Aspen Meadow Veterinary Specialists

Veterinary Voice

September 2012, Issue 47

AMVS Event Updates!

September was a great month for Aspen Meadow Veterinary Specialists. The Homeward Bound Harvest had a great turn out and raised a whopping $113,000 dollars for the Longmont Humane Society. The Longmont artwalk was great as well, despite the darkness there was a great turn out and the kid's especially enjoyed the glow sticks! It's great to have the opportunity to catchup with our local patients and their parents.

Upcoming Events!

Saturday, October 20th
AMVS will be sponsoring Wildlands Restoration Volunteers (WRV) in the aid of the St. Vrain Lafarge project. Volunteers will spend the day removing invasive Russian Olive Trees, creating fish habitat reef structures and planting 400 to 500 native Riparian Trees.

Wildlands Restoration Volunteers

To join this venture click on the logo above to sign up on WRV's website.

In This Issue

In addition to our regular ER hours, AMVS provides emergency and critical care services to your patients **Fridays, all day CALL** 303-678-8844

AMVS is: PACE certified, LEED certified, and a zero-waste facility.

Upcoming CE's

Animal Emergency and Critical Care is offering the first of many planned CE opportunity's

This lecture will cover how to treat some common and not so common toxicities. The lecture will focus on 4 primary toxins, albuterol, marijuana, xylitol, and baclofen. Following the lecture, there will be a one hour question and answer session with the doctors. There will be 3 additional lecturers present, each one with further information on one of these toxins. Doctors will be set up in stations and attendees will be able to ask each of these doctors further questions on their specific toxin.

Hosted by
Animal Emergency and Critical Care
104 South Main Street
Longmont, CO 80501

Lecture
Close Encounters: Emergency Toxicities

Speaker
Dana Dietrich, DVM (Primary Speaker)

Date and Time
Thursday, October 25, 2012
6:30pm Registration, Appetizers, and Drinks
7:00-8:00pm Lecture
8:00-9:00 Question and Answer Portion

Location
Front Range Community College- Community Room
2121 Miller Drive
Longmont, CO 80501

RSVP
303.678.8844

PACE

Archives of our past newsletters containing timely and useful medical information are on **our website**.

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Megaesophagus: Etiologies are Immeasurable - It's not always MG

By: Sacha Mace, DVM Internal Medicine Specialist

Practice Points:

- Regurgitation is the most common clinical sign of

megaesophagus at presentation

- Megaesophagus is common in dogs and less common in cats
- Diagnosis of megaesophagus is made radiographically, and the primary cause should be evaluated with appropriate diagnostic testing
- Management of megaesophagus is supportive unless an underlying cause is identified

- The prognosis for megaesophagus depends on the presence of aspiration pneumonia and the underlying condition

Some Etiologies for Megaesophagus:
Megaesophagus is a disorder of the esophagus characterized by diffuse dilation and decreased peristalsis. It is classified into congenital and acquired forms. Gastrointestinal, endocrine, immune-mediated, neuromuscular, paraneoplastic, and toxic disorders have been associated with acquired megaesophagus. Common clinical signs of megaesophagus are regurgitation, weight loss, coughing, and halitosis. Most cases of megaesophagus can be diagnosed using thoracic radiography; however, diagnosing the underlying cause requires a thorough history and additional diagnostics. The treatment, management, and prognosis of megaesophagus vary greatly depending on the underlying cause.

Acquired megaesophagus is sub classified into idiopathic and secondary forms. Congenital and idiopathic acquired megaesophagus disorders are suspected to be due to a combination of neurologic dysfunction. Secondary acquired megaesophagus can be caused by any disease that inhibits esophageal peristalsis by disrupting central, efferent, or afferent nerve pathways or by any disease of the esophageal musculature, including immune-mediated, infectious, and preneoplastic etiologies.

Congenital megaesophagus is documented in Newfoundlands, Parson Russell terriers, Samoyeds, Springer spaniels, smooth fox terriers, and Shar-Peis. These dogs typically present at the time of weaning with signs of regurgitation. Irish setters, Great Danes, German shepherds, Labrador retrievers, miniature schnauzers, and Newfoundlands have an increased prevalence for acquired megaesophagus. Dogs with acquired megaesophagus present from 7 to 15 years of age. In Newfoundlands, acquired megaesophagus and MG occur at a much younger age (≤2 years), and these dogs do not have a history of congenital megaesophagus.

Congenital and acquired megaesophagus has been documented in cats, with a familial disposition for the congenital form in the Siamese breed.

The suspected etiology for congenital megaesophagus is esophageal hypomotility. In some patients, this hypomotility is due to delayed maturation of esophageal

function that may or may not improve with age. Congenital MG is an inherited autosomal recessive condition in Parson Russell terriers, Springer spaniels, and smooth fox terriers that results in a deficiency or functional abnormality of acetylcholine receptors (AChRs) at the neuromuscular junction. The long-term prognosis for congenital MG is poor and patients generally succumb within 1 year. Congenital MG patients usually present with generalized weakness in addition to megaesophagus.

The etiology for acquired idiopathic megaesophagus is unknown. The diffuse neuromuscular dysfunction of acquired secondary megaesophagus can be caused by a variety of neuromuscular, immune-mediated, endocrine, gastrointestinal, paraneoplastic, and toxic diseases. The most common neuromuscular disorders associated with megaesophagus include MG and generalized inflammatory myopathies such as polymyositis and those associated with infectious diseases (Protozoal, rickettsial, spirochetal, fungal, Distemper, Tetanus infections). Less common neuromuscular disorders associated with megaesophagus include myopathies such as muscular dystrophies, dysautonomia, storage diseases, and neurogenic muscular atrophy. Because the canine esophagus is composed predominantly of striated muscle, any neuromuscular disease that affects limb muscles can affect the esophagus.

Of all acquired megaesophagus cases, approximately 25% are secondary to MG. Acquired MG can present in focal, generalized, and acute fulminating forms. Focal MG can present with various degrees of esophageal, facial, laryngeal, or pharyngeal dysfunction. Ninety percent of dogs with generalized MG have megaesophagus. Although acquired MG can affect dogs of any age older than a couple of months, most affected dogs are between 2 and 3 years of age or older than 9 years. Acquired MG occurs most often in German shepherds and golden retrievers. Affected feline breeds include the Abyssinian, Somali, and Siamese.

Preneoplastic syndromes differ from paraneoplastic syndromes in timing. Preneoplastic syndromes occur with occult cancer. Preneoplastic syndromes that can cause myositis include bronchogenic carcinoma, lymphoma, myeloid leukemia, and tonsillar carcinoma.

Megaesophagus associated with distemper is due to demyelination. Neurologic signs can develop 1 to 3 weeks or even months after initial recovery.

Although dysautonomia is rare, megaesophagus is a common finding in these patients. Dysautonomia is an idiopathic autonomic nerve disorder of cats and dogs that is suspected to be immune mediated. Within 1 to 7 days, patients experience a fulminant loss of autonomic nervous system function, followed by constipation, dry mucous membranes, pupillary dilation, prolapsed nictitating membranes, diminished pupillary light response, bradycardia, areflexic anus, and bladder atony.

Glycogen storage diseases (GSDs) are inborn errors of

glycogen metabolism. Only GSD II, which is documented in Swedish Lapland dogs, has been associated with megaesophagus.

A clinical presentation of megaesophagus associated with gait abnormalities and laryngeal paralysis is suggestive of laryngeal paralysis-polyneuropathy complex (LP-PNC). Megaesophagus is documented in most dogs that are affected with LP-PNC. LP-PNC is documented in Dalmatians, Leonbergers, Pyrenean mountain dogs, and Rottweilers. Puppies usually present between 2 and 6 months of age; however, in Leonbergers, onset is delayed to 1 to 9 years of age.

Endocrine causes
Hypoadrenocorticism and hypothyroidism are associated with reversible megaesophagus. Patients with hypoadrenocorticism may have megaesophagus due to electrolyte imbalances and a cortisol deficiency. Electrolyte imbalances cause altered membrane potentials, which results in decreased neuromuscular function. In addition, muscle weakness is a consequence of deficient cortisol. The association between megaesophagus and hypothyroidism has yet to be understood. Hypothyroidism is prevalent in some breeds that are predisposed to megaesophagus and laryngeal paralysis. Megaesophagus occurs in 3% of hypothyroid dogs. Resolution of megaesophagus once the thyroid is regulated has been reported. Aspiration pneumonia may cause a sick euthyroid syndrome that may be misdiagnosed as hypothyroidism.

Gastrointestinal Causes
Gastrointestinal disorders associated with acquired megaesophagus include esophagitis, esophageal obstruction, gastric dilatation-volvulus, and hiatal hernia. In cats, acquired secondary megaesophagus is due to pyloric dysfunction. Esophagitis is a common finding associated with megaesophagus. It may or may not precede megaesophagus. In patients with esophagitis, secondary megaesophagus develops due to chemical or obstructive irritation. Gastric reflux contains gastric acid, pepsin, bile salts, and trypsin, all of which cause esophageal inflammation and ultimately decrease esophageal motility.

Esophageal obstructions can be caused by esophageal foreign bodies, neoplasia, strictures, or vascular ring anomalies. Foreign bodies can cause a partial or complete mechanical obstruction. Peristaltic spasms over the retained foreign object cause tissue edema and mucosal abrasions. Although possible in any small dog, there seems to be a higher incidence of esophageal foreign bodies in young terriers. Because these terriers are young, this may be a condition of delayed esophageal maturation.

Foreign bodies or chronic gastroesophageal reflux (GER) can cause esophageal strictures, which occur secondary to mucosal healing attempts. Esophageal damage that penetrates the submucosa and muscularis layers causes

inflammation resulting in collagen deposition and fibrous connective tissue stricture.

Extraluminal esophageal obstruction is most commonly associated with vascular anomalies. In 95% of patients with secondary megaesophagus due to a vascular ring anomaly, the cause is a persistent right aortic arch. Other vascular anomalies associated with secondary megaesophagus include persistent right or left subclavian arteries, double aortic arch, persistent right dorsal aorta, left aortic arch, right ligamentum arteriosum, aberrant intercostal arteries, and persistent left cranial vena cava.

Dogs with chronic or recurrent gastric dilatation with or without volvulus have an increased risk of developing megaesophagus. In these dogs, megaesophagus is due to decreased lower esophageal sphincter (LES) tone caused by a combination of esophagitis from chronic GER or vomiting; chronic intermittent obstruction of the LES; increased intragastric and intra-abdominal pressures; and delayed gastric emptying.

In patients with hiatal hernia, the esophagus is essentially obstructed. Four types of hiatal hernias have been described in humans. Two of these types are applicable to animals. Type I is the "sliding" hernia, defined as intermittent cranial displacement of the abdominal esophagus, LES, and gastric cardia through the hiatus. Type II is the paraesophageal hernia, in which the gastroesophageal junction remains in its normal anatomic position; however, the stomach and abdominal organs enter the caudal mediastinum through a defect adjacent to the esophageal hiatus.

Paraneoplastic Causes
According to one study, megaesophagus was present in 40% of dogs with thymoma. The incidence of thymoma in dogs with MG is 3%; in cats with MG, the incidence is 26%. In humans, thymomas have increased production of CD4+CD8+ T cells and lack antigen-presenting cells that function for negative selection. This combination results in autoimmune disease. The prognosis for nonresectable thymoma in a dog with MG and megaesophagus is poor. However, complete thymic resection can result in resolution of megaesophagus and a decrease in AChR antibody titer.

Toxic Causes
Toxic substances that can cause megaesophagus include lead, organophosphates, and snake venom. Low-level lead exposure causes severe abdominal pain, vomiting, diarrhea, and megaesophagus. Lead intoxication can occur from ingestion of batteries, fishing line weights, lead-based paint, linoleum, and plumbing or solder supplies. Organophosphate toxicosis should be suspected if a patient presents with concurrent weakness and cerebellar signs. Organophosphates exist in flea collars and insecticides. They irreversibly bind to acetylcholinesterase, causing a cholinergic crisis (salivation, lacrimation, urination, defecation). Australian tiger snake envenomation causes a

rapidly progressing myopathy of skeletal muscle. If not lethal, Australian tiger snake envenomation has a 75% recovery rate for normal esophageal function.

Diagnostics for ME:
Imaging:
Thoracic radiography is diagnostic for most cases of megaesophagus. The degree of esophageal dilation has no diagnostic value in determining the etiology. Underlying causes of megaesophagus that may be revealed by radiography include neoplasia, foreign body, vascular ring anomaly, gastric dilatation-volvulus, and hiatal hernia. Normal midline tracheal location does not exclude a vascular ring anomaly; however, focal leftward deviation of the trachea near the cranial border of the heart on a dorso-ventral or ventro-dorsal view is a reliable sign of persistent right aortic arch in young dogs that regurgitate after eating solid food. Radiographic findings of megaesophagus with concurrent aspiration pneumonia or a distended stomach, small bowel, or urinary bladder should raise suspicion for dysautonomia. Incidental esophageal dilation does occur and is associated with excitement, aerophagia, general anesthesia, and vomiting.

If thoracic radiographic findings of megaesophagus are questionable, a barium contrast esophagram can confirm dilation and mechanical obstruction. Barium accumulates within the distended esophagus. Focal narrowing of the esophagus at the cardiac base is suggestive of a vascular ring anomaly. However, the diagnostic benefit of a contrast study should be weighed against the potential for aspiration of contrast agent.

Fluoroscopy evaluates pharyngeal motility and the presence and intensity of esophageal peristalsis. However, this diagnostic modality is not essential for diagnosis of megaesophagus. It can be helpful in cases of MG or esophagitis. MG can selectively affect only the pharyngeal and esophageal musculature without more overt clinical signs. Also, in cases of mild esophagitis, fluoroscopy may be of greater diagnostic value than a contrast esophagram in detecting hypomotility.

Esophagoscopy is rarely indicated for a diagnosis of megaesophagus, but it can be helpful for suspected cases of obstructive disease or reflux esophagitis. Esophagoscopy may identify an esophageal stricture due to a vascular ring anomaly, but it cannot differentiate the type of vascular ring anomaly.

Laboratory Testing
A complete blood count (CBC), serum chemistry panel that includes CK activity, and urinalysis should be performed for all regurgitating patients and those in which megaesophagus is suspected. In addition, an AChR antibody titer test should be performed in all cases of acquired megaesophagus. AChR antibody testing is performed by the Comparative Neuromuscular Laboratory in the School of Medicine at the University of California, San Diego. Information regarding sample submission can

be obtained at http://vetneuromuscular.ucsd.edu/. Corticosteroid therapy at immunosuppressive dosages for longer than 7 to 10 days lowers AChR antibody levels, so a pre-corticosteroid serum sample is recommended.

Additional diagnostics are performed based on the history, physical examination, and preliminary laboratory findings. The diagnostic objective is to determine whether the megaesophagus is associated with a potentially treatable disorder. For example, MG, hypothyroidism, hypoadrenocorticism, polymyositis, and lead poisoning all have specific treatments, whereas treatment for idiopathic megaesophagus is limited to supportive and symptomatic management.

Elevated serum CK activity occurs with muscle damage associated with some myopathies (inflammatory, necrotizing, and dystrophic) and muscle trauma and may be mildly elevated in patients in extended recumbency or after intramuscular injections.

Definitive diagnosis of acquired MG requires an AChR antibody titer test. However, this test is not useful in diagnosing congenital MG, which is a result of structural or functional AChR abnormalities and not immune-mediated damage. Therefore, congenitally affected dogs and cats do not have measurable circulating AChR antibodies.

Edrophonium chloride, a short-acting acetylcholinesterase drug, can be administered to support a presumptive diagnosis of congenital or acquired MG. Before administering the edrophonium be sure to exercise your patient until fatigued. The appearance of MG fatigue can include weakness, stiff gait, collapse, inspiratory stridor, or a reduced palpebral reflex. After fatigue is induced, edrophonium is administered IV (0.1 to 0.2 mg/kg). A positive response is characterized by improved muscle strength. This commonly occurs within 30 seconds of the edrophonium injection, and weakness returns within 5 minutes. Temporary improvement of generalized muscle weakness is suggestive of, but not definitive for, MG. The degree of megaesophagus is not affected by this test; however, improvement in motility may be observed if evaluated by fluoroscopy after administration of contrast agent.

Hypercholesterolemia, hypertriglyceridemia, and hyponatremia with or without the presence of a normochromic, normocytic, nonregenerative anemia is suggestive of hypothyroidism. In most cases, low total T4, elevated canine thyroid stimulating hormone, and low free T4 levels confirm the diagnosis of hypothyroidism.

Dogs with typical or atypical hypoadrenocorticism can present with megaesophagus. Hyperkalemia and hyponatremia are suggestive of typical hypoadrenocorticism. A low sodium to potassium ratio is not definitive for hypoadrenocorticism, even with studies that found a sodium to potassium ratio <15 to be more diagnostic for hypoadrenocorticism than a ratio of 27:1.

Typical and atypical hypoadrenocorticism are diagnosed with an ACTH stimulation test.

Nucleated erythrocytes, without anemia or basophilic stippling of red blood cells, suggests lead poisoning. These abnormalities are caused by transportation of lead to bone marrow. Serum blood lead tests are commercially available.

Organophosphate toxicosis can be excluded by measuring cholinesterase in a whole blood sample. A cholinesterase activity less than 25% to 50% of normal is suggestive of organophosphate toxicosis.

Additional diagnostic tests may include antibody titers for Toxoplasma gondii, Neospora caninum, Borrelia burgdorferi, Ehrlichia canis, and Rickettsia rickettsii; electromyography; measurement of nerve conduction velocity; and muscle and nerve biopsies to exclude myopathic and neuropathic disorders. Atropine, histamine, or pilocarpine tests can be performed to exclude dysautonomia. Atropine is administered IV (0.02 mg/kg) or SC (0.04 mg/kg); lack of an increase in heart rate is supportive of dysautonomia. When the intradermal histamine test (0.01 mg per dog) is used, absence of a wheal and flare within 15 minutes is supportive of dysautonomia. The histamine test has limited value in cats, as there is no significant difference in the histamine response between dysautonomic and control cats. If miosis does not occur after topical ophthalmic administration of pilocarpine 0.1%, a presumptive diagnosis of dysautonomia can be made.

Treatment

Treatment for idiopathic megaesophagus is largely supportive and symptomatic with periodic rechecks. Thoracic radiography is advised to monitor progress of esophageal dilation and aspiration pneumonia. Treatment for acquired secondary megaesophagus depends on managing the underlying specific disease process in addition to providing supportive and symptomatic care. Medications should be in liquid (not pill) form to enhance movement to the stomach and avoid accumulation within the esophagus, which can lead to esophageal irritation and nontherapeutic medication levels. If accumulated medication passes into the stomach, overdose may occur. Nutritional needs must be met and regurgitation minimized. This can be accomplished by frequent feeding of small, high-calorie meals with the patient in a cranially elevated position (FIGURE 1). The optimal food consistency to minimize regurgitation varies with each patient, so experimentation is encouraged. Nasoesophageal or esophageal tubes are not advised because they increase regurgitant volume, raising the risk of aspiration pneumonia. Aspiration pneumonia and esophagitis are the most common complications of megaesophagus.

Prognosis

The prognosis for megaesophagus varies with the

underlying etiology and presence of secondary complications. Aspiration pneumonia, dehydration, and malnutrition can significantly worsen the prognosis. Congenital megaesophagus has a guarded to poor prognosis; however, there is potential for improvement of esophageal motility with maturity up to 1 year of age. The prognosis for congenital MG is poor due to the mechanism of the condition, lack of a specific treatment, and high complication rate of aspiration pneumonia. Acquired idiopathic megaesophagus in general has a guarded to poor prognosis due to the common occurrence of aspiration pneumonia and malnutrition. Morbidity and mortality depend on the degree and nature of the underlying disease and client compliance. In the absence of severe aspiration pneumonia or thymoma, the success rate for acquired MG can be good with early diagnosis and appropriate management. Spontaneous remission of acquired MG with resolution of megaesophagus can also occur within an average of 6 months. However, many myasthenic dogs die of aspiration pneumonia during the first month after diagnosis, so the overall prognosis is still guarded. With the exception of the acute fulminating form of myasthenia, there is no association between the severity of MG and the possibility of remission. In one study, 39% of dogs with immune-mediated polymyositis had clinical improvement of their megaesophagus with continued medical management. However, early diagnosis and initiation of appropriate therapy are key to a good clinical outcome. Evaluation of muscle biopsy samples early in the course of the disease to establish a diagnosis is critical. The prognosis for pre- and paraneoplastic myositis is poor due to the underlying cancer. Dysautonomia is progressive, with a survival rate of <25% in cats over 18 months. Prognostic indicators include showing response to therapy (e.g., maintenance of body weight with oral feedings, fecal and urinary continence) within 7 to 10 days.

The original Bailey chair creator, Donna Koch, offers free Bailey chair building instructions:matthew1@earthlink.net & pawsup616@yahoo.com.
They also have a web group site at:
http://pets.groups.yahoo.com/group/megaesophagus/

Thank you for your continued support!
-Aspen Meadow Veterinary Specialists

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