

Megaesophagus in the Canine Patient



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INTRODUCTION

Megaesophagus is a condition that affects many species in veterinary medicine. It is a disorder defined by decreased peristalsis and segmental or diffuse dilation resulting in a flaccid esophagus unable to move ingesta properly.^{8, 15} The ingesta will remain in the enlarged, lax esophagus, instead of entering in to the stomach, which results in regurgitation of esophageal contents.⁷ It is a more common condition in dogs compared to cats, but it is the most common cause of regurgitation in canines and felines.⁸ Megaesophagus can be congenital or acquired and has breed predispositions.^{8,15} Congenital megaesophagus can be described as inherited, idiopathic, or secondary to a congenital condition, and it is usually diagnosed at weaning.⁸ Acquired megaesophagus can be idiopathic or secondary to a variety of medical conditions, and it has a better prognosis when the primary problem is diagnosed and treated.⁸ A serious complication of megaesophagus is aspiration pneumonia, which is the usual cause of death or euthanasia in these patients.² This manuscript will examine and discuss megaesophagus in the canine patient.

HISTORY AND PRESENTATION

Typical clinical signs for megaesophagus include regurgitation, which the owner may refer to as vomiting, rapid weight loss, increased or decreased appetite, poor growth, pain on palpation of the esophagus (if esophagitis is present), halitosis, nasal discharge (if rhinitis or pneumonia is present), cough, and fever (if aspiration pneumonia is present).^{8,15, 7}

Dogs with congenital megaesophagus commonly present at weaning when the pup starts eating solid food.^{9,8} Regurgitation can occur through the mouth or nose, and results in poor growth as the animal is not able to receive adequate nutrition with this defect.⁹ Breeds susceptible to

megaesophagus include Chinese Shar-pei, Fox Terrier, Great Dane, Golden Retriever, Irish Setter, Labrador Retriever, Miniature Schnauzer, Newfoundland, and Greyhound.^{8, 9, 15} Acquired megaesophagus generally occurs in middle-aged to older dogs, and breed predisposition for these cases coincides with the breeds that are predisposed to the primary diseases.^{9, 8}

PATHOPHYSIOLOGY

The pharynx, containing the oropharynx, nasopharynx, and laryngopharynx, is the common passageway of the digestive and respiratory systems.¹ The esophagus is a tubular tissue that connects the mouth to the stomach to transport food and liquids, and it is solely made of striated muscle in the dog.¹ It has motor and sensory innervation from the vagus nerve.¹⁵ The upper esophageal sphincter closes off the proximal end of the esophagus and prevents reflux of ingesta into the pharynx.¹ The lower esophageal sphincter is not a true sphincter, but instead an area of high intraluminal pressure, located distally at the gastroesophageal junction.¹

The swallow reflex is a tightly coordinated process between the tongue, hard and soft palates, pharyngeal muscles, esophagus, epiglottis, and upper and lower esophageal sphincters.⁴ When the tongue pushes food to the back of the throat, the swallow reflex ensures food enters into the esophagus by using the tongue to block the oral cavity, the soft palate to close off the oropharynx, and the epiglottis to block the larynx and entrance into the trachea.⁴ At the same time, the upper esophageal sphincter opens to allow the bolus of food to enter into the esophagus.⁴ Esophageal motility is stimulated by the presence of food and liquids via receptors in the esophagus which send impulses along the afferent pathway to the swallowing center in the reticular

formation of the brain.^{4, 15} Next, the efferent pathways of the vagus nerve carry motor impulses to myoneural junctions in the esophagus, causing peristalsis.¹⁵

Though megaesophagus is not completely understood, it is thought that esophageal innervation is reduced due to complete or partial interruptions within the neural pathways of the swallow reflex arch.^{8,15} This decrease in esophageal motility can be diffuse or segmental, and will lead to food accumulation in the affected areas of the esophagus.^{8, 15, 4} The stasis of food will cause the esophagus to expand as food accumulates, and these areas will lose tone, further resulting in the inability to coordinate movement down the esophagus and progressing the condition.^{8, 15, 4} Due to this, ingesta will tend to roll around in the esophagus and not reach the stomach, resulting in inevitable regurgitation of esophageal contents, rapid weight loss, ravenous appetite, and possible aspiration.^{8, 15}

ASPIRATION PNEUMONIA

Decreased innervation to the esophagus results in dysfunction of the proximal esophageal sphincter, whose job is to open for food to enter into the esophagus during the swallow reflex, and then close to prevent reflux into the pharynx.^{15,4} With a malfunction, ingesta can reflux from the esophagus, enter into the pharynx, and then migrate into the trachea, oral cavity, or nasal cavity, as the mechanisms of the swallow reflex are not active to protect these areas.⁴ Of these, the most concerning route is reflux entering into the trachea resulting in aspiration pneumonitis or aspiration pneumonia.^{4, 10} Aspiration pneumonitis occurs after inhalation of ingesta into the respiratory tract resulting in inflammation of the airways and pulmonary parenchyma.¹⁰ Aspiration pneumonia, a bacterial infection of the pulmonary parenchyma, can occur concurrently with aspiration

pneumonitis if the ingesta has bacterial components at the time of inhalation, or will occur directly after aspiration pneumonitis if the inflammation is colonized with a secondary bacterial infection.¹⁰

Clinical signs for aspiration pneumonia can include fever, inappetence, tachypnea, and dyspnea.¹⁰ A physical exam can reveal wheezes, crackles, and increased or decreased lung noises on thoracic auscultation.¹⁰

DIFFERENTIAL DIAGNOSES

Although regurgitation is the most common clinical sign of megaesophagus, owners may refer to this as vomiting, and it is important to differentiate the two with a thorough history and observation of the patient.¹ Regurgitation is a passive process which involves retrograde movement, or reflux, of usually undigested food that has yet to reach the stomach.^{18, 1} Vomiting is an active, neurologically mediated process that refers to a forceful expulsion of digested gastric contents through the mouth.^{18, 3} Table 1 compares and contrasts the clinical features of vomition and regurgitation. It is also important to note that regurgitation, or holding a large amount of food within the esophagus, can trigger the gag reflex, resulting in a mixed presentation of vomiting and regurgitating, making it hard to distinguish the true diagnosis.^F

Numerous other medical conditions present with regurgitation as a common clinical sign. Differential diagnoses include diseases that cause improper esophageal function, but do not cause secondary megaesophagus.⁸ Cricopharyngeal dysphagia, hiatal hernias, esophageal neoplasia, and cricopharyngeal achalasia should all be considered in addition to megaesophagus as differentials in a regurgitating dog.^{9, 14} Signalment and a thorough history are important details to keep in mind when diagnosing these cases.⁹

	Vomition	Regurgitation
Patient presentation	<ul style="list-style-type: none"> • Nausea causing hypersalivation, multiple swallowing attempts, and/or inappetence • Abdominal contractions or heaving • Projectile vomiting 	<ul style="list-style-type: none"> • No prodromal process or “warning signs” • Patient commonly has normal to increased appetite
Content characteristics	<ul style="list-style-type: none"> • Digested, bile-stained (yellow) ingesta • Acidic pH 	<ul style="list-style-type: none"> • Undigested food • Alkaline pH • Tube – shaped ingesta
Timing	<ul style="list-style-type: none"> • Can occur at any time, but commonly occurs minutes to hours after eating 	<ul style="list-style-type: none"> • Commonly occurs directly after a meal, but can occur several hours after a meal • Frequently occurs after or during exercise or a change in position

Table 1: Comparison of the Clinical Features of Vomition and Regurgitation^{1, 3, 18}

DIAGNOSTIC APPROACH

Signalment, history, and clinical signs are all helpful when diagnosing megaesophagus. In addition to these, thoracic radiographs, contrast studies, and videofluoroscopy, can all be used to make the diagnosis.¹ An endoscopic exam may be warranted to find a cause for the megaesophagus, however, it is not a good screening test as anesthesia can cause decreased esophageal tone leading to a false diagnosis.^{8, 14, 5} Thoracic radiographs alone are commonly enough to make a definitive diagnosis.^{8,15} On radiographs, a normal dog’s esophagus is usually not able to be seen since it is the same radiodensity of adjacent tissues.¹ A dog affected with

megaesophagus has diffuse or segmental dilation of the esophagus, which can be filled with air, liquid, or fluid, making it more obvious to visualize.^{8, 1} Once megaesophagus has been confirmed, it is important to differentiate if it is a congenital condition or acquired with an underlying cause.

As mentioned, congenital megaesophagus can be observed at weaning in pups. It can be inherited, idiopathic, or secondary due to congenital diseases including vascular ring anomaly (especially persistent right aortic arch), esophageal diverticula, or esophageal agenesis.^{8, 14, 1}

As for acquired megaesophagus, a variety of diseases can cause this condition, and extensive testing should be performed to find a primary disease.⁸ There is a better prognosis if a primary disease is diagnosed and treated, as the esophagus may revert to normal size and function in these cases.^{1, 8} Idiopathic, acquired megaesophagus is incurable and carries a poorer prognosis.¹ Therefore, it should only be a diagnosis of exclusion after in-depth testing for other conditions.^{1, 8} Table 2 provides a list of diseases known to be linked to acquired megaesophagus, along with the diagnostics used to test for these conditions.

If aspiration pneumonia is suspected, three-view thoracic radiographs should be taken, as radiography is the gold standard for a diagnosis.¹⁰ Radiographs will reveal interstitial, alveolar, and mixed pulmonary patterns in the pulmonary parenchyma.¹⁰ The most common lung lobes affected include the right middle, left cranial, and right cranial lobes, frequently with more than one of these involved.¹⁰ A definitive diagnosis can be obtained from cultures of exudate present in the airways via a transtracheal wash, bronchoalveolar lavage, bronchial scraping, or bronchial biopsy.¹⁰ It's common for the normal flora of the oropharyngeal cavity to be the causative agents found in aspiration pneumonia.¹⁰ *Escherichia coli*, *Pasteurella*, *Staphylococcus*, *Streptococcus*, *Klebsiella*, *Enterococcus*, and *Mycoplasma* infections show a predominance in diagnosis, with a mixed infection being more common.¹⁰

Disease	Diagnostic test	Treatment
Myasthenia gravis	Serum antibodies to acetylcholine receptors, Tensilon test	Anticholinesterase inhibitors and/or corticosteroids
Extraluminal mass (thymoma, thyroid carcinoma, etc.)	Thoracic radiographs, CT	Appropriate chemotherapy, radiotherapy, or surgery
Hypoadrenocorticism (Addison's disease)	Complete blood count, serum chemistry, ACTH stimulation test	Glucocorticoids and mineralocorticoids
Hypothyroidism	Thyroid function tests include free t4, total t4, and TSH	Thyroid supplementations
Tetanus	History, clinical signs, and bloodwork	Tetanus anti-toxin, antibiotics, and supportive care
Neuropathies (dysautonomia)	Neurologic examination, nerve biopsies, electromyography, pilocarpine/physostigmine testing	Symptomatic and supportive care
Toxicities (lead, organophosphate, tiger snake venom)	Appropriate blood tests, (complete blood count, serum chemistry, blood lead levels), clinical signs	Specific toxin treatment and supportive care
Botulism	ELISA, PCR assay, or toxin isolation	Botulism antitoxin and supportive care
Esophagitis	Esophagoscopy with or without biopsies	Antibiotics, medical therapy to reduce gastric acidity, corticosteroids, and/or gastrostomy tube placement
Esophageal obstruction (foreign body, intussusception, stricture)	Thoracic and abdominal radiographs, contrast study radiographs, esophagoscopy	Appropriate esophagoscopy procedure or surgery
<i>Spirocerca lupi</i>	Complete blood count, fecal smear/float, esophagoscopy	Doramectin or ivermectin
Polymyositis	Electromyography, muscle/nerve biopsy, antinuclear antibody assay (ANA)	Corticosteroids
Tick paralysis	Clinical signs and tick identification	Removal of ticks, supportive care

Table 2: Overview of Diagnostics and Treatments for Diseases Known to Cause Secondary

Megaesophagus^{5, 8, 12, 14, 17, 16}

TREATMENT AND MANAGEMENT OPTIONS

Treatment for megaesophagus includes treating the primary problem, if known, and preventing regurgitation.⁸ Table 2 provides a list of these diseases and treatments for them. Additionally, the patient should be fed in a vertical position using gravity to drain food and water through the lax esophagus and into the stomach.^{8, 15} Patients should stay in this vertical position at least thirty minutes after eating and drinking.^{8, 15} Vertical feeding can be achieved by a device called the “Bailey Chair”, which dogs can be trained to enter into for feedings, or, in smaller patients, owners can hold their animals in a vertical position.⁸ Bailey Chairs can be bought, or many resources are available to assist in building a Bailey Chair or to create a make-shift chair for vertical feeding.⁶

A high-calorie diet should be fed to the patient as small, frequent meals.^{8, 9} Patients tend to do better with keeping food down when their food is mixed with water and turned into a gruel, or if food is formed into “meatballs” as these can fall down the esophagus and into the stomach easier.⁸ Consideration can be given to placement of a gastric feeding tube, but regurgitation will still occur if the patient is not placed in a vertical position for at least thirty minutes after meals, risking aspiration.⁸ Also, patients’ heads can be propped up on a pillow or with use of an inflatable collar while sleeping to reduce the risk of aspiration during this time.⁸

Prokinetics, such as metoclopramide or cisapride, can be given to increase motility through the gastrointestinal tract to empty ingesta faster.⁸ These also increase lower esophageal sphincter tone and have anti-emetic properties to further reduce the risk of aspiration.⁸ Gastric acid reducers can also be given to reduce the risk of esophagitis or rhinitis if reflux from the stomach does occur.⁸

For treatment of aspiration pneumonia, a culture and sensitivity of the exudate present in the airways should ideally be performed before initiation of antibiotic therapy.¹¹ An in-house cytology and Gram stain can be done to help guide initial antibiotic therapy choices while awaiting official results of the culture and sensitivity.¹¹ Broad-spectrum antibiotic coverage should be used, preferably intravenously, as many of these patient are inappetent.¹¹ Gram-negative coverage can be obtained by use of fluoroquinolones, aminoglycosides, or ticarcillin-clavulanic acid.¹¹ Fluoroquinolones are known to adequately penetrate the blood-bronchial barrier.¹¹ For gram-positive coverage, ampicillin, first generation cephalosporin, or ticarcillin-clavulanic acid can be considered.¹¹ Ampicillin and cephalosporins penetrate the pulmonary parenchyma but are unable to cross the blood-bronchial barrier.¹¹ It is thought that the blood-bronchial barrier is broken down enough due to inflammation making it possible for these antibiotics to get through.¹¹ Additional therapy includes intravenous fluids, oxygen therapy, nebulization, coupage, mucolytics, antioxidants, and bronchodilators.¹¹ The patient should be monitored closely via vital signs, arterial blood gas, pulse oximetry, complete blood counts, serum chemistry, and subsequent thoracic radiographs, which should all be taken into consideration when making adjustments to the treatment plan.¹¹

A study completed with four dogs who had generalized megaesophagus and recurrent aspiration pneumonia used intermittent suctioning of esophageal contents to help in management of patients with recurrent aspiration pneumonia.² The decision for initiation of this treatment was based on increased frequency of aspiration events.² An indwelling esophagostomy tube was placed for intermittent, at-home suction of esophageal content 2- 4 times a day.² All dogs were also given a gastrostomy tube for feeding.² These dogs survived for a median of 13.5 months after suctioning

therapy was started.² After initiation of this treatment, regurgitation in all four dogs was rare and two of the dogs did not have additional episodes of aspiration pneumonia.²

EXPECTED OUTCOME AND PROGNOSIS

There is an overall 74% case fatality rate for canine patients with megaesophagus and the median survival time, from diagnosis to death or euthanasia, is 1-3 months.² The most common cause of death or euthanasia in these cases is aspiration pneumonia.² If aspiration pneumonia is evident on radiographs, the patient carries a 7.69 fold increased risk of death or euthanasia and is also 2.2 times more likely to die at any moment compared to megaesophagus dogs without aspiration pneumonia.² There is a better prognosis when megaesophagus is secondary and a primary cause has been identified and treated, as the esophagus can regain partial or complete function in these cases.^{2, 8} However, despite extensive testing, 50% of dogs with megaesophagus are still diagnosed with an idiopathic etiology.¹

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