Marlow and Me

Esophageal Stricture & Dysmotility in a Dog

Presented by:
Ethan L. Baggett
Mississippi State University
College of Veterinary Medicine
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Advisor:
Harry Cridge, MVB, MRCVS
Small Animal Internal Medicine Resident

Introduction

Esophageal strictures (ES) are relatively uncommon in the dog.[1-7] However, when they do occur, they are typically secondary to esophagitis extending into the muscle layers of the esophageal wall. This inflammation leads to circumferential mucosal ulceration, fibrosis and luminal compromise. The most common cause of ES in dogs is gastroesophageal reflux associated with general anesthesia.[5] However, any cause of esophagitis has the potential to cause an esophageal stricture. The most common causes of esophagitis include gastroesophageal reflux disease (GERD), esophageal foreign bodies, chronic vomiting, ingestion of caustic substances, medications, and infectious agents.[2]

In general, regurgitation is considered the cardinal sign of esophageal disease. [2] Strictures can cause a wide range of clinical signs including regurgitation, ptyalism, odynophagia, ravenous appetite, weight loss, and respiratory disease.[1-7] Clinical signs depend on the location and diameter of the stricture, which can occur at any point along the esophagus depending on the initiating cause.[2,3] Differential diagnoses include vascular ring anomalies, extraluminal masses, esophageal neoplasia, gastroesophageal intussusception, esophageal diverticulum, hiatal hernias, megaesophagus, and cricopharyngeal dysfunction.[7]

A strong clinical suspicion for esophageal disease and stricture can be determined based on evaluation of history, clinical signs, and the medical history of the patient. Diagnosis can be confirmed via a positive contrast barium esophagram, fluoroscopy or esophagoscopy .[2] An esophagogram will identify the number, location, and length of strictures.[2] Esophagoscopy has the benefit of mucosal evaluation; however, the endoscope may only be able to diagnose the most proximal stricture, if there is severe narrowing of the esophageal lumen.[1,3] It is strongly

recommended that routine bloodwork and imaging is performed to help rule out other differential diagnoses.

Restoration of adequate oral feeding to maintain hydration and nutrition is the minimal goal of treatment for ES.[4] Current treatment options include medical management, mechanical dilatation, stenting, or surgical resection and anastomosis.[4] The current standard-of-care therapy is a combination of medical management with mechanical dilatation, either by bouginage or balloon dilation.[4] Many animals require multiple mechanical dilations; however, it is impossible to predict the number of dilation procedures required for the individual patient.[3]

Most patients (up to 85%) with BES can become functional with help of dilation procedures and medical management.[7] However, prognosis is more guarded if the stricture is several centimeters long and severe esophagitis is present.[7] Concurrent esophageal dysmotility can also impact on prognosis.

History and Presentation

Marlow is an approximately 7-year-old female spayed mixed breed dog that presented to the Mississippi State University College of Veterinary Medicine Internal Medicine Department on July 8th, 2019 for an esophagoscopy with potential balloon dilation. Marlow was diagnosed with a presumptive esophageal stricture by her referring veterinarian via a positive contrast esophagogram (barium swallow study) on June 19th, 2019 after consultation with the MSU-CVM Internal Medicine Department.

Marlow originally presented to her referring veterinarian for chronic intermittent vomiting in December of 2018. She was treated symptomatically with maropitant citrate (2

mg/kg PO q24) and a barium swallow study was planned if her clinical signs did not resolve. In early January 2019, a positive contrast esophagogram was performed. The barium moved into the stomach without difficulty, and no barium was retained in the esophagus. Maropitant citrate was continued and she was offered referral to MSU-CVM or an abdominal exploratory if her clinical signs did not resolve. In March of 2019, an abdominal exploratory was performed by her referring veterinarian. During the surgery, there was mild splenomegaly. Otherwise, there were no significant findings. Full thickness biopsies were obtained of the stomach, duodenum, jejunum, spleen, and liver and submitted for histopathology at MSU-CVM.

On April 3rd, 2019, Marlow was diagnosed with idiopathic inflammatory bowel disease (IBD) via histopathology, and exclusion of other causes of canine chronic enteropathy (CCE). Marlow was prescribed prednisone (2 mg/kg PO q12h), modified cyclosporine (5 mg/kg PO q12h), and a hydrolyzed diet. Thirteen days post-operatively Marlow re-presented to her referring veterinarian due to regurgitation, and supportive care was continued. Marlow represented again in May and early June of 2019 for worsening clinical signs. The primary veterinarian consulted with the MSU-CVM Internal Medicine service and treatment for potential esophagitis was recommended. Marlow was started on sluried food and was instructed to be fed in a modified Bailey chair position. She was diagnosed with presumptive esophagitis and started on sucralfate (1 g PO q12h), omeprazole (20 mg PO q12h) and ondansetron (8 mg PO q12h). Prednisone (10 mg PO q12h) was continued; however, cyclosporine was not continued due to financial constraints. On June 19th, 2019, a second barium swallow was performed by her referring veterinarian and Marlow was diagnosed with a presumptive esophageal stricture at T1-T2, and she was referred to MSU-CVM.

Upon presentation to MSU-CVM, Marlow was bright, alert, and responsive. Her body condition score was 2/9 and she weighed 17.2 kg. Her vital parameters were within normal limits (T: 101.7°F , HR: 100bpm, RR: panting). Her heart and lungs auscultated normally with no murmur or crackles/wheezes. She had symmetrical muscle atrophy of her head and jaw, suspected to be associated with muscle wastage and prednisone administration. Otherwise her physical examination was unremarkable. Her medications at the time of presentation included sucralfate (1 g PO q12h), omeprazole (20 mg PO q12h), ondansetron (8 mg PO q12h), prednisone (10 mg PO q12h), and cisapride (5 mg PO q12h). She had not received any medication that morning and was adequately fasted for the anesthetic procedure. A complete blood count (CBC) and serum chemistry panel was performed 3 days prior to presentation by her referring veterinarian on July 5, 2019. She had a mild leukocytosis (20.25 K/uL), mild neutrophilia (18.23 K/uL), mild lymphopenia (0.98 K/uL), and a mild eosinopenia (0.01 K/uL). On the serum chemistry, her alkaline phosphatase was mildly increased (467 U/L) and she had a severely increased lipase (>6,000 U/L). Considering her physical exam and bloodwork, she was deemed a suitable candidate for anesthesia. Additional diagnostics were offered but were not pursued due to financial limitations.

Pathophysiology

In order to understand the pathophysiology of this disease it is important to review the fundamental anatomical structures of the canine esophagus. The esophageal wall is made up of four layers: the mucosa, submucosa, muscularis, and adventitia.[7] In dogs, the muscle layer is entirely skeletal muscle.[1,7] The esophagus contains an upper and a lower esophageal sphincter.

The lower esophageal sphincter (LES) relaxes during swallowing and allows ingesta to pass into the stomach.[1]

Esophagitis is inflammation and disruption of the esophageal mucosa which causes exposure of the submucosa.[1] The esophageal mucosal lining, the LES, clearance via peristalsis, alkaline salvia, and cell turnover are the normal physiologic defenses against esophageal inflammation.[1] Esophagitis is most commonly caused by gastroesophageal reflux, chronic vomiting, foreign bodies, caustic substances, or medications.[1] The most common cause of esophagitis in veterinary medicine is anesthesia-related gastroesophageal reflux.[1] Esophagitis is not the only cause of esophageal strictures. Esophageal strictures can be caused by congenital strictures, foreign bodies, neoplasia, and from previous esophageal surgery.[1]

Certain drugs used for anesthesia in veterinary medicine have been linked to decreasing LES tone, thus increasing the chance for reflux.[1] These drugs include: morphine, thiopentone, propofol, xylazine, and atropine.[1] Also, fasting for longer than 24 hours before anesthesia is more likely to cause reflux events than fasting for less than 4 hours.[1] Currently, body position is not associated with an increase in reflux.[1] Intra-abdominal surgeries are more likely to cause reflux compared to other procedures. The procedure most commonly associated with increased reflux is ovariohysterectomy.[1] However, the ovariohysterectomy is the most common intra-abdominal surgery performed in veterinary medicine.

After the extensive damage extending through the submucosa into the muscularis, formation of fibrous connective tissue can create an esophageal stricture.[2] A circular band of scar tissue (also known as a cicatrix) is formed and compromises the esophageal lumen.[2] A vicious cycle occurs once esophagitis has developed in which the inflammation causes a decrease in LES tone, predisposing to more esophageal reflux.[2] Severe esophagitis can also decrease esophageal

motility which can contribute to additional mucosal inflammation.[2] Depending on the cause, strictures can occur anywhere along the esophagus.[2]

Diagnostic Approach

Survey radiographs are insensitive for the diagnosis of esophageal strictures or esophagitis.[1] Positive contrast esophagography is used to determine the location, size, number, and length of the stricture.[6] Endoscopy is also an option for diagnosis; however, endoscopy may be limited to only viewing the most proximal stricture, if a severe proximal stricture limits evaluation of the distal esophagus via endoscopy. In general, a combination of contrast esophagography and esophagoscopy is used for a diagnosis. If fluoroscopy is available, this can be valuable to evaluate esophageal motility and possibly observe reflux episodes.[1]

In this case, Marlow was diagnosed with an esophageal stricture by the referring veterinarian via a positive contrast barium esophagogram. A lateral radiograph of a barium swallow study showed dilation of the cranial esophagus that tapered to a narrow stricture at the level of T1-T2. No other strictures were noted.

During esophagoscopy at MSU-CVM, the diagnosis was confirmed. Approximately 25cm from the mouth, there was a focal circumferential stricture, measuring approximately 2cm in length. However, the stricture was unusual in that the size of the stricture appeared dynamic and varied with esophageal contraction. At the narrowest, the stricture was approximately 1cm in diameter; however, its largest diameter was approximately 2.5cm. The esophageal mucosa was erythematous with mild nodular change. The mucosal vessels were prominent at the area of the

stricture. The endoscope was passed through the stricture and two small irregularly shaped metallic foreign bodies were identified within the gastric fundus.

Treatment and Management

The current standard-of-care for treatment and management of esophageal strictures is a combination of dilation and medical management.[4] Medical management is used to treat the esophagitis which involves protecting the esophageal mucosa from additional injury and eliminating the underlying cause.[2] A mainstay of treatment is to reduce gastric acid to eliminate further exposure of irritating gastric secretions.[2] Proton pump inhibitors, such as omeprazole, are more effective in the management of severe esophagitis (compared to H2-receptor antagonists) induced by gastroesophageal reflux.[2] Sucralfate is commonly used, although has unproved benefit, in small animal patients with suspected esophagitis.[2] It develops viscous properties in acidic environments and theoretically will be “activated” by the acid in the esophagus in the area of the inflammation, adhere, and protect the esophageal mucosa.[2] It is important to give this medication at least 30 minutes – 2 hours before food and before any other medications. Administration with food inhibits its ability to adhere to the damaged mucosa, and administration with other medications can decrease the absorption of those medications.[1] Glucocorticoids are often used with the goal to reduce esophageal inflammation and to inhibit further formation of fibrous connective tissue.[2] Systemically administered glucocorticoids have not been proven beneficial.[1] However, human studies have investigated the use of local glucocorticoid therapy administered during esophagoscopy, in which intralesional triamcinolone was associated with increased intervals between dilation episodes.[2] Prokinetics are also a mainstay of medical management. Metoclopramide and cisapride are used to increase LES tone and increase gastric

emptying.[1] However, cisapride seems to be more superior than metoclopramide.[1,2] Overall, the goal of medical management is to resolve regurgitation and minimize ongoing esophagitis.

When stricture dilation is indicated, the most common approach is either balloon dilation or esophageal bougienage.[2] Balloon dilation involves passing an inflatable balloon into the stricture under endoscopic guidance.[1] Once in place the balloon is expanded using saline to create a radial force on the stricture. This force causes it to break down and increases the size of the esophageal lumen.[2] Bougienage is an alternative technique which involves the passage of a rigid dilator of gradually increasing size through the stricture site. [2] There have been many debates on safety and effectiveness of balloon dilation vs. bougienage.[1] The balloon dilation exerts primarily radial forces at the stricture site, and the bougienage exerts radial and shear forces at the stricture site.[2] Generally, due the absence of the shear force, it has been suggested that balloon dilation is safer than using bougienage.[1] However, retrospective studies have shown no significant differences.[1] There is currently no established guidelines for the point at which to stop the dilation.[2] The basic idea is to stop dilating when the trauma is though to be a greater risk than waiting to see if the stricture recurs.[2] Potential complications of bouginage or balloon dilation include hemorrhage, esophageal rupture, esophageal diverticulum formation, infection, aspiration, and pain.[1]

In our case, Marlow was anesthetized and placed in left lateral recumbency for an esophagogastroduodenoscopy with potential for balloon dilatation. The focal circumferential stricture was located approximately 25cm from the mouth and measured approximately 2cm in length and was 2.5cm in diameter at its largest. A bougie was not available for this procedure. The largest esophageal balloon was 2cm in diameter and unfortunately did not provide sufficient

radial force against the esophageal stricture. Due to a lack of finance and inability to repeat the procedure, three esophageal balloons were insufflated concurrently within the stricture, in an attempt to provide sufficient radial force on the stricture site. No current studies have evaluated the performance of insufflation of multiple concurrent esophageal balloons, and further studies are recommended before this technique can be recommended. This resulted in mild stretching of the stricture. The balloons were inflated (using saline) and held for approximately one minute and this was repeated for a total of two one-minute inflations. Post inflation, a 2cm stomach tube was passed readily through the stricture, and the mucosa was mildly erythematous and there were no complications. Submucosal triamcinolone injection, mitomycin C application &/or PEG tube placement was considered; however, it was not performed due to cost. Mitomycin C is also inconsistently available.

Given the size of this stricture, it is unclear whether it was contributing to a mechanical cause of regurgitation, or whether the primary cause of regurgitation was esophageal dysmotility. Post-operatively, due to the potential of esophageal dysmotility, her cisapride dose frequency was increased. A topical analgesic (Magic mouthwash) was also prescribed. She was also discharged with instructions to continue the prednisone (10 mg PO q12h), ondansetron (8 mg PO q12h), omeprazole (20 mg PO q12h), and sucralfate (1g in a slurry PO q12h) as previously prescribed. At discharge, her family was informed that she may require multiple ballooning procedures and it is impossible to predict how many (if any) more she may need. It was also discussed that the stricture may not be the primary cause of the regurgitation and additional diagnostics are indicated.

Case Outcome

Marlow recovered from the procedure and anesthesia uneventfully, with no known episodes of regurgitation. Her owners were informed to continue Marlow on her slurried diet, and to continue feeding her in a modified Bailey chair position. She was discharged from MSU-CVM on July 8th, 2019 with instructions to have a recheck with her referring veterinarian in one week.

Despite initial improvement, in August 2019, Marlow presented to her referring veterinarian due to return of her original clinical signs. Marlow's owners were offered referral for additional diagnostics and treatment of esophageal dysmotility and/or a second esophageal ballooning procedure. However, due to her poor quality of life and the owner's financial limitation, her family elected to have her humanely euthanized.

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