Medical management of esophageal perforation secondary to esophageal foreign bodies in 5 dogs

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Abbreviations

EFB esophageal foreign body

PEG percutaneous endoscopically-placed gastrostomy

Abstract

Objective – To describe 5 cases of conservative management of substantial esophageal perforation in dogs.

Series summary – Five dogs presented with an esophageal foreign body (EFB) and resultant esophageal perforation. Clinical signs at presentation included tachycardia, tachypnea, and increased respiratory effort. Thoracic radiography was performed in all cases, and in each case pleural and mediastinal effusion was present suggesting esophageal perforation prior to endoscope-guided removal. A full thickness esophageal defect was visualized after foreign body removal in 4/5 cases. Treatment included IV crystalloid fluid therapy, IV antimicrobials, analgesia, and proton pump inhibitors in all cases. Two dogs had a percutaneous endoscopically-placed gastrostomy feeding tube placed and 1 dog received prednisolone sodium succinate IV because of marked pharyngeal inflammation. Complications after EFB removal included pneumothorax (n = 2) and pneumomediastinum (n = 4). Four of the 5 dogs survived to discharge and did not have complications 2 – 4 weeks following discharge. One dog was euthanized as result of aspiration pneumonia following EFB removal.

New or unique information provided – Traditionally, surgical management of esophageal perforations has been recommended. This can be a costly and invasive procedure and requires a high degree of surgical skill. In this report, conservative management of substantial esophageal perforation in 5 dogs is described; medical management may be a viable treatment option in dogs with perforation of the esophagus due to EFB.

Keywords: canine, endoscopy, esophagoscopy, esophagus, mediastinitis, pneumothorax

Introduction

Esophageal foreign body (EFB) obstruction is considered an emergency that requires prompt treatment.¹ Bones and osseous material are the most commonly reported EFB with a prevalence of 47% – 94%; however, fish hooks, rawhide chews, pieces of plastic or metal, balls, and children's figurines have also been reported as EFBs.²⁻¹⁰ Typical locations for EFBs are where the esophagus is narrower: at the pharyngeal esophagus, the thoracic inlet, near the heart base, and the distal esophagus.^{2-5, 7, 9-11} Many studies suggest that the distal esophagus is the most common location for EFBs.^{2, 4, 7, 9-12} Clinical signs associated with EFB obstruction include regurgitation, anorexia, salivation, and pain during swallowing; however in some cases, clinical signs can be absent.^{7, 8, 10, 13, 14} Small breed dogs are reported as being predisposed to EFBs.⁷⁻¹⁰ Diagnosis of EFB is usually confirmed by thoracic imaging^{5, 13} and treatment of choice is per-oral endoscopic removal or advancement of the foreign object into the stomach.^{9, 13, 15} Esophagitis, often with ulceration and subsequent stricture formation, is commonly reported.^{7, 9, 11, 13, 15} Higher number of complications during removal, esophageal perforation, haemorrhage within the esophagus and dogs that undergo surgery after attempted per endoscopic removal have been reported as risk factors for death associated with EFBs.¹⁰

Esophageal perforation may also be present due to pressure necrosis from prolonged contact, or as a result of the removal of a tightly lodged EFB.⁹ There are few reports of conservative management of esophageal perforation in the veterinary literature as this complication tends to be managed surgically.^{5, 8, 12} It has been reported by that 1 dog with esophageal perforation was managed medically for esophagitis and survived.⁸ There has also been report of esophageal perforations up to 12 mm that have healed without surgical intervention.⁵ This is in contradiction to a large study where there was a 100% mortality in 5 dogs after esophageal perforation had occurred with concurrent pneumothorax.¹⁰ Medical management for esophageal perforation could be considered in some cases, particularly where surgery has been declined. This case series reports the medical management of esophageal perforation in 5 dogs.

Series Summary

Between the years of 2005 – 2015, 5 dogs with esophageal perforations were treated using medical management, with 4 dogs surviving the incident. The cases were 4 female neutered dogs and a male neutered dog with ages ranging from 2 – 12 years. Breeds included 2 mixed breed dogs, a Border Collie, a Cavalier King Charles Spaniel, and a Shih Tzu. In each dog, there was a suspicion the EFB had been present for a duration of 2 – 7 days.

Upon presentation 4 dogs were quiet, alert, and responsive and 1 dog was distressed with increased breathing effort that required oxygen supplementation. On physical examination, common clinical signs included tachycardia (140 – 170/min), tachypnea (60/min), and increased respiratory effort. One dog was pyrexemic with a temperature of 39.8°C (102°F), with tachycardia and tachypnea. The remaining vital parameters were within normal

limits for all dogs in the series. Initial diagnostic investigation included a CBC ($n = 2$), venous blood gas analysis (1), serum biochemistry profile (1), and thoracic radiographs (5). The CBC showed a moderate neutrophilia in both dogs in which it was performed ($16.54 \times 10^9/L$ [$16,540/\mu L$] and $15.1 \times 10^9/L$ [$15,100/\mu L$]), and a mild regenerative left shift ($0.64 \times 10^9/L$ [$640/\mu L$]) with evidence of toxic change was present in 1. The serum biochemistry panel showed a moderately increased albumin concentration (42 g/L [4.2 g/dL]) and was otherwise unremarkable. Venous blood gas analysis showed a respiratory and metabolic alkalosis ($\text{pH } 7.417$), PvCO_2 37.9 mm Hg and bicarbonate concentration 24.0 mEq/L (24 mmol/L).

Radiography confirmed the presence of an EFB in the distal esophagus in 4 dogs, and in the cranial esophagus just caudal to the oropharynx in 1 dog (Figures 1 and 2). In addition, all 5 dogs displayed strong evidence of esophageal perforation manifesting as pleural effusion ($n = 4$) and mediastinal effusion (5) at initial evaluation. General anesthesia was induced with alfaxalone^a (2 mg/kg) or propofol^b (4 mg/kg) and maintained with inhalational isoflurane and oxygen. Esophageal endoscopy was then performed with the dogs in left lateral recumbency. In 4 of the 5 cases the entire EFB was removed orally (with moderate difficulty) and in 1 case 1 segment of the EFB was removed orally (with significant difficulty) and the second segment was pushed into the stomach for digestion. In 2 of the 5 cases, the type of EFB was recorded. The first was a lamb rib bone measuring $55 \times 9 \text{ mm}$ and the other was an ovine humeral bone measuring $40 \times 50 \text{ mm}$. In 4 cases, a full thickness defect with estimated size ranging from $5 - 10 \text{ mm}$ in diameter was visible endoscopically; therefore, according to the Savary Miller classification system these esophageal defects would be grade IV, the most severe grade.⁶ In 1 case, the defect could not be visualized; however, pneumomediastinum

was present on thoracic radiography prior to removal, suggesting a pre-existing perforation. Complications following endoscopic EFB removal in these cases included pneumothorax (n = 2) and pneumomediastinum (4). Pneumothorax was treated with needle thoracocentesis in both dogs when increased breathing effort was noted; there was no comment regarding the timing in relation to the EFB removal when thoracocentesis was performed.

Two of the 5 dogs required unilateral indwelling chest drains; these were the same dogs that required needle thoracocentesis. The nature and volume of the removed substance was not recorded. The chest drains were removed at 24 and 72 hours in each dog, respectively, as there was no ongoing fluid or air production.

All dogs received IV crystalloid fluid therapy at maintenance rates, parenteral antimicrobials (ampicillin^c 20 – 30 mg/kg q 8 h; metronidazole^d 10 mg/kg q 12 h; and in 1 case enrofloxacin^e 5 mg/kg q 24 h), analgesia (methadone^f 0.2 mg/kg IV as needed; buprenorphine^g 0.01 mg/kg IV as needed; and tramadol^h 2 mg/kg PO q 12 h) and IV proton pump inhibitors (pantoprazoleⁱ 0.5 – 1 mg/kg q 24 h). One dog was administered IV prednisolone sodium succinate^j (0.5 mg/kg q 24 h) because of marked pharyngeal inflammation; this was the dog that presented with increased breathing effort. Oral antimicrobials, analgesia, and proton pump inhibitors were continued for up to 14 days following discharge.

Two of the 5 dogs had percutaneous endoscopically-placed gastrostomy (PEG) feeding tubes (20-Fr) inserted prior to recovery from general anesthesia. Both dogs were disallowed oral food or water for the first 24 hours following removal of the EFB. Feeding was commenced at 1/3 of resting energy requirement with soft commercial canned dog food^k

via PEG tube 24 hours placement and was increased to full resting energy requirement by day 3. Both dogs began voluntary oral intake of small amounts of food and water within 4 days of EFB removal. The remaining 3 dogs did not have a feeding tube inserted and were disallowed food or water by mouth for 12 hours, 1 day, and 3 days after EFB removal, with small amounts of oral food and water being offered afterwards.

Four of the 5 dogs improved and were discharged 4 – 10 days after presentation. One dog developed severe aspiration pneumonia prior to EFB removal; this was the dog that was in respiratory distress at initial presentation. Mechanical ventilation was recommended but declined by the owners, and the dog was provided 70% fraction of inspired oxygen.

Euthanasia was elected based on financial limitations 24 hours after EFB removal due to continued deterioration. The remaining 4 dogs were clinically normal 2 – 4 weeks after discharge with no evidence of complications (no reported vomiting, regurgitation) and have remained clinically unaffected by the EFB to date of writing (range 6 – 36 months).

The cost of surgical management of esophageal perforation including post-operative care in comparison to endoscopic removal with aftercare is approximately 3.2:1 at the institutions involved in these cases. Endoscopic removal with placement of a PEG tube has a cost ratio of 1.6:1 compared to endoscopic removal alone. This cost calculation does not account for management of post-procedural complications such as aspiration pneumonia.

Discussion

This case series demonstrates successful medical management of esophageal perforation in 4/5 dogs (80%). In 1 dog, euthanasia was elected due to complications associated with aspiration pneumonia rather than due to complications associated with esophageal perforation

per se. Esophageal perforation may result from trans-mural pressure necrosis due to presence of the EFB or as a complication associated with removal, and carries a mortality rate ranging from 2.5% – 100% in published reports.^{8-10,13,14} Previous recommendations in people were that large defects in the esophageal wall, defects of any size with concurrent signs of sepsis, non-localized mediastinitis, or other thoracic cavity involvement required surgical exploration and repair.¹⁶ However, recommendations for management of esophageal perforations in humans is trending towards medical management.¹⁷ In adults, there was no difference in the outcome of patients managed medically compared to esophageal perforations managed surgically, with surgical patients experiencing longer hospitalization.¹⁷ In human neonates medical management of esophageal perforations is preferred.¹⁸ People managed medically for esophageal perforation generally receive parenteral antimicrobial therapy for 7 – 14 days, nil *per os*, parenteral nutrition, and draining procedures as required; clinical deterioration would be an indicator for surgical management.¹⁸ Successful medical management of esophageal perforation has been documented in dogs with defects of up to 12 mm.^{8,9,14}

Most of the veterinary literature supports surgical repair of esophageal perforations with little literature discussing medical therapy as an option.^{13, 14, 19} Surgical management of esophageal perforations is invasive, requires a high degree of surgical skill, and has been associated with a 14% risk of incisional dehiscence.^{5,14} The high dehiscence risk may be due to delayed wound healing as a result of the relatively poor esophageal blood supply and movement of the surgical site due to peristalsis.⁵ EFB that have been surgically removed have a reported mortality rate of 23.1% and the mortality rate doubles to 57% (4/7 dogs) for cases of esophageal perforations managed surgically.^{8,10} Burton et.al¹⁰ reported a 100% (5/5 dogs)

mortality for dogs that had EFB removed endoscopically where surgery had been declined; all dogs had sudden deaths resulting from esophageal perforation and pneumothorax. The mortality rate for conservative management was 42% (5/12 dogs) mortality in the study by Parker et al¹⁴ and 20% (1/5 dogs) in this case series. The reported 100% mortality rate¹⁰ from esophageal perforations during endoscopic removal could be biased as there would have been more aggressive attempts at removal as surgery had been declined in these cases. Whilst the case numbers with a successful outcome are small, they demonstrate the potential for survival following medical management of esophageal perforation associated with EFBs. The shift in management of esophageal perforations in human medicine is also supportive of the potential role of medical management.¹⁶⁻¹⁸

It should be noted that there are differences between people and dogs regarding the anatomy of the esophagus, etiology and duration to presentation for esophageal perforation. The human esophagus comprises proximally striated muscle and distally smooth muscle with a mixture of the 2 muscle fibers in the middle section¹⁷; in the dog, the esophagus is solely striated muscle.¹ We postulate that the differences in esophageal muscle composition may also play a role in the duration of healing. The majority of esophageal perforations in people are iatrogenic due to an endoscopy procedure or esophageal neoplasia, whereas EFBs are a major cause of esophageal perforation in dogs.^{1,17} The time lapse between onset of signs and treatment is usually longer in dogs.^{7,8,17} Also, in veterinary medicine financial limitations may play a larger role in treatment choice than in people.

The initial case reports detailing conservative management of esophageal perforations were published at a time when veterinary endoscopy was in its infancy and surgical repair may have been the only option for affected dogs other than euthanasia.^{5, 14} Less invasive

approaches continue to evolve and offer options for those who would otherwise have none.

The experimental use of over-the-scope clips was used successfully to repair iatrogenic esophageal perforations in dogs with an average repair time of 30 minutes.²⁰ In this study however, the esophageal perforations were created with a knife blade, and therefore are not a true representation of the esophageal lesions caused by EFBs seen in practice. These experimentally-induced lesions were simple incisions of healthy tissues that were repaired soon after injury. Therefore, the use of such a method may not be successful in the clinical setting.

Management of esophageal perforations and cases of necrotic esophagitis with pharyngostomy tube placement have been reported to decrease mortality and recovery times in people.¹⁷ A potential disadvantage of pharyngostomy tubes is that the physical presence of the tube may decrease healing of injured esophageal mucosa.¹⁷ A gastrostomy tube completely bypasses the esophagus and eliminates further esophageal injury^{7,9} however, we believe that the presence of food within the esophageal lumen may aid mucosal healing and prevent fibrosis. However, assisted feeding in cases of esophageal perforation did not decrease length of hospitalization.¹³ In this case series 2 surviving dogs were managed with PEG tubes and 2 were managed without. Due to the retrospective nature of this case series, there were no established criteria for the placement of a PEG tube, and decision to place a feeding tube was likely based on clinician preference and owner finances. In human neonates, assisted nutrition is the current recommendation and the decision for assisted nutrition should be based on the clinical case.¹⁸

This retrospective case series has several limitations. Ideally, a contrast esophagram should have been performed to definitively diagnose perforation in the 1 case in which it was

not directly visualized during esophagoscopy. In people the use of contrast agents is recommended for the definitive diagnosis of esophageal perforation.¹⁷ Contrast material can reduce endoscopic image quality, the leakage of barium into the mediastinum can result in mediastinitis and aspiration of iodinated contrast material can cause pulmonary edema; therefore the use of contrast agents in these cases can be controversial.⁷ The pneumomediastinum present in that case was highly suggestive of a perforation, and thus the dog was included in this series. A different clinician performed each esophagoscopy; therefore, there were likely variations in the assessment of esophagitis and perforation size was estimated, not definitively measured. The lesions were also endoscopically magnified making definitive measurement challenging. It would have been preferable to list the measurement as a percentage of the circumference of the esophagus. Despite the Savary-Miller classification system being used here, that system does not account for the general extent of esophageal damage, since an esophageal perforation classifies a case as a Class IV, which is the most severe class of injury.⁶ Unfortunately, none of the cases had follow up endoscopy.

Given the shift in recommendations in human literature and the cases reported herein, the authors believe medical management should be offered in cases of EFB in dogs when surgical repair is not financially feasible or when surgery must be meaningfully delayed due to lack of surgical expertise. The principles of treatment would be to manage concurrent esophagitis, administer antimicrobial treatment, and to therapeutically drain the pleural space if clinically indicated. The ideal method of nutritional management is unknown. Medical management should only be considered in dogs with small esophageal perforations.

Footnotes

^aAlfaxan, Jurox Pty. Ltd., NSW, Australia

^bFresofol 1%, Fresenius Kabi Australia Pty. Ltd, NSW, Australia

^cAmpicyn, Aspen Pharmacare Australia, NSW, Australia

^dMetronidazole BP, Baxter Healthcare, NSW, Australia

^eBaytril 50, Bayer Australia Ltd, NSW, Australia

^fMethadone hydrochloride 10mg/ml injection, Aspen Pharmacare, NSW, Australia

^gTemgesic, Reckitt Benckiser Australia Pty. Ltd., NSW, Australia

^hTramal, CSL Ltd., NSW, Australia

ⁱSomad, Takeda Pharmaceuticals Company, Zurich Switzerland

^jSolu-Delta-Cortef, Pfizer Inc, New York City, United States of America

^kHill's prescription AD, Hills Pet Nutrition Pty. Ltd, NSW, Australia

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Figure 1. Left lateral recumbency thoracic radiograph. Note the linear osseous opacity within the caudal thoracic esophagus representing the esophageal foreign body. This is surrounded by a soft tissue opacity, which may represent part of the foreign body, fluid within the esophagus or mediastinitis associated with esophageal perforation.

Figure 2. Dorsoventral view thoracic radiograph. There is a moderate left sided and mild right sided pleural effusion suggestive of esophageal perforation.

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