Megaesophagus, which can be divided into diffuse or segmental esophageal disorders, is more common in dogs than in cats. Generalized megaesophagus occurs with diffuse motility disorders related to inflammation (esophagitis), neuropathies, neuromuscular disease (myasthenia gravis [MG], dysautonomia), myopathies, or idiopathic acquired or congenital disorders. Acquired idiopathic megaesophagus is the most common primary cause of adult-onset generalized megaesophagus. MG is the most common cause of secondary megaesophagus in adult dogs. Megaesophagus can occur secondary to metabolic disease (hypoadrenocorticism, hypothyroidism) and toxicities (lead, organophosphates). Segmental megaesophagus can result from obstructive processes or local esophageal disorders (inflammation, neoplasia) that interfere with motility of a portion of the esophagus. Enlargement of the cranial esophagus with normal function of the distal esophagus in puppies suggests a vascular ring anomaly or esophageal stricture. Esophageal strictures usually occur in the distal third of the esophagus in dogs. Esophagitis of the lower third of the esophagus can result from gastric or duodenal fluid that comes in contact with the esophageal mucosa as a result of vomiting, reflux, or pooling during anesthesia. Esophageal foreign body is usually not a cause of regurgitation and megaesophagus.

Dogs with megaesophagus usually present for regurgitation that may occur several hours after food consumption. Regurgitation is distinguished from dysphagia and vomiting based on clinical signs. Regurgitation is passive and does not involve a neural reflex or the central nervous system. When the pharynx and esophagus cannot propel food toward the stomach, food and liquid accumulate. Gravity or passive changes in intrathoracic or intraabdominal pressure cause food to be propelled from the pharynx or esophagus out of the mouth. There are no antecedent events, such as abdominal contractions or retching, associated with regurgitation. The food is undigested and has a neutral to alkaline pH.

Usually dogs with megaesophagus do not have difficulty swallowing. However, the accumulation of food can act as a foreign body and can cause esophagitis. Esophagitis is painful, and anorexia, painful swallowing (odynophagia), repeated swallowing, and drooling may be present. Dogs with megaesophagus are predisposed to aspiration pneumonia, and respiratory complications are a common reason that owners elect euthanasia. Therefore, dyspnea, cough, and nasal discharge may be the presenting complaints. Weight loss occurs in animals that are unable to digest adequate calories due to megaesophagus. Thoracic auscultation may reveal raspy fluid sounds as a result of movement of fluid in the esophagus or moist rales or crackles in animals with aspiration pneumonia.

**DIAGNOSTIC CRITERIA**

**Historical Information**

**Gender Predisposition.** There are no pronounced gender predilections for megaesophagus.

**Age Predisposition.** Acquired idiopathic megaesophagus occurs in older dogs. Vascular ring anomalies and congenital megaesophagus are associated with regurgitation at weaning (<10 weeks of age).

**Inside this issue:**

**Peer-Reviewed Articles on**

1. Megaesophagus in Dogs
2. Chylothorax
Breed Predisposition. Acquired idiopathic megaesophagus most commonly occurs in large-breed dogs. Congenital megaesophagus is an inherited anomaly that has been reported in fox terriers, miniature schnauzers, Great Danes, German shepherds, Labrador retrievers, Newfoundlands, Chinese shar-peis, and Irish setters. Congenital MG is most often reported in Jack Russell terriers.

Owner Observations. Regurgitation and weight loss are the hallmarks of megaesophagus, although many pet owners do not distinguish between the regurgitation and vomiting. With an unclear history, it can be helpful to feed the dog in the exam room and observe for signs of vomiting or regurgitation. Nasal discharge, dyspnea, coughing, painful swallowing or multiple swallowing attempts, and hypersalivation may accompany regurgitation and weight loss.

Other Historical Considerations/Predispositions. Postanesthetic regurgitation is suggestive of esophagitis, while regurgitation that occurs 2 to 3 weeks following anesthesia is suggestive of esophageal stricture. Megaesophagus associated with hypoadrenocorticism is usually an incidental finding rather than a common cause of regurgitation. The association between hypothyroidism and megaesophagus is questionable and should only be considered after all other causes of megaesophagus have been excluded.

Physical Examination Findings
• Weight loss will be present, but dogs may be otherwise normal on physical examination.
• Dyspnea, cough, nasal discharge, and auscultable moist rales or crackles may be present in dogs with aspiration pneumonia.
• An enlarged fluid-filled esophagus may be palpated in the cervical region, and cervical auscultation may reveal raspy fluid sounds as a result of movement of fluid in the esophagus.
• Dogs with megaesophagus secondary to generalized MG may exhibit episodic weakness or gait abnormalities. Occasionally, dogs with acute decompensating MG will exhibit a rapid onset of marked weakness, megaesophagus, and usually pneumonia.
• Dogs with diffuse myopathies, peripheral neuropathies, or central nervous system disease may demonstrate muscle pain, lower motor neuron signs, and cranial nerve deficits, respectively.
• Dogs with megaesophagus secondary to lead toxicity may demonstrate neurologic abnormalities, vomiting, diarrhea, and pallor. Organophosphate toxicity is associated with hypersalivation, lacrimation, urination, defecation, and dyspnea.
• Dysautonomia is a rare condition in cats and dogs and should be suspected in animals that exhibit xerostomia, weakness, and myosis.

Laboratory Findings
• Complete blood count (CBC), chemistry profile, urinalysis, and fecal examination results are usually within normal limits.
• Dogs with hypoadrenocorticism may exhibit hyponatremia and hyperkalemia.

KEY TO COSTS
$ indicates relative costs of any diagnostic and treatment regimens listed.
$ costs under $250
$$ costs between $250 and $500
$$ costs between $500 and $1000
$$$$ costs over $1000
CBC results in dogs with megaesophagus secondary to lead toxicity may show nucleated erythrocytes, anemia, and basophilic stippling.

Creatine phosphokinase (CPK) levels may be elevated in dogs with inflammatory or infectious myositis.

Other Diagnostic Findings

- Radiographs of the cervical and thoracic esophagus should be obtained. Survey radiographs can be diagnostic if the cervical esophagus is air filled or the thoracic esophagus contains large amounts of food, fluid, or air. In these situations, the distended esophagus is outlined by a different radiographic density than the surrounding tissue.
- Positive contrast studies and videofluorography can be helpful in distinguishing obstructive lesions and localizing abnormal motility to specific regions of the esophagus. Enlargement of the cranial esophagus with normal motility of the distal esophagus suggests a vascular ring anomaly or esophageal stricture. Esophageal strictures usually occur in the distal third of the esophagus in dogs. Cranial strictures are more common in cats. Due to the risk of aspiration, contrast imaging studies should be performed only when necessary.
- Endoscopy is used to confirm a diagnosis of esophageal stricture or other intraluminal obstructive diseases. Endoscopy is of limited value in the diagnosis of motility disorders.
- Acquired MG is an immune-mediated disease directed against acetylcholine receptors. Diagnosis is based on a positive acetylcholine receptor antibody titer. Congenital MG is an anomalous condition in which dogs are born with insufficient acetylcholine receptors in the esophagus. Therefore, the acetylcholine receptor antibody test is not helpful diagnostically. Both congenital and acquired MG can be presumptively diagnosed with a Tensilon test.
- Electromyography (EMG) can provide supportive findings in MG and diffuse neuromyopathies and myopathies.

Summary of Diagnostic Criteria

- Weight loss and regurgitation are the hallmarks of megaesophagus.
- Megaesophagus is diagnosed with radiography. Most cases of generalized megaesophagus are evident on survey radiography. Contrast esophography and videofluorography can be used to distinguish segmental megaesophagus from focal motility disorders.
- In adult dogs, idiopathic megaesophagus is a diagnosis of exclusion: All other causes of secondary generalized megaesophagus must be excluded.
- Regurgitation occurring less than 3 weeks after anesthesia administration or surgery is suggestive of esophagitis or esophageal stricture.

Differential Diagnosis

In adult dogs with megaesophagus, the diagnostic challenge is to distinguish acquired idiopathic megaesophagus from secondary megaesophagus. Diagnostic tests that can be helpful include an acetylcholine receptor antibody titer, Tensilon test, adrenocorticotropic hormone (ACTH) stimulation test, functional thyroid screening tests, antinuclear antibody (ANA) titer, CPK level, EMG, blood lead levels, provocative ocular tests, muscle and nerve biopsy, and Toxoplasma and distemper titers. A diagnosis of acquired idiopathic megaesophagus is made only after secondary causes have been eliminated.

- MG: Acetylcholine receptor antibody titer, Tensilon test, EMG.
- Central nervous system disease: EMG, distemper, computed tomography.
- Neuropathies: ANA titer, blood lead level, nerve biopsy, EMG.
- Myositis/myopathies: ANA titer, CPK level, muscle biopsy, EMG, Toxoplasma titer.

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**TABLE 1**

**Clinical Signs Distinguishing Dysphagia, Regurgitation, and Vomiting**

<table>
<thead>
<tr>
<th>Clinical Sign</th>
<th>Pharynx/ Cranial Esophagus</th>
<th>Esophagus</th>
<th>Stomach (vomiting)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time of food ejection</td>
<td>Immediate</td>
<td>Delayed</td>
<td>Delayed</td>
</tr>
<tr>
<td>Character of food ejected</td>
<td>Undigested</td>
<td>Undigested</td>
<td>Partially digested, bile-stained, acid pH</td>
</tr>
<tr>
<td>Swallowing attempts</td>
<td>Multiple</td>
<td>Single</td>
<td>Single</td>
</tr>
<tr>
<td>Ability to drink</td>
<td>Poor</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Pain on swallowing</td>
<td>Possible</td>
<td>Common</td>
<td>Absent</td>
</tr>
<tr>
<td>Associated signs</td>
<td>Dyspnea, cough, salivation</td>
<td>Dyspnea, cough, salivation</td>
<td>Retching, salivation</td>
</tr>
</tbody>
</table>
The use of immunosuppressive dosages of corticosteroids in acquired MG is controversial. The potential benefits of suppressing immune-mediated destruction of acetylcholine receptors must be weighed against the potential deleterious effects of immune suppression in an animal at risk for aspiration pneumonia. In patients with pneumonia, it seems prudent to resolve the pneumonia with supportive care measures before initiating corticosteroid therapy.

Some clinicians recommend corticosteroid therapy as a preventive measure against esophageal fibrosis and stricture formation in patients with esophagitis or in conjunction with balloon dilation of existing esophageal strictures. There is no evidence that corticosteroids prevent or retard esophageal fibrosis or stricture formation.

Hypothyroidism: Total thyroxine (T₄) concentration, free T₄ concentration by equilibrium dialysis, endogenous thyroid-stimulating hormone concentration.

Hypoadrenocorticism: ACTH stimulation test.

In puppies less than 10 weeks of age, congenital megaesophagus, congenital MG, and vascular ring anomalies are the most likely diagnoses and can be distinguished using imaging studies and neurologic testing.

Esophagitis and esophageal stricture are confirmed with endoscopy.

**TREATMENT RECOMMENDATIONS**

- If pneumonia is present, aggressive therapy should be instituted, including fluid therapy, oxygen supplementation, IV antibiotics, nebulization, and coupage. Antibiotic therapy should be based on bacterial culture and sensitivity results of transtracheal wash fluid. $$$

- Gastrostomy feeding tube placement to rapidly achieve positive energy balance in severely debilitated patients. $$

- Oral anticholinesterase drugs (e.g., pyridostigmine bromide, 2 mg/kg PO bid–tid) are indicated in patients with acquired or congenital MG. In acquired MG, immunosuppressive dosages of corticosteroids (2–4 mg/kg/day PO or SC) and azathioprine (2 mg/kg PO qid–sid) can be used.

- Sulcrate slurry (1 g/30 kg dissolved in water PO tid) and metoclopramide (0.2–0.4 mg/kg SC tid–qid or 1–2 mg/kg IV by constant-rate infusion) are indicated in patients with esophagitis. H₂ receptor blockers such as ranitidine (1–2 mg/kg PO bid) or proton pump inhibitors such as omeprazole (1 mg/kg q24h) may also be beneficial.

- Esophageal stricture is treated with sequential balloon dilation of the stricture under endoscopic guidance. Balloon dilation should be repeated in 2 to 10 days and then every 1 to 3 weeks as needed. Recurrence is common. $$$–$$$$

- Megaeosophagus caused by hypoadrenocorticism, lead toxicity, and organophosphate toxicity resolves with treatment of the underlying disease. $$

- Some vascular ring anomalies, such as persistent right aortic arch, can be treated surgically. $$$$

**Alternative/Optional Treatments/Therapy**

- Cisapride (0.25 mg/kg PO tid) or metoclopramide (0.25–0.5 mg/kg SC, IV, or PO tid–qid or 1–2 mg/kg/day IV by constant-rate infusion) can be instituted as a prokinetic drug in patients to increase lower esophageal sphincter tone and gastric emptying. These drugs seem most effective in patients with primary esophagitis or megaesophagus complicated by esophagitis. They are of questionable benefit in other forms of megaesophagus.

- In patients with MG that are unable to tolerate oral anticholinesterase administration, neostigmine methylsulfate (0.01–0.04 mg/kg IM tid–qid) can be used.

**Supportive Treatment**

- Specialized feeding practices include oral feeding of liquid or semisolid food. Canned food “meat-balls” can also be helpful.

- Feeding from an elevated position (where the upper body and forelimbs are elevated at least 45˚ relative to the hind limbs), allows gravity to assist the aboral movement of food. Holding the pet in a vertical position for 5 to 10 minutes after eating can also be beneficial.

- Multiple feedings rather than one large meal help minimize accumulation in the esophagus. Placing ice cubes in the patient’s food also slows the feeding process, allowing longer periods for the aboral movement of food through the esophagus.

- Low-fat, high-protein diets physiologically increase lower esophageal tone and decrease gastric emptying time.

- Daily caloric intake should be calculated for dogs based on their ideal weight.

**Patient Monitoring**

- Any patient with megaesophagus is at risk for aspiration pneumonia. Patients should be monitored for dyspnea, coughing, nasal discharge, and fever.
• Patients should be weighed every 2 weeks. Failure to gain weight should prompt an increase in the feeding schedule or placement of a gastrostomy feeding tube.

• Repeat thoracic radiographs should be performed every 2 to 4 weeks in cases of potentially reversible megaesophagus to assess response to therapy and monitor for the presence of pneumonia. Repeat videofluorography is used to assess improvement of motility disorders.

Home Management
• The specialized feeding practices listed above should be instituted at home. Owners should be counseled that caring for a pet with megaesophagus is labor intensive.

• Patients that are unable to tolerate oral feedings and receive gastrostomy feedings exclusively will aggressively seek out opportunities to chew and eat anything available. Constant surveillance is necessary for these pets as these episodes of dietary indiscretion often lead to bouts of aspiration pneumonia.

Milestones/Recovery Time Frames
• Adult-onset acquired idiopathic megaesophagus is irreversible, and improvement of the patient’s condition is solely based on its ability to tolerate supportive feeding practices. Weight gain may not occur for several months. Usually, modest weight gain can be achieved but patients rarely achieve their ideal body weight.

• Approximately 50% of dogs with megaesophagus that are diagnosed with MG improve to some degree over a period of weeks to months. The severity of the megaesophagus seems to correlate with prognosis.

• In puppies, congenital megaesophagus may resolve by 1 year of age. Most puppies that will recover will exhibit significant improvement by 6 months of age.

• In puppies with megaesophagus caused by surgically correctable vascular ring anomalies, improvement can be seen immediately after surgery to months later. In 25% of cases, the damage to the esophagus due to entrapment by the ligamentum arteriosum is permanent, and megaesophagus is irreversible.

Treatment Contraindications
• Gastroesophageal achalasia is a cause of megaesophagus in humans. Treatment of achalasia in humans involves myotomy of the lower esophageal sphincter. This surgery was previously recommended in dogs with megaesophagus. Manometric pressure studies have shown that lower esophageal sphincter pressure is not increased in dogs with megaesophagus and that when the dogs are tilted vertically, food moves into the stomach by gravity flow. Results of gastroesophageal myotomy in dogs are poor, and this technique is no longer recommended.

• Anticholinergic drugs are not indicated in megaesophagus and will interfere with the actions of metoclopramide and cisapride.

• Domperidone and bethanocol, although potent prokinetic drugs, should be avoided due to vomiting and intestinal discomfort.

• Administration of oral pills, capsules, or tablets in animals with severe diffuse motility disorders may result in esophageal retention of the medication, thereby leading to failure of drug absorption and local esophageal inflammation.

PROGNOSIS

Favorable Criteria
• In cases of secondary megaesophagus, favorable signs include weight gain, tolerance of oral feeding, and improved radiographic appearance of the esophagus. Improvement in motility is best assessed with videofluorography.

• In cases of adult-onset acquired idiopathic megaesophagus, weight gain, tolerance of oral feeding, and avoidance of aspiration pneumonia are favorable short-term signs.

Unfavorable Criteria
• Recurrent bouts of aspiration pneumonia typically influence owners to choose euthanasia. Recovery from each subsequent episode of pneumonia is generally more intensive and expensive.

• In patients that are unable to tolerate oral feeding, failure to gain weight and the owner’s perception of poor quality of life generally lead to euthanasia.

• The occurrence of coincident disease, such as nonrespiratory infections, neoplasia, decubital ulceration, and other skin manifestations, indicates systemic consequences of malnutrition and a poor short-term prognosis.

RECOMMENDED READING


