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CASE REPORT

Duodenal stricture and portal gas accumulation secondary to ulcerative, fibronecrotising duodenitis in a foal

R. F. Dash[1] | G. Manso-Diaz[2] | A. Suarez-Bonnet[3] | M. Hewetson[1]

[1]Equine Referral Hospital, The Royal Veterinary College, Hertfordshire, UK

[2]Universidad Complutense de Madrid, Madrid, Spain

[3]Pathobiology and Population Sciences, The Royal Veterinary College, Hertfordshire, UK

Correspondence: R. F. Dash
Email: rdash1@rvc.ac.uk

Summary

Gastroduodenal ulcer disease with secondary stricture formation of the proximal duodenum and portal gas accumulation is described in a neonatal foal. Conventional diagnostics were performed to reach a diagnosis, including gastroscopy, ultrasonography and positive contrast radiography. In addition, a post-mortem CT was acquired with the contrast media in situ. This permitted visualisation of the stricture itself and demonstrated the location to be pyloric and proximal duodenal, which was confirmed on post-mortem examination. Hepatic gas was observed on the initial radiographic and ultrasonographic examination; however, it could not be determined whether this was within the biliary system or portal venous system. CT demonstrated that this gas was within the portal veins. Portal venous gas accumulation has been reported in small animals as a sequela of necrotising gastroenteritis, gastrointestinal ulceration and gastric trauma, but has never been reported in horses. Previously, hepatic gas seen in foals with this presentation was thought to be in the biliary tree.

KEYWORDS

horse, computed tomography, foal, gastroduodenal ulceration, portal gas, stricture

INTRODUCTION

Gastroduodenal ulcer disease (GDUD) occurs commonly in foals with a prevalence of 22% at post-mortem (Elfenbein & Sanchez, 2012) and 52% at gastroscopy (Murray et al., 1990). Its aetiology is incompletely understood with some proposing it is due to increased acid exposure (Becht & Byars, 1986) and others attribute it to alterations in mucosal perfusion (Ryan & Sanchez, 2005). In many cases, GDUD may be subclinical, but clinical signs include inappetence, colic, ptyalism, bruxism and gastro-oesophageal reflux (Becht & Byars, 1986; Murray, 1999). GDUD can affect foals as young as 2 days of age (Murray et al., 1990), although neonatal foals are less commonly affected (Elfenbein & Sanchez, 2012). There is evidence that systemic disease and stress may predispose to the disease (Becht & Byars, 1986; Ryan & Sanchez, 2005). In particular, enterocolitis has been noted as a preceding factor (Hewetson & McGuire, 2022; Zedler et al., 2009). A possible sequala of this disease is gastric outflow obstruction caused by stricture and mural thickening of the pylorus and/or duodenum (Barr, 2006; Kent et al., 2020). Treatment and prognosis are dependent on the location. With a pyloric obstruction, a gastroduodenostomy (Zedler et al., 2009) or Heineke–Mikulicz pyloroplasty (Kent et al., 2020) can be performed, with foals reaching racing age in 100% and 66% of cases, respectively. However, with duodenal obstruction, a gastrojejunostomy with or without jejunojejunostomy is performed and prognosis is poorer, with only 50–52% reaching racing age (Coleman et al., 2009; Zedler et al., 2009).

A diagnosis of gastric outflow obstruction is commonly achieved via gastroscopy and orally administered positive contrast

Abbreviations: BPM, beats per minute; CT, computed tomography; EGGD, equine glandular gastric disease; GDUD, gastroduodenal ulcer disease; IgG, immunoglobulin G.

radiography (Barr, 2006; Kent et al., 2020). Hepatic gas has been described as a possible concurrent radiographic finding with GDUD in foals and has been suggested to be within the biliary system (Butler et al., 2010). This case report aimed to discuss the location and pathogenesis of this hepatic gas and demonstrate its appearance on several imaging modalities. It also provides the opportunity to present the appearance of a gastric outflow obstruction on computed tomographic (CT) examination and discuss the merit of this modality in more specifically localising the obstruction.

CASE HISTORY

Initial referral

A 2-day-old Warmblood filly was referred to the Royal Veterinary College, Equine referral hospital, for lethargy and a reduced appetite. The foal had been treated with ceftiofur (5 mg/kg bwt i.m. q. 12 h) for 24 h prior to presentation; however, there was no improvement, which prompted referral. On presentation, the foal was quiet, alert and responsive. Physical examination revealed a heart rate of 120 beats per minute (BPM) and hypermotile intestinal sounds. All other parameters were within normal limits. Abdominal and umbilical ultrasound were unremarkable. An immunoglobulin G (IgG) snap test revealed partial failure of passive transfer with an IgG of 4 g/L (normal > 8 g/L). The foal was initially treated with 2 L of hyperimmune plasma, and ceftiofur (5 mg/kg bwt i.v. q. 12 h) was continued. A blood culture was taken, with no bacteria isolated after 48 h.

The following day profuse diarrhoea developed and there was a marked deterioration in the foal's demeanour. Treatment for enterocolitis was initiated with ditrioctahedral smectite (Bio-Sponge, Platinum Performance; 500 mg/kg bwt p.o. q. 12 h), and antimicrobials were changed empirically to marbofloxacin (5 mg/kg bwt i.v. q. 24 h). Intravenous boluses of crystalloids were administered to correct the fluid deficit and maintain hydration. A faecal sample was negative for salmonella and rotavirus A and B RNA. Later, the foal was noted to have an enlarged stomach on abdominal ultrasound and the passage of a nasogastric tube yielded 500 mL of gastric reflux. This was managed by milk withdrawal, repeated nasogastric intubation, a continuous rate infusion of glucose (4 mg/kg/min) and pantoprazole (1.5 mg/kg IV q24h). This continued for 24 h, during which time the foal became brighter, and the gastric reflux diminished. Periods of nursing were then introduced and gradually increased.

After 10 days of hospitalisation, the foal's demeanour had improved and her diarrhoea had resolved. She was discharged on sucralfate (20 mg/kg, p.o. q. 8 h), lactase (6000 Food Chemical Codex units PO q8h) and omeprazole (4 mg/kg bwt p.o. q. 24 h) with the recommendation that she be closely monitored and any deterioration should be reported to a veterinarian.

Re-referral

Four days following discharge from the hospital, the foal was readmitted. The owner reported that the foal had initially done well but then became progressively duller and inappetent. The foal would latch onto the teat but would then abruptly stop. This was accompanied by ptyalism and bruxism.

CLINICAL FINDINGS

Upon re-presentation, the foal was standing but weak with a low head carriage and depressed demeanour. She was in poor body

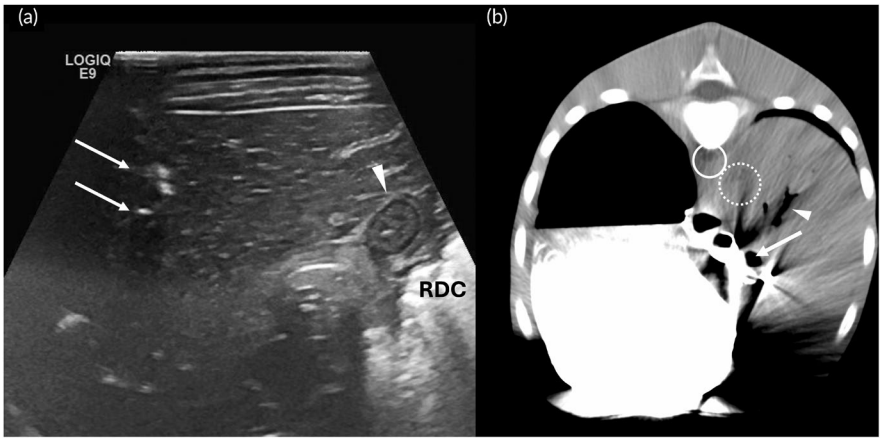

FIGURE 1 (a) Right lateral intercostal ultrasound window of the liver (dorsal is to the left), with hyperechoic foci (arrows) within the hepatic parenchyma which produces distal acoustic shadows. There is mild thickening of the wall of the duodenum (arrowhead). The right dorsal colon is labelled RDC. (b) Transverse computed tomographic image of the abdomen at the level of the T16, with a circle indicating the aorta, an interrupted circle indicating the caudal vena cava, an arrow at the gas filled portal vein and an arrowhead to the branching gas within the portal venous system (helical acquisition, 120 kV and 400 mAs, 512 × 512 matrix, 3 mm slice thickness, 1.5 mm slice interval, WW: 350, WL: 80, soft tissue kernel reconstruction).

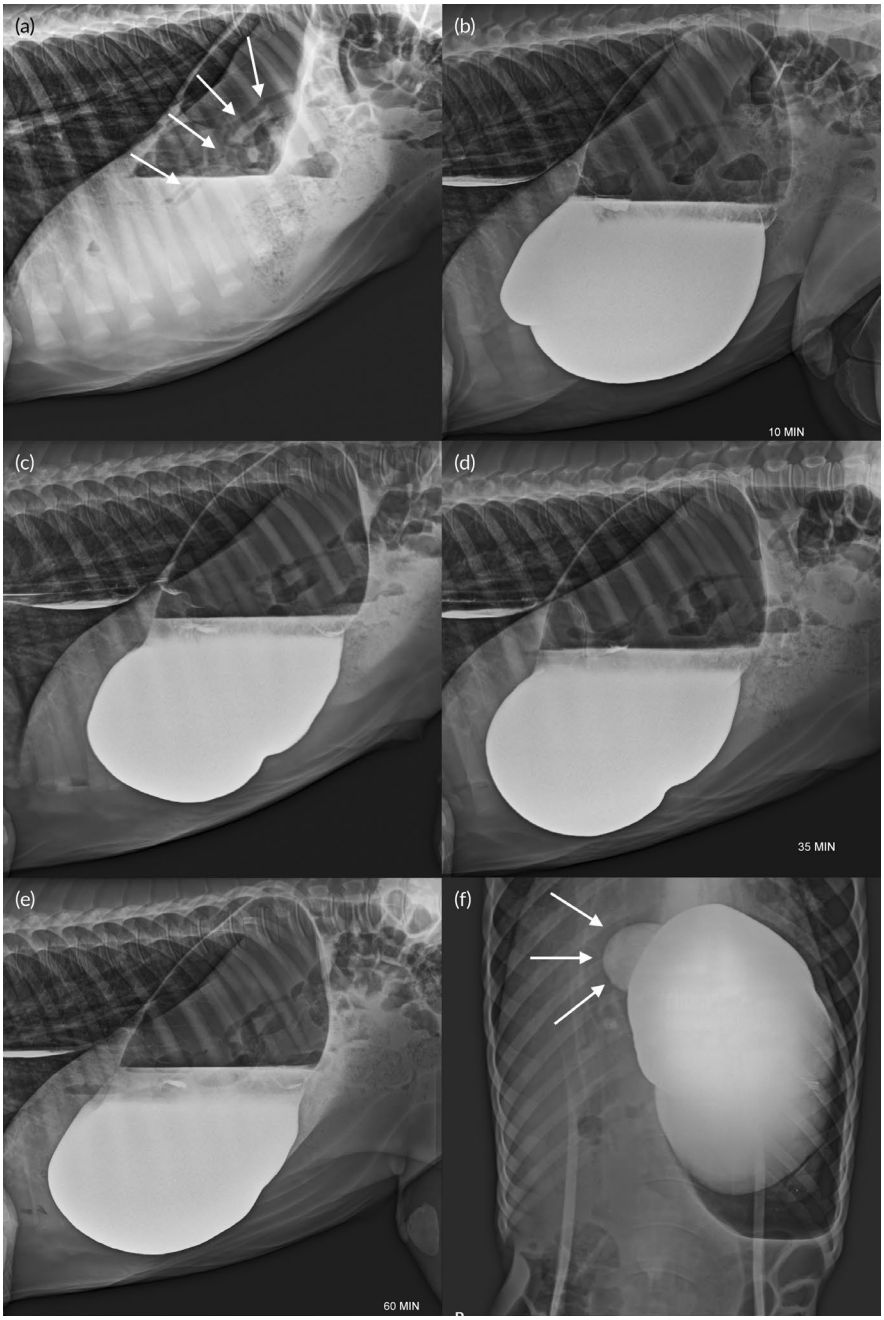

FIGURE 2 Positive contrast radiographic examination of the stomach. With laterolateral views that are (a) pre-contrast with arrows indicating the tubular gas opacities superimposed on the liver; (b) at 10 min; (c) at 20 min; (d) at 35 min; (e) at 60 min; and (f) a dorsoventral at 60 min with arrows indicating the abnormal truncation of the contrast at the level of the pyloric antrum.

condition and physical examination revealed a heart rate of 120 BPM. All other parameters were within normal limits. Abdominal ultrasound revealed a markedly fluid distended stomach, extending into the caudal abdomen. Nasogastric intubation yielded three litres of gastric reflux, and the ultrasound examination was continued. No free peritoneal fluid was seen. The small intestine was fluid-filled, but nondistended and motile throughout. The wall of the visible duodenum was subjectively mildly thickened (Figure 1a). Following this, gastroscopy was performed under light sedation. This revealed diffuse, severe squamous ulceration and a deformed, oedematous pylorus. The pylorus was amotile, and the lumen was not visible, suggestive of a gastric outflow obstruction. Treatment was re-commenced with pantoprazole (1.5 mg/kg bwt i.v. q. 24 h), marbofloxacin (5 mg/kg bwt i.v. q. 24 h), a glucose CRI (4 mg/kg/min) and intravenous isotonic fluid therapy.

The following morning, a positive contrast radiographic examination of the upper gastrointestinal tract was performed (Figure 2). Plain laterolateral radiographs of the abdomen were acquired, which showed mild gaseous distention of the stomach and several well-defined, tortuous, branching gas opacities superimposed on

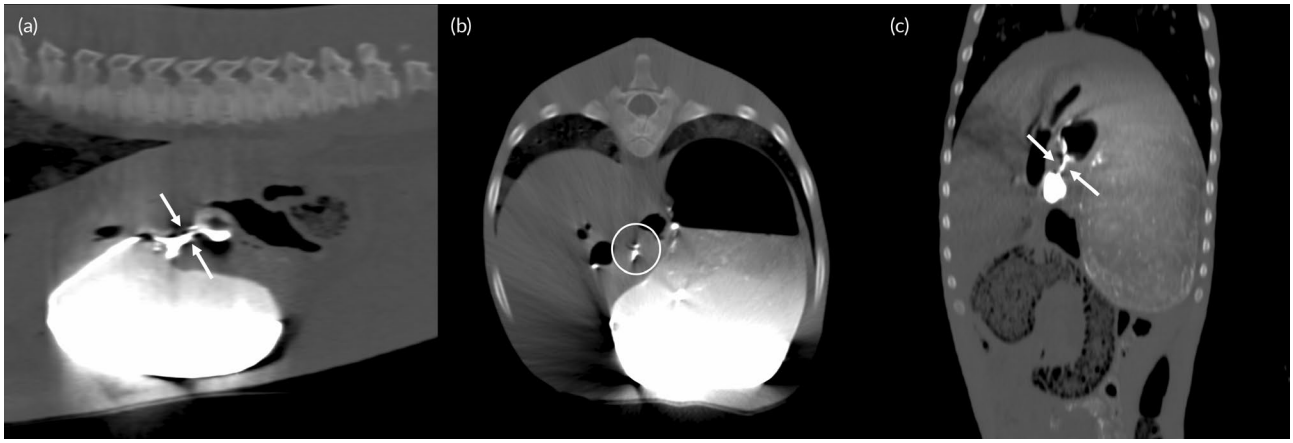

FIGURE 3 (a) Sagittal; (b) transverse; (c) dorsal reformatted computed tomographic images of the cranial abdomen demonstrating narrowing of the intraluminal contrast media at the level of the proximal duodenum (arrows and circle; helical acquisition, 120 kV and 400 mAs, 512 × 512 matrix, 0.6 mm slice thickness, 0.3 mm slice interval, WW: 2800, WL: 800, bone kernel reconstruction).

the hepatic silhouette, suggestive of gas in the biliary tree or portal venous system. A 30% weight by volume (WV) solution of barium sulfate was administered at 20 mL/kg via a nasogastric tube. Serial radiographic examination showed a complete lack of progression of the contrast medium beyond the stomach at 60 min, and a smooth rounded blunted appearance of the pyloric antrum. This was diagnostic of a gastric outflow obstruction, most likely the result of a stricture secondary to gastroduodenal ulceration. Other causes of mechanical obstruction such as an intussusception or volvulus were considered possible but much less likely. Additionally, within the ventral aspect of the caudal and accessory lung lobes, there was increased soft tissue opacity and faint air bronchograms indicative of an alveolar pattern. With the distribution and history, aspiration pneumonia was considered the most likely explanation. A focused abdominal ultrasound was performed to investigate the hepatic gas and demonstrated small, round, hyperechoic foci with distal acoustic shadows within the parenchyma of the liver, compatible with gas (Figure 1a). The exact location of the gas could not be established.

DIAGNOSIS

Findings were indicative of a gastric outflow obstruction due to a stricture of the pylorus or proximal duodenum, secondary to severe gastroduodenal ulcer disease. The hepatic gas was suggestive of portal venous gas secondary to the gastrointestinal ulceration or biliary gas due to incompetence of the sphincter of Oddi.

TREATMENT AND OUTCOME

Duodenal bypass surgery was offered to the owner, but with the comorbidity of aspiration pneumonia, a guarded prognosis was given, and they elected to euthanise the foal.

POST-MORTEM FINDINGS

Post-mortem CT

The body and pylorus of the stomach were moderately distended with contrast media. The lumen of the aborad aspect of the pyloric outflow tract and proximal duodenum tapered to a narrow point, such that only a thin irregularly marginated column of contrast media was present within a 20-mm segment of intestine (Figure 3). A small volume of contrast had progressed beyond this narrowing, demonstrating the normal diameter of the more aborad intestine.

Tubular branching gas-attenuating structures were present within the hepatic parenchyma which could be traced back to the hepatic portal vein (Figure 1b). The cranial half of the portal vein was also completely filled with gas.

The CT examination confirmed the precise location of the stricture to be the pylorus and proximal duodenum and also permitted the location of the hepatic gas to be established within the portal venous system.

Post-mortem examination

Gross examination revealed marked gastric dilation and severe diffuse ulceration. At the pyloric outflow tract and proximal duodenum, there was a focal, segmental, approximately 2 cm long region of marked constriction of the lumen with reduced patency. Additionally, bile duct distention was noted, and a scant amount of content was present within the remainder of the small intestine (Figure 4).

Microscopic examination through the site of duodenal stenosis identified a marked, acute ulcerative, fibrinonecrotising and haemorrhagic duodenitis, and moderate expansion of the submucosa with pyogranulomatous inflammatory infiltrates, including multinucleated giant cells, and areas of dystrophic mineralisation. The bile duct

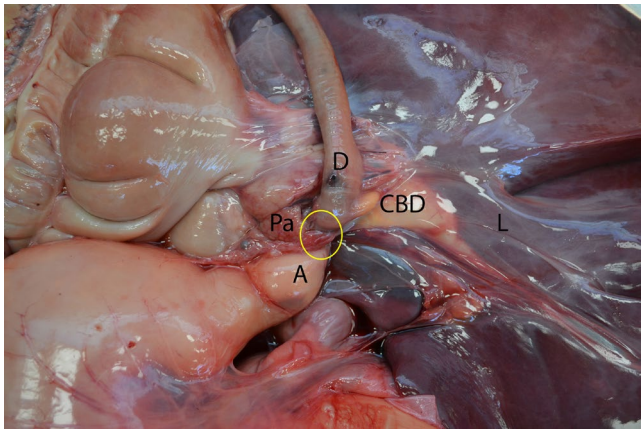

FIGURE 4 Gross post mortem photograph of the stricture of the proximal duodenum, with D: duodenum; Pa: pancreas; A: pyloric antrum; CBD: common bile duct; L: liver; a circle indicating the stricture.

was dilated and showed mild neutrophilic and lymphoplasmacytic inflammation. These findings were consistent with gastroduodenal ulcer disease and secondary stricture formation of the proximal duodenum (Palmer, 1985).

DISCUSSION

Hepatic gas has previously been described in foals with GDUD and was proposed to be within the biliary system (Butler et al., 2010). In cats and dogs, hepatic gas is reported to occur in both the biliary system (pneumobilia) and the portal venous system (Cordella & Bertolini, 2021; Moon Larson, 2018). Pneumobilia is associated with recent surgery or incompetence of the sphincter of the major duodenal papilla (sphincter of Oddi; Manfredi et al., 2019). Portal venous gas is associated with necrotising gastroenteritis, gastrointestinal ulceration and gastric trauma (Cartwright et al., 2016; Faverzani et al., 2009; Lee et al., 2015; Walczak et al., 2020). It has also been shown that portal gas is more tubular in appearance and more peripherally located within the liver (Cordella & Bertolini, 2021). The foal in the present case showed tubular gas within the liver which could be followed to the hepatic portal vein, which was also gas filled, resulting in a diagnosis of portal venous gas. Considering the pathogenesis of pneumobilia and portal venous gas in small animals, it also seems more likely that a foal with GDUD would develop secondary portal venous gas.

For a foal with gastric outflow obstruction, the prognosis with surgery depends on the location of the stricture. This can be pyloric/proximal duodenal or duodenal (Zedler et al., 2009). In this case, the post-mortem CT examination with orally administered positive contrast media determined the location of the obstruction to be pyloric/proximal duodenal. On this basis, the foal could have undergone a gastroduodenostomy, which offers a better prognosis. This information was not available until after the foal was euthanised, but could have been beneficial in providing a more accurate prognosis and assisting with surgical planning. CT examination is also financially costly, and in this case, there was evidence of concurrent aspiration pneumonia. Therefore, this information would have been unlikely to affect the owner's decision to euthanise the foal. In future cases, this CT procedure could be performed in an anaesthetised foal to establish the precise location of the stricture, with the appropriate precautions to prevent aspiration.

This foal presented initially with signs of enterocolitis and was discharged following treatment, before representing 4 days later with GDUD and a gastric outflow obstruction. The outflow obstruction was attributed to segmental ulcerative duodenitis and diffuse thickening of the duodenum that obstructed duodenal flow (Acland et al., 1983; Becht & Byars, 1986). The pathogenesis of GDUD in foals is poorly understood; however, affected foals are often reported to have had diarrhoea prior to developing clinical signs, suggesting that enterocolitis may play a role in the pathogenesis of the disease in this age group (Zedler et al., 2009). This is supported by the fact that gastroduodenal ulceration is more frequently associated with gastrointestinal disease in nonsurviving foals (Elfenbein & Sanchez, 2012). It has always been assumed that the primary inciting factor in foals that develop a gastric outflow obstruction is acid injury affecting the glandular mucosa of the pylorus and proximal duodenum. However, it is possible that primary enterocolitis is in fact the inciting cause in these cases, and that gastroduodenal ulceration is secondary, as was demonstrated in this case. The squamous mucosa was most severely affected, as would be expected in secondary squamous gastric disease resulting from delayed gastric emptying and chronic gastric distension. Whilst the pylorus appeared oedematous and deformed, the overlying glandular mucosa was intact, suggesting that primary glandular gastric disease was unlikely to have been the inciting cause in this case. This is in contrast to adult horses with gastric outflow obstruction, where duodenal lesions are rarely reported (Bezdekova et al., 2020).

This foal was treated with protected antimicrobials and justification for the antimicrobial choices is therefore warranted. Ceftiofur is justified in this case as a first-line broad-spectrum antimicrobial in a neonatal foal with suspected sepsis due to the high mortality (BEVA ProtectMe, 2020). Marbofloxacin is also justified in this case due to systemic deterioration and the development of acute onset enterocolitis despite treatment with ceftiofur. The choice of marbofloxacin was based on concerns over *Salmonella*, *Enterobacter* and other Gram-negative *Enterobacteriaceae* species. Faecal samples were submitted for culture but due to the delay in reporting results, it was decided to switch empirically to marbofloxacin due to the acute deterioration and risk to life. The foals responded favourably to the marbofloxacin. Upon representation, marbofloxacin was recommenced due to the initial improvement on this drug and acute deterioration following cessation of treatment.

In conclusion, portal venous gas can be seen in foals with GDUD. CT examination with orally administered positive contrast media could be useful to establish the location of a stricture, to inform on prognosis, and aid in surgical planning. Enterocolitis may be a factor in the pathogenesis of GDUD in foals.

AUTHOR CONTRIBUTIONS

R. F. Dash: Investigation; writing – original draft; writing – review and editing; resources. **G. Manso-Diaz:** Investigation; writing – original draft; writing – review and editing; resources. **A. Suarez-Bonnet:** Investigation; writing – original draft; writing – review and editing; resources. **M. Hewetson:** Investigation; writing – original draft; writing – review and editing; supervision; resources.

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CONFLICT OF INTEREST STATEMENT

The authors declare they have no conflicts of interest.

ETHICS STATEMENT

Ethical approval was not required according to the policy of the Clinical Research Ethical Review Board at The Royal Veterinary College. Informed client consent for inclusion in this study was obtained.

ORCID

R. F. Dash https://orcid.org/0000-0003-4955-8841
G. Manso-Diaz https://orcid.org/0000-0002-8548-669X
A. Suarez-Bonnet https://orcid.org/0000-0003-0296-5896
M. Hewetson https://orcid.org/0000-0001-6518-0864

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