Treatment of pyloric stenosis in a cat via pylorectomy and gastroduodenostomy (Billroth I procedure)

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Case Description—A 5-month-old 1.9-kg (4.2-lb) spayed female Siamese cat was referred to the Mississippi State University Animal Health Center for evaluation because of a history of decreased appetite, regurgitation, vomiting, and lack of weight gain. The clinical signs were first observed after an ovariohysterectomy performed by the referring veterinarian 22 days earlier. Regurgitation occurred after feeding, and the cat was vomiting approximately 3 times daily. Dietary changes were unsuccessful in resolving the decreased appetite. At the time of evaluation at the referral hospital, the owners had been spoon-feeding the cat 1 tablespoon of a canned high-calorie diet 2 every 2 to 4 hours. Examination revealed that the cat was approximately 5% dehydrated and lethargic, with a body condition score of 4 (scale, 1 to 9). The abdomen was tense when palpated. A CBC, serum biochemical analysis, urinalysis, fecal flotation, and FIV, FeLV, and feline heartworm antigen tests were performed, and survey abdominal radiographs were obtained.

Clinical Findings—Radiographic findings included a fluid- and gas-distended stomach with a small accumulation of mineral opacities. Ultrasoundographic examination confirmed severe fluid distention of the stomach with multiple hyperechoic structures present and revealed protrusion of the thickened pylorus into the gastric lumen, with normal pylorogastric serosal continuity. Endoscopy of the upper gastrointestinal tract revealed an abnormally shortened pyloric antrum and stenotic pyloric outflow orifice. Pyloric stenosis resulting in pyloric outflow obstruction was diagnosed.

Treatment and Outcome—A pylorectomy with end-to-end gastroduodenostomy (Billroth I procedure) was successfully performed, and a temporary gastrostomy tube was placed. Six days after surgery, the cat was eating and drinking normally, with the tube only used for administration of medications. The gastrostomy tube was removed 12 days after surgery. Results of follow-up examination by the referring veterinarian 3 weeks after surgery were normal. Occasional vomiting approximately 2 months after surgery was managed medically. Fifteen months after surgery, the owners reported that the cat seemed completely normal in appearance and behavior.

Clinical Relevance—Pyloric stenosis should be considered a differential diagnosis for young cats with pyloric outflow obstruction. The cat of this report was treated successfully with a Billroth I procedure. Histologic examination and immunohistochemical analysis of the excised tissue showed the stenosis to be associated with hypertrophy of the tunica muscularis. (J Am Vet Med Assoc 2013;242:792–797)
A minimal amount of anechoic free fluid was found within the abdomen. Ultrasonographic diagnoses included pyloric outflow obstruction with abnormal pyloric wall thickness and mild left renal pyelectasia.

The cat was anesthetized for endoscopic evaluation of the upper gastrointestinal tract. Following premedication with dexmedetomidine (5 µg/kg [2.3 µg/lb]) and butorphanol tartrate (0.4 mg/kg [0.18 mg/lb]), IM, an over-the-needle catheter was inserted in a cephalic vein, and anesthesia was induced via IV administration of propofol (2.5 mg/kg [1.1 mg/lb]). The cat was intubated, and anesthesia was maintained with isoflurane in oxygen. A flexible gastrointestinal videoscope with an insertion tube (outer diameter, 8.6 mm; working length, 1,030 mm) was used to evaluate the upper gastrointestinal tract. Results of esophageal evaluation were unremarkable. The stomach was dilated with liquid and ingesta. The pyloric antrum was abnormally shortened, and the pylorus was very difficult to identify; careful probing with a blunt-tipped forceps revealed the pyloric outflow orifice. The diameter of the pyloric antrum was estimated to measure 2 mm. These findings confirmed stenosis of the pyloric outflow tract.

On the basis of these results, a pylorectomy and gastroduodenostomy (Billroth I) procedure was recommended to the owner. The owner elected to pursue surgery, which was performed under the same anesthetic episode. Cefazolin (22 mg/kg [10.0 mg/lb]) was admin-

Figure 1—Survey abdominal radiographs (ventrodorsal [A], right lateral [B], and left lateral [C] views) of a 5-month-old spayed female Siamese cat evaluated because of intermittent vomiting, regurgitation, decreased appetite, and lack of weight gain. A—The stomach (arrowheads) is severely distended with fluid and gas, and other abdominal organs are displaced caudally. B—With the patient in right lateral recumbency, the distended pylorus (arrowheads) can be seen as a soft tissue opacity. C—With the patient in left lateral recumbency, the gas-filled lumen of the stomach (arrowheads) is evident, and a small accumulation of faint, pinpoint mineral opacities is present within the pylorus (circle). This finding, known as a gravel sign, is supportive of pyloric outflow obstruction.11

Figure 2—Ultrasonographic image of the stomach and pylorus of the same cat as in Figure 1, with the patient in dorsal recumbency (left side of the image is cranial). The pylorus is seen protruding into the gastric lumen, mimicking the appearance of an intussusception. The pyloric wall is thickened, and severe gastric distention with echogenic fluid and small hyperechoic structures are present.
istered IV. Exploratory surgery via a ventral midline celiotomy revealed palpable thickening of the pyloric region of the stomach. All other organs were grossly normal. Stay sutures (4-0 polydioxanone) were placed in the stomach and duodenum adjacent to the proposed pylorectomy site. These sutures were used to manipulate the stomach and duodenum throughout the procedure. Margins of resection for the pylorectomy were visualized 1 to 2 cm oral and aboral of the pylorus. Branches of the right gastric and gastroepiploic arteries and veins supplying the pylorus were identified, isolated, and cauterized by means of bipolar electrocautery. The cauterized vessels were then sharply divided. During dissection, care was taken to avoid the common bile duct and disruption of the blood supply to adjacent structures. Doyen forceps were placed oral and aboral to the planned area of resection. A straight Kelly forceps was then placed opposite each Doyen forceps across the area to be resected. The pylorus was transected oral and aboral to the Kelly forceps with a No. 15 scalpel blade. Starting from the lesser curvature, the pyloric antrum was closed in a simple interrupted pattern with 4-0 polydioxanone suture, leaving a stoma with approximately the same diameter as the duodenum. The duodenum and pyloric antrum were anastomosed with 4-0 polydioxanone suture in a simple interrupted pattern.

A purse string suture of 3-0 polydioxanone was then placed in the gastric body, and a full-thickness stab incision was made within the purse string. A 16F, 39-cm-long silicone gastrostomy tube was introduced into the gastric lumen, and the purse string suture was tied. A stab incision was made in the left body wall, just caudal to the last rib, and the gastrostomy tube was passed through this stab incision site. Gastropexy to the left abdominal wall was performed with 3-0 polydioxanone in an interlocking box pattern. Nylon suture (3-0) was used to secure the tube to the skin at the exit site in a fingertrap pattern. Lavage of the surgical region and peritoneal cavity was performed with sterile saline (0.9% NaCl) solution, and the abdomen was closed routinely. Recovery from anesthesia was uneventful.

Immediate postoperative care included IV fluid therapy with lactated Ringer’s solution (3 mL/kg/h [1.4 mL/lb/h]) and a constant rate infusion of fentanyl (5 µg/kg/h, IV) for analgesia. Maropitant citrate (1.0 mg/kg [4.5 mg/lb], q 24 h) and famotidine (0.1 mg/kg [0.05 mg/lb], q 12 h) were administered SC. Pyrantel pamoate (10 mg/kg [4.5 mg/lb]) was administered via the gastrostomy tube for treatment of the Toxocara spp infection. Gastric contents were aspirated via the gastrostomy tube, and the tube was flushed with 5 mL of warm water every 4 hours. The fentanyl infusion was incrementally decreased and was discontinued 24 hours after surgery. Beginning at that time, buprenorphine (0.01 mg/kg [0.005 mg/lb], IV) was administered every 8 hours. Sucralfate suspension (200 mg; 105.3 mg/kg [47.86 mg/lb]) was administered every 8 hours via the gastrostomy tube as prophylaxis against gastrointestinal ulceration, and feeding was reintroduced, starting with one-fourth of a can of a high-calorie moist diet every 6 hours via the gastrostomy tube. On the second day following surgery, the maropitant citrate was discontinued because of the cat’s excellent appetite and lack of vomiting.

Six days after admission, the cat was discharged to the owner. At that time, the cat was eating and drinking normally and the gastrostomy tube was only being used for administration of medications. The owner was instructed to administer famotidine (0.5 mg/kg [0.23 mg/lb], q 12 h) and sucralfate (200 mg, q 8 h) through the gastrostomy tube and buprenorphine (0.01 mg/kg, PO, q 8 h) and to return the cat for examination in 1 week. One day after discharge, the cat was reevaluated at the veterinary medical teaching hospital because it had disrupted the sutures securing the gastrostomy tube to the skin. These sutures were replaced, and at the owner’s request, the cat was readmitted to the hospital. All medications were continued at the same dosages. The cat was fed the same high-calorie canned diet (one-fourth of a can, q 6 h) and remained hospitalized for 5 days, at which time the gastrostomy tube was removed. Upon discharge, the owner was instructed to continue famotidine administration (0.5 mg/kg, PO, q 12 h) and to have a follow-up evaluation by the referring veterinarian in 5 to 7 days for assessment of the gastrostomy stoma and again in 3 weeks for a second treatment for Toxocara spp infection.

Histologic evaluation of the resected tissue revealed that the wall of the pylorus was abruptly and irregularly thickened by haphazardly arranged bundles of smooth muscle with some subdividing bands of fibrous connective tissue (Figure 3). Scattered lymphocytic aggregates, usually associated with blood vessels, were interspersed among these tissues. The submucosa also contained fibrovascular tissue with numerous profiles of arteries and veins, interspersed with mesenchymal cells, and minimal mononuclear infiltrates. Many of these mesenchymal cells tested positive for smooth muscle actin via immunohistochemical analysis, indicating smooth muscle myofiber or myofibroblast origin (Figure 4). A focus of mucosal ulceration, covered by a coagulum of fibrin, necrotic cellular debris, and small
numbers of degenerate leukocytes, was detected. Histologic and immunohistochemical findings were consistent with a diagnosis of hypertrophy of the tunica muscularis of the surgically excised pyloric tissue.

Results of a follow-up physical examination by the referring veterinarian 3 weeks after surgery were normal. The owner reported no vomiting at that time, and the cat’s appetite was good. Approximately 2 months after surgery, the owner reported occasional vomiting. Famotidine treatment was reinstituted at the described dose; because of problems with twice-daily pill administration, famotidine was discontinued, and treatment was initiated with an omeprazole suspension (0.6 mg/kg [0.27 mg/lb], PO, q 24 h). Approximately 3 months after surgery, the omeprazole was changed to every-other-day dosing, to be continued indefinitely. Through continued telephone communication up to 15 months following surgery, the owners reported that the cat seemed completely normal both in appearance and behavior.

Discussion

Pyloric stenosis of young cats has been reported rarely. In 1970, Twaddle reported pyloric stenosis in 3 cats that were between 11 and 14 months of age at the time of initial evaluation; 2 of these were sexually intact female Siamese, and 1 was a neutered male domestic shorthair. Two additional sexually intact female Siamese cats with the condition were reported by Twaddle in 1971; these kittens were 6 months old at the time of evaluation and were offspring of one of the female Siamese cats treated previously. The clinical signs described in those reports included persistent vomiting, a good appetite despite vomiting, thin body condition, and regurgitation of any solid food. Radiographic examinations of the upper gastrointestinal tract following oral administration of barium sulfate contrast medium were performed in all of those patients. A tentative diagnosis of pyloric stenosis was made on the basis of prolonged gastric emptying times and clinical history. All 5 cats were treated via Heineke-Mikulicz pyloroplasty. After surgery, one of the older cats vomited occasionally, with complete resolution after 1 month. In 2 of the younger cats, vomiting was seen 2 to 3 times daily during the first 10 days but became progressively less frequent, with complete resolution 2 months after surgery. Follow-up time was variable, but was as long as 1 year, and all cats were reported to be clinically normal when the last update was obtained.

In 1974, Pearson et al described 13 cats with delayed gastric emptying. All cats were ≤ 2 years of age, and 11 of 13 were of the Siamese breed. Nine of these 13 cats also had some degree of esophageal dilatation or dysfunction, determined on the basis of esophagoscopy results. All of these cats were treated via a Fredet-Ramstedt pyloromyotomy. Five cats were clinically normal following recovery from surgery, and 5 had intermittent vomiting or regurgitation; 3 had no improvement and died or were euthanized because of their clinical signs.

Overall, the previous reports of pyloric stenosis in cats treated by means of pyloroplasty or pyloromyotomy revealed favorable outcomes in many cases, with resolution or improvement of clinical signs. Because of the young ages of cats in reported cases and apparent heritability of the condition, a congenital etiology has been assumed. However, determining a specific etiology is difficult because no histopathologic findings have previously been described for patients with this condition.

Pyloric stenosis also occurs in dogs and is also known as antral pyloric hypertrophy syndrome or chronic hypertrophic pyloric gastropathy. The congenital form of the disease is less common and is characterized by selective hypertrophy of the muscularis of the pylorus; this most commonly develops in Boxers and Boston Terriers. The acquired form of the disease is more common in male dogs and small breeds such as Lhasa Apso, Shih Tzu, Pekingese, and Miniature Poodle. With this form of the disease, mucosal hypertrophy is present, with or without hypertrophy of the muscularis. In humans, pyloric stenosis is a frequently encountered condition of newborns and infants. It is characterized by hypertrophy of the pyloric musculature and typically treated via pyloromyotomy.

In the cat described in the present report, a presumptive diagnosis of congenital pyloric outflow obstruction was made on the basis of the clinical history of intermittent vomiting and anorexia and progressive weight loss, along with radiographic and ultrasonographic evidence of gastric distension. Diagnostic imaging of the abdomen facilitates the diagnosis of mechanical pyloric outflow obstruction. Abdominal radiographs reveal marked gastric distention with fluid and gas, as in the patient of this report. Ultrasonography is used in the evaluation of gastric wall thickness and the presence of intramural or extramural lesions, which may contribute to decreased gastric outflow.

The normal ultrasonographic appearance of the feline gastrointestinal tract has been described. The pylorus lies slightly more on the midline in cats than in dogs, with gastric wall thickness measuring between 1.1 and 3.6 mm. Measurements may be greater if obtained when
the stomach is empty and may also vary depending on whether the measurement is made in the region of a rugal fold or interrugal area. To the authors’ knowledge, the ultrasonographic appearance of pyloric stenosis in cats has not been described. In dogs, congenital pyloric hypertrophy can be visualized ultrasonographically as a localized, circumferential thickening of the pylorus. The thickened pylorus can protrude into the gastric lumen and mimic the appearance of an intussusception. In 1 study,13 gastric wall thickness was >9 mm in all dogs evaluated. In the cat of the present report, the pylorus was moderately thickened, measuring approximately 6 mm, with a multi-layered puckered pattern protruding into the gastric lumen. Gastric body wall thickness measured up to 3 mm, but the lumen was severely distended with fluid and ingesta. Upon further ultrasonographic evaluation, the serosal margins of the wall of the pylorus were identified and found to be contiguous with the gastric wall, eliminating the suspicion of intussusception. Although not pathognomonic for obstruction or hypertrophy in animals, food and fluid were not observed to pass through the lumen of the pylorus during ultrasonography, supporting decreased peristalsis. In a previous report11 that described ultrasonographic examination of 6 dogs with pyloric hypertrophy, 5 were noted to have no movement of gastric contents through the pylorus despite peristaltic activity. Additionally, a fluid- or food-distended stomach can aid in determining delayed gastric emptying, especially if the time of previous feeding is known.11 In children, diagnosis of pyloric stenosis is usually made ultrasonographically, and for experienced evaluators, accuracy of diagnosis approaches 100%.7 In these cases, the lesion can be seen as a thickened prepyloric antrum bridging the distended stomach and duodenal ampulla. Results of upper gastrointestinal endoscopy were definitive for stenosis in the cat of this report and prompted surgery.

To the best of the authors’ knowledge, this is the first report of a cat with histologic findings of smooth muscle hypertrophy and fibrosis supporting the clinical diagnosis of pyloric stenosis. This is most analogous to the congenital form of pyloric stenosis found in young Boxers and Boston Terriers and in human infants.5,7

In addition to the previously mentioned treatment options of Heineke-Mikulicz pyloroplasty and Fredet-Ramstedt pyloromyotomy described for cats with pyloric stenosis,2–4 reported treatments for dogs with pyloric stenosis have included Y-to-U pyloroplasty and pyloromyotomy.3 With pyloromyotomy, resected tissue can be submitted to help provide a definitive diagnosis. Pylorotomy has been used for primary treatment and as a revision surgery for the treatment of this condition in dogs. In 1 report,12 pylorotomy was advocated for as the best initial treatment for dogs with pyloric stenosis involving either large amounts of redundant mucosa or thickened pyloric musculature. Treatment of pyloric stenosis with a Billroth I procedure has not been previously reported in cats. In this case, it is the authors’ opinion that the stenosis was so severe that pyloromyotomy or pyloroplasty procedures may not have provided adequate relief from the outflow obstruction. Pylorotomy in cats has been described in 2 clinical reports.13,14 Runk et al13 described the use of this technique with a favorable outcome in a 1-year-old cat for treatment of duodenal perforation associated with oral administration of carprofen following ovariohysterectomy. After prolonged treatment for septic peritonitis, the cat recovered fully and was apparently normal according to the owners 6 months after surgery. A report by Shimamura et al14 described use of the Billroth I procedure in a 7-year-old cat for treatment of a pyloric mass, which was histologically confirmed as lymphoma. Although recovery from surgery was excellent, the patient began vomiting intermittently approximately 3 weeks after surgery. The vomiting was attributed to megaesophagus, which was reported as a complication of the pylorectomy, although the relationship between that condition and the surgery was not clear.

The prognosis following pylorectomy in dogs for benign causes of gastric outflow obstruction is good. However, complications can occur, including dehydration and septic peritonitis, gastric atonia, persistent or recurrent outflow obstruction, and inadvertent common bile duct trauma.12,13 Although the technique for gastroduodenostomy is similar to an intestinal end-to-end anastomosis, a thorough knowledge of pyloric anatomy is necessary to avoid serious surgical errors because of the proximity of the pancreas, bile duct, and major vessels.12,13 Congenital pyloric stenosis in cats is rare. However, it should be considered when a young cat, particularly of Siamese breed lineage, is evaluated because of signs of pyloric outflow obstruction. To the authors’ knowledge, this is the first report that describes ultrasonographic and histologic changes associated with pyloric stenosis in a cat. Additionally, this is the first report of use of the Billroth I procedure to treat this condition in a cat, and the treatment resulted in resolution of the clinical signs associated with pyloric obstruction.

References

10. Runk et al. Ultrasound evaluation of gastric contents through the pylorus despite peristalsis, supporting decreased peristalsis. In a previous report that described ultrasonographic examination of 6 dogs with pyloric hypertrophy, 5 were noted to have no movement of gastric contents through the pylorus despite peristalsis activity. Additionally, a fluid- or food-distended stomach can aid in determining delayed gastric emptying, especially if the time of previous feeding is known. In children, diagnosis of pyloric stenosis is usually made ultrasonographically, and for experienced evaluators, accuracy of diagnosis approaches 100%. In these cases, the lesion can be seen as a thickened prepyloric antrum bridging the distended stomach and duodenal ampulla. Results of upper gastrointestinal endoscopy were definitive for stenosis in the cat of this report and prompted surgery.

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References

From this month’s *AJVR*

**Effects of route of administration and feeding schedule on pharmacokinetics of robenacoxib in cats**

Jonathan N. King et al

**Objective**—To establish pharmacokinetics of robenacoxib after administration to cats via the IV, SC, and oral routes.

**Animals**—24 cats.

**Procedures**—In a crossover design, robenacoxib was administered IV, SC, and orally (experiment 1) and orally (experiment 2) to cats with different feeding regimens. Blood robenacoxib concentrations were assayed, with a lower limit of quantification of 3 ng/mL.

**Results**—In experiment 1, geometric mean pharmacokinetic values after IV administration of robenacoxib were as follows: blood clearance, 0.44 L/kg/h; plasma clearance, 0.29 L/kg/h; elimination half-life, 1.49 hours; and volume of distribution at steady state (determined from estimated plasma concentrations), 0.13 L/kg. Mean bioavailability was 69% and median time to maximum concentration (Cmax) was 1 hour for cats after SC administration of robenacoxib, whereas mean bioavailability was 49% and 10% and median time to Cmax was 1 hour and 30 minutes after oral administration to cats after food withholding and after cats were fed their entire ration, respectively. In experiment 2, geometric mean Cmax was 1,159, 1,200, and 692 ng/mL and area under the curve from 0 to infinity was 1,337, 1,383, and 1,069 ng·h/mL following oral administration to cats after food withholding, cats fed one-third of the daily ration, and cats fed the entire daily ration, respectively.

**Conclusions and Clinical Relevance**—For treatment of acute conditions in cats, it is recommended to administer robenacoxib by IV or SC injection, orally after food withholding, or orally with a small amount of food to obtain optimal bioavailability and Cmax. (*Am J Vet Res* 2013;74:465–472)