Canine Megaesophagus

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Profile

DEFINITION

A disorder characterized by esophageal hypomotility and dilation, progressive regurgitation, and loss of body condition. Two forms of the syndrome have been described: congenital and acquired megaesophagus.

Congenital megaesophagus is often recognized in puppies shortly after weaning, causing regurgitation and failure to thrive.

Acquired megaesophagus may develop secondary to several conditions. MG accounts for 25% to 30% of these cases.[1–3] In some cases of MG, regurgitation and weight loss may be the only presenting signs, whereas in most cases of MG, regurgitation is but one of many clinical signs that may be present, including peripheral muscle weakness. Acquired megaesophagus has also been associated with hypoadrenocorticism, lead poisoning, lupus myositis, dysautonomia, and severe forms of esophagitis. Hypothyroidism has been suggested as a cause of acquired megaesophagus, but this disorder has not been identified as an important cause in retrospective risk factor analysis.[1] Acquired megaesophagus also describes cases of adult-onset megaesophagus that have no known cause (idiopathic); most cases of megaesophagus fall into this category.

SIGNALMENT

Species. Megaesophagus is the most common cause of regurgitation in dogs.[1,4] Aside from dysautonomia, megaesophagus is rare in domestic cats. Congenital megaesophagus has been reported in several cats[5] and in one group of cats secondary to pyloric dysfunction.[6]

Breed predilection.
Congenital Megaesophagus: Increased incidence has been reported in Irish setters, Great Danes, German shepherds, Labrador retrievers, Chinese Shar-Peis, and Newfoundland breeds, and autosomal dominant inheritance has been demonstrated in miniature Schnauzers and fox terriers (summarized in reference 4).

Acquired Megaesophagus: No apparent breed predilection for acquired idiopathic megaesophagus. Some breeds (e.g., Akitas; terriers, including Scottish terriers; German shorthaired pointers; and Chihuahuas) may be at increased risk for MG.[3]

Age and range. Between 7 and 15 years of age for acquired megaesophagus; shortly after weaning for congenital megaesophagus.

Gender. No predilection.

CAUSES/PATHOPHYSIOLOGY

The pathogenesis of the **congenital form** is incompletely understood, although several studies have pointed to a defect in the vagal afferent innervation of the esophagus.[7–12]

Acquired megaesophagus has been compared erroneously with esophageal achalasia in humans. Achalasia is a failure of relaxation of the lower esophageal sphincter and ineffective peristalsis of the esophageal body. A similar disorder has never been rigorously documented in dogs, although case reports appear from time to time.[13] Several important differences between canine acquired megaesophagus and human achalasia have been documented.[7,8,14] Although causes have not been identified, some studies suggest a defect in the afferent neural response to esophageal distention, similar to that reported in congenital megaesophagus.[15]

RISK FACTORS

Risk factor analysis suggests that esophagitis markedly increases the risk for acquired megaesophagus. It is not yet clear whether esophagitis is a cause or consequence of megaesophagus. Hypothyroidism has been cited as an important cause of acquired megaesophagus in dogs, although risk factor analysis has not revealed a clear association.[1] MG accounts for 25% to 30% of secondary cases,[2,3] and hypoadrenocorticism, lead poisoning, dysautonomia, and lupus myositis have been associated with acquired megaesophagus.

CLINICAL SIGNS

Regurgitation is the most frequent clinical sign. Frequency of regurgitation may vary from one episode every few days to many episodes per day. As with many esophageal disorders, affected animals may become malnourished and may develop aspiration pneumonia. Physical examination often reveals excessive salivation, mild to moderate cachexia, coughing, and pulmonary crackles or wheezes.

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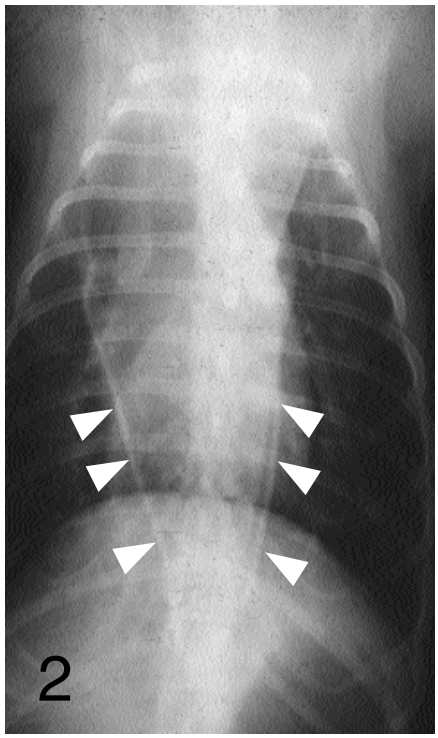
ACTH = adrenocorticotropic hormone; MG = myasthenia gravis

Diagnosis

Routine hematology, serum biochemistry, and urinalysis should be done to investigate secondary causes of acquired megaesophagus (e.g., hypoadrenocorticism). **Survey radiographs** are usually diagnostic for megaesophagus (**Figures 1** and **2**). **Contrast radiographs**, which are associated with a risk for aspiration pneumonia in some affected dogs, may be necessary to confirm the diagnosis, evaluate motility, and exclude foreign bodies or other causes of obstruction. **Endoscopy** has many uses in the diagnosis and treatment of esophageal disease. Endoscopy may be used to substantiate the diagnosis of megaesophagus, rule out obstructive lesions (e.g., foreign bodies and neoplasia), and confirm concurrent esophagitis, a frequent finding in canine acquired megaesophagus.[1] The decision to perform endoscopy in an individual patient should always be weighed against anesthetic risk, presence of aspiration pneumonia, and the likelihood of new findings.



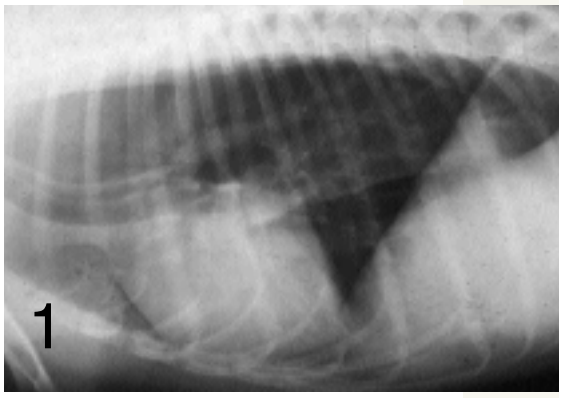
Ventrodorsal survey radiograph of the same dog with megaesophagus shown in Figure 1. The severity of the esophageal dilation is more evident in this view.

If secondary causes are suspected, additional diagnostic tests should be considered (e.g., serologic testing for nicotinic acetylcholine-receptor antibody, ACTH stimulation, antinuclear antibody testing, serum creatine phosphokinase activity, electromyography and nerve-conduction velocity, and muscle and nerve biopsy).[4] Ancillary testing is determined on a case-by-case basis. Thyroid function testing (e.g., endogenous TSH, TSH stimulation, T_4) should be performed in individual suspected cases (e.g., concurrent cutaneous or metabolic manifestations).

Treatment

If secondary disease can be excluded, therapy for congenital and acquired megaesophagus should be directed at nutritional management and treatment of aspiration pneumonia.

Animals with acquired megaesophagus should be appropriately differentiated from those with other esophageal disorders. Those with MG should be treated with pyridostigmine (1.0 to 3.0 mg/kg PO Q 12 H).[16] Those that do not respond to pyridostigmine may need immunosuppression with corticosteroids (prednisone, 1.0 to 2.0 mg/kg PO or SC Q 12 H) or azathioprine, 2 mg/kg PO Q 24 H initially then 0.5 to 1.0 mg/kg PO Q 48 H. Mycophenolate mofetil, a novel immunosuppressive drug with relative specificity for lymphocytes, has also been recommended, but this drug has not been sufficiently evaluated. Dogs with hypothyroidism should be treated with levothyroxine (22 µg/kg PO Q 12 H), and those affected with polymyositis should receive prednisone (1.0 to 2.0 mg/kg PO Q 12 H). A detailed review of treatment of these disorders may be found in reference 4.



Lateral survey radiograph of a nine-year-old dog with acquired megaesophagus

Smooth-muscle **prokinetic therapy** (e.g., metoclopramide or cisapride) has been advocated for stimulating esophageal peristalsis, but these drugs will probably not have much affect on the striated muscle of the canine esophageal body.[17–19] Esophageal 5-HT_4 receptors are present in many species but are apparently absent in canine esophageal striated muscle.[17] Bethanechol has been shown to stimulate esophageal propagating contractions in some affected dogs and may therefore be a more appropriate prokinetic agent.[14] Because of the high incidence of esophagitis in canine megaesophagus, affected animals should also be medicated with oral sucralfate suspensions (1 g Q 8 H for large dogs; 0.5 g Q 8 H for smaller dogs; 0.25 to 0.5 g Q 8 H to Q 12 H for cats).

Pulmonary infections should be identified by culture and sensitivity, and an appropriate antibiotic selected for the offending organism. This may be accomplished by trans- or endotracheal wash or by bronchoalveolar lavage at the time of endoscopy. Nebulization and coupage may be useful adjunctive therapy in the treatment of aspiration pneumonia.

Antiemetic therapy (α_2-adrenergic antagonists, D_2-dopaminergic antagonists, 5-HT_3 antagonists) and gastric antisecretory

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agents (H_2-histaminergic antagonists; H^+, K^+-ATPase inhibitors) may be used but have unproven efficacy in treatment of esophageal disorders other than esophagitis, gastroesophageal reflux, and hiatal hernia.

Surgery. Cardiomyotomy (myotomy of the gastroesophageal sphincter) had been recommended as a therapeutic measure in the belief that canine megaesophagus is similar to human achalasia. However, because most studies have reported normal gastroesophageal sphincter tone and appropriate relaxation with swallowing (summarized in reference 15), cardiomyotomy cannot be recommended. Indeed, animals treated with myotomy generally have less favorable outcomes than untreated animals.

NUTRITIONAL ASPECTS

Affected animals should be fed a high-calorie diet in small, frequent feedings, from an elevated or upright position to take advantage of gravity drainage through a hypomotile esophagus. Dietary consistency should be formulated to produce the fewest clinical signs. Some animals handle liquid diets quite well; others do better with solid meals. Animals that cannot maintain adequate nutritional balance with oral intake could be fed by a temporary or permanent gastrostomy tube, which can be placed surgically or percutaneously with endoscopic guidance.

Rx at a glance…

- **Treat any underlying cause**
  Myasthenia gravis:
  pyridostigmine, 1.0-3.0 mg/kg PO Q 12 H
  OR
  prednisone, 1.0-2.0 mg/kg PO or SC Q 12 H
  OR
  azathioprine, 2 mg/kg PO Q 24 H initially; then 0.5-1.0 mg/kg PO Q 48 H
  mycophenolate mofetil ?
  Hypothyroidism:
  levothyroxine, 22 µg/kg PO Q 12 H
  Polymyositis:
  prednisone, 1.0-2.0 mg/kg PO Q 12 H
- **Sucralfate**
  PO: 1 g Q 8 H for large dogs
  0.5 g Q 8 H for smaller dogs
  0.25-0.5 g Q 8 H–12 H for cats
- **Prokinetic therapy?**
  bethanechol
- **Antiemetic/gastric antisecretory agents?**
- **High-calorie diet in small, frequent feedings from elevated position.**
  Test consistencies from liquid to solid for tolerance.
- **Temporary or permanent gastrostomy tube feeding in recalcitrant cases**
- **Animal maintained in standing position for 15-30 min after feeding**
- **Antibiotics** (for aspiration pneumonia)

Follow-Up

PATIENT MONITORING

Patients should be monitored for appetite, frequency and severity of regurgitation, loss of body weight, and signs of pulmonary complications.

COMPLICATIONS

Affected animals may develop aspiration pneumonia at any point during the onset or chronic phase of the disease. Pet owners and veterinarians should monitor for coughing, loss of appetite, and general malaise. Recurrences of aspiration pneumonia may necessitate additional care, including hospitalization.

PROGNOSIS

Congenital megaesophagus is associated with a fair prognosis. With adequate attention to caloric needs and recurring episodes of aspiration pneumonia, many animals develop improved esophageal motility over several months. Pet owners must be committed to months of physical therapy and nutritional support. Affected animals must be managed with controlled feedings; ad libitum feeding increases risk for regurgitation and aspiration pneumonia. Elevated feedings with the animal held in a standing position for 15 to 30 minutes after feeding may improve prognosis and outcome. Pet owners should be advised to monitor for coughing, which may be an early sign of aspiration pneumonia.

Acquired megaesophagus has a guarded to poor prognosis. Chronic malnutrition and repeated episodes of aspiration pneumonia worsen the long-term prognosis.

Acquired megaesophagus has a more favorable prognosis if underlying disease can be promptly identified and successfully managed.[4] Refractory cases result from chronic esophageal distention, myenteric nerve degeneration, and muscle atrophy.

In General

RELATIVE COST OF TREATMENT

Depends on severity of clinical signs and recurrences of aspiration pneumonia. Many pet owners spend $500 to $1000/year managing the dog with megaesophagus.

FUTURE CONSIDERATIONS

Current clinical research is focused on identifying genetic predispositions and or mutations (congenital form) and the underlying cause of the disease (acquired form). The mechanism of acquired idiopathic megaesophagus appears to be a disorder of the afferent innervation of the esophagus, but the exact cause has not yet been determined. New therapies are needed, as most prokinetic drugs are ineffective in the treatment of this disease.

See Aids & Resources, back page, for references, contacts, and appendices.