Chylous ascites associated with abdominal trauma and intestinal resection-anastomosis in a pet ferret (*Mustela putorius furo*)

Lucile Chassang DMV

Isabelle Langlois DMV

Pauline Loos DMV

Mila Freire DMV, PhD

Elizabeth O'Toole DMV

From the Department of Veterinary Clinical Sciences, Faculty of Veterinary Medicine, University of Montréal, St-Hyacinthe, QC J2S 7C6, Canada. Dr. Chassang's present address is Exotics Department, Centre Hospitalier Vétérinaire Frégis, 94110 Arcueil, France. Dr. Loos' present address is Department of Infectious and Parasite Diseases, Veterinary Vaccinology, University of Liège, 4000 Liège, Belgium.

Address correspondence to Dr. Langlois (isabelle.langlois@umontreal.ca).

CASE DESCRIPTION

A 10-week-old 0.73-kg (1.6-lb) castrated male domestic ferret (*Mustela putorius furo*) was referred for exploratory laparotomy because of pneumoperitoneum and possible septic peritonitis after being bitten by the owner's dog.

CLINICAL FINDINGS

Abdominal exploration revealed a large laceration of the duodenum, tears of the jejunal mesentery, and 2 small tears in the abdominal wall. Chylous abdominal effusion developed 48 hours after surgery.

TREATMENT AND OUTCOME

Postoperative care included supportive treatment, analgesia, and antimicrobials. An abdominal drain was placed during the laparotomy and enabled monitoring of abdominal fluid production. Enteral feeding was provided through an esophagostomy tube. The chylous fluid production rapidly decreased after treatment with octreotide was initiated, and the ferret improved. Chyloabdomen resolved after 8 days of hospitalization and medical treatment.

CLINICAL RELEVANCE

Findings suggested that chylous ascites can potentially develop secondary to blunt abdominal trauma in ferrets. In this ferret, chyloabdomen was successfully treated with octreotide administration and abdominal drainage. (*J Am Vet Med Assoc* 2018;252:1272–1278)

A 10-week-old 0.73-kg (1.6-lb) castrated male domestic ferret (*Mustela putorius furo*) was referred to the Exotic Animal Clinical Service of the Veterinary Teaching Hospital at the University of Montréal for exploratory laparotomy because of pneumoperitoneum and possible septic peritonitis after being bitten by the owner's dog.

The ferret had been evaluated by the referring veterinarian a few hours after the traumatic incident. On physical examination, the patient was lethargic and recumbent and had a high-normal rectal temperature (39.8°C [103.6°F]; reference range, 37.8° to 40°C [100.4° to 104°F]).[1] The referring veterinarian observed abdominal distension and ecchymoses, but no associated penetrating bite wounds. Abdominal palpation elicited signs of pain. Initial stabilization by the referring veterinarian included IV crystalloid fluid therapy[a] (8 to 12 mL/kg/h [3.6 to 5.5 mL/lb/h]) and analgesia (hydromorphone, 0.1 mg/kg [0.045 mg/lb], IV, q 4 h). Survey radiography revealed loss of abdominal serosal detail consistent with abdominal effusion, free air in the abdomen, loss of integrity of the right abdominal wall with apparent herniation of the small intestines into the subcutaneous tissues, and contusions involving the right middle lung lobe. Given the patient's history and the results of physical examination and radiography, the referring veterinarian suspected the patient had a traumatic abdominal wall tear and perforation of a hollow viscus, resulting in septic peritonitis. Contrast radiography confirmed the presence of a perforated loop of intestine, and the ferret was referred for exploratory laparotomy and further treatment.

On referral examination, the ferret was stuporous, tachypneic (48 respirations/min; reference range, 33 to 36 respirations/min),[2] tachycardic (380 beats/min; reference range, 180 to 250 beats/min),[3] and laterally recumbent, with a rectal temperature of 39.9°C (104°F). The mucous membranes were pale and lacked observable capillary refill. The abdomen was distended, and signs of severe pain were elicited on palpation.

A tear involving the right lateral aspect of the abdominal wall was suspected. High-normal rectal temperature was attributed to the septic process because the referring veterinarian reported a similar rectal temperature before the ferret was transferred, reducing the likelihood that transportation, stress, or excitement contributed to this finding. Furthermore, the history and physical examination findings suggested hyperdynamic circulatory shock, a condition that may be associated with septic peritonitis.

Indirect blood pressure measured by means of Doppler ultrasonography was low (systolic blood pressure, 50 mm Hg; reference range, 80 to 120 mm Hg).[4,5] A CBC and serum biochemical analyses revealed a moderately low PCV (31%; reference range, 40%

ABBREVIATIONS

REE Resting energy expenditure

to 70%), markedly high BUN concentration (29.5 mmol/L; reference range, 4.8 to 16.9 mmol/L), and normal serum creatinine concentration (35 µmol/L; reference range, 23.0 to 76.7 µmol/L).[6] Moderate hypoproteinemia (48 g/L; reference range, 54.7 to 77.9 g/L)[6] was detected and attributed to septic peritonitis and gastrointestinal bleeding secondary to abdominal trauma. The patient's hyponatremia (131 mmol/L; reference range, 140.1 to 169.7 mmol/L)[6] was most likely caused by third-space fluid accumulation. Blood glucose concentration (8.4 mmol/L; reference range, 3.0 to 8.4 mmol/L)[6] was within reference limits.

A focused assessment with sonography for trauma was performed on the abdomen and revealed hyperechoic peritoneal effusion and small intestines distended with fluid. Further, no peristaltic movement was observed, consistent with generalized intestinal ileus.

For stabilization and in preparation for an emergency exploratory laparotomy, enrofloxacin (10 mg/kg [4.5 mg/lb]) and a bolus of lactated Ringer solution[b] (10 mL/kg) were administered IV. The ferret's heart rate decreased to 250 beats/min, and capillary refill time could now be assessed but was prolonged (< 3 seconds; reference range, ≤ 2 seconds).[7]

The ferret was premedicated with midazolam (0.3 mg/kg [0.14 mg/lb], IV) and fentanyl (8 µg/kg, IV) and was preoxygenated for 5 minutes via facemask. Anesthesia was induced with ketamine (5 mg/kg [2.3 mg/lb], IV), and the ferret was intubated with a cuffed endotracheal tube that had an inner diameter of 3 mm. Anesthesia was maintained with isoflurane, and intraoperative monitoring included capnography, ECG, and pulse oximetry performed with a multiparameter patient monitor.[c] Analgesia was provided with infusions of fentanyl (5 to 10 µg/kg/h, IV) and lidocaine (25 µg/kg/min [11.4 µg/lb/min], IV). Because of the septic shock and to improve blood pressure, a phenylephrine (1 to 2 µg/kg/min [0.45 to 0.9 µg/lb/min], IV) infusion was initiated at the time of anesthetic induction, and boluses of hydroxyethyl starch[d] (5 mL/kg, IV, divided over 3 boluses) and lactated Ringer solution[b] (5 mL/kg, IV) were administered. Placement of an arterial catheter for direct blood pressure measurements was unsuccessful, and a decision was made to monitor the patient's blood pressure indirectly by the oscillometric method. Because only a single measurement (systolic blood pressure, 90 mm Hg for 2 hours after anesthetic induction) was successfully obtained during the anesthetic procedure, the patient's heart rate was used as an indirect indicator of blood pressure for intraoperative fluid therapy decisions. Factors that contributed to unsuccessful monitoring of blood pressure by the oscillometric method included the patient's severe hypotension and the limited intraoperative access to perform these measurements.

A ventral midline laparotomy was performed. The abdomen contained serosanguineous fluid mixed with what appeared to be greenish intestinal contents **(Figure 1)**. A sample of the fluid was obtained for

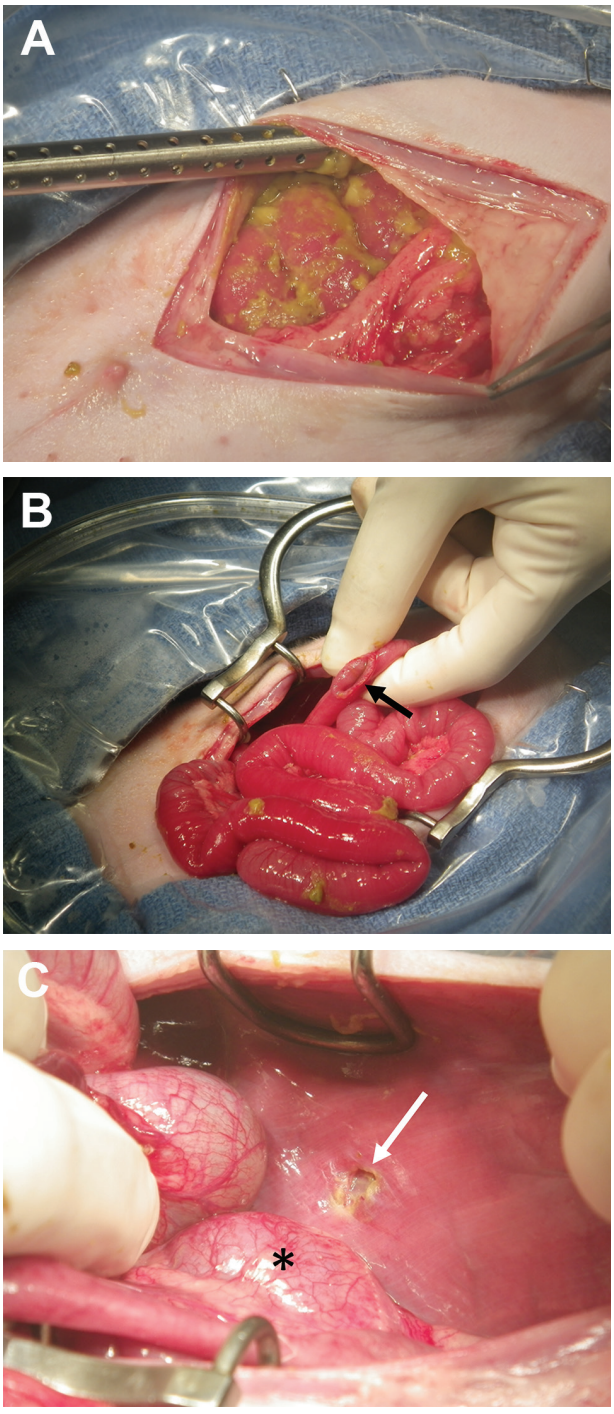

Figure 1—Intraoperative photographs obtained during exploratory laparotomy in a 10-week-old domestic ferret (*Mustela putorius furo*) that had been bitten by a dog. A—The abdominal cavity contained serosanguineous fluid mixed with intestinal contents. B—The jejunum was dilated, suggestive of paralytic ileus, and a laceration of the descending duodenum (arrow) was observed. C—The cranial right aspect of the abdomen had an abdominal wall perforation (arrow) next to the right kidney (asterisk).

aerobic and anaerobic bacterial culture. The descending duodenum had a large laceration perforating the mesenteric and antimesenteric aspects 1 cm distal to the major duodenal papilla. Several small mesenteric tears were present, and a large portion of the jejunum appeared dilated and paralyzed. Vascularization of the jejunum did not appear to be compromised. Two small perforations of the abdominal wall were present, one on each side near the right and left kidneys. A duodenal resection and anastomosis was performed. The mesenteric tears and the abdominal wall perforations were closed. Prior to abdominal closure, the abdomen was lavaged with warm sterile saline (0.9% NaCl) solution, and a 10-mm Jackson-Pratt surgical drain[e] was placed for postoperative abdominal drainage and fluid production monitoring. The anesthetic procedure required 4 hours and 30 minutes from the time of induction to extubation.

Immediate postoperative intensive care included fluid therapy with crystalloid (lactated Ringer solution,[b] 2.5 mL/kg/h [1.1 mL/lb/h], IV) and colloid (hydroxyethyl starch,[d] 1 mL/kg/h, IV) solutions and administration of enrofloxacin (10 mg/kg, IV, q 12 h) and cefazolin (22 mg/kg [10 mg/lb], IV, q 8 h). Phenylephrine (0.5 µg/kg/min [0.23 µg/lb/min], IV) administration was discontinued within the first hour after surgery, and the vasopressor, norepinephrine (0.1 to 1 µg/kg/min, IV) was administered instead. Nevertheless, severe systolic hypotension (35 to 44 mm Hg) persisted during the first 12 hours after surgery. Analgesia was provided with infusions of fentanyl (1.25 to 5 µg/kg/h [0.57 to 2.3 µg/lb/h], IV) and ketamine (0.1 to 0.4 mg/kg/h [0.045 to 0.18 mg/lb/h], IV). Constant rate infusions were used to avoid peaks and troughs observed with IM administration and to enable quick adjustments of dosages on the basis of patient requirements. Because hypoglycemia (1.1 to 2 mmol/L) was noted during anesthesia, blood glucose concentration was monitored every 4 to 8 hours. Norepinephrine was administered in 5% dextrose solution to prevent further hypoglycemia, and blood glucose concentration (3.7 to 8.4 mmol/L) remained within reference limits overnight. During the first 12 hours after the surgical procedure, a substantial volume of serosanguineous fluid (6.7 mL/kg/h [3.05 mL/lb/h]) had been collected through the abdominal drain. Drain care consisted of stabilization of the drainage bulb tubing to the ferret's body with an abdominal bandage and disinfection of the tubing and ports with alcohol during each manipulation of the apparatus. In the postoperative period, the ferret's hemodynamic status was closely monitored through frequent evaluation of systolic blood pressure by means of Doppler ultrasonography, urine output measurements, and a continuous ECG.

During the first 24 hours after the surgery, the ferret's general condition mildly improved; however, the ferret continued to be hypotensive with systolic blood pressure ranging from 60 to 65 mm Hg despite continuous infusion of norepinephrine. The ferret was stuporous and tachycardic (260 to 280 beats/min). No urine or feces were produced overnight. A CBC was performed and revealed severe leukopenia (1.36×10^9 WBCs/L; reference range, 3.0×10^9 WBCs/L to 16.7×10^9 WBCs/L) due to neutropenia (0.27×10^9 cells/L; reference range, 0.9×10^9 cells/L to 7.4×10^9 cells/L).[6] There was a left shift (bands, 0.16×10^9 cells/L; reference range, 0 to 0.1×10^9 cells/L)[6] with toxic changes present in the neutrophils consistent with a septic process. Serum biochemical analyses revealed a high alkaline phosphatase activity (312 U/L; reference range, 13.3 to 141.6 U/L), consistent with sepsis-induced cholestasis, and severe hypoalbuminemia (11.5 g/L; reference range, 28.0 to 43.9 g/L) and hypoglobulinemia (19.1 g/L; reference range, 20 to 29 g/L),[6] consistent with loss in the abdominal inflammatory effusion. Furthermore, a high creatinine concentration (110 µmol/L; reference range, 23.0 to 76.7 µmol/L)[6] combined with a urine specific gravity (1.040; reference range, 1.034 to 1.060)[8] within reference limits suggested prerenal azotemia. Therefore, fluid therapy was altered from lactated Ringer solution[b] to an alkalinizing solution[a] (10 mL/kg/h, IV) with a slightly higher sodium content to maintain the patient's serum sodium concentration within reference limits.

Given the ferret's critical state on postoperative day 1, further clinical support was provided. General anesthesia was induced for placement of a 10F esophagostomy tube and a urinary catheter. The urinary catheter was placed to enhance fluid balance monitoring by enabling accurate comparisons of the total fluids received by all methods to total fluids lost via the urinary catheter and abdominal drain. This monitoring continued every 4 hours until the urinary catheter was removed on postoperative day 5. Changes in fluid balance, body weight, and clinical condition were used to make fluid therapy decisions during this time. Also on postoperative day 1, additional medications were started to stimulate gastrointestinal motility and aid in tolerance of future enteral feedings (metoclopramide, 2 mg/kg/d, IV), reduce nausea (maropitant, 1 mg/kg, IV, q 12 h), and control and prevent potential gastritis (famotidine, 0.5 mg/kg, IV, q 8 h). At this time, the patient's analgesic treatment consisted of multiple constant rate infusions (fentanyl, 1.25 to 2 µg/kg/h, IV; ketamine, 0.2 to 0.3 mg/kg/h [0.09 to 0.14 mg/lb/h], IV; and lidocaine, 10 µg/kg/min, IV) to obtain an acceptable level of pain control. To broaden the antimicrobial spectrum, metronidazole (20 mg/kg [9.1 mg/lb], IV, q 12 h) administration was also initiated.

On day 2, treatment continued as previously described, and enteral feedings[f] through the esophagostomy tube were implemented when the ferret's systolic blood pressure was ≥ 80 mm Hg. Enteral feedings were started at a third of the patient's calculated daily REE, with feedings performed every 2 hours. Primary goals of the initial enteral feedings were to provide nutritional support to the intestinal tract and enhance local blood flow to optimize healing at the surgical sites. Because the ferret had higher

urine output (8 mL/kg/h; reference range, 1.08 to 1.17 mL/kg/h)[2] than expected and had developed severe diarrhea, the IV fluid rate was modified such that the volume administered by IV and enteral feeding equaled the total volume collected through the urinary catheter and abdominal drain.

On day 3, the ferret's general condition improved substantially. The ferret was more alert and responsive and began to move voluntarily in its cage. Heart rate, respiratory rate, and rectal temperature were within reference ranges, and even though the dosage of norepinephrine was decreased (0.7 µg/kg/min [0.32 µg/lb/min], IV), measurements of systolic blood pressure (70 to 105 mm Hg) were within the reference range. Monitoring of blood glucose concentration revealed hyperglycemia (18.6 mmol/L) that was attributed to the use of 5% dextrose as the IV fluid delivery solution for the norepinephrine infusion. Therefore, a single dose of a short-acting insulin[g] (0.5 U, SC) was administered, the constant rate infusion of norepinephrine in 5% dextrose solution was discontinued, and a constant rate infusion of norepinephrine in saline solution was initiated. The ferret responded well, and no further episodes of hyperglycemia were noted. Analgesic requirements progressively decreased, and administration of fentanyl was discontinued, with ongoing analgesia provided with ketamine (0.1 mg/kg/h, IV) and lidocaine (10 mg/kg/h, IV) infusions and the addition of buprenorphine (0.02 mg/kg [0.01 mg/lb], IV, q 8 h). Administration of metoclopramide and antimicrobials was continued as previously described. Modifications to gastrointestinal support were implemented with the substitution of omeprazole (2.5 mg/kg, PO, q 24 h) for famotidine and the discontinuation of maropitant. Enteral feedings via the esophagostomy tube were well tolerated, and the volume fed was increased to 50% of the patient's REE. A repeated CBC showed mild anemia (PCV, 33%) that was attributed to hemodilution, blood sampling, and inflammation. Neutrophil (6.39 × 10^9 cells/L), lymphocyte (3.24 × 10^9 cells/L; reference range, 0.6 × 10^9 cells/L to 10.5 × 10^9 cells/L),[6] and WBC (10.14 × 10^9 cells/L) counts were within reference ranges; however, a left shift (bands, 0.41 × 10^9 cells/L) and toxic changes in the neutrophils persisted. Serum biochemical analyses revealed persistent hypoalbuminemia (12.2 g/L) and hypoglobulinemia (18.4 g/L), consistent with ongoing loss via the digestive tract and the inflammatory processes associated with sepsis. Azotemia and hyponatremia had resolved. Abdominal fluid production (6 mL/kg/h [2.7 mL/lb/h]) remained high, and the fluid started to take on a cloudy white appearance.

On day 4, abdominal fluid production had increased (8.2 mL/kg/h [3.7 mL/lb/h]), and the fluid had a milky appearance **(Figure 2)**. Analysis of the fluid was performed, and results were consistent with a chylous effusion with nonseptic neutrophilic inflammation. Triglyceride concentration in the effusion was 5.99 mmol/L, compared with 0.99 mmol/L (reference range, 0.5 to 2.8 mmol/L)[6] in the serum. The effusion's total solids concentration, measured by refractometry, was 11 g/L, but was artificially high because of the high lipid content. Cytologic examination of the effusion revealed a total cell count of 1.05 cells/µL, with the predominant cells being lymphocytes (43%), nondegenerative neutrophils (38%), and monocytes (19%). No microorganisms were seen. On the basis of the effusion's cytologic and biochemical analyses results, a chylous effusion was diagnosed. Therefore, the enteral feedings were modified to a diet[h] specifically formulated for obligate carnivores such as ferrets.

By day 5, the ferret's clinical condition had improved further. Because of the ferret's mobility, continuous ECG and urine production monitoring were discontinued; however, the patient still required vasopressor support (norepinephrine, 0.4 to 0.7 µg/kg/min, IV) to maintain blood pressure within the reference range. Although the ferret seemed disinterested in food offered orally, enteral feedings through the esophagostomy tube were well tolerated, and the patient's caloric intake through the tube was increased to two-thirds of the REE. The patient began to have normal-appearing bowel movements. Bacterial culture of the abdominal fluid collected during surgery revealed growth of *Streptococcus* spp; however, antimicrobial susceptibility testing

Figure 2—Photograph of abdominal effusion obtained 4 days after exploratory laparotomy from the ferret in Figure 1. Notice the typical cloudy white appearance suggestive of chyle.

could not be performed given the fastidious nature of the isolate's growth. Because *Streptococcus* spp are usually susceptible to β-lactams, the antimicrobial spectrum was narrowed to IV administration of cefazolin (22 mg/kg, IV, q 8 h).

Over the following 48 hours (days 6 and 7), the ferret's clinical condition continued to improve. Metoclopramide, ketamine, lidocaine, and norepinephrine IV infusions were discontinued. The crystalloid and colloid fluid therapy continued, and the patient's enteral caloric intake was increased to 100% of the REE. Because the ferret's peripheral veins were affected by phlebitis at this time and because the ferret had begun to make retching and vomiting efforts, presumably as a result of the esophagostomy tube, the decision was made to induce general anesthesia (midazolam, 0.3 mg/kg, IM; butorphanol, 0.05 mg/kg [0.02 mg/lb], IM) for placement of a jugular catheter and repositioning of the esophagostomy tube. Positioning of the esophagostomy tube was confirmed radiographically. Abdominal fluid production continued to be high (up to 12.7 mL/kg/h [5.8 mL/lb/h]); therefore, octreotide[i] (10 µg/kg, SC, q 8 h), a somatostatin analog that has been used successfully in the treatment of chylothorax and chylous ascites, was initiated to decrease the production of the abdominal chylous effusion.[35,38,39] A CBC revealed a normal leukogram, but the lymphocyte count had decreased (1.3×10^9 cells/L), compared with the count on day 3, likely because of loss in the chylous effusion.

On day 8, the ferret was able to eat on its own, but was not able to meet its caloric requirement via this route. Fluid therapy consisted of infusion of crystalloids (15 to 20 mL/kg/h [6.8 to 9.1 mL/lb/h], IV) and colloids (0.4 to 0.7 mL/kg/h, IV). Analgesic and antimicrobial treatment consisted of buprenorphine and cefazolin, respectively, at the same dosages as previously described. The abdominal drain had functioned consistently until day 8, when it suddenly occluded. The occlusion was confirmed by detaching the bulb from the drain and attempting to flush the drain with sterile saline solution. This procedure was unsuccessful, and because there was evidence of continued abdominal effusion on focused assessment with sonography for trauma, the decision to place a second abdominal drain under general anesthesia was made.

Anesthesia was induced with butorphanol (0.05 mg/kg, IM), midazolam (0.3 mg/kg, IM), and propofol (4 mg/kg [1.8 mg/lb], IV). The ferret was then intubated with a cuffed endotracheal tube that had an inner diameter of 3 mm, and anesthesia was maintained with isoflurane. The abdominal drain was removed, and the entrance site of the drain sutured closed. A second abdominal drain, a 12-gauge tube,[j] was placed (Seldinger technique) to provide drainage of the chylous effusion. Abdominal fluid production (3.8 mL/kg/h [1.7 mL/lb/h]) decreased dramatically within 36 hours after octreotide administration was begun, and the effusion's appearance became less chylous. Fluid therapy was decreased and eventually discontinued, and amoxicillin-clavulanate (12.5 mg/kg [5.7 mg/lb], PO, q 12 h) replaced IV antimicrobial treatment.

From days 9 to 11, the ferret's general condition continued to improve. Its appetite was considered normal, and because the ferret was able to ingest sufficient food orally to meet its REE, the esophagostomy tube was removed. Abdominal fluid production continued to decrease; by day 11, it was 0.2 mL/kg/h, and the abdominal drain was removed. Serum biochemical analyses revealed improvement of the hypoalbuminemia (24.4 g/L) and a globulin concentration (24 g/L) within reference range. Results of hematologic evaluation revealed polychromasia and resolution of the toxic changes in the neutrophils.

The ferret remained hospitalized for an additional 24 hours for monitoring and was discharged on day 12 with instructions for the owner to administer amoxicillin-clavulanate (12.5 mg/kg, PO, q 12 h) for 7 days and octreotide (10 µg/kg, SC, q 8 h) for 3 days. Communication with the owner 2 months after surgery indicated that the ferret had continued to do well. Thirty months later, no long-term complications associated with the traumatic event had been reported by the owner.

Discussion

To the authors' knowledge, this was the first well-documented case report in the veterinary medical literature regarding the development of abdominal chylous effusion secondary to abdominal trauma and surgical intervention in a ferret. In veterinary medicine, chyloabdomen has been reported previously in various species, including dogs and cats,[9–18] horses,[19–24] bovids,[25] slender-tailed meerkats,[26] red pandas,[27] a cheetah,[28] a hedgehog,[29] and a Djungarian hamster.[k] On the authors' review of the pertinent veterinary medical literature, only 1 published report[30] of this condition in ferrets was found. Another report[31] has been documented in the gray literature, and at least 1 clinician has encountered this condition.[l]

Chylous ascites is an uncommon cause of abdominal effusion and is usually related to disruption of the lymphatic network. In humans, it may arise from congenital disorders, but in most cases is secondary to traumatic injury, obstruction, or external compression of the lymphatics, infections, hepatic cirrhosis, or inflammatory diseases.[32] In veterinary medicine, similar causes have been described, including suspected congenital defects in a calf[25] and in a foal,[19] neoplasia (the most common cause of chylous ascites in cats and the only reported cause in ferrets),[9,10,12,18,30] steatitis secondary to vitamin E deficiency in a cat,[10] feline infectious peritonitis,[15] pancreatitis in a dog and slender-tailed meerkats,[11,26] and hepatic cirrhosis.[10,28,29] Chylous ascites that developed in a horse following nephrectomy has been described,[24] but to the authors' knowledge, chylous ascites secondary to trauma has not been reported in the veterinary literature.

In human medicine, postoperative chylous ascites is a rare complication caused by incidental disrup-

tion of major retroperitoneal lymphatics such as may occur during procedures requiring extensive dissection in the retroperitoneal space or near the root of the mesentery. Typically, chylous effusions appear a few days to even a month after the surgical procedure.[32] In the ferret described in the present report, surgery was performed 2 days before the abdominal effusion acquired the appearance of chyle. The traumatic lesions in the ferret's abdomen were surgically corrected without manipulations or dissections near the major retroperitoneal lymphatic network or root of the mesentery. The abdominal wall tears secondary to trauma were located dorsally in the abdominal wall, close to the epaxial muscles and retroperitoneal space; however, these tears were small, and no other gross abnormalities were seen in the retroperitoneal spaces. The abdominal organ lesions were limited to the gastrointestinal tract, with the largest perforation present in the duodenum. Some perforations were also present in the mesentery of the jejunum, but did not compromise its vascular supply. Furthermore, no gross lesions of the vasculature or root of the mesentery were identified.

From the authors' perspectives, it was highly unlikely that iatrogenic surgical disruption of the lymphatic networks caused the chylous effusion. Similarly, other potential causes, such as congenital malformations, malignancy, or rupture of major lymphatic vessels, were also deemed unlikely because they typically require surgery to alleviate the chylous ascites.

Although the definitive cause of the ferret's chylous effusion was not identified, blunt abdominal trauma was suspected given the ferret's lack of clinical signs of disease prior to the traumatic incident and rapid improvement with medical treatment. Blunt abdominal trauma as a cause of chylous ascites is better described in the human pediatric literature[32–34] and accounts for up to 19% of such cases in literature reviews. In these children, blunt abdominal trauma is believed to cause chyloabdomen as a result of hyperextension and hyperflexion of lymphatic vessels, leading to rupture of lymphatic vessels and lymph leakage.[33]

Conservative treatment successfully resolved clinical signs of chyloabdomen after 48 hours in the ferret described in the present report. In humans, medical management is the initial therapeutic approach for chylous effusions,[32,33,35] with nutritional support being an important component to maintain the patient's nutritional status and influence chyle formation and flow through the thoracic duct. Total parenteral nutrition is generally advocated, although provision of a high-protein, low-fat diet with medium-chain triglycerides is an alternative.[33] Medium-chain triglycerides are absorbed directly by intestinal cells and then transported as glycerol and free fatty acids directly to the liver via the portal vein, unlike long-chain triglycerides, which are converted into monoglycerides and free fatty acids, and then transported as chylomicrons to the intestinal lymph ducts.[33] Such diets reduce the production and flow of chyle in humans[33] but do not seem to influence chyle production and flow in dogs.[m] In the ferret described in the present report, total parenteral nutrition was not recommended because of the need to maintain mucosal integrity of the small intestine, encourage healing by reversal of mucosal atrophy induced by starvation, and increase anastomotic collagen deposition and strength.[36] An esophagostomy tube was placed to support these nutritional goals, and enteral nutrition was implemented as soon as the patient's cardiovascular status stabilized (day 3). Initially, the ferret received a highly digestible, high-protein diet[e] (44.2% on a dry-matter basis) with a relatively high fat content (30.4% fat on a dry-matter basis) at a third of its REE. However, once the chyloabdomen was diagnosed, the ferret's diet was changed to a diet specifically formulated for obligate carnivores.[g] Unfortunately, this change did not affect the production of chyle.

To decrease the ferret's production of chyle, a synthetic long-acting somatostatin analog, octreotide, was administered. Octreotide has broad-ranging effects on the body, and its use in treating chylous effusions stems from its effects on the gastrointestinal system.[35,37] The exact mechanisms are unknown; however, the drug's multifactorial effects on the gastrointestinal system and reduction in splanchnic blood flow are thought to reduce the thoracic duct circulation and the triglyceride content of the chyle.[35,37] Octreotide has been shown in animals to shorten the time for chylous fistula closure and cessation of chylous drainage,[38] and the use of octreotide is often recommended in humans as an adjunct to other treatments for chylous effusions. Octreotide administered via continuous IV infusions and SC injections has been used successfully in humans, and the IV delivery may drastically decrease chylous fistula output after 24 to 72 hours of treatment.[39] In the ferret described in the present report, chylous effusion was successfully managed medically with octreotide, administered at a dosage previously reported in the veterinary literature,[40] combined with abdominal drainage. Neither advanced imaging nor a second surgical intervention was necessary for resolution of the chyloabdomen.

Footnotes

a. Plasma-Lyte A, Baxter Inc, Mississauga, ON, Canada.
b. Lactated Ringer solution, Baxter Inc, Mississauga, ON, Canada.
c. Life Window LW6000, DigiCare Animal Health, Boyton Beach, Fla.
d. Voluven, Fresenius Kabi Canada, Calea Ltd, Mississauga, ON, Canada.
e. Jackson-Pratt silicone flat drain, Cardinal Health Canada Inc, Pointe-Claire, QC, Canada.
f. Hill's Prescription Diet a/d, Hill's Pet Nutrition, Topeka, Kan.
g. Humulin R, Eli Lilly Canada Inc, Toronto, ON, Canada.
h. Oxbow Carnivore Care, Oxbow Animal Health, Murdoch, Neb.
i. Sandosatin 100 µg/mL, Novartis Pharmaceuticals Canada Inc, Dorval, QC, Canada.
j. Guidewire-inserted chest tube, MILA International, Erlanger, Ky.
k. Burballa A, Ramis A, Martorell J. Chyloabdomen due to chronic liver disease in a Djungarian hamster (*Phodopus sungorus*) (oral presentation). 1st Int Conf Avian Herpetol Exot Mamm Med, Wiesbaden, Germany, April 2013.
l. Huynh M, Exotics Department, Centre Hospitalier Vétérinaire Frégis, Arcueil, France: Personal communication, 2016.

m. Fossum TW. Chylothorax: pathophysiology and treatment options (abst), in *Proceedings*. 24th Annu Am Coll Vet Intern Med Forum 2006. Available at: www.vin.com/doc/?id=3856265. Accessed Apr 16, 2016.

References

1. Marini RP. Physical examination, preventive medicine, and diagnosis in the ferret. In: *Biology and diseases of the ferret*. 3rd ed. Ames, Iowa: Wiley-Blackwell, 2014;235–258.
2. Fox JG. Normal clinical and biological parameters. In: *Biology and diseases of the ferret*. 3rd ed. Ames, Iowa: Wiley-Blackwell, 2014;157–185.
3. Wagner RA. Ferret cardiology. *Vet Clin North Am Exot Anim Pract* 2009;12:115–134, vii.
4. Olin JM, Smith TJ, Talcott MR. Evaluation of noninvasive monitoring techniques in domestic ferrets (*Mustela putorius furo*). *Am J Vet Res* 1997;58:1065–1069.
5. Lichtenberger M, Ko J. Critical care monitoring. *Vet Clin North Am Exot Anim Pract* 2007;10:317–344.
6. Hein J, Spreyer F, Sauter-Louis C, et al. Reference ranges for laboratory parameters in ferrets. *Vet Rec* 2012;171:218.
7. Morissey JK, Kraus MS. Cardiovascular and other diseases. In: *Ferrets, rabbits and rodents: clinical medicine and surgery*. 3rd ed. St Louis: Elsevier Saunders, 2012;62–77.
8. Eshar D, Wyre NR, Brown DC. Urine specific gravity values in clinically healthy young pet ferrets (*Mustela furo*). *J Small Anim Pract* 2012;53:115–119.
9. Fossum TW, Hay WH, Boothe HW, et al. Chylous ascites in three dogs. *J Am Vet Med Assoc* 1992;200:70–76.
10. Gores BR, Berg J, Carpenter JL, et al. Chylous ascites in cats: nine cases (1978–1993). *J Am Vet Med Assoc* 1994;205:1161–1164.
11. Lott K, Mansfield C, Abraham LA. Acute chylous peritonitis associated with acute pancreatitis in a Staffordshire Bull Terrier. *N Z Vet J* 2015;63:125–126.
12. Myers NC III, Engler SJ, Jakowski RM. Chylothorax and chylous ascites in a dog with mediastinal lymphangiosarcoma. *J Am Anim Hosp Assoc* 1996;32:263–269.
13. Nelson KL. Chyloabdomen in a mature cat. *Can Vet J* 2001;42:381–383.
14. Peterson SL. Postcaval thrombosis and delayed shunt migration after pleuro-peritoneal venous shunting for concurrent chylothorax and chylous ascites in a dog. *Vet Surg* 1996;25:228–230.
15. Savary KC, Sellon RK, Law JM. Chylous abdominal effusion in a cat with feline infectious peritonitis. *J Am Anim Hosp Assoc* 2001;37:35–40.
16. Thompson MD, Carr AP. Hyponatremia and hyperkalemia associated with chylous pleural and peritoneal effusion in a cat. *Can Vet J* 2002;43:610–613.
17. Wright KN, Gompf RE, DeNovo RC. Peritoneal effusion in cats: 65 cases (1981–1997). *J Am Vet Med Assoc* 1999;214:375–381.
18. Fish EJ, Welles EG, Weiss RC, et al. What is your diagnosis? Abdominal fluid from a dog. *Vet Clin Pathol* 2015;44:457–458.
19. Campbell-Beggs CL, Johnson PJ, Wilson DA, et al. Chyloabdomen in a neonatal foal. *Vet Rec* 1995;137:96–98.
20. Fish EJ, Boes KM, Wilson KE, et al. Pathology in practice. Chylous ascites with secondary neutrophilic inflammation in a foal. *J Am Vet Med Assoc* 2015;246:857–859.
21. Hanselaer JR, Nyland TG. Chyloabdomen and ultrasonographic detection of an intra-abdominal abscess in a foal. *J Am Vet Med Assoc* 1983;183:1465–1467.
22. Mair TS, Lucke VM. Chyloperitoneum associated with torsion of the large colon in a horse. *Vet Rec* 1992;131:421.
23. May KA, Cheramie HS, Prater DA. Chyloperitoneum and abdominal adhesions in a miniature horse. *J Am Vet Med Assoc* 1999;215:676–678.
24. Arnold CE, Taylor T, Chaffin MK, et al. Nephrectomy via ventral median celiotomy in equids. *Vet Surg* 2013;42:275–279.
25. Cruz AM, Riley CB, MacDonald DG, et al. Use of mesenteric lymphangiography in a calf with chylothorax and chyloperitoneum. *J Am Vet Med Assoc* 1995;206:1567–1571.
26. Naples LM, Lacasse C, Landolfi JA, et al. Acute pancreatitis in slender-tailed meerkats (*Suricata suricatta*). *J Zoo Wildl Med* 2010;41:275–286.
27. Montali RJ, Roberts M, Freeman RA, et al. Pathology survey of the red panda (*Ailurus fulgens*). In: Ryder OA, Byrd ML, eds. *One medicine*. Berlin: Springer, 1984;128–140. Available at: link.springer.com/chapter/10.1007/978-3-642-61749-2_14. Accessed Sep 15, 2016.
28. Terrell SP, Fontenot DK, Miller MA, et al. Chylous ascites in a cheetah (*Acinonyx jubatus*) with venoocclusive liver disease. *J Zoo Wildl Med* 2003;34:380–384.
29. Roh Y-S, Kim E-J, Cho A, et al. Chylous ascites in a hedgehog (*Atelerix albiventris*). *J Zoo Wildl Med* 2014;45:951–954.
30. Vilalta L, Altuzarra R, Molina J, et al. Chylous ascites in 2 ferrets. *J Exot Pet Med* 2017;26:150–155.
31. Zorgniotti F. Ferret chyloabdomen—gross necrospy findings. Veterinary Information Network website. Available at: www.vin.com/Members/Boards/DiscussionViewer.aspx?documentid=3216704&ViewFirst=1. Accessed Jun 19, 2016.
32. Aalami OO, Allen DB, Organ CH. Chylous ascites: a collective review. *Surgery* 2000;128:761–778.
33. Al-Busafi SA, Ghali P, Deschênes M, et al. Chylous ascites: evaluation and management. *ISRN Hepatol* 2014;2014:240473.
34. Cochran WJ, Klish WJ, Brown MR, et al. Chylous ascites in infants and children: a case report and literature review. *J Pediatr Gastroenterol Nutr* 1985;4:668–673.
35. Ismail NA, Gordon J, Dunning J. The use of octreotide in the treatment of chylothorax following cardiothoracic surgery. *Interact Cardiovasc Thorac Surg* 2015;20:848–854.
36. Lewis SJ, Egger M, Sylvester PA, et al. Early enteral feeding versus "nil by mouth" after gastrointestinal surgery: systematic review and meta-analysis of controlled trials. *BMJ* 2001;323:773–776.
37. Stajich GV, Ashworth L. Octreotide. *Neonatal Netw* 2006;25:365–369.
38. Markham KM, Glover JL, Welsh RJ, et al. Octreotide in the treatment of thoracic duct injuries. *Am Surg* 2000;66:1165–1167.
39. Leibovitch I, Mor Y, Golomb J, et al. The diagnosis and management of postoperative chylous ascites. *J Urol* 2002;167:449–457.
40. Fossum TW. Surgery of the lower respiratory system: pleural cavity and diaphragm. In: *Small animal surgery*. 4th ed. St Louis: Elsevier Mosby, 2013;991–1032.