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# What Is Your Diagnosis?

In collaboration with the American College of Veterinary Radiology

## History

A 2-year-old 28-kg castrated male Australian Shepherd that was adopted from California at 8 weeks of age and traveled across the southwestern and southern US was referred for follow-up care and management after removal of a hard testicular mass (approx 2.5 to 5 mm in diameter) and castration by the primary veterinarian. The mass had been present for about 2 to 3 weeks prior to surgery, and purulent discharge during surgery was noted. In addition to surgery, the primary veterinarian performed a fecal examination, which revealed no evidence of parasitism. Monthly heartworm and flea and tick preventatives were prescribed. The mass was submitted for histopathology, the results of which indicated chronic, granulomatous, and fibrotic orchitis with intralesional larvae.

On physical examination, the dog was alert and responsive and had all vital signs within reference limits, a body condition score of 5/9, and mild scleral hyperemia bilaterally. No crackles or wheezes were auscultated, and clinically normal bronchovesicular lung sounds were present. No murmurs or arrhythmias were noted. The dog's abdomen was tense, but palpation did not elicit signs of pain. The dog was sedated with dexmedetomidine (2.5 µg/kg, IV) and butorphanol (0.2 mg/kg, IV) to facilitate further examination. Digital rectal examination revealed a symmetrically enlarged prostate. The dog's scrotum contained a large firm hematoma, and the prescrotal incision was healing well. No peripheral lymphadenopathy was palpated.

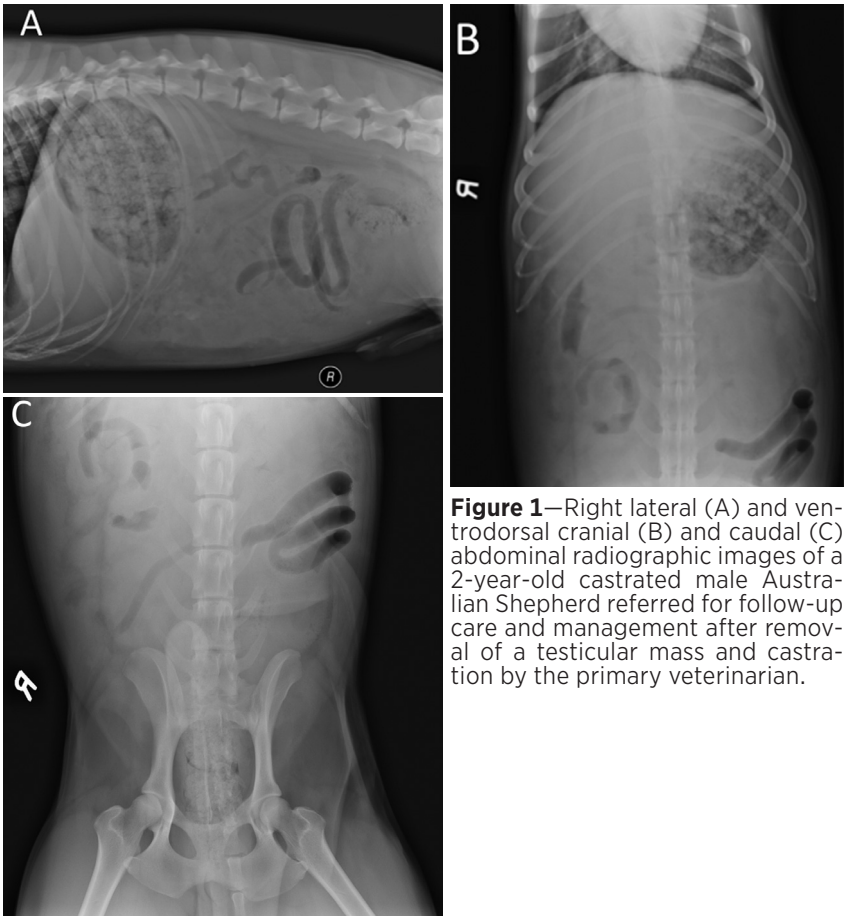


**Figure 1**—Right lateral (A) and ventrodorsal cranial (B) and caudal (C) abdominal radiographic images of a 2-year-old castrated male Australian Shepherd referred for follow-up care and management after removal of a testicular mass and castration by the primary veterinarian.

A CBC, serum biochemical analysis, and urinalysis were performed. The CBC revealed mild macrocytosis (mean corpuscular volume, 78.4 fL; reference range, 63.0 to 77.0 fL) and mild lymphopenia (1,168.0 cells/µL; reference range, 1,200 to 6,500 cells/µL). Serum biochemical analysis revealed mild hyperglycemia (128 mg/dL; reference range, 75 to 125 mg/dL), moderate hypoalbuminemia (1.9 g/dL; reference range, 2.5 to 3.9 g/dL), mild hyperglobulinemia (4.8 g/dL; reference range, 2.1 to 4.3 g/dL), and mild hypomagnesemia (1.3 mg/dL; reference range, 1.7 to 2.4 mg/dL). Urinalysis revealed a urine specific gravity of 1.026, pH of 8.0, trace proteinuria, no detection of sulfosalicylic acid, 0.2 mg/dL of urobilinogen, small amounts of blood with a total of 10 to 25 RBCs/hpf, occasional epithelial cells, occasional lipids, and no bacteria seen. Thoracic (not shown) and abdominal **(Figure 1)** radiography was performed.

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**Formulate differential diagnoses, then continue reading.**

## Diagnostic Imaging Findings and Interpretation

Findings on thoracic radiography were unremarkable (not shown). Abdominal radiography revealed abdominal distention with a diffuse, moderate loss of serosal detail, wispy soft tissue opaque material throughout the abdomen, and partial boarder effacement of the spleen and liver **(Figure 2)**. There was faint wispy mineral-opaque material within the ventral midabdomen that was not definitively within a loop of intestine. The radiographic conclusion was peritoneal effusion. The mineral noted on radiography was thought to have been due to dystrophic mineralization of abdominal soft tissues, as the mineral did not appear to be contained within the digestive tract. Due to the histology of the testicular mass revealing intralesional larvae, another differential for the mineral noted was calcareous corpuscles, which are remanent organelles of adult and larval cestodes organized in concentric rings consisting of calcium, phosphorus, magnesium, and carbonate.

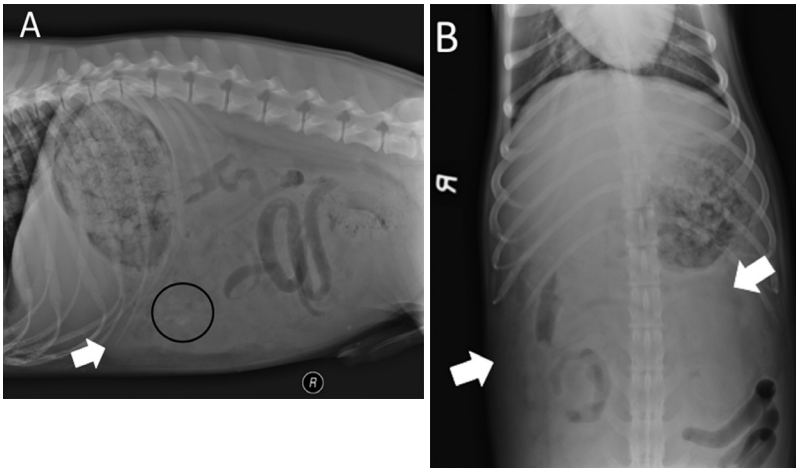


**Figure 2**—Same right lateral (A) and ventrodorsal cranial (B) radiographic images as in Figure 1. A—There is a diffuse, moderate loss of serosal detail with wispy soft tissue opaque material throughout the abdomen, resulting in border effacement with the liver and spleen (arrows). There is faint, wispy, mineral-opaque material (encircled) in the ventral midabdomen. B—The faint mineral-opaque material is not evident.

Abdominal ultrasonography, performed by a board-certified radiologist, revealed that the mesentery was hyperechoic and that the mesenteric fat had numerous anechoic structures with thin hyperechoic rims and distant acoustic enhancement **(Figure 3)**. Multiple heterogenous hyperechoic regions were present in the prostate. There was also mild to moderate medial iliac, jejunal, and colic lymphadenomegaly; the right medial iliac lymph node was the largest and measured up to 1.07 cm in thickness. The spleen was ultrasonographically normal. Given the historical mass removal, histopathology results, and travel history, the primary differential was parasitic cysts resulting in reactive lymphadenopathy and peritonitis; however, steatitis and metastatic neoplasia could not be excluded. The changes to the prostate were considered likely secondary to resolving benign prostatic hyperplasia.

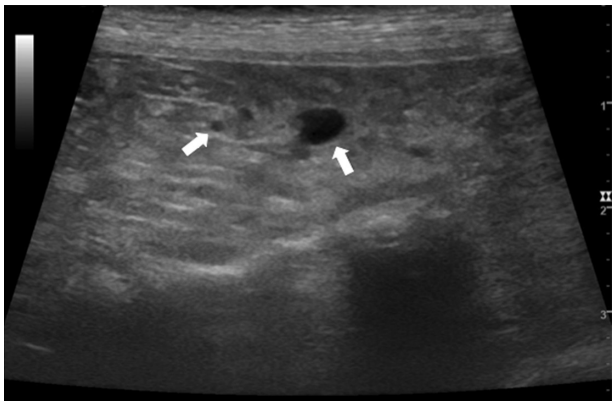

**Figure 3**—Transverse ultrasonographic image of the left caudal region of the abdomen of the dog described in Figure 1. The mesentery is heterogeneously hyperechoic, and there are multiple small, round to ovoid, thin-walled, anechoic, distally enhancing structures (arrows) throughout the mesentery, consistent with fluid-filled cystic structures. These changes were present diffusely throughout the abdomen. The scale on the right of the image is in centimeters.

## Treatment and Outcome

Ultrasound-guided fine-needle aspirate samples of an omental cyst and a medial iliac lymph node were obtained. Cytologic results for the omental cyst indicated *Mesocestoides* spp, which was diagnosed by the presence of several fragmented larvae throughout the slide preparations. The medial iliac lymph node cytology was inconclusive and had peripheral blood contamination with a population of inflammatory cells present that consisted of neutrophils, macrophages, small lymphocytes, eosinophils, and a few large immature lymphocytes. The dog was discharged with prescriptions of fenbendazole (100 mg/kg, PO, q 12 h, for 4 weeks) and a broad-spectrum dewormer (Drontal Plus; 1 tablet [680.4 mg of febantel, 136 mg of praziquantel, and 136 mg of pyrantel pamoate], PO, q 14 d for 1 month).

The patient was returned 4 weeks later for a recheck abdominal ultrasonographic examination, which revealed persistent hyperechoic mesentery and hyperechoic peritoneal fat with multiple, but fewer, ill-defined, variably sized, anechoic regions within the mesentery presumed to be resolving *Mesocestoides* cysts. The multifocal lymphadenopathy was improving but not resolved. A CBC, biochemical analysis, urinalysis, and urine protein-to-creatinine ratio were performed. Results of the CBC indicated newly developed mild leukopenia, mild monopenia, and persistent mild macrocytosis. Biochemical

analysis revealed a persistent mild hyperproteinemia characterized by moderate hypoalbuminemia (2.0 g/dL), mild hyperglobulinemia (4.8 g/dL), and persistent mild hypomagnesemia. Urinalysis showed newly developed proteinuria (+1) and the presence of sulfosalicylic acid (+1). The urine protein-to-creatinine ratio was 0.2, with a total urine protein concentration of 48 mg/g and urine creatinine concentration of 242.4 mg/g. Fenbendazole was continued at a lower dose interval (q 24 h) for 14 days due to the mild leukopenia presumptively caused by bone marrow toxicosis, a rare adverse effect of high doses of fenbendazole. A third dose of the same broad-spectrum dewormer (Drontal Plus, 1 tablet [680.4 mg of febantel, 136 mg of praziquantel, and 136 mg of pyrantel pamoate], PO, once) was prescribed.

The patient was returned 3 months later for recheck abdominal ultrasonography. The previously identified anechoic structures within the mesentery were no longer present, and the previously described changes to the prostate were no longer identified. The patient was presumed to have been in remission of the infection with *Mesocestoides* spp and doing well. Fenbendazole was discontinued.

## Comments

*Mesocestoides* spp are tapeworms of the class Cestoda and family Mesocestoididae.[1] They are most commonly found in the western US, Europe, Asia, and Africa.[1,2] *Mesocestoides* infection is infrequently diagnosed due lack of findings on routine bloodwork and fecal examination.[3] Larvae are typically found incidentally at necropsy, exploratory laparotomy, or ovariohysterectomy.[3] Canine peritoneal larval cestodiasis (CPLC) is the presence of adult tapeworms or tetrathyridia larval stages within the peritoneal cavity.[4] It is currently unclear whether CPLC develops following penetration of adult tapeworms through the intestinal wall into the abdominal cavity or following ingestion of the first intermediate host and thereby becomes an aberrant second intermediate host.[2,4,5] When CPLC occurs, the life cycle of the tapeworm is broken and tapeworms are not found within the gastrointestinal tract.[1]

Canine peritoneal larval cestodiasis can result in parasitic ascites, peritonitis, and nonspecific clinical signs including anorexia, weight loss, depression, tachypnea, dyspnea, vomiting, diarrhea, and abdominal pain.[1] The most common location of larvae and adult cestodes is the liver, spleen, abdominal wall, lungs, kidney, and scrotum.[1] About 22% (13/60) of dogs with CPLC are subclinical[4]; however, the condition may be fatal.[2]

Diagnosis of CPLC is challenging because there are no parasite stages passed in the feces and no available serologic tests.[2] Diagnosis in dogs that develop abdominal distention is typically first made after abdominocentesis or exploratory surgery.[2] A presumptive diagnosis may be made from ultrasonographic features consistent with *Mesocestoides* spp, including small anechoic cystic structures that are often multiloculated with a thin, hyperechoic wall; within transverse views of the cystic structures, larvae may be visualized with enlarged heads (5 to 6 mm) and slender tails (3 mm).[3] Parasites must be recovered from the peritoneal cavity to confirm a diagnosis of CPLC.[2] Calcareous corpuscles may also be recovered from the abdominal cavity and are considered pathognomonic for infection with a larval cestode.[5] As in this case, analysis of ultrasound-guided fine-needle aspirate samples of anechoic structures seen within the abdominal cavity or abdominal fluid from abdominocentesis may result in recovery of larvae or adult *Mesocestoides* spp.

*Mesocestoides* spp infestations are best treated with peritoneal lavage and fenbendazole at high doses of 50 to 100 mg/kg for at least 28 days.[2,5] Praziquantel (5 mg/kg, SC, q 14 d for 2 doses) may also be used if fenbendazole is not effective.[5] High doses of fenbendazole can be curative, but patients should be monitored for adverse effects such as leukopenia from toxicosis affecting the bone marrow, as seen in this case.[2]

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