

# Partial gastrointestinal obstruction for one month due to a linear foreign body in a cat

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A six-year-old male castrated domestic shorthair cat was presented to the referring veterinarian with a one week history of listlessness, anorexia, and intermittent vomiting. All vaccinations were up to date, including feline leukemia virus (FeLV), and another cat resident in the same household was healthy. Diet consisted of canned cat food and chicken livers. On presentation, the cat was afebrile and mildly dehydrated. Abdominal palpation was unremarkable. A complete blood count (CBC), serum biochemical analysis, and urinalysis were normal. An ELISA test for FeLV was negative. Abdominal radiographs revealed loss of abdominal contrast, but abdominocentesis failed to yield peritoneal fluid. Subcutaneous fluids were administered and the cat was discharged with a seven day course of amoxicillin/clavulanate at 13.7 mg/kg PO q12h (Clavamox, Beecham Laboratories, Bristol, Tennessee, USA). Transient improvement was seen, but vomiting and anorexia resumed after the antibiotic therapy was discontinued. Treatment with amoxicillin/clavulanate was repeated on the same regimen for 14 days. Intermittent vomiting continued and watery brown diarrhea developed. Results of a second serum biochemical analysis showed hypoalbuminemia (21 g/L, normal 26–40 g/L). A five day course of prednisone at 1.0 mg/kg PO q12h (Apo-Prednisone, Apotex Inc., Weston, Ontario) was instituted in addition to the antibiotics. Because of continued deterioration, the cat was referred to the Veterinary Teaching Hospital at the Western College of Veterinary Medicine.

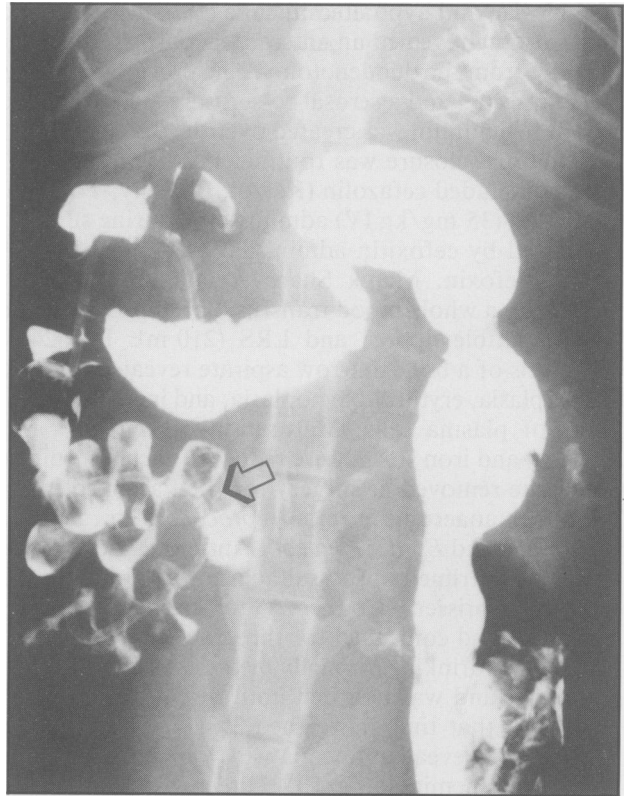
On presentation, the cat was afebrile, mildly dehydrated, and weighed 3.47 kg. Mucous membranes were pale and abdominal palpation revealed thickened bowel loops. Results of a CBC revealed neutrophilia ( $26.18 \times 10^9/L$ ) with a regenerative left shift ( $1.49 \times 10^9$  bands/L). The packed cell volume (PCV) was 0.32 L/L (normal 0.24–0.45 L/L). The serum biochemical analysis showed hypoproteinemia (47 g/L, normal 68–80 g/L) and hypoalbuminemia (19 g/L, normal 22–38 g/L). Results of urinalysis were normal and an ELISA test for FeLV was negative. Abdominal radiographs showed a linear foreign body within a fluid-filled, distended, and bunched segment of small intestine, and contrast radiographs confirmed partial obstruction due to the foreign body (Figure 1).

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**Figure 1.** Ventrodorsal abdominal projection made two hours after giving barium shows part of the small intestine arranged in a distinctive serpentine pattern along the foreign body (arrow).

Initial treatment consisted of intravenous lactated Ringer's solution (LRS) (200 mL q12h). Whole blood (60 mL) from a compatible donor was administered to correct the hypoproteinemia. A laparotomy was performed two days after presentation. At that time, the foreign body was palpated within the intestine, and it extended from the stomach to the cecum. No areas of perforation or compromised vascular supply were observed and, therefore, intestinal resection was not necessary. A 30 cm black elastic cord, similar to those attached to catnip-filled toys, was removed via distal duodenotomy, and a 3 cm diameter red plastic ring attached to a short strand of cord was removed via gastrotomy. Abdominal lavage was performed with warmed LRS and all incisions were closed routinely. The cat recovered from anesthesia without complications.

Postoperatively, the cat was depressed and anorexic although the abdomen remained nonpainful, the incision clean, and the temperature normal. Results of a CBC revealed nonregenerative anemia (PCV = 0.19 L/L) and hypoproteinemia (5.2 g/dL). Hydration was initially maintained by administration of LRS (100 mL SC q8h). Intravenous infusion of LRS (210 mL q24h), supplemented with potassium chloride (70 mEq/L), was established three days postopera-

tively to correct hypokalemia (3.1 mmol/L, normal 4.0–5.8 mmol/L) detected on a subsequent serum biochemical profile. The cat was force-fed baby food. On the seventh postoperative day, purulent material was expressed from the incision. Results of a CBC showed neutrophilia ( $17.59 \times 10^9/L$ ) with a significant left shift ( $3.31 \times 10^9$  bands/L), worsening anemia (PCV = 0.14 L/L), and hypoalbuminemia (18 g/L). A second laparotomy revealed an enterocutaneous fistula originating from the duodenotomy. The previous incisions were resected and a serosal-to-serosal patch using two loops of jejunum was created over the duodenotomy. Abdominal closure was routine. Perioperative treatments included cefazolin (Kefzol, Eli Lilly, Toronto, Ontario) (35 mg/kg IV) administered during surgery, followed by cefoxitin administered at 35 mg/kg IM q6h (Mefoxin, Merck Sharp & Dohme, Kirkland, Quebec), a whole blood transfusion (60 mL IV) from a compatible donor, and LRS (210 mL IV q24h). Analysis of a bone marrow aspirate revealed myeloid hyperplasia, erythroid hypoplasia, and increased numbers of plasma cells. Maturation of cell lines was normal and iron stores were reduced. Bacterial culture of tissue removed at surgery revealed a mixed infection with anaerobic cocci, *Streptococcus* sp., *Proteus mirabilis*, and *Escherichia coli*. Antibiotic therapy was changed to trimethoprim-sulfadiazine at 35 mg/kg PO q12h (Tribrissen, Coopers Agropharm Inc, Ajax, Ontario) and continued for three weeks. The cat was eating and drinking normally by the second postoperative day, and was boarded until discharge nine days later. At that time it weighed 3.61 kg. Results of a CBC still revealed anemia (PCV = 0.18 L/L) and hypoproteinemia (55 g/L). Three weeks after discharge, the cat was doing well and the anemia had improved (PCV = 0.28 L/L). Reassessment of the initial radiographs taken by the referring veterinarian confirmed the presence of the linear foreign body nearly one month prior to surgical removal of the cord.

The duration of obstruction due to a linear foreign body, measured from the onset of clinical signs to diagnosis, has been reported to range from 1–10 days (1,2). In this cat, vomiting started five weeks prior to surgery. Radiographs demonstrated a minimum four week duration of partial obstruction due to the linear foreign body. The chronicity of linear foreign body obstruction in this cat is unusual, but does illustrate that ingestion of a linear foreign body can result in partial obstruction for longer than previously believed. Linear foreign body obstruction should be considered a differential diagnosis for chronic gastrointestinal disease in cats. The location of the foreign body, type of material, and degree of intestinal hyperperistalsis were probably all factors involved in preventing complete obstruction, perforation, or peritonitis during the extended period of partial obstruction.

A diagnosis of linear foreign body obstruction can be made based on history, oral examination, abdominal palpation, and survey radiographs (1,3,4). Survey abdominal radiographs also can be normal, especially early in the course of disease (4). Contrast radiographs can be used to differentiate gas patterns associated with a linear foreign body from those associated with non-specific enteritis (3,4).

Linear foreign bodies are treated conservatively or surgically. Conservative management resulted in passage of the material in one to three days in nine of 24 cats, five of which had sublingual fixation of the foreign body (2). Surgery is indicated if the foreign body remains stationary, or if perforation and subsequent peritonitis are suggested by clinical, radiographic, and/or hematological findings (5). Surgery was indicated in this cat because of the chronicity of partial obstruction.

Hypoproteinemia in this cat was attributed to degradation of protein and reduced nitrogen absorption resulting from bacterial overgrowth in stagnant loops of small intestine (6). Extravasation of plasma proteins from mucosal vasculature and/or interstitial spaces, due to mucosal damage by the foreign body, may have contributed to the hypoproteinemia (6). Significant protein malassimilation rarely occurs in chronic small intestinal obstruction (6). Hypoproteinemia could have contributed to delayed healing of the enterotomy incisions and increased susceptibility to infection.

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#### **Linear foreign body obstruction can result in chronic, intermittent, gastrointestinal disease in cats**

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Perioperative antibiotic therapy is indicated in patients with small intestinal obstruction to reduce bacterial overgrowth, prevent peritonitis secondary to peritoneal contamination which may occur during intestinal surgery, and prevent absorption of endotoxins and migration of bacteria through the mucosal barrier into the systemic circulation (7,8). Antibiotics for prophylaxis in intestinal surgery are usually administered intravenously during induction of anesthesia and continued no further than 6–24 hours postoperatively (7). Perioperative antibiotics in this cat may have prevented the complications that resulted in a second laparotomy. The *E. coli* and *Streptococcus* sp. cultured from the enterocutaneous fistula were normal intestinal flora; *Proteus mirabilis* could have been a hospital contaminant.

Anemia of chronic inflammatory disease was considered due to the normal red cell indices. However, reduced marrow iron stores were more consistent with iron deficiency anemia (9). In this cat, chronic blood loss through damaged intestinal mucosa, or reduced iron intake due to anorexia, could have resulted in the low iron stores. The anemia improved with resolution of the gastrointestinal inflammation and return of the cat's appetite.

Linear foreign body obstruction can result in chronic, intermittent, gastrointestinal disease in cats. Careful examination of the mouth (including the base of the tongue), complete abdominal palpation, and attention to radiographic patterns consistent with linear foreign body obstruction are important in every cat with gastrointestinal disease so as not to miss this diagnosis.

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## BOOK REVIEW

## REVUE DE LIVRE

Jordan FTW, ed., **Poultry Diseases 3rd edition**. Philadelphia. Ballière Tindall (W.B. Saunders Company, Toronto), 1990. 497 pp. \$49.95

As stated by the Editor, the 3<sup>rd</sup> edition of *Poultry Diseases* is designed for veterinary students and graduate veterinarians studying avian medicine or working with the poultry industry. It would also be a useful reference for practitioners serving backyard, small farm flock, or fancy-fowl clients.

The text provides a concise description of the important poultry disease problems, although, as with many multiple author texts, a few of the experts have included details of interest only to specialists making some parts unnecessarily complicated. The book has a distinctly British flavor since all but four of the 28 authors are from the UK.

Most diseases are described by cause, epidemiology, diagnosis and control. The first chapter describes the European poultry industry with emphasis on the situation in the UK. Chapters 2-13 cover bacterial diseases and infections, including avian mycoplasma and chlamydia. The chapter on the Enterobacteriaceae is much the longest, but only the section on *S. arizona* is excessive. Colibacillosis, the most serious bacterial infection in the industry in Canada, is the only area that is inadequate. Except for *Pasteurella* infection in turkeys and staphylococcal osteomyelitis, other bacterial infections are up-to-date and very well described.

Chapters 14-20 cover viral diseases. The four chapters on the virus-induced lymphoproliferative diseases are very good. Hemorrhagic enteritis in turkeys is described in the chapter on Adenoviruses but is indexed only under "turkey hemorrhagic enteritis". The suggestion that the adenovirus of inclusion body hepatitis causes infectious anemia is wrong. This anemia is likely caused by the chicken anemia agent. The chapter on infectious bursal disease does not discuss the most significant problem of infection in baby chicks causing bursal destruction without causing clinical disease. The viruses that may be associated

with enteritis and infectious stunting in poults and chicks are covered under Reoviridae and Astroviruses, but "stunting syndrome" in chickens is in Miscellaneous Conditions (Chapter 38). Chapters on chicken anemia agent and (pneumoviral) rhinotracheitis of turkeys are included.

Chapters 31 and 32 discuss fungal and parasitic diseases.

Chapters 33 to 38 cover the musculoskeletal, urinary and cardiovascular systems, as well as nutritional and toxic problems and miscellaneous conditions. Many obscure lesions of doubtful clinical significance are described in the musculoskeletal chapter. The artificial division into axial and appendicular skeleton adds to the confusion. The description of renal disease is excellent, but it is not clear why the author dismisses the possibility of primary urate nephrosis from water deprivation as a cause of renal failure. Uric acid is extremely cytotoxic and high levels in the kidney cause tubular degeneration followed by hyperuricemia and visceral urate deposition. Sodium toxicity can occur without significant renal pathology.

Descriptions of sudden death in 6-16 week old male turkeys, flip-over (sudden death syndrome) in broiler chickens, and ascites caused by right heart failure from pulmonary hypertension can be found in the chapter on cardiovascular disease.

The final seven chapters cover a variety of topics in epidemiology and disease prevention. They include hygiene and disinfection, vaccines and vaccination, medication and welfare. The chapter on how to carry out a field investigation is very good. The last chapter is an appendix of psychologic, production and other data.

This is an up-to-date, well organized, easy to read poultry disease text. It includes many of the emerging problems in the poultry industry.

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