Washington State University Veterinary Hospital
Progress Report

115262 Riggs
Olsen, Michael
H:(509)592-0006 W:(509)592-6676
Canine –Welsh Corgi, Pembroke
10/10/95 M-C Brown & White

Appetite (A)	Bowel Movement (BM)
N= normal	N= normal
F= fair	A= abnormal
P= poor	0= none
0= none	

Date	A	BM	Temp	HR	RR	
6/07/07	N/A	N/A	101.3	96	40	**Case Summary:**

History: Riggs presented to WSU-VTH as a referral from Dr. Smith with Valley Veterinary Clinic. He came in with ascending flaccid paralysis beginning Monday June 4, 2007 in the rear limbs with ataxia and unwillingness to use his legs. By early Tuesday morning Riggs was thrashing all four limbs. The disease progressed to unwillingness to use all four limbs. Riggs was unable to stand upon presentation and was quiet and depressed. Riggs has no history of trauma or toxin ingestion and has not been on flea and tick preventive. Mr. and Mrs. Olsen have noticed ticks on their other 2 Corgis but none on Riggs.

Is this really the best term to use? What kind of disease processes/ pathophysiologic mechanisms are implied by this term?

Physical exam:
Weight: 15.2 kg
Body condition score: 4/5
Temperature : 100.0 F
Respirations: 30 breaths per minute
Heart rate: 64 beats per minute
Mucous Membranes: Pink
Capillary Refill time: <2 seconds
Riggs is quite alert and responsive, eyes ears nose and throat are all within normal limits. Auscultation of the heart reveals a normal sinus arrhythmia, lungs auscult within normal limits. Abdominal palpation is unremarkable. There is a firm mass on Riggs' back, it is round and firm to the left of midline over the L1/L2 vertebrae. Mr. Olsen believes it in the same area as a mass that was removed years back. There is a soft round mass on the right flank just caudal to the ribs. Riggs had a severely inflamed anus and perianal region. He was painful when a rectal was performed and the glove came out with soft feces and fresh blood. No other abnormalities were noted on physical exam.

The tick?

Neurological Exam:
Decreased palpebral reflex (0.5/2)
Decreased menace (1/2)
Decreased facial sensation (1/2)
Unable to stand
Absent cutaneous proprioception rear limbs (0/2)
Absent cutaneous proprioception right front limb (0/2)
Decreased conscious proprioception left front limb (1/2)
Absent cutaneous trunci reflex
Voluntary bladder, but unable to stand to release
Deep pain present in all four limbs and tail
All other aspects of neurologic exam normal
Localization: diffuse lower motor neuron

Problem List:
1: Diffuse Lower Motor Neuron Disease

NOTE: Comments in this style of box record handwritten feedback from Dr. R. Sellon

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						2: Firm Mass over L1/L2, soft mass right side 3: Bloody Diarrhea
						Problem 1: Diffuse Lower Motor Neuron Disease **SO:** Riggs presented to WSU-VTH with diffuse lower motor neuron disease referred to us by Dr. Smith with Valley Veterinary Clinic. This began on Monday June 4, 2007 in the rear limbs with ataxia and unwillingness to use legs. By early Tuesday morning Riggs was thrashing all four limbs. The disease progressed to unwillingness to use all four limbs. Riggs was unable to stand upon presentation, quiet and depressed. **A:** Diseases that affect the peripheral nerves and cell bodies result in only a few anatomical or physiological alterations in the neuronal cell body, axon or synapse. Ultimately there is a disruption of neural impulse generation or a destruction of neural elements. The four most common diseases to cause ascending paralysis in dogs are tick paralysis, acute myasthenia gravis crisis, coonhound paralysis (acute polyradiculoneuritis) and botulism. Tick paralysis is the most likely differential in this case due to the clinical signs of ascending flaccid paralysis, diffuse lower motor neuron signs and presence of an engorged female tick. There are four different species of ticks in the United States that have been documented to cause tick paralysis and one in Australia. *Dermacentor variabilia, D. andersoni, Amblyomma americanum* and *A. maculatum* are all found in the United States. *Dermacentor andersoni* is the tick found from the Cascades to the Rocky Mountains, the region where Riggs lives. In Australia *Ixodes holocylus* is the tick that cause tick paralysis. The exact neurotoxin secreted by *D. andersoni* is unknown; however it is similar to the holocyclotoxin secreted by *Ixodes holocyclus* in Australia. This neurotoxin is a presynaptic neurotoxin which inhibits the release of acetylcholine at the neuromuscular junction and is secreted in the saliva of the tick. If left untreated the ascending paralysis progresses to respiratory failure and is fatal. However removal of the tick results in recovery from paralysis in 24-72 hours. Paralysis begins 5-7 days after the tick attaches to the animal. Coonhound paralysis or polyradiculoneuritis causes similar clinical signs and is often associated with raccoon saliva, however there have been reports of coonhound paralysis without exposure to raccoon saliva and it is thought to be mediated by antigenic substances that trigger an immune mediated event. However, in coonhound paralysis animals often present with hyperesthesia, which was not seen in this case. Botulism works in a similar manner as the tick paralysis toxin in that it inhibits the release of acetylcholine and prevents the activation of lower motor neurons. Clinical signs are similar to tick paralysis and diagnosis is based on history of ingestion of carrion or spoiled food stuff (ingestion of the toxin elaborated by *Clostridium botulinum*) as well as exclusion of other lower motor neuron diseases. Riggs does not have history of eating spoiled food stuffs. Finally acute myasthenia gravis can cause an ascending paralysis. Myasthenia gravis is caused by either a lack of acetycholine receptors at the motor end plate (congenital) or antibodies made towards the nicotinic acetylcholine receptors at the motor endplate causing a decrease in functional receptors. Often the signs of myasthenia gravis are brought on by exercise leading to weakness; spinal reflexes

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stay intact. However, in the acute fulminating form of the disease it can be acute onset of lower motor neuron weakness and loss of reflexes. In light of the clinical signs associated with presence of a tick, myasthenia gravis is a less likely differential.

P: Monitor Riggs for the next 24-72 hours for recovery from paralysis. If paralysis worsens or remains unchanged consider other differentials, possibly run an acetylcholine antibody test or tensilon test for myasthenia gravis. Look at electromyography for coonhound paralysis, electrophysiology of neuromuscular tissue for coonhound paralysis or botulism. In this case due to the presence of ascending paralysis, diffuse lower motor neuron disease as well as presence of an engorged tick the most likely diagnosis is tick paralysis. Tick paralysis is of importance to public health because it affects man as it does animals. According to JAVMA zoonosis is defined as those diseases and infections, which are naturally transmitted between vertebrate animals and man. Tick paralysis does not fit this definition; however many think zoonoses should include the traditional definition as well as diseases induced by non-infective agents such as toxins and poisons – thus tick paralysis would be considered a zoonotic agent. Essentially tick paralysis is of importance to the general public, veterinarians and the animals they treat.

Is a muscle biopsy likely to confirm a dx of coonhound paralysis?

Excellent assessment of this problem.

Problem 2: Firm Mass
SO: A 1.5 cm in diameter firm mass above L1/L2 lumbar vertebrae to the left of midline was palpated during physical exam. This mass may or may not have been removed in the past.

Is it in the skin or underneath the skin?

As well as others

A: A firm mass in the skin of a dog could be a sebaceous adenoma, histiocytoma, squamous cell carcinoma or mast cell tumor. After palpating the mass it did not change in size making a mast cell tumor less likely. Often palpating a mast cell tumor will elaborate histamine and cause inflammation. Likely this mass is a sebaceous adenoma, one of the most common skin tumors of dogs, but without a fine needle aspirate or biopsy there is no way to tell. The soft mass is likely a lipoma.

Does this commonly happen w/ MCTs?

P: recommended fine needle aspirate of masses and cytology.

Problem 3: Bloody Diarrhea
SO: Bloody stool was seen on a glove after a rectal was preformed on Riggs. His anal and perianal area was warm, red and swollen, all indications of inflammation. The blood was not bright red but not black or tarry. Riggs has been on Dexamethazone at a dose of 2mg/kg.

Or distal small bowel.

A: Fresh blood in stool is indicative of rectal, perianal or colon bleeding. This results in what is called hematochezia or fresh blood in the stool. Blood originating in the gastrointestinal tract cranial to the colon results in melena or dark blood in the stool. This is due to the bacterial digestion of the red blood cells in the gastrointestinal tract. Hematochezia can be caused by trauma to the anus, colon

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						or rectum, colitis, proctitis, parvovirus, parasitism (whipworms, coccidian, round worms or hookworms) or neoplasia. Melena can be caused by disease of the pharynx, lungs (coughed up and swallowed), esophagus, stomach, small intestine and even the large bowel if transit time is slow enough. Taken into context with this case Riggs was on 2mg/Kg dexamethasone. Dexamethasone is the most potent of all commonly administered glucocorticoids and the most likely to cause gastrointestinal tract ulceration. Dexamethasone increases acid, pepsin and trypsin secretion, decreases mucus and bicarbonate production and has an effect to decrease prostaglandin production all leading to gastric ulceration. Likely in this case Riggs has gastrointestinal tract ulceration leading to bloody stools. **P:** discontinue dexamethasone, continue on 10mg Q12 hours famotidine, an H2 receptor antagonist that acts on parietal cells to reduce gastric acid formation. Continue to monitor Riggs' defecation for an improvement. Monitor mucous membranes looking for pale mucous membranes which could indicate excessive blood loss. Likely Riggs will improve with cessation of the dexamethasone. Signature of Student
6/08/07 6am	N/A	N/A	100.6	88	Panting	**Case Summary:** **Physical Exam:** Weight: 15.2 kg Body Condition Score: 4/5 Temperature: 101.6 F Respirations: panting Heart rate: 128 beats per minute. Mucous Membranes: pink Capillary Refill Time: <2 seconds Riggs is bright alert and responsive, eyes ears nose and throat are all within normal limits. Riggs' heart and lungs auscult within normal limits. Abdominal palpation is unremarkable. Riggs has a mass on his back just to the left of midline over L1/L2 vertebrae 1.5 cm diameter small firm. There was some blood on the thermometer after taking his temperature per rectum however his anus and perianal area while still inflamed is not swollen and inflamed. No other abnormalities were noted on physical exam. ***S/O*:** Riggs is bright alert and responsive this morning. He was able to get up and walk outside to urinate! He continues to have a slight ataxia. ***A*:** Riggs is now walking which is wonderful! He likely had tick paralysis treated with tick removal. See previous assessments of bloody stool and mass. **P:** Continue to monitor Riggs throughout the day, take him on short walks as he may still tire easily. Continue on 5mg famotadine tablets Q12 hours. Signature of Student

To put this dose into context, do you have a feel for what an equivalent dose of prednisone would be?

There is an association between colonic ulcers and dexamethasone tx in dogs w/ certain neurologic dz—is this dog an "at risk" patient?

Shows the importance of a really good PE, doesn't it?

Nice SOAP.

--R. Sellon