CASE REPORT

Collapse following gastrointestinal bleeding secondary to a congenital duodenal diverticulum in two littermate boxer pups

Two littermates, a young male and female boxer, were admitted to the Utrecht University's Department of Clinical Sciences of Companion Animals within a three month period. Both dogs suffered from anaemia caused by chronic intestinal blood loss, vomiting and weight loss. In both cases, there was no response to conservative medical management. Eventually, the dogs suffered significant gastrointestinal haemorrhage that resulted in collapse. Gastroduodenoscopy and exploratory surgery showed a duodenal diverticulum in both dogs. This is the first report that describes this congenital anomaly in two siblings.

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INTRODUCTION

Intestinal diverticula are bulging pouch-like herniations in the wall of the intestine that can occasionally produce signs of gastrointestinal disease. Diverticula can be classified as congenital or acquired, true (full-thickness) or false (mucosal), primary or secondary, and intraluminal or extraluminal (Scudamore and others 1982, Ryan and others 1984). Intestinal diverticula are unusual lesions in the dog.

Marshall and Hayes (1966) were the first to describe a Meckel's diverticulum in a dog. A Meckel's diverticulum is a remnant of the vitelline duct that connects the embryonic midgut to the yolk sac and normally atrophies when placental nutrition replaces the yolk sac. It is located in the terminal ileum and is the most common intestinal anomaly in humans (Margolies 2001).

Meckel originally described two types of intestinal diverticula: a congenital type that arises from the antimesenteric surface of the ileum and affects all layers of the normal intestine and an acquired type that arises on the mesenteric surface and consists only of mucosa (Dowse 1961). Since Meckel's original report, other types of intestinal diverticula, including those located at other places within the small intestine, have been described.

Congenital intestinal diverticula are uncommon in dogs and have been associated with a failure in embryonic development (Ablin and others 1991). This present report describes the clinical findings in two canine siblings with a duodenal diverticulum.

CASE HISTORIES

Case 1

An 11-month-old male boxer was referred because of collapse, vomiting, weight loss and anaemia. Over the preceding three months, the dog had had four episodes of collapse after exercise. Over the past eight weeks, the dog had lost weight and had vomited his food on a daily basis shortly after eating. Hypoallergenic diet resulted in no improvement. The dog had a good appetite and no abnormalities were noticed in drinking, urinating and defecating patterns.

At the time of presentation, the dog had pale mucous membranes, melena and was lethargic. Physical examination showed no abnormalities except for pale mucous membranes and a firm pulse rate of 164/minute. A biochemistry profile showed hypoproteinaemia, hypoalbuminaemia and hypoglobulinaemia. Haematology showed a non-regenerative anaemia and leucocytosis (Tables 1 and 2). Bone marrow aspirates showed mild erythroid hyperplasia and blood smear showed hypochromia, anisocytosis and microcytes which is characteristic for anaemia caused by iron deficiency. A blood transfusion was given, which increased the packed cell volume (PCV) to 17 per cent.

Survey radiographs of the abdomen appeared unremarkable. Abdominal ultrasonography showed a mildly distended stomach. Because of melena, therapy with 2 mg/kg ranitidine (Zantac;

Table 1. Biochemistry results of case 1 and case 2

	Case 1	Case 2	Reference range
Urea (mmol/l)	9·7	4·0	3·0-12·5
Creatinine (μmol/l)	54	73	50-129
Glucose (mmol/l)	7·1	6·3	4·2-5·8
Total protein (g/l)	28	47	55-72
Albumin (g/l)	18	22	26-37
Sodium (mmol/l)	140	146	141-150
Potassium (mmol/l)	3·4	3·8	3·6-5·6
Calcium (mmol/l)	2·10	2·49	1·98-2·97
Alkaline phosphatase (U/l)	20	29	<73

Table 2. Haematology results of case 1 and case 2

	Case 1	Case 2	Reference range
PCV (per cent)	7	26	42-61
Reticulocytes (per cent)	3·6	7·7	<1·5
Reticulocyte index	<1	>1	
Platelets ($\times 10^9$/l)	329	637	144-603
Leucocytes ($\times 10^9$/l)	35·2	17·1	4·5-14·6
Neutrophils ($\times 10^9$/l)	33·3	13·3	2·9-11·0
Lymphocytes ($\times 10^9$/l)	0·0	2·6	0·8-4·7
Monocytes ($\times 10^9$/l)	1·8	0·7	0·0-0·9
Eosinophils ($\times 10^9$/l)	0·4	0·5	0·0-1·6
MCV (fl)		63·3	63·5-72·9
MCH (fmol)		1·11	1·37-1·57
MCHC (mmol/l)		17·6	20·5-22·4

PCV Packed cell volume, MCV Mean corpuscular volume, MCH Mean cell haemoglobin, MCHC Mean cell haemoglobin concentration

GlaxoSmithKline) subcutaneously, twice daily, and 1 g sucralfate (Ulcogant; Merck BV) orally, four times a day, was started. Two days later, the dog collapsed with white mucous membranes. The PCV had dramatically decreased to 9 per cent. A second blood transfusion was administered. A grass-filled pocket with haemorrhagic fluid was observed by duodenoscopic examination. Exploratory coeliotomy was performed and a diverticulum located at caudal duodenal flexure was discovered and removed by partial enterectomy. A third blood transfusion was given intraoperatively. The dog recovered uneventfully and ranitidine, sucralfate, 12·5 mg/kg amoxicillin and clavulanic acid (Synulox; Pfizer) orally, twice daily, and 3 mg/kg ferrofumarate (Ferrofumaraat, Roche Nederland BV) orally, three times a day, were given postoperatively. The dog went home five days after surgery with a PCV of 24 per cent and evidence of appropriate regeneration.

Six weeks postoperatively, the dog was totally recovered and had gained 6 kg. Neither vomiting nor melena was observed postoperatively. The PCV had increased to 39 per cent. The dog is still free of clinical signs 28 months later.

Case 2

Three months after the first patient, a 14-month-old female boxer was presented with problems of collapse, weight loss and a systolic murmur (Fig 1). During the past two months, the dog had collapsed six times after physical activity. The dog was bright, alert and had a good appetite. No abnormalities were noticed in drinking, urinating and defecating patterns. Despite a good appetite, the dog had lost 4 kg bodyweight over the past two months. Physical examination showed no abnormalities except for pale pink mucous membranes and a systolic murmur (two of six) that was audible on all valves.

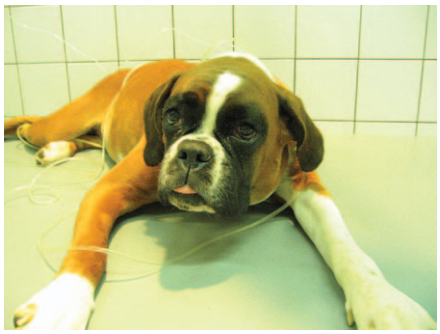

FIG 1. Female boxer that developed acute anaemia because of a duodenal diverticulum

A respiratory sinus arrhythmia was observed on ECG, and mild insufficiency of the mitral valve with minimal dilatation of the left atrium was found on ultrasonographic examination.

A biochemistry profile showed hypoproteinaemia and hypoalbuminaemia, and haematology showed a regenerative moderate anaemia and leucocytosis (Tables 1 and 2). A blood smear showed hypochromasia, polychromasia and anisocytosis of the red blood cells. An additional haematological profile was consistent with iron deficiency (Table 2). No parasites were detected on faecal examination. Because the dog was doing better and the anaemia was regenerative, the owner decided to postpone further diagnostics and treatment.

But one month after the first visit, the dog had a severe collapse and was brought to the emergency service with melena and pale mucous membranes. At that time, it became clear that this dog was a littermate of the first boxer. PCV at presentation was 10 per cent. A blood transfusion was administered and an abdominal ultrasound was performed. The stomach, and descending and ascending duodenum were distended with liquid contents. A foreign body seemed present in the distal duodenum. Endoscopic examination showed a duodenal diverticulum containing foreign material and haemorrhagic fluid. The diagnosis was confirmed by coeliotomy and the diverticulum, located at the identical location as the previous dog, was resected (Fig 2). The diverticulum contained vegetable material, grass and glass splinters (Fig 3). Postoperative management was identical to that of the first dog.

Three months after surgery, the PCV, total protein and albumin levels had normalised. The dog recovered completely with normal stools and without episodes of collapse.

Pathology

In both cases, the diverticula contained an ulcerated mucosa and a hypertrophied muscular layer. All layers (serosa, muscular

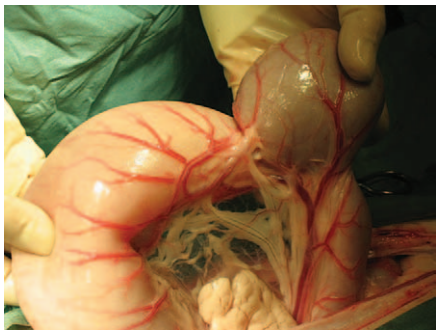

FIG 2. Intraoperative view of the duodenum with a diverticulum

layer, submucosa and mucosa) were present in the wall of both diverticula at histological examination (Fig 4). The submucosa showed some infiltration by plasma cells and neutrophils. The mucosal lining showed erosion with exudative inflammation and hyperplasia of the crypts (Fig 5).

DISCUSSION

Canine duodenal diverticula are rarely reported. Ablin and others (1991) identified only one dog with a duodenal diverticulum in a group of 13 dogs with

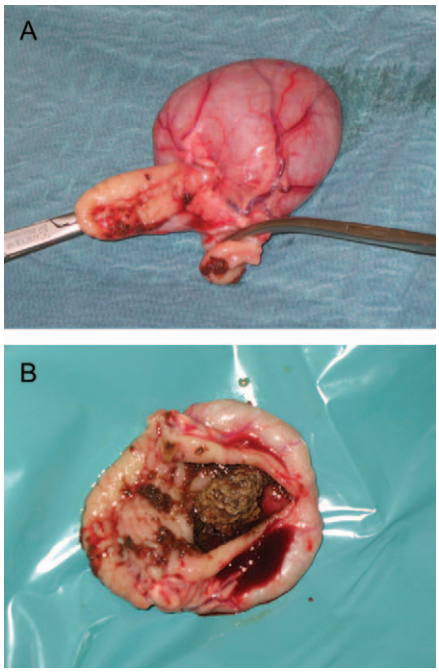

FIG 3. Diverticulum after resection and after incision showing vegetable material and haemorrhage

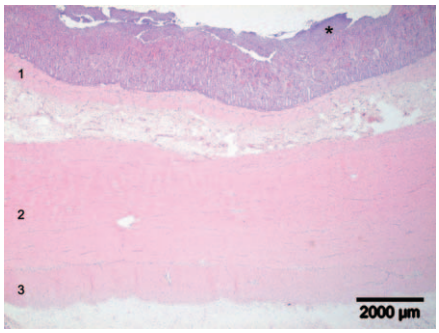

FIG 4. Transmural biopsy of the diverticulum in the duodenum. The mucosa lacks villi and is covered with purulent exudate (asterisk). The lamina muscularis mucosae (1), outer (longitudinal) muscular layer (3) and most notably the inner (circular) muscle layer (2) show marked hypertrophy without discontinuity. H&E

intestinal diverticula admitted between 1962 and 1988. In human beings, duodenal diverticula constitute about 5 per cent of all gastrointestinal diverticula (Wrenn and Hollabaugh 2000). Disruption of the process of endodermal proliferation and recanalisation during the embryological development of the primitive gut may result in various congenital anomalies, including these diverticula (Van der Gaag and Tibboel 1980, Abdel-Hafiz and others 1988). Two other possible aetiologies are mentioned in human literature. The first is an embryological pseudodiverticulum not including all layers of the bowel wall and resulting from a local weakening of the duodenal musculature. Obstruction of the common bile and pancreatic ducts

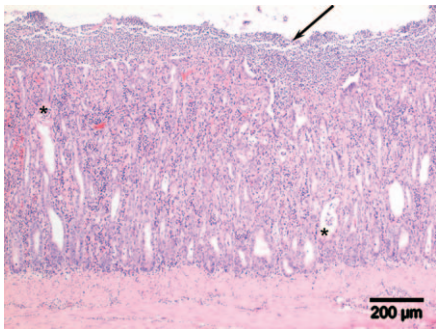

FIG 5. Mucosa of diverticulum. Elongated and frequently dilated crypts with absence of villi. The eroded mucosa is covered with purulent exudate (arrow). Lamina propria is moderately rich in inflammatory cells (plasma cells and towards the luminal side also neutrophils). Several crypt abscesses are visible (asterisk). H&E

are commonly caused by its location adjacent to the papilla of Vater. The second type is a true diverticulum caused by adhesion or extraluminal scarring related to peptic ulcers and are thus often located in the cranial duodenum (Knoefel and Rattner 2001). The duodenal diverticula reported here are most likely caused embryologically because of location and histological composition.

The initiating factor for this abnormal development in the two dogs remains obscure. The fact that these two boxers were littermates suggests a genetic origin or exposure to embryotoxic agents during gestation. No other littermates showed clinical signs of anaemia or intestinal problems and the mother was not exposed to any toxic agents according to the breeder. Screening the littermates by ultrasound was considered but omitted because ultrasound proved to be not sensitive enough in these two cases and barium-contrast radiography together with endoscopy was considered to be too invasive. Because the diverticulum had the same clinical manifestation, the same duodenal location and the same macroscopic and microscopic appearance, a genetic origin is likely.

The clinical signs of these boxers show similarity to the symptoms of children with a Meckel's diverticulum. A Meckel's diverticulum is the most frequent cause of painless, major gastrointestinal bleeding in a previously healthy infant. Fifty per cent occur before two years of age and more than 95 per cent of haemorrhaging Meckel's diverticula contain ectopic gastric mucosa, causing mucosal ulceration (Margolies 2001). Ectopic gastric mucosa is believed to result from maturation of multi-potential cells within the diverticular wall or from implantation of mature cell rests during maturation of the gut (Mackey and Dineen 1983). Ablin and others (1991) found ectopic gastric mucosa in three diverticula of the 13 intestinal diverticula examined. Ectopic gastric mucosa was not observed in the dogs reported here, and melena, severe anaemia and subsequent episodes of collapse were most likely caused by stasis of food and foreign material, fermentation and subsequent mucosal irritation, diverticulitis and rupture of superficial blood vessels.

Both dogs were treated with a partial enterectomy and end-to-end anastomosis to ensure lumen patency. Diverticulectomy with primary closure of the duodenal wall was not performed because the anatomical morphology of the intestinal wall was unknown at surgery. Prognosis after surgical resection of intestinal diverticula in dogs seems excellent unless intestinal perforation and peritonitis occur (Ablin and others 1991).

This is the first description of a duodenal diverticulum in two siblings. Although this congenital anomaly is rare, this diagnosis should not be ruled out in young dogs with a combination of persistent chronic anaemia and melena. Delayed diagnosis of the disease can lead to deterioration and collapse caused by intestinal haemorrhage and subsequent haemorrhagic shock.

References

Abdel-Hafiz, A. A., Birkett, D. H. & Ahmed, M. S. (1988) Congenital duodenal diverticula: a report of three cases and a review of the literature. *Surgery* **104**, 74-78

Ablin, L. W., Moore, F. M., Shields Henney, L. M. & Berg, J. (1991) Intestinal diverticular malformations in dogs and cats. *Compendium on Continuing Education for the Practicing Veterinarian* **13**, 426-430

Dowse, J. L. A. (1961) Meckel's diverticulum. *British Journal of Surgery* **48**, 392-399

Knoefel, W. T. & Rattner, D. W. (2001) Duodenal diverticula and duodenal tumours. In: Oxford Textbook of Surgery. 2nd edn. Eds P. J. Morris and W. C. Wood. Oxford University Press, Oxford. pp 943-946

Mackey, W. C. & Dineen, P. (1983) A fifty-year experience with a Meckel's diverticulum. *Surgery, Gynecology and Obstetrics* **156**, 56-64

Margolies, M. N. (2001) Diverticular disease of the small bowel. In: Oxford Textbook of Surgery. 2nd edn. Eds P. J. Morris and W. C. Wood. Oxford University Press, Oxford. pp 982-985

Marshall, W. S. & Hayes, M. J. (1966) An unusual congenital abnormality in the dog. *The Veterinary Record* **79**, 483-484

Ryan, M. E., Hamilton, J. W. & Morrissey, J. F. (1984) Gastrointestinal hemorrhage from a duodenal diverticulum. *Gastrointestinal Endoscopy* **30**, 84-87

Scudamore, C. H., Harrison, R. C. & White, T. T. (1982) Management of duodenal diverticula. *Canadian Journal of Surgery* **25**, 311-314

Van der Gaag, I. & Tibboel, D. (1980) Intestinal atresia and stenosis in animals: a report of 34 cases. *Veterinary Pathology* **17**, 565-574

Wrenn, E. L. & Hollabaugh, R. S. (2000) Alimentary tract duplications. In: Pediatric Surgery. 3rd edn. Eds K. W. Ashcraft, P. J. Murphy, R. J. Sharp, D. L. Sigalet and C. L. Snyder. W. B. Saunders, Philadelphia, PA, USA. pp 527-539