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DIAGNOSIS AND TREATMENT OF ESOPHAGEAL STRICTURE IN A DOG

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ABSTRACT

A 1.5-year-old male Corgi dog was presented by his owner to our institute with a 4-day history of dysphagia and regurgitation. The dog underwent a physical examination followed by hematological tests, X-rays, and endoscopy. Creatine kinase contents were above the average level, referring to inflammation in the dog. We detected narrowing of the upper esophagus was detected after feeding barium meal. We also found a stricture on narrowing the esophagus's lumen. Surgical resection of the stricture was performed. After the operation, we gave the dog anti-inflammatory and anti-regurgitation drugs for five days. With excellent postoperative care, the dog was eventually cured. Based on the above findings, the dog was diagnosed with esophageal stricture secondary to esophagitis. We concluded that surgical resection is the proper way to treat severe esophageal stricture in adult dog for complete recovery, which was successfully achieved in this case.

***Keywords*:** Esophageal stricture; Dog; Inflammation; Surgical resection

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1. INTRODUCTION

Canine esophageal stricture refers to the narrowing of the esophagus, which prevents food from entering the gastrointestinal correctly (Shalvey et al. 2022). The stricture can be congenital, resulting from malformation in the esophageal wall during development, and can be acquired due to trauma, inflammation, tumors, or other conditions (Luedtke et al. 2003). The incidence of congenital esophageal stricture is rarely low in small animal species (Shalvey et al. 2022), and acquired esophageal stricture is uncommon (Adamama-Moraito et al. 2002). Stricture may occur at any point along the esophagus, and the clinical signs include regurgitation, dysphagia, weight loss, and respiratory signs (Adamama-Moraito et al. 2002). According to pathological typing, esophageal strictures can be divided into benign and malignant strictures (Da Riz et al. 2021).

The accurate diagnosis of esophageal strictures relies on a combination of presumptive and definitive methods. The former, including esophagogram and esophagoscopy, provide complementary information. An esophagogram, for instance, not only pinpoints the location, diameter, and length of the stricture but also paints a vivid picture of the condition. On the other hand, an esophagoscopy offers a direct view of the stricture, allowing for a more detailed evaluation of the mucosa. The definitive diagnosis, however, is based on endoscopic biopsy, histopathology following surgical resection, or post-mortem examination, which provide a conclusive understanding of the condition.

The main therapeutic options for esophageal stricture include surgical resection, endoscopic dilation, and medical therapy (Pregun et al. 2009). Surgery is the most common way to treat esophageal stricture in dogs, which can effectively relieve the symptoms by removing the stricture, rebuilding the esophagus, and dilating the stricture (Huang et al. 2022). Dilatation is a non-invasive treatment that involves placing a dilator or stent in the stricture through an endoscope. It is often conducted in less severe cases of esophageal stricture. The disadvantages of dilatation include tissue growth, stent migration, stent removal, hemorrhage, perforation, and the requirement of multiple dilation procedures (Grabowski and Andrews 1996; Huynh 2019). Medical therapy is often conducted after surgery or endoscopic dilation to relieve symptoms and reduce the need for repeated dilations; commonly used medications include antibiotics and anti-inflammatory drugs (Patil et al. 2017).

This case report presents a 1.5-year-old male Corgi dog with a 4-day history of eating difficulty and vomiting, diagnosed with canine esophageal stricture. The dog was successfully treated by surgical resection.

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The following sections detail the diagnosis, treatment, and outcomes, providing valuable insights for clinical reference.

2. MATERIALS AND METHODS

2.1. Case Information

The Corgi dog was male and 1.5 years old. The dog owner described that the dog showed eating difficulty and vomiting four days ago, with no previous history and no abnormal food intake. We evaluated the general condition of the dog, and the results were listed below. The dog was in good mental condition, weighing 6.4kg, with a body temperature of 38.5°C, respiratory rate of 27 times/min, and heart rate of 115 beats/min. Some food residues were found in the anterior part of the mouth, and there was no sign of dehydration.

2.2. Complete Blood Count

After the physical examination, the whole blood was collected from the forelimb veins of the affected dog for blood count (Table 1). No obvious abnormality was observed; the total number of white blood cells, lymphocytes, and the percentage of lymphocytes were all within the normal range.

Table 1: Results of whole blood count

Parameters	Units	Results	Reference range
White blood cell count	10^9/L	8.57	6.00-17.00
Neutrophils	10^9/L	6.24	3.62-12.30
Lymphocytes	10^9/L	2.18	0.83-4.91
Monocytes	10^9/L	0.15	0.14-1.97
Eosinophils	10^9/L	0.00	0.04-1.62
Basophils	10^9/L	0.00	0.00-0.12
Neutrophil	%	72.8	52.0-81.0
Lymphocyte	%	25.5	12.0-33.0
Monocyte	%	1.7	2.0-13.0
Eosinophilic	%	0.5	0.5-10.0
Basophil	%	0.0	0.0-1.3
Red blood cell count	10^{12}/L	6.20	5.10-8.50
Hemoglobin	g/L	162	110-190
Hematocrit	%	45.3	33.0-56.0
Mean corpuscular volume	fL	73.1	60.0-76.0
mean corpuscular hemoglobin	pG	26.1	20.0-27.0
Mean corpuscular hemoglobin concentration	g/L	357	300-380
Red cell distribution width, coefficient variation	%	13.1	12.5-17.2
Red cell distribution width, standard variation	fL	38.7	33.2-46.3
Platelet count	10^9/L	380	117-490
Mean platelet volume	fL	8.2	8.0-14.1
Platelet distribution width	fL	15.6	12.0-17.5
Plateletcrit	%	0.31	0.090-0.580

2.3. Biochemical Examination

Plasma separated from the whole blood of the affected dog was used for biochemical examination. Albumin, total protein, globulin, albumin/globulin ratio, calcium, glucose, urea nitrogen, inorganic phosphorus, amylase, cholesterol, alanine transaminase, total bilirubin, alkaline phosphatase, creatinine, urea nitrogen/creatinine ratio and creatine kinase were tested for any abnormality in the body. Most of the biochemical parameters (Table 2) were within normal levels, except for alkaline phosphatase (ALP) and creatine kinase (CK). ALP decreased a little bit while CK increased. Low ALP may indicate malnutrition, liver disease, anemia, and other dog problems (Komnenou et al. 2006). CK is an enzyme found in muscle cells and released into the bloodstream when muscle tissue is damaged. A high creatine kinase may indicate muscle damage or inflammation in the dog (Kim and Wierzbicki 2021). These results suggest that inflammation caused by muscle damage existed in the dog, and the dog may be malnourished due to prolonged failure to eat properly.

2.4. X-ray Examination

Plain X-ray image showed no dilation of the esophagus and no foreign bodies throughout the esophagus (Fig. 1A; Fig. 1B). Further X-ray barium meal examination revealed that a small amount of barium residue and dilation were observed in the esophagus at C3-C4 of the anterior neck segment (Fig. 1C; Fig. 1D). Reexamination after

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barium feeding showed that some barium meals remained at C3-C4 levels in the anterior cervical segment, indicating possible esophageal stricture. As for the outline of the heart shadow and the density of the lung lobes, no apparent abnormalities were found.

Table 2: Results of blood biochemical examination

Parameters	Units	Results	Reference range	Organs related
Albumin	g/L	36.10	22-44	Liver and kidney
Total protein	g/L	64.10	52-82	Whole body
Globulin	g/L	28.00 2	3-52	Whole body
Albumin/globulin ratio		1.30		
Calcium	mmoL/L	2.44	1.98-2.95	Whole Body
Glucose	mmoL/L	7.46	3.89-7.95	Whole Body
Urea nitrogen	mmoL/L	3.01	2.5-9.6	Kidneys
Inorganic phosphorus	mmoL/L	0.94	0.81-2.2	Kidney
Amylase	U/L	1166	400-2500	pancreas
Cholesterol	mmoL/L	5.86	2.84-8.26	Whole Body
Alanine transaminase	mmoL/L	34	10-118	Liver
Total bilirubin	g/L	4.24	2-15	Whole body, liver
Alkaline phosphatase	U/L	19 ↓	20-150	Liver
Creatinine	mmoL/L	89	27-124	Kidney
Urea nitrogen/creatinine ratio		8		
Creatine kinase	U/L	314 ↑	20-200	Heart muscle, skeletal muscle

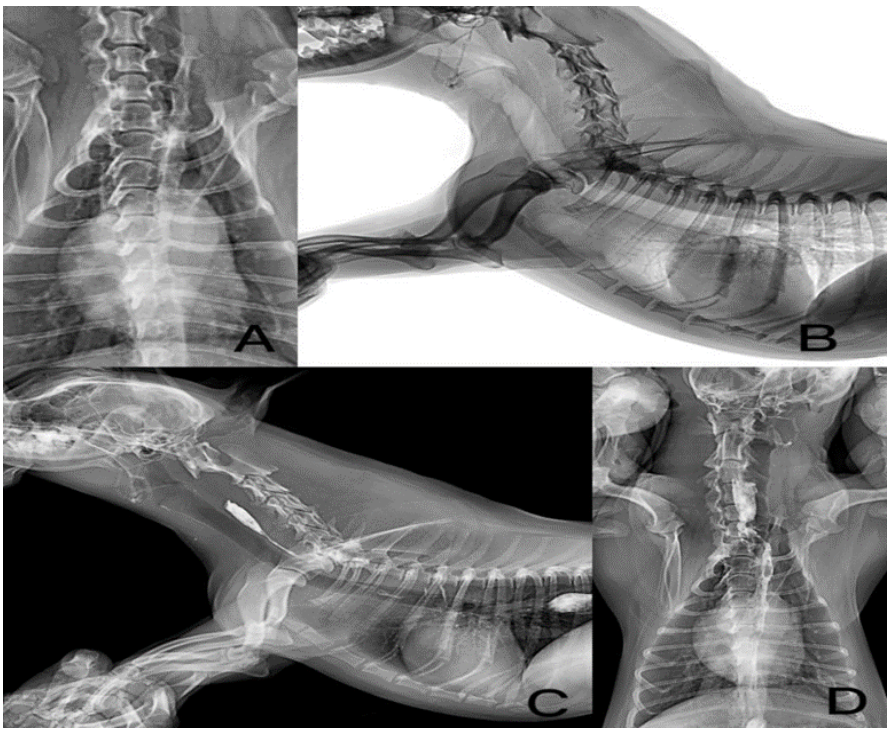

Fig. 1: X-ray images of the dog.

2.5. Endoscopy

Under endoscopic visualization, a stricture, approximately 1cm in length and 2mm in diameter, was revealed at the narrowing lumen of the upper esophagus, 23cm away from the oral incisor (data not shown). No significant abnormality was found in the posterior segment of the esophagus.

3. RESULTS

The results of the hematological tests showed the affected dog had a normal hematological profile, except for alkaline phosphatase and creatine kinase. The increase in creatine kinase indicates the dog had inflammation caused

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by muscle damage. X-ray barium meal examination revealed a possible esophageal stricture. Endoscopy of the esophagus confirmed the stricture, which was 1cm in length and 2mm in diameter.

Considering the above-mentioned results, the affected dog was determined to have an esophageal stricture secondary to esophagitis. Thus, the dog owner's chief complaint of vomiting must be regurgitation (Jiang 2023).

3.1. Surgical Treatment

The affected dog fasted for eight h before initiating the surgical procedure (Kuang et al. 2007). Half an hour before the operation, the dog was given atropine 1mg intramuscularly, Doapride 1 mL subcutaneously, and Bayri 1 mL subcutaneously. Then, a 4 mL propofol emulsion injection was administered intravenously to induce anesthesia, followed by endotracheal intubation.

The main details of the surgery were described below: 1.5% isoflurane was supplied to maintain dog anesthesia during the operation. An endoscope was inserted into the esophagus to look for the stricture (Fig. 2.1) and to facilitate surgical locating. An incision was made on the surface of the stricture, and the esophagus was exposed. Under the guidance of an endoscope (Fig. 2.2; Fig. 2.3), we confirmed the stricture position, and then we opened esophagus wall accordingly, the tissue hyperplasia on the stricture was exposed and excised (Fig. 2.4). Immediately after resection, endoscope was pushed through the esophagus (Fig. 2.5; Fig. 2.6), to check whether the esophagus is blocked. The endoscope passed the esophagus lumen, the excised esophagus was unobstructed, and the esophagus was sutured (Fig. 2.7). Endoscope was used again to confirm whether the sutured esophagus was unobstructed (Fig. 2.8). It turned out that the endoscopy was able to pass through the esophagus smoothly, after this confirmation we continued to close the surgical wound. The operation ended with the interrupted supply of isoflurane and the pulling out of the endotracheal tube when the dog had a laryngeal reflex. The dog was transferred to the inpatient unit for further treatment and nursing care.

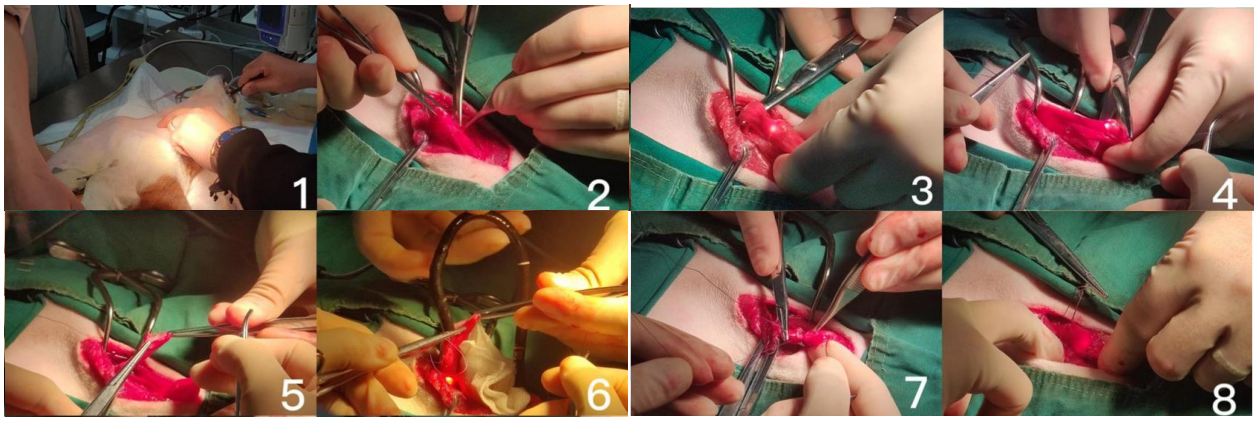

Fig. 2: Surgical resection of the stricture on the esophagus.

3.2. Postoperative Treatment

The postoperative wound of the dog was cleaned with iodine twice a day, respectively, in the morning and evening. Medical therapy was given to the dog for 5 days. The prescription was detailed as follows: 0.9% NaCl solution 100mL+ ampicillin sodium 0.5g, iv, to prevent secondary infection; 5% glucose solution 100mL+ Putron injection 10mL+VC 0.5mL+VB6 0.5mL, iv, and 5% glucose solution 100mL+ albumin (dog) 5mL, iv, to maintain the nutrition of the dog and promote wound healing; 5% glucose solution 100mL+ omeprazole 15mg, iv, to reduce regurgitation.

After five days of medical treatment, the dog was fed a small amount of liquid food. No regurgitation was detected, and the dog showed a good appetite and no other abnormal conditions.

4. DISCUSSION

In this case, the dog had a short history of dysphagia and regurgitation. We gave physical examination to the dog, and no obvious disorder was found except for some food residue left at the anterior part of the mouth. Because the dog had eating difficulty and no lesion on the mouth mucus, we suspect the dog had esophageal disease.

Most esophageal diseases have similar clinical symptoms, including regurgitation, dysphagia, hypersalivation, and may be accompanied by aspiration pneumonia (Chijimatsu et al. 1983; Funes and Ruaux 2020; Kollannur et al. 2024). To make an accurate diagnosis, we conducted a hematology test to investigate whether there was an inflammatory reaction in the dog, we took an X-ray examination to detect abnormalities in the digestive system and did an endoscopy to observe the lesions.

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Hematology tests and biochemical examination showed that the dog's alkaline phosphatase content was a little bit under the normal level, suggesting signs of malnutrition (Komnenou et al. 2006), which may have been caused by starving as the dog had eating difficulty; on the contrary, creatine kinase content was higher compared to the normal level, indicating muscle damage and inflammation in the dog (Kim and Wierzbicki 2021).

Plain X-rays failed to detect any abnormality in the dog; on the contrary, X-ray barium meal examination showed a narrowing of the esophagus, indicating esophageal stricture. Once suspected, the diagnosis of esophageal strictures is simple in most cases (Glazer et al. 2008). Through endoscopy of the esophagus, a stricture, 1cm in length and 2mm in diameter, was confirmed. The much-narrowed esophagus lumen could prevent the food from passing through the esophagus, by clinical symptoms of dysphagia and regurgitation.

As the narrowing of the esophagus in congenital esophageal stricture is present from birth, and typical regurgitation begins after implementation of a solid diet post-weaning (Shalvey et al. 2022), congenital esophageal stricture in this case is excluded because the symptom in this case begun at 1.5 years old. Thus, the stricture on the esophagus of the dog was acquired. Acquired benign esophageal stricture usually occurs secondary to esophagitis that extends into the muscle layers of the esophageal wall and results in scar tissue formation. The most common causes of esophagitis include chemical, thermal, traumatic, infectious agents, persistent vomiting, esophageal foreign body, and gastroesophageal regurgitation (Adamama-Moraito et al. 2002; Sellon and Willard 2003). Although the owner of the dog denied abnormal food intake from the onset of dysphagia and regurgitation, clinical signs of acquired esophageal stricture become evident till 8 to 21 d following the exposure of the animal to the suspected causative agent. A mature stricture needs time to form (Adamama-Moraito et al. 2002). Thus, we cannot exclude a possible insult on the esophagus of the affected dog before clinical signs occur. The inflammation caused by muscle damage indicated by biochemical tests further proved a potential insult. We strongly suspected the esophageal stricture observed in this case was caused by muscle damage on the esophageal wall. The disease was primarily diagnosed as esophageal stricture secondary to esophagitis. As we failed to conduct a biopsy on the stricture lesion, the pathological type of the stricture is unknown.

Esophageal stricture in dogs is a potentially severe digestive disorder that can worsen over time (Wijekoon et al. 2018). Considering the seriously narrowed esophageal lumen (2mm in diameter) caused by the stricture, we took an intervention procedure on the affected dog immediately after diagnosis. We did not choose endoscopic dilation because the stricture in this case is not elastic enough to place a stunt; there was a high risk of esophageal perforation and bleeding (Busch 1980). Thus, surgery was performed to remove the tissue hyperplasia causing the stricture, which is more radical and efficient (Faulkner et al. 1976). The surgery process was smooth, but we failed to find an exotic insult at the stricture site. A sharp object may have passed through the esophageal lumen that caused damage to the esophageal wall, resulting in esophageal inflammation and regurgitation and consequently stimulating the hyperplasia of fibrous tissue, forming the stricture. With post-operative medication and nursing care, the dog showed a good prognosis. The dog was discharged with our suggestion: pay more attention to the food intake and the overall well-being of the dog, avoid overeating, eat an unbalanced diet, and eat food with sharp objects.

In the follow-up visit, the dog was full of vitality and recovered completely. From the excellent outcome after surgical resection, we finally diagnosed the disease as benign esophageal stricture secondary to esophagitis.

5. CONCLUSION

The 1.5-year-old Corgi dog presented in this case with dysphagia and regurgitation was adequately diagnosed and treated. The disease was defined as benign esophageal stricture secondary to esophagitis. It is simple to confirm the stricture by X-rays and endoscopy upon suspicion. Dysphagia and regurgitation are the common symptoms associated with esophageal stricture. Veterinarians should observe the symptoms personally because the dog owner could be misleading, such as when regurgitation could be considered as vomiting. Treatment of canine esophageal stricture is based on the dog's physical condition and the stricture's severity. For much-narrowed cases or lesions that lack elasticity, surgical resection ensures the efficacy of treatment and the safety of the diseased animal. In addition, the excellent outcome of the surgery may help define the benign feature of the esophageal stricture. Postoperative medication was also essential to control regurgitation, inflammation, and wound infection.

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Author's Contribution

Wei Luo performed hematological tests, data interpretation, results analysis and discussion, manuscript writing and revision, Ping Liu did X-rays, endoscopy, treatment, formal analysis and revision of the manuscript.

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