Oesophageal adenocarcinoma presenting as a foreign body induced intrathoracic abscess in a dog

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CASE REPORT
Companion or pet animals

Abstract
A 9-year-old, male, entire Irish setter was referred for further investigation of a chronic cough, anorexia, weight loss and lethargy. Computed tomography revealed a large ovoid encapsulated lesion, with a gas and fluid attenuating centre, and an intrallesional mineral opacity. The lesion ran close to the caudal oesophagus, and extended cranially within the pulmonary parenchyma. Endoscopy revealed a communication between the lumen of the distal oesophagus and the lesion, which contained a purulent discharge and plant material. Based on the computed tomography and endoscopic findings, the diagnosis was expected to be a thoracic abscess, secondary to foreign body migration or penetration; however, surgical resection and histopathological analysis of the lesion revealed an oesophageal adenocarcinoma. This case details the unusual presentation of an oesophageal adenocarcinoma, with endoscopic and computed tomography findings that mimicked a thoracic abscess with an intrallesional foreign body.

BACKGROUND
Oesophageal adenocarcinomas are extremely rare in dogs, with only a single case report of a naturally occurring canine oesophageal adenocarcinoma. In comparison, in human medicine, oesophageal adenocarcinomas are the most common form of oesophageal tumour within Western populations. We present this case, as it details unusual computed tomography (CT) and endoscopic findings that are not consistent with the typical observations expected for an oesophageal tumour. In addition, to our knowledge, no tumour with an incidental intrallesional foreign body has been reported within the veterinary literature.

CASE PRESENTATION
A 9-year-old, male, entire Irish setter was referred to the Queen’s Veterinary School Hospital, University of Cambridge, for further investigation of a chronic cough, anorexia, and weight loss. The cough had a 9-month duration and had repeatedly improved following antibiotic therapy, but consistently deteriorated within 5–7 days of stopping treatment. Initially, the cough had been dry, but had progressed over time to become moist and productive. Diagnostics performed prior to referral documented a marked left-shifted neutrophilia. On radiography, a focal alveolar pattern within the caudalventral region of the left cranial lung lobe and an interstitial pattern within the caudodorsal region of the left caudal lung lobe were documented (Figure 1). Bronchoscopy was performed on two separate occasions, with bronchoalveolar lavage yielding a septic mucopurulent exudate and a profuse growth of Streptococcus canis. A course of amoxicillin–clavulanic acid had been initiated for this infection based on antimicrobial sensitivity testing; however, the clinical signs recurred after finishing the course.

Other than a prior history of gastric dilation following which a prophylactic gastropexy was performed, the patient had no other relevant medical history. The dog had pink and moist mucous membranes on presentation, with a normal capillary refill time. The dog was tachycardic (heart rate: 160 beats per minute) with poor pulse quality. Its respiratory rate was 24 breaths per minute, with an increase in respiratory effort. It was normothermic. Body condition score was 3/9.

INVESTIGATIONS
The patient was stabilised with intravenous (IV) fluid therapy following admission. To further characterise the pathology identified on thoracic radiography and ascertain whether...
there was abdominal involvement from a possible tracking thoracic foreign body, a helical-16-slice CT (Toshiba Aquilion) of the thorax and abdomen was performed under general anaesthesia, with the dog in sternal recumbency. Scan settings included a pitch of 0.8, tube potential of 125 kVp, reference tube current of 100 mA, slice thickness of 1 mm and matrix 512 × 512. A 2 mL/kg bolus of 300 mg iodine permillilitre of non-ionic iodinated contrast medium (Omnipaque, GE Healthcare), followed by 5 mL of saline solution was injected manually through a cephalic IV catheter. Postcontrast images were acquired 40 seconds after contrast medium administration.

The CT showed a large, ovoid, encapsulated soft tissue attenuating lesion within the dorsal aspect of the left caudal lung lobe, close to the termination of the left caudal lobar bronchus (Figure 2). The lesion contained a mixed gas and fluid attenuating centre and an intralesional mineral opacity, and ran close to the caudal oesophagus (Figure 3). It was postulated that the two structures may communicate as the contents of the lesion were similar in appearance to that of the ingesta within the stomach. The key differential for the encapsulated lesion was a pulmonary abscess secondary to foreign body inhalation, given the CT appearance and location within the lung lobe.

In addition, the left caudal lung lobe contained several variably sized, rounded areas of whispy soft tissue attenuation, suspected to be smaller abscesses (Figure 4).

The pylorus of the stomach was in close contact with the right ventral abdominal wall, which was consistent with the patient’s history of a gastropexy. Microhepatica and a left gastro-azygous extrahepatic portosystemic shunt were concurrently noted incidental findings.

Following CT, oesophagoscopy was performed to establish whether there was a communication between the oesophagus and the lesion, as this would impact the surgical plan. This documented a ring-like defect in the oesophageal wall immediately proximal to the distal oesophageal sphincter, leading to an encapsulated structure (corresponding to the lesion identified on CT) filled with purulent discharge and plant material.

**Differential Diagnosis**

Differential diagnoses for the intrathoracic mass lesion included:

- Abscessation: an oesophageal or bronchial foreign body migration/penetration, an oesophageal diverticulum with secondary infection/impaction, or less likely hematogenous aetiology.
- Neoplasia: a primary oesophageal neoplasia with extension into the pulmonary parenchyma or a pulmonary neoplasia with oesophageal invasion.
- Granulomatous lesion: as a result of *Spirocerca lupi*, mycobacterial or fungal disease.

Considering together the abnormalities identified on CT, oesophagoscopy and bronchoscopy/bronchoalveolar lavage, either a pulmonary abscess with a draining tract connecting to the oesophageal lumen, or an acquired broncho-oesophageal fistula was suspected. A congenital broncho-oesophageal fistula with secondary abscessation was considered unlikely given the patient’s age and clinical history. Based on the capsular structure and intraluminal foreign plant material, an abscess secondary to foreign body migration was favoured over a neoplastic aetiology. A granulomatous lesion was considered the least likely, as *S. lupi* is not endemic in the United Kingdom, fungal associated granulomas are very rare as well, and it would be an unusual presentation for a mycobacterium-associated granuloma. The patient had no history of foreign travel.

**Learning Points/Take-Home Messages**

- Oesophageal tumours can behave in locally aggressive ways and may not always present as an oesophageal wall thickening or intraluminal mass.
- While rare, the possibility of neoplasia should not be discounted when a lesion has a foreign body found within it.
- Thorough list of differential diagnoses should be made based on all available information to avoid overlooking rare underlying aetiologies.
- Histopathology should always be performed, regardless of expectation.

**Treatment**

A ninth intercostal thoracotomy was performed to gain surgical access to the lesion. The lesion was identified to be in communication with the oesophagus just cranial to the diaphragm, and appeared as an abnormal pouching of soft tissue measuring 45 × 35 × 25 mm. The cranidorsal aspect of it was adhered to the caudal tip of the left caudal lung lobe. The left caudal lung lobe was noted to be discolored and atelectatic. A full thickness excision of the area of oesophagus invaded by the lesion was made, and the oesophageal defect was closed in two layers using 2 M polydioxanone in a simple continuous pattern. The left caudal pulmonary ligament was sectioned to allow mobilisation of the lung lobe. A stab incision was made into the left crus of the diaphragm, and omentum was pulled through into the thoracic cavity and tacked to the oesophageal repair. The diaphragmatic defect was then reduced in size to prevent postoperative herniation.

A sixth intercostal thoracotomy was performed via the same skin incision. A left caudal lung lobectomy was performed at the hilum using a TX30 stapler.

The thorax was thoroughly lavaged with sterile saline, and the hilar stump was inspected for air leakage or haemorrhage; none was noted. A single-lumen narrow-bore chest drain (MILA, 14Fr) was placed via the eighth intercostal space, and both thoracotomies were closed routinely.

The left caudal lung lobe and lesion communicating with the oesophagus were submitted for histopathology.
FIGURE 1 A right lateral thoracic radiograph. There is an interstitial pattern within the caudodorsal region of the left caudal lung lobe (white arrows), which correlates with the location of the mass observed on computed tomography. In addition, there is an alveolar lung pattern within the caudoventral lung fields, most prominently observed over the cardiac silhouette. This pattern is likely associated with a secondary pneumonia. There is incidental osteoarthritis visualised at the glenohumeral joint.

FIGURE 2 Sagittal (Figure 4) lung window reconstruction algorithm of the thoracic computed tomography. The ovoid lesion (orange arrows) can be seen to be close to the end of the left caudal lobar bronchus (green arrows) within the caudodorsal aspect of the left caudal lung lobe.

OUTCOME AND FOLLOW-UP

On visual inspection, the resected tissue (45 × 35 × 25 mm) was a tubular structure filled with purulent material and multiple pieces of conifer leaf. Histopathology revealed invasive nests and trabeculae of neoplastic epithelial cells arising from the oesophageal mucosa. Areas of the tumour demonstrated glandular differentiation with mucin production (defined by a Periodic Acid Shift histochemical stain) and rare metaplastic parietal cells, demonstrating gastro-oesophageal origin. The final diagnosis was an oesophageal adenocarcinoma. Histological analysis of the left caudal lung lobe (100 × 130 × 30 mm) showed neoplastic invasion by the oesophageal adenocarcinoma and concurrent diffuse abscessation within the lobe.

Postoperatively, the patient was oxygen dependent and remained in the intensive care unit for supportive treatment. IV fluid therapy (Hartmann’s lactated ringers) was continued in addition to methadone (Methadyne; Jurox; 0.2 mg/kg IV), paracetamol (Braun; 15 mg/kg IV), cefuroxime (Zinacef, GlaxoSmithKline; 20 mg/kg IV) and bupivacaine (Marcain polyamp, Aspen; 2 mg/kg) via the thoracic MILA drain.

Twenty-four hours postoperatively, the patient deteriorated. Blood gas analysis showed a consistently low PaO2 (53 and 57 mmHg) despite oxygen supplementation. The patient was re-anaesthetised to allow for positive pressure ventilation and provision of a higher inspired oxygen fraction. Despite ventilation, cardiopulmonary arrest ensued, and attempts at resuscitation were not successful.

DISCUSSION

We report this case as an unusual presentation of oesophageal adenocarcinoma in a dog that mimicked thoracic abscess with an intraliteral foreign body. The appearance of the lesion on CT, an encapsulated lesion with a gas and fluid attenuating centre, largely satisfied the CT appearance of a pulmonary abscess. In addition, the chronic history, bronchoalveolar lavage cytology and culture results, favourable response to antibiotic therapy and endoscopic finding of plant material
with all biased our top differential diagnosis towards that of a foreign body-induced abscess. The CT findings were not consistent with published veterinary CT reports of canine oesophageal tumours. These reports include a soft tissue attenuating mass within the oesophageal wall or lumen, stricture formation and oesophageal distension cranial to the mass/neoplastic stricture.4–6 Oesophageal tumours are rare in dogs and make up less than 0.8% of all reported tumours.7–9 Osteosarcomas and fibrosarcomas are the most commonly reported canine oesophageal neoplasms. These tumours are frequently associated with the nematode S. lupi.10–14 These parasites migrate to, and then mature and remain within, the lower oesophagus in their canine host. Up to 22% of S. lupi-infected dogs have been found to develop malignant oesophageal neoplasms.15 It is unclear as to the exact pathophysiological process by which S. lupi induces neoplasia, but it is thought to be a result of sustained inflammation in the oesophageal tissue surrounding the nematode. In addition to neoplastic lesions, S. lupi can create non-neoplastic oesophageal soft tissue proliferations.16 The patient in this case report lived in a non-endemic S. lupi region and had no history of foreign travel. The most common oesophageal tumours other than those associated with S. lupi include squamous cell carcinomas and leiomyosarcomas.17

The locally invasive nature of the tumour presented in this case report is more similar to reports of human oesophageal tumours. This is likely attributed to the fact that oesophageal adenocarcinomas account for 42% of all human oesophageal neoplasms.18 Between 40% and 60% of human patients have unresectable oesophageal cancer on presentation due to invasion of surrounding structures.19–21 The oesophagus has no serosal layer, and oesophageal tumours can consequently grow extensively without producing symptoms. The published incidence of malignant oesophageal fistulas in humans is 13%, and on postmortem examination of human patients, up to 42% of oesophageal neoplasms show direct invasion into surrounding pulmonary tissues.22,23

There is only one report of a non-experimentally induced oesophageal adenocarcinoma within the veterinary literature, and thus there is scarce information on their pathophysiology and natural progression in dogs.1 In people, oesophageal adenocarcinomas have been found to arise from a premalignant change to the oesophageal mucosa, which is termed Barrett’s oesophagus.24–28 Specifically, the term Barrett’s oesophagus describes the transformation of normal squamous mucosa within the distal oesophagus into metaplastic columnar mucosal cells.28–30 Barrett’s oesophagus is associated with chronic gastroesophageal reflux, and occurs in up to 10%–15% of people with gastroesophageal reflux disease.31,32 While Barrett’s oesophagus has been found to be an important risk factor for the subsequent development of oesophageal adenocarcinoma, the annual incidence of oesophageal adenocarcinoma in Barrett’s oesophagus patients is low, and is thought to be around 0.33%–0.63%.28,33–37 Interestingly, dogs have been used as an experimental model of Barrett’s oesophagus via pharmacological or surgical interventions, creating acidic or alkaline reflux models. Out of 50 dogs in this study, 24 dogs developed Barrett’s oesophagus, nine of them progressed to show mild dysplasia, and in two dogs this progressed to oesophageal adenocarcinoma 60 months after the surgical alteration.38 It is thus presumed on the basis of this study and the data from human literature that spontaneous
occurrence of oesophageal adenocarcinoma in dogs may, in part, be due to gastroesophageal reflux in these patients.

The patient presented in this case had no reported history of vomiting or regurgitation. This is unusual given the eventual diagnosis of an oesophageal tumour. Given the tumour type, it is a possibility that the patient had been suffering from silent reflux, particularly considering the patient history of gastric dilation and gastropexy. Gastrointestinal disturbances following gastropexy surgery have been reported within the veterinary literature. Some of the short-term gastrointestinal disturbances may be attributed to surgical induced ileus; however, one report describes chronic vomiting due to pyloric outflow obstruction by a malpositioned prophylactic gastropexy. In this case, the vomiting ceased after surgical revision of the gastropexy. Within the human literature, delayed gastric emptying time has a role in the pathophysiology of gastro-oesophageal reflux in a proportion of human patients.44–46

In the case presented here, any effect of the gastropexy on gastromotility and the presence or absence of reflux was not defined during life; however, it remains a potential risk factor based on the published evidence reported above.

Another possible risk factor for the development of an oesophageal adenocarcinoma in this patient was the previously undetected gastro-azygous portosystemic shunt (PSS). Patients with PSS have an increased risk of gastrointestinal signs such as vomiting, diarrhoea and gastrointestinal bleeding.47,48 In addition, in one study, up to 21% of patients with intrahepatic PSSs were found to have gastrointestinal ulceration.49 It has been proposed that this may be due to elevated serum bile acid concentrations leading to hypergastrinemia and increased gastric acid production, abnormal blood flow, poor mucosal integrity and abnormal mucus production.50 Furthermore, one paper reports subjective prolongation of gastric emptying time in canine patients with PSSs.51 If delayed gastric emptying occurred in our patient, this may have increased his risk of gastro-oesophageal reflux disease. These sequelae attributed to PSSs, and the absence of treatment for this condition, may have predisposed this patient to both gastro-oesophageal reflux, and the subsequent development of the oesophageal adenocarcinoma. However, it is worth noting that many dogs suffer from naturally occurring gastro-oesophageal reflux disease and untreated PSSs, and yet as mentioned previously, there is only one report of a non-experimentally induced oesophageal adenocarcinoma within the canine veterinary literature. This suggests that the aetiology of oesophageal adenocarcinomas within dogs is likely complicated and currently poorly understood.

To the authors’ knowledge, this is the second report of a naturally occurring oesophageal adenocarcinoma in a dog and the first report of an incidental foreign body within a tumour in the veterinary literature. This shows that oesophageal adenocarcinoma should be on the list of differentials when a tumour associated with the oesophagus is identified. This paper also highlights that the presence of a foreign body within a lesion should not exclude the differential diagnosis of neoplasia.

CONFLICT OF INTEREST STATEMENT
The authors declare they have no conflicts of interest.

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ETHICS STATEMENT
No ethical approval was necessary for this retrospective case report. Informed owner consent was given for all investigations and treatments.

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AUTHOR CONTRIBUTIONS
Emily Brady wrote the initial draft of the article. Michael Heritage, Hannah Wong, Laura Owen and Andre Kortum revised the article.