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Ferret Gastroenterology

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KEYWORDS

• Zoonosis • Foreign body • Gastritis • Reflux • Regurgitation • Vomitus • Diarrhea • Melena

KEY POINTS

- Clinical signs of gastrointestinal disease in ferrets are often nonspecific and may include lethargy, anorexia, vomiting, and diarrhea. A systematic history-taking and work-up is required to make a diagnosis.
- Ferrets are notorious for ingesting foreign bodies. While gastric and esophageal foreign bodies often can be removed with flexible endoscopy, exploratory surgery is often indicated for intestinal foreign bodies.
- Enteritis in adult ferrets is often bacterial in origin and caused by feeding raw, uncooked diets.
- Hypoglycemia, hypovolemia, hypothermia, and anemia are not uncommon findings in ferrets presenting with gastrointestinal disease, and fast and adequate correction of these is vital.

INTRODUCTION

Spontaneous gastrointestinal (GI) tract diseases are common in ferrets (*Mustela putorius furo*), and as a result, ferrets have been used extensively as animal models for human GI tract diseases like reflux, ulcers, and neoplasia.[1] Despite the large variation in etiology, GI disease can be difficult to differentiate on initial presentation. Hence, a thorough history, physical examination, and systematic diagnostic approach are the key to obtaining a correct diagnosis. This review discusses the general work-up for ferret GI disease, including history taking, physical examination, and diagnostic tests. Furthermore, specific GI tract pathologies are described in oral to aboral direction, including their diagnosis and therapy. Additionally, the review will provide a short

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Abbreviations	
ALT	alanine aminotransferase
AP	alkaline phosphatase
AST	aspartate aminotransferase
CDV	canine distemper virus
CT	computed tomography
ECE	epizoonotic catarrhal enteritis
ELISA	enzyme-linked immunosorbent assay
FECV	ferret enteric coronavirus
GI	gastroinestinal
IBD	inflammatory bowel disease
PCR	polymerase chain reaction
SARS-CoV-2	severe acute respiratory syndrome coronavirus 2

overview of the anatomy and physiology of the digestive tract and its key features, as these are instrumental for the correct interpretation of clinical and diagnostic findings.

FERRET GASTROINTESTINAL ANATOMY AND PHYSIOLOGY

Ferrets are obligate carnivores with a short, simple digestive tract. A rapid GI transit time (1–8 hours, average 3–4 hours) combined with a small body weight, make ferrets prone to hypoglycemia with prolonged feeding regimens or vomitus.[2,3] GI tract motility is affected by vagal tone, which becomes clinically important when manipulation during tracheal intubation inadvertently stimulates the vagal nerve and decreases GI motility.[4]

Adult ferrets have a typical carnivore dentition with 34 brachydont teeth; 2(3/3, 1/1, 3/3, 1/2; **Fig. 1**).[5,6] Well-developed masticatory muscles provide the ferret with a powerful bite, grasping the prey tightly. The tongue is long, free, and mobile, and with a predilection site for penetrating grass awns in working ferrets. Five pairs of major salivary glands open into the oral cavity—parotid, submandibular, sublingual, molar, and zygomatic.[7] Fights between hobs can cause mucoceles, when bite wounds damage the salivary gland or ducts. The stomach is simple but has considerable storage capacity (~50 mL/kg). Unlike many other small mammals, the ferret lower esophageal sphincter allows for reflux and emesis. The small intestinal lumen is narrow, increasing the risk of foreign body entrapment. The remaining small intestine cannot be distinguished and passes directly into the ascending colon since neither an ileocolic junction or cecum is present.[8] The colon is approximately 7 cm in total, and divided into an ascending, transverse, and descending part.[9,10] The rectum and anus are 2 and 1 cm long, respectively. An internal sphincter of smooth muscle is followed by an external voluntary sphincter of striated muscle that covers the anal sac ducts, which open at approximately 4 and 8 o'clock positions with the ferret in dorsal recumbency.[11]

DIAGNOSING GASTROINTESTINAL DISEASE

History, Physical Examination, and Clinical Signs

Clinical signs of GI disease are often unspecific and may include anorexia, weight loss, lethargy, dehydration, and nausea (see also **Table 1**). Other clinical signs that are more specific, although not pathognomonic, to GI disease are regurgitation, vomiting, diarrhea, and melena.[12] A detailed clinical history should be obtained, with emphasis on the duration and progression of clinical signs, appetite, stool appearance and defecation, vomitus/regurgitation, housing, nutrition, contact with other ferrets, and possible

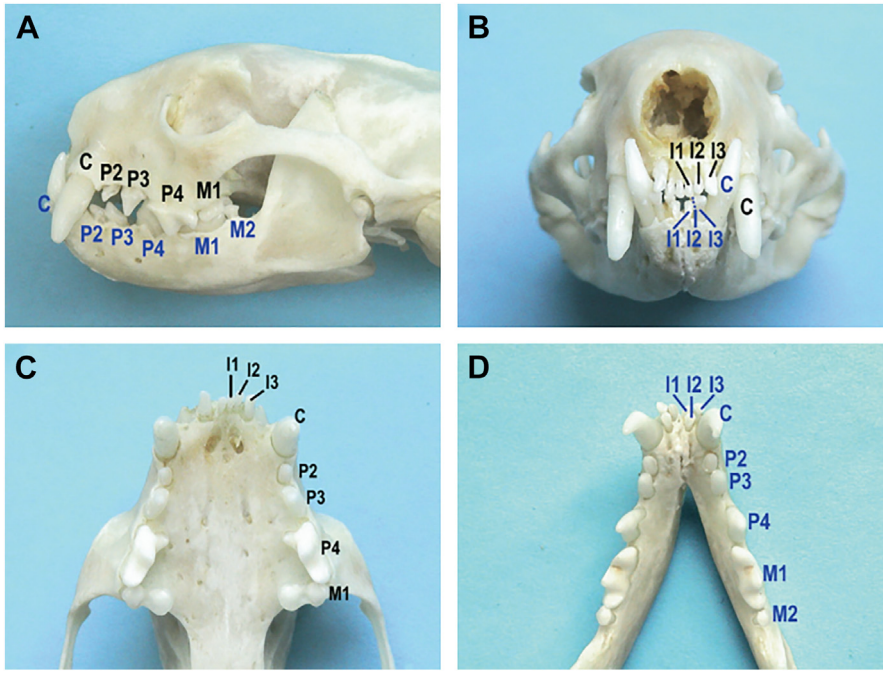

Fig. 1. Dentition of the ferret. Incisives (I), canines (C), premolars (P), molars (M) and their respective numbers are labeled. Note a number of dental abnormalities in this specimen, including malalignment of the incisors and blunting of the maxillary canine teeth. (*A*) Close-up of the dental arcades, lateral view. (*B*) Close-up of the dental arcades, rostral view. (*C*) Close-up of the maxillary arcades. Note the large carnassial tooth (fourth premolar or CT3) and the buccolingual direction of the molar tooth. (*D*) Close-up of the mandibular arcades. Note the large carnassial tooth (first molar or CT4) and that the second mandibular incisors are set back from the others. (*Courtesy of* Vittorio Capello, DVM. Previously published in "Johnson-Delaney CA. Diagnosis and treatment of dental disease in ferrets. J Exot Pet Med 2008;17(2):132–7".)

ingestion of toxins, drugs, or foreign bodies, which will greatly aid in the planning of any further workup and establishing a (differential) diagnosis. Asking the client to bring images or videos of excreted fluids and vomiting process can provide valuable information on color, consistency, and possible admixtures (eg, blood) in stool and/or vomit, as well as aid in differentiating regurgitation from vomiting, which can be challenging for clients as they may confuse reflux with gagging or coughing. Additionally, owners may not recognize pawing at the mouth as a sign of nausea.[13]

Due to their small body size, anorexic ferrets quickly become hypoglycemic, dehydrated, and hypothermic, especially if combined with fluid loss. Additionally, (prolonged) anorexia, vomiting, and/or diarrhea can lead to weight loss, muscle wasting, and depletion of fat reserves as a result of a negative energy balance. To discern whether aforementioned complicating sequela are present, a thorough physical examination, including at minimum an evaluation of mentation and alertness, body condition, posture, hydration status (skin turgor), pulse (frequency [200–400 bpm] quality), mucus membranes (color, moistness), capillary refill time, and body temperature (reference 37.8°–40°C [100–104°F]) will be needed.[14] Additionally, the physical examination should include a detailed examination of the digestive tract, including an

Table 1
Differential diagnosis, diagnostic work-up, and treatment options for common gastrointestinal disease presentations in ferrets

Clinical Sign	Differential Diagnoses	Diagnostic Work-Up Options	Treatment Options
Acute vomiting	GI foreign body Intoxication (food-related or other) Gastritis/gastroenteritis (eg, viral—coronavirus; bacterial—*Salmonella, E. coli*) Gastrointestinal (GI) obstruction/ileus Acute pancreatitis Acute renal failure Acute hepatitis Increased intracranial pressure (eg, head injury, meningitis) Motion sickness/vertigo Diabetic ketoacidosis (rare in ferrets) Sepsis, septic peritonitis	History (r/o toxicity, injury) Hematology and biochemistry (lipase, amylase, liver enzymes, bile acids, glucose, ketones, blood urea nitrogen [BUN], Ca, P, Na, K) Testing for specific pathogens Whole body radiographs Ultrasound Computed tomography Endoscopy (incl biopsy collection)	Anti-emetics: metoclopramide (Do not use in case of ileus/obstruction), maropitant Activated charcoal (in case of recent toxin ingestion) Laxatives (eg, lactulose) in case of trichobezoar Gastric protectants (eg, sucralfate) Antacids (eg, omeprazole, ranitidine) Antimicrobial therapy, for example, amoxicillin-clavulanic acid + metronidazole Nonsteroidal antiinflammatory drugs (meloxicam; CAVE: do not use in dehydrated patients or those with [suspected] gastrointestinal ulcers!) Assisted feeding Fluid therapy (if dehydrated, or diuresis is warranted eg, suspected intoxication or renal failure) Explorative laparotomy Endoscopic foreign body retrieval
Chronic vomiting	GI foreign body Gastric ulceration *Helicobacter mustelae* gastritis Gastritis/gastroenteritis Gastroparesis/delayed gastric emptying (eg, pyloric obstruction) GI neoplasia Azotemia (chronic renal insufficiency,	History Hematology and biochemistry (BUN, Ca, P, Ht, WBC, glucose, liver enzymes, bile acids, amylase, lipase, total protein, albumin, globulins, Na, K) (Contrast) radiography Fluoroscopy Ultrasound	Anti-emetics/prokinetics: metoclopramide, maropitant Antacids: omeprazole, ranitidine/cimetidine Gastric protectants, for example, sucralfate Antimicrobial therapy, for example, amoxicillin-clavulanic acid +

	Addisonian' s disease) Hypoglycemia Chronic hepatitis/hepatopathy Hypercalcemia Drug-related Food allergy/hypersensitivity Pyometra Chronic pancreatitis Central nervous system disorders leading to increased intracranial pressure (eg, meningitis)	Computed Tomography Endoscopy PCR (gastric/duodenal biopsies, feces) Histopathology (silver stain) Elimination and challenge diet	metronidazole Nonsteroidal antiinflammatory drugs Iron supplements Assisted feeding Fluid therapy (forced diuresis) Endoscopic removal of foreign bodies Explorative laparotomy Chemotherapy
Regurgitation	Megaesophagus Hiatal herniation[a] Esophageal stricture[a] Myasthenia gravis Esophageal foreign body Esophageal neoplasia Gastric reflux	(Contrast) radiography Fluoroscopy Computed tomography Endoscopy Tensilon test Serology - Antibody titer for Myasthenia gravis (San Diego, California, USA)	Feed at higher position and maintain animal in vertical position for 10–15 min after a meal Prokinetics: metoclopramide Antacids: omeprazole, ranitidine/cimetidine Gastric protectants: sucralfate Endoscopic removal or surgical intervention
Diarrhea	Stress Foreign body ingestion (rubber, foam, plastic, other) Trichobezoar Bacterial infection (primary or secondary, eg, *Salmonella* spp *E. coli, Clostridium* spp, *Campylobacter jejuni, Mycobacterium* spp.) Proliferative bowel disease (*Lawsonia, Desulfovibrio)* Viral infection (eg, rotavirus, FECV, canine distemper) Parasitic disease: coccidiosis, giardiasis Neoplasia (lymphoma most common) Inflammatory bowel disease Food hypersensitivity/food intolerance	Signalment History Abdominal palpation Culture and sensitivity Fecal flotation, wet mount Cytology Elimination diet (Contrast) radiographs Ultrasound Endoscopy Histopathology of biopsies PCR testing (eg, *Mycobacterium* spp, FECV, rotavirus)	Modify living conditions Dietary adjustments, hypoallergenic diet Laxatives (eg, lactulose) Antimicrobial therapy, preferably based on culture and sensitivity results Antiparasitics: metronidazole, paromomycin, toltrazuril Antiinflammatories/immune suppressants (glucocorticoids) Assisted feeding (if anorectic) Fluid therapy (ORS, SC/IV/IO) Endoscopic retrieval of foreign objects Explorative laparotomy Chemotherapy

(*continued on next page*)

Table 1
(*continued*)

Clinical Sign	Differential Diagnoses	Diagnostic Work-Up Options	Treatment Options
Melena (confirm erythrocytes in the stool with a fecal occult blood test)	Gastritis Gastric or duodenal ulceration (eg, due to *Helicobacter mustelae,* renal azotemia) Gastric/intestinal neoplasia/polyp(s) Pancreatitis Parasitic infestation (eg, giardiasis, coccidiosis or cryptosporidiosis in young ferrets) Ulcerogenic medication Rodenticide poisoning Dysbiosis (eg, *Salmonella* spp) Sepsis	Complete history (drugs, known ingestion of toxins/foreign bodies) Fecal flotation, wet mount Hematology/biochemistry (coagulation profile) Whole body radiography Endoscopy Histopathology of biopsies	Discontinue medication Vitamin K (injections, oral) Antimicrobial therapy, preferably based on culture and sensitivity results; amoxicillin-clavulanic acid + metronidazole for *Helicobacter* Antiparasitics: metronidazole, paromomycin, toltrazuril Gastric protectants: sucralfate Antacids: omeprazole, ranitidine/cimetidine Antiinflammatories Iron supplements Fluid therapy Activated charcoal
Hematochezia (with formed or soft/diarrheic feces+/− mucus)	Constipation, too firm/dry feces Passage of foreign material Parasitic disease (giardiasis, coccidiosis, whip/hookworms) Bacterial colitis (eg, *Clostridium perfringens/difficile, Salmonella* spp*, Campylobacter jejuni)* Fungal colitis[a] Inflammatory bowel disease Perianal fistula Anal sac disease (infection or impaction) Coagulopathy (thrombocytopenia, thrombocytopathy, rodenticide intoxication) Rectal polyps, neoplasia, other rectal/anal growths Infiltrative neoplasia (primarily lymphoma)	Complete history (drugs, known ingestion of toxins/foreign bodies) Complete blood count (CBC), coagulation profile Fecal flotation, wet mount Culture and sensitivity FNA for cytology Radiographs Ultrasound Colonoscopy/proctoscopy Biopsy for histopathology	Dietary adjustments, hypoallergenic diet Laxatives (eg, lactulose) Cisapride (only if no physical obstruction present) Antimicrobial therapy (eg, metronidazole, sulfasalazine, tylosin), preferably based on culture and sensitivity results, where possible Antiparasitics (eg, fenbendazole, ronidazole, sulfonamides) Immunosuppressant drugs (eg, cyclosporin A, glucocorticoids) Chemotherapy Cryotherapy, laser therapy Surgical intervention

Dyschezia and tenesmus	Colonic disease (parasitism including coccidiosis, bacterial colitis, inflammatory bowel disease, stress colitis, food hypersensitivity, neoplasia, megacolon) Rectal/anal disease (eg, perianal fistula/hernia, rectal prolaps/stricture/polyp, proctitis, anal sacculitis, rectal/anal neoplasia) Prostatic disease (eg, related to hyperadrenocorticism) Pelvic fracture, other trauma Dysuria/stranguria Dystocia	History Fecal flotation, wet mount Culture and sensitivity CBC and biochemistry (frequently unremarkable) FNA for cytology Radiography Ultrasound Colonoscopy/proctoscopy Biopsy for histopathology Computed tomography	Laxatives (eg, lactulose) Prokinetics (cisapride, only if no mechanical obstruction is present) Antiparasitics (eg, sulfonamides) Antimicrobial therapy, preferably based on culture and sensitivity Antiinflammatory drugs (eg, sulfasalazine, prednisolone) Immune modulatory drugs (cyclosporine A) Chemotherapy Cryotherapy, laser therapy Surgical intervention

Abbreviations: BUN, blood urea nitrogen; Ca, calcium; FECV, ferret enteric coronavirus; FNA, fine needle aspiration; Ht, hematocrit; IO, intraosseus; IV, intravenous; K, potassium; Na, sodium; ORS, oral rehydration salts; P, phosphate; PCR, polymerase chain reaction; r/o, rule out; SC, subcutaneous; WBC, white blood cell count.

[a] Not previously diagnosed in ferrets.

evaluation of the oral cavity (including teeth, tongue), neck (esophageal or pharyngeal masses), abdomen (inspection, superficial and deep palpation, undulation), and perineal area. In case of suspected oropharyngeal pathology, a more thorough evaluation of these structures under sedation or anesthesia is warranted (Fig. 2).

Diagnostic Workup

The work-up for diagnosing GI disease in ferrets is mostly similar to that in dogs and cats, and may include a fecal examination, microbiological culture and sensitivity testing, hematology and biochemistry, whole body radiographs (with or without contrast), ultrasonography, computed tomography (CT), and/or endoscopy with biopsy collection for histopathology (see Table 1).

Fecal Examination: Screening for Pathogens, Parasitology, Culture and Sensitivity, and Fecal Occult Blood Test

A fecal examination is a relatively simple and inexpensive, yet a valuable tool that can be employed to identify the cause of gastrointestinal problems observed in a ferret. While parasitic diseases are relatively uncommon in ferrets, routine parasite testing should be performed in any ferret with diarrhea, hematochezia, or melena to exclude helminthiasis (eg, nematodiasis due to infestation with *Toxacara cati, Toxascaris leonina, Ancylostoma* spp, or cestodiasis due to infestation with *Mesocestoides* spp, *Dipylidium caninum*), or protozoal disease (eg, giardiasis, coccidiosis, cryptosporidiosis [particularly in young ferrets]). Such testing should include both a direct smear of fresh fecal material and flotation.

Microbiological culture and sensitivity of the stool can be useful to identify primary or secondary bacterial pathogens. For certain pathogens (eg, *Salmonella* spp.), the use of specific culture media or multiple-day testing (to increase the chance of detecting a pathogen that is shed intermittently) are recommended. Specific polymerase chain reaction (PCR) tests are also available to identify presence of ferret enteric coronavirus (FECV), canine distemper, rotavirus, *Helicobacter mustelae* (which is common in ferrets but difficult to culture), *Salmonella* spp, and *Mycobacterium* spp.

Fig. 2. Oral inspection in a ferret. Ferrets often open their mouths when being scruffed. If the oral cavity cannot be inspected sufficiently, sedation is needed for a more thorough inspection.

Some gastrointestinal diseases (eg, *Helicobacter*-related gastritis) can lead to hematochezia, but this may not be macroscopically visible. In such cases, fecal occult blood testing is helpful to confirm the presence of blood. It should be remembered that a single negative occult fecal blood test does not rule out gastrointestinal hemorrhage, as bleeding may be intermittent. Additionally, the ferret should be placed on a nonmeat diet (eg, Emeraid carnivore, Oxbow carnivore care) for at least 24 to 36 hours to avoid false-positive test results due to dietary heme.[12]

Hematology and Biochemistry

In many ferrets with GI disease, hematologic and biochemical changes will be reflective of dehydration, anorexia, and wasting that have occurred due to ongoing disease. Nevertheless, a complete blood count and chemistry panel can help to provide clues to diagnose and assess the overall clinical condition of the patient. If larger volumes are needed, the jugular vein or cranial vena cava can be used (sedation or anesthesia is recommended), while the cephalic or saphenous veins can be sampled in case smaller volumes are required or if clotting disorders are suspected. In healthy ferrets, approximately 10% of the total blood volume (4–6 mL) can be safely collected, but if anemia and/or hypovolemia are present or the animal is critically ill, it is safer to limit this volume to 5% (2–3 mL). With repeated samplings, the total blood volume sampled ideally is recorded to avoid iatrogenic anemia.

Anemia is commonly found in ferrets with severe GI bleeding or chronic inflammation. Evaluation of the reticulocyte count can help to differentiate regenerative (eg, in case of bleeding) from nonregenerative anemia (eg, in case of chronic inflammation). Similar to dogs and cats, the mean cell volume, mean cell hemoglobin, and mean cell hemoglobin concentration can be used to gain further insight into the size of the erythrocytes and their hemoglobin concentration, which can be helpful to discern between the various causes of anemia. Leukocyte counts can be decreased in case of acute infections, while moderate leukocytosis is often present with chronic infections Neutrophilia and/or monocytosis may also be observed.

In ferrets, alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (AP), bilirubin, and bile acids can be used to assess liver function. Increases in AST and ALT can also be observed in animals with anorexia or primary intestinal disease. To evaluate kidney function, blood urea nitrogen, calcium, phosphate, potassium, and sodium can be evaluated. Creatinine has a very low sensitivity in ferrets and may remain normal in animals with severe kidney damage. Parameters used in many other species for diagnosing exocrine pancreatic insufficiency or pancreatitis are not properly established in ferrets. However, an increase in lipase (reference: 73–351 U/l) or amylase (reference: 19–62 U/l) can be associated with pancreatitis.[14] Glucose values <72 mg/dL (<4 mmol/L) in animals that have been fasted for 4 to 6 hours are highly suggestive of an insulinoma, although differentials may include severe septicemia, cachexia, or liver failure. Hypoproteinemia is frequently diagnosed in ferrets with GI disease and should ideally be corrected prior to anesthesia and surgical intervention (**Table 2**).[15] Elevated total protein can be seen in animals with acute or chronic inflammation, dehydration, liver disease, or lymphoma.

Diagnostic Imaging

Radiography can be useful in the diagnosis of GI disease. Survey radiographs can often reveal evidence of GI masses, thickened bowel loops, or abnormal gas patterns suggestive of foreign body obstruction or enteritis. Patency of the GI tract and transit time, megaesophagus, and obstructions are easily diagnosed with serial contrast

Table 2
Supportive care and medications commonly used in gastrointestinal disease in ferrets

	Product/Medication	Dose
Nutritional support (syringe-fed or via a nasogastric, esophageal or duodenojejunostomy tube)	Emeraid carnivore Oxbow critical care carnivore Royal Canine Recovery Liquid Trovet Recovery Liquid	Calculate a minimum daily intake of 400 kcal/kg BW, and divide into 4–6 feedings/day
Fluid therapy	Crystalloids, for example, lactated Ringer's	Replacement fluid volume (mL) with crystallloids = Estimated dehydration deficit (%) × body weight (kg) × 1000; maintenance = 70 mL/kg/day; PO/SC/IV/IO
	Colloids, for example, Hetastarch	3–5 mL/kg over 5 min IV/IO (for patients that are severely hypovolemic or hypoproteinemic)
	Glucose 5%, dextrose 2.5%–5%	0.25–2 mL IV (for hypoglycemia)
	Blood	6–12 mL/kg/h IV/IO (transfusion may be needed for anemic patients, PCV<25%); mix 1 mL of anticoagulant (citrate) with 6 mL of donor blood
Nutritional supplements	Cobalamin	25 μg/kg q7d SC × 6 wk, then q14 d SC × 6 wk (chronic diarrhea, with cobalamin malabsorption)
	Glutamine	0.5 g/kg q24 h PO (enterocyte supplementation with starvation)
	Iron dextran	10 mg/animal IM once (iron deficiency anemia)
	Pet-Tinic	0.2 mL/kg q24 h PO (iron deficiency anemia)
	Vitamin B12	½ capsule Cobalaplex q24 h PO
	Vitamin K	2.5–5 mg/kg q8–12 h SC/PO
Antacids/proton pump inhibitors	Cimetidine	5–10 mg/kg q6–8 h PO/SC/IM/IV
	Famotidine	0.25–2.5 mg/kg q24 h PO/SC/IV
	Omeprazole	1 mg/kg q12–24 h PO
	Ranitidine	2-5-5 mg/kg q12 h PO
Anti-diarrheal drugs	Loperamide	0.2 mg/kg q12 h PO

Antiemetics	Maropitant	1 mg/kg q24 h PO/IM
	Metoclopramide	0.2–1 mg/kg q6–12 h SC/PO/IV
	Ondansetron	0.5–1 mg/kg q12–24 h IM/IV/PO
Emetics	Apomorphine	0.04–0.2 mg/kg SC
	Ipecac (7%)	2.2–6.6 mL/animal PO
Gastrointestinal protectants	Bismuth subcitrate	6 mg/kg q12 h PO
	Bismuth subsalicylate	17.5 mg/kg q8 h PO
	Kaolin/pectin	1–2 mL/kg q2–6 h PO
	Sucralfate	25–125 mg/kg q6–12 h PO (give before meal, not simultaneous with other medication)
Laxatives	Feline hairball laxative	1–2 mL/animal q48 h PO
	Lactulose	0.15–0.75 mL/kg q12 h PO
Motility enhancers	Cisapride	0.5 mg/kg q8–12 h PO
	Metoclopramide	0.2–1 mg/kg q6–12 h SC/PO/IV
Antiparasitic drugs	Fenbendazole	20 mg/kg q24 h PO × 5 d (nematodiasis)
	Metronidazole	15–20 mg/kg q12 h PO × 5–14 d (gastrointestinal protozoa, eg, *Giardia*)
	Nitazoxanide	
	Paromomycin	5 mg/kg q12 h PO (cryptosporidiosis)
	Piperazine citrate	165 mg/kg q12 h PO × 5 d (cryptosporidiosis)
	Ponazuril	50–100 mg/kg q14 d PO (nematodiasis)
	Praziquantel	30–50 mg/kg q24 h PO × 3–7 d (coccidiosis)
	Sulfamethoxine	5–10 mg/kg PO/SC, repeat in 10 d (cestodiasis)
	Toltrazuril	25 mg/kg q24 h PO × 21 d (coccidiosis)
		20 mg/kg q24 h PO × 3–7 d (coccidiosis)
Antimicrobial agents	Amoxicillin + clavulanic acid	12.5–25 mg/kg q8–12 h PO
	Azithromycin	5 mg/kg q24 h PO
	Chloramphenicol	25–50 mg/kg q12 h PO/SC/IM/IV (14 d minimum for proliferative bowel disease)
	Clarithromycin	
	Enrofloxacine	12.5 mg/kg q8–12 h PO
	Gentamycin	5–10 mg/kg q12 h PO/SC/IM
	Metronidazole	2–5 mg/kg q12–24 h PO/SC/IM/IV
	Trimethoprim/sulfa	15–20 mg/kg q12 h PO
	Tylosin	15–30 mg/kg q12 h PO/SC
		5–10 mg/kg q12 h PO/SC/IM/IV

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Table 2
(*continued*)

	Product/Medication	Dose
Analgesics	Buprenorphine	0.01–0.05 mg/kg q6–12h transmucosal/SC/IM/IV
	Butorphanol	0.05–0.5 mg/kg q8-–12 h SC/IM (may cause sedation in higher dosages)
	Carprofen	1–5 mg/g q12–24 h PO (use with caution in dehydrated animals or those with gastritis/enteritis)
	Lidocaine	2 mg/kg q12–24 h topically
	Meloxicam	0.1–0.3 mg/kg q24 h PO/SC/IM (use with caution in dehydrated animals or those with gastritis/enteritis)
	Tramadol	5–10 mg/kg q12–24 h PO
Immune-modulating drugs	Azathioprine	0.9 mg/kg q24–7 2 h PO (chronic IBD, combine with prednisone)
	Cyclosporine	4–6 mg/kg q12 h PO
	Prednisone	0.5–2 mg/kg q12–24 h PO
	Sulfasalazine	62.5–125 mg/kg q8–12 h PO (colitis)
Miscellaneous	Activated charcoal	1–3 g/kg PO (adsorbent for gastrointestinal tract toxins/drug overdose)
	Cyproheptadine	0.5 mg/kg q12 h PO (appetite stimulant)
	Pyridostigmine	1 mg/kg q8–12 h PO (myasthenia gravis)

Abbreviations: IBD, inflammatory bowel disease; IM, intramuscular; IO, intraosseus; IV, intravenous; PCV, packed cell volume; PO, per os, orally; SC, subcutaneous.

radiographs (8–13 mL/kg, barium sulfate).[12] However, contrast should not be used in a sedated or anesthetized ferret without intubation as ferrets are likely to regurgitate and aspirate. Additionally, contrast agents such as barium can be damaging to the endoscopic equipment and should be avoided if endoscopy is planned.

Similar to dogs and cats, abdominal ultrasonography and CT can be extremely helpful for detailed visualization and evaluation of the size, shape, and density of the different parts of the GI tract and surrounding organs. Of the 2 techniques, ultrasound is cheaper and more readily available in clinical practice, but its sensitivity is highly operator dependent.[16] Additionally, newer technologies such as CT scans have become more affordable and available in private practices. Moreover, the disadvantage of the higher cost associated with CT scans is quickly outweighed by the fact that scans can be sent for external evaluation by a veterinary radiologist, if in doubt. Nevertheless, each technique has its merit for evaluating GI disease, as ultrasound is better at visualizing the individual intestinal layers, tissue blood flow, GI motility, and movement of chymus, while CT is superior at visualizing GI diameter, dilations, gastric trichobezoars, and foreign bodies.[15,17]

Endoscopy and Biopsy Collection for Histopathology

Despite their narrow diameter, the esophagus, stomach, duodenum, and upper jejunum are reachable by semiflexible or flexible endoscopy, allowing direct visualization of the intestinal lining and aiding in the diagnosis of, for example, gastric ulcers (**Fig. 3**). Similarly, colonoscopy is useful for visualization and obtaining biopsies during the diagnostic work up of chronic diarrhea, hematochezia on formed feces, or severe constipation. Because of the short large intestine, colonoscopy can be further advanced to a jejunoileoscopy. Flexible gastroscopy is commonly performed using a 100 cm long, 3 mm diameter bronchofiber flexible endoscope with a working channel of 1.2 mm, which is valuable for foreign body retrieval and biopsy sampling of the mucosa to help confirm the underlying cause of GI disease.[18] Alternatively, exploratory laparotomy can be considered for biopsy collection, especially in case of increased risk of intestinal wall perforation (eg, in case of intraluminal masses).

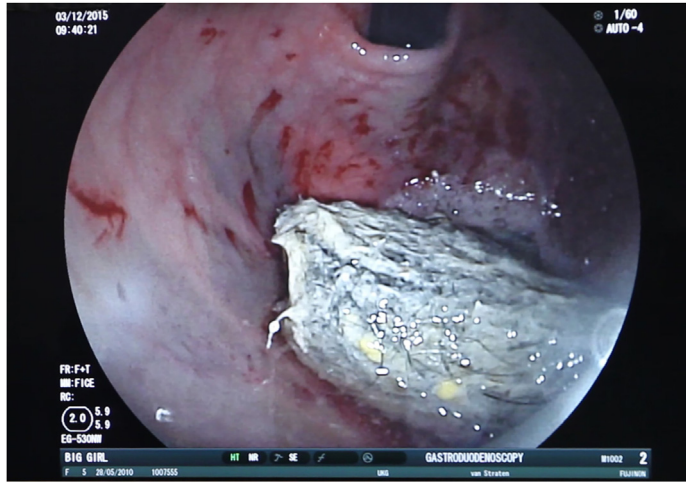

Fig. 3. Gastroscopic view of a hairball covered in sucralfate in a 5.5-year-old female chemically neutered ferret that was presented with chronic vomiting, anorexia, and weight loss.

Surgical biopsy also gives the added advantage of allowing visualization and targeting of grossly abnormal sections, and collection of full-thickness samples. Laparoscopy can be used for visualization and biopsy of the liver and/or pancreas via True-cut needle or simple clamshell or punch-style biopsy forceps.

For gastroduodenoscopy, a 4 to 6 hours fasting period will generally suffice, whereas colonoscopy can generally be accomplished following either a 12-hour fast or 8-hour provision of liquid carnivore diets (see **Table 2**) followed by a 4-hour fast. In fasted animals, sporadic monitoring of blood glucose may be considered to detect episodes of hypoglycemia, which should be treated prior to induction of anesthesia. During anesthesia, repeated boluses using a maximum of 20 mL/kg of warmed saline is introduced into the large intestine using a red rubber tube or large diameter urinary catheter, to empty remaining fecal content before commencing with the colonoscopy.[18]

SPECIFIC GASTROINTESTINAL DISEASE CONDITIONS

Dental Disease

Dental disease is common in ferrets, with a presentation, pathogenesis, and treatment similar to dental disease in dogs and cats.[19] Due to the high prevalence of acquired dental disease, a thorough inspection of the oral cavity is considered an important part of every physical examination, particularly in adult to older ferrets and patients that are hyporexic. Dental radiographs are helpful to exclude problems that are not readily visible during oral inspection.[20]

According to previous publications, common dental problems in ferrets include mild gingivitis (83.7%; **Fig. 4**), dental attrition (63%–72%), and fractures of the maxillary canines (30%–52%), while tooth resorption (21.9%) and severe periodontal disease (16.3%) are less commonly diagnosed.[20,21] Prevalence of malocclusion varies greatly among studies (24%–95%) with linguoversion of the secondary mandibular incisors being the most prevalent (93%).[20,21] Regardless of diet, dental calculus will gradually build up, leading to gingivitis, caries, periodontal disease, and eventually tooth root abscesses, if left untreated.[22] In rare instances, these abscesses may extend into the nasal cavity, leading to oronasal fistulas and unilateral nasal discharge.[23] To delay calculus build-up, daily tooth brushing is recommended, and often tolerated by ferrets if using meat flavored toothpaste.[24]

Oral Ulcerations and Neoplasia

Ferrets with oral ulcerations commonly present with pain, hypersalivation, nausea, anorexia, gagging, or pawing at the roof of the mouth. Such ulcerations can be

Fig. 4. Gingivitis and mild build-up of tartar seen around and on the maxillary teeth in a 6-year-old neutered female ferret that was presented for a routine check-up.

secondary to systemic or renal disease, gastric reflux, neoplasia, electrocution injuries, dental disease, or ingestion of caustic solutions. Ulcers can be directly visualized on the oral examination, and be present at any location.[6] Those associated with systemic disease are commonly found on the buccal mucosa, while those caused by ingestion of caustic substances are predominantly found on the rostral part of the tongue and mucosal lining.[25] A full work up including a detailed history, oral examination, bloodwork, urine analysis, and diagnostic imaging can aid in identifying the initiating cause.

Once the inciting cause is treated, ulcers will generally heal by themselves, but meanwhile alleviation of pain is recommended using analgesics such as tramadol, butorphanol, and/or nonsteroidal antiinflammatory drugs (NSAIDs). Additionally, oral liquid sucralfate can be provided, in addition to a soft to liquid diet to facilitate food intake.[26] For ferrets that are too painful and unwilling to eat by themselves, a nasogastric or esophageal tube can be placed to ensure adequate food intake while the oral lesions are allowed to heal. If left untreated, palatal ulcerations may erode through the hard palate to form oronasal fistulas. Healing of these is often complicated and will generally require surgical intervention.[27]

Mucosal lymphoma can occur in ferrets in association with oral ulcerations. Additionally, solid tumors of either benign or malignant origin have also been reported in ferrets. Cytology and/or histopathology of oral swellings is mandatory to differentiate neoplasia from abscesses and will help to determine the prognosis and course of treatment. While local resection of benign tumors like epulis can be achieved, more radical surgery should be considered in case of squamous cell carcinoma, osteoma, or multilobular tumors.[6] Hemimandibulectomies and adjunct radiation therapy may be considered in case of nonresectable tumors.[6,28]

Salivary Mucocele

Mucoceles have been reported in the zygomatic salivary glands in ferrets.[29,30] Obstruction of the salivary ducts can result from trauma, salivary microliths, oral neoplasia, or severe dental disease. The impaired drainage leads to progressive facial swelling with a build-up of mucous or seromucous fluids depending on which of the 5 types of salivary glands are affected.[7] Exophthalmos might be present.[29] Aside from the swelling, the ferret is usually unaffected.[11] Salivary mucoceles are diagnosed based on aspiration of the characteristic thick, viscous to mucinous, clear to light hemorrhagic fluid. CT is useful in identifying the affected gland and planning the surgical approach, which involves surgical drainage, marsupialization, or complete surgical resection to reduce the risk of reoccurrence.[6,29]

ESOPHAGEAL DISORDERS

Megaesophagus

Megaesophagus refers to dilation and lack of muscular motility of the esophagus. Thus far, megaesophagus has only been described in adult ferrets. The disease etiology is largely unknown.[31–33] Dilation of the esophagus can either be segmental or generalized, caused by neuromuscular dysfunction leading to a hypomotility and failure to transport the esophageal content to the stomach.[34] Intrathoracic, especially mediastinal, space-occupying lesions like thymomas and lymphomas compressing the esophagus can mimic the signs of megaesophagus, and should be excluded with diagnostic imaging.[35] Because of the hypomotility and delayed emptying, ferrets with megaesophagus often regurgitate after eating, and commonly display difficulties with swallowing. This results in coughing, choking, hyporexia, and weight loss.

Common complications include aspiration pneumonia, malnutrition, and hepatic lipidosis.[32] Affected ferrets often have concurrent gastritis and reflux, while a case reported myasthenia gravis as an underlying cause.[31] The dilated esophagus is best diagnosed on contrast radiographs using barium, while problems with motility can be assessed by fluoroscopy.[12,13] Treatment is often unrewarding, with survival times commonly being less than a week post diagnosis, except in 1 ferret with myasthenia gravis, that responded to pyridostigmine bromide (1 mg/kg, PO, q8h) treatment for a month.[31–33] Treatment consists of feeding liquid food at elevated locations, followed by restraining the ferret in an upright position for 10 minutes after the meal, medical prevention of (esophageal) ulcerations, and treatment of concurrent bronchopneumonia and GI-ulcerations, if present.[32]

Esophageal Foreign Bodies

The curious nature of young ferrets makes them prone to foreign body ingestion. Especially rubber and rubber-like materials, like ear plugs, seem to be highly preferred.[12] Foreign bodies can lodge throughout the GI tract, resulting in a variety of clinical signs, dependent on the size of the foreign body, the location, and the degree of luminal occlusion. Most often esophageal foreign bodies are identified on survey radiographs in ferrets with acute anorexia, vomiting, gagging, and lethargy, but some cases may require fluoroscopy, CT, or endoscopy to establish a diagnosis (**Fig. 5**).[36] Foreign body retrieval may be achieved endoscopically, whereby the object may be removed retrograde, or pushed into the stomach to allow natural passage or removal by gastrotomy.[13,36,37] Failure to dislodge the esophageal foreign body from the esophagus from both an oral and aboral (gastrotomy) direction complicates the removal and requires an esophagostomy through a thoracotomy. Temporary placement of a duodenojejunostomy tube can be used to allow for enteral nutrition during the healing and recovery.[38]

Esophagitis and Esophageal Strictures

Esophagitis is a common sequela to foreign bodies and other medical conditions, like megaesophagus and eosinophilic gastroenteritis. Gastric acid reflux during regurgitation or vomiting leads to erythema, inflammation, and erosion of the esophageal mucosal lining. Esophagoscopy can be used to assess the degree of esophagitis

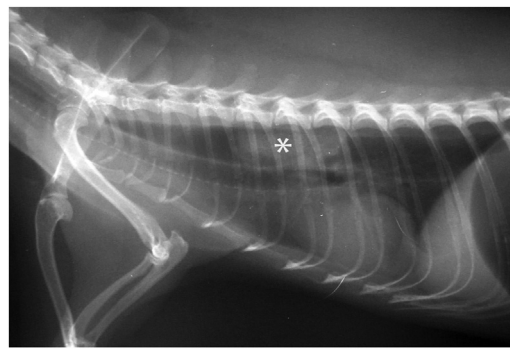

Fig. 5. Lateral projection of a radiograph of a 9-month-old intact male ferret that was presented with an acute onset of regurgitation directly after eating. A large foreign body can be seen craniodorsal to the heart (see *asterisk*). The foreign body proved to be a collapsed cat toy that could be removed endoscopically.

and presence of strictures.[18] Several reports on strictures exist, caused by chronic esophagitis, prolonged compression by a foreign body, or leakage of corrosive substances from such objects (eg, coin or button battery). Combined use of proton pump inhibitors, sucralfate, feeding of soft, liquefied foods, and surgical intervention with balloon dilation and stenting have shown moderate to good success in treating these conditions.[36,37,39,40]

DISORDERS OF THE STOMACH

Gastritis and Gastric Ulcerations

Signs associated with irritation of the gastric mucosa are generally mild and can include vomiting and hyporexia. A typical sign might be that the ferret shows interest in food, but once it starts to eat, it suddenly stops. If left untreated, gastritis may progress to gastric and duodenal ulceration or even perforation, with the ferret displaying hypersalivation, anorexia, weight loss, and teeth grinding as a result of abdominal pain, melena, anemia, and even death. Ferrets are sensitive to prolonged use of NSAIDs; hence, preventive use of gastroprotective drugs and/or antacids should be considered (see **Table 2**).[13] Renal azotemia and intoxications are other common causes, while neoplasia is rare. *H mustelae* is found in up to 100% of ferrets worldwide, and is commonly found in ferrets with gastritis, ulcerations, and even neoplasia (eg, gastric adenocarcinoma and mucosa-associated lymphoid tissue lymphoma).[41,42] However, many ferrets are subclinically infected, and disease seems mostly limited to individuals that are immunocompromised (eg, due to stress, crowding, other underlying disease). Hence, the relevance of the pathogen itself remains unclear. Radiographs are generally unremarkable, unless a gastric foreign body has caused gastric irritation or a pyloric perforation is present. Hematology may show varying levels of nonregenerative anemia, dependent on severity and chronicity of the bleeding. Gastroscopy is useful to assess the severity and distribution of gastric ulcerations (**Fig. 6**), and to obtain gastric and duodenal biopsies. *H mustelae* can be identified on histopathology using silver staining or following PCR analysis of biopsy specimens or fecal samples.[15] Blood serum immunoglobulin G enzyme-linked immunosorbent assay (ELISA) titers and a urea breath test can also be used to confirm *H mustelae*.[13] Nevertheless, due to the high prevalence of *H mustelae* in healthy ferrets, positive identification of the organism (without confirming its presence in lesions during histopathology) does not necessarily equal causation of clinical disease. Treatment is focused on gastroprotective drugs, antiemetics, supportive care, and antibiotics (see **Table 2**). In case of gastric neoplasia, due to the possible interaction with *H mustelae*, adjunct targeted antibiotic treatment is commonly advised in addition to chemotherapy.

Gastric Foreign Bodies and Gastric Distention

Ingested foreign bodies or trichobezoars are common in ferrets (see **Fig. 3**). Clinical signs are often nonspecific, with nausea, hypersalivation, hyporexia, and diarrhea. Vomiting is uncommon.[11,13] In rare cases, ferrets can present in lateral recumbency, and shock resulting from complete obstruction and associated gastric distention. Radiographs are diagnostic, and emergency treatment includes decompression of the stomach with an orogastric tube, treatment of hypovolemic shock, and stabilization of the patient prior to performing exploratory surgery.[13] Dependent on their nature, foreign bodies can easily be missed on initial survey radiographs, hence warranting 4 to 6 hours of fasting with follow-up radiographs in patients suspected of foreign body ingestion. Remaining gastric content at this point indicates delayed emptying

Fig. 6. Gastroscopy with a flexible endoscope in a 5.5-year-old female chemically neutered ferret that was presented with chronic vomiting, anorexia, and weight loss.

or entrapment of a foreign body. Additional techniques that can be used to assess gastric content include ultrasound and gastroscopy.[18] Endoscopic retrieval and/or surgical removal of a foreign body or trichobezoar may be attempted. Prevention is aimed at limiting exposure to rubber, foam, or plastic objects that the ferret can ingest. Additionally, frequent grooming and the use of cat laxatives are helpful to reduce the formation of trichobezoars.[43]

Neoplasia and Stenosis

Lymphoma is the most common gastric neoplasia in ferrets, while adenocarcinoma and neuroendocrine carcinomas are more rarely reported.[44] Both lymphoma and adenocarcinoma are suggested to coincide with *H mustelae* infection, as discussed previously.[41,42,45,46] Ferrets with gastric neoplasia commonly present with progressive lethargy, anorexia, vomiting, and weight loss (**Table 3**).[47,48] Radiographically, a cranial intraabdominal mass may be present, while ultrasonography may indicate thickening of the gastric mucosa or a nodular mass.[45,47] If the outflow of the pylorus is obstructed by a mass (or muscular hypertrophy), gastric distension may also be visible.[13,41,46–48] Gastric biopsies are required to obtain a definitive diagnosis. Samples can be obtained through endoscopy or laparotomy, and subsequently sent to a pathologist to help identify the tumor type and guide selection of the most appropriate chemotherapy protocol.[49] In case of confirmed pyloric neoplasia, euthanasia is generally

Table 3
Typical morphologic characteristics of diarrhea in ferrets caused by specific pathogens

Disease	Pathogen	Watery	Mucoid	Color	Hematochezia	Melena	Birdseed Like
Coccidiosis	*Isospora* spp, *Eimeria* spp				x	x	
Giardiasis	*Giardia intestinalis, Giardia duodenalis*						(x)
Cryptosporidiosis	*Cryptosporidium parvum*		x	Yellow			
Salmonellosis	*Salmonella* spp				x		
Campylobacteriosis	*Campylobacter jejuni, Campylobacter coli*	x	x	Green	x		
Proliferative bowel disease	*Lawsonia intracellularis*		x	Bright green	x		
Epizootic catarrhal enteritis	FECV		x	Bright green (acute stage)			x (chronic stage)
Parvo	Parvovirus					x	
Rotavirus diarrhea	Rotavirus		x	Green			
Canine distemper	Canine distemper virus					x	
Inflammatory bowel disease	N/A (immune mediated, possibly food intolerance/ hypersensitivity)	x					x
Eosinophilic gastroenteritis	N/A	(x)	(x)	Green/dark	(x)		

Abbreviation: N/A, not applicable.
x, common clinical presentation, (x), can be present.

recommended, especially if the animal is in a poor condition. In case of muscular hypertrophy, reconstruction of the pylorus can be attempted.[12]

INTESTINAL DISORDERS

Intestinal Parasitism

While intestinal parasites are not a common cause of clinical disease, newly acquired, young, immunocompromised, stressed individuals, or ferrets housed in unsanitary conditions or high stocking densities are at greater risk of infection. Initial work-up includes a direct smear and serial fecal flotations. Since ferrets are often asymptomatic, routine testing of a newly acquired ferret should be considered, especially in multiferret households.

Coccidiosis

Clinical signs of coccidiosis range from a mild to severe diarrhea, dependent on degree and extent of atrophic enteritis (**Table 3**). If infection is severe, additional signs may include dehydration, hematochezia or melena, rectal prolapse, weight loss, and even death.[50,51] While ferrets of all ages are susceptible, young and immunocompromised individuals are most frequently affected.[51,52] In a study, oocysts were identified on fecal flotation in 4.7% of submitted fecal samples (n = 132/2805), with *Cystoisospora* (*Isospora*) *laidlawi* being twice as prevalent as *Eimeria furonis*. The same study also found a markedly higher prevalence of coccidiosis in European compared to Canadian samples (13% and 3.5%, respectively).[50] Since oocysts are shed intermittently, pooled or serial samples are recommended (**Fig. 7**).[51] If coccidiosis remains a viable diagnosis despite negative flotations, PCR and identification of the parasite on intestinal biopsies can be used to confirm the diagnosis.[50,51] Prognosis is grave in severe cases, though symptomatic treatment (assisted feeding, fluid therapy, blood transfusions, supplemental heat, etc.; see **Table 2**) can be attempted in combination with sulfamethoxine or sulfadimethoxine, ponazuril, or toltrazuril. Of these, ponazuril and toltrazuril are coccidiocidal and thereby the drugs of choice as sulfamethoxine is coccidiostatic.[51,53] Biliary coccidiosis due to *E furonis* has been reported in ferrets, resulting in hyperplasia of the biliary tree, cholecystitis, and cholangiohepatitis. Treatment was unsuccessful in both cases.[53,54]

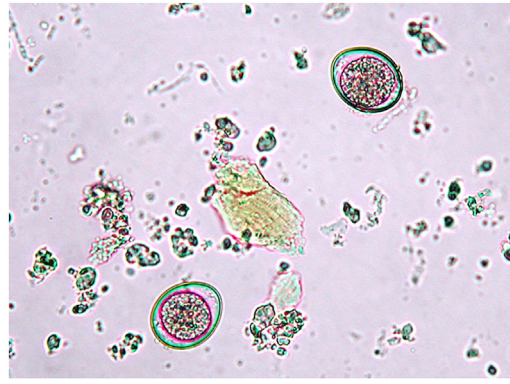

Fig. 7. Oocysts found after fecal flotation in a 4-month-old intact female ferret that was presented with persistent diarrhea.

Cryptosporidiosis

Similar to coccidia, cryptosporidia are single-celled protozoal organisms that can infest the gastrointestinal tract, predominantly the ileum, leading to chronic, watery to mucoid, yellow diarrhea (**Table 3**). In ferrets, cryptosporidiosis is most commonly associated with *Cryptosporidium parvum* which has zoonotic potential.[11] Ferrets of all ages can be affected, though the infection is usually subclinical in a large percentage of animals. Immunosuppression, caused by concurrent disease, suboptimal housing, or relocation, can escalate the diarrhea and result in anorexia, lethargy, and death occurring within 2 to 3 days after onset of clinical signs.[55,56] Affected animals excrete infective, sporulated oocysts in their feces, which can subsequently be identified in direct fresh fecal smears, following flotation, or using modified acid-fast staining or fluorescent antibodies. Alternative methods include the use of PCR, histopathology, or electron microscopy of intestinal biopsies.[55] A wide range of drugs (azithromycin, nitazoxanide, paromomycin) have been used to treat ferrets with cryptosporidiosis with variable success as continued autoinfection hinders successful eradication. In adult immunocompetent ferrets, the infection is usually self-limiting.

Giardiasis

Both *Giardia intestinalis* and *Giardia duodenalis* have been reported as a cause of giardiasis in ferrets.[57–59] *Giardia* spp infrequently cause anorexia and mild diarrhea in ferrets (**Table 3**), and may potentially be zoonotic, but rarely cause fatal disease in the absence of concurrent infections.[57–60] Fluorescent antibody coproscopy is considered the gold standard for diagnosing *Giardia,* but more commonly used methods include identification of motile flagellates or oocysts on fecal flotation (eg, Mini FLOTAC) with or without light microscopy or a positive *Giardia* coproantigen test.[61] In addition to symptomatic treatment, metronidazole can be used to eliminate infection (see **Table 2**).[55]

BACTERIAL GASTROENTERITIS

Feeding untreated or raw (whole) prey, eggs, and/or meat is the primary cause of bacterial gastroenteritis in ferrets.[13] As in other species, raw food products can be contaminated with *Campylobacter* spp, *Salmonella* spp, *Mycobacterium* spp, *Listeria* spp, and/or *Clostridium* spp. As these bacterial pathogens carry a potential zoonotic risk,[62] feeding raw food items to ferrets is discouraged at research facilities and in households with immunosuppressed family members.

Salmonellosis

Reports on *Salmonella* spp. causing enteritis in ferrets are rare, though infection may likely be underreported. *Salmonella* spp are readily transmitted by fecal-oral route,[63] with the serotype involved depending on the contaminated food source. Ferrets with clinical salmonellosis may demonstrate fever, lethargy, hemorrhagic diarrhea, anemia, balanitis, serous to seromucoid conjunctivitis, and pregnant jills might possibly abort (**Table 3**).[64] Mortality varies according to the strain involved. On necropsy, hyperemic small intestines with petechial hemorrhaging and bloody semi-solid contents may be observed. Histologically, the mucosal surface of the entire GI tract is inflamed with invasion of macrophages and lymphocytes, and missing intestinal tips on the villi. *Salmonella* is diagnosed on fecal cultures (repeated sampling is advised because of intermittent shedding), blood cultures (if septicemia is present), or culture of intestinal biopsies. Sensitivity testing is paramount to guide selection of antibiotic treatment. Sulfathalidine has been used with success in experimentally

infected ferrets.[63] In addition to appropriate antibiosis, fluid therapy (including correction of electrolyte imbalances, if any), assisted feeding, reduction of environmental stressors, and treatment of concurrent diseases are advised (see **Table 2**).[13]

Campylobacteriosis

Campylobacteriosis in ferrets is common. Most often, infection is caused by *Campylobacter jejuni*, and—to a lesser extent—by *Campylobacter coli.* Ingestion of contaminated food and direct transmission at multiferret locations are the primary causes of infection.[65] Adult ferrets are usually asymptomatic carriers, while kits and immunodeficient individuals commonly develop diarrhea, usually within 24 hours following ingestion of the pathogen (**Table 3**).[66] *Campylobacter* colonizes the entire intestinal tract, leading to intermittent, mucoid, watery, green, or bloody diarrhea. Disease is often self-limiting, but infections can persist for more than 4 weeks.[41,46,50] *Campylobacter* can also cause placentitis and abortion in up to 100% of pregnant jills.[67] Infection can be confirmed by culturing the feces on selective media at 37°C and 42°C (98.6°F and 107.6°F) under microaerobic conditions for 48 to 72 hours.[11] PCR of intestinal biopsies or liver tissue can also be used to identify the pathogen. Treatment consists of supportive care and antibiotics based on antimicrobial sensitivity testing, as needed (see **Table 2**).[13]

Mycobacteriosis

Mycobacterial infections in ferrets are uncommon, except in New Zealand feral ferrets.[68,69] Infection typically involves *Mycobacterium bovis, M avium,* and *M triplex.* The primary cause of disease transmission is feeding of unheated raw meat products, but direct/indirect transmission from in-contact ferrets or wild animals also carries a risk.[70] Clinical signs of mycobacteriosis are often nonspecific and may include anorexia, diarrhea, weight loss, and lethargy (**Table 3**). In a study, mortality related to mycobacteriosis was reported to be 41% in ferrets with clinical disease.[71] Necropsy commonly reveals a granulomatous enteritis, enteric lymphadenitis, and hepatosplenomegaly with granulomas present in both organs.[69,72] Clinical diagnosis of mycobacteriosis can be difficult to achieve as many ferrets have subclinical disease, show nonspecific signs, and/or only have a single granuloma or no gross lesions on exploratory laparotomy or necropsy.[11] Old granulomas may calcify and be identified on radiographs. A delayed diagnosis increases the risk of zoonotic transmission or spread within the population and can therefore be problematic.[70] On cytology or histopathology (with or without immunohistochemistry), characteristic acid-fast staining rods can be identified in the granulomatous lesions. PCR testing and culture can be used to confirm the strain involved.[73] Reactions to tuberculin vary with the strain; hence, this test is considered unreliable in ferrets. Ferrets infected with mycobacterium tuberculosis complex (*M tuberculosis* or *M bovis)* should be euthanized for zoonotic reasons.[11] Be aware that strains of mycobacterium tuberculosis complex may be a notifiable disease depending on geographic regulations. In case of nontuberculous mycobacteria, rifampicin with/without enrofloxacin and azithromycin has been used. However, because of the granulomatous nature of the disease, clinical disease is likely to reoccur when antibiotic treatment is withdrawn.[71,73,74] Surgical removal of granulomas compressing or obstructing the GI-tract can be attempted to alleviate clinical signs, but chance of recurrence is high, hence making the overall prognosis guarded to poor.[72]

Proliferative Bowel Disease

Lawsonia intracellularis, formerly known as intracellular *Campylobacter*-like organism, is an uncommon cause of chronic, bright green, mucohemorrhagic diarrhea in very

young or immunocompromised ferrets (Table 3).[13,75,76] *Lawsonia* invades the colonic intestinal wall, resulting in proliferation and infiltration of the intestinal wall with mixed inflammatory cells. This in turn leads to malabsorption. Upon clinical examination, thickened intestines may be palpable.[13] The ferret may cry in pain on defecation. Rectal prolapses may result from persisting chronic diarrhea and straining. Additionally, ferrets may show progressive anorexia, weight loss, and dehydration which can result in mortality if left untreated.[11,13,75] Coinfections with other pathogens like *Escherichia coli* may increase the risk of morbidity and mortality.[77] The thickened colonic wall can readily be diagnosed using abdominal ultrasound, while the organism can be diagnosed by PCR of feces or intestinal biopsies. Additionally, Warthin-Starry stain and immunohistochemistry can be used to identify the organism in the apical portion of the colonic enterocytes.[75,76,78] Treatment with oral chloramphenicol has been found effective.[79] Early treatment is vital to avoid chronic colonic malabsorption. Rectal prolapses, if present, usually do not require the use of purse-string sutures as these will generally not recur if the straining and diarrhea are resolved.

VIRAL GASTROENTERITIS

Epizootic Catarrhal Enteritis

Epizootic catarrhal enteritis (ECE) is a common cause of diarrhea caused by ferret enteric coronavirus.[80] Young ferrets are typically subclinical or only display very mild clinical signs, hence serving as an important reservoir for infecting adult ferrets. Adult ferrets, in contrast, develop smelly, bright green mucoid diarrhea within 2 to 3 days post infection, which lasts for weeks to even months (Table 3). As disease progresses, anorexia, vomiting, dehydration, weight loss, and lethargy may develop.[25] While mortality is low, morbidity may reach 100%.[11,13,81,82] Confirmation of infection can be achieved using PCR, identifying the coronavirus particles using transmission electron microscopy of fresh feces or intestinal biopsies or through detection of serum antibodies to ferret enteric virus using ELISA.[83] Histopathology of intestinal biopsies reveals atrophy and necrosis of intestinal villi and lymphocytic infiltration.[82] Ferrets with epizootic catarrhal enteritis must be isolated; supportive therapy, including prednisolone, is only necessary in anorexic or dehydrated ferrets. Nutritional support with an easily absorbed diet may improve the general clinical condition (see Table 2).[13] Chronic malabsorption, with feces typically containing undigested fat and starch and resembling birdseed (Fig. 8), is common in recovered ferrets. Due to the contagiousness of the disease, screening should be considered before introducing new individuals to multiferret households.

Fig. 8. Typical bird seed feces seen in a 4-year-old neutered male ferret that was presented with chronic poor appetite and irregular consistency of the feces.

Aside from causing ECE, it is speculated that ferret enteric coronavirus might mutate into a systemic form causing severe systemic disease, as known with feline coronavirus and feline infectious peritonitis.[25,84]

Rotavirus Diarrhea

Crowding and poor sanitary conditions are predisposing factors for rotavirus diarrhea. While adult ferrets usually display mild diarrhea or remain asymptomatic, morbidity and mortality can exceed 90% in 1 to 3 week old kits, especially in case of coinfections.[85] Transmission of the infection only requires ingestion of few fecal rotavirus particles, with kits developing a yellow to green mucoid diarrhea causing perineal soiling and erythema within 24 to 72 hours (**Table 3**).[13,86] Though clinical signs commonly only last for 3 to 4 days, the small size and undeveloped immune system of the kits predisposes them to severe dehydration, electrolyte imbalances, anorexia, and hypoglycemia.[87] Group A and C rotavirus infection can be demonstrated using electron microscopy, ELISA, or using reverse transcription PCR of fecal or tissue samples.[86,88,89] Gross postmortem examination of deceased kits will reveal an enlarged abdomen with gas-filled intestines, while histopathology confirms atrophic enteritis of the jejunum and ileum with blunted degenerated villous tips.[86,88] Treatment of affected kits generally requires intense supportive care (see **Table 2**). Rotavirus is almost impossible to eradicate in a population as virus particles are stable, especially in cooler climates. It is therefore important to maintain as sanitary conditions as possible, quarantine and screen new animals prior to introducing them into the population, and ensure ingestion of colostrum containing antibodies against rotavirus.[90]

Canine Distemper

Ferrets are highly susceptible to canine distemper virus (CDV), and readily develop disease following exposure to the virus if unvaccinated. Transmission occurs through direct contact or inhalation or ingestion of virus particles excreted in bodily fluids by other (unvaccinated) ferrets, as well as unvaccinated dogs, wild canines, mustelids, and procyonids.[89,91] Modified live virus vaccine-induced disease has also been documented.[92] After a 7 to 10-day incubation period, the ferret develops flu-like symptoms, and a pruritic rash which starts on the chin and progresses to the ventrum. GI signs including melena and diarrhea may be seen, though these are less common.[93,94] Progressing leukopenia results in secondary bacterial infections, including pneumonia. If the animal survives the initial stage, neurologic signs, including seizures, will develop prior to death.[13,89] In unvaccinated populations, mortality reaches up to 100%.[95] Infection with CDV can be confirmed by immunofluorescence test on blood or conjunctivital scrapings, or by reverse transcriptase-PCR on peripheral blood.[96] Treatment consists of supportive care, symptomatic therapy, and treatment of secondary bacterial infections using antibiosis, but is rarely successful, and may only prolong the development of neurologic signs.[89] Euthanasia should therefore be considered as a realistic alternative to palliative treatment. Prevention is by serial vaccination, which may also help to reduce risk of spread and severity of disease after exposure.[95] Currently, PureVax Ferret Distemper Vaccine (Boehringer-Ingelheim) is the only licensed vaccine for ferrets. Unfortunately, this vaccine is unavailable in many countries. Canine vaccines, most notably Nobivac Puppy-DPv (Merck Animal Health), have successfully been used as an alternative. However, efficacy and safety studies for this and many other vaccines are lacking. With some of these vaccines inducing myofasciitis, caution is warranted when considering using a new vaccine.[97]

Influenza

Enteritis in ferrets with influenza can occur when viral particles invade the intestinal epithelium. However, ferrets infected with influenza virus more commonly present with respiratory signs, pyrexia, anorexia, and conjunctivitis.[13,62] Influenza is a zoonosis, but ferrets are also susceptible to transmission from humans.[98,99] Diagnosis is based on clinical signs, and spontaneous recovery occurs within 7 to 10 days. Nutritional support, cough suppressants, and NSAIDs can be used to alleviate symptoms.

In young kits, infection can be fatal because of secondary bacterial pneumonia.[89,100] A thorough history and PCR are important to differentiate influenza from canine distemper, because of the difference in prognosis.

Coronavirus Disease 2019

Acute gastroenteritis with lethargy, anorexia, vomiting, and profuse mucoid diarrhea has been observed in a ferret that became infected with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) by a symptomatic human. The ferret recovered after symptomatic treatment, including amoxicillin and dexamethasone.[101] This report not only indicates that SARS-CoV-2 should be considered in the differential diagnosis for ferrets with GI disease, but also emphasizes the disease to be an anthropozoonosis. Hence, caution is warranted to prevent transmission to ferrets in the household in case of SARS-CoV-2-positive family members.

MISCELLANEOUS INTESTINAL DISORDERS

Inflammatory Bowel Disease

The cause of inflammatory bowel disease (IBD) in ferrets is unknown, but chronic inflammation of the GI tract caused by diet, atypical immune response, genetics, or hypersensitivity has been proposed.[13] IBD is common in young to middle-aged ferrets, with a study identifying GI inflammation in up to 92% of necropsied ferrets. Initially, clinical signs can be subtle and intermittent, including hyporexia, vomiting, and diarrhea; in the later stages of the disease, loose birdseed poop, anorexia, weight loss, and lethargy may be noted (**Table 3**).[11] If left untreated, IBD has been suggested to progress to intestinal lymphoma.[102]

Diagnostic workup is important to distinguish IBD from ferret coronavirus, lymphoma, eosinophilic gastroenteritis, and dietary indiscretion.[13] Initial history taking should include current diet, possible exposure to ferret corona virus, and response to a diet trial (if performed). Hematology and biochemistry are often unremarkable or nonspecific with increased liver enzymes, hyperglobulinemia, and lymphocytosis. A final diagnosis and differentiation from lymphoma and eosinophilic gastroenteritis can be made using full-thickness intestinal biopsies. Histology of jejunum shows villous atrophy, distended crypts, and an inflammation characterized by lymphocytes and plasma cells, and occasionally low numbers of eosinophils.[103,104] Immunohistochemistry (using CD3 and CD79a) is slightly superior in distinguishing IBD from lymphoma and should be considered.[102] Prednisone and/or azathioprine can be used to reduce intestinal inflammation, with both drugs being well tolerated by ferrets (see **Table 2**).[105] No hydrolyzed diets for ferrets are available; hence, the use of hypoallergenic diets for cats or a nutritionally balanced homemade diet using a novel protein source should be considered while keeping the general nutritional requirements for ferrets in mind.

Eosinophilic Gastroenteritis

Reports of confirmed eosinophilic gastroenteritis in ferrets are rare. Eosinophilic gastroenteritis is a noncontagious disease, characterized by eosinophilic infiltration of multiple

tissues (GI tract, associated lymph nodes, liver, and lungs) and persistent eosinophilia in peripheral blood.[11,13,106] Clinical signs are related to the degree of GI infiltration and affected segments, and include vomiting, thickened intestines, lethargy, weight loss, and green mucoid diarrhea (**Table 3**).[106] The condition has only been reported in adult ferrets and is thought to be triggered by genetics or chronic intestinal irritation caused by commercial diets low in protein (<35%–55% of dry matter).[1] Biopsies of intestines and associated tissues are required to identify the diffuse eosinophilia of multiple tissues, which allows distinction between eosinophilic gastroenteritis, intestinal lymphoma, and IBD. Reports on treatment include the use of azathioprine, prednisone, ampicillin, cyclosporine, ivermectin, hypoallergenic diets, or combinations of these.[13,106,107] Treatment in other species indicates that immunosuppressive therapy in combination with hypoallergenic diets might be useful (see **Table 2**).[1] Unfortunately, current data are insufficient to recommend a specific treatment regimen in ferrets.

Food Hypersensitivity

Confirmed cases of food allergy have not been reported in ferrets, but GI signs with mild diarrhea that resolve following a diet change are not uncommon. Reports on the effect of elimination diets alone are currently not available, but a hydrolyzed hypoallergic diet (Hill's Prescription Diet z/d for cats) did not seem to decrease GI inflammation in a ferret with eosinophilic gastroenteritis.[107] A nutritionally balanced, novel protein home cooked diet might be superior to commercial hypoallergenic cat food, while also meeting the specific nutritional requirements of the ferret, but further research is required. Furthermore, care should be taken if using grain-free diets, as these have been associated with development of cystine urolithiasis.[108]

Intoxications

Accidental ingestion of toxic agents like household chemicals, plants, creams, soaps, paint, human food items, or medications are common in ferrets. Ferret-proofing the environment is therefore paramount.[109,110] Even small amounts of ingested toxins can cause significant morbidity and mortality, due to the small body weight of the ferret.[111] A thorough history should be obtained, and owners should be encouraged to bring packaging from ingested materials to aid in identifying potential toxic ingredients. Common toxicoses causing GI symptoms are theobromine, caffeine, and ibuprofen. Treatment depends on the type and amount of the toxin that is ingested and the clinical signs that are observed.[109,112] In case of a recent ingestion of a noncorrosive toxin, emesis can be induced with apomorphine. Activated charcoal (1-3 mg/kg orally) can be used to reduce GI absorption.[109,112]

Aside from household toxins, toxicity may also result from enterotoxins produced by bacteria. For example, severe yellow mucoid diarrhea may develop in neonatal kits following exposure to enterotoxins produced by *Staphylococcus delphini*. *E coli* is another enterotoxin-producing bacterium that leads to diarrhea; the type of diarrhea seen is strain dependent.[113,114] Enterotoxemia can quickly lead to severe dehydration, and—together with septicemia—to high mortality rates. Diagnosis is rarely made antemortem. If enterotoxemia is suspected, treatment using antibiotics, fluid therapy, and electrolytes should be initiated as soon as possible. Bacterial culture and PCR may be used to identify the strain and serotype involved.[114]

Intestinal Neoplastic Disease

Both benign and malignant intestinal neoplasias have been described in ferrets. Polyps are often asymptomatic and incidentally found on endoscopy.[110]

Intestinal lymphoma is the most common malignant intestinal tumor, and often diagnosed in older ferrets (mean age: 6.8 years).[44,115,116] Nodules can be present, but more commonly intestinal lymphoma results in diffuse intestinal thickening, with enlarged mesenteric lymph nodes.[115,117] Clinical signs can be subtle with slowly progressive weight loss, chronic diarrhea, and malabsorption. Lymphoma is readily misdiagnosed as IBD or eosinophile gastroenteritis,[22] requiring histopathology and immunohistochemistry of full-thickness intestinal biopsies to differentiate lymphoma from other causes of chronic diarrhea, and allowing the neoplasia to be immunophenotyped as either T-cell or B-cell lymphoma. IBD has been suggested as a precursor for lymphoma, making early stage disease especially difficult to distinguish.[102,115] T-cell lymphoma is more common than B-cell lymphoma (56% and 31%), with a slightly shorter median survival time (5 and 8.4 months, respectively).[118] Lymphoma can be staged, depending on the degree of organ involvement, with lower stages resembling less infiltration and better prognosis.[110,116] Several chemotherapeutic protocols have been utilized, but further research is required to define the treatment of choice, considering efficacy, safety, and quality of life during treatment.[117]

Intestinal adenocarcinoma and leiomyoma have also been described in ferrets but are rare. Generally, these neoplasms cause distinct nodular masses in the intestinal wall, with the ferrets developing progressive weight loss and nonresponsive diarrhea. If the tumor obstructs the intestinal lumen, vomiting and anorexia may be seen, thereby mimicking the clinical signs of intestinal foreign body entrapment.[44,110,119] The nodular mass may be identified using diagnostic imaging or be discovered as an incidental finding during abdominal palpation or exploratory laparotomy. Prior to starting chemotherapy, enterectomy with end-to-end anastomosis should be performed to remove the neoplasia and obstructed portion of the bowel. As regional and distant metastasis may already be present at the time of diagnosis, prognosis in case of adenocarcinomas is usually grave, and euthanasia should be considered.

Foreign Bodies

In addition to the stomach, the jejunum and duodenum are common sites where gastrointestinal foreign bodies get lodged.[110] Clinical signs include acute anorexia, apathy, diarrhea, profuse vomiting, bruxism, and ptyalism; time of onset is related to the time of luminal obstruction.[120,121] While some intestinal foreign bodies are readily palpable on abdominal palpation, abdominal radiographs with/without contrast or abdominal ultrasound are often used to confirm their presence.[121] As ferrets are notorious for ingesting rubber, foam, or plastic objects, it is not uncommon for them to have ingested multiple foreign bodies at once. Therefore, the entire GI tract should be thoroughly palpated, even if a foreign body has already been identified.[110,122] Linear foreign bodies are rare and can be more difficult to diagnose on radiographs, unless a curled-up intestine is identified on abdominal radiographs. As intestinal foreign bodies are unlikely to pass, enterotomy is indicated after initial stabilization (see **Table 2**). Postoperative care includes assisted feeding with liquid diets, analgesia, heat, and fluid therapy, as needed (see **Table 2**). In older ferrets, full-thickness intestinal biopsies for lymphoma/IBD screening as well as careful evaluation and palpation of the pancreas are recommended during exploratory surgery. In absence of perforations and (pre-) perforative peritonitis, the prognosis after foreign body removal is good.[120–122]

Stress-Related Diarrhea

Some ferrets are very sensible to environmental change. Traveling, altered routines, or a new human/animal family member can pose enough stress to the ferret, and subsequently lead to (*Helicobacter*-associated) gastritis and intermittent diarrhea. Generally,

ferrets will show little, if any, other clinical signs and commonly have a good body condition, which enables distinction from other causes of diarrhea. Diagnosis is based on history and exclusion of other causes of diarrhea. Modifications of the living environment to reduce exposure to stressors will suffice to resolve the problem.

DISTAL ENTERIC DISEASE

Rectal Prolapse

Any disease causing straining or prolonged diarrhea (see **Table 1**) can result in rectal prolapse. In young ferrets, rectal prolapse is most commonly seen in animals with enteritis and profuse diarrhea. In older animals, in addition to primary GI disease, rectal prolapses are also commonly seen with other conditions, including urinary straining caused by prostatic disease, neoplasia, obstructive urolithiasis; dystocia; or sublumbar lymphadenopathy.[11,12,103] In countries where anal gland removal is not prohibited, poor surgical technique may also cause rectal prolapse.[110] Diagnosis and treatment are focused on identifying and alleviating the initiating cause, following which most prolapses will resolve by themselves. Irritation or discomfort from mild prolapse can be alleviated using hemorrhoid cream or lidocaine-containing gel/ointment. If the rectal prolapse is severe, placement of a purse-string suture can be considered to prevent recurrence and protect the mucosal lining. Sutures can be left in place for 2 up to 21 days while the underlying cause is being treated.[11,13,103,110] Necrotic tissue should be removed, if present.[110]

Rectal Masses and Fistulas

Mild intermittent hematochezia, straining, or rectal prolapse can be an indication of rectal masses. Typically, these masses protrude into the lumen or compress the rectum, as can be visualized during CT or proctoscopy. Rectal polyps are usually benign and can be biopsied and removed with proctoscopy.[13] Rectal neoplasia includes lymphoma and leiomyosarcoma, which require a more complex treatment using radical surgical debulking, possible rectoplasty, and chemotherapy with/without radiation, dependent on tumor type and initial staging.[119,123] Removal of the entire tumor is often not feasible because of its anatomic location, and the risk of regrowth with local invasion is high, resulting in a poor prognosis.[13]

Rectovaginal fistulas have been described in 2 ferrets with fecal incontinence through the vulva, causing chronic vaginitis. Contrast radiographs and CT were used to identify the fistula path, before attempting surgical closure. Refistulation is possible, and quality of life should be considered in case of reoccurrence.[124,125]

Anal Gland Disease

Reports of anal gland disease in ferrets are few, but likely underreported since anal glands are routinely surgically removed in many countries.[126] Anal sacs can become impacted or infected, resulting in unilateral/bilateral perianal swelling. Emptying should be performed under sedation in a well-ventilated room, for the sake of the veterinary personnel. Antibiotics are rarely required after emptying and flushing, but analgesics are recommended to prevent discomfort.[13,110] Anal gland neoplasia can easily be mistaken for anal gland impaction; hence, it is recommended to carefully palpate the anal sacs after emptying. Both anal gland squamous cell carcinoma and adenocarcinomas have been reported. Surgical removal in combination with radiotherapy and/or chemotherapy usually carries a good prognosis.[127–129] In case of malignancy, screening for metastases is important, as adenocarcinomas may metastasize to regional lymph nodes or distant organs, including liver, spleen, and lung.[127]

SUMMARY

Ferrets are commonly affected by GI diseases of both infectious and noninfectious origin, resulting in clinical signs that are often nonspecific such as lethargy, anorexia, vomiting, and diarrhea. Noninfectious disease is often related to ingestion of foreign objects, trichobezoars, gastric reflux, neoplasia, or dental disease. Amongst the infectious causes, viral and bacterial gastroenteritis are most commonly encountered, with the latter frequently being associated with consumption of raw food. Diagnostic work-up and therapeutic principles largely follow similar guidelines as those established in other companion animals, while preventive measures include maintaining the ferret on a high-protein diet, ensuring vaccination against canine distemper, ferret proofing the house, and adequate quarantine and routine fecal parasite screening when new ferrets enter the household.

CLINICS CARE POINTS

- Histopathology and immunohistochemistry of full-thickness intestinal biopsies are required to differentiate IBD from intestinal lymphoma.
- In contrast to many other small mammals, ferrets are prone to gastric reflux, causing esophagitis and oral ulcerations. Gastritis is also common, and often suggested to be related to *H mustelae* infection.
- Screening for epizootic catarrhal enteritis through fecal PCR or serum antibodies should be considered before introducing new ferrets into multiferret households, as young kits serve as a reservoir, transmitting disease to other, adult ferrets.
- While the cause at current remains uncertain, eosinophilic gastroenteritis in ferrets might be triggered by long-term feeding of a diet low in protein (<35–55% of dry matter).
- In elderly ferrets, full-thickness intestinal biopsies for lymphoma/inflammatory bowel disease screening as well as careful evaluation and palpation of the pancreas are recommended during exploratory surgery.

DISCLOSURE

The authors have nothing to disclose.

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