Sonographic Features of Septic Peritonitis Caused by Mesenteric Ischemia due to *Spirocerca lupi* Aberrant Migration in Dogs

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ABSTRACT

Spirocercosis is caused by the nematode *Spirocerca lupi (S. lupi*). Aberrant nematode migration may occur to various organs. Acute mesenteric infarction-like syndrome leading to focal intestinal wall necrosis perforation and septic peritonitis has been described in association with *S. lupi* infection. The aims of this study were: 1) to describe the ultrasonographic appearance of focal intestinal necrosis secondary to arterial thrombosis caused by *S. lupi* migration, and 2) to assess the efficacy of ultrasonography in differentiating between septic peritonitis secondary to ischemic necrosis of the intestines and other causes of septic peritonitis. Dogs diagnosed with septic peritonitis due to *S. lupi* aberrant migration between years 2017-2020 were included in the study. Control dogs were selected based on a diagnosis of septic peritonitis due to gastrointestinal pathology other than *S. lupi* infection. Thirty-two dogs were included (16 dogs in each study group). Partial or complete intestinal layering loss was significantly more common in the *S. lupi* group compared with the control group [15/16 (94%) vs. 8/16 (50%), respectively *P*=0.015]. There was no difference in the occurrence of other ultrasonographic parameters, nor was there a difference in the small intestine wall thickness between the *S. lupi* and control groups [3.4 mm (range 1.8-5.0), vs. 3.1 mm (range, 2.0-50.0), respectively *P*= 0.89]. In conclusion, ultrasonographic appearance of *S. lupi*-associated peritonitis share similarities to peritonitis of other causes. Yet, it should be considered a differential diagnosis in animals presented with septic peritonitis associated with loss of normal intestinal layering on ultrasonography in endemic areas.

Key words: *S. lupi*; Aberrant Nematode Migration; Intestinal Layering Loss; Septic Peritonitis.

INTRODUCTION

Spirocercosis is caused by the nematode *Spirocerca lupi (S. lupi*) (1). The disease is prevalent in tropical and subtropics areas. Dogs typically become infected through ingestion of infected intermediate hosts (e.g., coprophagous beetles), and less commonly by predation on of paratenic hosts (1). After ingestion, the infective larvae (L3) is released from the intermediate host and penetrates the gastric wall; it migrates through the gastric and celiac arteries to the thoracic aorta and from there to its final destination, the caudal thoracic oesophagus (1). The latter causes a local inflammatory process, resulting in formation of fibro-inflammatory nodules, which may undergo neoplastic transformation in up to 25% of cases (2). Clinical signs mostly result from the presence of a nodule in the caudal oesophagus, and include regurgitation, vomiting, dysphagia hypersalivation, mandibular sialoadenomegaly and weight loss (3). Less common manifestations of *S. lupi* infection include sudden death resulting from a rupture of the aorta, and pyothorax resulting from rupture of the oesophageal wall (4, 5).

Aberrant nematode migration may occur to various organs (6,7,8,9). Clinical signs vary according to the location of

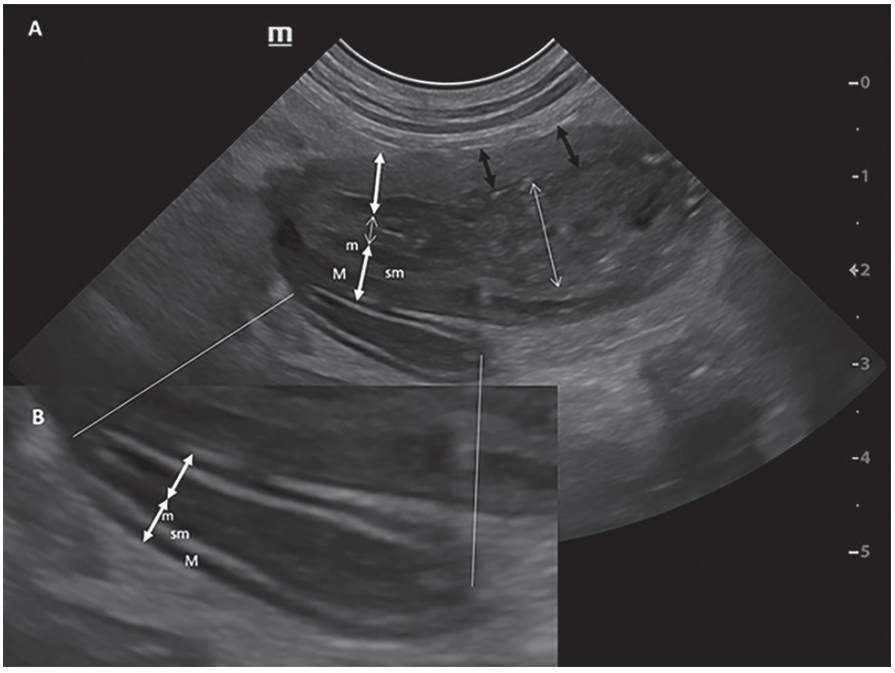

Figure 1: Typical appearance of the intestinal wall of a dog with *S. lupi* aberrant migration. **A.** The image depicts two intestinal segments (normal, at the bottom and abnormal on top of the image). The abnormal segment, showing increased thickeness and echogenicity along with complete loss of normal intestinal layering. **B.** Magnification of the normal intestinal segment. Wall thickness is marked by white thick double headed arrow. Layering includes the inner hypoechogenic mucosa (m), hyperechogenic submucosa (sm), and hypoechogenic external muscularis (M). The lumen widening is demonstrated with thin double headed arrow.

the aberrant migration. For example, variety of neurological signs, mostly asymmetric paraplegia or paraparesis have been reported due to aberrant migration in the nervous system (10). Thromboembolism is another potential sequela of *S. lupi* infection (11,12,13,14), resulting from the migration of the nematode through blood vessel walls (5). One of the common locations for thromboembolism in general, is the hind limbs, resulting in varying degrees of ischemia, depending on the degree of obstruction and its chronicity. Yet, thromboembolic events may also occur in the mesenteric arteries (15). Thrombi, which obstruct the tributaries of the mesenteric artery may cause local ischemia and segmental necrosis of the intestine (Figure 1), a condition termed acute mesenteric infarction-like syndrome, which may lead to intestinal wall perforation and septic peritonitis (15).

Septic peritonitis (SP) is a life-threatening condition, and without appropriate intervention it is likely to result in multi-organ dysfunction and death (16,17,18). The gastrointestinal tract is the most common source of secondary septic peritonitis (16, 17, 18). Diagnosis of septic peritonitis usually relies on identification of intracellular bacteria in the abdominal fluid. Other diagnostic tests rely on differences between serum and peritoneal fluids parameters, including pH, glucose, bicarbonate, lactate, white blood cells and neutrophil counts (19, 20). Treatment of septic peritonitis is intensive and costly, and includes administration of broad-spectrum antibiotics, cardiovascular supportive treatment, and timely surgery to identify and eliminate the source of infection. Despite aggressive medical and surgical treatment, mortality rate among animals with SP is relatively high, ranging between 20-80% (21, 22, 23).

Sonographic characteristics of gastrointestinal perforation in dogs may include increased regional mesenteric fat echogenicity, peritoneal effusion, fluid-filled stomach or intestines, gastrointestinal wall thickening, presence of abdominal free air, loss of gastrointestinal wall layering, regional lymphadenopathy, reduced gastrointestinal motility, corrugated intestines, and presence of a mass or foreign body (24).

Differentiation between septic peritonitis caused by *S. lupi* and other causes is challenging. The diagnosis *S. lupi* associated septic peritonitis relies mostly on ruling out other gastro-intestinal causes for peritonitis preoperatively, identification of segmental intestinal necrosis during surgery and histopathology of the affected intestinal segment (15). Typical histopathologic findings include intestinal transmural coagulative necrosis and mesenteric multifocal necrotizing eosinophilic arteritis, thrombosis, hemorrhage, and early fibroplasia (25).

We hypothesized that ultrasonography could be used to differentiate between peritonitis caused by *S. lupi* aberrant migration and other gastro-intestinal causes of peritonitis. The aims of this study were: 1) to describe the ultrasonographic appearance of focal intestinal necrosis secondary to arterial thrombosis caused by *S. lupi* migration. 2) to assess

the efficacy of ultrasound in differentiating between septic peritonitis secondary to intestinal perforation due to ischemic necrosis and other causes of septic peritonitis.

MATERIALS AND METHODS

This was a retrospective case control study. Dogs diagnosed with septic peritonitis due to *S. lupi* aberrant migration at the Hebrew University Veterinary Teaching Hospital (HUVTH) (years 2017-2020) were considered. Diagnosis was made based on the presence of larvae in mesenteric blood vessels, identified either macroscopically or histologically. Control dogs were selected based on a diagnosis of septic peritonitis due to gastrointestinal pathology other than *S. lupi* infection. Dogs were excluded if a complete ultrasonographic examination performed by a radiologist was not available. Data collected from the medical records included the date, signalment, body weight and ultrasonographic findings.

Ultrasonographic evaluation

Ultrasonographic evaluation of the abdominal structures was performed with a micro-convex array transducer in B-mode at a frequency range of 3-11 MHz (Mindray DC-8 and Mindray M-9, Mindray Bio-Medical Electronics, Shenzhen, China). The ultrasonographic reports were reviewed for the presence of echogenic mesenteric fat, abdominal effusion, gastro-intestinal (GI) fluid accumulation, GI decreased motility, GI wall thickening, partial or complete loss of GI wall layering, regional lymphadenopathy, and presence of mass or foreign body. Maximal jejunal wall thickness at the intestinal lesion was recorded and categorized as normal, decreased, or increased based on body weight, as previously described (26).

Table 1: Proportion of abnormal ultrasonographic findings in dogs with septic peritonitis due to *S. lupi* aberrant migration compared to dogs with secondary septic peritonitis due to other gastrointestinal pathologies.

Parameter	*S. lupi*	Control	*P* value
Loss of normal intestinal layering	15/16 (94%)	8/16 (50%)	0.015
Thickened intestinal wall	3/12 (25%)	5/16 (31%)	1.00
Intestinal lumen widening	10/16 (63%)	5/16 (31%)	0.07
Free abdominal fluid	14/16 (88%)	14/16 (88%)	1.00
Enlargement of mesenteric lymph nodes	11/16 (69%)	9/16 (56%)	0.72
Corrugation of small intestinal wall	2/16 (13%)	3/16 (19%)	1.00
Plication of intestines	0/16 (0%)	1/16 (6%)	1.00
Echogenic omentum	13/16 (81%)	14/16 (88%)	1.00

Statistical analyses

All continuous variables were tested for normal distribution using a Shapiro-Wilks test. Values are reported as median (and range). Difference in continuous variables between groups was analysed using Mann-Whitney U test. Proportion were compared by the Chi square test or the Fischer exact test, as appropriate. $P<0.05$ was considered statistically significant. All statistical analyses were performed using SPSS® 25 for windows (SPSS 22.0 for Windows, IBM Corp., Armonk, N.Y., USA).

RESULTS

Thirty-two dogs were included in this study (16 dogs in each study group). In both groups mixed-breed dogs were the most common (5 in the *S. lupi* group and 8 in the control group). There were 10 males (9 castrated) and 6 females (5 spayed) in the *S. lupi* group, and 7 males (3 castrated) and 9 females (7 spayed) in the control group. Median body weight of dogs in the *S. lupi* group was 32.0 kg (range, 9.8-46.8 kg) and was significantly ($P=0.005$) higher than the control group 19.1 kg (range, 4.0-39.8 kg).

The occurrence of partial or complete intestinal layering loss was significantly higher in the *S. lupi* group compared with the control group [15/16 (94%) vs 8/16 (50%), respectively $P=0.015$]. There was no significant difference in the occurrence of free abdominal fluid, intestinal lumen widening, enlargement of mesenteric lymph nodes, small intestine corrugation, small intestine plication, echogenic omentum or increased wall thickness between the study groups (Table 1). There was also no statistically significant difference in the small intestine wall thickness between the *S. lupi* and control groups [3.4 mm (range 1.8-5.0mm), vs. 3.1 mm (range, 2.0-50.0 mm], respectively $P=0.89$].

DISCUSSION

This study demonstrates that the ultrasonographic appearance of the gastrointestinal tract of dogs with *S. lupi*-associated peritonitis shares common characteristics with GI perforation of other causes. Partial or complete loss of normal intestinal wall layering is more prevalent and suggestive of *S. lupi*

associated peritonitis, but by no means can be considered pathognomonic.

Normal migration of *S. lupi* larvae involves the gastric arteries, the aorta and the oesophagus. Aberrant migration to multiple body organs, such as the urinary system and the CNS has been previously described (6, 7, 8, 9, 10) . Aberrant migration to mesenteric arteries results in segmental ischemia and intestinal perforation (15). In a recent case series of dogs with *S. lupi* associated septic peritonitis, thickening of the jejunal arteries, surrounding mesojejunum, and segmental necrosis were identified on exploratory laparotomy (15). In this study hematoma formation were found in other regions of the mesentery and histology revealed thrombotic mesenteric vessels with intra-lesional *S. lupi* nematode larvae (15).

Ultrasonography is a routine diagnostic tool utilized in cases of acute abdomen. Sensitivity of ultrasonography in identifying the cause of acute abdomen is 86.9% (27). In most cases, the cause is readily identifiable on ultrasound (e.g., foreign body), but in some cases the cause may not be identified prior to surgery (e.g. bile duct rupture) (27). Common ultrasonographic abnormalities of septic peritonitis include free abdominal fluid, increased mesenteric echogenicity, and enlarged lymph nodes (24). Other ultrasonographic abnormalities are related to the primary cause of peritonitis (e.g., foreign body, neoplasia, abscesses) and secondary complications (e.g., pancreatitis).

Treatment guidelines for septic peritonitis, regardless the primary cause, are identical and include surgical exploration to identify and eliminate the source of infection, removal all contaminants from the abdominal cavity and addressing secondary complications. Yet, it is important to identify the cause of intestinal perforation in order to facilitate immediate specific therapeutic intervention. Moreover, since treatment of septic peritonitis is costly, some owners might elect to euthanize their pet when the long-term prognosis is considered guarded to poor due to an incurable underlying cause. This is especially important in perforations suspected to result from gastrointestinal neoplasms (which are often malignant). Neoplasia cannot be diagnosed based on ultrasonographic appearance but is considered more likely when other causes cannot be identified and when changes in the gastrointestinal wall are present. Loss of normal intestinal wall layering is one of the hallmarks of the ultrasonographic appearance of intestinal tumors (28). It is thus important to recognize that loss of normal intestinal wall layering upon ultrasonographic examination is not pathognomonic to neoplasia in general, and in fact is extremely common in *S. lupi* associated perforation (94%). The latter, is associated with a favourable outcome in both the short and long term (15). In the aforementioned case series of dogs with *S. lupi* associated peritonitis, resection and anastomosis of the necrosed section was performed and all but one dog survived and were discharged within 1-6 days (15).

In theory, any thrombogenic condition may cause infarction of the mesenteric vessels; however, mesenteric infarction of the intestines is not commonly reported in dogs, and with the exception of sporadic case reports, the only pathology reported as causing mesenteric infarction in dogs is aberrant migration of *S. lupi* (15). Thus, this disease potentially can be used as a model for the ultrasonographic changes expected in mesenteric infarction and intestine ischemia.

There are only few reports describing ultrasonographic findings in ischemic diseases of the small intestine in small animals (29, 30, 31, 32, 33). In human patients, typical ultrasonographic changes of mesenteric infarction are well defined and include transition from anechoic to hyperechoic mucosa, and from hyperechoic to hypoechoic submucosa. As the condition progresses, intramural or intra-abdominal gas might accumulate due to intestinal perforation. In some cases of prolonged vascular compromise, necrosis of the intestinal wall and sloughing of the mucosa can lead to thinning of the intestinal wall and amorphous echogenic contents of the intestinal lumen (34). Other potential ultrasonographic findings of mesenteric ischemia include ileus, reduced or absent intestinal peristalsis, and peritoneal effusion (35).

Doppler ultrasonography is a potentially useful tool for identifying intestinal wall ischemia. In human patients, absent or decreased flow signal has been related to intestinal wall ischemia compared with increased flow signal in inflammatory bowel diseases (36, 37). However, color Doppler ultrasonography has some limitations in detecting blood flow, especially in small and slow-flowing vessels (38). In *S. lupi* related intestinal ischemia one should expect decreased to absent blood flow, as might occur in other ischemic causes such as mesenteric torsion, necrotizing neoplasm and pressure necrosis due foreign body. In this study Doppler was not consistently available and thus could not have been

used for evaluation as a diagnostic tool for *S. lupi* associated mesenteric infarction.

Contrast-enhanced ultrasonography (CEUS) is another modality that can be utilized to assist in identifying acute mesenteric infarction in animals. Despite its wide use in humans suffering from this condition (39, 40), there is one report describing the use of CEUS for the evaluation of intestine ischemia in veterinary medicine (41), showing a reduced or absent enhancement of the intestinal lesions in comparison to the surrounding perfused wall. Despite its potential, CEUS is not routinely used in veterinary practice and its availability is limited. Computed tomography with contrast medium is the gold standard for diagnosing acute mesenteric ischemia in humans (42). There are few reports describing the tomographic features of the small and large intestinal ischemic injury in dogs and cats (43, 44). Those reposts include identification of the thrombi in the mesenteric arteries and other vessels (43), distension of the mesenteric vasculature (44), streaky peritoneal fat and peritoneal effusion (44) and dilated and gas-filled loops of bowel with a "paper thin wall" which were considered indicative of necrotic bowel (43, 44). As in other imaging modalities, the primary cause for ischemia can be identified in some cases (e.g., intestinal torsion, thrombosis, intestinal foreign body and intestinal mass). The aforementioned modalities should be further evaluated prospectively to assess their utility in identification of mesenteric ischemia in general and specifically in *S. lupi* associated septic peritonitis, as pre-operative, noninvasive tools, in cases where a cause cannot be identified using a routine diagnostic workup.

This study has several limitations. The most important relates to the low number of dogs included in this study, which increases the risk for type II error, namely not identifying differences between the groups when the later in fact do exist. Secondly, this study is a retrospective one in which scans were reviewed retrospectively by one radiologist. A prospective design with standardized approach would likely have captured more information. Thirdly, ultrasonographic examination is a subjective procedure and was performed in this study by several radiologist, which add variability to the results.

In conclusion, ultrasonographic appearance of *S. lupi* associated peritonitis share similarities to peritonitis of other causes. Yet, it should be considered a differential diagnosis in animals presented with septic peritonitis associated with loss of normal intestinal layering on ultrasonography in endemic areas.

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