ACUTE SPINAL CORD INJURIES - WHEN NEUROSURGERY IS NOT THE ANSWER.

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Acute spinal cord diseases are common in veterinary clinics. The presentation of the clinical signs can be hyperacute (<6 hours) or acute (7-24 hours) but not all require surgery. It is important to understand the different causes and therefore identify situations where it is not necessary to go into the operating room. This talk will focus on the most common causes of spinal cord acute disease where the treatment is not based in neurosurgery. We will focus on recognizing the clinical signs consistent with an acute disease of the spinal cord and also in the clinical signs that may indicate the possibility that the underlying cause does not require surgical treatment.

Neurological examination and neuroanatomical localization

The clinical approach in animals presenting with an acute spinal disease begins with a detailed medical history (signalment, onset, progression of clinical signs, pain), unless the patient has suffered a traumatic event, where priority is focused on the patient stabilization. Anamnesis is followed by a general and neurological examination. The aims of the neurological examination are to establish an anatomical diagnosis (neurolocalization), assess the severity of neurological dysfunction and predict prognosis (in some cases). Animals with spinal cord disease have a normal mentation without alteration of the cranial nerves unless the cause may affect different regions of the nervous system and does not only cause myelopathy. After assessing the posture and movement / locomotion, postural reactions, spinal reflexes, muscle tone, sensitivity and palpation of the vertebral column, the lesion may be located in one of the four regions in which the spinal cord is functionally divided: Spinal cord segments C1-C5, C6-T2, T3-L4-S3 and L3 (Fig. 1). Neurolocalization can be more challenging in patients presenting with Schiff-Sherrington syndrome or the spinal shock syndrome. The Schiff-Sherrington posture (Schiff-Sherrington syndrome) may be seen in patients presenting with acute and severe lesions in T3-L3 spinal segments. This is a pathological posture that is observed when the patient is in lateral recumbency. It is characterised by hypertonia and extension of the thoracic limbs with a normal motor function and hypotonia of the pelvic limbs and a severely compromised motor function (fig. 2). Spinal reflexes are normal in the thoracic and pelvic limbs unless accompanied by spinal shock (in this case the spinal reflexes in the pelvic limbs may be decreased).

The Schiff-Sherrington posture is caused by an acute transient loss of function of the neurons that inhibit the extensor muscles of the thoracic limbs (known as border cells, located in the spinal cord segments...
L1-L5) (fig. 3). This posture resolves spontaneously within some days (10-14 days) and has no prognostic value even though it is due to an acute and severe lesion.

**Spinal shock** is a transient loss of muscle tone and spinal reflexes in hind limbs after an acute and severe spinal injury. This phenomenon occurs due to an acute loss of the stimulus for neural descending facilitating function, causing hyperpolarization and dysfunction of motor neuron excitability in the intumescences that causes a loss of spinal reflexes and hypotonia. In dogs, the first reflex that re-emerges is the perineal reflex, followed by the patellar and flexor reflexes in about 24-48 hours. Hypotonia of muscles may persist for 10 to 14 days, and is replaced by an increase in muscle tone. The area of spinal pain (where present) and the evaluation of the cutaneous trunk reflex can help localizing the lesion in case of acute diseases of the spinal cord with the presence of spinal shock.

Severe injuries of the spinal cord segments C1-T3 can cause lesions in the tectotegmental spinal tract and result in the sympathetic denervation of the ipsilateral eye (Horner's syndrome) (Fig.4)

**Vertebral fractures / luxations (VFL)**

VFL are a frequent cause of acute myelopathy in dogs and cats. Neuronal tissue damage is due to a primary mechanical damage in the parenchyma and associated vessels (contusion, compression, laceration, hemorrhage, and ischemia) and to a secondary damage characterized by biochemical alterations (cell failure, altered membrane permeability, production of free radicals, ex cytotoxicity, oxidative damage and inflammation) resulting in cellular necrosis. VFL’s etiology can be traumatic when there is an external force (road accident, horse kick, falls) or pathological when there is an abnormal bone tissue (primary or secondary neoplasia, hyperparathyroidism, osteomyelitis, discospondylitis). VFL are considered unstable when two or more of the 3 spinal compartments are affected. Unstable fractures can cause repetitive motions and more damage to the spinal cord.

Clinical signs due to VFL depend on the location and the severity, and the possibility of damage affecting other organs. Spinal hyperesthesia is commonly present. Animals where VFL is suspected must be immobilized in lateral recumbency on a rigid table and should be handled very carefully to avoid spinal cord damage. Radiography has sensitivity around 75% for the detection of VFL. The CT scan provides excellent bone detail and can identify VFLs not visible on plain radiographies. Magnetic resonance (MR) provides detailed information about the status of the spinal cord. Myelography should be avoided in animals with VFL because it can cause seizures and impaired neurological function. The initial treatment should focus on the perfusion of the spinal cord (blood pressure and oxygenation), external stabilization of the column (fig. 6) and the administration of analgesia. There is no scientific evidence that supports the use of glucocorticoids (including methylprednisolone) in spinal cord injuries. In stable VFLs (based on fig. 5), the **conservative treatment** (6 to 8 weeks of strict rest) may be indicated. Surgical treatment is indicated if the fracture is unstable or in case of spinal cord compression (disc extrusion or hemorrhage).

**Disc herniation - Acute lumbar disc herniation**

Acute disk extrusions may be due to Hansen type I extrusion of the degenerated nucleus pulposus or to the extrusion of the non degenerated nucleus pulposus (compressive or non-compressive). Disc extrusions of the degenerated nucleus pulposus (Hansen Type I) primarily affect chondrodystrophic breeds, but any canine breed can be affected. Clinical signs depend on the affected spinal segments and the severity of spinal cord compression (myelopathy C1-C5, L3-T3 o L4-S3). Diagnosis is based on imaging modalities such as myelography, computed tomography (CT) or MRI to confirm or to rule out the initial suspicion and to locate the level and severity of spinal cord compression. The use of the MR has allowed identifying acute extrusions of not degenerated nucleus pulposus that are probably associated with mechanical and supra-physiological stress during physical exercise. A portion of the nucleus pulposus goes explosively through the annulus fibrosus and bruises the spinal cord. This displacement and the spinal cord damage can be seen with the MR. This type of disc extrusion may be compressive or...
non-compressive. The **conservative treatment** is indicated in animals with minimal neurological dysfunction and **minimal spinal compression** (strict rest for 4 to 8 weeks) and analgesia. Surgical treatment (Hemilaminectomy, dorsal laminectomy, ventral slot, pediculectomy, Mini-hemilaminectomy) is indicated in animals with spinal cord or nerve roots compression and progressive neurological deficits. Patients with minimal neurological deficits and/or simply spinal pain but moderate nerve roots/spinal cord compression unresponsive to conservative treatment could also be considered surgical patients. Prognosis depends on the clinical signs, diagnostic findings and the type of treatment used. The presence of nociception before conservative or surgical treatment is crucial to consider a good recovery prognosis.

Acute non-compressive nucleus pulposus extrusions (ANNPE) are characterized by acute, generally lateralized, painless and non-progressive symptoms. Some patients exhibit pain upon palpation during the first hours after the extrusion. The extruded material (not degenerated nucleus pulposus) causes bruising to the spinal cord and extends into the epidural space without significant spinal cord compression. The presumptive diagnosis is made by advanced imaging tests (MR> CT). There is no evidence of the benefit of drug treatment for these cases. The treatment involves restriction of exercise and rehabilitation for 4-6 weeks. If there pain upon palpation, anti-inflammatory drugs may be given for a few days. Prognosis is favourable for most cases (67%). The severity of the neurological deficits on the initial examination and the the extent of the injury as seen on the resonance imaging can help predicting the prognosis.

Acute non-compressive hydrated nucleus pulposus extrusion HNPE are characterized by acute, severe (ambulatory tetraparesis/quadruplegia/ambulatory paraparesis/paraplegia), normally not lateralized, not painful and non-progressive symptoms. Some patients exhibit pain upon palpation during the first hours after the extrusion. The extruded material (not degenerated nucleus pulposus) causes bruising to the spinal cord and extends into the epidural space, normally ventral to the spinal cord, causing moderate/severe compression. The presumptive diagnosis is made by advanced imaging tests (MR> CT). There is no evidence of the benefit of drug treatment for these cases. However, even with moderate compression, most patients improve with conservative treatment within 24-48 hours. The conservative treatment involves restriction of exercise and rehabilitation for 4-6 weeks. If there pain upon palpation, anti-inflammatory drugs may be given for a few days. Prognosis is favourable for most cases. The severity of the neurological deficits on the initial examination and the the extent of the injury as seen on the resonance imaging can help predicting the prognosis.

**Ischemic myelopathy - Fibrocartilaginous embolism (FCE)**

Myelopathy caused by FCE is considered the most common cause of ischemic myelopathy in dogs and cats. It is caused by the embolisation of a piece of material that is histologically equal to the nucleus pulposus into one of the intrinsic spinal cord arteries, which cause ischemia of the area irrigated by the artery and associated clinical signs. It occurs most commonly in adult dogs, but is described in dogs from 3 months to 13 years of age. Clinical signs are characterized by presentation of hyperacute myelopathy (may affect any section of the spinal cord) (<6 hours) non-progressive after 24 hours, not painful and commonly lateralized. The diagnostic test of choice for the presumptive diagnosis is MRI, but definitive diagnosis is based on histopathology. The treatment is based on maintaining the perfusion of the spinal cord (oxygenation and blood pressure) and intensive physiotherapy. The prognosis is generally favourable in animals with nociception. If the intumescences are affected, a permanent neurological dysfunction is possible.

**Meningomyelitis**

Meningomyelitis is an inflammation of the spinal cord and the meninges. Meningomyelitis occurs commonly with accompanying inflammation of the brain (meningoencephalomyelitis), resulting in a multifocal or diffuse localization that affects the nervous system. The most common causes include Feline Infectious Peritonitis, canine distemper virus, bacteria, protozoa (Neosporosis, Toxoplasmosis), fungal
myelopathies (Cryptococcus, Histoplasmosis and Coccidioidomycosis) and parasitic (spinal spirocerosis), and granulomatous meningomyelitis. Steroid responsive meningitis arteritis is the most common cause of meningitis in dogs and occasionally affects the parenchyma of the spinal cord. The diagnosis is based on Imaging tests (MR), cerebrospinal fluid analysis, and serology/PCR/culture for certain infectious agents. The treatment is based on the underlying cause and the prognosis is variable depending on the etiology, the extent and severity of the damage in the meninges and neural tissue.

Readings


