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

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**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. Chyloous 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 chyloous 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 chyloous 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)*
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). Moderate hypoproteinemia (48 g/L; reference range, 54.7 to 77.9 g/L) 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) 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) 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).

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. 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
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 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, 2.5 mL/kg/h [1.1 mL/lb/h], IV) and colloids (hydroxyethyl starch, 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 (0.1 to 1 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, urinary 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 X 10⁹ WBCs/L; reference range, 3.0 X 10⁹ WBCs/L to 16.7 X 10⁹ WBCs/L) due to neutropenia (0.27 X 10⁹ cells/L; reference range, 0.9 X 10⁹ cells/L to 7.4 X 10⁹ cells/L). There was a left shift (bands, 0.16 X 10⁹ cells/L; reference range, 0 to 0.1 X 10⁹ cells/L) 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 hypogobulinemia (19.1 g/L; reference range, 20 to 29 g/L), 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) combined with a urine specific gravity (1.040; reference range, 1.034 to 1.060) within reference limits suggested prerenal azotemia. Therefore, fluid therapy was altered from lactated Ringer solution to an alkalinizing solution (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, 0.5 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 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 RER, 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) 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 (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 two-thirds of the REE. The patient’s caloric intake through the tube increased to three-quarters of the REE, and the patient’s REE. A repeated CBC showed mild anemia (PCV, 33%) that was attributed to hemodilution, blood sampling, and inflammation. Neutrophil (6.39 X 10^9 cells/L), lymphocyte (3.24 X 10^9 cells/L; reference range, 0.6 X 10^9 cells/L to 10.5 X 10^9 cells/L), and WBC (10.14 X 10^9 cells/L) counts were within reference ranges; however, a left shift (bands, 0.41 X 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) 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 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](image)
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 nopep-nephrine 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 (10 µg/kg, SC, q 8 h) was used to decrease 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, chylous effusions and ascites secondary to traumatic injury or external compression of the lymphatics, infections, hepatic cirrhosis, or inflammatory diseases. In veterinary medicine, similar causes have been described, including suspected congenital defects in a calf, and a foal, neoplasia (the most common cause of chylous ascites in cats and the only reported cause in ferrets), steatitis secondary to vitamin E deficiency in a cat, feline infectious peritonitis, pancreatitis in a dog and slender-tailed meerkats, and hepatic cirrhosis. Chylous ascites that developed in a horse following nephrectomy has been described, 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-

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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. 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 and accounts for up to 19% of such cases in literature reviews. In these children, blunt abdominal trauma is believed to cause chylous abdomens as a result of hyperextension and hyperflexion of lymphatic vessels, leading to rupture of lymphatic vessels and lymph leakage.

Conservative treatment successfully resolved clinical signs of chylous abdomens after 48 hours in the ferret described in the present report. In humans, medical management is the initial therapeutic approach for chylous effusions, 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 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. 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 (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 chylous abdomen was diagnosed, the ferret’s diet was changed to a diet specifically formulated for obligate carnivores. 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. 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. Octreotide has been shown in animals to shorten the time for chylous fistula closure and cessation of chylous drainage, 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. 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, combined with abdominal drainage. Neither advanced imaging nor a second surgical intervention was necessary for resolution of the chylous abdomen.

**Footnotes**


b. Lactated Ringer solution, Baxter Inc, Mississauga, ON, Canada.

c. Life Window LW6000, DigiCare Animal Health, Boynton 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.


g. Humulin R, Eli Lilly Canada Inc, Toronto, ON, Canada.

h. Oxbow Carnivore Care, Oxbow Animal Health, Murdock, Neb.

i. Sondosatin 100 µg/mL, Novartis Pharmaceuticals Canada Inc, Dorval, QC, Canada.


l. Huynh M, Exotics Department, Centre Hospitalier Vétérinaire Frégis, Arcueil, France: Personal communication, 2016.

References


