Sclerosing encapsulating peritonitis: a very rare disease entity in cats

"Sclerosing encapsulating peritonitis": een zeer zeldzame ziekte bij katten

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ABSTRACT

Sclerosing encapsulating peritonitis (SEP), also known as encapsulating peritoneal sclerosis, is a very rare disease described in humans as well as in animals. This condition is characterized by a chronic, diffuse, fibrocollageneous thickening of parietal and visceral peritoneum with secondary encapsulation of abdominal organs, mostly small intestines. Etiopathogenesis remains incompletely understood. SEP can be divided into primary, idiopathic forms and secondary forms. Secondary SEP can be caused by many different underlying disorders of which peritoneal dialysis is the most common one in humans. Diagnosis of SEP remains difficult due to its vague clinical symptoms; therefore, a combination of medical imaging, surgery and histopathology is warranted. Treatment is challenging and the prognosis is variable, depending on the severity of the disease. A combination of surgery, medicinal therapy, nutritional support and treatment of underlying disorders is used. In this article, two feline cases of SEP are described, followed by a review of the literature.

SAMENVATTING

"Sclerosing encapsulating peritonitis" (SEP), ook bekend als "encapsulating peritoneal sclerosis", is een zeer zeldzame aandoening die voorkomt bij zowel mensen als dieren. De aandoening wordt gekenmerkt door een chronische, diffuse, fibrocollageneuze verdikking van de pariëtale en viscerale pleura met secundair inkapseling van de abdominale organen, meestal de dunne darmen. De etiopathogenese blijft onduidelijk. SEP kan opgesplitst worden in primaire, idiopathische vormen of secundaire vormen, die veroorzaakt kunnen worden door een groot aantal aandoeningen, waarvan in de humane geneeskunde peritoneale dialyse de meest voorkomende oorzaak is. Diagnose van SEP is moeilijk door de vage klinische symptomen, waardoor ze het beste gebeurt met behulp van medische beeldvorming, chirurgie en histopathologie. Behandeling is niet gemakkelijk en de prognose is wisselend, afhankelijk van de ernst van de ziekte. Een combinatietherapie van chirurgie, medicatie, nutritionele ondersteuning en behandeling van onderliggende ziekten kan worden toegepast. In dit artikel worden twee katten met SEP beschreven gevolgd door een overzicht van de literatuur.

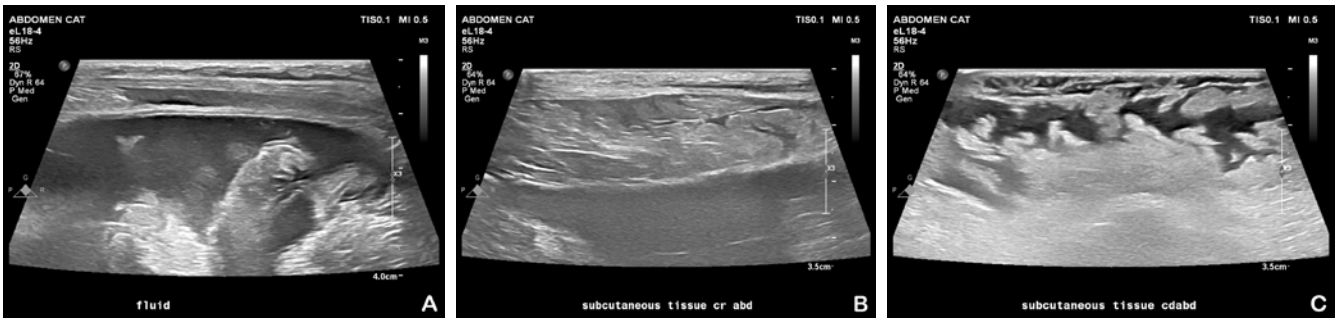

Figure 1. Abdominal ultrasound images of the first cat. A. A large amount of hypoechogenic, flocculent fluid was found in the peritoneal cavity. Thickened and hyperechogenic peritoneum and subcutaneous tissue were recognized in B. the cranial abdomen and C. the caudal abdomen.

CASE REPORTS

Two cats were presented for necropsy and were diagnosed with sclerosing encapsulating peritonitis after macroscopic and histologic examination.

Cat 1

The first cat was a female sterilized European Shorthair of 12.5 years old. She was presented at the Faculty of Veterinary Medicine of Ghent University in emergency with complaints of a hard, tense abdomen, diarrhea and lethargy. An abdominal ultrasound was performed, which showed a moderate amount of heterogenous and fibrinous abdominal effusion (Figure 1A), a severely thickened and hyperechoic peritoneum (Figures 1B and 1C) with several adhesions to the visceral organs and diffuse hepato- and splenomegaly. Blood examination showed no abnormalities. Differential diagnoses were neoplasia, inflammation (feline infectious peritonitis (FIP)), hepatic degeneration and/or regeneration, granulomatous inflammation and sclerosing encapsulating peritonitis (SEP). Due to the grave prognosis, the cat was euthanized and referred for necropsy.

During necropsy, the general condition of the cat was evaluated. The cat had a poor body condition and generalized muscle atrophy. She was also severely dehydrated. Two hundred mL of serohemorrhagic fluid was present in the abdominal cavity. The parietal peritoneum had a prominent grey aspect and was moderately thickened. Multiple adhesions and fibrin strands were visible between the peritoneum and the different abdominal organs (Figure 2A). The small intestines showed a severely tortuous aspect starting from the jejunum up to cranial colon (Figure 2B). On transection, the intestinal wall was severely thickened with intraluminal little to no mucous content. No obvious mesentery was recognizable. The gastric wall had a normal thickness. The liver was very small and completely encapsulated by a thick light grey capsule, with complete loss of its normal lobular anatomy and with severe fibrous adhesions to the diaphragm and the stomach (Figure 2C). On transection, the normal dark red hepatic color was still present and the gall bladder was completely encapsulated by the liver parenchyma. The spleen was also severely decreased in size with loss of its normal morphology (Figure 2D) and contained a thickened splenic capsule that was moderately attached to the stomach. The pancreas was not clearly recognizable and was hidden in the fat and connective tissue in between the stomach, spleen and intestines. The left cranial lung lobe showed a diffuse atelectatic and dark red aspect, which sank in formalin. The rest of the lungs had no abnormalities with a soft consistency and a light pink color. No lesions were found in the other organs.

Histopathology was performed using formalin fixation, paraffin embedding, and standard hematoxylin and eosin (H&E) stain, combined with a Masson's trichrome (MT) stain specifically for connective fibers. Small intestines, liver, spleen and pancreas showed a severely thickened peritoneal layer (Figures 3A, B and C for H&E, and D, E and F for MT; for comparison: H&E and MT stains of intestines, liver and spleen with a normal peritoneum are added in Figures 4A-F). This thickened peritoneal layer showed variable maturity depending on the location. Mature connective tissue consisted of a large amount of connective tissue fibers with only small amounts of spindloid fibroblasts, while more immature areas showed presence of larger amounts of more plump fibroblasts in a more myxoid stroma. Some parts of the peritoneum showed increased presence of small, tortuous blood vessels with plump endothelial cells (neovascularization), admixed with connective tissue fibers, which is indicative for granulation tissue. Multifocally, there was mild lymphoplasmacytic inflammation. The stomach did not show a thickened peritoneum. There was a mild mucosal lymphoplasmacytic infiltration in the intestinal walls. In the liver, there were multifocal intracytoplasmic accumulations of hemosiderin pigment in Kupffer cells and an increased amount of Ito-cells. The spleen had a very contracted appearance. Pancreatic parenchyma showed no abnormalities. Lung tissue of the cranial left lung lobe showed a focal suppurative bronchopneumonia, mixed with large amounts of fibrin and edema.

Based on the macroscopic and histologic lesions, the diagnosis of SEP with involvement of the small

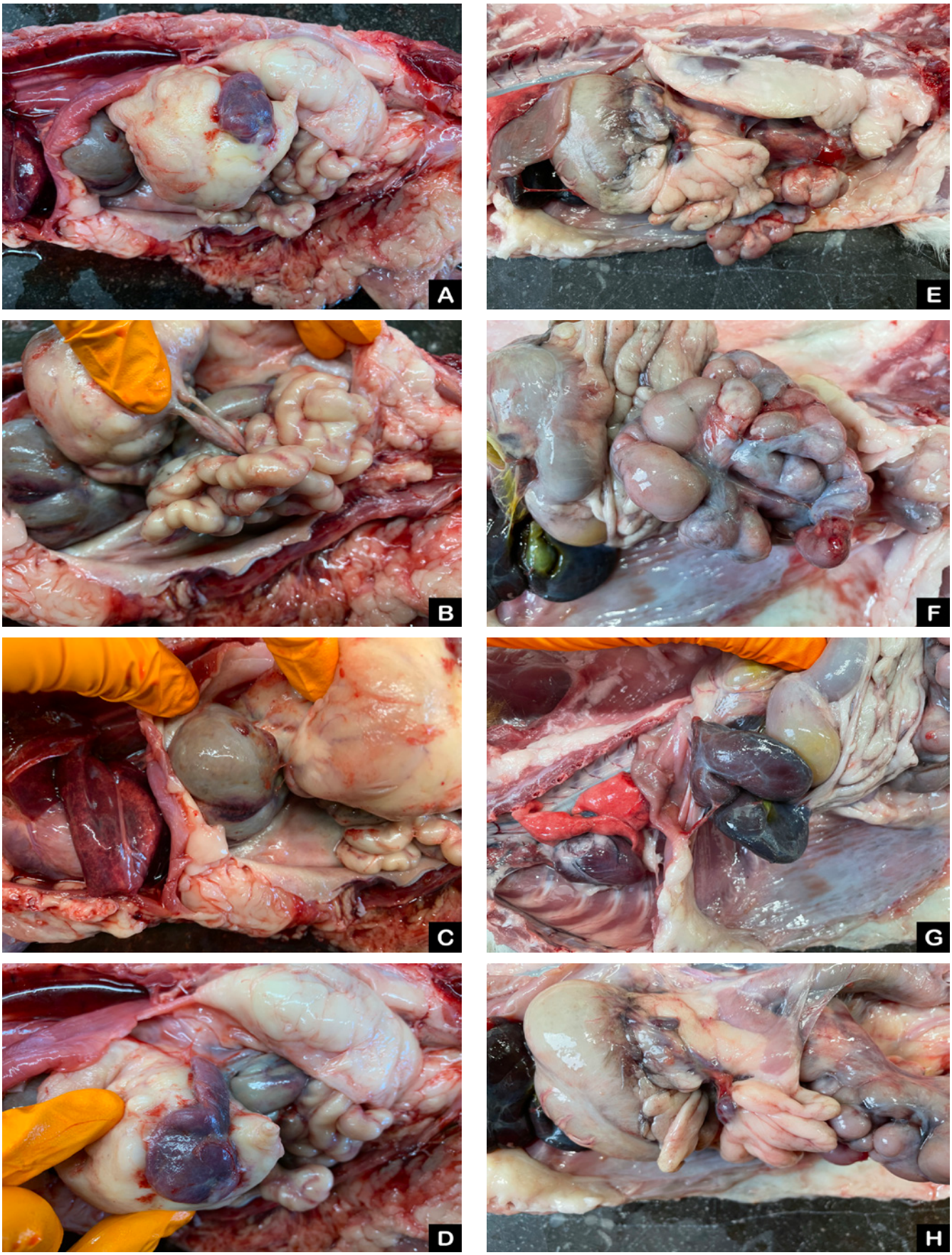

Figure 2. Macroscopic images of both cats. A. In situ image of the first cat with generalized tortuous aspect of B. the small intestines, C. a small grey liver with round edges and D. a small irregular spleen. E. In situ image of the second cat with generalized tortuous aspect of F. the small intestines, G. an irregular, small liver with round edges and H. a very small spleen in between the fat tissue. Notice in the background of images F. and G. the very pale color of the peritoneum.

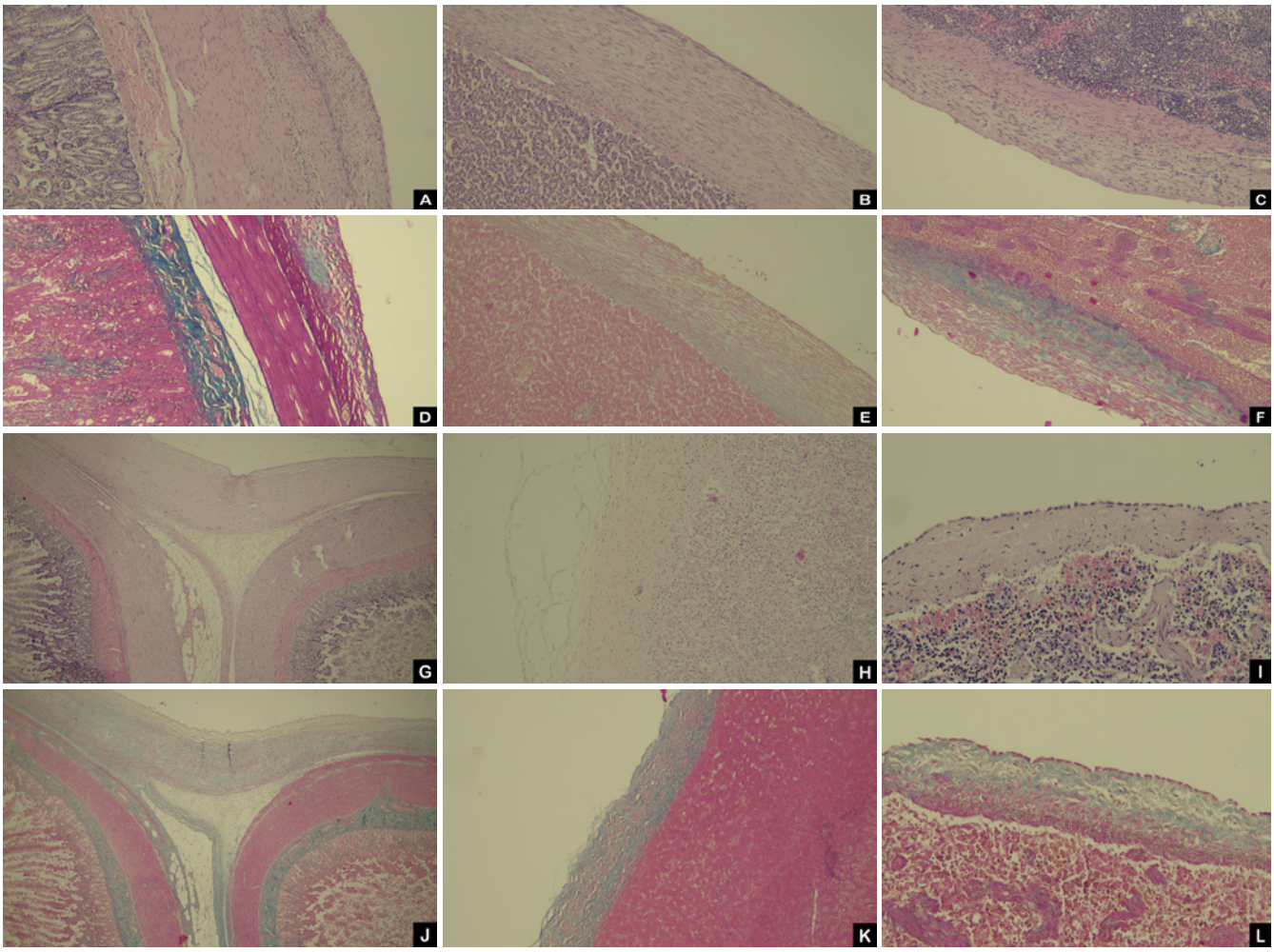

Figure 3. Histologic images of the thickened peritoneum of different visceral organs in both cats. Images A. through F. are of the first cat: A. (H&E, 100x) and D. (MT, 100x) show small intestines, B. (H&E, 100x) and E. (MT, 100x) show liver and C. (H&E, 100x) and F. (MT, 100x) show spleen. Images G. through L. are of the second cat: G. (H&E, 40x) and J. (MT, 40x) show small intestines, H. (H&E, 100x) and K. (MT, 100x) show liver and I. (H&E, 200x) and L. (MT, 200x) show spleen.

intestines, liver, spleen and pancreas, was made. Additionally a focal suppurative bronchopneumonia in the cranial left lung lobe was found, suspected to be caused by an aspiration pneumonia.

Cat 2

The cat was a female sterilized kitten (*Felis vulgaris*) of only five months old, presented in a local veterinary clinic. Complaints of the owner were partial anorexia and polydipsia. No vomiting or diarrhea were mentioned at that time. However, the kitten had a history of intermittent diarrhea. Clinically, an extreme amount of abdominal fluid was noticed, confirmed by ultrasound of the abdomen, after which FIP was suspected. Blood examination and echocardiography showed no abnormalities. Laparoscopy was performed, after which a suspicion of SEP was made. The cat died spontaneously during the following night.

Necropsy showed moderate dehydration, with a normal body condition and muscle configuration. The peritoneum was diffusely thickened and showed an obvious dark grey color due to the increased amount of connective tissue. All organs were abnormally adhered to each other (Figure 2E). The small intestines had a diffuse severe thickened intestinal wall and were very tortuously adhered to each other, starting from jejunum up to caudal ileum (Figure 2F). The duodenum had a normal wall thickness but was moderately dilated. The colon and cecum showed no abnormalities. A normal mesentery was not present. The liver was small with ventrally rounded edges and had a diffuse dark black parenchyma with a light grey capsule with multifocal linear white lesions (Figure 2G). The spleen was severely decreased in size with a very shriveled appearance, surrounded by a thick layer of connective tissue (Figure 2H). No lesions were found in the other organs.

Histopathology was performed using formalin fixation, paraffin embedding and standard H&E stain, combined with a MT stain. Small intestines, liver,

spleen and pancreas showed a severely thickened peritoneal layer (Figures 3G, 3H and 3I for H&E, and 3J, 3K and 3L for TM), with the same appearance as seen in the first case. Presence of fibroblasts, mature and immature connective tissue and mild to moderate lymphoplasmacytic inflammation in the peritoneum was seen in the small intestines, liver and spleen. The small intestines showed multifocal necrosis in the outer layers of its peritoneum, and lymphoplasmacytic inflammation was seen in the peritoneum as well as in the superficial layers of the tunica muscularis. Diffusely, hepatocytes were severely shrunken, with only a small amount of normal cytoplasm and a small nucleus, indicative for liver atrophy. A prominent amount of neutrophils was seen in the liver sinusoids. The spleen had a contracted and activated appearance.

A diagnosis of SEP was made, with involvement of the small intestines, liver and spleen, combined with moderate to severe hepatic atrophy.

LITERATURE REVIEW

Definition

Sclerosing encapsulating peritonitis (SEP) is known by many different names in the literature, of which SEP is most commonly used (Machado, 2016; Tannoury and Abboud, 2021). This disease was first diagnosed in humans by Owtschinnikow in 1907, and was originally called 'peritonitis chronica fibrosa incapsulata' (Machado, 2016; Danford et al., 2018; Tannoury and Abboud, 2021). Other commonly used names are 'encapsulating peritoneal sclerosis' (EPS) (Danford et al., 2018), 'fibroblastic peritonitis' or 'icing sugar bowel' (Machado, 2016). The designation 'abdominal cocoon syndrome' is a specific idiopathic type of SEP, mostly seen in adolescent girls of tropical and subtropical regions without clinical symptoms (Machado, 2016; Danford et al., 2018; Tannoury and Abboud, 2021). However, terms are not standardized and some publications use them interchangeably. Important differentiation needs to be made with some similar diseases, such as peritoneal encapsulation, which is an incidental congenital condition, characterized by encapsulation of the small intestines, mesocolon and omentum without any overt adhesions (Danford et al., 2018; Machado, 2016).

SEP is a condition characterized by chronic, diffuse, fibrocollageneous thickening of the parietal and visceral peritoneum ('sclerosing'), with secondary encapsulation and formation of adhesions between and constrictions of the abdominal organs (encapsulating) with presence of an ongoing mainly mononuclear inflammation in the peritoneum (peritonitis) (Machado, 2016; Danford et al., 2018). In human medicine, SEP is classified into four different types depending on the extent of the abdominal organs which are involved (Alshomini et al., 2021) (Table 1); however, in some publications, only the first three types are mentioned (Machado, 2016; Tannoury and Abboud, 2021).

Prevalence

SEP is a rare disease, most commonly reported in humans (Machado, 2016; Danford et al., 2018). The exact prevalence is unclear and due to its rarity, no presumptions about possible predisposing factors can be made (Danford et al., 2018). In the human literature, it has been reported that SEP can occur at all ages, with a mean age of 39 years (Machado, 2016) and the oldest age of 90 years (Zhang et al., 2020). Men appear to be more sensitive (Danford et al., 2018). In veterinary medicine, SEP is extremely rarely seen, with most reports only describing one to five cases (Hardie et al., 1994; Adamama-Moraitou et al., 2004; Etchepareborde et al., 2010; Barnes, 2015; Veiga-Parga et al., 2015; Sonck et al., 2018; Tsukada et al., 2022). To the best of the authors' knowledge, only 13 canine cases have been described up until now (Hardie et al., 1994; Adamama-Moraitou et al., 2004; Etchepareborde et al., 2010; Barnes, 2015; Veiga-Parga et al., 2015; Tsukada et al., 2022). In cats, the disease is even more rare, with only two previously reported cases (Hardie et al., 1994; Sonck et al., 2018). A variety of ages, breeds and sexes was found in dogs, but due to limited cases in the literature, no further conclusions can be made regarding in specific predispositions.

Etiopathogenesis

The etiopathogenesis of SEP remains incompletely understood (Machado, 2016).

The primary idiopathic form of SEP in humans, previously mentioned as the 'abdominal cocoon syndrome' is not associated with any obvious causes.

Table 1. The four different types of sclerosing encapsulating peritonitis (Machado, 2016; Alshomini et al., 2021; Tannoury and Abboud, 2021).

Type	Abdominal organs involved in the encapsulation
1	Part of the small intestines
2	All the small intestines
3	All the small intestines + stomach, cecum, colon, liver or ovaries
4	Entire peritoneal cavity

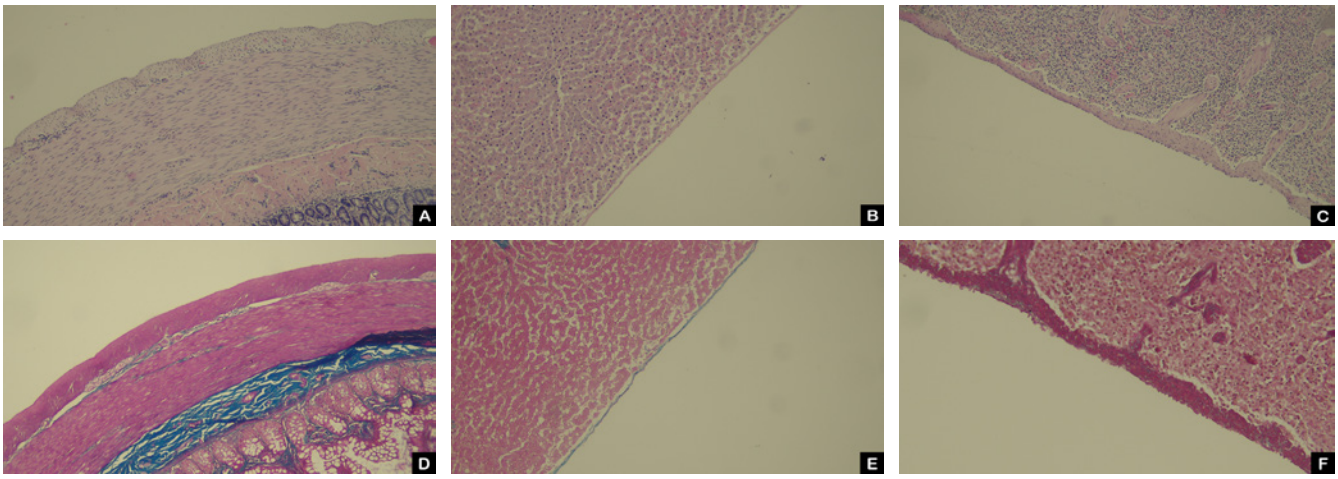

Figure 4. Histologic images of the thin peritoneum of different visceral organs of normal, healthy cats for comparison. A. (H&E, 100x) and D. (MT, 100x) show small intestines, B. (H&E, 100x) and E. (MT, 100x) show liver and C. (H&E, 100x) and F. (MT, 100x) show spleen.

Multiple hypotheses have been made, such as retrograde menstruation superimposed with viral infection, retrograde peritonitis originating in the fallopian tubes and other reproductive disorders. However, these hypotheses don't explain all cases, since 75% of patients are men, premenstrual women or children. Other possible explanations are several developmental diseases, mainly concerning development of mesenteric blood vessels or omentum (Machado, 2016; Tannoury and Abboud, 2021).

Secondary SEP can be caused by several diseases characterized by chronic low-grade inflammation of the peritoneum, leading to progressive fibrosis and sclerosis (Adamama-Moraitou et al., 2004; Alshomini et al., 2021). The most common cause is peritoneal dialysis used in renal patients, in which the most significant risk factors are the duration of the therapy, recurrent additional peritoneal infections, acetate in the dialysate and chlorhexidine usage for disinfection (Machado, 2016; Danford et al., 2018; Tannoury and Abboud, 2021). Other less common etiologies are peritoneal venous shunts, infectious peritonitis (tuberculosis, other bacteria, cytomegalovirus, fungi, parasites), adminis-tration of certain medications (beta blockers, antiepileptic drugs, etc.), intraperitoneal chemotherapy or other irritants (powder of surgical gloves), abdominal sarcoidosis, cirrhosis, blunt abdominal trauma, intraabdominal surgery (hysterectomy, liver transplantation), asbestos, systemic inflammatory disorders, recurrent polyserositis (Mediterranean fever), autoimmune diseases (systemic lupus erythematosus), endometriosis, peritoneal carcinomatosis caused by ovarian, gastric, pancreatic or renal carcinoma or advanced midgut neuroendocrine tumors, etc. (Machado, 2016; Tannoury and Abboud, 2021).

SEP is suspected to need a combination of different factors to give rise to disease (Machado, 2016; Danford et al., 2018). Patients come in contact with (one or multiple) of the inciting factors mentioned above, which induce non-inflammatory peritoneal sclerosis. A pro-inflammatory second hit then starts a cascade of different pro-inflammatory factors, of which transforming growth factor beta-1 (TGF-β1) and interleukin-1 (IL-1) are the most important. TGF-β1 is known to promote differentiation of normal mesothelial peritoneal cells to fibroblasts, which induces an increased production of extracellular matrix components such as collagen (Danford et al., 2018). IL-1 is produced by activated peritoneal macrophages and induces differentiation of submesothelial mesenchymal stem cells to fibroblasts (Hardie et al., 1994). Genetic predisposition may be present; however, no unmistakable proof of this has been published yet (Danford et al., 2018).

In the human literature, a staging system has been proposed to be able to differentiate between the different pathogenic stages of the disease, which might have an impact on therapy response (Danford et al., 2018) (Table 2). The stages are based on a combination of clinical symptoms, presence or absence of inflammation and encapsulation, and intestinal pathology.

SEP in dogs cannot always be clearly associated with an underlying disease, which is why multiple cases are defined as being idiopathic (Adamama-Moraitou et al., 2004; Veiga-Parga et al., 2015). Probable causes in some cases are previous abdominal surgery, steatitis, chronic infectious peritonitis (bacteria, leishmania), perforating foreign objects, ruptured pyometra, fiberglass ingestion, abdominal neoplasia, such as gastric, pancreatic or intestinal adenocarcinoma, hepatocellular carcinoma or sclerosing mesothelioma (Hardie et al., 1994; Adamama-Moraitou et al., 2004; Etchepareborde et al., 2010; Barnes, 2015; Tsukada et al., 2022). Of the two reported feline cases, one was presumably caused by steatitis and fat necrosis of unknown origin (Hardie et al., 1994), while the other one was suspected to be caused by an allergic reaction (Sonck et al., 2018).

Clinical symptoms

SEP can give a wide variety of vague symptoms in humans. Patients are most commonly presented with intermittent and recurrent, moderate to severe abdominal pain, caused by intestinal obstruction and necrosis (Machado, 2016; Danford et al., 2018; Zhang et al., 2020; Alshomini et al., 2021; Tannoury and Abboud, 2021). This is mostly in combination with a malnourished appearance, abdominal distention, palpable abdominal mass, nausea and vomiting (Machado, 2016; Danford et al., 2018; Zhang et al., 2020; Tannoury and Abboud, 2021). Other vague possible symptoms are fever, weight loss, loss of appetite and ascites (Machado, 2016; Tannoury and Abboud, 2021). It is important to notice that some cases of SEP are incidental and do not cause any clinical complaints (Machado, 2016; Zhang et al., 2020; Tannoury and Abboud, 2021). Blood examination does not give any specific abnormalities and are mostly related to the clinical malnutrition, inflammation and possible underlying disease (Danford et al., 2018).

Common clinical symptoms in canine cases are also vague, and can include vomiting, diarrhea, soft feces, anorexia, depression or lethargy, enlarged abdomen and abdominal pain (Hardie et al., 1994; Adamama-Moraitou et al., 2004; Etchepareborde et al., 2010; Barnes, 2015; Tsukada et al., 2022). Chronic cases can show moderate to severe low body condition and muscle atrophy (Hardie et al., 1994; Etchepareborde et al., 2010; Barnes, 2015), combined with symptoms specific for the underlying etiology (Hardie et al., 1994). The chronic weight loss is a consequence of different factors (Hardie et al., 1994), of which the most important are malabsorption secondary to fibrosis with decreased gastrointestinal motility, abdominal pain with secondary anorexia, and cachexia caused by chronic release of inflammatory mediators. Blood examination is similarly vague as seen in humans, and is mostly a representation of the underlying disorder, including electrolyte abnormalities, hypoalbuminemia and anemia, caused by the effusions and the chronic inflammation (Barnes, 2015).

Cats show similar symptoms as seen in dogs: anorexia, intermittent vomiting, rare diarrhea, weight loss, abdominal distention and sensitivity (Hardie et al., 1994; Sonck et al., 2018). Blood examination does not show any specific findings; however, anemia has been described once (Sonck et al., 2018).

Macroscopic lesions

SEP in humans and animals gives a very typical gross thick collagenous encapsulation of the small intestines with secondary adhesions between the intestinal loops, with a very tortuous and mass-like appearance in the central abdomen (Machado, 2016; Tannoury and Abboud, 2021). Depending on the type of the disease, other organs can be additionally involved, such as stomach, cecum, colon, liver or ovaries (Machado, 2016; Tannoury and Abboud, 2021). The typical peritoneal encapsulation normally needs several years to develop in humans; however, a progression for a short period of twelve weeks has been described in the literature (Machado, 2016). Organs that are encapsulated in fibrous tissue, can be misshapen and unrecognizable, as is commonly seen with the liver (Etchepareborde et al., 2010; Tsukada et al., 2022). The parietal peritoneum becomes diffusely thickened (Alshomini et al., 2021; Tsukada et al., 2022).

Human SEP is known to cause intestinal obstruction and necrosis in a high percentage of patients (Danford et al., 2018; Zhang et al., 2020; Alshomini et al., 2021). Rarely, there is formation of enterocutaneous fistulae and ascites (Hardie et al., 1994). Intestinal obstruction and necrosis, very commonly noticed in humans, are not regularly seen in dogs and cats (Hardie et al., 1994; Etchepareborde et al., 2010; Adamama-Moraitou et al., 2004;), presumably due to their very active fibrinolytic system. Less fibrous tissue is produced and more granulation tissue is formed, which has less obstructive capacities.

Canine cases are mostly presented with ascites, a finding not typically seen in humans (Hardie et al., 1994; Adamama-Moraitou et al., 2004; Etchepareborde et al., 2010; Barnes, 2015; Veiga-Parga et al., 2015; Zhang et al., 2020; Tsukada et al., 2022). The ascites can be caused by multiple factors, such as altered vascular permeability due to immature collagen in capillary walls in granulation tissue, lymphatic obstruction due to fibrous tissue or inflammation, or chronic hepatic or biliary disease with secondary portal hypertension (Hardie et al., 1994; Adamama-Moraitou et al., 2004; Barnes, 2015). The abdominal fluid is typically serohemorrhagic containing large amounts of erythrocytes, mixed inflammatory cells, reactive mesothelial cells and fibroblasts (Hardie et al., 1994; Barnes, 2015). Cats show similar lesions as

Table 2. The four different stages of sclerosing encapsulating peritonitis (Danford et al., 2018).

Stage	Characteristics
1	Pre-sclerosing encapsulating peritonitis
2	Inflammatory sclerosing encapsulating peritonitis
3	Encapsulating or progressive sclerosing encapsulating peritonitis
4	Chronic fibrotic sclerosing encapsulating peritonitis

seen in dogs: typical connective tissue encapsulation of small intestines with intestinal adhesions and clustering centrally in the abdominal cavity, combined with serohemorrhagic ascites and a grey, thickened parietal peritoneum (Hardie et al., 1994; Sonck et al., 2018).

Histopathologic lesions

SEP causes very characteristic visceral and parietal peritoneum with an uneven, diffusely, moderately to severely thickened appearance (Hardie et al., 1994; Adamama-Moraitou et al., 2004; Etchepareborde et al., 2010; Machado, 2016; Sonck et al., 2018; Alshomini et al., 2021; Tannoury and Abboud, 2021; Tsukada et al., 2022). Humans mostly show presence of fibroblast proliferation, combined with deposition of fibrocollagenous tissue and fibrin deposition (Machado, 2016; Danford et al., 2018). In contrast, different layers can be seen in the thickened peritoneum of animals. The deepest layer shows mature collagenous connective tissue with densely packed collagen fibers, while the more superficial layers are built up of granulation tissue, characterized by loose collagenous stroma with presence of numerous plump fibroblasts, mixed with abundant, small, tortuous blood vessels lined by plump endothelium (neovascularization), and a mild mucinous deposition (Hardie et al., 1994; Etchepareborde et al., 2010; Veiga-Parga et al., 2015; Sonck et al., 2018). In humans and animals, mild to moderate, mostly mononuclear (lymphocytes, plasma cells and some macrophages) inflammation can be seen in the thickened peritoneum (Hardie et al., 1994; Etchepareborde et al., 2010; Veiga-Parga et al., 2015; Danford et al., 2018; Sonck et al., 2018; Zhang et al., 2020; Tannoury and Abboud, 2021; Tsukada et al., 2022). Multifocal lymphatic dilation can be found (Sonck et al., 2018; Tannoury and Abboud, 2021). Mesothelial lining can be multifocally discontinuous or absent (Hardie et al., 1994; Malchado, 2016).

Diagnosis

Due to its vague clinical symptoms and rarity, preoperative diagnosis of SEP is challenging (Machado, 2016; Danford et al., 2018; Tannoury and Abboud, 2021). In most cases, a combination of history, pre-existing predisposing factors, clinical symptoms and abdominal imaging is needed for the diagnosis (Machado, 2016). In human medicine, diagnostic algorithms based on history, physical symptoms, lab examinations, medical imaging, surgery and histopathology are proposed (Danford et al., 2018). Since a high percentage of SEP patients are presented in emergency, diagnosis is often made during surgery (Machado, 2016).

Medical imaging has different modalities that help in diagnosing SEP, and lesions are similar in humans and animals. The most ideal technique is computed tomography (CT) scan, which shows very characteristic clumping of the intestinal loops in the central abdomen, encased by a thick peritoneal layer (Etchepareborde et al., 2010; Barnes, 2015; Veiga-Parga et al., 2015; Machado, 2016; Danford et al., 2018; Alshomini et al., 2021; Tannoury and Abboud, 2021; Tsukada et al., 2022). In cases with ascites, the abdominal fluid will also be clearly visible (Barnes, 2015; Danford et al., 2018; Alshomini et al., 2021; Tsukada et al., 2022). Other possible lesions recognized in humans are intestinal obstruction, mineralization of the peritoneum and/or the capsule of other organs, increased density of mesenteric fat, partial intestinal dilation and lymphadenopathy (Machado, 2016; Danford et al., 2018; Zhang et al., 2020; Tannoury and Abboud, 2021). Magnetic resonance imaging (MRI) likely has similar abilities to diagnose SEP; however this imaging technique is not commonly used (Machado, 2016; Danford et al., 2018).

Standard radiographic (RX) and ultrasound (US) abdominal images can also give a presumptuous diagnosis in humans as well as in animals; however, these are not very specific. RX will show typical gathering of intestinal loops in the central or dorsal abdomen (Machado, 2016; Tannoury and Abboud, 2021). Other lesions that can be found are intestinal dilation and peritoneal calcification (Machado, 2016; Danford et al., 2018). Sometimes RX can be complicated by loss of contrast (Sonck et al., 2018). A combination with contrast fluid will show delayed transit, distention of the intestinal pre-obstruction and a typical 'cauliflower' or 'accordion' appearance due to compression of adhered intestines (Machado, 2016; Danford et al., 2018; Tannoury and Abboud, 2021). US will show thickened, hyperechoic intestinal serosa, sometimes combined with hyperechoic string-like fibrous adhesions in between the intestinal loops (Adamama-Moraitou et al., 2004; Machado, 2016; Danford et al., 2018; Tannoury and Abboud, 2021). The intestinal loops are typically tethered together and dilated, with a very irregular trilaminar appearance (Danford et al., 2018; Tannoury and Abboud, 2021). In animals, large amounts of hyperflocculent ascites can be recognized (Hardie et al., 1994; Adamama-Moraitou et al., 2004; Sonck et al., 2018), which can complicate recognizing the other typical US lesions.

Abdominocentesis with microscopic examination of the abdominal fluid can be used to exclude certain differentials or to diagnose underlying etiologies, such as neoplasia or septic peritonitis (Hardie et al., 1994; Adamama-Moraitou et al., 2004). A definitive diagnosis can be made using histopathological examination of the thickened peritoneal capsule surrounding the small intestines (Machado, 2016; Danford et al., 2018), which can be visualized even better with specialized connective tissue stains, such as MT staining.

Differential diagnoses

Differential diagnoses in humans are congenital peritoneal encapsulation, septic peritonitis, tubercu-

lous peritonitis, intestinal malrotations and/or herniations, voluminous intussusception, chronic idiopathic intestinal pseudo-obstructions, peritoneal mesothelioma or other types of malignant neoplasia (primary or metastasis), autoimmune conditions and pseudomyxoma peritonei (Machado, 2016; Danford et al., 2018; Tannoury and Abboud, 2021). In dogs, differentials include different types of abdominal neoplasia, abscesses, hematomas, granulomas (Barnes, 2015). In cats, FIP, gastrointestinal neoplasia, sclerosing peritoneal mesothelioma and peritonitis secondary to perforation due to an ingested foreign object need to be in the differential list (Sonck et al., 2018).

Therapy

SEP in humans has a grave prognosis, with a mortality rate of up to 50-60% one year after diagnosis (Danford et al., 2018) and a mortality rate of up to 80% a few months post-surgery (Tannoury and Abboud, 2021). The prognosis is known to be worse when multiple abdominal organs are involved and when the peritoneal thickening is severe, which makes surgery more difficult. Prognosis in dogs has also been described to be very grave, with 93% of mortality within 13 months after diagnosis (Tsukada et al., 2022). Information about therapy is mainly based on human medicine and includes medication, surgery, nutritional support and therapy or cessation of the underlying disorders (Danford et al., 2018; Tannoury and Abboud, 2021). The therapy of choice differs from case to case, and in human medicine, it is decided based on the stage of the disease (Danford et al., 2018). However, these stages are not easy to differentiate from each other, so therapy with multiple aspects is most commonly used.

Multiple types of medications are used in human cases of SEP, of which corticosteroids, tamoxifen and colchicine are the most common (Machado, 2016; Danford et al., 2018; Tannoury and Abboud, 2021). The best medication depends on the stage of the disease (Danford et al., 2018). Corticosteroids can best be used in the inflammatory stage and have an immunosuppressive and anti-inflammatory function (Machado, 2016; Danford et al., 2018). They decrease formation of fibrous tissue and decrease the inflammatory response, which both slow the progression of the disease (Machado, 2016; Danford et al., 2018). Tamoxifen is a nonsteroidal and antiestrogen drug and is best used in the fibrotic stage (Machado, 2016; Danford et al., 2018). It produces TGF- β and metalloproteinase that both promote the degradation of collagen (Machado, 2016; Danford et al., 2018). Colchicin inhibits the messenger ribonucleic acid expression of TGF-β, and therefore also exhibits an anti-inflammatory response (Machado, 2016). Depending on the publication, all of these medicines show variable results (Danford et al., 2018). Medicinal therapies for SEP in animals has been rarely reported in the literature. In a study by Etchepareborde et al. (2010), treatment with tamoxifen of a German shepherd dog has been described with good results.

Different surgical techniques are used in humans (Danford et al., 2018). It is very important to realize the risks of these surgeries, as complicated and fatal results are not uncommon (Danford et al., 2018). This is why surgery is reserved only in case medicinal therapy does not have the desired effect (Danford et al., 2018) or when there are severe symptoms of intestinal obstruction (Machado, 2016). The most commonly used surgical technique is active total intestinal enterolysis, also called adhesiolysis, during which an acute ablation of the fibrous capsule and intestinal adhesions is performed (Machado, 2016; Tannoury and Abboud, 2021). This surgery needs to be combined with the removal of any necrotic organ parts and opening of possible fluid pockets. If possible, resection with secondary anastomosis of intestines should be avoided, since it is known to cause a higher incidence of post-surgical complications (Machado, 2016; Zhang et al., 2020; Tannoury and Abboud, 2021). Other surgical complications described in humans are early bowel obstructions, intra-abdominal infections, enterocutaneous fistulae, short bowel syndrome and bowel perforations (Machado, 2016; Tannoury and Abboud, 2021). Surgical techniques in dogs and cats have not successfully been described in the literature, with most of them causing multiple intestinal perforations during surgery with subsequent euthanasia (Etchepareborde et al., 2010; Barnes, 2015; Veiga-Parga et al., 2015).

Nutritional support by ensuring adequate nutrition is very important, mostly achieved by total parenteral nutrition (Danford et al., 2018). However, bowel rest has been described as a necessary aspect of therapy as well (Machado, 2016; Danford et al., 2018).

DISCUSSION AND CONCLUSION

Sclerosing encapsulating peritonitis is a rare disease, described in humans and in animals. In the two feline cases in this paper, the physical complaints of swollen and painful abdomen, anorexia and lethargy are described. Prominent vomiting and diarrhea, as seen in most veterinary cases and reported in the only two earlier described feline cases (Hardie et al., 1994; Sonck et al., 2018), were not noticed in the two cases of the present study.

Abdominal ultrasound was used in both cases and showed ascites and a thickened and irregular aspect of intestinal walls, as can be seen in SEP in dogs. No treatment was started in the cats, as one of them died the night after the ultrasound examination and as the other one was euthanatized due to the grave prognosis. Typical macroscopic and histological lesions were found. Etiopathogenesis of both cases remains unclear. Both cats were sterilized in the past. No ex-

act data regarding the sterilization of the first cat was known, but since this cat was already 12.5 years old, sterilization most likely happened several years ago. The second cat was sterilized three weeks before the consultation, and no significant abnormalities had been mentioned during that surgery. It is possible that in both these cats, sterilization was the triggering factor that started the SEP, as it has been described in humans that abdominal surgery and hysterectomy can cause SEP (Machado, 2016; Tannoury and Abboud, 2021). No other possible predisposing factors were noted in these cats.

Unfortunately, due to the extreme rarity of SEP in cats, a lot remains unknown regarding the exact etiopathogenesis, clinical symptoms, diagnostic methods and ideal treatment. In this article, the possible clinical symptoms and pathological findings during abdominal ultrasound, necropsy and histopathological examination were emphasized.

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