CASE REPORT

Survival of two dogs with pyothorax secondary to perforating oesophageal foreign body

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Case report Two dogs with an oesophageal foreign body (FB) were diagnosed with secondary pyothorax at the time of presentation. One dog was managed with surgical FB removal, pleural lavage and thoracotomy tube placement. Following surgery, it was admitted to the intensive care unit for oxygen therapy, antimicrobial and analgesic administration, fluid therapy, thoracic drainage and enteral nutrition. The other dog was managed with endoscopic FB removal, thoracotomy tube placement with continuous suction, and similar aftercare in the intensive care unit. Both survived to discharge from hospital.

Conclusion This report details the treatment and survival of two dogs with secondary pyothorax associated with oesophageal FB, with successful management through supportive care, oesophageal rest and treatment of infection.

Keywords dogs; endoscopy; sepsis; thoracotomy

Abbreviations CRI, constant-rate infusion; FB, foreign body; ICU, intensive care unit; RER, resting energy requirement

Management of oesophageal foreign bodies (FBs) in dogs is typically uncomplicated. Treatment options include using endoscopic assistance or fluoroscopic guidance to facilitate oral removal or advancement of the FB into the stomach, or removal through thoracotomy and oesophagotomy.1–6 All methods of removal have a similar reported frequency of complications, ranging from 10% to 29% with an overall survival rate of 79–93%.7–4,6,7 Longer duration of clinical signs has been associated with an increased risk of complications in some studies.2,4 The major complication arising from an oesophageal FB is perforation of the oesophagus, with subsequent pneumomediastinum, pyomediastinum and, in severe cases, pneumothorax and pyothorax.

Pyothorax is an infrequent consequence of oesophageal perforation8–9 and requires concurrent perforation of the mediastinum. There are a few reports of pyothorax secondary to oesophageal FB, with most describing grave outcomes. One dog presenting with a confirmed pyothorax secondary to an oesophageal FB died following oesophagectomy.8 Another study7 reported one dog with effusion that was described as pyothorax. The dog was euthanased at surgery and details of cytological or microbial culture results to confirm pyothorax were not reported. A third study reported two cases of oesophageal FBs presenting with pleural effusion, with one of the dogs dying and the other being euthanased.2 In these cases, cytology or microbial culture was not performed to determine the type of effusion. In one further case report, a dog with septic pyothorax secondary to an oesophageal FB was reported to survive, but specific details of this case were not presented in this large retrospective study of pleural and mediastinal effusions.8 From these reports, practitioners may assume a grave prognosis for pyothorax secondary to an oesophageal FB, but there is little evidence to qualify this assumption.

We describe the management of two dogs with an oesophageal FB, both of which presented with pyothorax.

Case reports

Case 1

A 3-month-old 4.24-kg entire female Bull Terrier-cross was presented with pyothorax. The dog had been recently acquired, so its previous history was unclear. The dog presented with tachypnoea (respiratory rate of 48 breaths/min) and laboured breathing with increased inspiratory and expiratory effort. Lung sounds were inaudible bilaterally in all quadrants on thoracic auscultation. The dog displayed signs of vasoconstricted shock with obtunded mentation, pale mucous membranes, a capillary refill time of 3 s and hypothermia (rectal temperature of 33.8°C). The dog did not show signs of interstitial dehydration. Non-invasive blood pressure was unable to be measured. Initial bloodwork included packed cell volume and complete biochemical panel was unable to be run because the sample volume was too small. Pertinent findings are shown in Table 1.

Initial stabilisation was achieved with flow-past oxygen supplementation, and IV boluses of compound sodium lactate (50 mL/kg Hartmann’s Solution; Baxter, NSW, Aust) and hydroxyethyl starch 130/0.4 (Voluven; Fresenius Kabi, NSW, Aust; total dose not recorded) to achieve a mean oscillometric arterial blood pressure of 70 mmHg. Antimicrobial therapy was initiated with ceftazidime (Cezfazidime; Sandoz, NSW, Aust) at 25 mg/kg IV and amoxicillin (Amoxil; GlaxoSmithKline, VIC, Aust) at 23 mg/kg IV within 1 h of admission. Needle thoracocentesis collected 180 mL of purulent fluid which, on cytological examination, was consistent with septic supplicative inflammation, characterised by degenerate neutrophils and...

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in intracellular rods and cocci. Right lateral and ventrodorsal thoracic radiographs identified a mineral opacity 28 mm long × 25 mm wide visible in the mediastinum in the region of the heart base (Figure 1). There was also increased soft-tissue and gas opacities in the pleural spaces, consistent with the presence of pleural fluid and pneumothorax. Activated partial thromboplastin time (SCA2000; Synbiotics) performed 4 h after presentation on citrated blood was prolonged at 225 s (reference 72–102 s) and a concurrent prothrombin time (SCA2000; Synbiotics) was normal at 17 s (reference 11–17 s). Given the coagulation times were performed within the fluid resuscitation period, they could have at least in part reflected dilutional coagulopathy. Regardless, 30 mL/kg of fresh frozen plasma was administered IV; 20 mL/kg prior to surgery and a further 10 mL/kg during surgery. Multiple further venous blood gas and electrolyte analyses were performed during the resuscitation period.

A right lateral thoracotomy was performed under general anaesthesia. Ongoing fluid support with compound sodium lactate (total volume 17 mL/kg) and hydroxyethyl starch 130/0.4 (total volume 4 mL/kg) IV was provided and vasopressor support was provided with continuous-rate infusions (CRI) of dopamine (DBL Sterile Dopamine Concentrate; Hospira, VIC, Aust) at 15 μg/kg/min IV and phenylephrine (Neo-synephrine; Hospira) at 1–2 μg/kg/min IV. Tachycardia (heart rate of 180–200 beats/min) and hypotension (Doppler systolic blood pressure of 40–60 mmHg) were persistent for the first hour of anaesthesia. The heart rate progressively decreased and non-invasive blood pressure progressively increased during the second hour of anaesthesia, concurrent with the commencement of the transfusion of additional 10 mL/kg fresh frozen plasma. Ceftazidime was continued at 25 mg/kg IV every 90 min during anaesthesia.

A large amount of turbid, yellowish brown fluid was removed by suction from the thoracic cavity. Following retraction of the lung lobes, a bone FB in the mid-oesophagus was noted, with an oesophageal perforation at the craniodorsal aspect of the FB. An oesophagotomy was performed caudal to the location of the perforation to allow removal of the FB. The edges of the perforation were debrided and the oesophagus was sutured in a single-layer full-thickness simple continuous pattern with 4/0 polydioxanone (PDS*II, Ethicon). Fibrinous material was collected for aerobic and anaerobic cultures and susceptibilities. The pleural space was lavaged and suctioned to remove all visible fibrinous material. A thoracostomy tube (Kendall Argyle Trochar Tube 10Fr; Tyco Healthcare [Covidien], NSW, Aust) was placed. A gastrostomy feeding tube (Surgivet PT16XL 16Fr; Smiths Medical, NSW, Aust) was placed in the left fundus after making a small left flank incision with a left fundic gastropexy. The duration of anaesthesia was approximately 2 h and the duration of surgery was approximately 1.5 h.

The dog was transferred to the intensive care unit (ICU) immediately after surgery. Vasopressor support was continued with a CRI of dopamine at 5 μg/kg/min IV for 8 h until a mean oscillometric arterial pressure of 100 mmHg was recorded and maintained. Mean arterial pressure remained stable at 80–100 mmHg following this. Postoperative oxygen therapy in an oxygen cage (fraction of inspired oxygen of 0.5) was administered to correct hypoxaemia (oxygen saturation (SpO₂) 88% on room air, 99% with oxygen) and tachypnoea (respiratory rate 60–80 on room air, 35–50 with oxygen). In addition to further analgesia, the dog’s vital signs stabilised with this intervention and oxygen therapy was ceased after 48 h when the dog maintained SpO₂ of 97% on room air.

Other ICU management included thoracic drainage, isotonic crystalloid IV fluid therapy, enteral nutrition and analgesic and antimicrobial administration. Intermittent thoracic drainage was performed via the thoracostomy tube every 4–6 h for 4 days. Initial fluid production was approximately 2 mL/kg/h. After 4 days, the volume of fluid aspirated from the tube had dropped to < 0.5 mL/kg/h and the tube was removed. Intravenous fluid therapy was provided with compound sodium lactate containing 20 mmol/L of supplemental potassium chloride to account for maintenance requirements and additional abnormal ongoing losses for 5 days. A metoclopramide (Metomide; Ceva Animal Health, NSW, Aust) CRI was provided at 1–2 mg/kg/day for days 3–5 of hospitalisation for treatment of ileus and regurgitation. A high-calorie enteral diet (Recovery; Royal
Canin, VIC, Aust) was provided at 6 feeds/day via the gastrostomy tube. The volume of food was calculated to provide half of the dog’s resting energy requirement (RER) on the first day and full RER from the second day onwards. Analgesia was provided with a fentanyl (Fentanyl Injection; AstraZeneca, NSW, Aust) CRI at 2 μg/kg/h IV for 36 h and then tramadol at 2 mg/kg every 12 h administered SC for 4 days (Tramal 100; CSL, VIC, Aust), then via feeding tube (Tramadol 10 mg compounded capsules; Bova Compoundings, NSW, Aust). The tramadol was started 24 h prior to ceasing the fentanyl and was continued until discharge. Antimicrobial therapy was provided with amoxicillin at 23 mg/kg IV every 6 h and ceftazidime at 34 mg/kg every 8 h for 5 days. Subsequent results of microbial culture reported a beta-haemolytic Streptococcus species susceptible to all tested antimicrobials, including amoxicillin, and a Fusobacterium species. Antimicrobial therapy was subsequently changed to amoxicillin–clavulanate (Clavulox 50 mg tablets; Pfizer [Zoetis], NSW, Aust) at 23 mg/kg every 12 h via feeding tube. Serial bloodwork, including complete blood count, electrolytes and venous blood gases, was performed during hospitalisation.

At 6 days after presentation, the dog was discharged from hospital with the gastrostomy tube in place. No food was to be offered orally for a further week to allow the oesophagotomy to heal, with feeding to continue via the gastrostomy tube. Amoxicillin–clavulanate per feeding tube was prescribed to continue twice daily for 28 days. Tramadol (Tramadol 10 mg compounded capsules; Bova) was prescribed to continue twice daily as needed. The dog was recovering well at last follow-up, 3 weeks after discharge. Further management was performed by the owner, who is a veterinarian.

**Case 2**
A 2-year-old 5.3-kg spayed female Shih Tzu was referred for management of an oesophageal FB that had been present for 7 days. The dog had been fed a chicken carcass 7 days previously and had shown signs of anorexia and lethargy since that time. After 7 days of clinical signs the dog was presented to the referring clinic. Thoracic radiography at the referring clinic identified a mineral opacity in the caudal mediastinum. Intravenous fluid therapy with 0.9% sodium chloride (0.9% Sodium Chloride; Baxter) and antimicrobial therapy with amoxicillin–clavulanic acid SC (dose not recorded) and enrofloxacin 6.6 mg/kg SC were provided prior to referral.

The dog presented with tachypnoea (respiratory rate of 40 breaths/min) and laboured breathing. The dog was tachycardic (heart rate of 160 beats/min). Rectal temperature was 38.4°C. Lung sounds were loud in the right hemithorax and normal in the left hemithorax on thoracic auscultation. Pulse oximetry recorded a SpO₂ of 88% on room air. An arterial blood gas sample taken while the dog was breathing room air was consistent with hypoxaemia (arterial partial pressure of oxygen 71.8 mmHg, reference interval 95.3–108.9 mmHg) with an elevated alveolar–arterial oxygen gradient of 49 mmHg (reference < 15 mmHg). Repeat right lateral, left lateral and ventrodorsal thoracic radiographs were taken to further characterise the FB and assess for signs of perforation. Imaging identified a poorly defined structure in the caudal mediastinum that measured approximately 76 mm long × ≈35 mm wide and had a mixture of soft-tissue and mineral opacities. There was also increased soft-tissue opacity in the pleural spaces, consistent with the presence of pleural fluid (Figure 2). Initial bloodwork included packed cell volume, total plasma protein, electrolytes, venous blood gases, glucose, lactate and complete blood count. A complete biochemical panel was not run because of the owner’s financial concerns. Pertinent findings are shown in Table 1.

Thoracotomy was recommended, but the owner declined surgical management. Thoracocentesis removed a small volume of purulent fluid which, on cytological examination, was consistent with septic suppurative inflammation, characterised by degenerate neutrophils and extracellular and intracellular rods and cocci. Antimicrobial therapy was immediately initiated with ampicillin (Austrapen; Lennnon Healthcare, NSW, Aust) at 22 mg/kg IV and metronidazole (Metrodazole BP; Baxter) at 10 mg/kg IV. The fluid was submitted for aerobic microbial culture and susceptibility testing. Endoscopic FB removal under general anaesthesia was then performed. One piece of bone was removed orally while another was advanced into the stomach. A small full-thickness oesophageal perforation was noted. A pneumothorax developed during anaesthesia, necessitating a single-needle thoracocentesis (volume of air removed not recorded). A thoracostomy tube (left hemithorax; 12Fr; V-TPTL-1230 V; Cook Animal Health, IN, USA) was placed. A gastrostomy feeding tube (16Fr; Cook Animal Health) was placed percutaneously using endoscopic guidance. The pleural space was not lavaged.

ICU management included 24-h monitoring, oxygen therapy, thoracic drainage, fluid therapy, antacid therapy, analgesic and antimicrobial administration, and enteral nutrition. Oxygen therapy was provided via an oxygen cage at a fraction of inspired oxygen of 0.45–0.5 for the first 12 h following anaesthetic recovery, after which the dog maintained arterial oxygen saturation at 96% breathing room air and oxygen therapy was stopped. Thoracic drainage was performed via the thoracostomy tube with constant suction for 12 h using a three-chamber collection system (Thora-Seal; Covidien, NSW, Aust) and then intermittent drainage every 4 h for a further
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24 h. Pleural fluid production progressively dropped from 2 mL/kg/h to < 0.2 mL/kg/h at the time of removal, 36 h after placement. Further fluid cytology was not performed. Intravenous fluid therapy was provided with compound sodium lactate with 20 mmol/L of supplemental potassium chloride at 5 mL/kg/h for 1 day, 2.5 mL/kg/h for the next day and 1.25 mL/kg/h from the third day until discharge. Antacid therapy was provided with pantoprazole (Pantoprazole; Sandoz) at 0.5 mg/kg IV once daily for the duration of hospitalisation. Analgesia was provided with a fentanyl transdermal patch at 2.3 μg/kg/h (Durogesic 12; Janssen-Cilag, NSW, Aust) for 24 h, with concurrent methadone (Methadone; Ilium, NSW, Aust) at 0.3 mg/kg SC as required every 4–8 h for 12 h. Additionally, tramadol was concurrently started at 2 mg/kg SC and continued twice daily for the duration of hospitalisation. Bupivacaine (Bupivacaine Injection BP 0.5%; Pfizer) was also administered at 1.47 mg/kg via the thoracostomy tube twice in the 24 h after placement. Antimicrobial therapy was provided with ampicillin at 22 mg/kg IV every 8 h and metronidazole at 10 mg/kg IV twice daily for the duration of hospitalisation. A high-calorie enteral diet (Hills Prescription Diet™ a/d™; Hill’s Pet Nutrition, NSW, Aust) was administered via the gastrostomy tube at 5 feeds/day. The volume of food was calculated to provide one-third of the dog’s RER on the first day, two-thirds on the second day and full RER from the third day onwards. The results of aerobic microbial culture of the pleural fluid showed a light, mixed growth. Identification of individual bacterial species was not reported. Electrolytes and venous blood gases were serially monitored during hospitalisation.

At 6 days after presentation, the dog was discharged from hospital with the gastrostomy tube in place and amoxicillin–clavulanate (Clavulox 250 mg tablets; Pfizer) at 11.8 mg/kg via feeding tube twice daily for 20 days. Feeding solely via the gastrostomy tube was continued for 1 week, following which oral feeding was reintroduced. Ongoing antacid therapy with omeprazole (Losec 10 mg Tablets; AstraZeneca) at 0.47 mg/kg via feeding tube once daily for 12 days was also provided. The dog returned 17 days later for re-examination. It was clinically well. Repeat thoracic radiography was within normal limits. Repeat oesophagoscopy under general anaesthesia was performed to assess for stricture formation. There was a focal mild thickening of the oesophageal mucosa at the site of the perforation, such as penetrating thoracic injuries and extension of pulmonary infection, can be good.6 Both surgical10 and non-surgical management11 have been associated with long-term survival. Non-surgical management of pyothorax secondary to oesophageal perforation was also associated with a good outcome in four out of five dogs with spirocercosis.12

Our case report is unique because the two dogs presented with pyothorax, rather than developing it as a consequence of treatment or management. Treatment followed the standard principles of canine pyothorax therapy as reported previously,10,11 including drainage of infected fluid, pleural lavage (in case 1), thoracostomy tube placement and antimicrobial therapy. Antimicrobial therapy should be broad-spectrum, and in retrospect, the dog in case 2 should ideally have been treated with greater Gram-negative coverage. Bypass of the oesophagus is standard of care for management of oesophageal perforation. A gastrostomy tube was placed in both cases to help prevent mechanical disruption of the healing oesophagus and in the case of the dog managed without surgery, to prevent further contamination of the mediastinum and pleural space.

Both dogs had severe sepsis at presentation, having met at least two criteria for the systemic inflammatory response syndrome13 with evidence of infection, and having organ dysfunction: respiratory dysfunction in both cases and coagulation dysfunction and cardiovascular dysfunction (septic shock) in dog 1. Patients with severe sepsis require a high level of care. Continuous monitoring and supportive care in a 24-h ICU were key features in both cases. Supportive care for septic dogs must include aggressive management of organ dysfunction occurring as a result of sepsis. For these dogs, the supportive care included oxygen therapy, IV fluid therapy, vasopressor medications, opioid analgesia, enteral nutrition, antacids and prokinetics. It is not possible to attribute the outcome to any individual treatment in these two dogs, but it is considered unlikely that successful outcomes would have been reached without intensive care.

Surgery is generally recommended to treat cases of oesophageal FB with radiographic or endoscopic evidence of oesophageal perforation.2,4 In this report, dog 1 was managed surgically but not dog 2. Surgery was recommended as the preferred treatment in both cases to provide source control for the infection and allow for debridement and primary repair of the oesophageal perforation. However, in case 2 the dog was successfully treated without the recommended surgical intervention. Management included endoscopic FB removal, thoracostomy tube placement and oesophageal rest. A major risk of endoscopic FB removal in cases of oesophageal perforation is the rapid development of a life-threatening pneumothorax from leakage of insufflated air, which occurred in this case. Being prepared to rapidly perform a thoracocentesis, if needed, is vital to avoid the fatal consequences of this complication. We speculate that complete bypass of the oesophagus by gastrostomy tube feeding was important in facilitating the successful outcome, as maintaining the oesophagus in a collapsed state allowed the small feeding to heal. Another factor that may have facilitated the successful outcome was the apparently low volume of pleural effusion, based on radiographic assessment. The positive outcome in this case is in accordance with previous reports that oesophageal perforation can be successfully managed without surgery in some cases.7

In conclusion, the outcome in these two cases supports that pyothorax secondary to oesophageal FB in dogs can be successfully managed with supportive care, treatment of infection and

Discussion

Previous studies describing oesophageal FBs in dogs have focused on the method of removal2,6,7 or the frequency of complications.3,4,9 Pyothorax is reported infrequently and is usually discussed as a complication of therapy, rather than existing at presentation. Pyothorax secondary to oesophageal perforation has been associated with a poor outcome in a few studies,2,6,7 as previously noted. However, the outcome of pyothorax in dogs secondary to causes other than oesophageal perforation, such as penetrating thoracic injuries and extension of pulmonary infection, can be good.6 Both surgical10 and non-surgical management11 have been associated with long-term survival. Non-surgical management of pyothorax secondary to

The first priority of veterinarians is the health and welfare of the animals they treat. The never-ending engagement in animal welfare issues can be frustrating for practitioners, creating unique ethical dilemmas for veterinarians who are constantly balancing their concern for animal welfare with the demands of clients, industries and employers without necessarily having the training or tools to help them.

Animal welfare in veterinary practice, a book aimed specifically at practitioners, fills this void and importantly highlights the crucial role that everyday practitioners have in improving and promoting animal welfare at the ‘grass roots’ practical level. Author James Yeates, Chief Veterinary Officer at the RSPCA, is well known in the animal welfare world and has widely published on topics such as companion animal welfare assessment.

This book takes a positive and pragmatic approach to the challenges of practice, where the veterinarian’s ability to improve animal welfare can depend more on the ability to influence client behaviour than clinical knowledge. A concern for animal welfare and solving everyday ethical dilemmas can be viewed as a positive aspect of practice, and this book provides practical and realistic methods to assist veterinarians in carrying out this role. Chapters 3 and 4, entitled Welfare assessment and Clinical choices, respectively, describe various methods of welfare assessment and discuss how this information can be used to provide practical solutions to help improve animal welfare.

The book is written in a concise, easy to read style with some excellent tables and chapters focusing on patients, clients, welfare assessment, clinical choices and achieving animal welfare goals. The book also extends beyond the clinic, outlining how veterinarians can be proactive ‘animal welfare ambassadors’ to their clients and others, and the importance of professional collaboration in improving animal welfare. The book presents realistic and practical methods for working with owners to improve patients’ welfare in everyday practice, as well as explaining quality of life assessments and how to resolve difficult ethical dilemmas.

As a long-term practitioner who enjoys practice because of everyday engagement with ‘ethical dilemmas’ and firmly believes that practitioners have the ability to individually and collectively improve animal welfare, I enjoyed this book with its informative and uplifting message and practical information.

All veterinarians and veterinary students would enjoy and benefit from reading this common-sense and informative book.

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