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HELMINTHOLOGY - SHORT COMMUNICATION

Urinary incontinence associated with *Mesocestoides vogae* infection in a dog

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Abstract

Peritoneal larval cestodiasis caused by *Mesocestoides* spp. is a rare infection in dogs. A 6-year-old female dog was presented for veterinary care with urinary incontinence which started 1 year earlier. After performing hematology, ultrasound, and computerized tomography, an exploratory laparotomy revealed canine peritoneal larval cestodiasis (CPLC) with the presence of *Mesocestoides vogae* (syn. *Mesocestoides corti*) tetrathyridia confirmed by morphological identification and PCR and DNA sequencing. Parasitic cysts were found around the urinary bladder and appeared to inhibit its normal function. An initial treatment with 5 mg/kg praziquantel subcutaneously every 2 weeks for four treatments failed to alleviate the clinical signs, and only treatment with fenbendazole at 100 mg/kg P.O. twice daily for 28 days was associated with the disappearance of ascites and regaining of urinary control. This is the first report of CPLC associated with urinary incontinence in dogs and the first description of this cyclophyllidean cestode in dogs in Israel.

Keywords Canine · *Mesocestoides vogae* · Urinary incontinence

Introduction

Mesocestoides spp. are cestode worms that belong to the family Mesocestoididae. The life cycle of *Mesocestoides* spp. has yet to be fully clarified. Currently, a 3-host life cycle is assumed for this genus (Padgett and Boyce 2004), with carnivores, birds of prey, and, occasionally, humans, as definitive hosts (Padgett and Boyce 2004). Coprophagous ground dwelling arthropods are the putative first intermediate host; however, all attempts to identify these or other invertebrates vectors have failed so far. In the first intermediate host, oncospheres (first larval stage) are presumed to develop into cysticercoids (second larval stage). The third life-cycle stage of *Mesocestoides* spp., termed the tetrathyridium, is the stage that has been recovered from various amphibians, reptiles, birds, and mammals. These infective larvae are characterized by an elongated or oval body about 2–70 mm long, filled with parenchyma; the scolex at the anterior end is usually invaginated and has four suckers and no rostellum and resembles that of the adult worm (Deplazes et al. 2016, pp. 213–214). Tetrathyridia develop in the body cavities, subserosal cysts, lungs, liver, and other organs. The final adult form develops within the intestines of the definitive hosts, approximately 2–3 weeks after ingestion of an intermediate host infected with tetrathyridia (Caruso et al. 2003). However, dogs can harbor both the intermediate stage and adult tapeworm at the same time if the tetrathyridium migrates through the intestinal wall, reaching the peritoneal cavity and abdominal organs (Toplu et al. 2004). *Mesocestoides corti* was described in the adult worm form in mice by Hoeppli in 1925 (Hoeppli 1925). Later on, Specht and Voge (1965) attributed the same species name to tetrathyridia found in the fence lizard (*Sceloporus occidentalis*), which were capable of multiplication and partial development in dogs following experimental infection (Eckert et al. 1969). In 1991, Etges suggested that tetrathyridia

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previously considered to be of *M. corti* be reclassified to a new species, *M. vogae*, based mostly on developmental differences (Etges 1991). Results of pylogenetic analysis done using the ITS2 gene (Crosbie et al. 2000a) and mitochondrial genes (Varcasia et al. 2018) also show that mesocestoides from dogs fall into a lineage distinct from that of *M. corti*. However, to date, there is still no conclusive evidence whether these are indeed two distinct species, and both names are sometimes used synonymously. One unique feature of *M. vogae* is its ability to engage in asexual reproduction (Crosbie et al. 2000a). Both adult worm as well as larval infection in the final hosts by *Mesocestoides* spp. is usually asymptomatic. Therefore, diagnosis of this infection is often an incidental finding during necropsy or laparotomy. However, some symptomatic clinical cases have been reported in association with larval cestodiasis caused by *M. vogae* (Boyce et al. 2011; Tamura et al. 2014; Häußler et al. 2016) including life-threatening peritonitis with severe peritoneal effusion in dogs (Bonfanti et al. 2004).

We hereby describe the findings of a case of urinary incontinence associated with *M. vogae* infection in a dog from Israel. To the best of our knowledge, this is the first report of *Mesocestoides* spp. infection associated with urinary incontinence in dogs and the first report of *M. vogae* infection in dogs in Israel.

Case report

A 6-year-old Husky mixed breed neutered female dog weighing 20 kg was presented to a veterinary practice in Holon, Israel, after it displayed signs of urge incontinence. Specifically, the dog was not able to restrain itself from urinating inappropriately. The dog had been adopted from an animal shelter in Israel at 4 months of age and was since housed predominantly indoors with free access to an outside garden. It had been dewormed every 6 months with praziquantel (5 mg/kg), pyrantel pamoate (6.5 mg/kg), and febantel (34 mg/kg; Drontal Plus, Bayer, Kansas, USA). When it was 5 years old, the owners temporarily left Israel with their dog for a period of 1 year to Massachusetts, USA, where it was housed entirely indoors. Urinary incontinence was noticed there for the first time, but treatment was not initiated.

At presentation, the physical examination was unremarkable. The dog's body score was adequate, and the owners explained that its appetite had remained consistently healthy since its urinary incontinence had begun 1 year previously. Complete blood count (CBC) and urinalysis were unremarkable. The dog was treated with phenylpropanolamine (Proin, Pegasus labs, Inc., FA, USA) at 3.25 mg/kg P.O. twice daily for several weeks, but it remained urinary incontinent even though mild improvement was noted by the owner. The dog was admitted again 4 months after its first presentation to the veterinary clinic with the same complaint. An abdominal ultrasound was performed and showed small liver size and a large volume of hyperechoic ascitic fluids with multiple abdominal cysts which were filled by amorphic-appearing fluid. Aspirated abdominal fluid appeared clear with numerous tiny whitish specks suspended within. The fluid had a total solid fraction (TS) of 1 and a packed cell volume (PCV) of zero. Cytology of the fluid stained by Giemsa indicated an inflammatory reaction with a small number of pyknotic neutrophils and macrophages, with no evident bacterial infection. It was decided to perform imaging of the abdomen with computerized tomography (CT) prior to exploratory laparotomy (Fig. 1a–c). CT findings included a reduced liver size with multiple irregular cavitated cystic structures, some of which communicated with the abdominal cavity (Fig. 1a) and ascites (Fig. 1b). Abnormal appearing tissue encased the urinary bladder (Fig. 1c). Differential diagnosis at this stage included mesothelial hypertrophy, neoplasia such as mesothelioma, and peritoneal cestodiasis. Exploratory laparotomy was thus performed, revealing extensive adhesions in the cranial abdomen, with numerous cysts that were adherent to the abdominal viscera and the urinary bladder. Surgical manipulation of the adherent viscera was not attempted. However, an open friable cyst, about 7 cm in size which contained many spaces filled with clear fluid was resected and submitted for histopathology. Tissues were trimmed, embedded in paraffin, sectioned, and stained with hematoxylin and eosin.

The histopathology findings in the tissue sections examined were mild fibrosis and mononuclear cell infiltration. There were many scattered cystic spaces composed of fibrous tissue with an inner layer of palisading macrophages surrounding degenerate cestode larvae with a thick, smooth tegument, a subjacent layer of somatic cells, and a loose body cavity with numerous calcareous corpuscles (Fig. 2a, b). No scolices were observed. Since it was now strongly suspected that the result of these pathological findings was due to proliferating larval cestodes, such as *Mesocestoides* or *Spirometra* species, ascitic material suspected to contain cestode larvae was collected and submitted to the Department of Parasitology, Kimron Veterinary Institute, Israel, and slide preparations were prepared and examined under an optical microscope. Whitish parasites, 1–3 mm in size with calcareous corpuscles, were observed. The parasites were morphologically identified as acephalic tetrathyridia, most likely belonging to *Mesocestoides* spp. (Fig. 3a, b).

DNA of the parasites was extracted using the QIAamp DNA Stool Mini Kit (Qiagen, CA, USA) according to the manufacturer's instructions, and two PCR procedures, targeting the mitochondrial cytochrome c oxidase subunit I (cox1; Bowles and McManus 1994) and the small subunit of ribosomal RNA (rrN) genes (Trachsel et al. 2007), were performed. Amplicons on electrophoresis gels were examined using UV illumination.

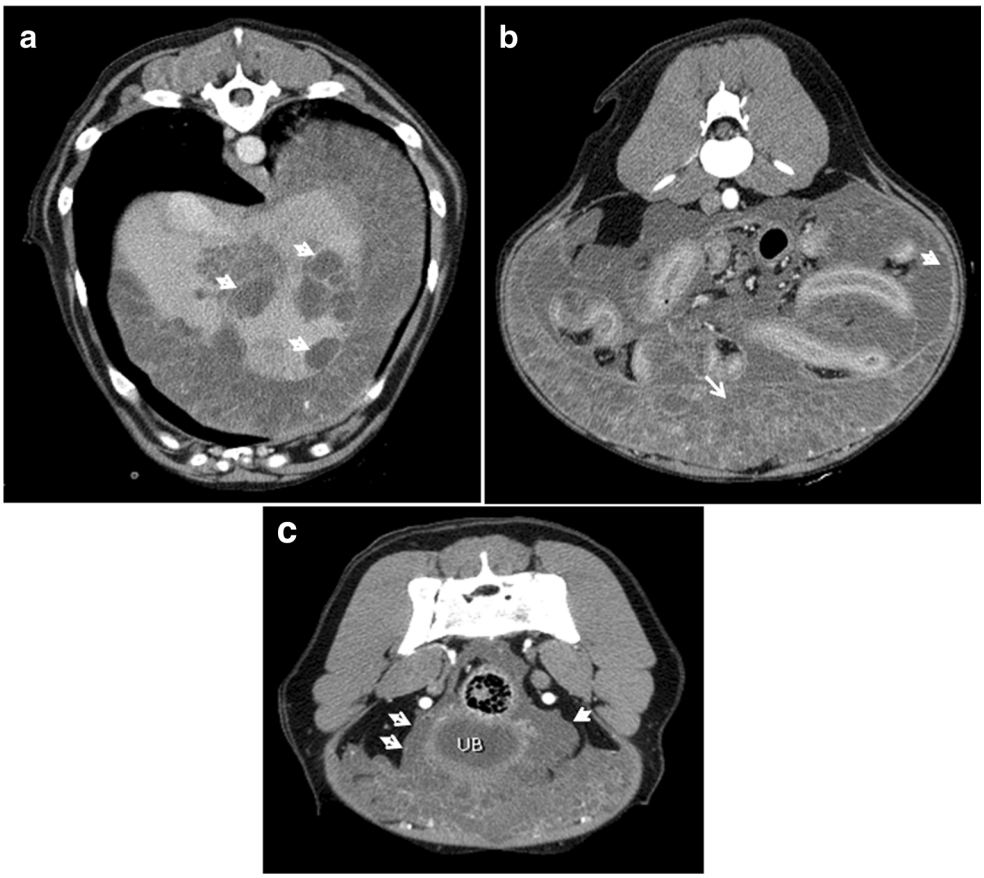

Fig. 1 Image from computed tomography (CT) scan of the abdomen of the infected dog. Image was taken after injection of iohexol contrast solution (OMNIPAQUE, GE Healthcare) at 1294 mg/kg. **a** Cranial abdomen, liver showing multiple irregular cystic structures (arrowheads). **b** Mid abdomen, ascites (arrowhead) and thick cystic tissue showing distinct heterogeneous contrast uptake close to the peritoneal lining (arrow). **c** Caudal abdomen, urinary bladder (UB) encased within proliferative cystic tissue (arrowheads)

The resultant PCR amplicons were sequenced. DNA sequence analysis by BLAST (https://blast.ncbi.nlm.nih.gov/Blast.cgi) showed that amplicons shared 99% identity with *Mesocestoides* spp. cox1 gene sequence (accession number KP941432.2) and 100% identity with those of *M. corti/vogae* rrN gene sequence (accession number HM011122.1). The sequences from the cox1 and rrN amplicons were deposited in GenBank as accession numbers MK095584 (amplicon size 407 bp) and MH843161.1 (amplicon size 267 bp), respectively. Fecal flotations from the dog using Sheather's sugar solution (SG = 1.27) were consistently negative for *Mesocestoides* eggs.

Treatment with a praziquantel solution (Vetbancid, CP-Pharma GmbH, Burgdorf, Germany) injected subcutaneously at 5 mg/kg once every 12–13 days was repeated four times. Interestingly, the owners reported that their dog became hyperthermic and weak with periodic vomiting after each deworming.

On physical examination 2 months after the beginning of therapy, the dog was still ascitic with roughly the same volume of abdominal fluid as assessed by ultrasonography. However, it had lost about 0.5 kg in body weight. Aspirated abdominal fluid contained viable *Mesocestoides* tetrathyridia evident by light microscopy of unstained fluid. In CBC, marked eosinophilia was noted (eosinophils 2.74 K/μl, RI 0.06–1.23 K/μl), and in addition, a high serum alkaline phosphatase activity (ALKP 218 U/L, RI 23–212 U/L) and high globulins level (GLOB 5.2 g/dL, RI 2.5–4.5 g/dL) were present. Treatment was thus switched to a 4-week course of fenbendazole at 100 mg/kg P.O. q 12 h (Fenbendazole, Vetmarket, Shoham, Israel) as described by Boyce et al. (2011).

Following 28 days of treatment with fenbenzazole, the dog appeared to be in good health with normal appetite and regained normal urinary control. In addition, it had less clinically noticeable swelling of its abdomen although it gained about 1 kg in weight. Ultrasound imaging of the peritoneal cavity showed no visual free fluid fraction but still depicted cysts. Lastly, it was not possible to retrieve any free fluid on an abdominal tap, although a few partially digested parasitic *Mesocestoides* tetrathyroidia were aspirated from cysts.

Discussion

Mesocestoides spp. are widely distributed in the northern and southern hemisphere, infecting wild carnivore populations with prevalence rates as high as 84.1% (Karamon et al. 2018). In Europe, at least two species of *Mesocestoides* are known to occur in carnivores, including dogs, cats, and wild foxes: *Mesocestoides litteratus*, which is the most common

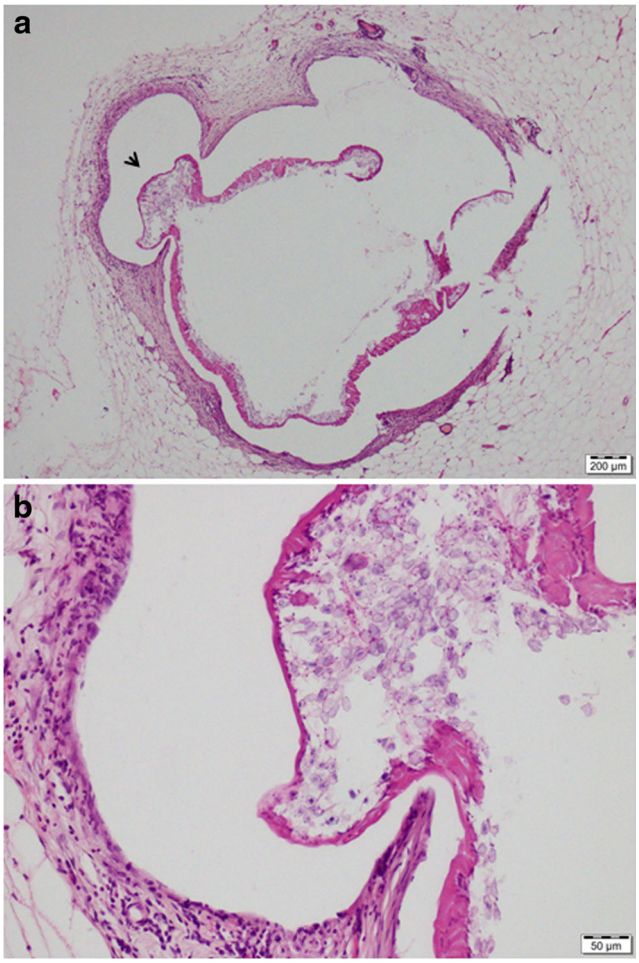

Fig. 2 Histopathologic preparation of cyst tissue resected from the dog abdomen, showing cestode larvae. **a** Mesenteric fat with a thin fibrous capsule surrounding a larva. Arrowhead indicates area seen in detail in **b**. Hematoxylin and eosin (H&E) × 4 magnification. **b** Higher magnification from **a**. The larva is surrounded by a thin wall of fibrous tissue with minimal to mild lymphocyte infiltration. Note the smooth tegument and fine basophilic calcareous corpuscles. H&E, × 20 magnification

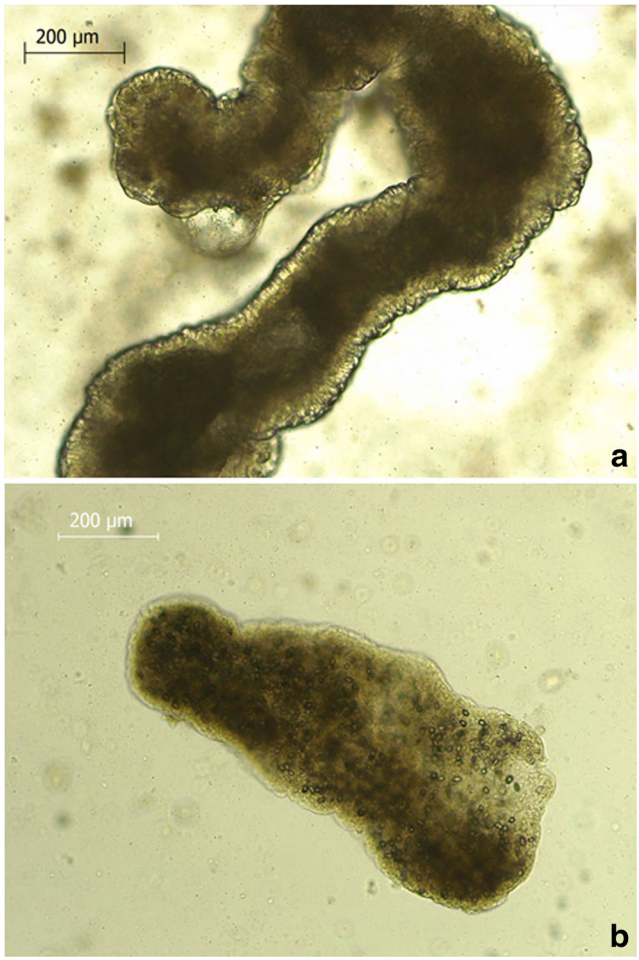

Fig. 3 **a**, **b** Photomicrographs of acephalic tetrathyridia removed from abdominal effusion

species, and *Mesocestoides lineatus* (Deplazes et al. 2016, pp. 213–214). Tetrathyridia of *M. vogae* and of unidentified other species are known to occasionally cause CLPC in mammals in Europe, the USA, and elsewhere (Crosbie et al. 1998; Kashiide et al. 2014). Although an infrequent observation, infection from *Mesocestoides* spp. has been reported in humans from Korea (Eom et al. 1992) and the USA (Fuentes et al. 2003).

Since the dog in the current report was housed entirely indoors in Massachusetts, USA, exposure to *Mesocestoides* from an infected intermediate host is unlikely. In addition, *Mesocestoides* spp. have not been described in Eastern United States so far (Crosbie et al. 2000b). *Mesocestoides* spp. have been previously reported in Israel, but only in wild carnivores such as a mongoose (*Herpestes ichneumon*), three golden jackals (*Canis aureus*), three red foxes (*Vulpes vulpes*), and a hyena (*Hyaena hyaena*; Loos-Frank 1990). However, cases of *Mesocestoides* infection in dogs have been reported elsewhere in the Middle East and in neighboring countries (El-Shehabi et al. 1999; Dalimi et al. 2006). The reported dog had free access to the outdoor area in Israel, and therefore, exposure to potentially infected intermediate hosts was highly feasible, supporting the fact that in the case presented here, infection is likely to be autochthonous.

Although the dog described in this report had signs of urinary incontinence, it was vibrant and active and had a healthy appetite. Blood tests from the dog showed only eosinophilia, suggesting the presence of helminth-associated infection, and high alkaline phosphatase activity, suggesting inflammation of the bile system or possibly a side effect to the drug used for anthelmintic treatment. If initial radiography and ultrasound had not been performed, parasitic ascites would not have been suspected. The ultrasound findings were typical of CPLC as previously described (Venco et al. 2005), including the presence of ascites and abdominal cysts. However, to the best of our knowledge, this report is unique in describing urinary incontinence associated with *M. vogae* infection. Crosbie and others (1998) have described a case of dysuria

in a female dog infected with *Mesocestoides* spp. but it did not display loss of urination control as in the present case. Observation of fibrous tissue encasing the urinary bladder in the dog reported here was visualized after examination by CT and was confirmed after performing a laparotomy. The fibrous-lined cystic tissue likely adhered to urinary bladder wall limiting its free expansion or disturbed the closure of the urethral sphincter, thus causing the dog to show signs of urinary incontinence.

Routine fecal examinations of the dog were always negative. The sensitivity of fecal flotation for the detection of *Mesocestoides* eggs has been found by others to be poor (Széll et al. 2015). The preferred diagnostic method for morphological description would be microscopic evaluation of a whole proglottid, which is rarely found in feces (Tassi and Widenhorn 1977; Barutzki and Schaper 2011). Thus, *Mesocestoides* spp. infection cannot be ruled out based on negative fecal examination. Since no adult stage is known this far for *M. vogae*, this may also explain lack of the detection of eggs in the feces. Accurate morphological description of the *Mesocestoides* spp. was not possible, and it is not feasible even using morphological keys as few if any distinctive morphological features are present in tetrathyridia, let alone where acephalic larvae are considered as was in this case. Only sequencing of amplified DNA from the larvae showed amplicons homologous to *M. vogae*. Even though phenylpropanolamine was administered to the dog to alleviate its urinary incontinence, no significant improvement was observed until effective treatment against *Mesocestoides* was initiated.

Praziquantel (Kashiide et al. 2014) and fenbendazole (Crosbie et al. 1998) are the two main drugs reported to be used in CPLC in dogs, with varying effectivity. Praziquantel is the drug most commonly used against cestode infections, and it has marked anthelmintic activity against a wide range of adult and larval cestodes (Miró et al. 2007). In the case presented here, praziquantel did not reduce the viability of worms in the abdominal parasitic ascitic fluid. This observation may have been influenced by several factors, including a late diagnosis of infection at the stage where an overwhelming number of viable peritoneal tetrathyridia had managed to reproduce and persist or the use of a low dose of praziquantel (5 mg/kg). A survival analysis report of dogs diagnosed with CPLC due to *Mesocestoides* spp. (Boyce et al. 2011) has also found that praziquantel was not effective against *Mesocestoides* larvae in the peritoneal cavity. Instead, the authors observed better survival of dogs with peritoneal parasitic cestodiasis that were treated with fenbendazole. The authors recommend that affected dogs be given 100 mg/kg P.O. twice daily for 28 consecutive days. This treatment regime also proved to be effective in the described case by alleviating ascites. Most importantly for the owners of the dog, treatment enabled it to regain urinary control. Improvement in urine bladder function may be the result of a reduction in the size of parasitic cysts that surrounded it due to elimination of the larvae, the alleviation of pressure exerted on it by ascitic fluids, or a combination of both factors.

Side effects of nausea and lethargy have been reported after treatment of dogs with praziquantel, especially after injection (Plumb 2008). This may explain the dog's clinical signs after each treatment with this drug. Alternatively, death of larvae with release of toxins may also be responsible for this phenomenon.

In conclusion, although *M. vogae* is rarely observed as a cause of disease in dogs, migration of larval forms outside of the intestinal tract may cause various clinical syndromes, including urinary incontinence as demonstrated in this case. This dog, like others that had received fenbendazole therapy for CPLC, fully recovered from urinary incontinence after treatment and the resultant elimination of parasitic larvae in the peritoneal cavity. CPLC should be added as a differential in the list of diseases causing urinary incontinence in dogs in areas where *Mesocestoides* spp. are present, which presently include Israel.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

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