Vol 30, Issue 5, October 2016

VETERINARIA

Peritoneal cestodiasis in a dog of Central Italy

Fabio Spina[1*], Med Vet, PhD

Silvia Rossi[2], Med Vet, Dipl. ECVCD

Giliola Spattini[3], Med Vet, PhD, Dipl. ECVDI

Peritoneal cestodiasis is a parasitic disease, well described in the veterinary literature, consisting of exudative peritonitis caused by larval stage (Tetrathyridium) proliferations of *Mesocestoides* spp. This disease syndrome has been termed canine peritoneal larval cestodiasis (CPLC). The disease is unusual in Italy and the only cases reported were in Northern Italy. This report describes a case of asymptomatic peritoneal cestodiasis in Central Italy (Rome). A 7-year-old, male, cross-breed dog was referred for abdominal ultrasound for a severe asymmetric testicle hyperplasia. The images revealed peritoneal reactivity with diffuse abdominal cystic structures and diffuse hyperechoic fluid. Abdominocentesis detected the presence of lactescent fluid. The cytological examination was consistent with neutrophilic peritonitis associated with peritoneal cestodiasis.

Key words - Peritoneal cestodiasis, dog, parasitosis, *Mesocestoides*, CPLC.

INTRODUCTION

Adult parasites of the genus *Mesocestoides* spp. are responsible for the infestation of dogs, cats and wild animals in Europe, North America and Asia.[1] The life cycle of the parasite is not yet fully understood but it is believed to require two intermediate and a definitive host.[2] In the dog and cat, the asexual larval forms of the parasite (tetrahyiridium) may be the cause of a severe form of peritonitis, characterised by abundant effusion. The syndrome is defined as canine peritoneal larval cestodiasis (CPLC). The disease has been sporadically reported also in the cat.[3,4] Dogs with this condition may be asymptomatic or have nonspecific symptoms such as ascites, anorexia and reduced athletic performance. Although CPLC is believed to be potentially fatal, subclinical infections detected accidentally during surgery have been reported.[1,5]
Biochemical blood tests and coprological examinations are generally not diagnostic; many authors have suggested that cytology, with identification of the larval stages of the parasite or of the calcareous corpuscles in the abdominal fluid, and Polymerase Chain Reaction (PCR) of the larval tissues are essential for the *in vivo* diagnosis.[1,6]

The larvae of *Mesocestoides* spp. are responsible for a syndrome termed canine peritoneal cestodiasis (CPLC).

CLINICAL CASE

A 30 kg, 7-year-old, intact, male, cross-breed dog was presented for an ultrasound urogenital evaluation for severe hyperplasia of the left testicle. The clinical history reported a previous diagnosis of a form of leishmaniasis and the patient was currently under treatment with allopurinol (10 mg/kg PO q12h). The dog was regularly vaccinated and treated annually with anthelmintic formulations based on Pyrantel, Febantel and Praziquantel. Food and water intake appeared

[1] Libero professionista, Roma
[2] Laboratorio di analisi CDVet, Roma
[3] Clinica Veterinaria Castellarano (RE)
*Corresponding Author (fabiospina69@gmail.com)

Received: 22/09/2015 - Accepted: 05/07/2016

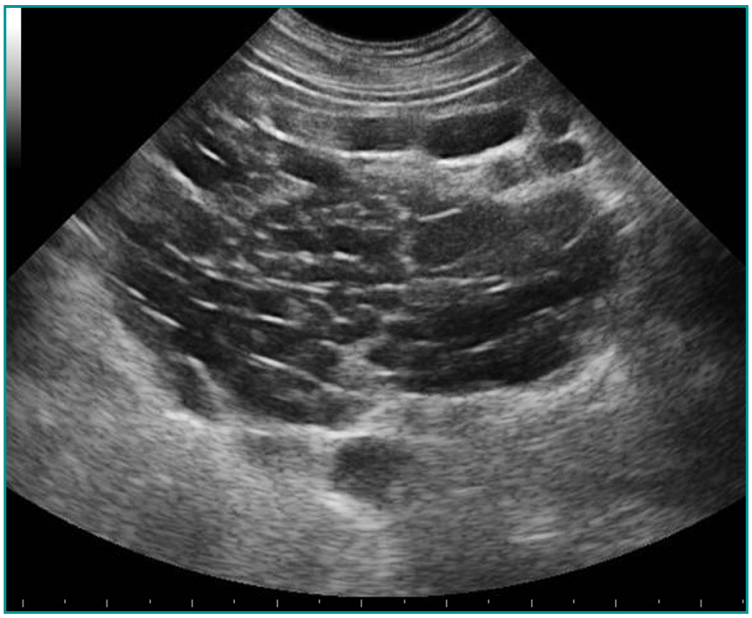

Figure 1 - Ultrasound image of a large-size, septated cyst. Left paramedian longitudinal scan.

unchanged and no organic function abnormality was reported.

The patient was in good nutritional status and properly hydrated. The physical examination was normal and the only detectable abnormalities consisted in testicular hypertrophy and moderate abdominal distention without signs of pain on palpation.

Blood and biochemical tests detected the presence of neutrophilic leukocytosis (20.19 10^3/µl; range 5.2-13.9), with the presence of toxic neutrophils associated with monocytosis (1352.7/µl; range 200-1100) and oeosinophilia, (807.6/µl; range 0-600) and hyperamylasaemia (1612 IU/L; range 450-1200). The electrophoresis of serum proteins detected the presence of increased β globulins (2.69 /dl; range 0.50 to 2.40) and a reduction in the Alb/Glob ratio (0.59; range 0.80-1.90).

Abdominal ultrasonography revealed the presence of diffuse peritoneal fat hyperechogenicity, free fluid rich with hyperechoic foci in suspension and numerous cystic lesions at omental level. The cysts were of variable size, but still under 3 cm, with a thin hyperechoic wall and characterised by the presence of septa within the predominantly anechoic content. Splenomegaly was also present, associated with the presence of a non-specific focal lesion, enlargement of the mesenteric and right colic lymphocentres, increased prostatic diameter associated with the presence of a diffuse microcystic pattern (suggestive of cystic prostatic hyperplasia) and the presence of a large-size heterogeneous focal lesion (mass) of suspected neoplastic origin at the level of left testicle, cause of the clinically-detected hypertrophy.

Abdominocentesis allowed to collect 2.5 ml of lactescent fluid that was placed in a test tube containing K3EDTA and sent to the reference laboratory for cytology. The microscopic examination of the sample revealed increased cellularity with a clearly prevalent population of partially degenerated neutrophilic granulocytes and the absence of overt bacterial phagocytosis. Occasional macrophages and voluminous clusters of cells were also present, with small pink granular nuclei and abundant, basophilic and non-homogeneous cytoplasm, containing rounded, oval or polygonal refractive granules (calcareous corpuscles). In view of the sample's characteristics (presence of debris and suspended material) the instrumental total cell count was not possible; the protein content, measured by refractometer, was of 3.8 g/dl. The cytological pattern was compatible with neutrophilic inflammation associated with peritoneal cestodiasis.

Diffuse peritoneal cysts were detected in an asymptomatic patient undergoing and ultrasound examination of the genital tract.

The patient was treated with Fenbendazole (Panacur®, MSD Animal Health Srl, Segrate, Milan, Italy), 50 mg/kg PO daily for 10 days and Praziquantel (Droncit®, Bayer AG, Leverkusen, Germany), 5 mg/kg SC in a single dose repeated after 15 days.

After 30 days the patient returned for a clinical and ultrasonographic control. In the clinical history nothing unusual was reported. At physical examination the ab-

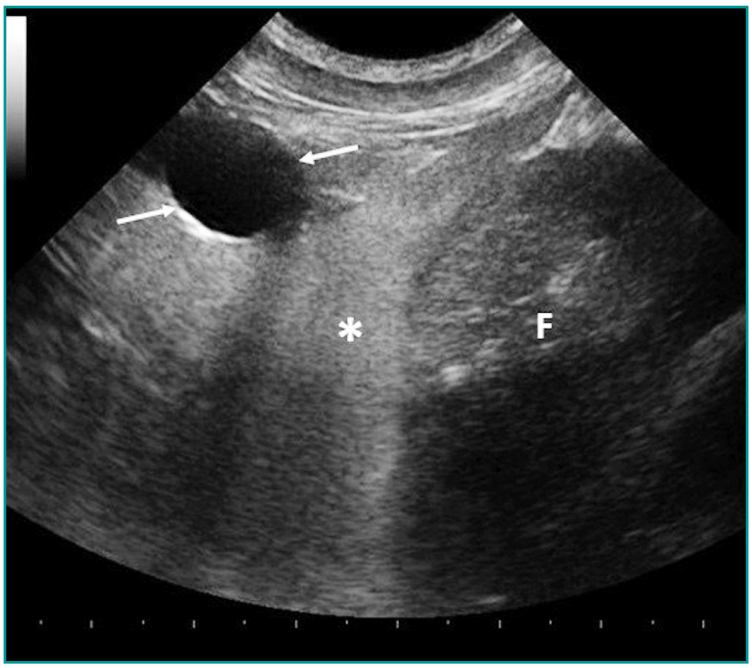

Figure 2 - Ultrasound image obtained from the median longitudinal scan of the abdomen. Thin-walled anechoic cyst (arrows) cranial to the liver (L). The lesion appears surrounded by hyperechoic fluid (*).

dominal distension appeared reduced but persistent. The ultrasound examination showed the persistence of the cystic lesions and of the abdominal fluid, which however appeared significantly reduced in number and in volume. The previously encountered lymphadenomegaly was instead no longer appreciable. The other findings were overlapping with those of the previous assessment.

In view of the asymptomatic status of the patient a follow-up clinical and ultrasonographic control was suggested after thirty days, in order to evaluate the opportunity for an additional therapeutic cycle. The owner decided instead not to proceed with any additional control or therapy.

DISCUSSION

Larval forms of *Mesocestoides* spp. are the cause of a pathological syndrome known as canine peritoneal larval cestodiasis (CPLC). In the scientific literature cases have been reported in the USA[2,6] and, sporadically, in Italy, Turkey, Japan and Germany.[1,3,4,5,7,8,9]

The biological cycle of the parasite has not been fully explained in all species, although it appears to include at least two intermediate hosts in which the larvae develop. The dog is infested by ingesting the intermediate hosts, in which the metacestoid stage of the parasite (tetrathyridium) is present (amphibians, reptiles or birds). Once in the intestine, the larvae mature into the adult form; some may however penetrate through the gut and invade the abdominal cavity.

Although the syndrome has been reported to be fatal in both the dog and the cat, many asymptomatic forms discovered by chance have been reported,[1,2,8] often during routine surgical procedures (sterilisation). In view of these findings the disease is probably underdiagnosed. In other cases, affected patients present nonspecific symptoms such as anorexia, vomiting, weight loss, depression and abdominal distension.[1,2,3,4]

The treatment of peritoneal cestodiasis is challenging and the complete removal of the larval forms of the parasite from infested patients is often not possible.

In our case, the patient was completely asymptomatic and the detection of the infestation was secondary to an ultrasound examination performed for reasons not related to the disease. The peritoneal infestation and the consequent multiplication of the parasite was caused by a mechanism not fully known still today. Some experimental studies in mice have shown an increased sensitivity to infestation in male subjects undergoing immunosuppressive therapy with corticosteroids. On the contrary, in a 2011 study on 60 dogs Boyce *et al.* did not find any correlation between the genre of the infected subjects and their survival, although some of the patients that showed major clinical symptoms were indeed undergoing corticosteroid treatment for various reasons. Chronic treatment with corticosteroids in patients with peritoneal cestodiasis has also been reported by other authors.[5]

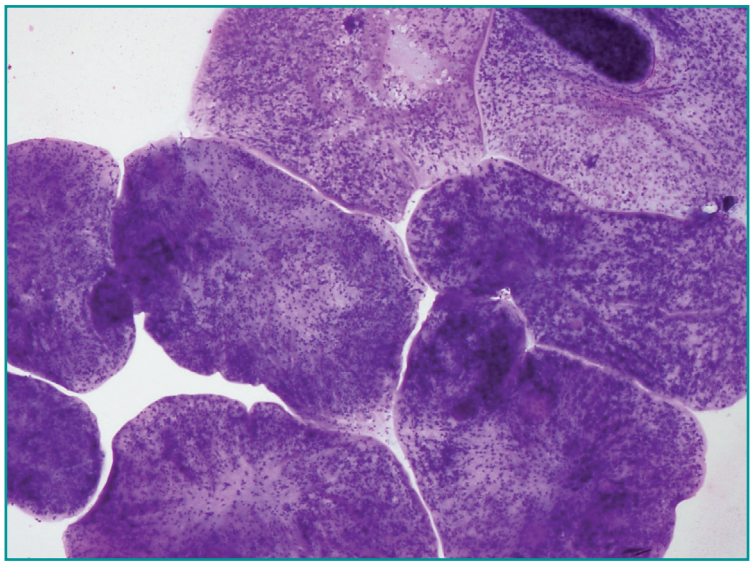

Figure 3 - Acephalic larval forms. (May Grunwald Giemsa, 100X).

The treatment of peritoneal cestodiasis is challenging and the complete removal of the larval forms of the parasite from infested patients is often not possible.[5]

To date, reported treatments include the use of fenbendazole, alone or in combination with surgical removal of the cysts and peritoneal lavage, or of praziquantel, alone or in combination with the administration of fenbendazole.

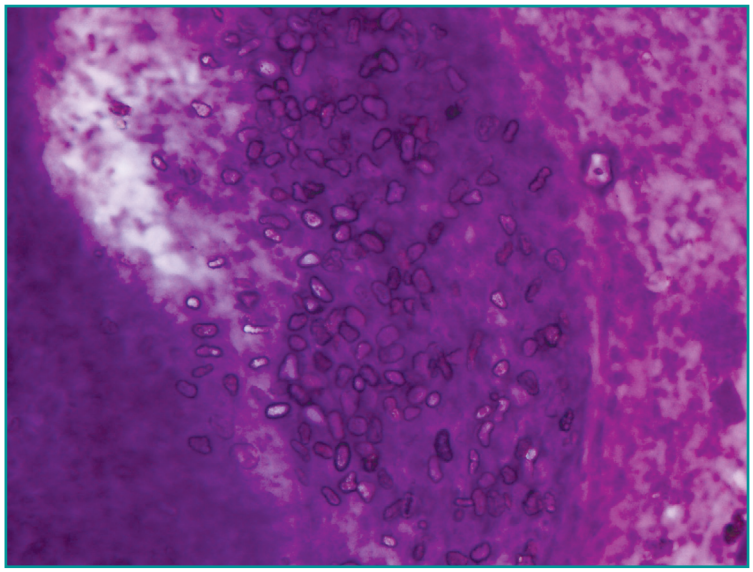

Figure 4 - Calcareous corpuscles immersed in a larval tissue fragment. (May Grunwald Giemsa, 400X)

Several authors have reported the efficacy of praziquantel subcutaneously at the dose of 5 mg/kg in a single dose repeated after 14 days.[5,6,8] In other papers, fenbendazole was used at the dose of 50-100 mg/kg BID orally for variable time periods.[1,8] It should however be reported that these studies have always concerned a limited number of animals (1-2 dogs). The only study carried out on a larger sample (Boyce, 60 dogs) reported the lack of efficacy of praziquantel in eliminating the larval forms in the course of peritoneal cestodiasis. The authors concluded that fenbendazole at the dose of 100 mg/kg BID for 28 days in combination with peritoneal lavage resulted to be the most effective therapeutic option for the resolution of the pathology.

The lethal dose of fenbendazole is very high and its therapeutic use at high doses is well tolerated by patients.[2] However, peritoneal cestodiasis may still relapse and hence a continuous follow-up is necessary for the proper monitoring of the disorder.

In the patient examined, at subjective evaluation the therapy used reduced the severity of the infestation, reducing the number of cysts identified ultrasonographically and the amount of free fluid present. However, it did not eliminate the larvae *in toto*. It is also necessary to point out that in resistant and/or relapsing cases some authors have reported the chronic treatment with fenbendazole (100 mg/kg BID *per os*), possibly alternated with short periods of suspension in order to reduce the risk of drug toxicity.

It should finally be recalled that in the case described since it was not possible to carry out a targeted molecular assay (PCR) - due to the owner's non-compliance - the diagnosis of mesocestodiasis was presumptive and based on the rarity of conditions caused by other tapeworms.

To our knowledge, this is the first case of peritoneal cestodiasis reported in Rome, an extremely relevant epidemiological event.

ACKNOWLEDGEMENTS

The authors thank Dr. Mercedes Paolillo for having referred the case.

KEY POINTS

- ??????????????
- ??????????????
- ??????????????

BIBLIOGRAFIA

1. Venco L, Kramer L, Pagliaro L *et al.* Ultrasonographic features of peritoneal cestodiasis caused by Mesocestoides sp. in a dog and in a cat. Veterinary Radiology & Ultrasound 46(5):417-22, Sep-Oct 2005.
2. Patten P, Rick L, Zaks K *et al.* Cestode infection in 2 dogs: cytologic findings in liver and a mesenteric lymph node. Veterinary Clinical Pathology 42(1): 103-8, Mar 2013.
3. Eleni C, Scaramozzino P, Busi M *et al.* Proliferative peritoneal and pleural cestodiasis in a cat caused by Metacestodes of *Mesocestoides Spp.* Anatomohistopatological findings and genetic identification. Parasite 14(1): 71-6, Mar 2007.
4. Haziroglu R, Ozgencil E, Guvenc T *et al.* Peritoneal tetrathyridiosis in a Siamese cat - a case report. Veterinarski Arhiv 75(5): 453-8, 2005.
5. Kashiide T, Matsumoto J, Yamaja Y e*t al.* Case report: First confirmed case of canine peritoneal larval cestodiasis caused by Mesocestoides vogae (syn. M. corti) in Japan. Veterinary Parasitology 17; 201(1-2): 154-7, Mar 2014.
6. Boyce W, Shender L, Schulz L *et al.* Survival analysis of dogs diagnosed with canine peritoneal larval cestodiasis (*Mesocestoides* spp.). Veterinary Parasitology 25; 180(3-4): 256-61, Aug 2011.
7. Bonfanti U, Bertazzolo W, Pagliaro L *et al.* Clinical, cytological and molecular evidence of *Mesocestoides* sp. Infection in a dog from Italy. Journal of Veterinary medicine A. Physiology, pathology and clinical medicine 51(9-10): 435-8, Dec 2004.
8. Papini R, Matteini A, Bandinelli P *et al.* Effectiveness of Praziquantel for treatment of peritoneal larval cestodiasis in dogs: A case report. Veterinary Parasitology 28; 170(1-2): 158-61, May 2010.
9. Wirtherle N, Wiemann A, Ottenjann M *et al.* First case of canine peritoneal larval cestodosis caused by *Mesocestoides lineatus* in Germany. Parasitology International 56(4): 317-20, Dec 2007.