A CONGENITAL OESOPHAGOTRACHEAL FISTULA IN A TWO-YEAR-OLD DOG

LEA STOGDALE*, D.G. STEYN* AND B.C. THOMPSON†


INTRODUCTION

Congenital oesophagotracheal fistulas are very rare in both humans and animals. Van der Gaag, Kuiper and Kroneman reviewed the literature and could locate descriptions of congenital oesophagotracheal fistulas in a calf, 3 dogs and 1 cat. In addition they described a case in a calf. These authors were able to find 3 reports of oesophagobronchial fistulas, 1 in a cat and 2 in dogs. Van der Gaag et al. discussed the embryological development of the oesophagus and the pulmonary airways, relating this to the variations of congenital malformations affecting the oesophagus. This case report describes the clinical symptoms, radiographic appearance, surgical correction and post mortem findings of a congenital oesophagotracheal fistula in a two-year-old Yorkshire Terrier.

CAS HISTORY

A two-year-old male Yorkshire Terrier, weighing 2 kg, was referred to the Department of Medicine with a history of coughing, dysphagia and vomiting. Before the onset of these symptoms the dog had been lively, in good condition and had suffered no previous illnesses. Two weeks prior to admission, it had suddenly commenced coughing persistently, eating and drinking with difficulty and frequently vomited food or water during and after ingestion. The animal was treated by the referring veterinarian with antibiotics and supportive therapy. As no response was obtained it was referred to the Faculty of Veterinary Science.

At the initial clinical examination (Day 1) the dog was found to be in poor condition; he was very depressed, refused to eat and was unwilling to walk. The mucous membranes were slightly pale and the animal was considered to be 5% dehydrated as judged by skin elasticity. The respiratory rate was 40/min with a moist productive cough occurring approximately every 5 min. Respiration was forced and was mainly diaphragmatic with little movement of the ribs. Each inspiration was preceded by grunting noises and the alveolar and bronchial sounds were markedly increased. Moist rales and clear gurgling noises were audible during the initial part of each expiration. The cervical oesophagus was palpably distended with gas and the abdomen was enlarged due to the presence of excess gas in the stomach. The dog eructated periodically with a gurgling noise. No abnormal mass in the abdomen could be palpated. Haematological examinations showed a macrocytic anaemia and a neutrophilic leukocytosis.

RADIOGRAPHIC FINDINGS

Plain radiographs were taken of the thorax and abdomen. On both the lateral and dorso-ventral radiographs the stomach and small intestines were seen to be markedly distended with gas. Foci of increased density were distributed throughout the lung field.

Fig. 1 Lateral radiograph of the neck and thorax 10 minutes after a barium swallow. The inflated oesophagus is outlined by the barium, which has adhered to the wall from the level of the 3rd to 6th thoracic vertebrae. Barium has passed into the lungs delineating the trachea, bronchi and bronchioles.

A barium swallow was given to the dog and its passage along the gastro-intestinal tract was observed with a fluoroscope and recorded on radiographs taken 10 min, 30 min, 3 h and 24 h after the barium was administered. The barium passed rapidly along the cervical oesophagus and then remained in the thoracic oesophagus for 1 to 2 min before peristaltic movements propelled it into the stomach. During the time that the barium remained in the thoracic oesophagus some of...
the contrast medium was seen to pass into the lungs. The trachea, bronchi and bronchioles were clearly outlined by the barium (Fig. 1). Barium adhered to the oesophagus from the level of the 3rd to 6th thoracic vertebrae indicating an oesophagitis. The oesophagotracheal fistula was not obvious on any of the radiographs but is suggested by a small barium shadow running ventrally from the oesophagus to the trachea at the level of the 5th rib, just caudally to the cranial bronchus branch (Fig. 2).

Three hours after the barium was given, radiographs (Fig. 3) showed that the barium was passing through the gas-filled intestines. Radiographs taken 24 h after the barium was given showed that some of the contrast medium had reached the rectum but that some had remained in the area of the duodenum, suggesting a partial obstruction or a porous foreign body.

Based on clinical, laboratory and radiographic findings, the provisional diagnosis made was an upper intestinal tract obstruction (such as a sponge) and, in addition, a defect in the swallowing mechanism or in oesophageal function.

Supportive therapy was commenced as soon as the dog arrived in the hospital and was continued daily. Each day, 100 ml of dextrose-saline solution* and 50 ml of balanced electrolyte solution† were administered intravenously. Eighty mg theophylline ethylenediamine‡ and 20 mg ampicillin§ were given intramuscularly 4 times each day. On the first day 0.2 ml of glucocorticoid mixture¶ and 1 ml of vitamin B complex** were injected intramuscularly. Since a haematological examination on Day 3 showed a decrease in the haematocrit reading, 20 ml of packed red blood cells were administered intravenously.

Surgical intervention was considered essential to diagnose the aetiology of the pneumonia and the gastro-intestinal dilatation, and to remove the cause. Despite reasonably intensive care and supportive therapy the little dog was a very poor surgical risk. On Day 3, general anaesthesia was induced with 0.5 ml of a 5% thiopentone sodium solution†† intravenously. The dog was intubated and anaesthesia was maintained with 1% halothane‡‡ in oxygen using a closed circuit anaesthetic machine.§§. A continuous intravenous infusion of Ringer-lactate solution¶¶ was given throughout the surgery. A laparotomy was performed via a mid-ventral incision. The abdominal cavity was explored and all the abdominal contents were thoroughly checked for abnormalities. None were found, thus ruling out intestinal obstruction. However, when the rebreathing bag was compressed the stomach dilated. This led the surgeon to suspect an oesophagotracheal fistula. The abdomen was closed using a standard technique.

In order to confirm the suspected diagnosis of oesophagotracheal fistula, an oesophagoscopy and a tracheoscopy were carried out immediately using a human pediatric gastroscope. Oesophageal examination revealed a slit-like aperture about 3 mm long in the ventral oesophageal wall, dorsally to the heart. Bubbles of air were seen coming from this opening when the rebreathing bag was compressed, causing forced inspiration. Tracheoscopic examination also revealed a small opening, of about 3 mm diameter, in the dorsal wall of the trachea, situated above the heart and just caudal to the aperture of the cranial bronchus.

Thus, the presence of an oesophagotracheal fistula was diagnosed and a thoracotomy was performed to correct the defect. Manual positive pressure ventilation was commenced as the right side of the thorax was opened by a dorso-ventral incision in the 6th intercostal space. The oesophagotracheal fistula was isolated directly above the heart at the level of the 5th rib. It was about 4 mm long and extended ventrally from the oesophagus to the trachea. The fistula was ligated at both ends with 2 sutures of No 1 silk. The thoracic space was opened by a dorso-ventral incision in the 6th intercostal space. The oesophagotracheal fistula was isolated directly above the heart at the level of the 5th rib. It was about 4 mm long and extended ventrally from the oesophagus to the trachea. The fistula was ligated at both ends with 2 sutures of No 1 silk. The thoracic

**"Sodium Chloride and Dextrose Injection B.P. 0.45% (m/v); 2.5% (m/v)" Baxter
†"Plasmalyte B", Baxter
‡"Aminophylline", Searle
§"Penbritin", Beecham Research Lab.
¶"Opticortenot-S", Ciba Geigy
***"Vitamin B Complex injection", Centaur Labs (Pty.) Ltd.
****"Penothal", Abbotts Labs.
††"Fluothane", I.C.I.
†‡"Fluotec, type FRM", Cyprane Ltd.
*** Ringer-Lactate solution B.P.
*†† Ringer-Lactate solution B.P.
musculature was then closed routinely. At this stage spontaneous respiration had not started and the heart beat was fast and weak. The cardiac stimulant, heptamacrine hydrochloridet and the respiratory stimulant doxapram hydrochloride† were injected intravenously. The dog failed to recover from the anaesthetic and died 15 minutes after the completion of the surgery.

AUTOPSY FINDINGS

At post mortem, the oesophagotracheal fistula was carefully examined. It was 4 mm long, running ventrally from the oesophagus to the trachea. When the 2 silk sutures were removed, the fistula was seen to have parallel sides and a patent lumen. There was no fibrous tissue around it. The diameter of the fistula was 2 mm. There was a 3 mm slit-like opening in the oesophagus and an oval opening of similar size in the trachea. It was concluded that this was a congenital fistula. There was a subacute, diffuse foreign-body pneumonia with focal areas of emphysema. The oesophagus was inflamed throughout much of its length. The gastro-intestinal tract was gas-filled and devoid of contents apart from a small amount of barium in the caecum. The pathological aetiological diagnosis was foreign body pneumonia as a result of a congenital oesophagotracheal fistula.

†"Cortensor", Wander
‡"Doxapram", Robins Co.

DISCUSSION

Oesophagotracheal fistulas may be congenital or acquired as a result of trauma, infection or neoplasia. Congenital fistulas are usually detected during the first few days of life as choking and cyanosis recur with each attempt at nursing, and there may be an excessive amount of gas in the gastro-intestinal tract. In the case described here, clinical symptoms relating to the oesophageal fistula only started when the dog was 2 years of age. The fistula had a slit-like opening into the oesophagus and this must have remained closed (perhaps filled with mucoid material) during the asymptomatic 2 years.

ACKNOWLEDGEMENTS

The authors wish to thank Dr. A. Fourie for referring the case, Miss M. A. Groenland for translating one of the references, Dr. L. Bomzon for revising the original paper and Mrs. J. Oberholzer for typing the manuscript.

REFERENCES


INLIGTING

APLASTIC ANAEMIA FROM VETERINARY PHENYLBUTAZONE

Recently a 20-year-old professional jockey died of aplastic anaemia after taking phenylbutazone prepared for veterinary use. The drug, an anti-inflammatory agent, is widely used to treat both animals and humans; however, the dose is much greater for horses than for humans (for horses 2-4 g a day per 1 000 pounds of body weight, for humans 300-600 mg a day). The patient was hospitalized on December 16, 1975, with a 2-week history of spontaneous bruising and listlessness. He admitted having taken phenylbutazone in the form of crumbled 1-gram horse tablets about 20 times in the previous 2 years. One month before he was admitted to the hospital, he took 2 g a day of the veterinary product for more than 3 days for mild but painful injuries sustained in a fall. He was also taking furosemide, 40 mg per day, and cathartics occasionally.

Studies of multiple bone-marrow biopsy specimens demonstrated fatty marrow with almost complete aplasia. Bone marrow was transplanted, but the patient died 40 days post-transplant of disseminated candidiasis without evidence that the graft had taken.

It is a well-known fact among race track personnel that for minor aches and pains jockeys, grooms, and trainers often take phenylbutazone that is manufactured for horses. The patient confirmed the widespread use of "bute" at the race track and only after specific questioning admitted using it himself. He was not aware of any adverse effects of the drug.

Only chloramphenicol causes more cases of drug-induced aplastic anaemia than phenylbutazone. Although the mechanism of phenylbutazone-induced haematologic toxicity is not known, there is evidence that it is dose related. Phenylbutazone and aminopyrine have been reported to cause a granulocytosis when taken in certain Chinese herbal preparations.

The use of phenylbutazone by persons working around stables and race tracks is not surprising when one considers its availability and effectiveness. Physicians and veterinarians should be aware of this practice and counsel against it since veterinary phenylbutazone (greater strength than that manufactured for human use) can be fatal to man.

Source: Journal American Medical Association 236(9): 1049, 1976