently on the ventral nerve roots and peripheral nerves. The disease is initiated by coon bites one to two weeks before signs appear. Hallmarks are pain without motor function. Ninety percent show paresis and hyporeflexia in the posterior limbs first and dysphonia. Progression is to tetraplegia with areflexia, normal mental altertness, and normal temperature. Most commonly affected are adult hunting dogs.[4,6]

TOXIC POLYNEUROPATHY— TICK PARALYSIS

The exact mode of pathogenesis is unknown, however, the initiating agent is a neurotoxin from a female tick (usually *Dermacentor andersoni*). Only one tick is needed to show signs.[4,6]

Clinical presentation is of an ascending flaccid paralysis, with little or no sensory impairment. Progression is from hyporeflexia and ataxia to paresis to paralysis and areflexia. Signs are seen five to seven days after attachment of the ticks. Differential diagnosis must include coonhound paralysis. Finding ticks on the dog is suggestive.[4,6]

INFECTIOUS VERTEBRAL OSTEOMYELITIS-DISKOSPONDYLITIS

Diskospondylitis is an inflammation of the vertebral bodies and associated intervertebral discs. Typically the source is hematogenous, although contiguous tissue infection may be associated. A variety of organisms have been cultured, including *Staph. aureus, Strep. canis, Brucella canis, Corynebacterium spp., Pseudomonas* and others. Cultures are negative twenty-five percent of the time and sepsis may not be demonstrated by biopsy.[2,5,6]

Diagnostically, the most important sign is severe vertebral column pain associated with a progressive paraparesis and ataxia. Neurologic deficits result from compression due to bone proliferation associated with the lesion. Fever and blood chemistry abnormalities may be absent. Radiographs may reveal bony proliferation of vertebral bodies with lysis present.[2,5,6]

SPINAL BIFIDA

Spinal bifida is a congenital defect in the dorsal laminae of the vertebrae, often resulting in herniation of meninges. The pathogenesis of this condition is not completely understood. Clinical signs depend on the location of the lesion, with trauma more easily causing injury to the spinal cord becasue of lack of bony protection. Diagnosis is by radiography of the spine, myelography, and breed. The condition is most common in the "screw-tailed" breeds (Boston Terrier, Pug, French and English Bulldog).[2,4,6]

SPINAL DYSRAPHISM

Spinal dysraphism is an inherited incomplete closure of the neural tube in Weimaraners. It may be associated with syringomyelia, which is seen as tube-like cavities in the spinal cord.[4]

Clinical signs include a hopping gait on the hind legs, crouching wide stance, and scratching reflex in both hind legs at once with paresis. The signs do not improve or worsen. Diagnosis is subjective and ascertained by breed, clinical signs and history. Radiographs and CSF tap help determine that no treatable disease is present.[4,6]

HEMIVERTEBRAE

Hemivertebrae is a condition of uncertain pathogenesis, consisting of failure of fusion of right and left halves of vertebrae. The "screw-tailed" breeds, Boston Terriers, Pugs, French and English Bulldogs, are most often afflicted.[4]

Clinical signs are rare. They are caused by intermittent trauma to the spinal cord due to instability of the vertebral column. Clinical signs are usually progressive and include posterior paresis, urinary and fecal incontinence, muscle atrophy, abnormal conformation and pain. Paresis may be acute. Diagnosis is by myelography.[2,4]

CAUDA EQUINA SYNDROME

Cauda equina syndrome is a nerological condition that results from the compression, destruction, or displacement of the nerve roots forming the cauda equina. The condition usually occurs in older dogs, presenting as spinal nerve root signs resulting in disorders of the pelvic limbs, sphincters, and tail/perineal areas.[2,6]

The most common sign is pain in the lumbosacral area and tail. Other signs include pelvic limb lameness, difficulty rising, paresis of the tail, urinary and/or fecal incontinence, perineal hyperalgesia, self-mutilation of tail and perineal area and decreased lumbosacral spinal reflexes. The clinical signs vary greatly due to

multiple etiologies and varying degrees of compression. The early signs can be confused with hip dysplasia. Differential diagnosis includes degenerative myelopathy, type II disc protrusion and spinal neoplasia.[2,6]

DEGENERATIVE MYELOPATHY OF GERMAN SHEPHERDS

Degenerative myelopathy occurs in aging German Shepherds and is characterized by a slowly progressive paraparesis and ataxia of the pelvic limbs. The onset is insidious and may continue for five to six months. Proprioception is the first reflex to go, along with crossed limbs and hyperreflexia of the pelvic limbs. Signs can be asymmetrical. Often a disuse atrophy of the muscles in the caudal thoracic and lumbosacral region is evident.[5,6]

Geriatric intervertebral disc disease can be ruled out by plain radiography and myelogram. The myelogram would demonstrate a normal subarachnoid space not compressed by space-occupying mass (neoplasia) or excessive dural ossification, or encroachment of spondylosis deformans. CSF is normal.[5]

The lesion is of multisystemic degeneration consisting of diffuse white matter degeneration in both ascending and descending spinal cord tracts in all segments of the cord. Both myelin sheaths and axons degenerate. Peripheral nerves may be involved, resulting in lower motor neuron signs. Diagnosis is usually based on breed, age, typical history, clinical signs, and inability to find other lesions.[5,6]

VASCULAR ACCIDENT—FIBROCARTILAGINOUS EMBOLI

A vascular accident involving the spinal cord is caused by occlusion of vessels of leptomeninges and spinal cord parenchyma by fibrocartilaginous emboli derived from herniated disc material. Presenting signs are an acute (developing in less than six hours), unilateral hemiparesis or hemiplegia. This may occur from the cervical region caudal to the caudal vertebrae, showing signs referable to C_3 to L_7, varying with the area affected.[2,5]

Often recovery is spontaneous, especially if some voluntary motion is retained in the affected limbs. Improvement is usually noted during the first week, possibly due to resolution of hemorrhage and edema and to collateral circulation reaching ischemic areas. Later improvement may be due to compensation for permanent loss of spinal cord parenchyma.[5]

The syndrome should be differentiated from other spinal cord lesions demonstrating acute onset. Possibilities include acute disc herniation and osteomyelitis, while neoplasia and spinal cord infection more commonly manifest as deterioration over several months' time. Since there is a large area over which this can occur and thus show signs, the differential must be made more on the basis of acute onset with improvement following and on unilateral signs rather than location.[2]

PATHOLOGICAL VERTEBRAL FRACTURE

Chronic renal insufficiency in older dogs from interstitial nephritis, glomerulonephritis or amyloidosis results in hypocalcemia. The resultant increase in parathormone (PTH) induces accelerated release of stored calcium from bone. Skeletal involvement is general, but certain lesions are more apparent in given areas. Loss of alveolar socket bone and lamina dura dentes occurs early, loosening the teeth. The maxilla and mandible are softened and readily pliable (rubber jaw syndrome). Lameness and stiffness of gait occur. Vertebral fractures after relatively minor trauma can cause spinal cord damage. The fracture may occur anywhere along the vertebral column, thus signs may be referable to most any area. Other signs that may aid in diagnosis include jaw softening, a generalized decrease in bone density, and abnormalities found in urine analysis and on general workup.[6,11]

Neoplasia can also cause lysis of vertebrae and resultant fracture (see section on neoplasia).

Differential diagnosis should again include other spinal cord lesions demonstrating acute onset, such as vascular accident, acute disc herniation, and osteomyelitis.

TRAUMA

The most likely sources of external trauma are auto collision, gunshot, and fight wounds, presenting with sudden onset relating to the time of incident or within a few hours. Signs are generally nonprogressive, especially greater than twenty-four hours post-trauma. If signs continue to develop over the next twenty-four hours, they indicate continual bleeding or excessive movement at the site of vertebral injury, thus allowing continued cord trauma. Thereafter signs are stable or improved.[5,6,13]

The thoraco-lumbar junction is the most common fracture site. It should be realized that radiographs show only the present degree of luxation and do not show the extent at the time of injury. Presenting signs may include Schiff-Sherrington syndrome, with normal postural reactions and efforts of voluntary movement. Spinal reflexes caudal to the lesion are normal or exaggerated. Lesion level may be determined by panniculus response.[5,13]

"Spinal shock" is a physiological phenomena, commonly seen in primates, that causes complete atonia and areflexia caudal to the lesion for two to three weeks. While a distinct entity in primates, in domestic animals it is not outstanding and carries no clinical significance. By the time the veterinarian examines the patient, spinal reflexes are always present caudal to the lesion.[5,6,13]

Location of injuries associated with trauma should be done with minimal patient manipulation, with the animal in lateral recumbency. To differentiate the Schiff-Sherrington phenomenon from cervical injury in cases of thoracic limb hypertonia, check for pain and voluntary movement. In the Schiff-Sherrington syndrome they are present in the forelegs and not in the rear. With cervical spinal cord injury, pain and voluntary movement deficit is more equal.[5,6,13]

INTERVERTEBRAL DISC DISEASE

Intervertebral disc disease occurs in all breeds, being seen more commonly in chondrodystrophic breeds (Dachshund, Pekinese, French Bulldog) and can occur at a young age. The condition is thought to be related to the early spontaneous degeneration of the intervertebral discs that takes place in these breeds. Occurence is considered unlikely at less than one year of age, occasionally at two years of age, and quite often at three or more years of age. Miniature poodles, beagles, and cocker spaniels also undergo this type of degeneration. In nonchondrodystrophic breeds it is also seen and is associated with aging, occurring in dogs five years of age and older.[5,10]

Signs of disc protrusion in the cervical region from C_2 to T_1 include a "stiff neck" appearance as a classical sign. Also seen are an arched back, reluctance to move, and pain with vocal manifestations, which may even be elicited by touch. Tremors of muscle groups of the thoracic limb and neck, along with knuckling over, are seen. The C_2-C_3 disc and C_5-C_6 disc most commonly are involved.[2]

Thoracolumbar disc protrusion is likely to show less pain. The dog may show aggressiveness, refusal to move, and pain when handled. The lumbar muscles are rigid and kyphosis of the spine may be evident. Local hyperesthesia at the site of herniation may be present. Motor signs include changes in gait and progression from ataxia to knuckling over to paresis and finally paralysis.[2]

Eight percent of disc protrusions are at T_{11}-L_3. An intercapital ligament coursing dorsally over the discs from T_2-T_{10} is a major factor in low incidence of disc protrusion here.[13,14] Pressure from protrusions or extrusions produces varying degrees of ischemic myelopathy in the spinal cord by interfering with blood circulation in the parenchyma. A sudden large extrusion may produce focal hemorrhage and myelomalacia of grey and white matter.[5]

In all cases spinal reflexes are intact. Most often they are hyperreactive and manipulating limbs reveals hypertonia, ie. classic signs of upper motor neuron disease. Pain sensation is almost always intact in paraparetic cases. Paraplegic animals with pain sensation have a better prognosis than those with no cerebral response to deep pain, such as that elicited by forceps pressure at the base of the toenail. In these cases, the panniculus test is helpful.[5]

The examiner must establish the degree of paresis, spinal reflex function and cerebral response to pain in order to establish prognosis and treatment. Radiographs and a myelogram are also very helpful in precise localization of the lesion.

CONCLUSION

A good history and clinical neurological exam are very important in cases of spinal cord disease. A knowledge of spinal column anatomy and physiology is necessary for an understanding of clinical signs and interpretation of neurological tests. This knowlege, along with an understanding of the pathology and clinical signs associated with spinal cord disease, will enable the clinician to give an accurate estimate of functional cord damage, localize the lesion, and suggest optimal therapy. Spinal cord problem diagnosis and treatment can then become a very rewarding experience for the practicing veterinarian.

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