One of the more confusing neurolocalizations is that of diffuse Lower Motor Neuron (LMN) disease. These patients frequently are referred for either cervical spinal cord or intracranial disease because of the presence of clinical signs in all four legs. This newsletter will focus on the four primary differential diagnoses for dogs that present with an acute, rapidly progressive LMN tetraparesis. In addition to the information presented here, video case examples are available on our website at www.intownvet.com/intown/newsletters.html.

**Basic Neuroanatomy**

Before discussing each of these diseases, a quick refresher on Upper Motor Neurons (UMN) versus Lower Motor Neurons (LMN) may be useful. UMN cell bodies are located in the brain and their axons synapse on LMNs at various levels of the brain and spinal cord. These UMNs are generally excitatory to flexor muscles and inhibitory to extensor muscles. Since people and animals rely predominantly on the extensor muscles to stand and walk, diseases affecting the UMN system are detected as a lack of inhibition on the LMN, leading to increased tone and exaggerated spinal reflexes in the limbs caudal to the lesion. LMNs exist at all levels of the spinal cord and innervate skeletal muscles. The LMNs that are most important clinically are those that give rise to peripheral nerves of the limbs originating in the cervical (C6-T2) or lumbar (L4-S3) intumescence. Diseases affecting the LMN will result in weakness to flaccid paralysis of the muscles.

Distinguishing UMN from LMN disease (Table I) is achieved by performing a thorough neurological exam. Test the segmental spinal reflexes (e.g., patellar reflex) and withdrawal in all four legs, assess the degree of muscle tone and palpate for evidence of muscle atrophy. Following the neurological exam, one should be able to more accurately localize the lesion to the spinal cord or peripheral nervous system (Table II). If LMN signs are present in all four legs, a diffuse LMN disease is most likely. It is possible, although uncommon, to have diffuse spinal cord disease (e.g., inflammation/infection) that is affecting both intumescences giving rise to LMN signs in all four legs.

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**Table I: Distinguishing UMN signs from LMN signs**

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<thead>
<tr>
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<th>UMN</th>
<th>LMN</th>
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<tbody>
<tr>
<td>Spinal Reflexes</td>
<td>Normal to Exaggerated</td>
<td>Decreased to Absent</td>
</tr>
<tr>
<td>Withdrawal</td>
<td>Normal</td>
<td>Decreased to Absent</td>
</tr>
<tr>
<td>Muscle Tone</td>
<td>Normal to Increased</td>
<td>Decreased to Absent</td>
</tr>
<tr>
<td>Muscle Atrophy</td>
<td>Late (Disuse)</td>
<td>Early (Denervation)</td>
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<tr>
<td>Postural Reactions</td>
<td>Decreased to Absent</td>
<td>Decreased to Absent</td>
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Differential diagnoses for acute, rapidly progressive LMN tetraparesis

1. Acute Idiopathic Polyradiculoneuritis / Coonhound Paralysis
2. Tick Paralysis
3. Botulism
4. Acute, Fulminant Myasthenia Gravis

Acute Idiopathic Polyradiculoneuritis / Coonhound Paralysis

Acute Idiopathic Polyradiculoneuritis is an inflammatory disorder that primarily affects the axons and myelin of the ventral roots. The inciting cause is unknown, but theories include recent illness or vaccination, immune-mediated disease, and viral infection.

Coonhound Paralysis appears to be an identical clinical disease in dogs that have been exposed to a raccoon bite or scratch 1-2 weeks prior to the onset of clinical signs. It is thought that an antigen in the raccoon saliva triggers the condition. In both diseases, the history typically reflects a sudden onset of hind limb weakness that rapidly progresses to nonambulatory tetraparesis within 48-72 hours.

Neurological examination reveals typical LMN signs as described above in the affected limbs with flaccid tetraparesis or tetraplegia, decreased to absent reflexes, and decreased to absent withdrawal. Loss of voice or change in character of the bark is common and some patients will have facial weakness. Urinary & anal sphincter tone usually remain normal and these patients typically do not become incontinent. Sensory function remains intact in the limbs. In fact, some patients appear hyperesthetic when the limbs are manipulated. Postural reactions may be normal if sufficient muscle strength remains, but they often are absent. The clinical signs can progress for up to 1 week after onset.

Diagnosis is based on history, potential exposure to raccoons and neurological exam findings. Electrodiagnostics can help differentiate this disease from the others listed below. A special type of nerve conduction (F wave) that looks at transmission through the ventral roots often is abnormal. Cerebrospinal fluid (CSF) often demonstrates an elevated protein level.

There is no specific treatment for this condition other than time, nursing care (e.g., hydration, nutritional support, prevention of pressure sores) and intense physical therapy. Glucocorticoids have not been shown to shorten the duration of disease or improve the degree of recovery. In fact, glucocorticoids cause neuromuscular weakness at higher doses making it difficult to assess the patient’s degree of recovery.

The prognosis for most patients is good to excellent. However, recovery may take several weeks to months. The most important monitoring parameter is ventilation as some patients develop a severe, potentially life-threatening, respiratory paresis due to involvement of the phrenic nerve and intercostal nerves. These patients may need to be mechanically ventilated.

After recovery, avoid exposure to raccoons or other factors that may have triggered the clinical signs as survival does not confer immunity.

Tick Paralysis

Tick Paralysis is characterized by a flaccid, rapidly ascending LMN paralysis in dogs following exposure to a salivary neurotoxin released by adult ticks. Many species of ticks have the potential to produce Tick Paralysis, but the tick species that are most important in the United States are Dermacentor variabilis (American dog tick, common wood tick) and Dermacentor andersoni (Rocky Mountain wood tick). This disease occurs only after exposure to gravid female ticks.

Affected dogs typically begin displaying clinical signs 5-9 days after tick attachment. They usually present with paraparesis that rapidly progresses to tetraparesis within 12-72 hours. The clinical signs and neurological exam findings are indistinguishable from Idiopathic Polyradiculoneuritis/Coonhound Paralysis.

Diagnosis is based on removing an engorged tick followed by quick resolution of clinical signs. Failure to find a tick does not rule out Tick Paralysis as the tick may have dropped off. In most cases, we recommend shaving the entire dog as it can be extremely difficult to find all of the ticks. DO NOT STOP LOOKING after finding an engorged tick as there is often more than one tick on the dog.

Be sure to use forceps or a hemostat to remove the entire tick including the mouthparts. If the owner does not allow you to shave the dog, the patient should be treated with an acaricide (tick dip). Frontline or similar spot-on tick treatment SHOULD NOT BE USED AS THE SOLE TREATMENT because these medications take 36-48 hours to take effect.
In severe cases, respiratory paralysis or aspiration pneumonia may occur during this time. Tick serology does not need to be performed. A positive test only shows prior exposure to a tick-borne disease and is not diagnostic for Tick Paralysis.

Removal of the offending tick generally results in a rapid recovery. Improvement often is noted within hours of tick removal and full recovery can occur within 2-3 days. In the meantime, provide good nursing care, including physical therapy, prevention of pressure sores, pneumonia, and maintain hydration and nutritional status.

**Botulism**

Botulism is caused by ingestion of a preformed exotoxin produced by *Clostridium botulinum*. The toxin usually is ingested in uncooked, spoiled food, or carrion. In rare cases, the organism produces the exotoxin after colonization of the gastrointestinal tract (GIT).

Eight types of botulinum toxin have been identified, with type-C1 being the most common toxin found in dogs. After absorption from the upper GIT, the toxin enters the circulatory system and eventually binds to cholinergic synapses, including the neuromuscular junction (NMJ). The toxin blocks the release of acetylcholine (ACh) at both NMJ and cholinergic synapses in the autonomic nervous system (ANS), which in turn leads to flaccid paralysis and dysfunction of the ANS.

Onset of signs is rapid, beginning within 12 hours of ingestion. As with the diseases listed above, the clinical signs often appear as a progressive weakness to flaccid paralysis starting in the pelvic limbs and rapidly progressing to the thoracic limbs. Pain perception in the limbs remains intact. In severe cases, respiratory paralysis can lead to patient death.

Unlike the other LMN diseases, cranial nerve deficits are common, including facial nerve paralysis, decreased gag reflex, decreased jaw tone, and megaesophagus. ANS signs also are common, including altered heart rate (increased or decreased), dilated pupils with decreased pupillary light reflex, keratoconjunctivitis sicca, urinary retention, and constipation.

Diagnosis is based on clinical signs and potential exposure. Definitive diagnosis requires identification of the toxin in blood, feces, vomitus, or food. Electrodiagnostics can be helpful in distinguishing botulism from other diseases.

Treatment primarily consists of supportive care involving routine nursing care (thick bedding, prevention of bed sores, physical therapy, and bladder and bowel care (enemas and bladder expression or catheterization). Antitoxin can be given in early cases (within 5 days), or in severe cases, but often the toxin has already been internalized into neurons and is inaccessible to antitoxin. If giving antitoxin, perform an intradermal test injection prior to giving the full dose to test for potential anaphylactic reaction. Patients do not develop immunity to the toxin, but repeat exposure is rare.

**Acute, Fulminant Myasthenia Gravis**

This is an uncommon form of Myasthenia Gravis (MG) in which there is a severe and rapidly progressive form of generalized muscle weakness. Acquired MG is caused by antibodies directed against the ACh receptor on skeletal muscle, resulting in decreased receptors available for ACh binding and subsequent muscle weakness.

Patients with this form of MG often present in lateral recumbency and are nonambulatory tetraparetic. Aspiration pneumonia is common due to the presence of laryngeal and pharyngeal weakness. Unlike the three diseases listed above, these patients often look very ill.

Definitive diagnosis is based on demonstration of anti-ACh receptor antibodies in serum. A Tensilon Test (edrophonium) can be performed as a rapid in-hospital screening test, but these patients frequently have a false negative test due to the severity of the weakness.

Thoracic radiographs should be taken to screen for megaesophagus, aspiration pneumonia and presence of a thymoma (which causes a paraneoplastic MG). Routine screening tests, such as complete blood count, biochemical profile and abdominal ultrasound, often are normal, but should be performed to search for an underlying cause of MG.

Treatment involves early and aggressive intensive care with anticholinesterase therapy (oral pyridostigmine or injectable neostigmine), potentially immunosuppressive medications (used very cautiously if aspiration pneumonia is present and with the knowledge that steroids can cause additional neuromuscular weakness), IV fluids and nutritional support. In human medicine, plasmapharesis and/or intravenous immunoglobulin have been used. This is used infrequently in veterinary medicine due to cost and limited availability. This form of MG has a high mortality rate, but fortunately it is the least common form of MG.
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