ILEOCECOCOLIC STRICTURES IN TWO CAPTIVE CHEETAHS (ACINONYX JUBATUS JUBATUS)

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ILEOCECOCOLIC STRICTURES IN TWO CAPTIVE CHEETAHS
(ACINONYX JUBATUS JUBATUS)


Abstract: Intestinal strictures were diagnosed in two captive cheetahs (Acinonyx jubatus jubatus). The cheetahs presented with lethargy, anorexia, diarrhea, and weight loss. The first cheetah had a stricture of the ileoceccocolic junction diagnosed at necropsy. The second had an ileoceccocolic stricture causing obstruction that was diagnosed at surgery. After resection and anastomosis, the cheetah recovered well. The etiology of the strictures remains undetermined. Intestinal stricture, particularly of the ileoceccocolic junction, should be considered as a differential diagnosis for cheetahs with nonspecific gastrointestinal signs.

Key words: Acinonyx jubatus jubatus, cheetah, ileoceccocolic junction, intestine, stricture.

INTRODUCTION
Captive cheetahs frequently show evidence of gastrointestinal disease. The two cheetahs described in this case report were evaluated for nonspecific gastrointestinal signs. Both animals were found to have strictures at the ileoceccocolic junction, and the cause of the strictures could not be determined. Currently, it is uncertain whether ileoceccocolic stricture is unique to one environment or if it may affect the wider captive cheetah population.

CASE REPORT
Case 1
A 15-yr-old female, 31.5-kg cheetah was examined for chronic intermittent soft stools and 1 wk of decreased diet intake. The animal was anesthetized for physical examination, which revealed thin body condition and generalized muscle wasting. Radiographs of the thorax and abdomen were unremarkable except for a small hepatic silhouette. On endoscopy, the distal esophageal and gastric mucosa had a glistening edematous appearance and no visible rugal folds. Multiple endoscopic biopsies were collected, but the samples were nondiagnostic. Compared to the International Species Information System (ISIS) reference ranges for cheetahs, the complete blood count (CBC) showed leukocytosis (23.3 × 10^9/L; reference range 10.4 ± 3.5 × 10^9/L) with a neutrophilia (20.5 × 10^9/L; reference range 7.0 ± 2.7 × 10^9/L), immature neutrophils (1.1 × 10^9/L; reference range 0.4 ± 0.8 × 10^9/L), and lymphopenia (0.9 × 10^9/L; reference range 2.0 ± 1.0 × 10^9/L). No significant changes were seen in the serum chemistry panel. Serology for feline immunodeficiency virus (FIV) via kinetic enzyme-linked immunosorbent assay (ELISA) and Western blot and feline leukemia virus (FeLV) via ELISA was negative. Serology for feline immunodeficiency virus (FIV) via kinetic enzyme-linked immunosorbent assay (ELISA) and Western blot and feline leukemia virus (FeLV) via ELISA was negative (Cornell University, Animal Health Diagnostic Center, Ithaca, New York 14853, USA). Fecal cytology showed a diverse bacterial population with occasional spiral bacteria. A fecal culture grew Escherichia coli and Proteus sp. The animal received amoxicillin (Penn Labs, Inc., Philadelphia, Pennsylvania 19102, USA; 15.9 mg/kg by mouth [p.o.], twice a day [b.i.d.] for 21 days) and pyrantel pamoate (Equi-Phar, Phoenix Scientific, Inc., St. Joseph, Missouri 64503, USA; 7.5 mg/kg p.o. for one dose).

Over the next 3.5 wk, the cheetah consumed approximately half of its regular diet in small meals. Metronidazole (Pliva, Inc., East Hanover, New Jersey 07936, USA; 7.9 mg/kg p.o., b.i.d. for 10 days) and bismuth subsalicylate (Pepto-Bismol, Procter & Gamble, Cincinnati, Ohio 45202, USA; 8.3 mg/kg p.o., b.i.d.) were prescribed. Six weeks after the diagnostic exam, body condition had improved, although intermittent diarrhea continued. No parasites or abnormal cell populations were found on fecal flotation or cytology, respectively. Repeated treatment with amoxicillin, followed by treatment with trimethoprim–sulfamethaxazole (Schering-Plough Co., Kenilworth, New Jersey 07033, USA; 30.5 mg/kg p.o., s.i.d. for 10 days) produced no clinical improvement. The animal became lethargic, its condition continued to decline, and it was humanely euthanized.

At postmortem examination, the most significant lesion was a constrictive band (1.5 cm thick) at the ileoceccocolic junction, resulting in marked disten-
A stricture (s) characterized by a thickened and ulcerated ring, incorporating the cecal opening (arrow), has caused marked dilatation of the distal ileum (i) (11-cm circumference), with severe partial obstruction of the colon (c) (4-cm circumference). Bar = 1 cm.

**Figure 1.** Ileocecocolic junction, cheetah, case 1. A stricture (s) characterized by a thickened and ulcerated ring, incorporating the cecal opening (arrow), has caused marked dilatation of the distal ileum (i) (11-cm circumference), with severe partial obstruction of the colon (c) (4-cm circumference). Bar = 1 cm.

**Figure 2.** Ileum, cheetah, case 1. Photomicrograph illustrates the abrupt margin of the ulcer (arrow), with villous retention on the right. Hematoxylin and eosin (H&E); bar = 250 μm.

**Figure 3.** Ileum, cheetah, case 1. Photomicrograph shows the pattern of granulation tissue deep to the ileal ulcer. The submucosa is markedly thickened; vessels are arranged perpendicular to the lumen (arrows), with fibroblasts parallel to the surface. Hematoxylin and eosin (H&E); bar = 250 μm.

The mucosa at the ileoceccolic junction was ulcerated and the surface was irregularly proliferative. Histologically, the mucosal surface at the constriction was ulcerated (Fig. 2) and the wall thickness was doubled with superficial granulation tissue (vessels arranged perpendicular to the luminal surface with intervening fibroblasts parallel to the surface) (Fig. 3) and neutrophils. No intraluminal organisms were identified on Gram, Ziehl–Neelsen, Warthin–Starry, or Gomori’s methenamine silver–stained sections. The other main finding in the gastrointestinal tract was low numbers of spiral bacteria in the gastric glands in association with mild lymphoplasmacytic deposits in the lamina propria.

**Case 2**

A 6.5-yr-old, 39-kg female cheetah had a 1-mo history of intermittent poor appetite, loose stool, hematochezia, lethargy, and weight loss. The animal was anesthetized for physical examination, radiographs, and an electrocardiogram, all of which were within expected limits. Endoscopic examination revealed normal esophageal, gastric, and colonic mucosal surfaces; biopsies were not collected. A CBC and serum chemistry panel were unremarkable. Serology for FIV and FeLV was negative. No parasites were seen on fecal flotation or direct examination. A fecal culture produced a heavy growth of *E. coli*, and metronidazole (12.8 mg/kg p.o., b.i.d. for 14 days) was prescribed.

Over the next 2 wk, the animal did not eat well, and its fecal consistency varied from liquid to semi-formed. Prednisone (West-ward Pharmaceutical Corp., Eatontown, New Jersey 07724, USA; 1 mg/kg p.o., s.i.d. for 5 days, followed by 0.5 mg/kg p.o., s.i.d. for 5 days) was administered, and one dose of ivermectin (ProMectin, Phoenix Scientific; 0.3 mg/kg, administered intramuscularly [i.m.] via dart) was given. The following week the cheetah was anorexic, and retching and vomiting were observed. The animal was anesthetized for examination and had lost 5.4 kg in the 3 wk since its initial evaluation. Physical examination revealed severe dehydration and a markedly distended intestinal loop in the caudal abdomen. A CBC showed leukocytosis (31.9 × 10⁹/L) with absolute neutrophilia (29.0 × 10⁹/L), relative lymphopenia (1.9 × 10⁹/L), and hemocoagulation (hematocrit [Hct] 0.53, reference...
Trophilia (22.2 /H11003/H11006/range 0.38

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Leukocytosis (33.6 /H11003/H11006) and reticulocytosis were present. Chemistry abnormalities included elevated amylase (4,068 U/L; reference range 1,308 ± 330 U/L), hyperbilirubinemia (97 µmol/L; reference range 5 ± 3µmol/L), hypophosphatemia (0.9 mmol/L; reference range 1.9 ± 0.6 mmol/L), and hypoalbuminemia (25 g/L; reference range 36 ± 4 g/L).

A contrast study of the upper gastrointestinal tract with barium sulfate (Lafayette Pharmaceuticals, Inc., Kansas City, Missouri 64137, USA; 150 ml p.o. via stomach tube) showed peristaltic contractions of the stomach and small intestine without distension or obstruction. The study was discontinued before the contrast medium reached the large intestine because of concerns about anesthetic time and hypothermia; however, the gas seen on radiographs was concluded to be colonic. A dose of long-acting ceftriaxone crystalline-free acid (Excede, Pfizer Animal Health, Exton, Pennsylvania 19341, USA; 7.1 mg/kg, administered subcutaneously) and metronidazole (Abbott Laboratories, North Chicago, Illinois 60064, USA; 14.9 mg/kg, administered intravenously [i.v.]) were administered. Midazolam (Abbott Laboratories; 0.12 mg/kg i.m.) and cyproheptadine (Merck & Co., Whitehouse Station, New Jersey 08889, USA; 1.2 mg/kg p.o.) were given as appetite stimulants, and famotidine (Pepcid, Merck & Co., Inc.; 0.59 mg/kg i.v.) and sucralfate (Aventis Pharmaceutical, Inc., Kansas City, Missouri 64137, USA; 59 mg/kg p.o.) were given as gastric protectants. The cheetah was fed by stomach tube while under sedation (Carnivore Care, Oxbow, Murdock, Nebraska 68407, USA; and canned cat food). The cheetah started to eat small meals the following afternoon, and its appetite and activity continued to improve over the next 36 hr. Treatment continued with sucralfate (59 mg/kg p.o. for 5 days), enrofloxacin (6 mg/kg p.o., i.d. for 10 days), and metronidazole (14.7 mg/kg p.o., b.i.d. for 10 days). Treatment was discontinued 17 days postsurgery, when the animal was alert, active, and eating well.

DISCUSSION

Intestinal obstruction has been described in association with extraluminal compression, mural thickening, or intraluminal obstruction, and there are many etiologies for each of these categories. In the cheetahs described, there was no evidence of neoplasia, foreign body, abscess, polyp, or endoparasitism, which commonly cause intestinal ob-
Histopathologic examination did not reveal an inciting cause for the constriction and inflammation seen in both cheetahs; however, infectious, inflammatory, neoplastic, and idiopathic causes were considered. With respect to viral infections, feline infectious peritonitis (FIP), caused by a feline coronavivirus, often manifests as a nonspecific multisystem disease with atrophy of the intestinal tissue. Occasionally solitary mural masses at the ileocecal junction or in the colon have been described as pyogranulomas in FIP; but this lesion was not present in the cheetahs.

Bacterial agents leading to intestinal lesions were considered. *Anaerobiospirillum* spp. are found in healthy cats but may be a cause of feline ileocolitis. Histologically, *Anaerobiospirillum* infections result in spiral bacteria within the intestinal crypt, leading to crypt abscesses; no spiral bacteria were seen in the cheetah intestinal samples. The presence of a few gastric spiral bacteria with mild lymphoplasmacytic deposits in the lamina propria in case 1 was not thought to be significant to the development of the stricture. Although *E. coli* was cultured from feces from both cheetahs and from the ileoceccolic junction of the cheetah in case 2, bacteria were not seen in the intestinal wall sections.

Proliferative enteropathy, caused by *Lawsonia intracellularis*, is a recognized disease in pigs and hamsters, and similar proliferative enteric disease has been identified in multiple species, including raptors, nonhuman primates, and horses. Hamsters may present with ileocolic junction stenosis and fatal obstruction. The proliferative lesions seen in the cheetahs consisted of chronic inflammation, with granulation tissue as the major component, while glandular proliferation is characteristic of *Lawsonia* sp. infections. Another differential considered was intestinal thickening due to mycobacteriosis, which is recognized in a number of species. *Mycobacterium avium* complex have been reported to cause thickened intestinal loops in domestic cats. Neither granulomas nor significant histiocytic inflammation were seen in the cheetahs, and acid-fast bacilli were not identified with Ziehl–Neelsen stain.

The cheetah tissues were evaluated for mycotic agents that may cause intestinal thickening. Histoplasmosis causes granulomatous respiratory lesions or disseminated disease, although dogs with histoplasmosis are more likely than cats to have gastrointestinal tract involvement, with signs of diarrhea and wasting. Intestinal histoplasmosis results in thickened intestinal walls, with areas of necrosis and granulomatous inflammation. *Pythium insidiosum* also causes intestinal thickening resulting in anorexia, weight loss, vomiting, and diarrhea. Pythiosis can produce intestinal masses in domestic cats. No intraluminal fungal organisms were seen with special stains in the cheetah samples.

Infiltrates are seen in the lamina propria and submucosa in inflammatory bowel disease (IBD), an idiopathic condition in the domestic cat, causing vomiting, weight loss, and diarrhea. One report describes a stricture of the small intestines and partial obstruction due to IBD. The characteristic histologic findings of feline IBD are lymphoplasmacytic enteritis and colitis. Lymphoplasmacytic deposits were not a significant component of the lesions seen in the cheetahs. There are two presentations of idiopathic inflammatory bowel disease in humans: Crohn’s disease and ulcerative colitis. Crohn’s disease affects the distal small intestine and colon, specifically targeting the crypts, transmural inflammation, and mucosal ulceration; and stricture formation is a common feature. Ulcerative colitis extends cranially from the rectum. Regional enterocolitis, a rare syndrome in domestic dogs, produces transmural granulomatous inflammation that can cause complete luminal obstruction. The pattern and distribution of the histologic lesions in the cheetahs were distinct from these IBDs. Intestinal neoplasia, common in felids, may manifest as mural infiltration. Alimentary lymphosarcoma may present as discrete masses, while intestinal adenocarcinoma may result in segmental thickening to constriction. The pleomorphic cell population at the ileoceccolic junctions of the cheetahs was not consistent with diagnoses of neoplasia.

Clinical signs for the most frequent cheetah diseases are often vague and nonspecific. The cheetahs in this case report had signs similar to those with gastritis, veno-occlusive disease, or enteritis and colitis. However, none of these diseases were present in the described cases, except for mild gastric inflammation in association with spiral bacteria in case 1. The impetus to present this case information was to highlight the potential for clinical signs commonly recognized in cheetah disorders to lead to an unexpected diagnosis. Stricture at the ileoceccolic junction was not initially considered in either of these cases. The findings of this study emphasize the need to conduct a thorough investigation of the gastrointestinal tract in cheetahs with nonspecific presenting signs to assess the possibil-
ity of intestinal stricture, particularly at the ileocecal junction. The precise cause of the strictures was not determined, despite evaluation for infectious and neoplastic etiologies.

LITERATURE CITED

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