



This case report illustrates the interest of PURINA® PRO PLAN® VETERINARY DIETS Feline EN ST/Ox Gastrointestinal and FortiFlora® in an Abyssinian cat infected with *Tritrichomonas foetus*

Benefits of FortiFlora® and Feline EN ST/Ox Gastrointestinal diet in an Abyssinian cat infected with *Tritrichomonas foetus*

Dr Marine Hugonnard

Maître de Conférences en Médecine, Université de Lyon, VetAgro Sup,
F-69280, MARCY L'ETOILE, FRANCE

Case History

Fakir, a one year old entire male Abyssinian cat, was presented for chronic diarrhoea with haematochezia, which had developed since acquiring him at a show at the age of 8 months. The faeces were normally liquid, and the owner reported bouts of faecal incontinence. He was fully vaccinated and dewormed with moxidectin 4 months ago. He lived in an apartment with a 3 year old non-pedigree cat, which showed no signs of digestive problems. Various changes of diet (standard range) had not improved the symptoms, neither had symptomatic treatments (digestive tract protectants, motility regulators) and a one month course of antibiotics (sulphonamides). The cat remained alert and his appetite was unchanged.



Figure 1: *Tritrichomonas foetus*, Lugol stain, enlargement x40 .

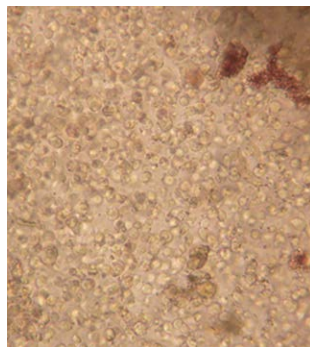


Figure 2: *Tritrichomonas foetus* in culture, inverted microscope, magnification x20.

Physical Examination

Fakir was lively and weighed 2.1kg. He was thin but had a shiny coat. A low-grade gingivitis was noted, together with erythema and slight dilatation of the anus. The rest of the examination was normal.

Differential diagnosis

The principal hypotheses considered were parasitic infection (particularly *Giardia* or *Tritrichomonas*), dietary intolerance or allergy, or an unbalanced intestinal microflora. An underlying retrovirus or coronavirus infection could not be ruled out. Metabolic diseases (cholangitis, pancreatitis) were less likely, as the clinical picture would not normally be dominated by diarrhoea. Exocrine pancreatic insufficiency was also a possible hypothesis. Nevertheless, this disease is rare and seborrhoea is almost always seen as a result of vitamin B group deficiency. Idiopathic inflammatory bowel disease was not included at this stage as this is effectively a diagnosis by elimination after excluding other causes.

Additional Examinations

Faecal examination for *Giardia* was negative. Culture of a rectal swab for *Tritrichomonas foetus* was strongly positive. (Figures 1 and 2).

A rapid screening test for feline retrovirus was negative. The biochemistry profile was normal apart from a low-grade hypoalbuminaemia (26g/l, compared to normal values of 29 to 39g/l).

Ultrasound appearance of the liver, pancreas, bile ducts and digestive tract were normal. Enlargement of the ileocolic and jejunal lymph nodes was observed, as well as a thickening of the caecal wall (Figure 3).

Culture of a rectal swab of the asymptomatic companion cat was carried out, to determine if it was a healthy carrier. This proved negative.

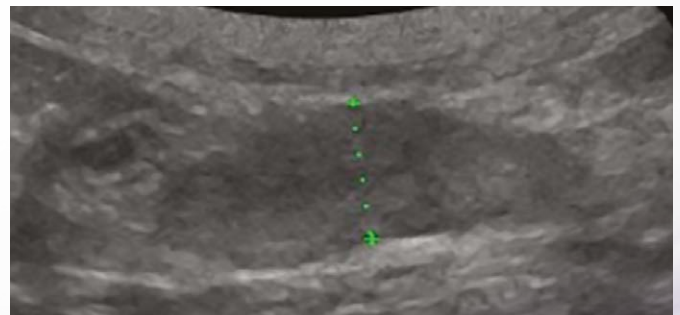


Figure 3: Abdominal ultrasound with enlarged jejunal lymph node.

Diagnosis

A diagnosis was made of *Tritrichomonas foetus* infection without favouring factors or apparent comorbidity.

Treatment and Follow-up

Whilst awaiting the results of the faecal tests and culture for *Tritrichomonas foetus*, treatment was started with fenbendazole at 50 mg/kg/day for 3 days. This was prescribed for both cats in the household without any improvement in Fakir's symptoms.

On reception of the culture results, a 30 day course of a probiotic (FortiFlora[®]) was prescribed for Fakir. It was recommended that the litter tray should be disinfected with bleach. Within 15 days, the haematochezia and the faecal incontinence had disappeared and there was an improvement in faecal consistency. This changed from liquid (faecal score of 7 on the Nestlé PURINA[®] faecal score chart) to soft (faecal score of 6).

However, there was an onset of faecal tenesmus and flatulence. A dietary transition to PURINA[®] PRO PLAN[®] VETERINARY DIETS Feline EN St/Ox Gastrointestinal dry food was then started, following which the tenesmus and flatulence disappeared. Within the space of 3 weeks, the faecal score further improved, varying between 5 and 6. In addition, body weight increased by 300g in 5 weeks. At this stage, another culture for *Tritrichomonas foetus* was performed. The results were once again positive.

Discussion

Tritrichomonas foetus is a flagellate protozoan phylogenetically and morphologically close to *Giardia*. The carrier rate in the feline community varies between 10 and 31% according to studies [1,2,3]. It could become an emerging pathogenic agent in this species [4]. The cat may be a healthy carrier and isolating the parasite does not necessarily signify that it is the cause of the symptoms. In Fakir's case, the epidemiology (a young pure-bred cat raised in a cattery) [5], the previous therapeutic failures and the lack of other identified possible cause of the diarrhoea reinforce the hypothesis that *Tritrichomonas* was very likely to be the origin of the symptoms.

To diagnose tritrichomoniasis, three techniques are possible: direct examination of freshly passed faeces under a light microscope, culture of a rectal swab or a PCR test. The sensitivity of culture and PCR are markedly superior to direct examination [1].

An association of *Tritrichomonas foetus* with a retrovirus is rare. On the other hand, *Tritrichomonas foetus* and *Giardia* can coexist in the same individual (12% co-infection according to the study of Gookin et al. [1], 27% in the study of Kuehner et al. [3]). An isolated faecal examination does not exclude giardiasis. Treatment with fenbendazole is therefore recommended, even though this drug is not effective against *Tritrichomonas foetus* [1].

In theory, specific treatment of tritrichomoniasis requires the use of antibiotics from the nitroimidazole family. Only ronidazole is effective most of the time at an oral dose rate for cats of 30 mg/kg/day for 14 days [6]. Cases of resistance to ronidazole have however been recently reported [7]. Moreover, this antibiotic does not have market authorisation

for use in the cat and potentially serious nervous side effects have been described in this species. This treatment must therefore only be undertaken with the informed consent of the owners. In this case, Fakir's owners did not wish to take risks and opted for a dietary treatment combining easily digestible food sources with probiotics. This strategy could be defended insofar as in the absence of treatment, the symptoms of tritrichomoniasis resolve spontaneously over a period of about one year on average (between 5 months and 2 years) [8]. Following the disappearance of diarrhoea, the animal may continue to carry the organism [7,8]. Dietary stability appears to be a factor that leads to a more rapid regression of symptoms. This probably contributes by stabilising the intestinal microflora closely linked with the immunity of the mucous membrane barrier. Leading on from this principle, an easily digestible food source rich in prebiotics in association with encapsulated probiotics adapted to the intended species are likely to favour a clinical cure. In the case of Fakir, the association of the diet Feline EN St/Ox Gastrointestinal with FortiFlora[®] produced a clear clinical improvement and return to a quality of life that the owners judged satisfying. Given the current state of knowledge and the lack of an effective and safe specific treatment, this dietary approach could be an interesting alternative to ronidazole following diagnosis of tritrichomoniasis.

Further Reading

1. GOOKIN JL, STEBBINS ME, HUNT E, et al. (2004) Prevalence of and risk factors for feline *Tritrichomonas foetus* and *Giardia* infection. *J Clin Microbiol*; **42**:2707-2710.
2. BRIGUI N., HENAFF M., POLACK, B. (2007) Prevalence of *Tritrichomonas foetus* in cats in France. 21st International Conference of the World Association for the Advancement of Veterinary Parasitology (Gent, Belgium), p 352.
3. KUEHNER KA, MARKS SL, KASS PH et al. (2011). *Tritrichomonas foetus* infection in purebred cats in Germany: prevalence of clinical signs and the role of co-infection with other enteroparasites. *J Feline med Surg*; **13**:251-258.
4. GOOKIN JL. (2009) *Tritrichomonas foetus* – an emerging cause of feline diarrhea. Proceedings 19th ECVIM-CA Congress, Porto.
5. GUNN-MOORE DA, McCANN TM, REED N et al. (2007). Prevalence of *Tritrichomonas foetus* infection in cats with diarrhea in the UK. *J Feline Med Surg*; **9**:214-218.
6. GOOKIN JL, COPPLE CN, PAPICH MG et al (2006). Efficacy of ronidazole for treatment of feline *Tritrichomonas foetus* infection. *J Vet Intern Med*; **20**:536-543.
7. GOOKIN JL, STAUFFER SH, DYBAS D et al (2010). Documentation of *in vivo* and *in vitro* aerobic resistance of feline *Tritrichomonas foetus* isolates to ronidazole. *J Vet Intern Med*; **24**:1003-1007.
8. FOSTER DM, GOOKIN JL, MOORE MF et al (2004). Outcome of cats with diarrhea and *Tritrichomonas foetus* infection. *J Am Vet Med Assoc*; **225**:888-892.

(Photo credit for pictures 1 and 2: Parasitology department, VetAgro Sup)