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Introduction:

Quadriparesis (weakness and ataxia of all 4 limbs) and quadriplegia (paralysis of all 4 limbs) are common problems in all animals. Once the neurologist, faced with an animal who has neurologic disease affecting all 4 limbs, has determined that the lesion is below the foramen magnum (meaning a spinal cord or peripheral disease), there are 4 possible anatomic locations for the disease process: 1) if there is UMN dysfunction in all 4 legs, the lesion is most likely to be in the spinal cord between C1-C5; 2) if there is LMN dysfunction in the fore legs and UMN dysfunction to the rear legs, the lesion is severe and involves spinal cord segments C6-T2; 3) if there is UMN dysfunction to the rear legs and "root signature" (lameness due to nerve root involvement) in the forelegs, the lesion is mild and affecting spinal cord segments C6-T2; or, 4) if there is LMN dysfunction in all 4 limbs, the lesion is due to a diffuse LMN disease.

In developing the differential diagnosis for quadriparesis, the basic mechanisms of disease must be considered along with the signalment and history. Congenital diseases are not uncommon in the cervical spinal column of dogs. These include agenesis of the dens (with resultant atlantoaxial subluxation), blocked vertebra, multiple cartilaginous exostoses, leukoencephalomyelopathy of Rottweilers, and hereditary ataxia of Jack Russell and Smooth-haired Fox terriers. In older animals, degenerative intervertebral disc (IVD) disease, inflammatory meningomyelitis and neoplasia are not uncommon. If the signs are symmetrical, then nutritional, metabolic and toxic diseases must be considered. On the other hand, most asymmetrical diseases can be separated into their most likely causes, which must be included in the differential. These causes are discospondylitis, meningomyelitis, IVD disease and neoplasia.

Diagnostic Approach:

Like the rest of the nervous system, the neurologic examination is the single most important diagnostic method to localize diseases of the cervical spine, providing an indication from which to make a tentative differential diagnostic list. On the other hand, localizing diseases in the cervical spinal column to a specific spinal segment can be difficult, since tests like the

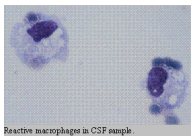
panniculus response cannot be performed there. Hyperpathia can be difficult to elicit and hyperesthesia is not easily mapped.

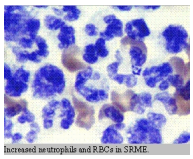
The ancillary diagnostic tests for spinal cord disease are similar regardless of the cause and include the minimum data base, spinal radiographs, EMG, CSF tap and analysis, myelography and MRI. The minimum data base will often be normal or may need to be expanded based upon the physical and neurologic examinations. In older patients, routine chest and abdominal radiographs and abdominal ultrasound may help make a diagnosis of the cervical disease or assist in making the prognosis. Spinal radiographs may show signs of degenerative disc disease, congenital malformation, spinal arthritis or discospondylitis. The later disease being the only disease diagnosis which can be made on plain spinal radiographs. The other diseases will need additional imaging techniques to confirm that they are the source of the problem. In acute diseases, the EMG may not help identify denervation until 5-7 days have past; however, nerve conduction velocity studies may help identify damaged nerves or diffuse LMN disorders. On the other hand, in chronic diseases, the EMG may help to localize the disease process, so that radiographs can concentrate on the lesion. The CSF tap can help determine the presence of inflammation or infection in cervical diseases. The problem of inflammatory myelitis is increasing, making CSF tap and analysis critical in assessing cervical neurologic disease. Even when other neurologic conditions are identified, myelitis may be present. Unfortunately, many patients are treated with corticosteroids before being adequately worked-up for cervical disease. The work-up performed in the face of the steroids may be erroneous. As such, surgical intervention may be performed, only later to discover the cause of neck pain was inflammatory meningomyelitis. Spinal myelography helps to contrast the spinal cord when looking for mass lesions. It can be an extremely valuable diagnostic aid in determining the need for surgical intervention and what surgical approach is best. In cervical vertebral malformation complex, the lesion is dynamic. The only imaging technique which can provide dynamic views is the myelogram. Myelography, therefore, remains the single most important imaging technique for assessing surgical diseases in the cervical spine. When the myelographic data is lacking or when it is not clear what the lesion represents, MRI can add diagnostic detail. MRI may be important in assessing neoplastic disease processes, including nerve root tumors. The sequence of diagnostic tests logically follows the pattern of minimum data base, EMG, spinal radiographs, CSF tap, myelography and, finally, MRI. If an accurate diagnosis is made along the way, the remaining test may not be needed.

Specific Disorders

Meningomyelitis:

As stated before, meningomyelitis appears to be on the rise. Twenty years ago, it was rare to diagnose meningomyelitis and most of these were secondary to canine distemper virus with the remainder being due to toxoplasmosis. Today, it is almost impossible to deal with animals with neck pain and not be suspicious of meningomyelitis. For this reason, even with signs of early degenerative disc disease, I do not consider surgery until I have ruled-out meningitis. While some neurologists are unconcerned about performing myelography on patients who have meningomyelitis, most contrast agents are inflammatory by nature. In the face on meningomyelitis, myelography can exacerbate the clinical signs and is, therefore, generally contraindicated in meningomyelitis.


Reactive macrophages in CSF sample


Increased neutrophils and RBCs in SRME

The clinical signs of meningomyelitis are, generally, neck pain and asymmetrical neurologic deficits. The deficits depend upon which pathways are involved in the disease process. The signs are usually progressive, but may develop acutely. In dogs and cats, the causes of meningomyelitis are, in order of likelihood, viral, inflammatory, protozoal, fungal, rickettsial and bacterial diseases. The viral disease most commonly seen in dogs is canine distemper (even in vaccinated dogs). In cats, feline leukemia virus (FeLV), feline infectious peritonitis (FIP) and feline immunodeficiency virus (FIV) are the most common viral infections. Toxoplasmosis can occur in both dogs and cats, while dog also may develop *Neospora caninum* infections. Aspergillosis is not uncommon in dogs, while cryptococcosis is more common in cats. Cats do not appear to have rickettsial diseases, but dogs have been shown to develop meningomyelitis from both ehrlichiosis and Rocky Mountain spotted fever. Titers for these agents should be performed on the serum and/or CSF when presented with meningomyelitis.

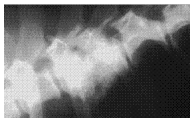
The diagnosis is made on CSF tap and analysis. Generally, we approach animals with neck pain and quadriparesis by performing a minimum data base including a CBC, chemistry profile, urinalysis and appropriate radiographs. With the CBC, we run plasma fibrinogen levels. This is a crude estimate of systemic inflammation, but a valuable tool in assessing the potential for meningomyelitis. It may be the only abnormality noted in the CBC. Once the minimal data base is evaluated, we proceed with anesthesia and CSF tap. While this is being processed, spinal radiographs are taken. If the CSF indicates inflammation by increase in cells and protein and the survey radiographs do not demonstrate significant findings, we then treat the inflammation rather than proceed with myelography. Based upon the response to therapy, we reassess the need for further tests. CSF titers are submitted for the relevant infectious agents providing

confirmation of the specific disease causing organism. In those cases where a specific disease causing organism can be found, the treatment is adjusted appropriately. When no organism is found, the tentative diagnosis of inflammatory meningomyelitis is made. Many newer forms of meningomyelitis are now recognized including steroid-responsive meningomyelitis. This is usually associated with an increase in blood vessel fragility and may lead to an apparently blood-contaminated CSF tap. On examination, however, there is a marked increase in non-degenerative neutrophils in the CSF.

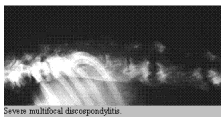
As in beagles with necrotizing vasculitis (beagle neck pain syndrome), many patients with steroid-responsive meningomyelitis have elevations in alpha 2 globulins on serum electrophoresis. Steroid-responsive meningomyelitis probably represents a form of vasculitis which results in inflammation in the CNS. Conventional therapy with corticosteroids will not always resolve this condition, since steroids only suppress the symptoms of the disease. Although some dogs recover from this disease following corticosteroid management, many would probably benefit from alternative therapy. Conventional therapy involves giving prednisolone at 1 mg/kg/day in three divided doses. Once the signs resolve (usually within 72 hours), the dosage is reduced to twice a day. This is further reduced to daily medication in the morning and, finally, to alternate day therapy. We find that many patients will benefit from anti-oxidant therapy, including vitamin E, vitamin C and selenium. Additional medications of benefit include omega-3-fatty acids, ginkgo biloba extract and green tea. When pain is present, garlic, ginger and feverfew may help reduce the inflammation without causing additional gastrointestinal signs. Some patients will be relieved by the alternative medication, reducing or replacing the corticosteroid.

Discospondylitis:

Discospondylitis represents an infection of the vertebrae associated with abscessation of the intervertebral space. It may be secondary to a migrating foreign body; but, often, no specific source of the infections is found. It is thought that, in most cases, there is a hematogenous spread of the infection which isolates into a degenerative disc. Although some cases are associated with vegetative endocarditis, most do not demonstrate a source of infection. It may be that agents enter through inflamed tissues associated with periodontal disease. In cases where there is persistent or intermittent fever, blood cultures may provide information about the infection. This is, however, less common than finding the organism in the urine.



Radiographic changes consisting of endplate destruction with lysis and sclerosis.


Severe multifocal discospondylitis.

The primary complaint in discospondylitis is pain at the site of infection. In severe cases, quadriparesis and anorexia may be present with cervical discospondylitis. The diagnosis is confirmed by routine spinal radiographs showing characteristic lysis and sclerosis of the adjacent endplates of the vertebrae. This is one of the few neurologic conditions where the diagnosis can be made on routine radiographic examination. The minimum data base includes a CBC (with a marker of inflammation such as the plasma fibrinogen level), urinalysis (with culture), fecal examination, *Brucella canis* titer, and spinal radiographs. Chest radiographs and echocardiography may be indicated if there is a heart murmur. Since the radiography changes may not occur until 2-3 weeks from the start of clinical signs, repeat radiographic examination is indicated when discospondylitis is high on the differential list. The CBC may reflect changes consistent with infection (including neutrophilia) or be normal. On of the important monitors is the marker of inflammation. We use fibrinogen, since it is easy and inexpensive to run. When the fibrinogen levels are elevated, this is a good indicator of a disease with much tissue reaction. On the other hand, when the fibrinogen is low, I am particularly concerned about the possibility of fungal disease. In the later case, I usually perform a routine chest radiograph looking for discospondylitic-like lesions between the sternebrae. When lesions are also present between the sternebrae, most often fungal infection is the cause of the discospondylitis lesions.

The causative agents are bacteria (*Staphylococcus, Streptococcus* and *Corynebacterium* are the most common, although
Brucella
can occasionally be seen as a cause), parasitic (
Spirocerca lupi
in thoracic discospondylitis), and fungal (
Aspergillus
and
Nocardia
). As such, the treatment and prognosis vary depending upon the organism causing the infection. Parasitic infections are rare except in the Southwestern US and usually represent advanced cases of parasitism.
Brucella canis
infection is not uncommon, but much less so than the other bacterial causes. When
Brucella
appears to be the cause, antibiotic therapy must take this into account (usually, I use doxycycline). Fungal infections with
Aspergillus
do not respond well to antifungal drugs. Recently, there have been reports of controlling the infection for extended period using itraconazole. I use raw garlic in hopes that it will help control

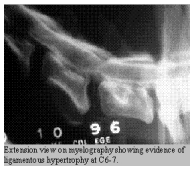
the problem.

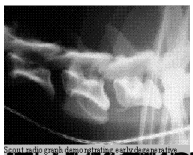
By in large, the most common causes of discospondylitis are secondary to bacteria which can be treated using a combination of sulfa drugs (sulfadimethozine, 15 mg/kg every 12 hours) and either cephalosporins (22 mg/kg every 8-12 hours) or enrofloxacin (5-7.5 mg/kg every 12 hours). I prefer the former combination and treat the infection for a minimum of 6-8 weeks. Radiographic repair usually lags behind remission of the infection; however, following the response to therapy and continuing therapy beyond the time of radiographic quiescence seem the best policy. In cases which do not respond, the urine should be reexamined and abdominal ultrasound of the kidneys performed, looking for evidence that fungal disease was the real cause. Rarely, the infection will result in bony compression or instability requiring surgical intervention. Most often, spinal cord compression is the result of soft tissue inflammation which subsides quickly with appropriate antibiotic therapy.

Cervical Vertebral Malformation Complex:

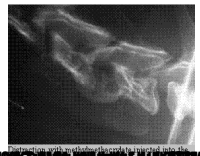
Wobbler's disease occurs in young and old animals. In young animals, it appears to be secondary to inherited malformation and mis-articulation of the cervical vertebrae which is accentuated by high protein diets. In older animals, it appears to be secondary to chronic degenerative disc disease. Although other large breeds can be affected, it is said to be a disease of young Great Danes and old Doberman Pinchers. When a Doberman Pincher presents with signs of rear leg ataxia with "root signature" in the forelegs, there is a high probability that the dog has Wobbler's disease.

The onset of clinical signs can be acute or slow and insidious. There is evidence of ataxia in all four limbs with the pelvic limbs being more affected. There will be both conscious and unconscious proprioceptive dysfunction with a wide-based stance in the rear legs. The forelegs may show a stiff and stilted gait with atrophy or fasciculations of the deltoideus, biceps and infra- and supraspinatus muscles. There is usually some degree of neck pain on palpation and neck manipulations. One sign of this is a reluctance to hop medially with the forelegs.


Extension view on myelography showing evidence of ligamentous hypertrophy at C6-7.



Screw and polypropylene washer to maintain distraction at the surgical site.



Neck brace to provide post-surgical support during the first month post-operatively.