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**Case Report**

# Sclerosing encapsulating peritonitis in a dog

Carolina Fontana[1], Eliane Novaski[2], Karin Christine Passe Montagnini[1], Simone Cerqueira Lima[2], Juliana Teixeira Druziani[5], Elton Rodrigues dos Santos[2], Juliana Figueredo Vizzu[2], Giovane Frachesco de Carvalho[4], Giovane Baron Quinagli[5], Monica Regina de Mattos[3], Fabiola Bono Fukushima[5], Olicies da Cunha[4], Monica Kanashiro Oyafuso[2], Mariana Pavelski[2], Aline de Marco Viott[1]*

[1] Anatomic Pathology Laboratory, Federal University of Paraná (UFPR), Palotina Campus, Rua Pioneiro, 2153, Jardim Dallas,85950-000, Palotina, PR, Brazil.
[2] Section of Small Animal Clinical Sciences, Federal University of Paraná (UFPR), Palotina Campus.
[3] Anatomic Pathology Laboratory, Pontifical Catholic University of Paraná (PUC-PR), Toledo Campus, Avenida União, 500, Vila Becker, 85902-532, Toledo, PR, Brazil.
[4] Section of Small Animal Surgery, Federal University of Paraná (UFPR), Palotina Campus.
[5] Section of Veterinary Anesthesia, Federal University of Paraná (UFPR), Palotina Campus.

***Corresponding author:** E-mail: alinedemarco@yahoo.com.br

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**Abstract**

Sclerosing encapsulating peritonitis (SEP) is a rare condition which consists of reactive fibrous tissue proliferation with mixed inflammatory infiltration within the abdominal cavity. The present report describes an additional case of SEP affecting a mixed-breed immature female dog presented with persistent vomiting, progressive weight loss, and ascites. Abdominal radiographic and ultrasonographic findings suggested abdominal neoformation resulting in gastric displacement, in addition ascitic fluid was evaluated and cytology showed large numbers of inflammatory effusion. An exploratory laparotomy was performed and were detected multiple thick peritoneal adhesions which restricted mobility of abdominal viscera. Biopsy specimens of these lesions were submitted for histopathological examination. Microscopically, the external serous surfaces of the abdominal organs were covered with dense fibrous connective tissue characterized by intense mature collagen deposition and moderate angiogenesis. The animal was euthanized due to poor prognosis and sent for necropsy. The clinical, imaging, gross and microscopic findings were compatible with SEP.

**Key words:** Neoformation, intestinal obstruction, peritoneal adhesions, chronic inflammation.

## Introduction

Sclerosing encapsulating peritonitis (SEP) is a condition characterized by the formation of thick sheets of fibrous connective tissue accompanied by aggregates of inflammatory cells along the parietal and visceral peritoneum. It is rarely diagnosed in humans, dogs, and cats. This reactive mesenchymal proliferation may result in peritoneal adhesion formation between the intestinal loops resulting in intestinal obstruction as thick bands of connective tissue encapsulate groups of small intestinal loops and compress abdominal organs (3,6,13).

In dogs, SEP is multifactorial and has been associated with chronic bacterial peritonitis, foreign body irritation, fiberglass ingestion, intestinal rupture, neoplasms, leishmaniasis, and may be idiopathic (1,6,11). In cats, the disease may occur due to pancreatitis secondary to vitamin E deficiency or may also be idiopathic (6). In humans, this condition may occur as a result of prolonged peritoneal dialysis, bacterial peritonitis, abdominal trauma, and chronic exposure to asbestos (3).

The progression of the disease process is established according to the clinical signs and is divided into four phases or stages. The first stage of the disease is asymptomatic in which only ascites develops. During the second stage of this

condition, considered as the inflammatory phase of the disease, clinical signs include weight loss and dysorexia. In the third stage, there is proliferation of fibrous tissue with encapsulation of abdominal viscera. Partial or complete obstruction of the intestines may occur in the fourth stage of the disease since sclerotic lesions cover the serosal surface of adjacent abdominal organs (10,14). The treatment is supportive and aims to correct the underlying cause whenever feasible (7,14).

SEP is a disease of complex etiology which clinical diagnosis and treatment are difficult. Radiology and ultrasonography may be useful to diagnose this condition (3). However, the definitive diagnosis is dependent upon microscopic examination of biopsy samples of this lesion collected during exploratory laparotomy (2,8,11).

To date, there have been few published reports of SEP in the veterinary literature. Therefore, the aim of this study is to describe an unusual case of Sclerosing encapsulating peritonitis in a dog with emphasis on the diagnostic approach, clinical picture, imaging, postsurgical follow-up care, gross necropsy changes, and microscopic findings of this condition.

## Case Description

An intact immature mixed-breed female dog weighing 13 kg was referred to a veterinary hospital with a 10-day history of vomiting, abdominal distension, and progressive weight loss. The animal had been rescued and therefore no previous clinical history was available. During physical examination, a 4/9 body condition score (BCS), 7% dehydration, borborygmi which were audible without the stethoscope, eructation, nausea, and a distended abdominal cavity with a positive ballottement wave were noted.

Ancillary routine laboratory tests were requested. Complete blood count and serum biochemical profile showed leukocytosis (27900 cel/mm³, reference < 17000 cel/mm³) due to neutrophilia (22459 cel/mm³, reference < 11400 cel/mm³) [neutrophilic leukocytosis], monocytosis (4743 cel/mm³, reference <1350 cel/mm³), and an increase in liver enzymes (ALT 158, reference < 80 IU/L; ALP 532 IU/L, reference < 80 IU/L).

Laboratory analysis of the abdominocentesis fluid showed that the cavitary effusion was an exudate: density 1028, protein 167.5 g/dL, nucleated cell counts 8500 µL, 79% neutrophils, 17% macrophages, and 4% lymphocytes. Abdominal ultrasound revealed the presence of hyperechoic tissue within the effusion; multiple anechoic cavitations which imparted the lesion a fishing net appearance or cystic aspect. Segmental small intestinal pleating was observed in the epigastric region. Ultrasonographic findings were suggestive of neoformation (Fig. 1A). Laterolateral and ventrodorsal radiographs showed ascites and dorsal displacement of the stomach and intestinal loops.

Treatment was formulated based on the clinical, imaging, and laboratory findings and consisted of antiemetics (maropitant 2mg/kg, SID; metoclopramide 0.5mg/kg, BID). The patient did not respond to therapy. Gastrointestinal transit was evaluated using barium contrast in order to rule out intestinal obstruction. Contrast radiographs showed increased gastrointestinal filling/transit time - the contrast material was observed at the level of the rectum after 5 hours, esophageal dilation cranial to the cardia, and partial obstruction of the intestinal loops (Fig. 1B).

Due to the imaging findings and lack of response to clinical treatment, the patient was referred for exploratory laparotomy to further assess the gastrointestinal disorder. The dog underwent pre-retro-umbilical celiotomy. A peritoneal neoformation which completely encased the intestinal loops was found at surgery (Fig. 2A and Fig. 2B). The veterinarian

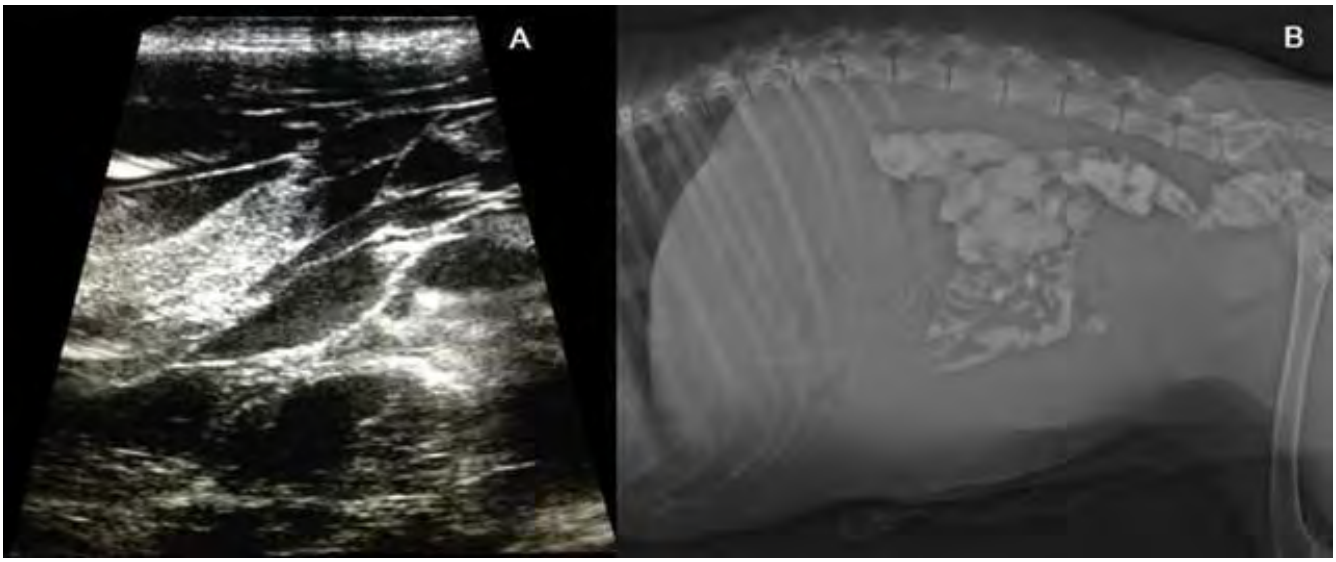


**Figure 1.** Abdominal cavity of a dog with Sclerosing encapsulating peritonitis (SEP). (A) Abdominal ultrasound shows an extensive area of heterogeneous texture and hyperechoic lines with anechoic areas (cavitations). (B) Laterolateral contrast barium X-ray. Dorsal displacement of the intestinal loops with partial segmental obstruction of the small intestine and esophageal dilation cranial to the cardia.

opted for surgical correction of this intra-abdominal lesion. Gastropexy and esophageal plication were also performed to fix the displaced stomach. Samples of the peritoneal lesion were submitted for histopathological examination. Microscopically, this lesion consisted of long thick bundles of fibrous connective tissue accompanied by moderate to large numbers of small blood vessels which were embedded in a dense collagenous extracellular matrix. In addition, there was multifocal to coalescent lymphohistiocytic and neutrophilic inflammatory infiltrates. The histological findings were compatible with chronic organizing peritonitis.

Postoperative treatment included intravenous fluid therapy, maropitant (2mg/kg, SC, SID), ondansetron (0.1mg/kg, IV, TID), omeprazole (1mg/kg, IV, SID), enrofloxacin (5mg/kg, IV, BID), cephalothin (30mg/kg, IV, BID), dexamethasone (0.15mg/kg, IV, SID), tramadol (3mg/kg, SC, IDB), furosemide (2m/kg, IV, IDB), and hypercaloric enteral feeding. Despite improvement of the gastrointestinal disorder three days after treatment was instituted, dysorexia, ascites, apathy, and postprandial vomiting resumed. The patient had progressive weight loss unresponsive to therapy. Euthanasia was elected due to poor prognosis and the animal was submitted for postmortem examination.

At necropsy, the parietal peritoneum was yellowish and markedly thickened and had multifocal moderate petechiae and ecchymoses. There was approximately 300 ml of serosanguineous fluid within the abdominal cavity. The viceral serosal of all abdominal organs was covered by a dense layer of fibrous connective tissue (Fig. 3A), which was 0.2 to 0.8 cm thick, yellowish, had an irregular surface and a rubbery fibroelastic consistency, and firmly adhered to the viscera (Fig. 3B). In some areas, abdominal organs - specially the stomach, liver, and intestines – were trapped and encased by the abdominal mass (Fig. 3C) resulting in segmental intestinal stenosis and adhesive obstruction. Additional gross findings included moderate esophageal dilation anterior to the esophageal hiatus, the presence of a pyloric ulcer 2 cm in diameter. Multiple tissue samples were collected at necropsy, fixed in 10% formaldehyde, embedded in paraffin, routinely processed for histopathology, and stained with hematoxylin and eosin (HE).

Microscopically, there was severe multifocal lymphohistiocytic and neutrophilic inflammation in the visceral peritoneum accompanied by extensive and intense proliferation of fibrous connective tissue arranged in thick elongated bundles. In these peritoneal lesions, moderate to marked angiogenesis was noted within an abundant collagenous extracellular matrix and moderate multifocal areas of necrosis were also present.

There was marked thickening of the splenic capsule with lymphohistiocytic inflammatory infiltrates and the presence of numerous intralesional basophilic gram-negative bacterial colonies multifocally in sections of pericapsular splenic tissue stained with Brown-Brenn (BB) (Fig. 4A). The serosa of the liver and intestines were tightly adhered to the parietal peritoneum. Collagen fibers were blue in sections of diaphragm, liver, kidney, and spleen stained with Masson's trichrome (Fig. 4B). There was no birefringence under polarized light in the affected peritoneum.

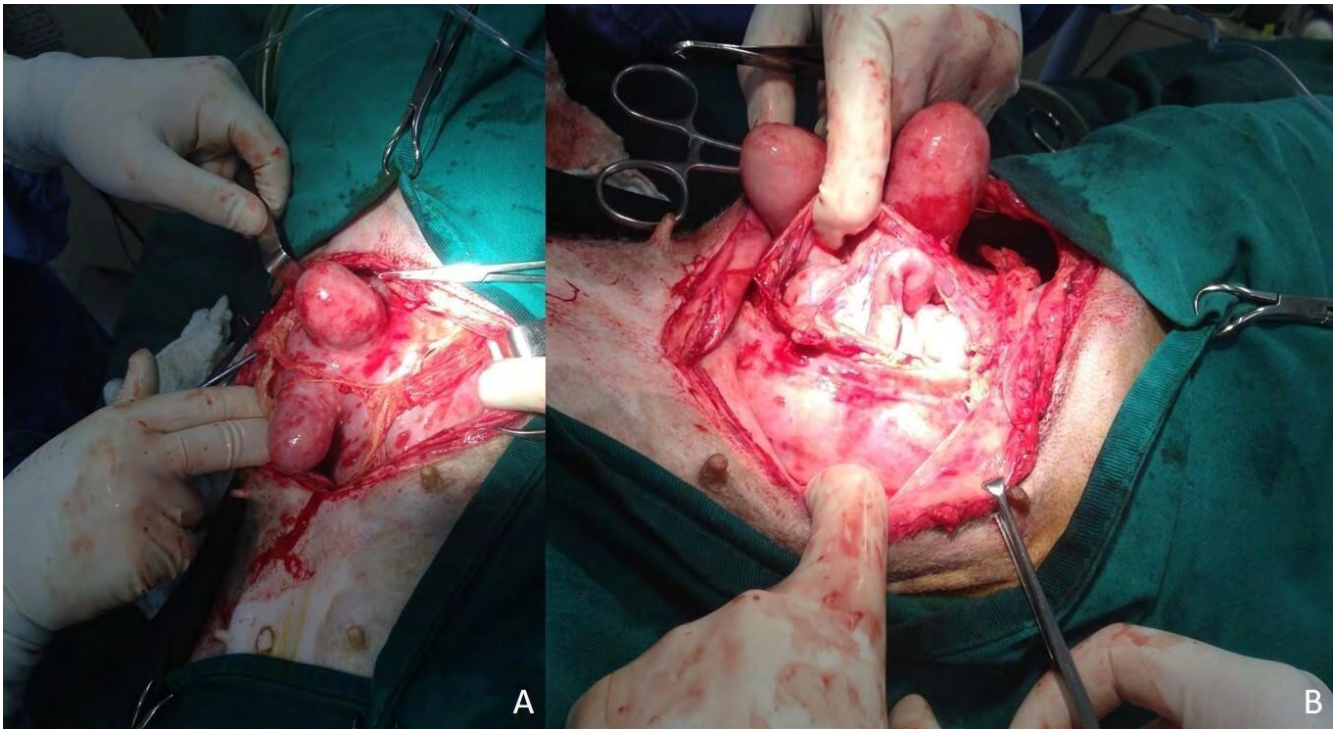


**Figure 2.** Sclerosing encapsulating peritonitis (SEP), canine, female, immature, mixed-breed. Exploratory laparotomy. (A) Intraabdominal peritoneal neoformation compresses and distorts the greater curvature of the stomach. (B) The intestinal loops are markedly encapsulated with thickened. peritoneum. The peritoneal capsular surface of the abdominal organs is extensively covered by fibrotic tissue.

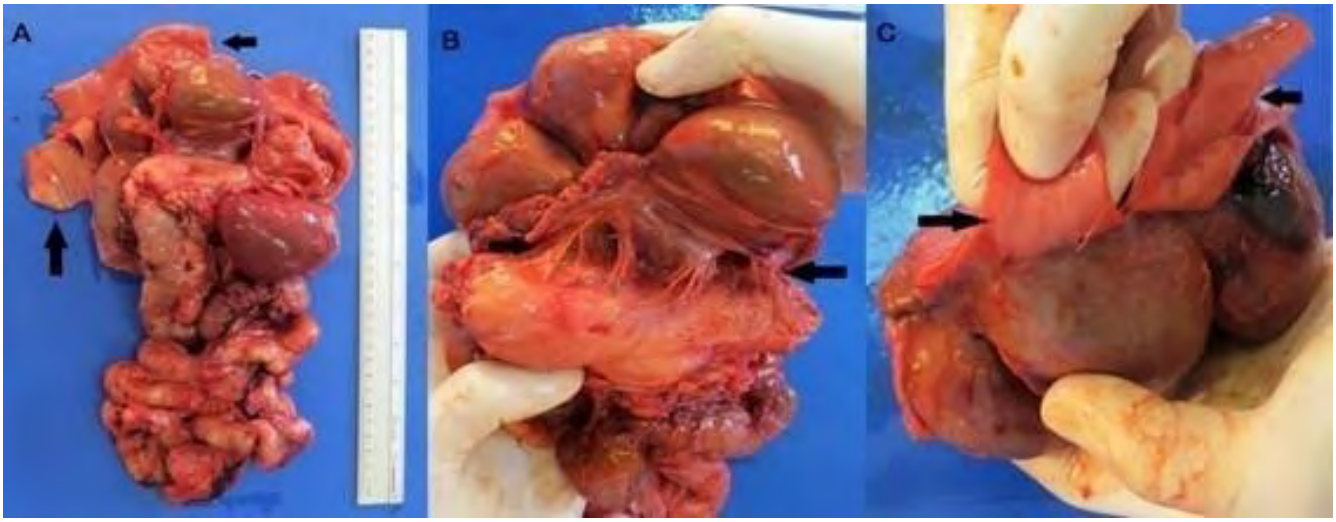


**Figure 3.** Sclerosing encapsulating peritonitis (SEP), canine, female, immature, mixed-breed. (A) The abdominal organs are clumped and tightly adhered to each other by abundant fibrous connective tissue (arrows) with pleating of the intestinal loops and irregularity of the serosal surface of the stomach. (B) There is fibrous adhesion between abdominal viscera with the presence of fibrous tags between the liver and stomach (arrow). (C) Mild diffuse yellowish discoloration liver which is smaller and have rounded edges rounded and a thick capsule of fibrous connective tissue covering and compressing the entire organ (arrow).

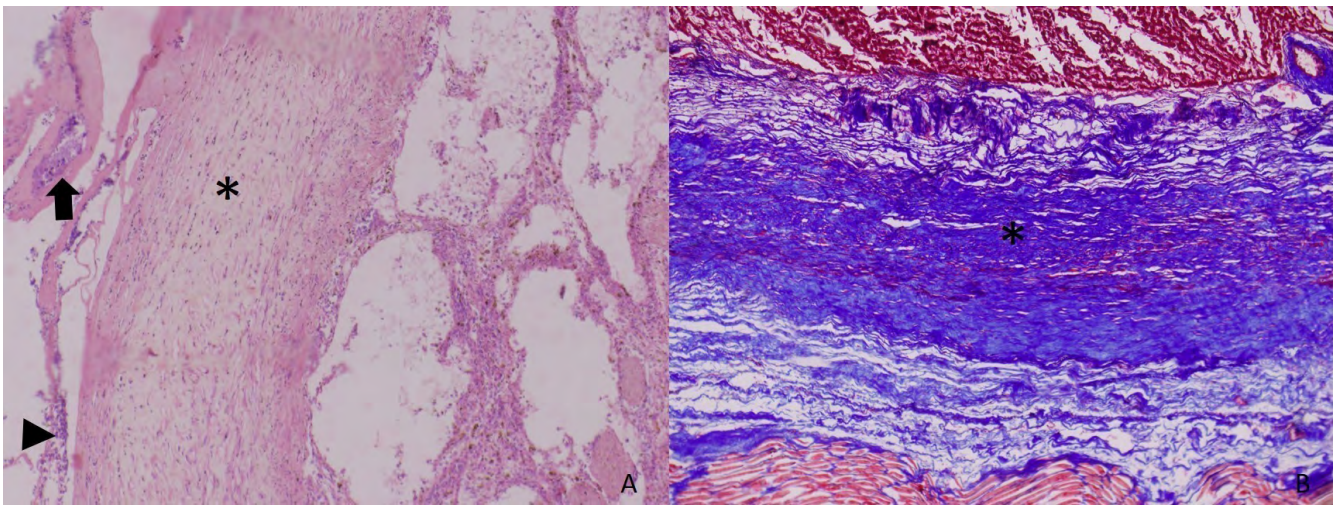


**Figure 4.** Sclerosing encapsulating peritonitis (SEP), canine, female, immature, mixed-breed. Histopathology. (A) Spleen. Markedly thickened splenic capsule. Moderate multifocal lymphohistiocytic inflammatory infiltrate (arrowhead) and moderate numbers of intralesional basophilic bacterial colonies multifocally (arrow) (bacterial periesplenitis) (HE, 6x). (B) There is a marked collagen fiber proliferation between the diaphragm and the liver tissue which stain blue (asterisk) (Masson trichrome, 4x).

## Discussion

Although SEP is an uncommon disease, there are a number of causative factors that may be involved in this disorder (3,13). An excessive inflammatory reaction or exaggerated immune response to chronic bacterial peritonitis has been reported as the usual cause of this disease in animals and humans, and clinical signs including nausea, emesis, and abdominal effusion have been observed in these cases (6,10).

The gastrointestinal obstruction occurs in 92% of humans diagnosed with this condition (8). In the present report, partial gastric and intestinal obstruction was observed which is a clinical sign typically observed during the fourth stage of this disease (10,14). In our case, this obstructive disorder secondary to SEP was confirmed during exploratory laparotomy and at necropsy. According to the literature, SEP should be suspected when there is persistent vomiting, progressive weight loss, systemic inflammatory reaction, and imaging findings (ultrasonography, radiography, tomography) consistent with this disorder such as the presence of a large intraabdominal mass and gastrointestinal displacement and/or obstruction (3,9,14).

The ultrasound findings in our case such as abdominal effusion and the presence of hyperechoic solid intraabdominal structures interspersed with anechoic cystic

areas may also suggest peritoneal carcinomatosis (4). These ultrasonographic findings combined with the radiographic findings of small intestinal displacement and partial segmental intestinal obstruction led to the clinical diagnosis of disseminated epithelial malignant neoplasia or chronic peritonitis. SEP was confirmed by histopathology in biopsy samples collected during laparotomy.

The presence of bacterial colonies on the splenic capsule strongly indicates that these microorganisms triggered the development of SEP in this dog. Other factors are that have been incriminated as causes of this disorder include previous surgical procedures and fiberglass intake (8). The latter was ruled out by polarized light microscopy.

The findings of leukocytosis and abdominal exudative effusion are compatible with peritonitis (1,7). However, the source of the infection was not identified. In addition to leukocytosis, there was also an increase in liver enzymes (ALT and AF), possibly due to strangulation of the gallbladder and liver lobes by abundant dense connective tissue.

In human medicine, the list of differential diagnoses for SEP includes tuberculous peritonitis, peritoneal mesothelioma, post-transplant carcinomatosis, or small-bowel lymphoma, and is easily differentiated in histological findings (15). In the present study, these possibilities were discarded in the absence of neoplastic processes or the presence of granulomas.

In the present case, the constrictive nature and thick dense aspect of fibrous connective tissue covering the abdominal organs and the microscopic examination in these biopsies confirmed the diagnosis of SEP. These findings underscore the importance of the gross and microscopic evaluation of this tissue to reach a definitive diagnosis of this condition (3,14).

In humans, corticosteroid therapy is the most effective treatment for SEP as this anti-inflammatory drug inhibits collagen synthesis. This drug has also been successfully used for the treatment of this condition in animals and therefore was instituted in the postoperative treatment in our case (7,9,14). However, there was no improvement in the condition. There are reports of the successful use of tamoxifen in humans and animals affected with SEP since this drug has a potent antifibrotic effect. The use of tamoxifen in combination with corticosteroids offers a therapeutic alternative for the treatment of SEP as this regimen promotes progressive regression of the neoformed exuberant fibrous tissue (5,10,13).

Surgical correction of SEP may improve the clinical signs of this disorder but is insufficient as a sole treatment to this condition due to its high recurrence rate (1,8,14). In the present case, a slight improvement of the clinical picture was observed in the first 72 hours postoperatively, with a decrease in the number of emetic episodes. However, there was recurrence of clinical signs five days after treatment was started. According to some authors, despite the several therapeutic measures for SEP that has been described in the literature, none efficiently induces regression of the neoformation. Therefore, the prognosis is guarded to poor in this disease as seen in the present report (7,11 ,14).

## Conclusion

Sclerosing encapsulating peritonitis (SEP) must be considered as differential diagnosis for dogs presenting with weight loss, abdominal distention, and persistent emesis. This condition is rare, multifactorial, and only partially understood. While clinical diagnosis is difficult and can require advanced imaging techniques, laparotomy or gross examination findings are characteristic. Surgical intervention can be palliative, but new treatment options are necessary to decrease recurrence of clinical signs. In this case, the presence of bacteria along the peritoneal surface suggests an infectious cause, however, other causes have been described. Additional investigation is necessary to understand the triggers that can lead to SEP.

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