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CLINICAL CASE

Strongyloides stercoralis hyperinfection in an immunosuppressed dog from



France*,**,***

M. Cervone^{a,*}, A. Giannelli^b, D. Otranto^b, S. Perrucci^a

^a Medicine University of Pisa, Department of Veterinary, Viale delle Piagge 2, 56124 Pisa, Italy
 ^b Medicine University of Bari, Department of Veterinary, Str. prov. per Casamassima km 3, 70010 Valenzano (Bari), Italy

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KEYWORDS

Strongyloides stercoralis; Hyperinfection syndrome; Immunosuppressed dog; Concomitant giardiosis Summary Strongyloides stercoralis is a threadworm, whose adult females parasitize the small intestine of mammals causing severe clinical presentations in immunosuppressed animals and puppies. A 10-month-old male Chihuahua dog was referred due to chronic diarrhoeic haematochezia, hematemesis, weight loss, pruritus and cough. During the clinical examination, severe weight loss and alopecia on the abdomen were observed. Stool examinations revealed the presence of alive larvae of *S. stercoralis*, as well as cysts and trophozoites of *Giardia duodenalis*. The negativity to *S. stercoralis* infection was achieved only after administration of ivermectin. Results of this study confirm that routine copromicroscopic methods may fail to diagnose *S. stercoralis* infection. In addition, although fenbendazole is considered the drug of choice for the treatment of canine strongyloidiasis, ivermectin may be a valid alternative.

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The work was done at Small Animal Veterinary Clinic Paris III, Paris, France.

^{***} Authors declare off-label use of ivermectin (use of drug in a non approved category of animal). A blood test to detect the genetic mutation responsible for the ivermectin sensitivity in dog (MDR1) was performed.

 $^{^{*}}$ Corresponding author. Small Animal Veterinary Clinic Paris III, 17, boulevard des Filles-du-Calvaire, 75003 Paris, France.

E-mail addresses: mariocervone@live.it (M. Cervone), giannelli.alessio@gmail.com (A. Giannelli), domenico.otranto@uniba.it (D. Otranto), stefania.perrucci@unipi.it (S. Perrucci).

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Introduction

Intestinal parasites of dogs comprise a number of species of zoonotic concern (e.g., Toxocara canis, Ancylostoma spp.), including Strongyloides stercoralis Bayay, 1876 [1,2]. Parthenogenetic females of this nematode live threaded in the epithelium of the small intestine in dogs, cats and primates [3]. They produce eggs which hatch inside the intestine in first stage larvae (L1s). Hosts shed L1s (and sometimes eggs) in their faeces. In the environment, S. stercoralis L1s may then develop either to the infective third-stage larvae (L3s) or moult into free-living adult nematodes, mainly depending on environmental conditions [1]. In the latter case, after mating, females produce eggs that hatch into L1s [1,3], mature into infective L3s and are able to infect a new susceptible host in which migrate to the small intestine via lungs and, after two additional moults, they develop to parthenogenetic adult females. The infection by S. stercoralis occurs percutaneously and, rarely, per os. Infection through lactation (mainly between the first and third day post-birth) has been described but remains controversial [4]. In addition, in immunosuppressed animals, autoinfections may occur spontaneously with potential dissemination of migrating L3s through the intestinal wall or the perianal skin [1,5]. In the infected host, strongyloidosis may result in self-curing, acute and chronic forms supported by autoinfection, and hyperinfection [6,7]. Difference between chronic form and hyperinfection depends on the severity of clinical manifestations and the number of adults in the intestine. Indeed, hosts chronically infected are asymptomatic or exhibit only minor symptoms; while hyperinfection is more likely related with severe gastrointestinal and respiratory alterations [7,8]. Severe forms are usually associated with immunosuppressive or debilitating disorders in human patients [7], but they are observed in young dogs with no know factors of immunodeficiency.

The prevalence of canine strongyloidiasis is underreported, probably because of the diagnostic limitation due to the fluctuations in amount of larvae excreted in the faeces [6,9]. Indeed, the parasitological diagnosis of S. stercoralis infection is based on the Baermann test, the agar plate method of Koga [10] or on the direct smear of fresh faeces [11]. In asymptomatic patients, a single stool examination fails to detect larvae in up to 70% of cases, so repeated stool examination are required to increase diagnostic sensitivity. Baermann method and especially agar plate method of Koga are more sensitive than direct smear to detect larvae in faeces [7]. However, due to lack of standard procedures in most parasitology laboratories and low sensitivity of direct parasitological methods, serological tests are also employed [6,12], including indirect fluorescent antibody (IFA) test [13], an intradermal reaction test and radial immunodiffusion [13,14].

S. stercoralis is commonly diagnosed in dogs from tropical and subtropical areas [15] with prevalence up to 24.3% in Asia [16]. In Europe, canine strongyloidosis has been reported in Germany [17], Finland [1], Greece [18], Portugal [19] and Italy [20,21] with an overall mean prevalence of about 0.8% [20].

The aim of the present paper is to describe a case of *S. stercoralis* hyperinfection in an immunosuppressed dog from France, coinfected by *Giardia duodenalis*. Data

recorded would clarify the spectrum of the clinical consequences and importance of diagnostic methods for this zoonotic infection.

Observation

A 10-month-old male Chihuahua dog of 1.5 kg bodyweight (Body Condition Score 2/9, according to WSAVA, Global Nutrition Committee), purchased in an animal-shop in Paris (France), was referred to a veterinary clinic because of chronic diarrhoeic haematochezia, hematemesis, weight loss and pruritus over a period of six months. During the previous week, the owner also reported that the dog suffered for persistent respiratory abnormalities and cough. Faecal examinations by flotation test, X-rays and abdominal ultrasounds were performed two months before and any abnormality was recorded. Therefore, inflammatory bowel disease (IBD) was suspected and the animal was put under regime of an intestinal diet (I/D, Hill's), an antidiarrhoeic-antibacterian association (Canidiarix®; TVM, Lempdes, France; i.e., sulfaguanidine 50 mg/kg/day + framycetine 8.4 mg/kg/day + atropine 0.0021 mg/kg/day for 5 days, repeated three times with an unknown interval), prednisone (Dermipred®; SOGEVAL, Laval, France) 1 mg/kg/day for two months and a nutritional complement (FortiFlora®; Purina, Missouri, USA), but no improvement in clinical condition was observed. Additionally, two months before the dog was referred, a treatment with fenbendazole 50 mg/kg/day (Panacur®; Intervet, Angers, France) for three days was performed and repeated 15 days later, based on suspected intestinal protozoan infection.

During the clinical examination, severe weight loss and alopecia on the abdomen were the only physical alterations observed. Blood tests revealed hypoalbuminaemia (2.3 g/dL), increased level of alanine aminotrasferase (ALT, 345 IU/L), anaemia (Hct, 28.82%), basal cortisol levels < 5.52 nmol/L and low eosinophil percentage of 1.20%. Radiographic examination of the thorax revealed the presence of exudative fluid in the peri-ilar region, associated to bronchogram. The image of striking bronchial pattern associated with an alveolar pattern was compatible with diagnosis of severe exudative or haemorrhagic pulmonary disease. Abdominal ultrasound was then performed with a 5-7.5 MHz convex probe (EASEOTE-MyLab™ 25Gold, Maastricht, Netherlands) in order to examine the intestinal tract, but no significant abnormalities were found. A fresh faecal sample was collected and promptly analysed by flotation test with a low-density solution (specific gravity 1.2), by direct smear and by the Baermann method. Stool examinations revealed alive L1s of S. stercoralis (Fig. 1A and B) and cysts and trophozoites of G. duodenalis. A real-time PCR of the material collected via oro-pharyngeal brushing revealed the presence of *Bordetella bronchiseptica*. Therefore, the diagnosis of intestinal co-infection by S. stercoralis and Giardia intestinalis was posed, associated with pulmonary infection by B. bronchiseptica and iatrogenic Cushing syndrome. The dog was hospitalised and patient received IV NaCl 0.9% fluids (108 mL/day) and SC maropitan citrate (Cerenia®, PFIZER, Sandwich kent, UK, 1 mg/kg/day), in order to restore normal hydration and to stop emesis

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