"A Life Saving Neuter"

A Case Report of Canine Peritoneal Larval Cestodiasis caused by *Mesocestoides*

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Class of 2020

Clinicopathological Conference

July 26th, 2019

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Introduction

Mesocestoides is a genus of tapeworms that in the United States is found in the western states, mainly California[2]. This tapeworm has the unique ability to invade the peritoneal cavity and undergo asexual reproduction, resulting in marked parasitic proliferation within the host animal[2]. This is the only genus of tapeworms in which asexual reproduction resulting in a massive infection has been reported[6]. Definitive hosts are mainly wild and domestic canids, although multiple species, including humans, can become infected. Veterinary interest in the species is largely related to the rare migration of ingested tetrathyridia into the abdominal cavity of domestic dogs and occasionally cats[7]. *Mesocestoides* infection in domestic dogs is termed canine peritoneal larval cestodiasis (CPLC) when involving the peritoneal cavity. Dogs become infected when they ingest either the first intermediate host, a coprophagous arthropod, or the second intermediate host which could be a rodent, bird, lizard, or snake[2].

The third larval stage will replicate and then proliferate within the peritoneal cavity. This causes peritoneal effusion and the development of a pyogranulomatous peritonitis with adhesions[6]. Clinical signs are vague and include lethargy, weight loss, vomiting, and ascites[3,5]. Occasionally subclinical cases are identified during routine ovariohysterectomies or castrations[5]. Secondary scrotal cestodiasis can occur and may be an early indicator of peritoneal cestodiasis in male dogs[8]. Scrotal swelling was one of the initial presenting complaints in several reported cases of *Mesocestoides*[7]. This can range from mild scrotal swelling to extensive testicular enlargement and necrosis[6]. Diagnosis of *Mesocestoides* is based on cytology of the abdominal fluid, which is usually exudative, and identification of the organism via cytology of the cysts[2]. Unfortunately, due to the invasive and proliferative nature of this condition, canine peritoneal larval cestodiasis is a life-threatening disease with a guarded to grave prognosis[1].

History and Presentation

Copper, a two-year-old male neutered Australian Shepherd, was adopted in California at eight weeks old. His previous medical history includes a parvovirus infection, tapeworms, and tick infestations. From there, he moved with his owner to Texas and then to Mississippi. Per his owners, Copper has always had digestive aliments including chronic diarrhea, vomiting bile when fed late in the morning, and periodic vomiting (once per month) after eating. He was previously treated with metronidazole and probiotics, and was switched to Hill's Sensitive Stomach, which improved his stool consistency. He receives Heartgard and Nexgard as monthly parasite preventives and is up to date on vaccines.

Copper presented on February 15, 2019 to his rDVM after a 1/4 x 1/2 inch nodule was noted on his testicle two to three weeks prior. Five days later, a castration was performed, and the affected testicle was submitted for histopathology. A fecal flotation was also completed at that time and no ova were observed. The pathology report was returned on February 25th and described a chronic, granulomatous and fibrosing orchitis and funiculitis with intralesional larval cestodes suggestive of *Mesocestoides* spp. He was then referred to the MSU-CVM Small Animal Internal Medicine Service for a thorough *Mesocestoides* work up.

Upon presentation to MSU-CVM on March 4th, 2019, Copper was nervous, alert, and responsive. He had a heart rate of 120 bpm, was panting, and had a temperature of 102.3°F. He had mild scleral hyperemia bilaterally and the buccal surface of the right maxillary carnassial tooth was chipped. No crackles or wheezes were auscultated, and no murmurs or arrhythmias were noted. His abdomen was tense but non-painful. Sedated digital rectal exam revealed a bilaterally enlarged prostate. His scrotum contained a large firm hematoma, and the prescrotal

incision was healing well. No peripheral lymphadenopathy was palpated. All other physical exam parameters were within normal limits.

Pathophysiology

The life cycle of the *Mesocestoides* tapeworm is not fully understood, but it is known to involve two intermediate hosts and one definitive host. It is thought the egg (first larval stage) is ingested by a coprophagous arthropod and develops into the second larval stage. The arthropod (first intermediate host) is ingested by a second intermediate host such as a rodent, bird, lizard, snake, or frog. Within the peritoneal cavity of the second intermediate host, the second larval stage develops into the third stage (tetrathyridium). The final adult form develops when the second intermediate host is ingested by the definitive host which can be raccoons, coyotes, foxes, cats, or even humans. Reported cases in dogs in the United States are mainly found in California. This regional distribution may be attributed to the dense coyote and western fence lizard populations, which are definitive and second intermediate hosts, respectively. The adult form of the tapeworm develops within the intestines of the second intermediate host. The dog can serve as either the second intermediate host or the definitive host. Peritoneal infections can occur in both the second intermediate host and the definitive host because ingested tetrathyridia can penetrate the intestinal wall[2]. Tetrathyridia, the third larval stage, will replicate within the intestines, penetrate the gut wall, and proliferate in the peritoneal cavity, causing peritoneal effusion with the development of a pyogranulomatous peritonitis with adhesions[6].

Clinical signs include depression, anorexia, vomiting, diarrhea, weight loss, pyrexia, and poor haircoat, and eventually ascites abdominal distention, and sometimes dyspnea. Lesions of the peritoneal cavity include ascites with severe fibrinous peritonitis, villous proliferation,

serosal adhesions, and cyst formation. The cysts contain larvae, inflammatory cells, and calcified granules called calcareous corpuscles which may be free or within the larvae[2]. Larval invasion into the viscera indicates a more systemic infection and a worse prognosis. Peritoneal cestodiasis with secondary scrotal cestodiasis is caused by the migration of the tetrathyridia from the abdominal cavity through the scrotum via the tunica vaginalis and then into the testicular parenchyma from the scrotum[4,6]. The severity of the infection depends on when it is first observed clinically. It can be a mild scrotal swelling, as in Copper's case, or it can cause extensive testicular enlargement and necrosis, as well as numerous tetrathyridia occupying the testicular parenchyma[6].

Diagnostic Approach & Considerations

Copper's case is unusual since he had a diagnosis upon presentation to MSU-CVM. The testicle submitted for histopathology by his referring DVM identified that his scrotal swelling was a result of a *Mesocestoides* infection. This was an incidental finding as Copper was not displaying any obvious clinical signs besides occasional vomiting and diarrhea. *Mesocestoides* would not be a top differential for these signs due to its rare nature. The negative result from the fecal floatation performed by the rDVM is common in dogs with *Mesocestoides* larval infection, possibly indicating that ingestion of the first intermediate host is more common than ingestion of the second intermediate host[2]. This stresses the importance of submitting any lesions which appear abnormal for histopathology, to confirm what they may be. The referring veterinarian was concerned about the nodule on the testicle being testicular neoplasia, but the histopathology report identified the lesions as being suggestive of *Mesocestoides* spp.

Upon presenting to MSU-CVM, several diagnostic tests were performed to work-up this unusual condition. A serum chemistry and complete blood count were conducted. The chemistry showed moderate hypoalbuminemia (1.9 g/dl with reference range 2.5-3.9 g/dl), mild hyperglobulinemia (4.8 g/dl with reference range 2.1-4.3 g/dl) and mild hypomagnesemia (1.3 mg/dl with reference range 1.7-2.4 mg/dl). The changes in albumin and globulins are consistent with a parasitic infection. The chemistry also showed a mild elevation in glucose (128 mg/dl with reference range 74-125 mg/dl). On CBC the MCV was 78.4fL (reference range 63-77fL) and lymphocytes 1168/uL (reference range 1200-6500/uL). No other abnormalities were noted on bloodwork. Urinalysis showed a pH of 8.0. Thoracic and abdominal radiographs were taken, and an abdominal ultrasound was also performed with fine needle aspirates of abdominal lymph nodes and cystic structures to be submitted for cytology.

Thoracic radiographs revealed a mild, diffuse unstructured interstitial pulmonary pattern, which could be attributed to atelectasis, pneumonitis, or infectious etiologies. Abdominal radiographs revealed moderately decreased abdominal serosal detail with wispy soft tissue opaque material superimposed over the ventral falciform fat. The decreased abdominal serosal detail prevented complete evaluation of most abdominal organs (liver, spleen, urinary bladder, and small bowel) and may have been due to peritonitis secondary to the reported *Mesocestoides* infection. Other possible etiologies include peritoneal effusion such as a modified transudate, exudate, or hemorrhage.

Abdominal ultrasound found that the mesentery and peritoneal fat (including the falciform fat) were diffusely hyperechoic. Differentials for this included peritonitis and steatitis (inflammation of fatty tissue). There were also multiple variably sized, ovoid, smoothly marginated, hypoechoic nodules with thin, hyperechoic rims within the mesentery and peritoneal

fat, which demonstrated distal acoustic shadowing. The largest of these measured up to 0.48 cm in thickness. As explained in the radiology report, the hypoechoic nodules throughout the mesentery and peritoneal fat were considered most likely to be parasitic cysts, with lesser consideration given to granulomatous disease or neoplasia (as with carcinomatosis). There were a few mild to moderately enlarged, hypo- to hyperechoic lymph nodes (splenic, caudal aortic, left and right medial iliac, and internal iliac lymph nodes). The largest of these was the right medial iliac lymph node which measured up to 1.07 cm in thickness. Differentials for the multifocal lymphadenopathy included reactive lymphadenopathy or metastatic neoplasia. The prostate was heterogeneous in echogenicity with multiple partially well-defined hyperechoic regions. The appearance of the prostate could have been due to resolving benign prostatic hyperplasia (given the recent history of castration), with prostatitis or neoplasia not entirely excluded, but considered less likely.

While sedated for imaging, fine needle aspirates of the iliac lymph nodes and cystic structures were taken and submitted for cytological analysis. The lymph node aspirate came back as inconclusive with peripheral blood constituents. The aspirated omental cyst came back as a *Mesocestoides* infection with moderate suppurative inflammation. Scattered throughout the preparations were several fragmented larvae which appeared compatible with *Mesocestoides*. Internally, the organisms contained significant amounts of a blue pink fibrillar-like substance, numerous pyknotic appearing nuclei and numerous crystalline-like spheres which were compatible with calcareous corpuscles. Calcareous corpuscles are remnant organelles of both larvae and adults and are specific to cestodes. When observed in peritoneal fluid, they are pathopneumonic for infection with a larval cestode[2]. There was also an increased number of neutrophils present with lower numbers of macrophages and small lymphocytes among them.

All of these findings were consistent with Canine Peritoneal Larval Cestodiasis (CPLC) by *Mesocestoides* spp. infection.

Treatment & Management

With canine peritoneal larval cestodiasis, aggressive treatment strongly influences the likelihood of survival. Recommended treatment involves peritoneal lavage and/or long-term treatment with fenbendazole[3,5]. In one study looking at eleven dogs infected with peritoneal infections caused by *Mesocestoides* spp., five dogs were treated with praziquantel and albendazole and six were treated with fenbendazole. Those treated with praziquantel and albendazole did not see resolution, however five of the six dogs treated with fenbendazole successfully cleared their *Mesocestoides* infections[3]. Dogs treated using high doses of fenbendazole at 100 mg/kg orally twice per day for 28 consecutive days or by both peritoneal lavage/surgery and any dose of fenbendazole had less than 20% the risk of dying compared to those not similarly treated. Praziquantel, which is commonly used to treat adult tapeworm infections in the small intestines was not found to be effective against *Mesocestoides* larvae in the peritoneal cavity.

Unfortunately, even after 28 days of high dose treatment, some dogs suffered a reoccurrence of clinical disease with larvae again recovered from their peritoneal cavity months or years later[1].

A survival analysis study was performed on dogs with canine peritoneal larval cestodiasis and found that CPLC is a life-threatening disease with a guarded to grave prognosis. Survival at six months and one-year post-diagnosis were only 72.3% and 60.5% respectively. Ascites is an important diagnostic and prognostic indicator since the volume of fluid is related to parasite

multiplication and accumulation. For example, in one of the cases in the survival study, the authors determined that over ten million larvae were present in four liters of fluid recovered by peritoneal lavage. They also attempted to evaluate factors influencing initial infection and subsequent parasite multiplication in the peritoneal cavity of dogs. Although studies with rodents suggest that glucocorticoid administration and male sex may increase susceptibility, the survival study authors did not find any significant relationship between survival and either sex or spay/neuter status. It is possible that severely affected dogs are immunocompromised and unable to control parasite multiplication in the peritoneal cavity. Although several dogs in survival study with high clinical severity scores had a history of glucocorticoid administration (given for other disorders), the study lacked sufficient sample size and data to draw any conclusions about the influence of immunity or glucocorticoid administration on host survival[1]. This is a reasonable theory in Copper's case since a parvovirus infection could have left him immunocompromised during the time he may have been exposed to the *Mesocestoides* larvae.

The plan for Copper was to monitor him for signs of worsening disease such as abdominal distension, vomiting, diarrhea, inappetence, and difficulty breathing. He was sent home with fenbendazole at 100 mg/kg to be given every twelve hours for one month and Drontal Plus (praziquantel) with one tablet being given by mouth every fourteen days for one month. He was then scheduled for a recheck examination with the MSU-CVM Small Animal Internal Medicine service one month from his initial visit for a repeat abdominal ultrasound with possible aspirates. Four days after his initial visit, however, Copper was switched to the granular form of fenbendazole which can be mixed into his food since he was unable to keep the large volume (26 mls BID) of liquid down.

In conclusion, canine peritoneal larval cestodiasis is an uncommon cause of ascites in dogs and can cause a severe fibrinous peritonitis that can be fatal, especially if there is deep involvement of the viscera. It carries a guarded to grave prognosis with many previously reported cases being fatal or necessitating euthanasia[2]. However, treatment with high doses of fenbendazole have shown success in some cases with the possibility of reoccurrence months to years later.

Case Outcome

At Copper's recheck one month later on April 4th, a repeat chemistry, CBC, urinalysis, urine protein creatinine ratio and abdominal ultrasound were performed. Upon physical exam his abdomen was tense but non-painful and a pea size mass was palpated on the left side in front of the inguinal area. His scrotum was healing well and contained thickened scar tissue. No peripheral lymphadenopathy was palpated. The remainder of his physical exam was unchanged. The chemistry showed moderate, but stable hypoalbuminemia, mild hyperglobulinemia, and mild hypomagnesemia. The CBC revealed a mild leukopenia and a monocytopenia. A urinalysis was checked to make sure a protein-losing nephropathy was not contributing to the persistent hypoalbuminemia. No significant proteinuria was noted. The urine protein creatinine ratio was normal at 0.2 with the normal in a dog being less than 0.5. The hypoalbuminemia was likely due to residual parasitism; however, if the albumin did not begin to increase, additional diagnostics were suggested during future visits, such as attempting re-aspiration of any residual lesions in the abdomen and/or consulting with a parasitologist.

The abdominal ultrasound showed the mesentery and peritoneal fat remained mildly, diffusely hyperechoic, and there were now multiple ill-defined, variably sized, hypoechoic

regions within the mesentery. The previously described anechoic nodules within the mesentery and peritoneal fat were no longer identified. The hypoechoic regions within the mesentery may be due to peritonitis and/or resolving *Mesocestoides*. The splenic, caudal aortic, and internal iliac lymph nodes were no longer enlarged. The previously described bilateral medial iliac lymphadenopathy was persistent, with the right iliac measuring up to 0.72 cm in thickness. There was also a mildly enlarged right colic lymph node, which measured up to 0.74 cm in thickness. This indicated that the prior reactive lymphadenopathy was improving.

The plan for Copper was to continue monitoring him for signs of worsening disease such as abdominal distension, vomiting, diarrhea, inappetence, and difficulty breathing. He was sent home with more Fenbendazole at 100 mg/kg, but the dose was reduced from twice per day to once per day. The original dose, which has been rarely reported to cause bone marrow suppression, was decreased due to his leukopenia. He was also sent home with more Drontal Plus being given at one tablet by mouth every fourteen days for one month. It was recommended for Copper to have repeat bloodwork done either with MSU-CVM or his referring veterinarian in one week (April 11th) to recheck his albumin levels. No follow up with the owner has been successful.

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