Student Paper Communication étudiante

Acute pancreatitis attributed to dietary indiscretion in a female mixed breed canine

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Abstract — A female, mixed-breed dog was presented with signs of abdominal discomfort and vomiting of 24 h duration following an episode of dietary indiscretion. Clinical signs, previous medical history, and diagnostic tests supported a diagnosis of acute pancreatitis. Specific and supportive treatment was instituted, and clinical signs resolved 10 d after presentation.

Résumé — Pancréatite aiguë attribuée à une indiscrétion alimentaire chez une chienne de race croisée. Une chienne de race croisée est présentée avec des signes d'inconfort abdominal et des vomissements depuis 24 heures après un épisode d'indiscrétion alimentaire. Les signes cliniques, les antécédents médicaux et les tests diagnostiques appuient le diagnostic de pancréatite aiguë. Un traitement spécifique et de soutien est institué et les signes cliniques se résorbent 10 jours après la présentation.

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An 11-year-old female, yellow Labrador retriever crossbred was presented to the Arthur Veterinary Clinic, Arthur, Ontario with signs of anorexia and vomiting of over 24 h duration after having consumed household wastes. The dog had a medical history of several bouts of mild to moderate abdominal pain, vomiting and diarrhea, subsequent to periods of dietary indiscretion. To the owners' knowledge, no toxins, or materials known to be difficult to digest, were present in the waste.

Case description

Upon initial presentation, the dog appeared extremely lethargic and weak. The weakness was noted to be especially severe in the pelvic limbs, with the dog being unable to walk more than a few steps without collapsing. The physical examination revealed an increased rate and effort of open-mouthed respiration, injected oral mucous membranes, a prolonged (> 3 s) capillary refill time (CRT), and the presence of white foam on the tongue. The dog was in good body condition (3/5 body condition score) and was estimated to be 7% dehydrated. Tachycardia (a heart rate of 170 beats/min) and mild hypothermia (37.1°C) were noted. A mildly distended and extremely painful abdomen was detected on abdominal palpation; however, there was no evidence of organomegaly.

Differential diagnoses based on the history, clinical signs, and physical examination included: acute pancreatitis, foreign body or toxin ingestion, liver or renal disease, gastric dilatation volvulus (GDV), and leptospirosis. Initial supportive treatments consisted of crystalloid intravenous fluids (Lactated Ringer; Baxter Corporation, Mississauga, Ontario) at a rate of 112 mL/h for 6 h (maintenance plus 7% dehydration), hydromorphone hydrochloride (Hydromorphone HP 10; Sandoz, Boucherville, Quebec) 0.1 mg/kg body weight (BW), IV, and 12.375 mg trans-dermal fentanyl (ratio-fentanyl; Ratiopharm, Mississauga, Ontario) delivered at a rate of 75 μg/h for a total of 72 h.

Preliminary diagnostic tests included abdominal radiographs, a complete blood (cell) count (CBC), a full biochemical profile, and a canine pancreatic lipase SNAP test (Canine SNAP cPL; IDEXX Laboratories, Westbrook, Maine, USA). Ventro-dorsal and right-lateral abdominal radiographs were normal. The CBC and biochemistry profile revealed a mild, mature neutrophilia; a high normal hematocrit and globulins; moderate hypochloremia, hyperphosphatemia, and hypercholesterolemia; a mild elevation in urea; a high-normal creatinine value; and a severely elevated alkaline phosphatase (ALKP). Numeric results of these tests are summarized in Table 1.

No toxic changes were noted on the blood smear and the neutrophilia was attributed to inflammation. The elevated urea, high-normal creatinine, and hyperphosphatemia were suspected to be due to pre-renal causes; this was further supported by the high-normal hematocrit and total protein values, as well as the physical examination finding of dehydration. The hypochloremia was most likely due to a loss of chloride in the vomitus. The severely elevated ALKP was hypothesized to be due to inflammation of the liver, secondary to inflammation of the pancreas, or

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Table 1. Complete blood (cell) count and biochemical profile results prior to treatment, June 2, 2009

Parameter	Result	Reference interval
Segmented neutrophils	13.7 H	3.3 to 12.0 × 10^9 cells/L
Hematocrit	0.54	0.37 to 0.55 L/L
Total protein	77	52 to 82 g/L
Globulins	45	25 to 45 g/L
Chloride	96 L	109 to 122 mmol/L
Phosphorus	2.45 H	0.81 to 2.19 mmol/L
Cholesterol	12.93 H	2.84 to 8.27 mmol/L
Urea	11.6 H	2.5 to 9.6 mmol/L
Creatinine	142	44 to 159 μmol/L
ALKP	1740 H	23 to 212 U/L

H — high, L — low, ALKP — alkaline phosphatase.

Table 2. Complete blood (cell) count and biochemical profile results post treatment, June 5, 2009

Parameter	Results	Reference interval
Segmented neutrophils	9.0	3.3 to 12.0 × 10^9 cells/L
Hematocrit	0.54	0.37 to 0.55 L/L
Total protein	75	52 to 82 g/L
Globulins	46 H	25 to 45 g/L
Chloride	94 L	109 to 122 mmol/L
Phosphorus	1.75	0.81 to 2.19 mmol/L
Cholesterol	11.10 H	2.84 to 8.27 mmol/L
Urea	10.9 H	2.5 to 9.6 mmol/L
Creatinine	142	44 to 159 μmol/L
ALKP	1177 H	23 to 212 U/L

H — high, L — low, ALKP — alkaline phosphatase.

possibly due to hyperadrenocorticism. The hypercholesterolemia was suspected to be due to acute pancreatitis. The Canine SNAP cPL test returned a positive result and this, combined with the dog's history, physical examination, radiographic results, and blood and biochemistry profile, established a preliminary diagnosis of acute pancreatitis. The dog was therefore treated with ampicillin sodium (Novopharm, Toronto, Ontario), 500 mg (22 mg/kg), IV, q8h to prevent proliferation of bacteria in the damaged pancreas, and was fasted for 24 h. She was reassessed 8 h after being admitted, and since dehydration was no longer clinically detectable, the fluid rate was reduced to 35 mL/h (maintenance).

Twenty-four hours after the dog had been presented to the AVC, she was assessed to be mentally more alert and her respiratory rate and effort had begun to decrease. The dog had also not vomited since being admitted to the hospital and she was able to drink small quantities of water without vomiting. A leashed walk revealed that her pelvic limb weakness had begun to improve and she was able to walk at a slow, controlled pace without collapsing. The dog was discharged from the hospital with cephalexin (Novo-lexin; Novopharm) 500 mg (22 mg/kg), PO, q12h for 5 d, maropitant citrate (Cerenia; Pfizer Animal Health, Kirkland, Quebec) 4 mg/kg PO, q24h, in case she should require an anti-emetic, and a canned intestinal diet (Canine I/D diet; Hill's Pet Nutrition, Topeka, Kansas, USA). The fentanyl patch was to remain in place for the following 48 h. The owners were instructed to feed the dog a tablespoon of the diet, and if the dog did not vomit, a small meal could be offered. The clients were asked to bring the dog back in a week's time, provided the clinical signs did not reoccur, so that her clinical status could be re-evaluated and her CBC and biochemistry profile could be repeated.

Shortly after discharge from the hospital, the dog began vomiting after drinking water. A 24-h period of fasting and administration of maropitant citrate tablets (60 mg, PO) resulted in transient improvement and the dog was able to eat small amounts of the intestinal diet. The dog then became anorexic. The owners were instructed to remove the fentanyl patch in case this had been modifying the dog's behavior, causing the anorexia to persist. However, 4 h after the fentanyl patch had been removed, the dog vomited and was re-admitted to the hospital.

Upon re-presentation at the clinic, the owners informed the veterinarian that the dog was able to keep water and medication down, but had eaten very little since being discharged. The dog appeared lethargic upon presentation, but according to the clients, did appear brighter and more alert after removal of the fentanyl patch. No bowel movements or diarrhea were reported, and the dog was observed to experience full body tremors intermittently. Temperature and pulse were normal and the mucous membranes were pink and moist, with a CRT of < 2 s. Excessive panting was noted and the dog had a tense and painful abdomen. The owners reported that the dog had postured to defecate on a number of occasions since leaving the hospital, but had not produced feces. A rectal examination was performed, but was normal. In light of the physical examination findings and the dog's general appearance, a CBC and biochemical profile were recommended. The results of the CBC and biochemical profile revealed that blood values were beginning to normalize. A urine specific gravity was normal (1.018). The results of the CBC and biochemical profile are summarized in Table 2.

The dog's painful abdomen warranted the administration of meloxicam (Metacam; Boehringer Ingelheim, Burlington, Ontario), 0.1 mg/kg, BW, SC and tramadol hydrochloride (Chiron Compounding Pharmacy, Guelph, Ontario), 2 mg/kg, BW, PO, q8h. A crystalloid solution (500 mL) was administered SC, as subclinical dehydration was suspected in light of the high-normal hematocrit and total protein values. The clients were advised to withhold food for 48 h, and then introduce the intestinal diet slowly. Seventy-two hours after re-presentation, the owners reported that the dog had begun to eat small quantities of food and no vomiting had been observed. She was also noted to be more active and no longer lethargic or weak. The clients were instructed to continue feeding small meals of the intestinal diet, and to bring the dog in once a week to obtain accurate body weights.

Ten days after initial presentation no further problems were reported. The owners were advised to carefully monitor the dog for any signs of vomiting or abdominal pain, and to strictly adhere to feeding her an intestinal diet to prevent reoccurrence of the problem.

Discussion

Pancreatitis may occur in chronic and acute forms (1,2) and is reportedly the most common disease of the exocrine pancreas in dogs (3). This disease is believed to be multi-factorial in

origin, involving a complex interplay between factors including: breed, age, weight, trauma, drug use, and concurrent endocrine disease (4). Episodes of pancreatitis can range from mild to severe, where patients may exhibit a plethora of signs from mild lethargy to multiple organ failure or death (4). It is important to remember the variability of the clinical signs that may be displayed by dogs with pancreatitis (2) and its implications for diagnosis. In spite of this observed variability, clinical signs reported to be common include dehydration, anorexia, vomiting, and weakness (3).

Pancreatitis results from the inappropriate activation of exocrine pancreatic digestive enzymes within the parenchyma of the pancreas (1,4). In the normal pancreas, these digestive enzymes exist as inactive precursors, called zymogens, which become activated by the enzyme enterokinase, present in the small intestine (1,5). In pancreatitis, this important safety feature, which normally prevents autodigestion of the pancreatic tissue, is disrupted, and the zymogens become prematurely activated (5). Several factors may be involved in the disruption of this mechanism and the inciting cause is often unidentified (4,5). The disorder is observed more commonly in dogs than in cats (5), and poses a significant challenge to the practitioner with respect to both diagnosis and treatment (3).

Due to the vague clinical signs reported in the majority of animals with pancreatitis, the diagnostic workup of these cases is often extensive. A CBC, biochemical profile, urinalysis, and abdominal radiographs are indicated for most patients, acting to rule out other differential diagnoses, rather than to provide specific results to support a diagnosis of pancreatitis (4,6). Traditionally, serum amylase and serum lipase were used to diagnose pancreatitis in dogs but these enzymes are neither sensitive nor specific for the canine pancreas (2,3) and pancreatitis cannot be ruled out based on finding normal levels of these enzymes in the dog (6). Recently, new diagnostic tests, including tests measuring species-specific pancreatic lipase immunoreactivity (PLI) have been validated (2). Studies have revealed that PLI is limited to the acinar cells of the canine pancreas, and thus is a specific marker indicating damage to the pancreas (1,2). Pancreatic lipase immunoreactivity is currently considered to be the most reliable and sensitive blood test for diagnosis of canine pancreatitis (3,6). These promising findings have lead to the development of a cage-side SNAP canine pancreatic lipase immunoreactivity (cPLI) test; however, it is still recommended that a positive SNAP test result be followed with measurement of serum PLI levels (6). Abdominal ultrasonography is cited as being a useful aid in the diagnosis of canine pancreatitis (2,3), and is considered superior to survey radiographs (3,5) but is operator-dependent. Again it should be noted that normal findings on ultrasound cannot rule out pancreatitis, and higher resolution equipment may actually allow for over-interpretation of findings leading to false positives (6). Histopathology remains the gold standard in terms of diagnosis, and is the only modality that may distinguish acute and chronic pancreatitis (3,6).

Treatment of acute pancreatitis in the dog is complex and involves both general supportive therapy and patient-specific treatment regimens (4,5). Fundamentals of treatment involve removing the inciting cause (if identified), providing appropriate fluid therapy and traditionally, being strictly adherent to the "treatment" of "nothing by mouth" (4,5). However, although fasting is recommended to give the organ a respite, it has more recently been shown that appropriate nutritional therapy is critical for the maintenance of homeostasis and to support healing (4,6). It is, therefore, generally not advisable to withhold nutrition beyond 2 to 3 d, including the length of time the dog may have been anorexic before presentation (4). Complicating factors must also be appropriately managed, including the potential for bacterial infection in the damaged pancreas (5); however, antibiotic therapy should not be applied empirically, and is indicated in cases where complications are likely, or have been identified as bacterial in origin (6). Aggressive pain control is also critical for the well-being of the patient and is necessary to speed recovery (4,5); appropriate first-line analgesics include opioids, such as morphine and buprenorphine, whereas long-term pain control may be achieved with fentanyl patches and non-steroidal anti-inflammatory drugs (NSAIDs) (4,6). In spite of the need to provide the patient with appropriate and adequate analgesia, side effects associated with opioid administration, including vomiting, constipation and ileus, should be considered (4). In addition, caution should be exercised when prescribing NSAIDs to patients suffering from diarrhea, and those which are vomiting or dehydrated (6). Additional adjunctive therapy may include the use of antiemetics, such as dolasetron and maropitant, if vomition continues (5,6).

Canine pancreatitis is a difficult disease to manage, where the interplay between inciting causes and risk factors remains enigmatic and, in spite of the development of more specific and sensitive tests, the diagnosis may be elusive. The accurate and timely treatment of canine pancreatitis is, therefore, reliant upon the practitioner's ability to incorporate historical data, physical examination findings, and nonspecific and specific testing to arrive at a diagnosis and devise an effective and patient-specific treatment plan.

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