Spinal Cord Tumors, Spinal Cord Infarcts, and Degenerative Myelopathy

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- Tumors of the Spinal Cord
- Spinal Cord Infarcts
- Degenerative Myelopathy
- References

**Tumors of the Spinal Cord**

Perhaps the feature of spinal cord tumors that distinguishes them from most orthopaedic causes of spinal dysfunction is the clinical history. Tumors of the spinal cord most often begin with very subtle deficits that slowly progress to marked dysfunction. Pain is seldom a major feature of the clinical course, although its presence or absence does not rule definitively on the existence of a tumor. It is true that occasional cases of disk prolapse will present with relatively long and progressive histories, and that a myelogram (and possibly surgery) might be required to clearly distinguish between these atypical disk prolapses and tumors in the spinal canal. In general spinal cord signs that progress over more than a 2-week period strongly suggest either tumor or degenerative myelopathy (see Table 64-1).

The nature of the progressive neural deficits depends, of course, on the location of the tumor within and along the spinal cord. Often the deficits begin or are most marked on one side or the other. For spinal tumors this is the side on which the lesion is predominantly located. As the tumor expands to occupy more space, the severity of the deficits becomes more marked and more deficits may begin to appear.

Possibly because of the slower growth, proportionately more compromise of the spinal canal seems to be required by tumors than by disk prolapse to produce equivalent clinical deficits. Consequently, if an animal's condition is severe enough to warrant myelography, the radiographic results are often diagnostic in the case of spinal tumors. On occasion, however, a tumor may so impede the flow of dye in the subarachnoid space that only one limit of the tumor may be defined by a single injection. In such situations it is a good idea to make a second injection (i.e., a lumbar injection if the first was cisternal) to define the other limit of the tumor so that the entire extent of the tumor can be determined prior to the final decision regarding the feasibility of surgery.

Other special studies such as needle electromyography (EMG) may also be used to confirm the location of spinal pathology produced by tumors. The value of these studies is dubious at best, however, since myelographic evidence is required
before surgery is contemplated. Further, the value of EMG studies in the location of focal spinal cord pathology is lessened by the fact that many older dogs have mild focal signs of denervation in the epaxial musculature that may be related to mild subclinical root disease. Needle EMG may help serve as evidence against focal gray-matter disease in certain suspected spinal tumor cases in which myelography has failed to demonstrate a space-occupying lesion (see below). Tumors of the spinal canal can be divided into three types on the basis of their location with respect to the spinal cord and dura: Extradural, outside the spinal cord; intradural extramedullary, in the dura but not invading the neural substance of the spinal cord; and intramedullary, growing within the substance of the cord. The prognosis of each case can be based on two principal factors: the anatomical type of the tumor, which can often be discerned from the myelogram, and the cell type of the tumor, which can be determined only from histopathology. A more complete discussion of tumor types has been presented by deLahunta.

Prognosis for spinal tumors is fair for those that are extradural if they are situated in an area accessible to the surgeon for removal. If the entire tumor is removable, the prognosis is dependent upon the histologic tumor type. Unfortunately, many of the extradural tumors are metastatic in nature. Intramedullary tumors are essentially inoperable and have a poor prognosis.

**Spinal Cord Infarcts**

Spinal cord infarcts represent another form of spinal pathology that can easily be confused with disk prolapse. The feature that most often distinguishes such cases from those of disk prolapse is the absence of pain associated with the rapid onset of the signs of spinal cord dysfunction. According to deLahunta, spinal ischemia due to fibrocartilaginous embolization occurs most often in the cervical spinal cord. It can occur in lower sections of the cord as well.

The characteristic presentation of the disease is one of sudden onset of spinal cord signs without considerable evidence of pain associated with it. Often the onset of signs is preceded by a period of moderately vigorous exercise. In those animals in whom the lesion is located in the cervical cord, deficits in the forelimbs may be much more marked than those in the hindlimbs, suggesting that the ischemic area includes more of the central gray matter than of the peripheral white matter of the cord. The condition is usually nonprogressive after the first few hours.

Myelography, unless done very soon after the initial insult to the cord, usually reveals nothing, and surgical intervention is contraindicated. Most dogs improve within a week to 10 days. Few become perfectly normal, but many regain adequate spinal cord function. There is no specific treatment for the condition other than supportive therapy during the convalescence.

**Degenerative Myelopathy**

Occasionally, although clinical signs suggest either an atypically slow disk prolapse or spinal cord tumor, the myelogram will fail to demonstrate the presence of a compromised subarachnoid space even though the neurologic examination has convincingly demonstrated abnormalities indicative of a well-defined spinal cord lesion. Needle EMG also, if used in these cases, generally fails to confirm the existence of spinal motor pathology. Under such circumstances a presumptive diagnosis of degenerative myelopathy is warranted although a positive diagnosis requires histologic confirmation.

Degenerative myelopathies comprise a set of diseases of the spinal cord that are diagnostically frustrating and therapeutically impossible. Perhaps the most notorious form of this condition is that which affects aging German shepherds and which has been well described by Averill. However, the condition clearly affects dogs of other breeds as well as mongrels. It is also not clear whether there may be several forms of the condition that are histopathologically distinguishable even if they are clinically similar. Generally the time course of the disease prior to presentation is of a slowly progressive nature, making the condition difficult to distinguish from spinal cord tumor. Signs can be variable but are most often reflective of a partial loss of spinal cord function between the segments of the brachial and lumbar intumescences. Signs generally include gait abnormalities of the hindlimbs, which appear as either weakness or ataxia; proprioceptive deficits in the hindlimbs; normal to slightly depressed pain sensation in the hindquarters without apparent bowel or bladder dysfunction; and normal to exaggerated hindleg myotactic reflexes. Perhaps the most consistent finding is a disproportionate loss of proprioception relative to other sensory modalities.

We have seen several cases (not German shepherds) in which the presenting signs suggested a spinal lesion at or very near the cord segments contributing to the femoral nerve. In many of these cases the signs suggested a laterality to the lesion in that one hindleg appeared to be more severely affected than the other. In two of the cases it was believed that the lesion was
located exactly in the area of the cell bodies comprising one femoral nerve because the signs in that limb included depressed knee-jerk reflexes with exaggerated reflexes caudally on the more severely affected side and slightly exaggerated reflexes, including the knee-jerk, on the opposite side.

A presumptive diagnosis of degenerative myelopathy requires a myelogram without gross abnormalities. In some cases, in which the progression of signs warrants it, a second myelogram at a later date might be indicated to rule out a spinal cord tumor that was too small to be detected on the original. Generally speaking, however, tumors that are large enough to produce signs detectable by the gross neurologic examination are large enough to be detected by myelography. In some circumstances, in the German shepherd in particular, the expense and danger of myelography coupled with the expense of possible surgery may preclude its performance.

In the German shepherd the condition is fairly well defined. Age of onset of the disease may be as early as 5 years but generally is from 7 to 9 years. The signs are often initially unilateral and progress to include the other hind leg. There is a marked difference in the degree of deficit in proprioceptive and pain sensations, the former being much more severe. Bowel and bladder control is almost never affected even though the animal may be only barely capable of supporting himself in the hindquarters.

Histopathologic changes are related predominantly to the white matter of the cord. They are most severe in the thoracic area but can be found in other areas as well. (1) In some cases the central gray matter may also be involved, which may account for signs related to specific peripheral nerves in some cases.

Prognosis for the degenerative myelopathies is poor in that there is no known effective treatment for either reversal of the signs or abatement of their progression. Although there are no published controlled experiments relating to therapy for these conditions, we have tried massive corticosteroid therapy, multiple vitamin and mineral supplementation, dietary manipulation, and acupuncture, all to no avail. The signs of neural deficiency tend to progress, and the victims of the condition become less and less able to ambulate. The time course of this progression is variable, and it can take from several weeks to many months for minimal signs to progress to complete disuse of the hindquarters.

References


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