CASE REPORT

ISSN Online 1678-4456

Small loop bowel rupture due to feline gastrointestinal eosinophilic sclerosing fibroplasia: a case report

Ruptura de alça intestinal devido à fibroplasia esclerosante eosinofílica gastrointestinal felina: relato de caso

Daniel Pedro Bonjorno Maia Souto[1]; Carolina de Oliveira Ghirelli[1]

[1] Universidade Santo Amaro, São Paulo – SP, Brazil

ABSTRACT

A 1-year-old male Persian presented intense prostration and sporadic emesis. Abdominal ultrasound examination revealed an intestinal mass, thick loops, and lymph node enlargement associated with abdominal free gas and fluid. In surgery, a thickened and ruptured duodenum was noted. Histopathological and immunohistochemistry examination revealed feline gastrointestinal eosinophilic sclerosing fibroplasia. This article is relevant because of the poor literature that has been published. Besides, this is the first case of spontaneous rupture of bowel loops due to necrosis secondary to feline gastrointestinal eosinophilic sclerosing fibroplasia without a foreign body in a young cat.

Keywords: Cat. Intestine. Inflammation. Pneumoperitoneum.

RESUMO

Um gato macho, persa, apresentando prostração intensa e êmese esporádica. Avaliação ultrassonográfica abdominal revelou uma formação intestinal, segmentos intestinais espessados e linfonodomegalia, juntamente líquido e gás livres. Na cirurgia, o duodeno estava espesso e rompido. A avaliação histopatológica e imuno-histoquímica resultou em fibroplasia esclerosante gastrointestinal felina. Este artigo é relevante levando em conta a escassa literatura, principalmente por este ser o primeiro caso de fibroplasia esclerosante gastrointestinal felina com ruptura espontânea de um segmento intestinal por necrose e não pela presença de corpo estranho.

Palavras-chave: Gato. Intestino. Inflamação. Pneumoperitôneo.

Correspondence to:
Daniel Pedro Bonjorno Maia Souto
Universidade Santo Amaro
Rua Prof. Enéas de Siqueira Neto, 340, Jardim das Imbuias
CEP: 04829-300, São Paulo – SP, Brazil
e-mail: dpbonjorno@unisa.br

Received: September 01, 2023
Approved: June 24, 2024

How to cite: Souto DPBM, Ghirelli CO. Small loop bowel rupture due to feline gastrointestinal eosinophilic sclerosing fibroplasia: a case report. Braz J Vet Res Anim Sci. 2024;61:e215601. https://doi.org/10.11606/issn.1678-4456.bjvras.2024.215601.

Eosinophils are defense cells that, when unbalanced, can induce unique inflammatory issues in cats, such as eosinophilic granuloma, feline indolent ulcer, and hypereosinophilic syndrome (Buckley & Nuttall, 2012). The feline gastrointestinal eosinophilic sclerosing fibroplasia (FGESF) is one of those diseases which does not have a specific age range to occur, regardless of breed or gender predisposition (Craig et al., 2009; Linton et al., 2015). Macroscopically, it is observed as a rough, intramural, often ulcerated mass, more frequently in the gastrointestinal tract, especially in the pyloric sphincter region and the ileocecocolic junction (Craig et al., 2009; Davidson et al., 2021; Eckstrand et al., 2013; Linton et al., 2015; Martineau et al., 2023; Ozaki et al., 2003). However, it has been reported elsewhere (Craig et al., 2009; Goffart et al., 2022; Kambe et al., 2020; Thieme et al., 2019). In addition, descriptions of similar lesions in the subcutaneous and cervical have been reported (Ozaki et al., 2003; Zampieri et al., 2022). Their microscopic aspect is peculiar, observing big to irregular spindle-shaped cells, along with an extracellular pink matrix and a trabecular pattern of dense collagen that often resembles osteoid, with numerous eosinophils, neutrophils, and in some cases, a large population of mast cells. These aspects can lead to erroneous diagnoses of osteosarcoma or mastocytoma (Craig et al., 2009; Linton et al., 2015; Porras et al., 2022; Suzuki et al., 2013; Thieme et al., 2019). Bacteria can occasionally be present in the cytoplasm of neutrophils or the extracellular space, scattered across the bottom, in the form of micro abscesses and necrotic focus (Craig et al., 2009; Zampieri et al., 2022), in addition to other organisms such as fungi or parasites (Crossland et al., 2015; Eckstrand et al., 2013; Grau-Roma et al., 2014; Martineau et al., 2023). Usually, the lymph nodes can also be affected by the same sclerotic lesion of the intestine or eosinophilic lymphadenitis, with more typical fibrosis (Craig et al., 2009; Suzuki et al., 2013).

The pathophysiology of the lesion remains unknown. However, some hypotheses considered are intracellular bacterial infection by Staphylococcus aureus (Craig et al., 2009; Suzuki et al., 2013), deregulation of eosinophils, food hypersensitivity, dysbiosis, parasites and penetrating wounds due to foreign bodies (Craig et al., 2009; Crossland et al., 2015; Eckstrand et al., 2013; Linton et al., 2015). The role of bacteria and other agents in the development and perpetuation of the lesion is to provide a stimulus for exacerbated eosinophilic reaction, which remains in a vicious cycle of inflammation and fibrosis, even after the disappearance of the antigen (Craig et al., 2009; Grau-Roma et al., 2014; Linton et al., 2015). This is reinforced due to the similar appearance of pyogranulomatous lesions described in other studies (Eckstrand et al., 2013; Ozaki et al., 2003) and also because infectious agents are not found in all cases (Craig et al., 2009; Linton et al., 2015). It is not known how the antigen lodges in the intestinal wall, but the predisposition to occur in the pyloric sphincter or at the ileocolic junction suggests physical forces, such as penetration by foreign bodies (Craig et al., 2009; Linton et al., 2015).

The clinical signs are nonspecific, but the most common, in cases of intestinal involvement, are the history of chronic emesis, diarrhea, constipation, apathy, anorexia, and weight loss (Craig et al., 2009; Kambe et al., 2020; Linton et al., 2015; Martineau et al., 2023), which can occur in other illnesses such as inflammatory bowel disease or intestinal lymphoma (Garraway et al., 2018).

Laboratory tests can reveal neutrophilia, hyperglobulinemia with concurrent hyperproteinemia, hyperkalemia, and eosinophilia, although the latter is not seen in all cases (Kambe et al., 2020; Linton et al., 2015; Munday et al., 2014; Weissman et al., 2013).

In abdominal ultrasound, focal intestinal thickening with loss of stratification of layers is observed. These are also common aspects in neoplastic disorders, and some areas of high echogenicity interspersed inside, possibly related to areas of fibrosis (Larson & Biller, 2009; Weissman et al., 2013), there may also be free fluid and gas, hyperechogenicity of the mesentery, dilation of the intestinal loops and decreased motility, in cases of perforation and necrosis (Bernardin et al., 2015; Boysen et al., 2003). Lymph nodes adjacent to the mass may also show signs of involvement, in addition to being able to mimic metastatic nodules in some cases (Munday et al., 2014). The conclusive diagnosis is made only through biopsies. Immunohistochemistry is sometimes necessary (Craig et al., 2009; Martineau et al., 2023; Ozaki et al., 2003; Weissman et al., 2013;).

Despite having characteristics similar to the neoplastic process, the inflammatory context gradually transitions to more typical granulation tissue. This is associated with the fact that it can occur in very young animals, which are not aspects consistent with a neoplastic lesion (Craig et al., 2009).

The recommended treatment is surgical excision of the affected segment to avoid possible obstructions or ruptures. Besides that, some authors demonstrate that corticoid therapy can increase survival rate (Craig et al., 2009; Linton et al., 2015) when associated with immunomodulatory agents and antibiotic therapy, especially in cases where excision is not possible (Kambe et al., 2020). The injury prognosis is guarded, influenced by the patient's general condition, the lesion's location, and the treatment established. However, cases with good results have been reported (Craig et al., 2009; Kambe et al., 2020; Linton et al., 2015; Thieme et al., 2019; Weissman et al., 2013).

This article aims to report a case of FGESF in a young adult patient who developed a rupture of the affected intestinal segment.

A Persian-breed feline male, 1 year and 2 months old, was extremely prostrate with a history of sporadic emesis for seven days. Physical examination showed severe dehydration (10%), weak pulse, hypothermia, cyanosis, and systemic blood pressure of 90 mmHg. The animal was unaware of the environment, and the abdomen was rigid on palpation.

Upon admission, venous access was cannulated to perform fluid therapy. The minimum database was indicated for complete blood count, biochemical profile, and abdominal ultrasound.

Five months before, the patient had an abdominal ultrasound examination performed that showed a thickening of the duodenal segment (0.7 cm) and mesenteric lymph node enlargement. A biopsy was indicated then, but the owner declined the procedure. On ultrasound examination performed at the presentation, the duodenum persisted with markedly thickening and hypoechoic walls, with loss of stratification (0.83 cm) (Figure 1). Other intestinal segments were thickened and corrugated, with preserved stratification (0.34 cm). Mesenteric lymph node enlargement, free abdominal gas (Figure 2), and free fluid collection with high cellularity were also observed. The liver was homogeneous and markedly hypoechogenic, which may suggest toxemia.

Systemic blood pressure was reevaluated, showing a value of 60 mm Hg, even with fluid therapy at 7 mL/kg/h. Blood sample collection was not possible due to hypotension and severe dehydration. Because of sonographic findings and poor general condition, an exploratory laparotomy was indicated for diagnostic and therapeutic research, in addition to necessary interventions.

During the pre-surgical period, the patient underwent supportive treatment consisting of fluid therapy with Ringer Lactate (7 ml/kg/h), amoxicillin with potassium clavulanate (22 mg/kg/IV), metronidazole (10 mg/kg/IV), tramadol hydrochloride (2 mg/kg/IV), ranitidine hydrochloride (2 mg/kg/SC) and maropitant citrate (0.1 ml/kg/SC). Dobutamine (5 mcg/kg/min/IV), a vasoactive medication, was started due to decreased systolic blood pressure.

During surgery, a large amount of free fluid and thickening of the duodenum were identified, with rupture of its antimesenteric margin (Figure 3). However, due to the location, it was not possible to perform a resection. A biopsy of the bowel and lymph node was performed, and the rupture was sutured. Free abdominal fluid was blurred and sent for analysis, where the presence of red blood cells, degenerated neutrophils, vacuolated macrophages, lymphocytes, and eosinophils was identified, as well as cocos and bacilli of the Clostridium genus. In microbiological culture, Escherichia coli grew.

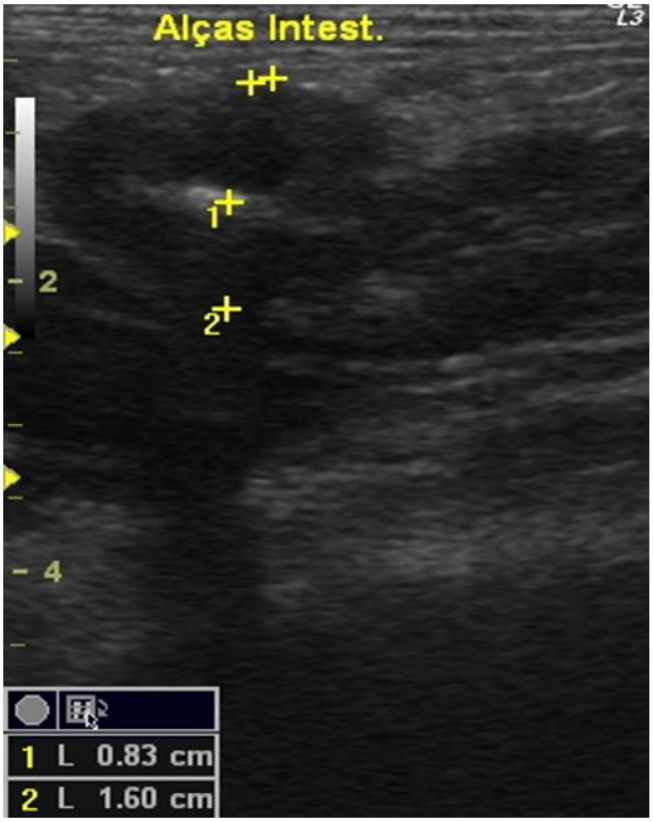

Figure 1 – Ultrasonographic transversal plane of duodenal mass. The duodenal loop here is thicker beyond the superior limit, often observed in cats (between cursors number 1 – 0.83 cm), and has a great diameter (between cursors number 2 – 1.60 cm). Examiner: Carolina de Oliveira Ghirelli.

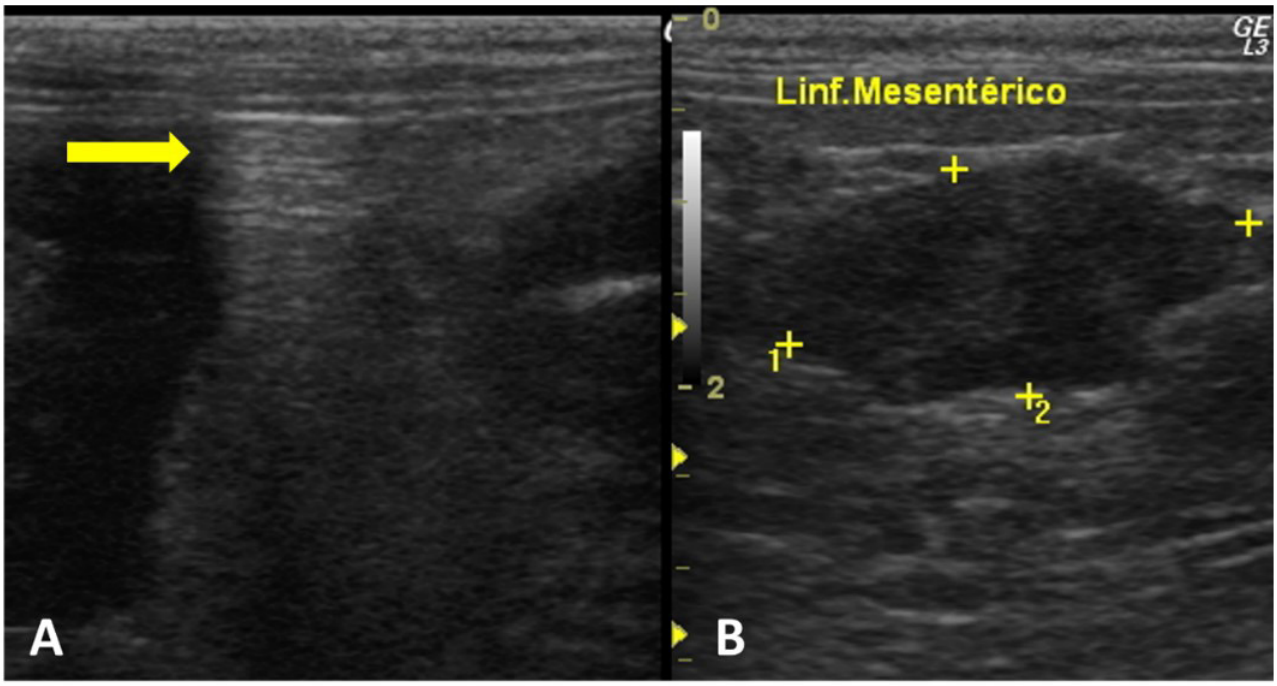

Figure 2 – (A) Reverberating hyperechoic free gas content observed as subtle hyperechoic horizontal lines (arrow) and (B) lymph node enlargement (between cursors). Note that the lymph node has a rough echotexture and is hypoechoic, demonstrating an essential reactional process. Examiner: Carolina de Oliveira Ghirelli.

After surgery, the patient remained hospitalized with the first protocol medication prescriptions. However, after approximately 4 h post-surgery, the patient suffered cardiorespiratory arrest and died.

In histopathology, feline gastrointestinal eosinophilic sclerosing fibroplasia was diagnosed. Histopathological material was also submitted to an immunohistochemical study using vimentin, smooth muscle actin, and desmin antibodies. The results were positive for all tested antibodies.

The present case reports a male cat diagnosed with FGESF, a disease first described in 2009 (Craig et al., 2009), which presented ultrasound changes since seven months old, with emesis as the primary manifestation, like in other reports (Craig et al., 2009; Kambe et al., 2020; Linton et al., 2015). The wide age range of patients affected by this condition, evidenced in the literature (Craig et al., 2009; Eckstrand et al., 2013), shows the importance of including this differential diagnosis even in kittens (Craig et al., 2009; Linton et al., 2015).

As the tutors decided not to investigate the alterations found at that time, the disease course was progressive, leading to acute abdomen five months later. At this point, ultrasound aspects were worse in intestinal wall thickness and mesenteric lymph node enlargement (Craig et al., 2009; Linton et al., 2015; Weissman et al., 2013), in addition to aspects consistent with rupture of the gastrointestinal tract (Boysen et al., 2003). It is worth mentioning that no case in the literature has been reported with these characteristics (Grau-Roma et al., 2014; Linton et al., 2015; Martineau et al., 2023; Weissman et al., 2013), although in one case, intestinal perforation due to FGESF was observed at necropsy (Craig et al., 2009).

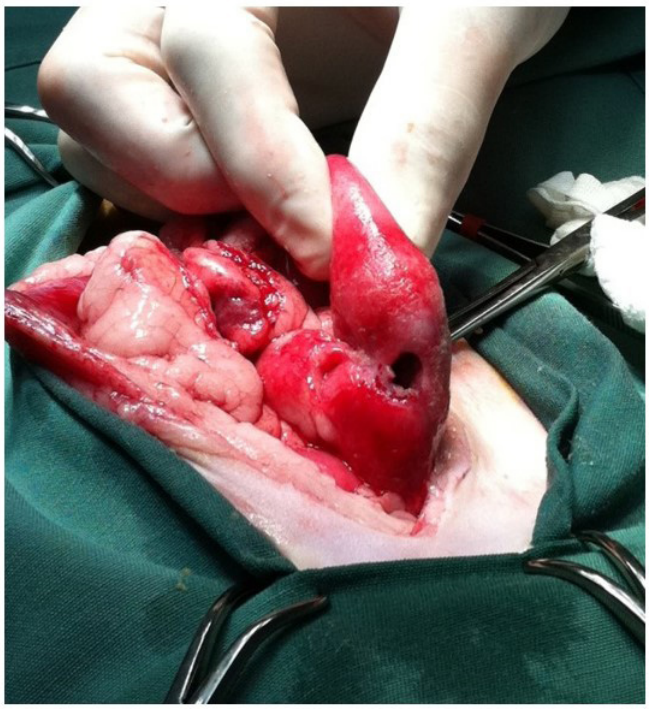

Figure 3 – Antimesenteric duodenal margin ulcerated. The loop also presents an exacerbated congestion and rough aspect. Photography: Carolina de Oliveira Ghirelli.

In this case, depending on the lesion location, it is not possible to remove it surgically (Craig et al., 2009; Goffart et al., 2022; Kambe et al., 2020; Thieme et al., 2019). The histopathological description of fragments is compatible with those described in other articles (Craig et al., 2009; Linton et al., 2015; Suzuki et al., 2013). The knowledge of veterinarian pathologists is vital to differentiate this lesion from neoplastic processes (Craig et al., 2009; Linton et al., 2015). Fragments were also submitted to immunohistochemical analysis, testing positive for all antibodies as observed in previous studies (Craig et al., 2009; Linton et al., 2015; Suzuki et al., 2013).

Due to severe clinical condition, unfortunately, patient died within hours after surgical procedure, making any comparison with literature data regarding animal's survival or the drugs used in cases of FGESF impossible (Craig et al., 2009; Linton et al., 2015; Martineau et al., 2023).

There are currently few reports of FGESF in literature. Its final diagnosis is a significant clinical, ultrasound, and histopathological challenge due to its similarity with other conditions, such as neoplasms and inflammatory bowel disease. This disease should be considered in cats with emesis, regardless of age, and when ultrasound examination identifies intestinal wall thickening and mesenteric lymph node enlargement. Although uncommon, it should be considered in felines presenting an acute abdomen due to intestinal perforation.

Conflict of Interest

The authors declare that there is no conflict of interest.

Ethics Statement

It was not necessary to submit it to the ethics committee as it was a case seen in the routine of a veterinary service.

References

Bernardin F, Martinez Rivera L, Ragetly G, Gomes E, Hernandez J. Spontaneous gastrointestinal perforation in cats: a retrospective study of 13 cases. J Feline Med Surg. 2015;17(10):873-9. http://doi.org/10.1177/1098612X14558894. PMid:25406178.

Boysen SR, Tidwell AS, Penninck DG. Ultrasonographic findings in dogs and cats with gastrointestinal perforation. Vet Radiol Ultrasound. 2003;44(5):556-64. http://doi.org/10.1111/j.1740-8261.2003.tb00507.x. PMid:14599169.

Buckley L, Nuttall T. Feline eosinophilic granuloma complex(ities): some clinical clarification. J Feline Med Surg. 2012;14(7):471-81. http://doi.org/10.1177/1098612X12451549. PMid:22736681.

Craig LE, Hardam EE, Hertzke DM, Flatland B, Rohrbach BW, Moore RR. Feline gastrointestinal eosinophilic sclerosing fibroplasia. Vet Pathol. 2009;46(1):63-70. http://doi.org/10.1354/vp.46-1-63. PMid:19112117.

Crossland NA, Hanks CR, Ferguson JA, Kent ML, Sanders JL, Del Piero F. First report of *Cylicospirura felineus* in a feral domestic shorthair cat in North America. JFMS Open Rep. 2015;1(2):2055116915593964. http://doi.org/10.1177/2055116915593964. PMid:28491368.

Davidson GA, Taylor SS, Dobromylskyj MJ, Gemignani F, Renfrew H. A case of an intramural, cavitated feline gastrointestinal eosinophilic sclerosing fibroplasia of the cranial abdomen in a domestic longhair cat. JFMS Open Rep. 2021;7(1):2055116921995396. http://doi.org/10.1177/2055116921995396. PMid:33747539.

Eckstrand CD, Barr BC, Woods LW, Spangler T, Murphy B. Nematode-associated intramural alimentary nodules in pumas are histologically similar to gastrointestinal eosinophilic sclerosing fibroplasia of domestic cats. J Comp Pathol. 2013;148(4):405-9. http://doi.org/10.1016/j.jcpa.2012.07.003. PMid:22925264.

Garraway K, Johannes CM, Bryan A, Peauroi J, Rossi G, Zhang M, Wang C, Allenspach K, Jergens AE. Relationship of the mucosal microbiota to gastrointestinal inflammation and small cell intestinal lymphoma in cats. J Vet Intern Med. 2018;32(5):1692-702. http://doi.org/10.1111/jvim.15291. PMid:30084202.

Goffart LM, Durand A, Dettwiler M, Vincenti S. Feline gastrointestinal eosinophilic sclerosing fibroplasia presenting as a rectal mass. JFMS Open Rep. 2022;8(2):20551169221114330. http://doi.org/10.1177/20551169221114330. PMid:35966900.

Grau-Roma L, Galindo-Cardiel I, Isidoro-Ayza M, Fernandez M, Majó N. A Case of feline gastrointestinal eosinophilic sclerosing fibroplasia associated with phycomycetes. J Comp Pathol. 2014;151(4):318-21. http://doi.org/10.1016/j.jcpa.2014.08.005. PMid:25444079.

Kambe N, Okabe R, Osada H, Ogawa M, Kishimoto M, Fukushima R, Kondo H, Ohmori K. A case of feline gastrointestinal eosinophilic sclerosing fibroplasia limited

to the mesentery. J Small Anim Pract. 2020;61(1):64-7. http://doi.org/10.1111/jsap.12847. PMid:29708273.

Larson MM, Biller DS. Ultrasound of the gastrointestinal tract. Vet Clin North Am Small Anim Pract. 2009;39(4):747-59. http://doi.org/10.1016/j.cvsm.2009.04.010. PMid:19531399.

Linton M, Nimmo JS, Norris JM, Churcher R, Haynes S, Zoltowska A, Hughes S, Lessels NS, Wright M, Malik R. Feline gastrointestinal eosinophilic sclerosing fibroplasia: 13 cases and review of an emerging clinical entity. J Feline Med Surg. 2015;17(5):392-404. http://doi.org/10.1177/1098612X14568170. PMid:25896239.

Martineau M, Tilmant C, Risco Castillo V, Guillot J, Reyes-Gomez E, Benchekroun G, Freiche V. A case of feline gastrointestinal eosinophilic sclerosing fibroplasia associated with fungal colonisation: endoscopic features, treatment and follow-up. JFMS Open Rep. 2023;9(1):20551169231165246. http://doi.org/10.1177/20551169231165246. PMid:37205163.

Munday JS, Martinez AW, Soo M. A case of feline gastrointestinal eosinophilic sclerosing fibroplasia mimicking metastatic neoplasia. N Z Vet J. 2014;62(6):356-60. http://doi.org/10.1080/00480169.2014.933905. PMid:24926904.

Ozaki K, Yamagami T, Nomura K, Haritani M, Tsutsumi Y, Narama I. Abscess-forming inflammatory granulation tissue with gram- positive cocci and prominent eosinophil infiltration in cats: possible infection of methicillin-resistant *Staphylococcus.* Vet Pathol. 2003;40(3):283-7. http://doi.org/10.1354/vp.40-3-283. PMid:12724569.

Porras N, Rebollada-Merino A, Rodríguez-Franco F, Calvo-Ibbitson A, Rodríguez-Bertos A. Feline gastrointestinal eosinophilic sclerosing fibroplasia: extracellular matrix proteins and TGF-β1 immunoexpression. Vet Sci. 2022;9(6):1-16. http://doi.org/10.3390/vetsci9060291. PMid:35737343.

Suzuki M, Onchi M, Ozaki M. A case of feline gastrointestinal eosinophilic sclerosing fibroplasia. J Toxicol Pathol. 2013;26(1):51-3. http://doi.org/10.1293/tox.26.51. PMid:23723568.

Thieme ME, Olsen AM, Woolcock AD, Miller MA, Simons MC. Diagnosis and management of a case of retroperitoneal eosinophilic sclerosing fibroplasia in a cat. JFMS Open Rep. 2019;5(2):2055116919867178. http://doi.org/10.1177/2055116919867178. PMid:31452913.

Weissman A, Penninck D, Webster C, Hecht S, Keating J, Craig LE. Ultrasonographic and clinicopathological features of feline gastrointestinal eosinophilic sclerosing fibroplasia in four cats. JFMS Open Rep. 2013;15(2):148-54. http://doi.org/10.1177/1098612X12464224. PMid:23076597.

Zampieri B, Church ME, Walsh K, Lennon EM. Feline eosinophilic sclerosing fibroplasia: a characteristic inflammatory response in sites beyond the gastrointestinal tract: case report and proposed nomenclature. JFMS Open Rep. 2022;8(2):20551169221117516. http://doi.org/10.1177/20551169221117516. PMid:36003069.

Financial Support: None.