Gastric *Ollulanus tricuspis* infection identified in captive cheetahs (*Acinonyx jubatus*) with chronic vomiting

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ABSTRACT

Gastritis, vomition and weight loss are common in captive cheetahs (*Acinonyx jubatus*). Gastric spiral bacteria (*Helicobacter* spp.) and the very small, viviparous nematode *Ollulanus tricuspis,* a stomach worm of cats, are believed to be important causes. Three sibling cheetahs at Wellington Zoo, New Zealand, developed chronic vomiting, diarrhoea and debility. Their parents were both South African-born. Response to antibacterial treatment was poor. Endoscopic examinations revealed chronic lymphoplasmacytic gastritis and *Ollulanus* infection. Treatment with oxfendazole and pyrantel embonate resulted in clinical improvement; however, 1 cheetah, which died 7 months later as a result of a ruptured liver due to hepatic amyloidosis, still had *Ollulanus* worms present in her stomach. *Ollulanus tricuspis* is a significant cause of gastritis and vomiting in captive cheetahs, lions and tigers, as well as wild cougars and tigers. The parasite has not yet been found in sub-Saharan Africa. Because of the unusual characteristics of this parasite, the literature on its life history and techniques for diagnosis is reviewed.

Key words: cheetah, gastritis, ill-thrift, nematoda, *Ollulanus tricuspis,* parasitology, pathology, vomiting, wildlife, zoological garden.

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INTRODUCTION

Chronic lymphoplasmacytic gastritis associated with intermittent vomition, progressive weight loss and debility is a common and widespread problem in captive cheetahs (*Acinonyx jubatus*)[13,35,36]. Outbreaks have been associated with gastric spiral bacteria[12,36,41], including *Helicobacter heilmannii* (formerly *Gastrospirillum*) and *H. acinonyx*[11]. These bacteria are believed to be aetiologically significant in cheetah gastritis. A variety of antibiotic treatment regimens have been employed in affected cheetahs[13,41,48]. Although treatment resulted in the improvement of clinical signs and gastric lesions in many cases, continuing evidence of gastritis with positive follow-up cultures was demonstrated in some, indicating either treatment failure or rapid reinfection[41,48].

Infection of captive cheetahs in Munich (Germany)[23,27] and in Taiwan[29] with the very small gastric trichostrongyloid nematode, *Ollulanus tricuspis,* has also been associated with gastritis and vomiting. First discovered by Leuckart in 1865, *O. tricuspis* is most commonly found as a clinically inapparent infection in cats. It has also been causally associated with anorexia, vomiting, diarrhoea and chronic hypertrophic gastritis in this species[2,16,18,39,47,49]. The worm has been reported in cats from most European countries[24,43], the United States[16,22,39,44], Canada[30,38], Australia[14], New Zealand[9,34] and Chile[46]. More than 40 % of free-ranging cats in some surveys have been shown to harbour the parasites[17,24,26]. The only African report to date was from Egypt[25]. These nematodes have also been recovered from the stomachs of feral cats, wild cats (*Felis silvestris*)[7], dogs[28,45], red foxes (*Vulpes vulpes*)[31] and pigs[42]. Investigations in a number of European zoological gardens have revealed debilitating and/or fatal infections in captive tigers (*Panthera tigris*) and lions (*P. leo*)[6,24]. In this respect, Böhm was the first to associate *Ollulanus* infection with disease in zoo tigers and lions[27]. *Ollulanus tricuspis* has also been found in free-ranging cougars (*Felis concolor*)[40] and tigers[33] in North America and India respectively.

This report deals with the identification and attempted treatment of *O. tricuspis* infection in vomiting cheetahs kept at Wellington Zoo, Wellington, New Zealand.

CASE HISTORY

In 1989, a litter of 4 cheetah cubs was born to Marah and Claudius at Western Plains Zoo, Dubbo, New South Wales, Australia. Both parents originally came from South Africa. Marah originated from Des Varaday's Cheetah Sanctuary and the sire, Claudius, was wild-caught and hand-raised after his dam was shot[5]. The cubs were vaccinated against feline parvovirus, rhinotracheitis virus and calicivirus and were treated (anthelmintic not recorded) for a 'moderate infestation with roundworms (*Toxascaris leonina*) causing intermittent vomiting and diarrhoea'[5]. At 9 months of age, over-supplementation of calcium resulted in the development of valgus deformities due to retained cartilaginous cores in the distal ulna in all 4 cubs[5]. This problem was addressed by correction of the dietary imbalance and surgery[5].

In late 1991, 3 of the siblings, Malindi (a female), Asante and Etosha (both males) were translocated to Wellington Zoo, New Zealand. Before this, Malindi had shown inappetence and vomiting with undigested meat in her faeces and all 3 had been treated for suspected thiamine deficiency, following an episode of disorientation and apparent ataxia[3]. The cheetahs are housed together in a grassed enclosure with trees and logs and a den/yard area that has a concrete floor. A chain-link roof and walls prevent access by feral domestic cats[3]. The animals are vaccinated annually against feline viral diseases and faecal flotations are performed twice yearly. *Toxascaris* eggs have been found regularly, both before and after arrival in New Zealand[3].

During 1994, the condition of all the animals began to deteriorate. Clinical signs included frequent vomiting, inappetance, intermittent loose faeces that occasionally contained frank blood with mucus, repeated gingivitis and ill-thrift. Malindi was the worst affected. Tests for

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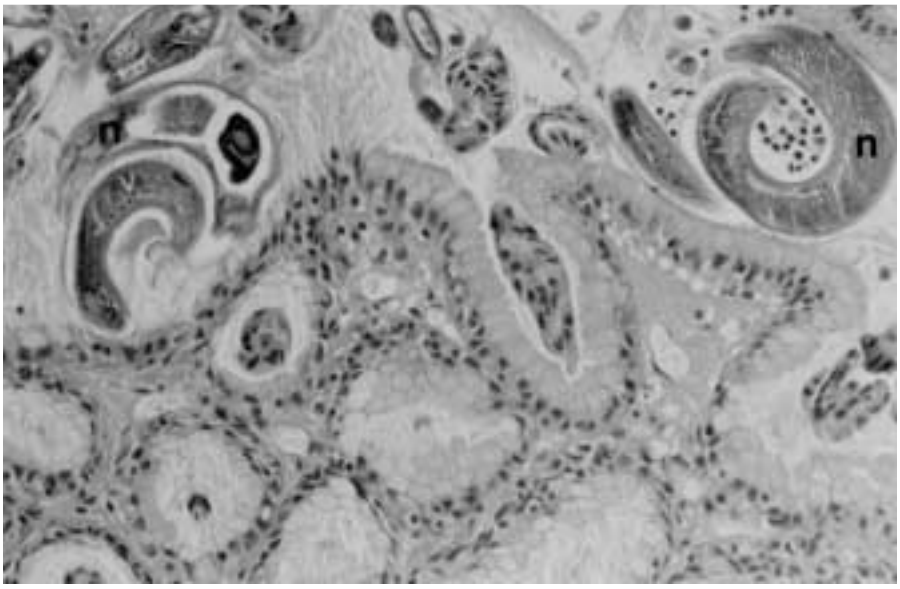

Fig. 1: **Micrograph of a gastric endoscopic biopsy from Asante. Six nematode** (n) **cross-sectional profiles are visible in the surface mucus on top of the mucosa**. HE, ×200.

feline leukaemia virus (FeLV) and feline immunodeficiency virus (FIV) were negative. *Helicobacter* infection was suspected and all 3 cheetahs were treated with a variety of antibacterials (enrofloxacin, amoxycillin, spiramycin, metronidazole) and other drugs (oral ranitidine and bismuth, dexamethasone, prednisolone, diazepam) for the following 18 months[3]. They appeared to respond briefly to treatment and then relapse with loose stools, bleeding gums and frequent vomiting of any food eaten. Hand feeding was often necessary and horse meat, pork, chicken and a proprietary diet (Hill's Prescription Diet C/D) were fed at intervals. Malindi and Etosha became debilitated and incontinent and the latter passed frank blood in his faeces. Blood tests revealed 6 % eosinophils in Malindi and 19 % in Asante. Treatment of Etosha with a variety of drugs (not specified) and an injection of 1 % ivermectin (dose not given) led to a slow recovery[3].

Gastric endoscopic examination of all 3 in October 1996 revealed that Malindi had an area of 'cobblestoned' mucosa, while Asante had small, raised, pale-pink, plaque-like foci scattered throughout the mucosa. Abdominal palpation revealed excessive gastric fluid and a turgid bowel in 2 animals. Gastric, duodenal and colonic biopsies from all 3 cheetahs were submitted for histopathological examination.

Lesions in all 3 stomachs were similar and consisted of prominent, locally extensive infiltrates of intra-epithelial globule leukocytes and increased numbers of plasma cells, lymphocytes, fibroblasts and collagen fibres and occasional, deeply situated nodular lymphoid aggregates in the lamina propria. Small nematodes (Fig. 1) with birefringent copulatory spicules (Fig. 2) in males and prominent longitudinal cuticular ridges (Fig. 3) were identified in the surface mucus and within dilated gastric crypts of at least 1 gastric biopsy from both Asante (numerous worms present) and Malindi. Additional serial sections performed on the original gastric biopsies subsequently revealed similar worms in Etosha. No spiral bacteria were seen with either HE or Warthin-Starry staining in any of the gastric biopsies. In the duodenums of all 3, neutrophils were evident in the lamina propria and prominent oedematous 'blebs' (large spaces formed between epithelium and lamina proprial connective tissue without endothelial lining) were present in the tips of many villi. The colonic biopsies from all 3 appeared normal.

The identity of the nematodes was confirmed following the biopsies when several *O. tricuspis* were identified in a specimen of vomitus from Malindi. Following this, all 3 cheetahs were treated with oxfendazole (10 mg/ kg) on 3 consecutive days and a month later given a single dose of 500 mg of pyrantel embonate. Two weeks after the endoscopy examinations, faecal cultures revealed *Salmonella* Typhimurium in Malindi and Etosha, and the cheetahs were treated with potentiated sulphonamides orally for 5 days. The cheetahs' appetites improved, the gingivitis disappeared, they regained their weight and the consistency and smell of their faeces returned to normal. Clinical signs of vomiting subsided but 1 cheetah began vomiting again 4 months later in April 1997 and all 3 were again treated with oxfendazole (10 mg/ kg) in 2 doses 14 days apart. In the years before their ill-health developed all 3 had been treated regularly with 'Canex Plus' (pyrantel embonate + oxantel embonate) (Pfizer Animal Health) and 'Drontal Plus' (pyrantel pamoate + febantel + praziquantel) (Bayer, New Zealand).

Malindi died in May 1997, 7 months after the endoscopic biopsies were taken. The necropsy revealed haemoperitoneum due to capsular rupture of the liver, which was swollen and diffusely pale brown. Histopathological examination revealed amyloidosis of the liver; no amyloid was present in the kidneys, lymphoid tissues, gastrointestinal tract or pancreas. Splenic lymphoid tissue was severely atrophied. A section of the stomach revealed intra-epithelial globule leukocytes, a moderate, diffuse lymphoplasmacytic infiltration with patchy fibrosis in the lamina propria,

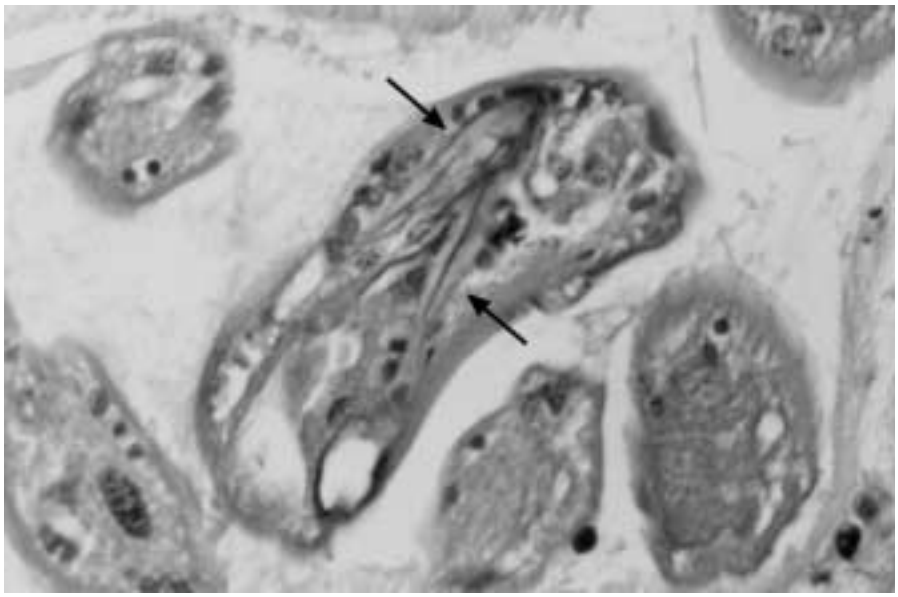

Fig. 2: **Micrograph of a gastric endoscopic biopsy from Asante. Six nematode cross-sectional profiles are visible in the surface mucus. The arrows indicate the split copulatory spicules of a male *Ollulanus*.** HE, ×800.

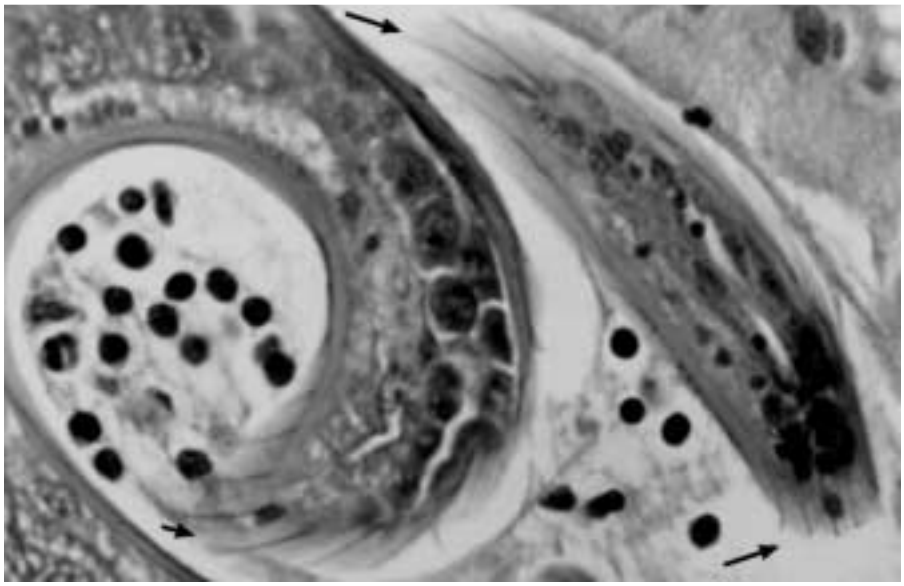

Fig. 3: **Micrograph of a gastric endoscopic biopsy from Asante. Two nematode profiles reveal the prominent cuticular ridges** (arrows). HE, ×800.

occasional glandular crypts containing neutrophils and sloughed cells, evidence of focal glandular atrophy and regeneration and isolated lymphoid foci in the deep mucosa and beneath the muscularis mucosae (Fig. 4). Additional sections cut from the same block revealed *Ollulanus* nematodes in gastric crypts. Numerous globule leukocytes were seen in a section of jejunum.

The males survive and appear well although both have had episodes of vomiting and nausea particularly after eating whole chickens[3]. The latter are now minced before being fed to the cheetahs. The concrete yard is steam-cleaned every 6 months and the cheetahs are drenched alternatively with oxfendazole and pyrantel embonate every 3 months[3].

LIFE HISTORY OF *OLLULANUS TRICUSPIS*

Ollulanus tricuspis worms are very small and difficult to see with the naked eye[22,24]. The adult females are 0.8 to 1 mm long and have 3 major (hence the name) and up to 3 minor tail cusps; adult males are 0.7 –0.8 mm long and have split copulatory spicules and a well-developed terminal bursa instead of the tricuspid tail[4,19,24,39,49,50]. Both male and female worms have characteristic longitudinal cuticular ridges[4,20,39,49]. The worms are viviparous, with eggs (of which there are only 1–3) through to 3rd-stage larvae developing in the uterus[8]. The number of worms in an infected animal therefore increases even without an exogenous cycle[24,44]; the endogenous generation interval lasts 30–37 days[50]. The life cycle is direct and infection is spread when a non-infected animal eats the vomitus of an infected one[19,44]. Vomitus normally consists of undigested or partially digested food covered in mucus[44]. It has been suggested that infected long-haired cats are more likely to transmit infection because they vomit more frequently than short-haired cats owing to their tendency to develop hairballs after grooming[22]. One infected animal can easily infect many others[20]. The numbers of worms in individual infected animals varies from very few to over 11 000[17,26], while a single sample of vomitus can contain none to more than 170[44]. The parasites remain viable in vomitus for up to 12 days[16,50]. No paratenic or intermediate hosts have been identified[44].

Ollulanus tricuspis larvae and adults should be differentiated from the larvae of other nematodes, such as *Aelurostrongylus abstrusus, Aonchotheca* (*Capillaria*) *putorii, Physaloptera* spp., *Trichostrongylus* spp., as well as oxyurids of prey animals (such as mice), which may also be found in feline stomachs[10,17,20,49].

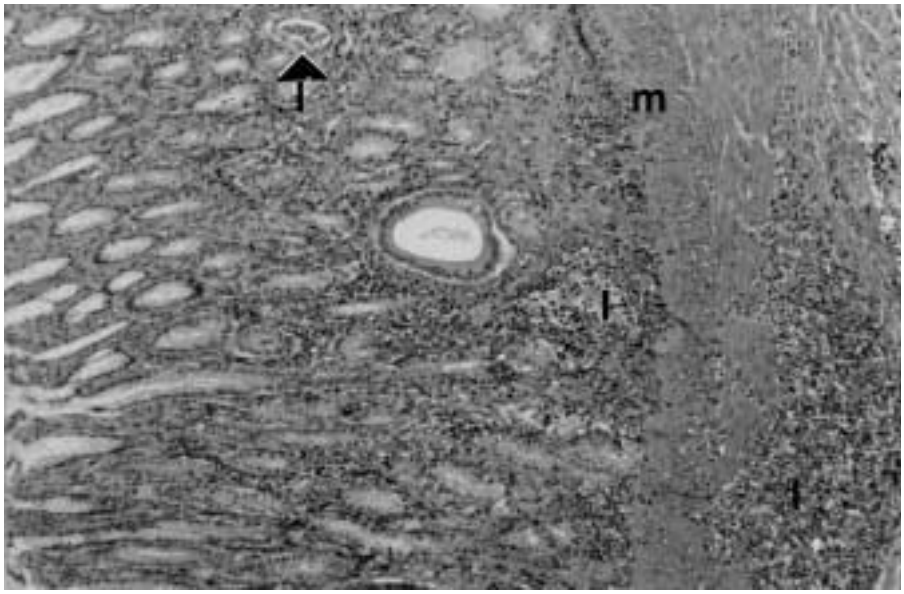

Fig. 4: **Micrograph of the stomach of Malindi *post mortem*. Note the lymphoplasmacytic infiltrates** (l) **in the lamina propria and beneath the muscularis mucosae** (m)**, a hyperplastic glandular crypt in the centre and inflammatory cells within 1 of the crypts** (arrow). HE, ×80.

TECHNIQUES FOR DIAGNOSIS

In order to diagnose infection antemortem, vomitus, gastric washings or gastric mucosal scrapings should be examined for worms with a dissecting microscope and gastric endoscopic biopsies should be examined histologically[22,27]. Examination of gastric contents and washings by repeated dilution and sedimentation is the most reliable method[17]. Worms can be concentrated using a Baermann's apparatus[24,44]. Several samples of fresh vomitus should be examined before ruling out an infection[44]. Faecal flotation or sedimentation is generally unrewarding, since L3, L4 and adult *Ollulanus* die in the higher pH of the intestine and are usually digested before faecal excretion[24]; however, *Ollulanus* worms have once been found in the faeces of a cat that had diarrhoea[19]. *Ollulanus* eggs are never seen in vomitus or faeces[49]. Peptic digestion of samples of stomach wall can be performed in dead animals[17,24].

DISCUSSION

Ollulanus probably played a role in the complex medical condition, particularly the vomiting and possibly the diarrhoea, affecting these cheetahs. Since clinical signs of vomiting and diarrhoea were present when the cheetahs were young cubs, it is quite likely that the animals were infected by their dam[3].

As far as the authors are aware, *O. tricuspis* has not yet been identified in any species of animal in sub-Saharan Africa. This may be because the parasites are

absent from those parts of the world, or because they are not amenable to detection *via* conventional coprological procedures, owing to their minute size and the fact that they are normally not found in faeces.

In this investigation, worms were seen in 2 of the 3 cheetahs on initial examination of gastric endoscopic biopsies. A subsequent search through serial sections of the same biopsies confirmed infection in the third. Additional sections of the necropsy stomach specimen were also required to confirm the presence of worms. This means that finding worms is probably fortuitous and false negatives can be expected especially if specimens are small and if only single sections are examined. This finding is supported by Guy[17], who compared various methods for the *post mortem* diagnosis of *Ollulanus* infection. Mucosal sampling, which is comparable to endoscopic biopsy or necropsy histopathology, detected only 29 %, while pepsin/HCl digestion detected 71 % of positive cases[17]. The accuracy of histology as a diagnostic tool, therefore, is probably proportional to the numbers of worms present[17,20].

Apart from small nematodes in surface mucus and within dilated gastric crypts, the 3 major histological changes in the stomachs of *Ollulanus*-infected cats are a patchy increase in mucosal fibrous tissue, an increase in proprial lymphoid aggregates and a significant increase in globule leukocytes[20,39]. If these lesions are found in feline gastric biopsies, *Ollulanus* infection is highly likely even if no worms are seen histologically[20]. The histological findings in these cheetahs resembled those described in cats with *Ollulanus* infection[20–22].

Increased numbers of globule leukocytes in gastrointestinal epithelium are commonly associated with helminth infections[15]. In Eaton *et al.*'s report on the association of gastric spiral bacteria with gastritis in cheetahs[12], globule leukocytes were prominent in the gastric biopsies of 16 % of the animals. No *Ollulanus* were found 'in spite of (an) extensive gross and histologic examination'. In the light of the above, it would be intriguing to know what had triggered the globule leukocyte response in the animals of their report. Where globule leukocytes are present, additional biopsies or serial sections should be examined to confirm or rule out *Ollulanus* infection.

In surveys[35,36] on the diseases of captive cheetahs in the United States and South Africa, no mention was made of *Ollulanus* as a possible cause of chronic gastritis. Chronic gastritis was noted in 91 % of the US study population, nearly all of which had gastric *Helicobacter* spp.[35]. Some of these animals were debilitated[35]. Gastritis accounted for 40 % of the South African mortalities[36]. Five cheetahs in the US survey had mild hepatic amyloidosis[35], and systemic amyloidosis was also noted in the South African survey[36]. In another survey[37], renal medullary type AA amyloidosis was found in 38 % of captive cheetahs. Many of these animals also had subsinusoidal hepatic AA amyloid deposits. All the animals had inflammatory diseases, the most common being chronic lymphoplasmacytic gastritis. The authors[37] concluded that the high prevalence of amyloidosis was in response to inflammation.

Possible causes of gingivitis in felids include FeLV and/or FIV infections, calicivirus infection, immune-mediated disease, infection with spirochaetes, spread from periodontitis, and uraemia[1]. The cause of the gingivitis in the Wellington cheetahs was never established[3]. Conceivably, the chronic vomiting could have precipitated a vitamin deficiency, culminating in gingivitis.

'Stationary management' of captive cheetahs and other large Felidae in zoos, as well as cats in breeding or research colonies, is likely to play a major part in increasing the prevalence of *Ollulanus* infection[2,27]. *Ollulanus* infection, therefore, should be strongly suspected in any felid with vomiting and loss of condition with or without diarrhoea.

According to previous reports, it appears that controlled studies on the anthelmintic efficacy against *O. tricuspis* infection in cats are lacking. In cats, tetramisole 2.5 % at 5 mg/kg has been claimed to be effective[24,50], while morantel (0.5 g/cat) and dichlorvos (10.3 mg/kg) were ineffective[50]. Greve[16], on the other hand, claimed success with 2 treatments of dichlorvos (11 mg/kg) at an interval of 1 month. Wilson and Presnell[49] referred to work of Zakhryalov and Chekushkina on experimentally infected kittens where fenbendazole at 10 mg/kg as a 10 % oral suspension was 99 % effective. For infected cats, Bell[2] suggested oxfendazole at 10 mg/kg twice daily for 5 consecutive days; lower dosages and less frequent treatments reduce but do not eliminate infections. Severe infection in a tigress and in 2 successive litters was eventually controlled with 20 mg/kg levamisole[32]. No information is available on the efficacy of ivermectin, which was administered to Etosha on 1 occasion[3]. Because of the inapparent nature of most feline infections, cessation of vomiting should not be regarded as evidence of clinical cure[44]. The fact that Malindi still had histological evidence of *Ollulanus* when she died more than 6 months after she and the other cheetahs were treated with oxfendazole and pyrantel embonate, indicates that these anthelmintics did not completely eradicate the infection. Further work on effective treatment of *Ollulanus* infection is needed.

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