# A PAIN IN THE NECK – WHAT TO CONSIDER FOR DOGS WITH SPINAL PAIN

Simon R. Platt BVM&S FRCVS Dipl. ACVIM (Neurology) Dipl.ECVN

College of Veterinary Medicine, University of Georgia, Athens, GA

**Abstract:** In addition to intervertebral disc disease which is discussed in another session, there are multiple considerations for the causes of neck and back pain in dogs. In this session we will address the common possibilities, which include discospondylitis, meningitis, spinal tumors, trauma and Chiari-like malformation.

**Keywords**: Discospondylitis, Chiari, neoplasia, Atlanto-axial subluxation

## Discospondylitis/osteomyelitis

Discospondylitis is due to infection of the intervertebral disc and adjacent vertebral endplates; if the infection is confined to the vertebral body, it is called vertebral osteomyelitis or spondylitis. The sites most commonly affected are L7-S1, caudal cervical, midthoracic, and thoracolumbar spine. The infection is usually slowly progressive but can result in acute signs due to secondary pathological vertebral fractures and intervertebral disc disease. Its most common clinical sign is that of spinal pain but neurologic signs are seen and realte to the localization. An association with empyema has been documented in several dogs, which may represent an extension of the disease and should be considered when considering diagnostic tests and or when dealing with a refractory case. Coagulase positive *Staphylococcus spp. (S. intermedius* or *S. aureus*) is the most common etiological agent associated with canine discospondylitis; other less commonly identified organisms include *Streptococcus* spp., *Escherichia coli, Actinomyces* spp. and *Brucella*

*canis,* as well as *Aspergillus* spp. Young German Shepherd bitches seem to be predisposed to aspergillosis, whereas young Basset Hounds contract discospondylitis due to systemic tuberculosis. Spinal pain is the most common initial clinical sign in this disease, which is most frequently seen in large intact male young to middle-aged dogs. With proliferation of inflammatory tissue, compression of neural tissue can lead to ataxia, paresis and occasionally paralysis dependent on where the lesion is located. Although it can occur in any animal, the condition is less common in toy and chondrodystrophoid breeds of dog, and rare in cats. Purebred dogs seem more commonly affected than mixed-breeds. Approximately 30% of dogs have signs of systemic illness such as fever and weight loss. Hematogenous spread from distant foci of infection (urogenital tract, skin, dental disease), penetrating wounds, surgery, or plant material migration can cause direct infection of the disc space or vertebrae, the latter of which is usually seen at the level of L2–4 at the insertion of the diaphragmatic crus. Immunosuppression due to factors such as hyperadrenocorticism is considered a predisposing cause. Infection causes osseous lysis, proliferation and soft tissue reaction, which can cause neural compression. Hematological changes are usually not present unless there are concurrent conditions such as endocarditis. Urine cytology may reveal bacterial or fungal agents. Blood and urine cultures should be performed in all suspected cases and are positive in up to 75% and 50% of cases, respectively. Ideally these should be performed prior to initiating antibiotic therapy. Serology for brucellosis should also be performed, especially in view of its zoonotic potential; this has been reported to be positive in up to 10% of cases.

Definitive diagnosis is usually made with spinal radiographs, although radiographic change may not be evident in the first 2–4 weeks of infection. The most commonly affected site is L7–S1, but other frequently affected sites include the caudal cervical/cranial thoracic vertebrae and the

thoracolumbar junction. As this can be a multifocal disease, the entire spine should be radiographed. Radiographic evidence of disease includes narrowing of the disc space, accompanied by subtle irregularity of both endplates through to gross lysis and osseous proliferation of the adjacent vertebral bone and even fractures. Radiography can also be used to monitor the response to treatment or the progression of the disease, although clinical progression is equally important, as radiographic change can lag behind clinical improvement.

Computed tomography (CT) can identify subtle endplate erosion and paravertebral soft tissue swelling more readily than radiography. Post-myelogram CT clearly defines compression of the neural tissues by infected tissues, as does MRI. Discospondylitis appears to have increased signal intensity on T2-weighted images and decreased signal intensity on T1-weighted images, changes also seen in the paravertebral tissues in all cases. MRI can also highlight the inflammation in the surrounding muscles.

If urine and blood culture, and brucellosis serology, have not identified an etiological agent in cases of discospondylitis, percutaneous needle aspiration of the disc space can be a safe procedure to obtain tissue for bacterial and fungal cultures and cytology. However, this procedure requires general anesthesia, sterile surgical preparation and fluoroscopic or CT guidance of the needle, and is usually only performed in patients unresponsive to initial broad-spectrum antibiotics. The procedure has been documented to be up to 75% sensitive; open biopsy of the vertebrae may be considered if needle aspiration is unrewarding. This has yielded positive cultures in approximately 80% of patients. In all cases, diagnostic investigation of potential systemic infectious foci should be considered. This should include abdominal ultrasonography for prostatic or renal disease, thoracic radiographs for pulmonary disease, and cardiac ultrasonography for endocardial disease.

Once imaging based radiographic evidence of discospondylitis is present, treatment for the common potential pathogen *Staphylococcus spp.intermedius* infection may be started. The initial treatment of discospondylitis consists of antibiotics (potentiated amoxicillin or cephalexin), cage rest and analgesics. Results of cultures may require alteration of this choice.

Intravenous antibiotics should be considered if severe neurological compromise or signs of sepsis are present; otherwise, oral antibiotics are acceptable. However quickly the patient improves, continuation of the antibiotics for 8-16 weeks is recommended. Resolution of clinical signs, such as pain and fever, should be expected within 5 days of initiating therapy; however, complete neurological resolution may take 2–3 months. Residual deficits may remain, but persistent pain indicates an active disease, and these patients should be treated with an additional antibiotic and considered for further diagnostics as they may have a potential fungal infection or surgical lesion. Discospondylitis associated with Aspergillus spp. has been treated with itraconazole (5 mg/kg of body weight, PO, q 24 h) although long term reports of success are lacking with the belief being that chronic recurrence and progression is likely.

Surgical decompression is rarely needed, and should only be considered in refractory cases or those with severe neurological deficits that show no sign of improvement within 3–5 days. Although internal fixation can be acceptable even at the site of an infection, it may be more appropriate to consider external skeletal fixation as has been successfully described in dogs with lumbosacral discospondylitis.

The prognosis for this disease is generally very good unless the etiology is fungal, there are multiple lesions, vertebral fracture or subluxation occurs or there is endocarditis; the potential for recurrence should be considered, especially if brucellosis has been diagnosed or an underlying

immunosuppressive condition is present. Residual neurological deficits are possible, and in those cases that have severe neurological deficits associated with the infection the prognosis should initially be guarded.

**Chiari-like malformation and Syringomyelia (CM/SM):**

Chiari-like malformation (CM) and syringomyelia (SM) often occur together, although both may occur independently of the other. Syringomyelia is a condition characterized by the presence of a fluid filled cavity (syrinx) or cavities within the parenchyma of the spinal cord. SM is secondary to abnormal cerebrospinal fluid movement and is usually associated with Chiari-like malformation, although it may be associated with other conditions such as congenital malformations, trauma, inflammation, and neoplasia. Chiari-like malformation is defined as a decreased caudal fossa volume with herniation of the cerebellum and often brainstem into or through the foramen magnum. In people, this condition is referred to as Chiari malformation, which has several types.

The term syringomyelia is accepted to describe fluid accumulation within the spinal cord, whether it be secondary to central canal dilation (hydromyelia) or secondary to fluid accumulation within the spinal cord parenchyma (syringomyelia or syringhydromyelia). It is difficult to determine the location of the fluid using Magnetic Resonance Imaging (MRI) and these cavities often communicate with each other. Syringomyelia frequently occurs with Chiari-like malformation in dogs and the terms Chiari-like malformation and syringomyelia (CM/SM) have been adopted to describe the canine condition.

Onset of signs may be acute or chronic in dogs ranging from 6 months to 10 years of age. The most common sign of CM/SM is pain, predominately isolated to the cervical region, occurring in 35% of affected dogs and 80% of people with the similar condition. Syrinx width, measured by MRI, has been shown to be the strongest predictor of pain in dogs where a wider syrinx was significantly associated with discomfort. Additionally, the location of the syrinx within the dorsal aspect of the spinal cord affecting the dorsal horn is thought to be one mechanism behind the development of pain, specifically neuropathic pain. Neuropathic pain is secondary to disordered processing of sensory information within the nervous system and results in spontaneous pain, paresthesia, dysthesia, allodynia, or hyperpathia. As a result, dogs may dislike touch to the skin of their neck or they may scratch with or without making contact to the skin on their neck. This "phantom scratching" has frequently been described in affected dogs. Pain may also be to CM alone as seen in dogs without SM secondary to compression of the brainstem or first cervical nerve.

Other clinical signs depend on the location of the syrinx, although the cervical spinal cord is predominately affected. These clinical signs include scoliosis and neurological deficits relating to cervical spinal cord dysfunction. Intracranial signs, such as facial paresis and vestibular dysfunction, have also been reported. However, dogs may also be asymptomatic for CM/SM.

The human classification of Chiari type I, which is the most similar to the canine condition, necessitates elongation and caudal displacement of the cerebellar tonsils (vermis and paravermal lobes) through the foramen magnum into the cranial cervical vertebral canal. A similar condition has been documented in dogs particularly affecting toy or small breed dogs. Predisposition to CM/SM has been seen in Cavalier King Charles Spaniels (CKCS) and Brussels Griffon dog. In CKCS, CM/SM is a hereditary condition, possibly autosomal recessive with incomplete

penetrance. CM is caused by congenital hypoplasia of the supraoccipital bone resulting in overcrowding of the structures within the caudal fossa. As a result, the cerebellum herniates through or into the foramen magnum, the medulla becomes kinked, and the dorsal subarachnoid space at the craniocervical junction is obstructed. The flow of CSF through the foramen magnum is disrupted as a result. Many theories behind why syringomyelia develops as a result of this obstruction have been postulated although a single theory has not been proven.

These structural abnormalities are best diagnosed with MRI, but they may be clinically silent; therefore, their significance must be carefully considered when such abnormalities are discovered.

Treatment may not be necessary in asymptomatic dogs or dogs with mild nonprogressive signs. Dogs exhibiting pain, more severe neurological deficits, or progressive signs can be treated either medically or surgically. Typically, medical therapy is pursued initially involving the use of analgesics and drugs that reduce CSF formation. Furosemide (1-2 mg/kg orally q12h) and prednisone (0.5-1 mg/kg orally 24h, tapering dose) are frequently used. Treatment of neuropathic pain with drugs such as Gabapentin (10 mg/kg PO q 8 h) is also an important aspect of therapy.

**Steroid Responsive Meningitis-Arteritis**

A severe form of steroid responsive meningitis-arteritis (SRMA) has been reported in Beagles, Bernese Mountain Dogs, Boxers, German Short-Haired Pointers, and sporadically in other breeds. This condition has a worldwide distribution and represents one of the most important

inflammatory diseases of the canine CNS.

Affected animals usually are most commonly young adults between 8 and 18 months of age, although the age range may extend from 4 months to 7 years. The clinical course is typically acute with recurrences. A more protracted form of the disease may be seen following relapses and inadequate treatment. Signs include recurring fever, hyperesthesia, cervical rigidity, and anorexia. There may be a creeping gait, arching of the back with head held down, and crouched posture. Some dogs with protracted disease may show clinical signs of parenchymal involvement such as ataxia, paresis, tetraparesis or paraplegia. Hematological studies often reveal a peripheral neutrophilia with a left shift, increased erythrocyte sedimentation rate, and in some cases, an elevated a-2-globulin fraction. CSF studies indicate increased protein and neutrophilic pleocytosis.

The prognosis is guarded to favorable, especially in dogs with acute disease that are treated promptly using immunosuppressive doses of corticosteroids. Untreated dogs tend to have a remitting and relapsing course. Tipold recommends the following long-term therapy (e.g., for at least 6 months), especially in any dog that has had a relapse: prednisolone at 4 mg/kg/day, PO or IV initially. After 2 days, the dose is reduced to 2 mg/kg daily for 1 to 2 weeks, followed by 1 mg/kg daily. Dogs are re-examined, including CSF analysis and hematology, every 4 to 6 weeks. When signs and CSF are normal, the dose can be reduced to half of the previous dosage until a dosage of 0.5 mg/kg every 48 to 72 hours is attained. Treatment is stopped 6 months after clinical examination, CSF, and blood profiles are normal. In refractory cases, other immunosuppressive drugs such as azathioprine (at 1.5 mg/kg PO every 48 hours) may be used in combination with steroids (e.g., alternating each drug every other day). Antibiotics are ineffective. Results of a long-term treatment protocol (up to 20 months) involving 10 dogs with

SRMA have been recently published. Eight of the 10 dogs were without clinical signs up to 29 months after the treatment was terminated. Long-term glucocorticosteroid treatment resulted only in mild clinical side effects, such as polyuria/polydipsia, polyphagia and weight gain, which were reversible after the therapy was discontinued. It was noted that elevated serum and CSF IgA levels did not decrease to normal values during prednisolone treatment and were still slightly increased after the therapy was discontinued. Monitoring of CSF cell count in dogs with this condition was a sensitive indicator of success of treatment. In addition, older dogs with high IgA levels in the CSF and frequent relapses seemed to require a longer duration of therapy and had a less favorable prognosis long term.

Note that Akitas, Bernese Mountain dogs, and other breeds with immune-mediated polyarthritis

**Spinal Meningiomas**

Tumors affecting the spinal cord are described based on their location as extradural, intradural–extramedullary and intramedullary. Intradural–extramedullary tumors that occur include meningioma and nerve sheath tumors. Meningiomas affecting the spinal cord are most common in the neck. Meningiomas of the spinal cord in dogs and cats tend to present with a progressive paraparesis. The clinical signs represent the spinal cord region involved. Paraspinal pain may or may not be present. Spinal meningiomas are the most common primary spinal cord tumor in cats older than 8 years of age. Mean age for spinal meningiomas in dogs is 9 years of age. Spinal meningiomas in dogs are most common in the cervical spinal cord, but occur in any region of the spinal cord. Myelography typically shows an intradural extramedullary compressive lesion. With MRI, these tumours are iso- to hypointense on T1-weighted (T1W) images, hyperintense on T2-weighted (T2W) images, and demonstrate strong, uniform contrast enhancement. These lesions should be surgically explored because many meningiomas can be completely or partially

removed and therefore may be associated with prolonged survival after surgery. Postoperative radiation therapy may be used adjunctively to prolong survival in dogs with incompletely excised tumors. Treatment with surgery and radiation therapy can result in an improved outcome and prevent recurrence. Surgical results are guarded when meningiomas are associated with an intumescence and tumors with ventral location and invasion of the neural parenchyma.

**Vertebral body tumors**

Tumors affecting the spinal cord are described based on their location as extradural, intradural–extramedullary and intramedullary. Extramedullary tumors are the most frequent and most commonly are primary vertrebral tumors. Clinical signs may be focal or multifocal depending upon the extension of the tumor. Signs include pain and paraparesis or paralysis. Pathological fractures of the vertebral body result in an acute onset of neurological deficits. Vertebral body tumors are primary or metastatic tumors most frequently reported in large and giant-breed dogs. Commonly described tumors in dogs include: osteosarcoma; fibrosarcoma; chondrosarcoma; hemangiosarcoma; plasma cell tumor; carcinoma; lymphoma; and liposarcoma. Small-breed dogs have a higher rate of vertebral metastasis than large-breed dogs. In cats, the most commonly described vertebral body tumor is osteosarcoma. Primary vertebral body tumors will cause a secondary myelopathy by compression or direct spinal cord invasion. The diagnosis is often based on survey radiographic findings, such as lysis, and pathological fractures secondary to tumor destruction of the bone. Other supportive diagnostic techniques, such as CT, MRI and myelography, are used to determine lesion extent. MRI and scintigraphy can be used to detect multiple metastases. Fluoroscopic-guided needle aspiration or surgical biopsy can be used to obtain a definitive diagnosis. Palliative treatment options include surgery, radiation therapy, chemotherapy or various combinations of the three. A vertebrectomy with a bone allograft fusion

has been used for the treatment of a primary vertebral neoplasm in a dog. Decompression or stabilization techniques are used in patients that are rapidly deteriorating. The overall prognosis is considered guarded for dogs and cats with vertebral neoplasia. Survival is not impacted greatly by various treatments but is often determined by the neurological deficits at the time of diagnosis.