Gastric dilatation and enterotoxemia in ten captive felids

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CASE DESCRIPTION
10 large felids at 8 facilities were determined or suspected to have developed gastric dilatation with or without enterotoxemia over a 20-year period. Four felids were found dead with no premonitory signs.

CLINICAL FINDINGS
4 felids (2 male snow leopards [*Uncia uncia*], 1 male Amur tiger [*Panthera tigris altaica*], and 1 male Sumatran tiger [*Panthera tigris sumatrae*]) were found dead or died before they could be evaluated. Six felids had hematemesis (1 male and 1 female African lion [*Panthera leo*] and 1 male jaguar [*Panthera onca*]) or abdominal distention and signs of lethargy with or without vomiting (1 male African lion, 1 male Malayan tiger [*Panthera tigris jacksoni*], and 1 female Sumatran tiger). Gastric dilatation was radiographically and surgically confirmed in the male Malayan and female Sumatran tigers and the jaguar.

TREATMENT AND OUTCOME
In 3 felids with an antemortem diagnosis, the gastric dilatation resolved with decompressive laparotomy but then recurred in 1 felid, which subsequently died. Three others died at various points during hospitalization. Although *Clostridium perfringens* type A was recovered from 3 of the 5 felids for which microbial culture was performed, and 2 felids had a recent increase in the amount fed, no single factor was definitively identified that might have incited or contributed to the gastric dilatation.

CLINICAL RELEVANCE
Gastric dilatation was a life-threatening condition in the large felids of this report, causing sudden death or clinical signs of hematemesis, abdominal distention, or vomiting. Even with rapid diagnosis and surgical decompression, the prognosis was poor. Research is needed into the factors that contribute to this emergent condition in large felids so that preventive measures might be taken. (*J Am Vet Med Assoc* 2018;253:918–925)

A 6-month-old 30-kg (66-lb) sexually intact male Malayan tiger (*Panthera tigris jacksoni*) was evaluated after sudden development of signs of depression, pyrexia (rectal temperature, 40.6°C [105°F]; reference range, 38.1° to 39.2°C [100.5° to 102.5°F]), loose feces, and a bloated appearance. The tiger had been fed a commercial horsemeat diet,[a] beef chunks,[b] and occasional whole cow's milk.

The tiger was anesthetized for radiographic evaluation with midazolam hydrochloride (0.2 mg/kg [0.09 mg/lb], IM), ketamine hydrochloride (3.3 mg/kg [1.5 mg/lb], IM), and medetomidine hydrochloride (0.04 mg/kg [0.02 mg/lb], IM), supplemented with isoflurane in oxygen delivered via an endotracheal tube. A dose of metronidazole was also administered (16.7 mg/kg [7.6 mg/lb], PO). Abdominal radiography revealed a markedly distended, fluid-filled stomach and colon with gas-dilated intestinal loops **(Figure 1)**. Attempts at orogastric intubation were unsuccessful, and the decision was made to pursue exploratory laparotomy. Given the perceived anesthetic risk to the tiger, the effects of the previously administered medetomidine were reversed early in the anesthetic period with atipamezole (0.2 mg/kg, IV). Crystalloid fluid (IV), antimicrobials (cefazolin at 22 mg/kg [10 mg/lb], IV, q 2 h), buprenorphine (0.006 mg/kg [0.003 mg/lb], IM), and meloxicam (0.1 mg/kg [0.05 mg/lb], SC) were administered during surgery.

Surgical findings included an ingesta-filled, distended stomach with no apparent foreign body or stricture, enlarged and edematous mesenteric and cecal lymph nodes, and a small region of hyperemia in the small intestine. The colon was also dilated with ingesta, and the serosa was white. Biopsy specimens were collected from the cecal lymph node prior to surgical closure of the abdomen.

During the anesthetic recovery period, the tiger failed to regain appropriate mentation and developed systemic hypertension (MAP, 175 mm Hg; reference range, 85 to 120 mm Hg). Mannitol (600 mg/kg [272 mg/lb]) was administered IV because of concerns regarding cerebral edema. After mannitol administration, audible rales, gurgles, and wheezes were detected on thoracic auscultation, and moderate hypoxemia (arterial oxygen saturation, 70% to 80%) was noted. Furosemide administration (4.4 mg/kg [2.0 mg/lb], IV, q 1 to 2 h) was initiated in

ABBREVIATIONS
MAP Mean arterial blood pressure

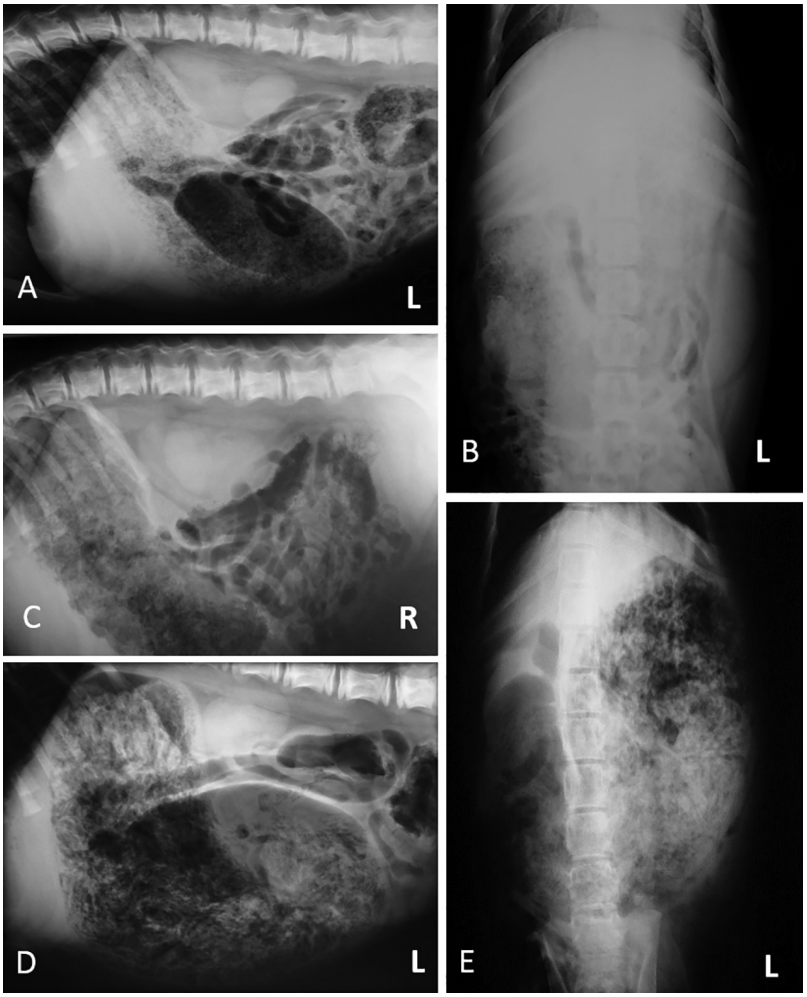

Figure 1—Lateral (A, C, and D) and ventrodorsal (B and E) abdominal radiographic views of a 6-month-old sexually intact male Malayan tiger (*Panthera tigris jacksoni*; A to C) and a 6-month-old sexually intact female Sumatran tiger (*Panthera tigris sumatrae*; D and E) with gastric dilatation. Notice that the stomach appears markedly distended with ingesta and gas in all images. L = Left. R = Right.

response to suspected acute respiratory distress syndrome, and a nasal line was placed to provide high-flow oxygen administration. At that point, the tiger also received maropitant (1 mg/kg [0.45 mg/lb], SC), penicillin benzathine (20,000 U/kg [9,091 U/lb], SC), famotidine (0.5 mg/kg [0.23 mg/lb], SC), ranitidine (0.5 mg/kg, SC, q 12 h), metoclopramide (0.2 mg/kg, SC, q 4 h), and metronidazole (15 mg/kg [6.8 mg/lb], IV, q 12 h), and administration of fluids, cefazolin, meloxicam, and buprenorphine was continued as during surgery. Midazolam (0.2 mg/kg, SC, IV, or IM) was administered as needed to ensure safety of attending personnel. A CBC and serum biochemical analysis performed at the time of surgery revealed a mild increase in serum alkaline phosphatase activity (108 U/L) and band neutrophilia (5%), with an unremarkable leukocyte count.[1]

The tiger was managed intensively over the next 72 hours. Twenty-four hours into care, a bronchodilator (terbutaline at 0.005 mg/kg [0.002 mg/lb], SC, q 8 h) and promotility agent (erythromycin at 0.8 mg/kg [0.36 mg/lb], IV, q 8 h) were added to treatment, and metoclopramide administration was initiated as a constant rate infusion. Enemas were administered, and nasoesophageal-tube feeding was initiated. The tiger remained pyrexic (rectal temperature, 39.4°C [103°F]), tachycardic, and tachypneic and required frequent sedation to manage signs of agitation that led to hypoxemia and exhaustion. By 60 hours into intensive care, normoxemia had been restored on a lower nasal oxygen flow rate, but the tiger remained pyrexic. Over the subsequent 8 hours, cognitive function began to improve (ie, the tiger attempted to stand and began vocalizing and biting at a towel) and remained normoxemic on the low nasal oxygen flow rate. Rectal temperature decreased to a range of 38.6° to 38.8°C (101.5° to 101.9°F). A CBC revealed leukopenia (4,900 WBCs/µL) with band neutrophilia (4%).[1] Results of serologic viral testing (FeLV, FIV, feline coronavirus, feline panleukopenia, and feline parvovirus) were negative.

The tiger appeared to gradually regain strength and had an increased frequency of its typical mentation status and behavior. Supplemental oxygen administration was halted, and the tiger remained normoxemic and appeared capable of drinking and eating. After typical behavior had been observed for several hours, the tiger was returned to holding facilities (72 hours after initial evaluation) and continued to receive the antimicrobial, gastrointestinal protectant, and promotility agent. A CBC at that point revealed leukocytosis (20,300 WBCs/µL) and normocytic, nonregenerative anemia (Hct, 35.7%).[1] Results of microbial culture of feces obtained via enema 3 days previously indicated moderate growth of *Clostridium perfringens* type A.

Histologic examination of the lymph node biopsy specimen revealed severe, subacute, pyogranulomatous lymphadenitis. Results of Fite acid-fast, Gram, and Gomori methenamine–silver nitrate staining were negative for bacterial organisms and fungal elements; Warthin-Starry staining for spirochetes also yielded negative results. Gimenez staining revealed minute cytoplasmic granules in a few histiocytic cells.

The tiger recovered completely with no lasting adverse effects on its health or behavior and was discharged from the hospital with prescriptions for metronidazole, cisapride (0.56 mg/kg, [0.25 mg/lb], PO, q 8 h), famotidine, amoxicillin–clavulanic acid

(25 mg/kg [11.36 mg/lb], PO, q 12 h), and metoclopramide. All medications were gradually discontinued, and oral cisapride administration was continued for 12 months. Four years after initial evaluation, the tiger remained healthy.

Five months after the aforementioned tiger was initially evaluated, routine fecal testing of felids housed at the same facility revealed positive results for a previously undescribed, novel *Clostridium* sp. This organism was isolated from the Malayan tiger and its male Sumatran tiger (*Panthera tigris sumatrae*) conspecific as well as from clouded leopards (*Neofelis nebulosa*) but not from a clouded leopard cub or a young, female Sumatran tiger that was being hand reared.

Three years previous to this incident, and at the same facility, a 6-month-old sexually intact female Sumatran tiger had been evaluated because of a sudden onset of signs of lethargy, depression, mild ataxia, and inappetence. At that time, it had been fed a commercial carnivore diet,[c] beef chunks, and bones once weekly. Physical examination with the tiger anesthetized revealed pyrexia (38.7°C [101.6°F]) and abdominal distention. High serum alanine aminotransferase activity (318 U/L), mild hypokalemia (serum potassium concentration, 3.4 mmol/L), and mild anisocytosis were identified on hematologic testing.[1] Radiography revealed a markedly distended stomach filled with ingesta (Figure 1).

Endoscopy was performed in an attempt to decompress the stomach but was unsuccessful, so gastrotomy was performed, during which several bone fragments were identified near the pylorus. Supportive care during surgery included IV fluid administration, antimicrobials, and analgesics. Toward the end of the procedure, the tiger became markedly hypertensive (MAP range, 111 to 237 mm Hg) and developed cardiac arrest. Cardiac resuscitation with intracardiac administration of epinephrine (0.05 mg/kg [0.02 mg/lb], IV) and doxapram hydrochloride (5 mg/kg [2.4 mg/lb], IV) was successful.

The tiger failed to recover well from anesthesia and became progressively pyrexic (rectal temperature, 40.2°C [104.3°F]) and tachypneic. Treatment for presumptive cerebral edema was instituted with mannitol (1 g/kg, IV) and furosemide (0.5 mg/kg, IV). After 3 mannitol and furosemide treatments were administered, the tiger began to have paddling behavior and had signs of noise sensitivity. Phenobarbital (3 mg/kg [1.4 mg/lb], IV, q 12 h) and midazolam (dose range, 0.1 to 0.2 mg/kg, q 2 to 4 h) were administered as needed to maintain mild sedation. The tiger remained under intensive care for 48 hours, after which it began to have signs of awareness but was unable to use its hind limbs and did not appear to be visually competent but was responsive to light. At 72 hours after cardiac resuscitation, the tiger began to eat and drink on its own when given food and water. Daily improvement in mentation status and gait was observed, and 2 weeks after the initial incident, the tiger was reintroduced to its family group (dam and male sibling) without complication.

Eight months later, this tiger developed a second episode of gastric distension that required surgical intervention. At that time, the tiger was being fed a commercial beef diet[d] and meat chunks. Following surgery, the juvenile developed cardiac arrest and, despite resuscitation attempts, died. Microbial culture of a gastric tissue sample yielded *C perfringens* type A. Pooled tissue samples (stomach and intestine) were submitted for PCR assay for *Clostridium* spp.[e] Results were negative for *Clostridium difficile* (toxins A and B) and positive for *C perfringens* genotype A. Gross necropsy revealed moderately enlarged mesenteric lymph nodes, mildly enlarged bronchiolar lymph nodes, pulmonary congestion and hemorrhage at the base of the trachea, dark red mucosa in the distal portion of the jejunum, and dilated and enlarged hepatic sinusoids. Histologic examination of various tissues revealed findings consistent with acute systemic shock (stomach or intestinal origin), periportal hemorrhage supportive of ascending enterotoxemia, and excessive adipose tissue in the gastric wall with fibrous serosal adhesion.

A 9-year-old sexually intact male jaguar (*Panthera onca*) housed at a zoological facility was evaluated because of sudden-onset hematemesis and signs of lethargy and weakness. Its diet varied according to a rotating schedule and included raw poultry, ground and solid horsemeat, bovine bones, and other items. Gastroscopy, performed while the jaguar was anesthetized, revealed multiple blood clots within the cardia of the stomach. Following gastroscopy, the stomach could not be decompressed, and MAP (measured indirectly via a forelimb) declined sharply, from 110 to 42 mm Hg. With the jaguar still in left lateral recumbency, a 14-gauge, 5-inch catheter was placed to decompress the stomach. The MAP immediately improved to 68 mm Hg and then to the prebloat value. Gastric dilatation-volvulus was confirmed by radiography. Surgical correction of the volvulus was initiated, during which the jaguar received corticosteroid drugs, antimicrobials, and crystalloid fluid IV and iron dextran IM. Approximately 400 mL of hemorrhagic fluid was observed within the abdominal cavity. The stomach was successfully derotated, and gastrotomy was performed to evaluate the gastric mucosa, which appeared edematous but otherwise unremarkable. Belt-loop gastropexy was performed to minimize the risk of recurrence, and the stomach and abdomen were routinely closed.

Postoperative care included amoxicillin (10.6 mg/kg [4.8 mg/lb], q 12 h) and a gastroprotectant (sucralfate at 17.7 mg/kg [8.0 mg/lb], PO, q 8 h) and 72 hours of food withholding. The diet was subsequently restricted to ground chicken with a total volume of < 0.45 kg (1 lb)/d for 5 to 7 days, with a gradual increase to the regularly fed volume. No gristle or bones were fed for 2 months after surgery. Results of histologic examination of a gastric biopsy specimen were unre-

markable, with no evidence of underlying infectious or inflammatory process. This jaguar died of unrelated causes 10 years after initial evaluation with no additional episodes of gastric dilatation reported.

Several other captive felids from various institutions were also identified as having gastric dilatation with or without enterotoxemia from 1994 through 2014, and none survived. A 10.5-year-old sexually intact male Sumatran tiger from a second facility was found dead after having had no prior clinical signs. When the tiger was 6 years old, *Salmonella* organisms were recovered from a fecal sample; however, all isolates were non–host adapted and not believed to reflect a carrier state. At 7 years of age, this tiger had been evaluated for weight loss, poor hair coat, and intermittent loose feces containing maldigested food. A CBC and serum biochemical analysis had revealed hypoalbuminemia (serum albumin concentration, 3.0 g/dL), with leukocytosis (17,800 WBCs/μL) and hyperglobulinemia (serum globulin concentration, 4.2 g/dL).[1] Histologic examination of colonic and gastric biopsy specimens had revealed severe lymphoplasmacytic-neutrophilic gastritis of the pyloric and fundic mucosa with moderate lymphoplasmacytic colitis. Treatment had been initiated with prednisone, enrofloxacin, and metronidazole, and chlorambucil was added 60 days later owing to incomplete remission of the disease. Prednisone and metronidazole doses were tapered and adjusted throughout the tiger's life in response to its apparent appetite, fecal quality, and behavior. Several dietary changes (lamb, beef, chicken, and turkey) were also made in an attempt to manage clinical signs. Seven months into treatment for inflammatory bowel disease, *Salmonella* organisms were recovered from a fecal sample, but no antimicrobial treatment was initiated because of the history of positive culture results with no clinical signs.

After 1 year of medical and dietary management for inflammatory bowel disease, remission was achieved and all medications were discontinued. One episode of gastroenteritis had been noticed 9 months after treatment discontinuation, but this had resolved within 24 hours without treatment. One month prior to death (3 years after apparent remission from inflammatory bowel disease), the tiger had reportedly been healthy on its current diet without treatment, with an increasing number of soft-feces incidents. Oral lipase powder administration had been instituted, and fecal consistency improved. One week after lipase powder administration was discontinued, the tiger was found dead in its enclosure. Necropsy revealed 180° gastric volvulus and dilatation with subcutaneous emphysema. Histologic examination of various tissues revealed findings consistent with acute shock, reactive lymphoid hyperplasia (mesenteric lymph nodes), and moderate nephrosclerosis.

A 10-year-old sexually intact male Amur tiger (*Panthera tigris altaica*) was found dead in right lateral recumbency with severe bloating and in rigor mortis; no clinical signs had been observed previously. Two small fecal boluses with frank blood and mucus were noticed on the floor where it was kept, and blood-tinged vomitus was visible on the ventral abdominal region. Microbial culture of dietary carcass meat, gastric tissue, and feces yielded various organisms, most notably *C perfringens* type A (all specimens), *Clostridium bifermentans* (all specimens), and *Clostridium sordelli* (carcass meat and feces). Necropsy revealed gastric rupture, and histologic examination of various tissues revealed findings consistent with acute fibrinous peritonitis and endocarditis, with a deposition disorder of the muscular tunics of the blood vessels of the stomach and gastric vessels.

An 11-year-old sexually intact male African lion (*Panthera leo*) at a wildlife park died following acute hematemesis. This lion had been hand raised and had arrived at a private facility at 2 months of age with loose feces and flea bite dermatitis that resolved with medical management. Clinical history was unremarkable except for a single episode of vomiting froth and blood at 5 years of age, which resolved without treatment. Gross necropsy revealed hemorrhagic gastritis; gastric torsion, dilatation, and rupture; and mucosal pallor. Histologic examination of gastric tissues revealed marked congestion and hemorrhage of the lamina propria and submucosa with extensive necrosis of submucosal glands.

An 11-month-old sexually intact female African lion at a private institution was evaluated because of hematemesis of unknown duration. Survey radiographs were nondiagnostic, and exploratory laparotomy was performed. Surgical exploration revealed hemoabdomen that appeared to have originated from a severely necrotic stomach. Euthanasia was elected owing to the poor prognosis. Gastric torsion and dilatation that had resolved spontaneously was suspected given the clinician's experience with this disorder in large dogs and findings of gastric necrosis on gross necropsy. No microbial culture or histologic examination was performed.

A 5-year-old sexually intact male African lion at a zoologic facility was evaluated for vomiting bile and progressive signs of lethargy over a 36-hour period. The lion had been fed a commercial horsemeat diet[a] (4.5 kg [10 lb]/d). Endoscopy revealed a large, spherical object consistent with a bezoar in the lumen of the stomach. Exploratory laparotomy was performed, during which 850 mL of serosanguineous fluid was removed from the peritoneal cavity by suction. Multiple regions of serosal hemorrhage and hyperemia (each 7 to 10 cm long) were identified intermittently along the jejunum. Gastrotomy was performed to remove the 10-cm trichobezoar. During the procedure, the lion developed respiratory arrest and then cardiac arrest and, despite resuscitation efforts, died. Microbial culture of jejunal fluid yielded mixed anaerobic bacteria (normal flora; identities not reported); *Candida krusei* was recovered from abdominal fluid. Gross necropsy revealed segmental jejunal hemorrhage and

the gastric trichobezoar. Histologic examination of gastrointestinal tissues revealed transmural congestion, hemorrhage, edema, thrombosis, and mixed inflammation of the intestine and pylorus, consistent with a partial gastrointestinal torsion. Mild interstitial pneumonia was attributed to sepsis from the gastrointestinal lesion. Results of cytologic assessment of the ascitic fluid indicated low cellularity with atypical mastocytosis and intralesional yeast organisms identified as *C krusei*. Two years after the lion's death, a *Clostridium* toxin panel was performed on a frozen liver sample, revealing negative results for *C perfringens* antigen and for α toxin, β toxin, β2 toxin, cytotoxin netF, and type A enterotoxin (CPE).

A 3.5-year-old sexually intact male snow leopard (*Uncia uncia*) was found dead at a zoological facility 48 hours following a routine preshipment examination facilitated by anesthesia. Six days per week, it was fed a commercial horsemeat diet[f] (1.4 kg [3 lb/d]; recently increased by 20% in response to lean body condition score), and an oxtail was fed the remaining day of the week. Gross necropsy revealed a 180° gastric torsion along the long axis with a rupture near the esophageal sphincter, with hemorrhage and edema of the gastric tear, emphysema of the thoracic diaphragm and esophagus, hemorrhagic and necrotic gastric mucosa, free blood in the abdominal cavity, hemorrhagic and necrotic colonic mucosa, and peritonitis. Histologic examination of gastrointestinal tissues revealed multiple hemorrhages within the lamina propria, marked edema and fibrin separating the villi, necrosis of the duodenal villi, fibrin indicative of disseminated intravascular coagulation, and characteristics of edematous and fibrinous enteropathy, with intralesional bacteria in the jejunum.

A 15.5-year-old sexually intact male snow leopard housed at the same institution as the other snow leopard was also found dead within 48 hours after the other. Dietary conditions were similar to those of the other snow leopard. Health history was generally unremarkable, except for intermittent signs of partial anorexia. Microbial culture of feces yielded *Plesiomonas* spp and of jejunal tissue yielded various organisms. Gross necropsy revealed a 270° gastric torsion along the long axis, with a rupture along the esophageal sphincter and segmental intestinal serosal erythema. Histologic examination of gastrointestinal tissues revealed dilated gastric serosal lymphatics with necrosis or autolysis of the mucosa.

Discussion

To the authors' knowledge, the present report represents the first of gastric dilatation with or without volvulus in large felids. Overall, 6 species of large felids (3 African lions, 2 snow leopards, 2 Sumatran tigers, 1 Amur tiger, 1 Malayan tiger, and 1 jaguar) were represented. Mean age was 6 years (median, 5 years). Most felids (8/10) were male. Four felids (2 snow leopards and 2 tigers) were found dead or died before they could be evaluated. For the 3 felids in which gastric dilatation was diagnosed before death, clinical signs at initial evaluation included abdominal distention, hematemesis, lethargy, vomiting, or a combination of these signs.

The 2 tigers that survived (1 of which later died) had signs of abdominal pain, and abdominal distention was confirmed radiographically as gastric dilatation. Both were 6 months old at initial evaluation and were housed at the same facility, although the gastric dilatation occurred 2 to 3 years apart. Passage of an orogastric tube was unsuccessful in both tigers, requiring surgical decompression. Both developed life-threatening complications following surgery. One had recurrent gastric dilatation at 1 year of age and died of associated complications. That felid (a Sumatran tiger) had excessive adipose tissue in the gastric mucosa, and this, combined with serosal fibrosis, was postulated to have contributed to delayed gastric emptying and enterotoxemia. The other felid (a Malayan tiger) remained alive at the time of final follow-up (3 years later), with no repeated episodes of dilatation. The jaguar that survived gastric dilatation developed minor complications during gastrotomy (hypotension and hyperkalemia) but did not develop the systemic signs associated with enterotoxemia that were observed in the tigers.

Three African lions had histologic or gross evidence of recent gastric dilatation and torsion that had resolved spontaneously. Two of these lions had hematemesis, and the third was vomiting bile. For the remaining felids, clear gross or surgical evidence of gastric dilatation or torsion was evident. The reason the African lions appeared prone to spontaneous resolution of gastric dilatation or torsion and not the other felids was unclear. Two of the 3 African lions had hematemesis prior to death, and of the other included felids, only the jaguar also had hematemesis.

In large-breed dogs, gastric dilatation with and without torsion is common and has been well researched. Several risk factors have been proposed, including impaired gastric motility, inflammatory bowel disease, consumption of large volumes of food, feeding once daily, underweight body condition, rapid ingestion, eating from a platform, male sex, and advanced age.[2,3] Results of other studies[4–8] implicate genetics, oil and fat content of dry food, deep-chested conformation, and temperament (easily stressed). In dogs with severe gastric distention, the hepatic portal vein and posterior vena cava may become obstructed, leading to a decrease in cardiac output, shock, disseminated intravascular coagulation, and mesenteric congestion.[2] However, despite decades of research, the cause of this disorder in large-breed dogs remains poorly understood. Other species in which gastric dilatation can occur with and without torsion include both captive and free-ranging wildlife, including polar bears,[9] fur seals,[10] red wolves,[11] echidnas,[12] rabbits,[13] guinea pigs,[14,15] red pandas,[16] sloths,[17] black-footed ferrets,[18] and various nonhuman primate species.[19,20]

In 4 felids of the present report (1 lion, 1 tiger, and 2 snow leopards), gastric rupture was diagnosed

on necropsy. Spontaneous gastrointestinal rupture has been reported to occur in domestic cats and in 1 study[21] was associated with lymphoma, inflammatory bowel disease, and necrotic suppurative enteritis. Other than the 1-year-old female Sumatran tiger with no gastric rupture, most felids of the present report had no histologic lesions that might predispose them to gastric rupture, making idiopathic gastric dilatation and prolonged volvulus the most probable cause of rupture.

Few reports[22–26] exist of enterotoxemia in large felids, and in these species, enterotoxemia may be a cause or consequence of gastric dilatation with or without volvulus. In particular, *C perfringens* has been hypothesized to contribute to the development of gastric dilatation in simian species and ferrets and has been recovered from the gastric contents of dogs with gastric dilatation.[18,19,27] Despite this, *C perfringens*, when inoculated into healthy primates, is incapable of triggering gastric dilatation.[19] Clostridial enterotoxemia without gastric dilatation has been identified in Amur leopards (*Panthera pardus orientalis*), cheetahs (*Acinonyx jubatus*), Bengal tigers (*Panthera tigris tigris*), African lions, and Amur tigers. One affected tiger reportedly developed neurotoxicosis from *Clostridium* toxins,[23] whereas the remainder developed gastrointestinal signs or were found dead.[22,24–26]

Clostridium spp are gram-positive, anaerobic, spore-forming bacteria that have been associated with disease in both wildlife and humans. *Clostridium perfringens* types A, B, C, and D are considered important pathogens of domestic animals.[28] This bacterium can be found in the feces, soil, and gastrointestinal tract of humans and other animals.[28] In domestic species, dietary and environmental changes can predispose animals to the development of clostridial disease.[28] However, positive results of microbial culture do not always reflect the pathogenicity of the organism.

The possibility existed that the Malayan tiger and Sumatran tiger from the same facility in the present report had acquired a novel pathogen. A novel *Clostridium* sp was identified in felids housed at the same institution shortly after the Sumatran tiger recovered, the importance of which was unclear. This novel organism had not been recovered at the time of initial evaluation, and no other felids have developed gastric dilatation at this institution. Additionally, this facility has since had 2 litters of Sumatran tigers with no reported incidents of gastrointestinal disease. The enterotoxemia in the 2 affected tigers was likely secondary to the α toxin or an as-yet unidentified toxin and may have been compounded by subclinical illness in these tigers.

Clostridium perfringens type A is a widespread pathogen that is known to cause enterotoxemia in lambs, goats, and calves.[29] It is has been attributed with causing enterotoxemia in horses, mink, pigs, camels, and water buffalo; necrotic enteritis in chickens; and hemorrhagic gastroenteritis in dogs.[29] Additionally, as observed in black-footed ferrets, *C perfringens* type A can cause gastric dilatation and death in overfed kits.[18] This organism has also been associated with the death of an Amur tiger and lion at 1 facility.[24] *Clostridium perfringens* type A produces α toxin and occasionally *C perfringens* enterotoxin, which is known for its enterotoxigenic properties.[29] The Malayan tiger in the present report was tested for this enterotoxin, and results were negative; the assay was not performed on samples from the other included felids. *Clostridium perfringens* type A was recovered from 3 of 4 tigers and was identified in 3 of 5 felids for which cultures were performed.

In a case report[22] of fatal clostridial enterotoxemia in 2 Amur leopards, it was postulated that incomplete thawing of meat may have contributed to the development of the infection. The USDA recommends that meat fed to captive exotic animals be used within 24 hours after thawing.[30] In such circumstances, meat should be thawed under refrigeration, and the temperature of the meat should not exceed 7.2°C (45°F) at any time.[30] Unconsumed food should be removed no later than 12 hours after feeding. Meat for use in zoological institutions may arrive with heavy microbial concentrations, with 60% of products tested in 1 study[31] identified as containing *Salmonella* spp and all samples as containing *Listeria monocytogenes*. This is not surprising given that clostridial contamination remains a concern in the human food industry as well. In a historic study, *C perfringens* was cultured from 43.1% of samples of meat meant for human consumption; and more recently, *Clostridium* organisms were identified in 100% of ground turkey and 60% of ground beef samples.[32,33] Commercial canine and feline raw diets are also susceptible to contamination with *Clostridium* spp, found in 20% of samples in 1 study.[34] Although culture results were unknown for many felids of the present report, clostridial enterotoxemia remains an important differential diagnosis for felids with acute gastrointestinal signs.

For the jaguar of the present report, chronic gastritis was believed to have incited the gastric torsion. In addition to this jaguar, other medium-sized felids at this facility were noted as having an increase in frequency of vomiting following the opening of a newly completed facility where bromination was used to maintain water quality in exhibit water features (as separate from water sources intended for potable use). This jaguar had clinical signs consistent with chronic gastritis for 2 years after the new facility opened, prior to development of gastric dilatation and torsion. Subsequently, it was determined that the bromination system was oversized for the volume of water requiring disinfection, resulting in residual bromine in exhibit water features. Bromine toxicosis reportedly causes respiratory, gastrointestinal, and neurologic dysfunction in animals and humans.[35–39] After bromine use was discontinued, the frequency of vomition among felids housed in this exhibit returned to usual.

In 2 felids of the present report, the presence of a gastric foreign body could have predisposed them to

the development of gastric dilatation. Gastric foreign bodies have been associated with gastric dilatation-volvulus in dogs,[40] and triggering of chronic vomiting and gastritis is a possible explanation for this association.[41] Gastric foreign bodies may cause an outflow obstruction, leading to delayed gastric emptying and chronic gastritis.[41] In the African lion with the gastric trichobezoar, the trichobezoar may have caused an intermittent outflow obstruction leading to gastrointestinal displacement that resolved prior to surgical intervention. For the Sumatran tiger at the first facility in the present report, the bone fragments found near the pyloric outflow may have had a similar effect.

The 2 snow leopards in the present report were from the same institution, and both had received a 20% increase in the amount of horsemeat fed in response to a lean body condition score. In large-breed dogs, food composition and volume are reported risk factors for gastric dilatation-volvulus,[5,6] and these factors may have contributed to the gastric dilatation in the snow leopards as well.

No pattern was identified in the cases of gastric dilatation with or without volvulus described in the present report that might have suggested a particular risk factor for the condition. Possible risk factors include chronic gastritis, presence of a gastric foreign body, delayed gastrointestinal motility, bacterial overgrowth of dietary items, and diet composition, including recent increases in dietary volume. Several felids had no data available regarding dietary history or microbial culture results, and this limited our ability to identify any dietary or microbial factors that might have been associated with gastric dilatation. When performed, microbial culture frequently yielded *C perfringens* type A, and it remains unclear whether this organism could have caused or contributed to gastric dilatation in the large felids. Although more males than females were affected, no conclusions can be drawn regarding whether male sex is a risk factor for gastric dilatation, as has been reported for domestic dogs.[7] In most large felids that survived the episode of gastric dilatation with or without volvulus, aggressive surgical and postsurgical management was necessary to counteract the impact of enterotoxemia. Even when early intervention was provided, gastric dilatation appeared to be an important concern with a poor prognosis. Research into the cause or causes of this disorder in large felids is recommended.

Acknowledgments

The authors thank Marla Waddell for record assistance, Julie Lemon for technical support, and Telena Welsh, Paul Povey, Christy Webster, and Steven Ok for caring for hospitalized tigers.

Footnotes

a. Nebraska horsemeat diet, Central Nebraska Packaging Inc, North Platte, Neb.
b. Nebraska brand, Central Nebraska Packaging Inc, North Platte, Neb.
c. Nebraska premium carnivore diet (beef), Central Nebraska Packaging Inc, North Platte, Neb.
d. Natural Balance (beef), Natural Balance Pet Foods Inc, Burbank, Calif.
e. Department of Medicine and Epidemiology, University of California-Davis, Davis, Calif.
f. 10% horsemeat diet, Dallas Crown Inc, Kaufman, Tex.

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