Pentatrichomonas hominis infection in four kittens

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- Pentatrichomonas hominis is a trichomonad parasite that can inhabit the large intestine of humans, cats, dogs, monkeys, and guinea pigs.
- Although the pathogenic potential of P hominis is debated, it appears that infection with the organism may be associated with chronic diarrhea in some cats.
- Direct examination of a fresh fecal smear is necessary for identification of P hominis in feces.

A 14-week-old 1.7-kg (3.7-lb) female Maine Coon kitten (cat 1) undergoing a routine physical examination was found to have a strong odor of feces. The cat appeared healthy otherwise and did not have fecal soiling of the perineum. The cat had been purchased the previous day from an overcrowded and ill-managed cattery with poor sanitation. It had been vaccinated with a killed herpesvirus-calicivirus-panleukopenia vaccine and treated 1 month previously with praziquantel and pyrantel pamoate by the breeder (dosages were not reported) but otherwise was not known to have received any veterinary care. Whether the cat had any parasites prior to anthelmintic treatment was not known.

A fecal sample obtained with a fecal loop was soft, foamy, and light tan and had an overpowering odor. Examination of a smear of the fecal sample stained with new methylene blue stain revealed numerous Pentatrichomonas hominis trophozoites. Leukocytes and other parasites were not seen.

The cat was isolated and treated with metronidazole (75 mg, PO, q 24 h) for 14 days, and the diarrhea resolved. The cat subsequently delivered 2 litters of kittens, at 1 and 2 years of age. Kittens in both litters developed diarrhea and hematochezia at 10 weeks of age, but fecal smears from these kittens were unremarkable. The cat also developed diarrhea during both lactation periods; however, other than small numbers of coccidial oocysts, results of fecal examinations were unremarkable.

A 2-month-old 1.3-kg (2.9-lb) male Pixie-Bob kitten (cat 2) was examined because of diarrhea and dyschezia. Results of physical examination were unremarkable. A nylon fecal loop was inserted into the anus, and the colon wall was gently scraped. Examination of a smear of the scrapings stained with new methylene blue stain revealed large numbers of polymorphonuclear leukocytes associated with colonic mucus. No parasites were observed. The kitten was treated with enrofloxacin (2.5 mg, PO, q 12 h) for 14 days and metronidazole (75 mg, PO, q 24 h) for 7 days. Ten days later, the owner reported the diarrhea was unchanged. Large numbers of leukocytes were still evident in the feces, but they were no longer associated with mucus. Results of immunofluorescent antibody tests for Giardia spp and Cryptosporidium spp were negative at this time, and no parasites were observed in the feces. The cat’s diet was changed to a high-fiber diet, and amoxicillin was prescribed (25 mg, PO, q 12 h, for 14 days) in case diarrhea in this cat was a result of bacterial colonization of colonic glands and crypts. Bacterial colonization of colonic glands and crypts has previously been observed in intestinal biopsy specimens from a juvenile Pixie-Bob cat with protracted diarrhea.

Despite the change in treatment, the cat’s diarrhea continued unabated. The cat was reexamined at 4 months of age. The owner reported at that time that the cat passed fetid, liquid bowel movements daily, and frequently defecated outside its litter pan. Large numbers of P hominis trophozoites and moderate numbers of polymorphonuclear leukocytes were detected in the feces. The owner requested that the cat be euthanatized because of the frequent housesoiling.

A necropsy was performed, and specimens were submitted for histologic examination. Histologic lesions were not evident in specimens from the stomach; however, specimens from the duodenum, jejunum, and ileum had widespread, mild to moderate plasmacytosis and plasmacytic enteritis and mild to moderate eosinophilic enteritis with numerous eosinophilic globular leukocytes. Colonic specimens had moderate to marked plasmacytosis and plasmacytic colitis, moderate suppurative colitis with goblet cell hyperplasia, mild to moderate eosinophilic colitis with eosinophilic globular leukocyte hyperplasia, and moderate submucosal follicular lymphoid hyperplasia. Cecal specimens had moderate plasmacytosis and plasmacytic typhlitis, mild to moderate eosinophilic typhlitis with eosinophilic globular leukocyte hyperplasia, marked submucosal follicular lymphoid hyperplasia with multifocal intrafollicular lymphocytosis, multifocal single-cell crypt epithelial cell necrosis, moderate number of intraluminal neutrophils, and moderate mucosal goblet cell hyperplasia. Examination of sections stained with Warthin-Starry, methenamine silver, and Giemsa stains revealed pear-shaped protozoan organisms in the intraluminal contents and close to the mucosal epithelial surfaces of the colon and cecum.

The histologic diagnosis was lymphoplasmacytic enterocolitis with eosinophils and eosinophilic globular leukocytes, neutrophils in the mucosa of the colon and within intraluminal contents of the cecum, and P hominis trophozoites in intraluminal contents of the colon and cecum. Histologic findings were similar to those reported for a cat with chronic diarrhea that was infected with P hominis.
A 4-month-old male Pixie-Bob kitten (cat 3), a littermate of cat 2, was examined because of diarrhea since weaning. As with cat 2, this cat frequently defecated outside its litter box and passed fetid, liquid feces. The cat had not been examined by a veterinarian previously. A fecal loop was inserted into the anus, and the wall of the colon was gently scraped. Examination of a smear of the scraping stained with new methylene blue stain revealed large numbers of *P. hominis* trophozoites and moderate numbers of polymorphonuclear leukocytes and RBC. The kitten was euthanized, and a necropsy was performed. Histologic findings were similar for findings for cat 2.

The dam of cats 2 and 3 had a long, well-documented history of chronic diarrhea and was passing fetid, liquid feces at the time of purchase from a cattery at 6 months of age. Results of immunofluorescent antibody tests for *Giardia* spp and *Cryptosporidium* spp were negative, and neither *P. hominis* nor any other parasites were detected during multiple fecal examinations. However, the cat frequently had large numbers of polymorphonuclear leukocytes in its stool. During a period of 14 months, the cat had normal stools as long as it was receiving enrofloxacin (20 mg, PO, q 24 h). If treatment with enrofloxacin was stopped, or if amoxicillin was administered instead of enrofloxacin, bowel movements again became loose, fetid, and explosive. After 14 months, treatment with enrofloxacin was discontinued, and the cat had normal feces for the next 12 months.

A 10-week-old 1.1-kg (2.4-lb) female Bengal kitten was examined 2 weeks after being purchased from a cattery. The owner did not report any problems at this time, and results of physical examination were unremarkable. A fecal smear was not examined.

The cat was reexamined 2 weeks later because of diarrhea and dyschezia. At this time, the cat was slightly lethargic and mildly (8%) dehydrated; rectal temperature was 39.2 °C (102.5 °F). Feces were tan, fetid, and flecked with fresh blood. Examination of a fecal smear stained with new methylene blue stain revealed numerous *P. hominis* trophozoites, but polymorphonuclear leukocytes and other parasites were not seen. The owner reported that neither of the 2 adult cats in the household had diarrhea or other clinical abnormalities. Results of serum biochemical testing and a CBC were within reference ranges, and the feline coronavirus titer was 1:400. The kitten was given lactated Ringer’s solution (75 ml, SC) because of the dehydration and was treated with metronidazole (75 mg, PO, q 24 h) for 10 days and enrofloxacin (5 mg, PO, q 24 h) for 21 days. Enrofloxacin was chosen because of previous reports of efficacy in treatment of cats with diarrhea associated with *P. hominis* infection, and because quinolone antibiotics are the drugs of choice for empiric treatment of suspected bacterial diarrhea in humans.

The cat was reexamined 2 days later. Feces were odorless and pasty, and trichomonads were not seen in a fecal smear. Three weeks later, the kitten was returned to the veterinary hospital for routine vaccinations. The owner reported that consistency of the feces varied from firm to soft. Examination of a smear of colonic contents revealed a small number of polymorphonuclear leukocytes, but parasites were not seen. The owner was instructed to continue treatment with enrofloxacin (10 mg, PO, q 24 h) indefinitely. At 6 months of age, the kitten was spayed. At this time, results of examination of a fecal smear were unremarkable. The cat continued to receive enrofloxacin for several additional months, because the owner observed that the feces became very soft within 24 hours after antibiotic treatment was discontinued. When examined at 1 year of age, the cat appeared healthy, and the owner reported that the cat’s feces were firm and appeared normal, even though treatment with enrofloxacin had been discontinued several months earlier. A fecal smear was not examined at that time. *Pentatrichomonas hominis* has been reported to inhabit the large intestines of humans, cats, dogs, monkeys, and guinea pigs. It exists only in a free-swimming form called a trophozoite and does not produce cysts. The trophozoites are fragile and do not persist for extended periods in the environment. Transmission of *P. hominis* is thought to be direct via the fecal-oral route. The risk of zoonotic infection with this organism has not been rigorously determined but is thought to be low. In undeveloped countries, *P. hominis* can be detected in feces of approximately 1% of humans with chronic diarrhea, but its importance as a pathogen has been questioned. Usually, these patients are infected with multiple parasites, and the role each parasite plays in causing or perpetuating diarrhea is not easily established. The medical literature does, however, contain a report of diarrhea in an infant in which *P. hominis* was the only parasite identified; the diarrhea resolved with drug treatment. The importance of *P. hominis* as a primary pathogen in cats is controversial.

Although trophozoites of *P. hominis* are about the same size as those of *Giardia* spp (roughly 4 x 10 µm), there is no difficulty in distinguishing between these parasites. *Pentatrichomonas hominis* has a distinctive undulating membrane that extends the length of its body, along with a rod-like structure called an axostyle, that extends the length of its body and protrudes posteriorly to form a pointed spine. *Pentatrichomonas hominis* also has 3 to 5 anterior flagella, but these flagella are not normally visible in vivo. A nucleus located at the anterior end of *P. hominis* may occasionally be seen as a round, refractile mass, but its presence or absence does not contribute to identification of the parasite. Staining is not required to identify *P. hominis*, but closing down the aperture of the microscope’s iris diaphragm aids visualization. Flotation techniques commonly used in veterinary practice to detect parasite ova in feces will not demonstrate trichomonads, and diagnosis requires examination of a fresh fecal smear.

Diarrhea in cats infected with *P. hominis* can be difficult to treat, and disappearance of trophozoites from fecal smears does not necessarily correlate with improvement in clinical signs. Cats described in the present report reflect the variability in clinical course of the disease and response to treatment that may be characteristic of *P. hominis*-associated diarrhea. One cat had severe colitis that was not responsive to treatment with metronidazole and enrofloxacin; diarrhea
developed prior to the appearance of trichomonads in the feces. A second cat had loose feces for several months after disappearance of trichomonads from the feces, and consistency of the feces was normal when enrofloxacin was given. A third cat responded promptly and completely to a single 14-day course of metronidazole treatment. Although treatment with metronidazole typically reduced the number of trichomonads observed in a fecal smear from thousands to 0 or nearly 0 within a day or 2, this dramatic reduction in parasite load was not necessarily associated with any improvement in clinical signs. Addition of enrofloxacin to the treatment plan seemed to improve consistency of the feces, and long-term treatment with enrofloxacin was required in some cats to maintain normal fecal consistency. The author now routinely treats cats infected with *P. hominis* with metronidazole for 7 days and enrofloxacin for at least a month.

A previous report described *P. hominis* infection in 3 Pixie-Bob cats with chronic diarrhea. Including the 4 cats described in the present report, therefore, 5 of 7 cats in which the author has detected *P. hominis* infection were Pixie-Bob cats. During the time these cats were treated, the author's practice consisted of approximately 4,000 patients, only 30 of which were Pixie-Bob cats. All 3 Pixie-Bob cats infected with *P. hominis* that underwent intestinal biopsy or necropsy were found to have histologic evidence of inflammatory bowel disease. At the time these cats were examined, all Pixie-Bob cats in Washington state were closely related and originated from a handful of regional catteries. A breed or familial predisposition for chronic diarrhea associated with inflammatory bowel disease and *P. hominis* infection or a common source of infection are 2 potential explanations for the seemingly high prevalence of *P. hominis*-related diarrhea in Pixie-Bob cats. Surprisingly, no mixed-breed cats living in the same households with infected Pixie-Bob cats developed diarrhea.

References