



Case Report

Feline gastrointestinal eosinophilic sclerosing fibroplasia

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Abstract

A female Persian cat arrives for clinical assessment with a 3-month history of weight loss and sporadic vomiting. The clinical and paraclinical findings were hypodynamia cachexia, leukocytosis and presence of a mass in duodenum. Histopathological evaluation revealed a non-neoplastic tumor proliferation, which was organized into dense, sclerotic-like connective tissue trabeculae that anastomosed, with cells of spindle-shaped morphology, elongated and rounded nuclei with prominent nucleoli and fine granular chromatin. These cells were intermingled with abundant eosinophils and in smaller proportion lymphocytes, macrophages, plasma cells, with transmural distribution. Masson's trichrome differential staining trabeculae of collagen fibers. Based on the clinical and microscopic findings, the diagnosis of feline eosinophilic sclerosing fibroplasia is established, being this pathology's first documented report in Colombia.

Key words: cat, intestinal obstruction, abdominal mass, non-neoplastic.

Introduction

Feline gastrointestinal eosinophilic sclerosing fibroplasia (FGESF) is a recently documented non-neoplastic inflammatory fibroproliferative disease, usually limited to the gastrointestinal tract and associated lymph nodes (9, 2). This disease has been classified as an emerging entity; therefore, the prevalence is still unknown (9). Reports indicate that the pathology is present in several countries such as Brazil, United States, Japan, New Zealand, United Kingdom, among others (6, 13). At the same time, the etiology is still unknown. Several factors have been documented that may be related to the appearance or development of the disease (5, 11). The typical clinical manifestation is characterized by chronic vomiting and/or diarrhea, associated with weight loss, anorexia and lethargy, in addition to the presence of a mass on abdominal palpation which may be accompanied by pain. The location of the mass is usually preferentially in the stomach, pyloric sphincter, ileum and ileocecal junction, where ultrasound is usually necessary to confirm its location (4, 9,10). Hematologic findings are variable; however, eosinophilia may be present in

approximately 50-60% of cases (9). Definitive diagnosis is made by histopathology in correlation with clinical findings (6,11). Microscopic changes that guide the diagnosis are given by the presence of long fascicles of mature collagen organized as trabeculae accompanied by inflammatory infiltrate rich in eosinophils (3, 8,11). There is no defined therapeutic protocol as of writing date. However, it has been observed that surgical resection and the inclusion of a corticosteroid and/or an immunomodulatory agent such as cyclosporine improves patient survival (4,9). The prognosis is guarded but may improve with timely treatment (5).

Case Description

A 6-year-old female Persian cat from Ipiales-Nariño was admitted to the veterinary clinic with a clinical history of chronic sporadic vomiting, progressive weight loss, decay, leukocytosis and thrombocytosis. Clinical examination revealed a mass on abdominal palpation which was confirmed by ultrasound. It was decided to perform a celiotomy, surgical excision of the mass and histopathological evaluation.



Figure 1. Abdominal US; duodenal mass. Note mass mixed echogenicity, which is also well defined and without apparent liquid content, approximately 18.5 mm wide and 35.2 mm long.

Diagnostic Examination

Ultrasonographic findings

Ultrasonographic (US) imaging revealed a mass located in duodenum (Fig. 1).

Exploratory celiotomy

A firm mass was identified within the cranial abdomen, whitish in color and irregular surface, dimensions 4.0 cm X 2.0 X 3.5 cm (Fig. 3). Surgical exeresis was performed and the mass was preserved in 10% buffered formaldehyde to perform analyses with haematoxylin and eosin (H&E) staining.

Histopathological evaluation

A non-neoplastic proliferation was observed forming



Figure 2. Exploratory celiotomy; duodenum segment. Note the mass in the cranial region of the duodenum. There is evidence of serosa of multilobulated aspect and irregular surface.

trabeculae made up of dense connective tissue of sclerotic appearance, which formed shunts in foci, separated by a population of spindled cells, elongated nuclei y fine granular chromatin. This lesion was accompanied by eosinophils, lymphocytes, macrophages, and plasma cells, with transmural distribution. In mucosa, extense ulcer associated with inflammatory infiltrate made up of neutrophils, lymphocytes and bacterial colonies.

Histochemical staining

Based on H&E findings, a Masson's trichrome differential staining was performed to determine collagenous component presence.

According to clinical, US and histopathological findings, FGESF is confirmed as the pathological entity, making this the first documented report of this pathology in Colombia.



Figure 3. Duodenum, ventral view. Firm mass, oval-shaped, with ulcerated appearance, concurring with ultrasonographic findings.



Figure 4. Duodenum. H&E. Note non-neoplastic transmural cell proliferation, characterized by dense connective tissue trabeculae (arrowheads)



Figure 5. Duodenal mass. H&E. Dense connective tissue trabeculae formation that shunt in foci (black arrows). Between trabeculae, considerable inflammatory cell presence made up of lymphocytes, macrophages, plasma cells and eosinophils. Left inferior corner. Multiple eosinophils (arrowheads) are evidenced accompanying the fibroblast proliferation.



Figure 6. Masson's trichrome staining. Staining was positive for collagen, confirming connective tissue trabeculae of coarse collagen, characteristic of Feline Gastrointestinal Eosinophilic Sclerosing Fibroplasia.

Discussion

Feline gastrointestinal sclerosing fibroplasia has been described in countries such as Brazil, United States, Japan, New Zealand and United Kingdom and this disease is probably of worldwide distribution. It has been observed that the mean age of presentation is approximately between 7 and 8.8 years (4,9); however, studies with a larger number of cases are required to establish the age range of presentation. In turn, the Ragdoll breed is described as having a higher frequency of presentation (9); however, at present, a breed predisposition for the presentation of this disease cannot be established. In the studied case, the patient is a 6-year-old Persian breed, like what has been reported.

Considering the macroscopic and microscopic features of the lesion, differential diagnoses should be considered. The macroscopic findings correspond to a hard mass that usually presents resistance to cutting, which can be associated with lymphoma, granuloma and intestinal adenocarcinoma (3). However, microscopic analysis is decisive in the diagnosis of the disease. The microscopic characteristics are defined by a distinctive network of dense connective tissue trabeculae, a finding that must be differentiated from other pathologies such as intestinal mastocytoma, fibrosarcoma or extraskeletal osteosarcoma. That is why the determining factors in the diagnosis of FGESF are, on the one hand, the large population of fibroblasts, and on the other hand, the nature of the inflammatory infiltrate (3, 10,11). In addition, Masson's trichrome differential staining can be used to confirm evidence of a collagen matrix (1). These factors in correlation with the patient's clinical presentation allow the definitive diagnosis to be established.

The pathophysiology of the disease is not well defined. It has been suggested that cats that develop eosinophilic reactions have an inherited eosinophili dysregulation, leading to an inappropriate eosinophilic inflammatory response that may respond to a variety of dietary, environmental, parasitic or intestinal dysbiosis-associated stimuli (7). On the other hand, several bacterial agents such as E. coli, Clostridium, Staphylococcus and mixed anaerobic species have been implicated in the lesions; however, the role of these agents to date is not clearly defined as primary or secondary (9, 13). In this report, bacterial colonies associated to the lesion were evidenced; therefore, it is recommended in cases suspicious to this entity to perform a microbiological culture in addition to the histopathological analysis.

Finally, it should be noted in this case the large number of eosinophils that accompanied the lesion. In the pathophysiology of the disease, it has been considered that sclerotic fibrosis is promoted by eosinophilic mediators, which may be key to the development of sclerotic fibrosis (14). Regarding the latter factor, eosinophils are known to stimulate fibrosis through the production of mediators such as transforming growth factor beta (TGF- β), IL-1 β , major basic protein (MBP) and IL-6 (5). Therefore, it is presumed that this process may result in the formation of abdominal mass.

There is no clarity regarding the ideal therapeutic protocol in patients with FGESF; however, it has been observed that the inclusion of a corticosteroid favors a longer survival time in individuals. In a study, Linton and collaborators (9) evaluated three therapeutic regimens. The "combination" group was treated with immunomodulatory agents, antibiotics and surgical resection. The group classified as "medical management" was treated with antibiotics, immunomodulatory agents and/or dietary management. The third group represented by "surgery" was treated only with surgical resection and antibiotics. The study suggests that individuals who were treated with immunomodulatory agents had the highest survival time, and in turn those who were treated with a combination of surgical resection, antibiotics and immunomodulators had the longest survival time. Therefore, although there is no well-defined therapeutic protocol, multimodal therapy seems to be the best option. In the present case, "combination" therapy was initially used and subsequently, four months later, recurrence of the mass occurred, and "medical management" was chosen, achieving stabilization in the progression of mass growth up to the date of the present report.

A relevant clinical aspect is the location of the lesion, because there is a relationship with the survival of the patients. It has been observed that survival time is shorter when the lesion is in the pyloric sphincter, while it is longer when the mass is in the intestine (4). In this case, the lesion was in the cranial region of the duodenum, relapsing four months after surgical exercise.

Conclusion

This is the first case report in Colombia. Although it is a rare pathology, it should be considered in the list of differential diagnoses in felines with masses in the gastrointestinal tract. The macroscopic characteristics (hard masses of ulcerated appearance located in the stomach, pyloric region, ileum and ileocecal region) are important for the surgeon to suspect the disease. Histopathology is a fundamental tool, since it allows to accurately differentiate a neoplastic lesion from a nonneoplastic tumor pathology, therefore, it is recommended that all masses in the abdominal cavity be referred for histopathological analysis.

Conflict of interests

Authors declare no conflict of interests in regard to investigation, authorship and/or article publication.

References

- Agulla B, Díaz D, García M, Rodríguez F, Villaescusa A, Rodríguez A, Pérez C, Sainz A. Remission of feline gastrointestinal eosinophilic sclerosing fibroplasia in a cat treated with corticotherapy. Pak. Vet. J. 2021; 41(2):309-12.
- 2. Brloznik M, Faraguna S, Goc M, Svara T. Recurrent feline gastrointestinal eosinophilic sclerosing fibroplasia and presumptive eosinophilic cystitis in a domestic short-haired cat: a case report. Vet Med (Praha). 2017; 62(5):295-300.
- Cho M J., Kim M, Seo K. Feline Gastrointestinal Eosinophilic Sclerosing Fibroplasia in a Bengal Cat. J. Vet. Clin. 2017; 34(6):481-3.
- Craig L, Hardam E, Hertzke D, Flatland B, Rohrbach B, Moore R. Feline gastrointestinal eosinophilic sclerosing fibroplasia. Vet. Pathol. 2009; 46(1):63-70.
- Davidson G, Taylor S, Dobromylskyj M, Gemignani F, Renfrew H. A case of an intramural, cavitated feline gastrointestinal eosinophilic sclerosing fibroplasia of the cranial abdomen in a domestic longhair cat. J. Feline Med. Surg. Open Reports. 2021; 7(1):1-6.
- De Souza G, De Oliveira K, Pereira R. Fibroplasia esclerosante eosinofilica gastrointestinal felina: relato de caso. Ciência Animal. 2017; 27(3):99-109.
- Grau L, Galindo I, Isidoro M, Fernandez M, Majó N. A case of feline gastrointestinal eosinophilic sclerosing fibroplasia associated with phycomycetes. J. Comp. Pathol. 2014; 151(4):318-21.
- 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.
- Linton M, Nimmo J, Norris J, Churcher R, Haynes S, Zoltowska A, Huges S, Lessels N, 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.
- Martín C, Oliver N, Manzano E, Pérez A. Fibroplasia esclerosante eosinofilica gastrointestinal felina: a propósito de un caso clínico. Madrid, Facultad de Medicina Universidad Complutense de Madrid (UCM). XIV Congreso Nacional de Investigación para Estudiantes Pregraduados de Ciencias de la Salud. 2021;192-8.
- Munday J, Martinez A, Soo M. A case of feline gastrointestinal eosinophilic sclerosing fibroplasia mimicking metastatic neoplasia. N Z Vet J. 2014; 62(6):356-60.
- Suzuki M, Onchi M, Ozaki, M. A case of feline gastrointestinal eosinophilic sclerosing fibroplasia. J. Toxicol. Pathol. 2013; 26(1):51-3.

- Thieme M, Olsen A, Woolcock A, Miller M, Simons M. Diagnosis and management of a case of retroperitoneal eosinophilic sclerosing fibroplasia in a cat. J. Feline Med. Surg. Open Reports. 2019; 5(2):1-7.
- Weissman A, Penninck D, Webster C, Hecht S, Keating J, Craig L. Ultrasonographic and clinicopathological features of feline gastrointestinal eosinophilic sclerosing fibroplasia in four cats. J. Feline Med. Surg. 2012; 15(2):148-54.