Acute pancreatitis in two dogs

Acute pancreatitis bij twee honden

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ABSTRACT

Acute pancreatitis in 2 dogs is discussed. A possible underlying cause was present in 1 dog. The clinical signs and routine blood results were unspecific and insufficient to confirm the diagnosis. Acute pancreatitis was diagnosed during exploratory celiotomy (case 1) and by abdominal ultrasonography (case 2). Aggressive medical treatment consisted of fluid administration, analgesics, anti-ulcer therapy, antibiotics, transfusion of fresh frozen plasma (FFP) and nutritional support. Despite the presence of negative prognostic factors, intensive therapy and monitoring resulted in complete recovery in both patients.

SAMENVATTING

In dit artikel worden 2 honden met acute pancreatitis besproken. Een mogelijke onderliggende oorzaak was aanwezig in 1 geval. De klinische presentatie en de algemene bloedresultaten waren aspecifiek en onvoldoende om de diagnose te bevestigen. De diagnose van acute pancreatitis werd bevestigd tijdens een exploratieve celiotomie in geval 1 en met behulp van abdominale echografie in geval 2. De agressieve behandeling bestond uit vloeistoftherapie, pijn- en antiulcermedicatie, antibiotica, plasmatransfusie en nutritionele ondersteuning. Ondanks de aanwezigheid van negatieve prognostische factoren resulteerden een intensieve therapie en opvolging in een compleet herstel van beide patiënten.

CASE REPORTS

Patient 1

A two-year-old, spayed female Bull terrier (25.3 kg, moderately obese) was referred to the Department of Small Animal Medicine and Clinical Biology at the Faculty of Veterinary Medicine (Ghent University) with a five-day -history of lethargy, nausea, acute vomiting and eating grass. She had been anorectic for 2 days. Uptake of a foreign body and intoxication were possible causes according to the owner. Her maintenance food consisted of Science Plan Adult Beef dry food (Hill's®, Breda, The Netherlands). The last 13 months she had been receiving corticosteroids (Prednisolone® 0.4 mg/kg every other day) and cephalosporine (Cephadroxil® 25mg/kg twice a day) for the treatment of an allergic dermatitis. Physical examination revealed weak femoral pulses and strong breathing sounds on auscultation. The dog had a tense abdomen and showed a prayer position. Rectal examination was unremarkable. Initial blood examination (complete blood count (CBC), serum biochemistry) showed anemia, hyponatraemia, hypertryglyceridemia, azotemia and increased alkaline phosphatase activity (Table 1).

Abdominal radiographs were performed because of abdominal pain and the possibility of a gastrointestinal foreign body (Figures 1a and 1b). Focal loss of serosal detail in the dorsal and right central abdomen suggested free abdominal fluid and a localized peritonitis. In the cardia of the stomach, 2 foreign bodies were present. Radiographic signs suggestive of gastrointestinal obstruction were not present.

The dog was rehydrated with Hartmann's solution (90 ml/kg/24 hours). An intravenous treatment with ranitidine, amoxicillin-clavulanic acid and methadone was started (Table 2). No food or water was given orally. Eight hours after onset of medical treatment the dog underwent an exploratory celiotomy during which a gastrotomy was performed to remove the foreign bodies (2 pieces of rubber). Severe necrotizing pancreatitis was noticed and the pancreas showed adhesions to the liver, right kidney and abdominal wall (Figure 2). A moderate amount of free fluid was present and sampled. This revealed a modified transudate that contained non-degenerative neutrophils and red blood cells. The results of aerobic and anaerobic bacterial cultures of the abdominal fluid were negative. An abdominal lavage was performed in an effort to remove the enzymes and cytokines present (Caronna *et al.*, 2009).

Table 1. Results of CBC, biochemistry and coagulation profile of patient 1.

Blood results	Patient 1	Reference range
Hematocrit (%)	32.9	43 - 59
White blood cells (10^3/mm$^{3)}$	6.6	6 - 12
Thrombocytes (10^3/mm^3)	189	150 - 500
Albumin (g/l)	22	20 - 30
Total protein (g/l)	60	60 - 80
Blood ureum nitrogen (mmol/l)	25.4	< 12
Creatinine (µmol/l)	358	< 72 + 1.2 x BW
Alkaline phosphatase (IU/l)	285	< 147
Alanine aminotransferase (IU/l)	41	< 120
Bile acids (µmol/l)	28	< 12
Glucose (mmol/l)	4.2	2.2 - 8.2
Sodium (mmol/l)	135.3	145 - 155
Potassium (mmol/l)	4.79	2.7 - 5
Calcium (mmol/l)	2.27	2.21 - 2.79
Cholesterol (mmol/l)	8.38	2.40 - 9.67
Triglycerides (mmol/l)	10.9	0.07 - 1.53
Prothrombine time (sec)	10.5	5 - 11
APTT (sec)	72.1	10 – 20
D-Dimers (ng/ml)	889	< 250 (reference for humans)
Fibrinogen (mg/dl)	770	100 – 460

APTT = activated partial thromboplastin time

Table 2. Medications used during and after hospitalization in patients 1 and 2.

Active component	Product used	Dose
Amoxicillin-clavulanic acid	Augmentin®	20 mg/kg IV q 8h
	Clavubactin®	12.5 mg/kg PO q 12h
Enrofloxacine*	Baytril®	5 mg/kg IV q 24h or 2.5 mg/kg PO q 12h
Heparin	Heparin LEO®	75-150 IU/kg IV or SC q 6h
Methadone	Mephenon®	0.1-0.2 mg/kg IV q 4h
Metoclopramide	Primperan®	0.3 mg/kg IV or PO q 8h
Phenobarbital+	Natrium Phenobarbital®	5 mg/kg IM q 12h
	Gardenal®	50 mg PO q 12h
Ranitidine	Zantac®	2 mg/kg IV or PO q 12h

*: only used in patient 1; +: only used in patient 2

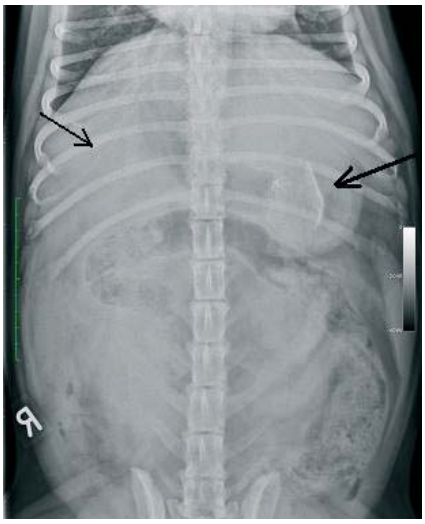

Figure 1a. Ventrodorsal radiographic image of the abdomen of patient 1: loss of serosal detail in the right cranial region (small arrow). Two corpora aliena are present in the cardia of the stomach (big arrow).

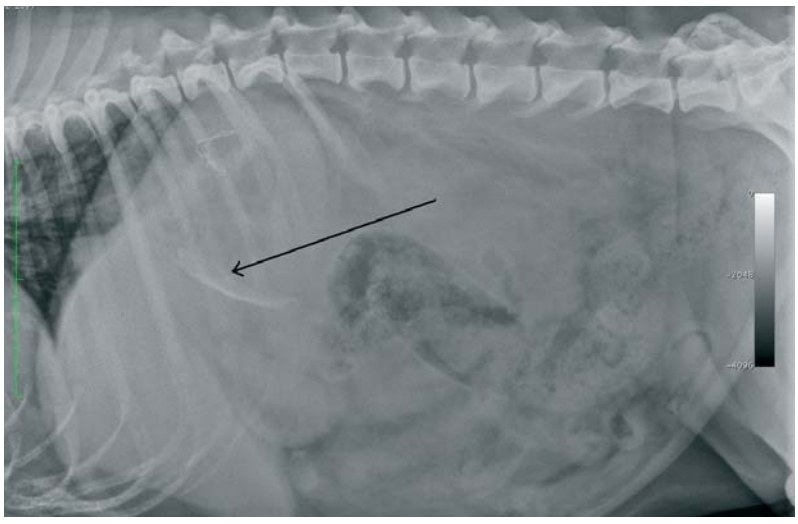

Figure 1b. Lateral radiographic image of the abdomen of patient 1. The arrow points towards the 2 corpora aliena present in the cardia of the stomach.

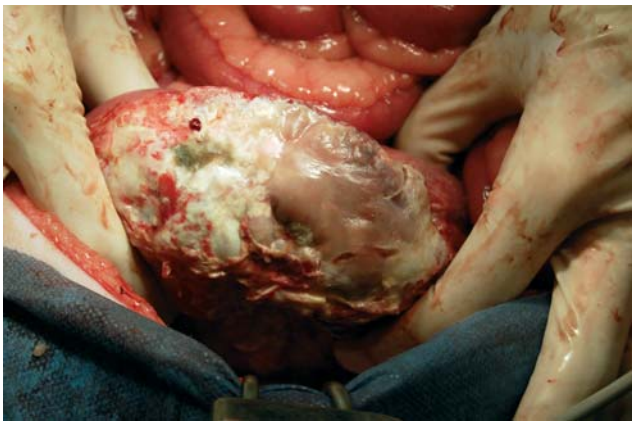

Figure 2. Right pancreatic limb demonstrating necrotic areas covered by fibrin.

To ensure appropriate post-operative nutritional support without stimulating pancreatic secretion, a transcutaneous jejunal feeding tube (V-PFT-5-35, Cook Medical Australia, Queensland) was inserted. Further, to monitor urine production and ensure hygiene, a 10Fr indwelling urinary Foley catheter (Kendall Curity, Tyco Healthcare UK, Hampshire) was placed.

Post-operatively, the dog was installed in the intensive care unit and received maintenance (Sterofundin®43.2 ml/kg/24hours) and replacement (NaCl 0.9% 43.2 ml/kg/24hours) fluids. Treatment with intravenous amoxicillin-clavulanic acid and ranitidine was continued for 5 and 7 days, respectively. Intravenous metoclopramide was initiated post-operatively, and intravenous methadone was continued for 5 days and then weaned off over the next 2 days (Table 2). The vital parameters, as well as urine production, blood pressure and blood glucose levels, were monitored during hospitalization. To provide α-macroglobulines and because of the possibility of developing DIC, a fresh frozen plasma transfusion (FFP) (60 ml/kg/24hours) was started immediately after surgery. Unfractionated heparin was added to the plasma to activate antithrombin, and this was followed by subcutaneous administration of heparin over the next 3 days (Table 2). The blood work was repeated 1 hour after surgery: the anemia had worsened mildly, probably due to dilution by the pre- and intra-operative fluid therapy, and mild thrombocytopenia and hypoalbuminemia had developed, but the renal values were decreasing. Because thrombocytopenia can indicate the development of disseminated intravascular coagulation (DIC), a citrate-coagulated blood sample was taken to evaluate the coagulation parameters. The coagulation results 1 day after surgery confirmed the presence of DIC, based on thrombocytopenia, moderately increased d-dimers and prolonged activated partial thromboplastin time (Table 1). Because the dog developed fever the first day post-operatively, intravenous followed by oral administration of enrofloxacine was added to the antibiotic treatment for the next 4 days (Table 2). One day after surgery, enteral feeding was started by the jejunal feeding tube with Convalescense Support Instant (Royal Canin®, Aimargues, France) (1/3 of Resting Energy Requirements or RER=3077 kJ Metabolized Energy) divided over 7 meals a day. The caloric amount was slowly increased over 3 days to the RER, observing that no vomiting occurred. Two days after surgery, small amounts of water and food (Digestive Low Fat canned food, Royal Canin®, Aimargues, France) were presented orally every three hours. The dog's appetite returned soon and the amount of food presented orally was increased during the further hospitalization, while the infusion rate was gradually decreased and discontinued on day 5. Tube feeding was discontinued on the third day post-operatively, and the jejunal tube was removed 3 days later. After 8 days of hospitalization, the dog was discharged on an oral therapy of amoxicillin-clavulanic acid and ranitidine for 1 week (Table 2). Her diet consisted of Digestive Low Fat dry food (Royal Canin®, Aimargues, France), divided over 3 meals each day. The dog was followed by the referring veterinarian, who informed us by telephone 2 months after discharge that the dog had made a perfect clinical recovery, and that the measurement of canine trypsin like immunoreactivity (cTLI) 2 months after discharge was within reference range.

Patient 2

A 4.5-year-old, male intact Boston terrier (8.9 kg, slightly obese) was referred to the Department of Small Animal Medicine and Clinical Biology at the Faculty of Veterinary Medicine (Ghent University) with a two-day history of dullness, anorexia and acute vomiting. The dog had a history of primary epilepsy for 2 years, for which it was receiving phenobarbital (Gardenal® 7 mg/kg twice daily) in combination with potassium bromide (Epikal®). The epilepsy was well controlled. A mild nasal and pharyngeal stridor due to brachycephalic airway syndrome was present. At physical examination, all vital parameters were normal. A tense and painful abdomen was the only abnormality observed. Rectal examination was unremarkable. Blood analysis (CBC, serum biochemistry) showed a severe leukocytosis with a left shift, thrombocytosis, mild hypoproteinemia, hypoalbuminemia, azotemia, hyperglycemia and increased liver enzymes (alkaline phosphatase, alanine aminotransferase, gamma-glutamyl-transferase). Phenobarbital levels were within normal limits, in contrast to those of potassium bromide, which were above the toxic serum levels. Urinalysis revealed isosthenuria, pyuria and hemoglobinuria (Table 3).

A diagnosis of severe acute pancreatitis was established on abdominal ultrasound: the entire right lobe of the pancreas was swollen, hypoechoic and surrounded by hyperechoic fat and a small amount of anechoic fluid (Figure 3). A blood sample taken for coagulation profile revealed severely increased d-dimers and fibrinogen (Table 3).

The dog was hospitalized and received a FFP transfusion at a rate of 60 ml/kg/24hours supplemented with unfractionated heparin, NaCl 0.9% (81.6 ml/kg/24hours), methadone, amoxicillin-clavulanic acid, ranitidine, metoclopramide and phenobarbital (Table 2). The dog was fasted for 24 hours and then re-

Table 3. Results of CBC, biochemistry, coagulation profile and urinalysis of patient 2.

	Patient 2	Reference range
Blood results		
Hematocrit (%)	49.6	43 - 59
White blood cells (/µl)	43120	6000 - 16000
Band neutrophils (/µl)	3881	0 - 300
Thrombocytes (/µl)	645000	164000 - 510000
Albumin (g/l)	25.2	31 - 44
Total protein (g/l)	49	54 - 76
Blood ureum nitrogen (mmol/l)	11.32	1.66 - 8.65
Creatinine (µmol/l)	50.4	60 +BW
Alkaline phosphatase (U/l)	565	< 123
Alanine aminotransferase(U/l)	247	< 52
Aspartate aminotransferase (U/l)	70	< 44
Gamma-glutamyl transferase (U/l)	31	< 8
Total bilirubin (µmol/l)	< 1.7	< 3.9
Direct bilirubin (µmol/l)	< 1.7	< 10.2
Glucose (mmol/l)	6.22	3.05 - 4.99
Sodium (mmol/l)	149	136 - 154
Potassium (mmol/l)	4.3	4 - 5.5
Cholesterol (mmol/l)	7.84	2.4 - 9.67
Triglycerides (mmol/l)	1.23	0.07 - 1.53
Prothrombine time (sec)	6.7	5 - 11
APTT (sec)	20.2	10 - 20
D-Dimers (ng/ml)	2807	< 250 (reference in humans)
Fibrinogen (mg/dl)	968	100 - 460
Phenobarbital (mg/l)	26	20 - 30
Potassium bromide (mg/l) toxic dose > 2000	2400.25	750 – 1550
Urinalysis		
White blood cells (/µl)	50	< 25
Red blood cells (/µl)	4	< 25
Crystals	negative	
pH	7	4.5 - 7
Specific gravity	1.016	1.015 – 1.035
Hemoglobin	positive	
Aceton, urobilinogen, bilirubin, leucocyte esterase, nitrite	negative	
Protein/creatinine ratio	10.53	< 0.5
Glucose (mmol/l)	1.943	0.333 – 1.11
Culture	negative	

APTT = activated partial thromboplastin time; BW = body weight

ceived small amounts of Digestive Low Fat canned food (Royal Canin®, Aimargues, France) divided over 7 small meals per day. The dog vomited twice after his second meal, and was therefore kept sober for an additional 12 hours before feeding was recommenced.

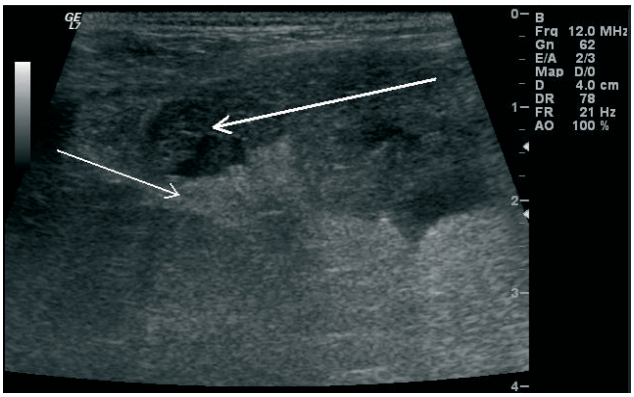

Figure 3. Ultrasonographic image in patient 2: hypoechoic enlarged pancreas (big arrow) lined by hyperechoic mesentery (small arrow).

Food intake was then gradually increased. After 4 days of hospitalization, the dog was discharged on this diet, and he also received oral amoxicillin-clavulanic acid for 10 days and phenobarbital (Table 2). The dosage of potassium bromide was slowly decreased over a period of 15 days and then completely discontinued. Ranitidine and metoclopramide were continued for 1 extra week (Table 2). At home, the dog recovered well. A follow-up visit at 5 months after discharge revealed no problems. His diet consisted of Digestive Low Fat dry food (Royal Canin®, Aimargues, France), which the owner combined with pasta, rice and chicken, supplemented with omega 3 fatty acids (fish oil). The changes to the anti-epileptic therapy treatment did not result in the occurrence of any seizures in the following 2 months.

DISCUSSION

The signalment of the 2 patients described is grossly compatible with the current literature on acute pan-

creatitis in companion animals, although the Bull terrier was rather young (Cook *et al*., 1993; Hess *et al.,* 1999; Bunch, 2003; Watson, 2004; Mix and Jones, 2006). The majority of acute pancreatitis cases are classified as idiopathic (Watson, 2004; Watson, 2007b), but the history of both patients revealed possible risk factors. At the present time, corticosteroids have only been proven to be a possible risk factor if high doses are given for the treatment of intervertebral disc disease (Watson, 2004). Since this was not the case in the Bull terrier, which had only received a low dose of prednisolone, it is questionable whether this medication was the underlying cause of the disease. However, because corticosteroids inhibit the clearance of proteases bound to α-macroglobulines (Watson, 2004), it was advised to cease this treatment. Gaskill and Cribb (2000) detected a 29 times greater risk for developing acute pancreatitis in epileptic dogs treated with potassium bromide/phenobarbital combination therapy instead of phenobarbital monotherapy. It was not clear, however, whether the increased risk was due to the potassium bromide or to the combination therapy. Because the level of potassium bromide exceeded the toxic dose in the Boston terrier, the medication was tapered and finally discontinued since the epilepsy was well controlled.

As expected with acute pancreatitis, the initial complaints and blood work were rather vague in both dogs, and too unspecific to be diagnostic. The rapid resolution of the azotemia in both dogs after initiation of fluid therapy confirmed that the azotemia was rather pre-renal (dehydration) than renal in origin. The increase in alkaline phosphatase activity in both dogs could have been due to cholestasis or to enzyme induction by corticosteroids or phenobarbital. The increase in alanine aminotransferase in patient 2, an enzyme associated with hepatocellular damage, can be explained by hypoxia, ischemia, inflammation or phenobarbital administration. Because of the severe pancreatitis, the coagulation profile was determined in both dogs. Disseminated intravascular coagulation (DIC) could only be confirmed in the Bull terrier. The Boston terrier only had severely increased d-dimers, an abnormality that can be a sign of hypercoaguability and thus a risk for developing DIC (Bunch, 2003).

Because it was impossible to get a diagnosis solely based on the history and blood results, medical imaging was performed in both patients. In the Bull terrier, abdominal radiographs revealed signs of a localized peritonitis suggestive of acute pancreatitis (Bunch; 2003; Ruaux, 2003; Watson, 2004), but many other causes could also explain this image. The most important differential diagnosis in this dog was the presence of a focal septic peritonitis caused by perforation of the gastrointestinal tract secondary to the foreign bodies. Because the 2 gastrointestinal foreign bodies were already an indication to perform a surgical exploration, no further diagnostics (such as ultrasound or abdominocentesis) were performed to look for other possible causes. The final diagnosis of necrotizing pancreatitis was made during macroscopic examination. Most likely, the pancreatitis was the major contributor to the clinical signs since the gastrointestinal foreign bodies did not cause severe mucosal irritation or mechanical obstruction. In the second patient, a diagnosis of acute pancreatitis was highly suspected on abdominal ultrasonography, which is a reliable test, but very machine and operator dependent (Simpson and Lamb, 1995; Ruaux, 2003; Watson, 2004; Mix and Jones, 2006).

In none of the patients were serum amylase and lipase measured, because of the disadvantages of these tests. In dogs, neither lipase nor amylase is pancreas specific, and their values depend on renal clearance, the use of steroids and the severity of the disease (Simpson and Lamb, 1995; Bunch, 2003; Ruaux, 2003; Watson, 2004). Although serum amylase and lipase were historically the standard tests for diagnosing acute pancreatitis in companion animals, these tests are no longer recommended (Simpson and Lamb, 1995; Bunch, 2003; Ruaux, 2003; Watson, 2004; Mix and Jones, 2006). Nowadays immunoassays, such as canine pancreatic lipase immunoreactivity (cPLI) and cTLI, are preferred over enzymatic assays, because of the higher specificity and sensitivity (Ruaux, 2003; Watson, 2004). In our patients, neither cTLI nor cPLI was measured during the disease, as the diagnosis had already been either made during surgery or suspected at abdominal ultrasonography. In addition, it would have taken several days to get the results of the 2 tests.

Biopsy of the pancreas remains the golden standard to diagnose pancreatic pathologies (Newman, 2006). Instead of invasive exploratory surgery, minimally invasive laparoscopic biopsy techniques are available that have been proven successful in the diagnosis of pancreatitis (Webb and Trott, 2008). Having histological biopsies to confirm the diagnosis is especially important in low-grade disease where other diagnostic tests are non-conclusive. This was not the case in our patients, however. Although macroscopic assessment during surgical exploration (first patient) and ultrasound examination (second patient) are considered to be less reliable diagnostic techniques, they were easily capable of making a distinct diagnosis in these 2 dogs.

The basic rules for treating acute pancreatitis in dogs are fluid therapy and the prevention of oral intake (Bunch, 2003). Because of the presence of necrotizing pancreatitis in the Bull terrier, it was expected that oral intake needed to be delayed for a substantial amount of time (Watson, 2007a). Therefore a choice was made to place a jejunal feeding tube allowing early enteral nutrition (Qin 2007). Enteral nutrition is the most natural route for administering nutrients and it improves the integrity and function of the intestinal mucosa (Qin *et al*., 2002; Qin *et al*., 2003; Wortinger, 2006). Moreover, a recent study by Mansfield (2008) showed that clinical abnormalities indicating poor intestinal integrity (e.g. no enteral food intake > 3 days, hematochezia, melena, regurgitation) are some of the most important negative prognostic indicators in dogs with acute pancreatitis. This demonstrates the importance of early enteral nutrition for these patients. Both

Table 4. Key values of Royal Canin Digestive Low Fat.

Values	RC Digestive Low Fat
Protein (%)	22
Fat (%)	5
Carbohydrate (%)	55.1
Crude Fibre (%)	1.7
Calcium (%)	1
Phosphorus (%)	0.8
ME (kJ/100g)	1.479

dogs received Digestive Low Fat (Royal Canin®, Aimargues, France), which contains only 5% fat, while the low fiber content ensures high digestibility and nutrient absorption. The key values of this type of diet are summarized in Table 4.

Both patients were given FFP combined with unfractionated heparin to activate antithrombin. Plasma also delivers proteinase inhibitors that bind pancreatic enzymes (Watson, 2004). Antibiotics were used in both dogs, because one had a severe form of necrotizing pancreatitis and developed fever, while the other had a severe leukocytosis with a left shift. The use of both enrofloxacine and amoxicillin-clavulanic acid can be justified: the first type penetrates well into pancreatic tissue and has a wide spectrum, while the other type of antibiotic is effective against many Gram-negative and Gram-positive aerobe micro-organisms and obligate anaerobes (Ramsey, 2008). Both dogs received metoclopramide as anti-emetic therapy. The use of this medication can be debated, because as a gastroprokineticum it can cause stimulation of the pancreas (De la Puente-Redondo *et al*., 2007). Maropitant, a strong anti-emeticum that inhibits both central and peripheral causes of vomiting (De la Puente-Redondo *et al*., 2007), seems more appropriate for these patients, but was not yet available at the time. Both patients recovered well, even though the prognosis in the Bull terrier was initially guarded because of the severity of the pancreatitis and the development of DIC.

CONCLUSION

Although pancreatitis is usually idiopathic, searching for an underlying risk factor is mandatory to successfully treat the episode and prevent recurrence. Aggressive medical treatment with fluids, pancreatic rest, early nutritional support, transfusion of FFP, antibiotic therapy, anti-ulcer medication and analgesics is mandatory. Although negative prognostic factors were present, intensive care monitoring and therapy led to favorable outcome in both dogs.

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