GASTROINTESTINAL DISEASE IN EXOTIC SMALL MAMMALS

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Abstract

Exotic small mammal medicine is a relatively new specialty area within veterinary medicine. Ferrets, rabbits, and rodents have long been used as animal models in human medical research investigations, resulting in a body of basic anatomic and physiologic information that can be used by veterinarians treating these species. Unfortunately, there is a paucity of veterinary articles that describe clinical presentation, diagnosis, and treatment options of gastrointestinal (GI) disease as it affects exotic small mammals. Although there is little reference material relating to exotic small mammal GI disease, patients are commonly presented to veterinary hospitals with digestive tract disorders. This article provides the latest information available for GI disease in ferrets (*Helicobacter mustelae* gastritis, inflammatory bowel disease [IBD], GI lymphoma, systemic coronavirus, coccidiosis, and liver disease), rabbits (GI motility disorders, liver lobe torsion, astrovirus, and coccidiosis), guinea pigs (gastric dilatation volvulus [GDV]), rats (*Taenia taeniaeformis*), and hamsters (*Clostridium difficile*). Both noninfectious diseases and emerging infectious diseases are reviewed as well as the most up-to-date diagnostics and treatment options. Copyright 2013 Elsevier Inc. All rights reserved.

Key words: exotic small mammal; gastrointestinal; digestive; diarrhea; infectious; stasis

G astrointestinal (GI) diseases are commonly diagnosed in exotic pet mammals. With veterinarians treating carnivorous, omnivorous, and herbivorous species of exotic small mammals, it is imperative that the latest in medical overviews of GI diseases affecting these animals are incorporated into clinical protocols. This article details the latest information regarding small exotic pet GI diseases. Although dental diseases and nutrition are major issues in the area of small exotic mammal GI health, they are beyond the scope of this article and are not reviewed here.

FERRETS

Noninfectious Disease

As ferrets share similar digestive physiology to dogs and cats, there are certain GI disease conditions (e.g., inflammatory bowel disease [IBD]) that will be common across these groups of animals. However, ferrets are also unique enough that they may present with abnormalities that are more specific to their body system. Ferrets are often presented with a nonspecific history of chronic diarrhea and cachexia, which usually corresponds to a significant diagnostic challenge (Table 1).

Although frequently diagnosed, there is a relative lack of information in the scientific literature regarding ferret idiopathic IBD.[1,2] When IBD is suspected, biopsy samples of the intestinal mucosa are used to differentiate lymphoplasmacytic infiltrates from other disease conditions that have similar clinical signs (e.g., eosinophilic infiltrate and neoplasia).

Gastroscopy provides a noninvasive tool to examine the gastric mucosa of ferrets (weighing

TABLE 1. Diagnostic plan: Ferret gastroenteritis

Complete history
   Diet, diarrhea, vomiting, weight loss, dysphagia,
     anorexia, lethargy dehydration, weakness, and fever
Clinical examination
   Adrenomegaly and abdominal pain
Blood examination
   Peripheral eosinophilia
Fecal examination
   Coproscopy
     Giardia sp.
     Eimeria sp.
   Bacteriology
     Salmonella sp.
     Campylobacter sp.
   PCR sequencing
     Ferret enteric coronavirus (FRECV)
     Rotavirus
     Helicobacter
     Canine distemper
Ultrasonography
   Lymph node
   Adnexa (liver, pancreas)
   Infiltration of the gastric and enteric lamina
Cytology and/or histopathology of the tissue
   Gastric
     Lymphoplasmacytic infiltrate (intestine and
       lymph node): inflammatory bowel disease
     ±Atrophy of the villi: PCR sequencing Rotavirus/
       FRECV
     Eosinophilic infiltrate (intestine and lymph
       node): eosinophilic enteritis
     Lymphoma: immunohistochemistry typing
   Intestine
     Lymphoplasmacytic infiltrate (intestine and
       lymph node): inflammatory bowel disease
     ±Atrophy of the villi: PCR sequencing Rotavirus/
       FRECV/distemper
     Eosinophilic infiltrate (intestine and lymph
       node): eosinophilic enteritis
     Lymphoma: immunohistochemistry typing
   Colon
     Proliferative colitis: *Lawsonia intracellularis*,
     Desulfovibrio
     Adenomatous polyp
     Lymphoma: immunohistochemistry typing
   Lymph node
     Lymphoplasmacytic infiltrate (intestine and
       lymph node): inflammatory bowel disease
     Eosinophilic infiltrate (intestine and lymph
       node): eosinophilic enteritis
     Pyogranulomatous infiltrate (lymph node): PCR
       sequencing ferret systemic coronavirus/
       Mycobacterium sp.
     Lymphoma: immunohistochemistry typing

> 0.8 kg) and characterize different gastric disease (Fig. 1; Table 2).[3] Gastroscopy also allows direct observation of the GI tract lumen for the collection of diagnostic tissue samples from gastric and duodenal mucosa (Fig. 2A and B). Unfortunately, and for obvious reasons, full-thickness biopsies cannot be harvested through a gastroscopy procedure. For certain GI disease conditions a full-thickness tissue sample is required for a definitive diagnosis. When lymphoplasmacytic infiltrates are identified in the GI tract of ferrets, the etiologic agent is rarely identified.[1] In a review of 9 ferret cases in which lymphoplasmacytic infiltrates were identified, spiral-shaped bacteria consistent with *Helicobacter* sp. was diagnosed in 1 animal whereas the other cases were characterized with moderate to severe lymphoplasmacytic gastritis and duodenitis. One case was described as having an eosinophilic infiltrate of the gastric mucosa.

Eosinophilic gastroenteritis has been described as a GI disease of ferrets <5 years old.[2,4,5] Clinical signs include vomiting, lethargy, and green diarrhea. Eosinophilic gastroenteritis has to be distinguished from an eosinophilic condition present in the lamina propria or in the lymph node in case of IBD (Fig. 3).[5] When eosinophilic gastritis is present, eosinophilic infiltrates are found in the digestive tract, associated lymph nodes, liver, and lungs.[4,5] There may be an association between dietary hypersensitivity and eosinophilic gastroenteritis. Protein digestibility in ferrets is limited compared with cats.[6,7] Therefore, ferrets require food items that contain highly digestible animal protein (35% to 55% of dry matter; e.g., whole prey rat contains 55% protein).[7] Recommended medical management for eosinophilic gastroenteritis cases includes immunosuppressive therapy using prednisone (2 mg/kg, once a day), cyclosporine (4 mg/kg, twice a day), or azathioprine (0.9 mg/kg, once every 2 days).[1,5,8]

Although rare, a case of adenomatous polyp causing tenesmus and diarrhea mimicking a colitis associated with *Lawsonia intracellularis* infection was described in a ferret.[9] Conversely, GI neoplasms (e.g., lymphoma) are commonly described in ferrets.[10-12] Mayer and Burgess[13] recently suggested standardizing the grading scheme for lymphoma in ferrets based on clinical stage, histomorphology, and immunophenotype. Immunohistochemistry is an invaluable tool to identify rare GI tumors and help one establish a more accurate prognosis for the patient.[14] In a survey of 20 ferrets diagnosed with lymphoma, 45% were of GI origin.[10] Digestive tract lymphomas are typically of T-cell origin and predominantly high-grade immunoblastic neoplasias involving the mesenteric lymph node.[10] In another study evaluating 29 ferrets affected by

FIGURE 1. Gastric endoscopy (3 mm diameter flexible scope) in an anesthetized ferret.

lymphoma, the median survival time for a T-cell lymphoma was 5 months (range 0.5 to 14) compared with 8.4 months (range 2.0 to 19.0) for B-cell lymphoma.[11] Chemotherapy has been proposed as a treatment option but further studies are needed to assess the therapeutic efficacy and adverse side effects, as well as quality of life during the treatment period.[11]

Infectious Disease

Publications of ferret gastric disease are dominated by *Helicobacter mustelae*.[15,16] The pathogenicity of *Helicobacter* spp. associated with gastritis in canine and feline species is unknown but may be more prevalent than once thought.[17,18] In a review of ferret gastritis cases, *Helicobacter* spp.-like organisms were only identified once through

histological examination (Table 2). Although polymerase chain reaction (PCR) diagnostic testing on affected tissues may be beneficial to definitively identify the organism, correlation between isolation of *Helicobacter* spp. by PCR and gastritis remain difficult to interpret.[18]

Ferret coronavirus infection is a very important GI disease of ferrets.[19] Coronaviruses are positive-stranded ribonucleic acid (RNA) viruses that are currently placed into 3 different classification groups. Ferret coronavirus is in group 1 along with feline coronavirus. Although closely related, there are 2 genetically distinct forms of ferret coronavirus: a ferret enteric coronavirus (FRECV) and a ferret systemic coronavirus (FRSCV). FRECV has been recently characterized and is linked with the disease condition known as epizootic catarrhal enteritis.[20] This is a highly contagious disease that infects ferrets of all ages, but older ferrets commonly present with more severe clinical disease. Clinical signs include green mucoid diarrhea and, later in the disease course, "birdseed-like" consistency of the fecal material. Villous atrophy and lymphocytic enteritis are common histological findings.

FRSCV is an emergent form of feline infectious peritonitis (FIP)-like coronavirus infection described in ferrets.[21,22] Typically, FRSCV affects young ferrets that are presented with generalized abdominal polyadenomegaly, diarrhea, weight loss, anorexia, and vomiting. Less common signs associated with FRSCV include hindlimb paresis,

| **TABLE 2. Signalment, clinical signs, and histologic diagnosis of ferrets following gastric endoscopic procedures using a 3 mm diameter flexible fiberscope**				
Age (y)	**Sex**	**Weight (kg)**	**Clinical Signs**	**Histopathological Findings**
5	Male	1.38	Chronic diarrhea	Diffuse minimal lymphocytic gastritis
4	Male	1.4	Chronic diarrhea	Moderate multifocal lymphocytic gastritis severe diffuse lymphoplasmacytic duodenitis
4	Male	1.28	Vomiting	Marked chronic lymphoplasmocytic gastritis with intralesional spiral-shaped bacteria consistent with *Helicobacter* sp.
4	Female	0.80	Dysorexia	Moderate chronic multifocal lymphoplasmacytic gastritis
5	Male	0.99	Chronic diarrhea	Diffuse chronic severe lymphoplasmacytic gastritis and enteritis
1	Male	1.2	Vomiting and ptyalism	Moderate lymphoplasmacytic gastritis and enteritis
3	Male	1.08	Diarrhea	Moderate diffuse lymphoplasmacytic gastritis and enteritis
4	Male	1.2	Dysphagia and anorexia	Marked diffuse lymphoplasmacytic gastritis and enteritis
3	Female	0.8	Vomiting	Marked chronic lymphocytic and eosinophilic gastritis
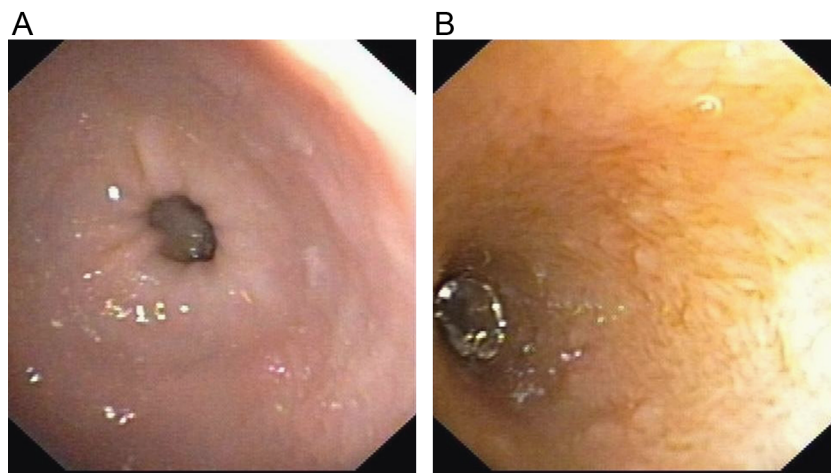
FIGURE 2. (A) Endoscopic view of a ferret's pylorus. (B) Endoscopic view of the proximal duodenum in a ferret.

seizures, respiratory signs, bruxism, icterus, erythema of the skin, colored urine, and rectal prolapse. Ultrasonography may be beneficial when trying to diagnose FRSCV.[23] Serology to detect antibodies to ferret coronavirus, immunohistochemistry staining, PCR, and specific reverse transcriptase PCR (RT-PCR) have all been described as diagnostic tests to detect FRSCV in ferrets, but all have serious flaws in the ability to detect this disease.[24-27] Necropsy findings from ferrets that die from FRSCV include polyadenomegaly and white nodules on the serosal surface, grossly resembling the dry form of FIP in cats. Histologically, pyogranulomatous infiltrates are commonly observed in the lymph nodes and the nodules of FRSCV infected ferrets (Fig. 4A and B).

Other viruses (e.g., group C rotaviruses) have recently been described as a cause of enteritis in juvenile ferrets.[28] Affected individuals usually present with severe dehydration and diarrhea.[28] Gross pathological findings include a thin-walled intestinal tract, and microscopic examination of these tissues reveals superficial atrophic enteritis with necrosis of the epithelial cells at the villous tips.[28] An RT-PCR specific for group C rotaviruses was developed that allows for accurate identification of this virus in juvenile ferrets.

Eimeria furonis infection can be pathogenic (e.g., dehydration, weight loss, and mortality) in immunosuppressed ferrets.[29] Diagnosing coccidiosis in ferrets is achieved using fecal flotation. Sulfadimethoxine has been used to control coccidiosis in a group of ferrets, but did not totally clear the organism from all individuals.[29] The use of recently released coccidiocidal drugs (e.g., ponazuril, 30 mg/kg, per os) may offer better treatment success against these parasites.[30] *Giardia duodenalis* has been isolated from ferrets, but the prevalence of this organism remains unknown.[31,32] Veterinary personnel and ferret owners should understand the potential zoonotic threat associated with animals diagnosed with giardiasis.[33]

Miscellaneous

Extrahepatic obstructions and cholestasis have been described in ferrets (Fig. 5).[34,35] Affected ferrets exhibited lethargy, icterus, hyperbilirubinemia, elevated alanine aminotransferase, and bilirubinuria. Sonographic results obtained from ferret patients with suspect extrahepatic obstructions included severe thickening of the bile duct. The underlying etiology of the obstruction has yet to be identified, although a green proteinaceous plug has been observed in 2 cases and sediment in 1 case.[34,35]
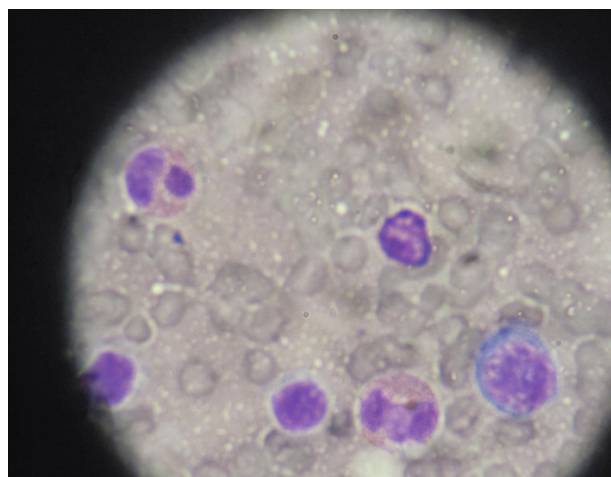

FIGURE 3. Eosinophilic infiltrate in the mesenteric lymph node of a ferret (40×).
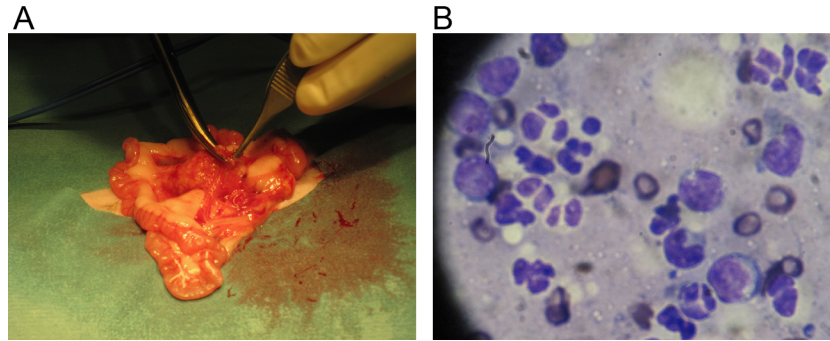
FIGURE 4. (A) Biopsy of an enlarged mesenteric lymph node. (B) Pyogranulomatous infiltrate in a mesenteric lymph node (40×).

Surgical management was necessary for resolution in all cases.

Although pancreatitis is considered a rare disease in ferrets, a review of pathological findings in ferrets revealed 50 cases of acute or chronic pancreatitis.[36] The ferret pancreatitis cases were classified as acute if necrosis or neutrophilic inflammation was present. Cases were classified as chronic if lymphocytic or pyogranulomatous infiltrate or fibrosis was observed. There is an isolated histopathological report of pancreatitis associated with β-cell islet tumor.[37]

GDV has been reported in a 1-year-old ferret with no predisposing or etiologic factor identified (Fig. 6).[38] Treatment of GDV in dogs requires aggressive fluid therapy and surgery.[39]

RABBIT

GI Stasis

GI stasis is one of the most common disease conditions diagnosed in domestic rabbits. Interpreting the cause of decreased fecal output and anorexia associated with gastric stasis, dysbiosis, or cecal impaction remain major diagnostic challenges for the clinician due to the nonspecific clinical presentation associated with the condition. A focused diagnostic plan should be developed (Table 3) to give one the best opportunity for a fast and accurate definitive diagnosis. Once a definitive diagnosis has been determined, an aggressive, effective treatment plan can be initiated.

Common clinical signs of rabbits diagnosed with GI stasis include acute lethargy/depression, bruxism, stretched or hunched body position, and unresponsiveness to external stimuli.[40,41] Acute death associated with GI stasis in rabbits has also been reported.[41] Abdominal palpation of an effected animal will often elicit a pain response

and a dilated stomach.[40] Radiographic signs of GI stasis in a rabbit include gas in the stomach or in the small intestine without gas in the cecum (Fig. 7A and B).[40] When distended, the stomach is round and can be filled with fluid or gas; a concurrent gas pattern in the duodenum consistent with segmental ileus is also common.[42] Although commonly used in practice, references for rabbit abdominal ultrasonography are limited to assessment of intestinal motility.[43] Other diagnostic imaging modalities, such as fluoroscopy, may prove useful for diagnosing and assessing treatment response of rabbit GI stasis in the future once standardized references have been established for this species.

Hyperglycemia has been promoted as an indicator of instestinal obstruction in the rabbit.[44] Eighteen rabbits with intestinal obstruction had a mean blood glucose measurement of 24.7 mmol/L (444.6 mg/dL) and 51 rabbits with gut stasis had a
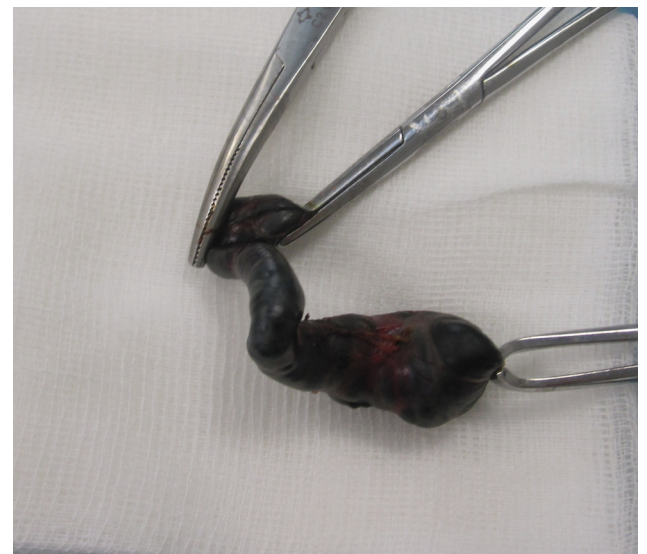

FIGURE 5. Resected gallbladder due to severe cholestasis in ferret. Note the enlarged common bile duct.
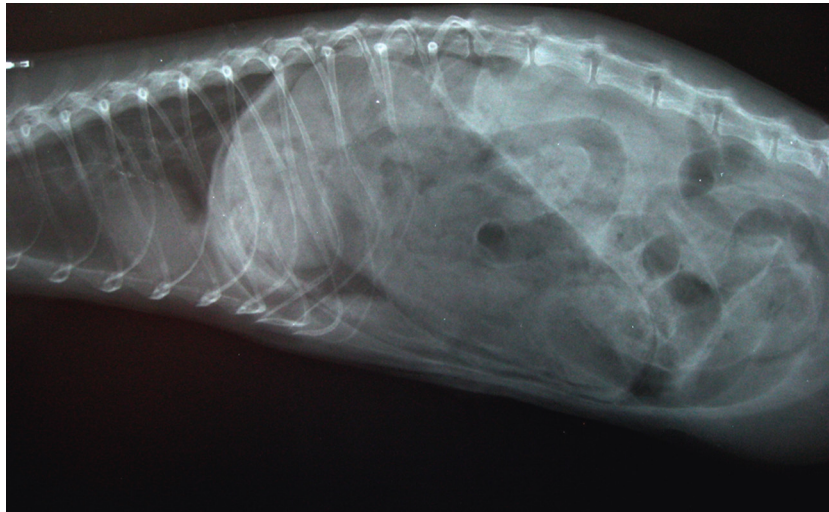
FIGURE 6. Lateral radiographic image of a gastric dilatation/volvulus in a ferret. The stomach is severely dilated, filled with air and deviated from its axis. Gaseous distentions in the distal intestine loops are also observed.

measurement of 8.5 mmol/L (153 mg/dL).[44] Further work to characterize the biological meaning of this clinical finding is needed.

Blood L-Lactate measurements are commonly used as a prognostic factor for dogs with gastric dilatation/volvulus. A relative and persistent high lactatemia in dogs with gastric dilatation/volvulus has been associated with tissue anoxia, necrosis, and a poor prognosis.[45] Very few blood L-Lactate scientific investigations have been performed in rabbits to study lactatemia and its association with gastric dilatation/volvulus. Two studies reported L-Lactate mean values in normal rabbits from 7.3 (±2.9) mmol/L (65.7 mg/dL ± 26.1 mg/dL) to 5.1 (±2.1) mmol/L (45.9 mg/dL ± 18.9 mg/dL), which are higher than physiologic values in carnivores (2.4 mmol/L; 21.6 mg/dL).[46,47] However, it remains unclear whether high lactate values have any clinical significance in obstructed rabbits and further studies are necessary to verify this hypothesis.

Various causes of obstruction have been identified in rabbits. In a review of 76 rabbits diagnosed with GI obstruction, 49 were found with pellets and hair in the stomach (Fig. 8).[48] The incidence of gastric trichobezoar has been investigated in at least 1 laboratory colony of New Zealand white rabbits and found to be as high as 3.2%.[49] Other authors report the mortality rate to be higher in long-haired rabbits (e.g., Angora rabbits), with trichobezoars accounting for almost 29% of mortalities.[50]

After trichobezoars, GI tumors are the second most frequent cause of intestinal obstruction in rabbits. Five cases of rabbit intestinal obstruction were caused by localized or disseminated tumors (lymphoma) in a series of 64 patient admissions. In a recent report, cecal lymphoma was described in a rabbit (Fig. 9).[51] Other known causes of GI obstruction in rabbits include carpet fibers,

TABLE 3. Diagnostic plan: Rabbit gastrointestinal stasis
Complete history
Diet, anorexia, weight loss, ptyalism, decreased or lack of fecal output, smaller faeces, clumped faeces, and diarrhea
Clinical examination
Abdominal distension, tympanism, abdominal pain, stomach distension, and reduced abdominal sounds
Oral examination
Dental disease, ulcers, and abscess
Blood examination
Enzymes and electrolytes
Glycemia
Lactate
Uremia and creatinine
Radiograph (consider serial radiographs for monitoring)
Gastric stasis: gas distension in the stomach
Caecal stasis: gas distension in the cecum
Paralytic ileus: generalized digestive distension
Ultrasound
Decreased motility
Obstruction
Soft tissue mass effect (neoplasia)
Increased thickness of the digestive wall
Liver lobe torsion
Reproductive tract disease
Renal disease

tapeworm cysts, postoperative adhesions, and locust bean seeds.[48]

Although treatment is often unrewarding in rabbits diagnosed with cecal impactions, pain medication and intravenous fluids are recommended to stabilize the patient. Treatment with prostaglandin F_2 alpha (0.2 mg/kg) once every 24 to 36 hours after oral administration of liquid paraffin has been suggested as a treatment alternative for cecal impaction in rabbits.[52] In a series of case reports, 3 rabbits treated with prostaglandin and liquid paraffin evacuated their cecal contents within 24 to 48 hours of treatment and all of the animals fully recovered. However, prostaglandin, as with any therapeutic agent, can produce a number of adverse side effects in the patient being treated.

Treatment options for GI obstruction in rabbits include medical management and surgical treatment. Surgical management for GI obstruction in rabbits has been associated with high complication rates (up to 50% in affected animals).[48] The standard gastrotomy or enterotomy is difficult to perform because the rabbit intestinal wall is very thin.[48]

Medical treatment for rabbits diagnosed with GI obstruction relies on fluid therapy and pain management.[42] Recognition of hypovolemic shock is important as it requires more aggressive fluid therapy than GI stasis. Bradycardia (<180 bpm), hypothermia ($97°F/36.1°C$), and hypotension (<90 mm Hg) are commonly observed in rabbits with GI obstruction.[42] Combination of hypertonic saline, colloid, and crystalloid fluid therapy are necessary to restore blood pressure until the patient's body temperature is stabilized. Fluid maintenance is higher in rabbits than in carnivores (~ 5 mL/kg/h).[42] Gastric decompression has also been recommended in case of severe tympany by orogastric or nasogastric tubes.[42]

Medical Treatment Nonobstructive Gastric Stasis

To increase GI motility in nonobstructive cases, several pharmaceutical agents have been used. Although metoclopramide has been reported as a prokinetic agent, no pharmacokinetic or pharmacodynamic data in rabbits are available to validate its use in this species.[41] Antacid has also been suggested as a treatment option for gastric ulceration in rabbits, a common postmortem finding.[40] Use of ranitidine has been recommended but no studies have been published describing its effectiveness in rabbits. Experimental data show some synergistic action with cisapride,
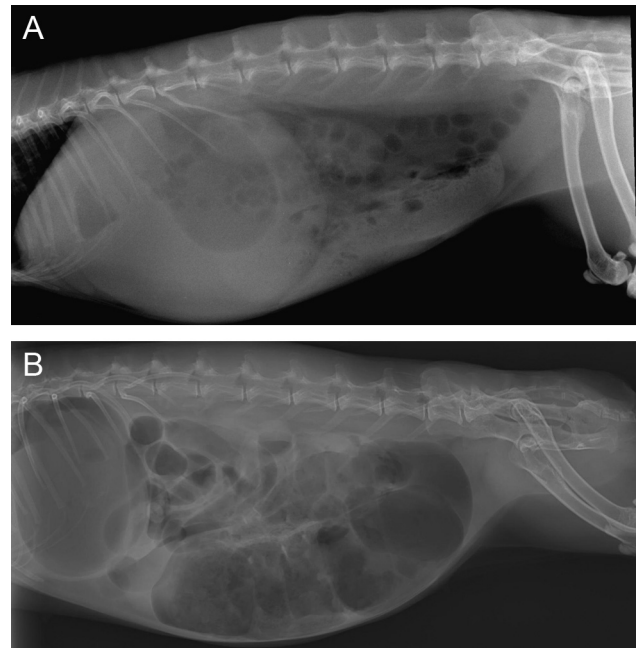
FIGURE 7. (A) Lateral radiographic image of a gastric dilatation of the stomach in a rabbit, consistent with obstruction. (B) Lateral radiographic image of cecal distention in a rabbit. Note that this finding can be related to stasis and not obstruction.

but there appears to be antagonist action as the dose increases.[53] Domperidone is also influenced by various drugs including ranitidine.[54] Recent research focusing on the effects of domperidone and trimebutine in rabbits has determined that domperidone stimulates contraction of the sphincter of Oddi.[55] Trimebutine can either stimulate or inhibit sphincter of Oddi contraction with a dose-dependent mechanism.[55] Although few data are available, those experimental results emphasize the need for further research to

FIGURE 8. Perioperative view of a hairball removal in a rabbit. A gastrotomy is performed on the large curvature of the stomach.
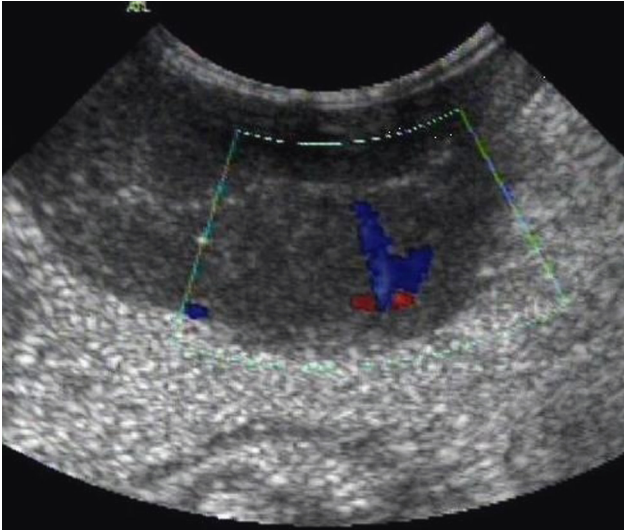
FIGURE 9. Ultrasound of a cecal lymphoma in a rabbit. Note the thickening of the cecal wall.

determine the recommended treatment options for nonobstructive GI stasis in rabbit patients.

Miscellaneous

Liver lobe torsion is an emerging disease condition in rabbits (Fig. 10).[56-58] Rabbits that are young to middle-aged (1.5 to 6 years old) appear to be most likely affected by this disease, which is associated with nonspecific clinical signs and decreased fecal output. Elevated hepatic enzymes are often observed but have not proven to be repeatable across rabbit liver lobe torsion cases. Ultrasonographic findings may reveal abdominal effusion, lack of blood flow in the affected lobe, and heterogenous parenchyma with anechoic to hypoechoic striations.[56,57] The liver lobe most frequently affected in rabbit is the caudate lobe, but other lobes are susceptible.[56,57] The underlying etiology for liver lobe torsion in rabbits includes adjacent organ distension and the narrow attachment of the caudate lobe.[56] Treatment by hepatectomy is curative and is associated with a good prognosis.[56] The surgical technique describes circumferential ligatures around the hilus of the affected lobe and transection distal to the ligatures.[56]

Biliary cystadenoma is another hepatic disease condition found in rabbits.[59] Biliary cystadenomas appear as a well-differentiated, cystic, multiseptate mass emanating from the surrounding hepatic tissue. Although recommended, a fine-needle aspirate may not achieve a definitive diagnosis.[59] In most cases, abdominal sonography is the diagnostic test of choice to confirm a diagnosis of biliary cystadenoma in a rabbit. A partial hepatectomy is considered curative for biliary cystadenoma.[59]

Infectious Disease

A novel astrovirus was described in rabbits after screening animals with enteritis complex.[60] Astroviruses were first discovered in 1975 using electron microscopy to investigate an unusual cluster of human diarrhea cases.[61] Astroviruses are small icosahedral virus with a diameter of 28 to 35 nm. They have a characteristic 5- or 6-pointed star-like surface structure when viewed by electron microscopy. The astrovirus has an RNA genome within a nonenveloped icosahedral capsid and has been isolated from lambs,[62] calves,[63] red deer,[64] cats,[65] mice,[66] mink,[67] bats,[68] turkeys,[69] and chickens.[70] The epidemiology of astrovirus infection in rabbits is still unknown, but in human medicine, it has been determined that the immunocompromised are most susceptible to the disease.[61] This virus has been isolated in rabbits presenting with enterocolitis or enteritis complex. Clinical signs in rabbits from which astrovirus has been isolated are diarrhea, dehydration, abdominal distension, and hyperthermia. Astrovirus has also been isolated from animals without diarrhea, which suggest a subclinical disease state. Currently it is unknown if this virus can act as primary agent of enteritis in rabbits. Histopathologically, macroscopic lesions of astrovirus infected tissue are noted by the presence of hemorrhagic foci within the tissue of the small intestine. Microscopically the villi are shortened and thickened, with dilatation of the intestinal

FIGURE 10. Perioperative view of a liver lobe torsion in a rabbit. Note the brown color and the friable aspect of the caudate lobe.

lamina propria and capillaries. In mice and mink, the recommended control and preventive measures are based on environmental disinfection (i.e., cleaning the environment, separation of the affected from noninfected animals).[63,66] Antibiotic therapy for both infected and exposed animals may reduce the occurrence of concurrent secondary bacterial infections, thereby improving the patient's prognosis.

Coccidia are frequently a cause of illness in young rabbits (<6 months old) and, in general, the most common parasites of the rabbit GI tract. Ten species of intestinal coccidia, all members of the genus *Eimeria*, are reported to infect domestic rabbits.[71] The *Eimeria* species described in this article are considered species specific for the rabbit, and exposure is through fecal-oral transmission. Adult carriers often serve as a source of infection for newborn rabbits. Rabbits at 2 to 3 months of age appear to be most susceptible to the pathogenicity of intestinal coccidiosis.[72,73] The number of young animals affected in rabbit colonies can vary, with some cases reaching 100%.[71] The most important species of intestinal coccidia as it relates to overall rabbit health are *Eimeria perforans*, *Eimeria magna*, and *Eimeria media*, with E. *perforans* being the most common and the least pathogenic species for laboratory rabbits.[71,74] In 1 pet rabbit research study, *E. magna* was identified as the dominant coccidia species (60%), along with *E. media* (30%) and *Eimeria intestinalis* (10%).[75] *E. intestinalis* is generally considered the most pathogenic, E. *magna* moderately pathogenic with important subclinical effects, and *E. media* relatively nonpathogenic to rabbits.[75]

Rabbits may have subclinical coccidiosis with no overt clinical signs. If clinical disease is present, the patient may be cachexic, dehydrated, and have intermittent to severe, dark and watery diarrhea with mucus or blood.[41] Animals with severe diarrhea may develop intussusception. When a rabbit dies as a result of cocciodiosis it is most often attributed to dehydration and secondary intestinal dysbiosis. Postmortem examination may reveal ulceration of the intestinal epithelium. Microscopically, villous atrophy, cellular congestion, edema, and epithelial necrosis may be present in the affected tissue. The presence of coccidial organisms in fecal samples (e.g., direct smear, flotation, and centrifugation) or intestinal preparations (e.g., smears and histopathology) of clinically diseased animals supports a presumptive diagnosis. A definitive diagnosis of coccidiosis in rabbits is based on histologic findings.

Numerous therapeutic agents have been used as preventatives and treatments for rabbit intestinal and hepatic coccidiosis, including amprolium (amprolium 9.6% in drinking water 0.5 mL per 500 mL),[76] sulfadimethoxine (15 mg/kg orally, twice a day for 10 days),[41,73] trimethoprim-sulfamethoxazole (30 mg/kg orally, twice a day for 10 days),[41] and toltrazuril (10 mg/kg orally).[77,78] A recent research investigation revealed that a single oral dose of 2.5 mg/kg or 5.0 mg/kg toltrazuril, or a single oral dose of 50 mg/kg sulfadimethoxine followed by placement in drinking water at 1 g/4 L water for 9 days, were found to significantly reduce the fecal oocyst count in rabbits by 73% to 99%.[75] The results of the study also reported that the extent of fecal oocyst reduction was not dependent on the dose of toltrazuril.[75] Oocyst counts began to rise again in the days after the cessation of treatment, suggesting that reinfection may be the cause of the increased parasite numbers.[75] Based on the results of the research investigation described previously, effective treatment for rabbits diagnosed with pathogenic coccidiosis can be achieved with a single dose of toltrazuril, coupled with cleaning and a disinfection of the litter tray and rabbit access areas.[75]

GUINEA PIGS

Recently, several reports of GDV in guinea pigs have been published.[79-81] The common sequela of gastric dilation in guinea pigs involves the stomach being filled with an increased amount of air that displaces this organ from its axis leading to severe volvulus. This disease condition has been identified in humans, dogs, swine, cats, and ferrets.[79] Guinea pigs affected with GDV typically range in age from 1.5 to 3 years old.[79] Tachypnea, lethargy, anorexia, and an absence of fecal production are common clinical signs associated with gastric dilatation. Survey radiographic images often show a severe gas distention, filling 50% of the abdominal cavity, and must be differentiated from gastric stasis (Fig. 11).

No predisposing factor has been clearly identified as the underlying cause of gastric dilatation in guinea pigs, therefore, a multifactorial etiology is likely. Stress, an underlying factor of gastric dilatation in dogs, could have a similar affect on guinea pigs as they are sensitive animals.[82] Gastric stasis, rapid ingestion of large amount of food and water, and dilation secondary to anesthesia disorders have also been suggested as contributing factors to this condition in guinea pigs.[79,80-84]

FIGURE 11. Lateral radiographic image of a gastric volvulus in a guinea pig. Note the gas-filled intestinal loop cranial to the stomach; this is characteristic of a volvulus.

It has been suggested that gastric dilatation in guinea pigs may be associated with gestation.[79] As guinea piglets are relatively large and precocious at birth, organ displacement in the sow's abdomen late in pregnancy could theoretically predispose the animal to gastric dilatation.[79] Additional work is required to determine whether this risk factor is important.

Surgical attempts to correct GDV in guinea pigs have been made, but there have not been any reported successes. Although these cases carry a grave prognosis, supportive therapy including aggressive intravenous fluid therapy, pain management, and decompression of the stomach through nasogastric or orogastric tubes are recommended.[83]

RAT

Taenia taeniaeformis is a predisposing factor for the development of hepatic sarcoma in rats.[85] Clinical signs commonly observed in rats diagnosed with hepatic *T. taeniaeformis*/sarcoma are nonspecific and may include lethargy, weight loss, anorexia, and sudden death. Laboratory findings in infected rats can include mild decreases in serum cholesterol concentration; increases in the serum activity of alanine aminotransferase and/or aspartate aminotransferase; and increases in the numbers of peripheral blood neutrophils, lymphocytes, and/or eosinophils. Rats infected with larvae of *T. taeniaeformis* may have decreased gastric acid secretion and hyperplasia of the gastric and intestinal mucosa due to substances secreted by the parasitic larvae.[86] Moreover, *T. taeniaeformis* infestation may adversely affect a rat's reproductive function.[87]

Diagnosis of *T. taeniaeformis* infection in the laboratory rat may be achieved with the use of ultrasonographic and/or radiologic imaging. In experimental studies with laboratory rats,

ultrasonography was used to detect parasitic cysts in the liver, and contrast radiography was used to detect hepatomegaly and gastric and intestinal mucosal changes.[88,89] Although imaging has been described in the laboratory setting, the clinical utility of ultrasonography, radiography, and serology for the diagnosis of T. *taeniaeformis* in pet rats remains to be investigated.

Treatment options for pet rats infected with *T. taeniaeformis* are unknown. However, experimental studies indicate that praziquantel is effective at killing both adult and larval forms of *T. taeniaeformis*.[90] The safety of praziquantel for the treatment of encysted larvae in rats is unknown, and it is possible that killed larvae will elicit a marked host immune response that is harmful to the rat. The recommended dose of praziquantel for pet rats is 30 mg/kg orally every 14 days for 3 treatments. *T. taeniaeformis* infected rats and mice pose a potential zoonotic disease risk.

HAMSTER

Clostridium difficile has been described as the primary causative agent of GI disease in hamster colonies.[91] *C. difficile* is a highly diverse, Gram-positive, anaerobic, spore-forming bacterium. Detailed data concerning the prevalence of subclinical carriers in many species, including the Syrian hamster, is lacking. However, among common laboratory animal species, Syrian hamsters are the most sensitive to naturally acquired disease from *C. difficile* infection.[92] Affected hamsters often die from diarrhea due to typhlitis and colitis or without any obvious clinical signs of disease.[91,93] Lesions are predominantly cecal, with occasional involvement of the ileum or colon.[91] The cecum is often distended by fluid, with multiple petechial to ecchymotic hemorrhages of the cecal wall. Histopathological analysis usually reveals lesions associated with

acute toxic changes (e.g., mucosal hemorrhage, and edema). PCR detection of *C. difficile* is highly sensitive and can discriminate between toxigenic and nontoxigenic strains of the organism by detecting toxin-producing genes.[94] Other diagnostic measures include isolation of the pathogen from fecal samples and detection of *C. difficile* toxins by enzyme-linked immunosorbent assay (ELISA) methodology.[94] Microbiological culture to isolate the pathogen can be done, but it is difficult because *C. difficile* is an anaerobic bacterium.

A recent survey on young hamsters sold through pet stores that died revealed that the most common disease condition that caused death was enteritis, followed by nematodes, cestodes, bacteria, and *Spironucleus* spp.[95] Hamsters are commonly infected with the mouse pinworm, *Syphacia obvelata*. This pinworm is often isolated from wild and pet rodents. *S. obvelata* has a direct life cycle, there is orofecal exposure, and the parasite eggs are found around the anus of the infected host. The eggs have a sticky outer layer and are resistant to rapid environmental degradation. In immunocompetent animals, *S. obvelata* infestation does not result in overt clinical disease. Clinical disease signs attributed to the pinworm include rectal prolapse, poor hair coat, and cachexia.[96] Diagnosis of pinworms may be accomplished through fecal flotation or transparent tape slide test of the patient's anal area. Within the body, *S. obvelata* are generally found in the cecum and, to a lesser extent, the colon, and may be associated with signs of mild enteritis. The recommended treatment for hamster pinworms is fenbendazole 20 to 50 mg/kg orally once a day for 5 days or ivermectin 2 mg/kg topically once.[97] As eggs are resistant to desiccation, a rigorous environmental decontamination is required. Although, pinworm eggs may be resistant to common disinfectants, they are susceptible to high environmental temperatures.

Hymenolepis nana is the primary cestode (dwarf tapeworm) found in the small intestine of rats, mice, and hamsters, and has zoonotic implications. This common tapeworm has both direct and indirect life cycles. Clinical signs associated with heavy infestations include poor weight gain, abdominal distention, and diarrhea. As with other rodent cestodes, a definitive diagnosis is made through fecal flotation. *H. nana* can be treated with praziquantel (5 mg/kg orally or subcutaneously, every 10 days). Another cestode, *Hymenolepis diminuta*, also infects hamsters and may be diagnosed using the same methods.

MICE

Spironucleus muris, a flagellated protozoa, is frequently isolated from mice, rats, gerbils, and hamsters. The infection generally results in clinical disease in young animals (3 to 6 weeks) when there are predisposing immunocompromising factors. The clinical signs can include depression, weight loss, dehydration, diarrhea, and death. The recommended treatment protocol for *S. muris* is metronidazole (2 treatments of 10 to 40 mg/kg orally, 5 days apart).[98]

Gastric yeast appears to be an incidental GI microbe in mice, hamsters, guinea pigs, gerbils, chinchillas, and rats,[96] and the morphology of the yeast organisms within the gastric sections is consistent with *Candida albicans*. However, there have been reports of extensive gastric candidiasis with mortality in immunocompromised mice. The typical filamentous structures, with pseudohyphae formation, of a severe pathogenic yeast infection are readily visualized with periodic acid-Schiff (PAS) or silver stains. Contaminated bedding is a possible source for a variety of fungal agents. The recommended treatment for *C. albicans* is nystatin 60,000 to 90,000 IU/kg orally, twice a day for 7 to 10 days.

Several *Eimeria* species have been identified as a primary cause of murine intestinal infections. *Eimeria falciformis* is the coccidial organism most often isolated from mice with intestinal infections. The recommended treatment for coccidiosis in mice is toltrazuril 0.5% at 10 to 20 mg/kg orally given for 3 days, pause for 5 days, and then repeat once more.[98]

When treating other murine endoparasites and ectoparasites, ivermectin was found to be efficient. Ivermectin can be formulated as a compounded spray (ivermectin 1% in propylene glycol dilutes 10-fold in distilled water—shake well before using), where it is sprayed over the entire cage area including the animal, substrate, and cage wall.[99,100] The compounded ivermectin spray has been effective in treating fur mites (*Myobia musculi* and *Myocoptes* spp.) and pinworms (*S. obvelata* and *Aspiculuris tetraptera*).

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