



A case of an intramural, cavitated feline gastrointestinal eosinophilic sclerosing fibroplasia of the cranial abdomen in a domestic longhair cat

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Abstract

Case summary A 5-year-old neutered male domestic longhair cat was presented for the investigation of a cranial abdominal mass following a 1-month history of inappetence and lethargy. Abdominal ultrasound revealed a large cavitated mass confluent with the mesenteric aspect of the descending duodenum. At surgery, the mass was found to involve the pylorus, proximal duodenum and pancreas, and was non-resectable. Histopathological examination of surgical biopsies revealed a non-neoplastic process involving eosinophils and fibroplasia.

Relevance and novel information This case report describes an uncommon feline gastrointestinal pathology with an unusual appearance that may provide an additional differential diagnosis other than neoplasia or abdominal abscess when confronted with a cavitated abdominal mass in cats.

Keywords: Mass; cranial abdomen; fibrosis; eosinophils; non-neoplastic; sclerosing fibroplasia

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Case description

A 5-year, 2-month-old neutered male domestic longhair cat presented to the primary veterinary surgeon after a 1-month history of inappetence, polydipsia and lethargy. Physical examination revealed a firm, non-painful cranial abdominal mass. Eosinophilia ($1.49 \times 10^9/l$; reference interval [RI] $0.00\text{--}1.00 \times 10^9/l$), neutrophilia ($18.47 \times 10^9/l$; RI $2.50\text{--}14.0 \times 10^9/l$) and a monocytosis ($2.25 \times 10^9/l$; RI $0.00\text{--}1.50 \times 10^9/l$) were observed on the complete blood count performed in-house. A mildly elevated urea (12.9 mmol/l ; RI $3.6\text{--}10.0 \text{ mmol/l}$), creatinine ($192 \text{ }\mu\text{mol/l}$; RI $27\text{--}186 \text{ }\mu\text{mol/l}$) and total protein (89 g/l ; RI $54\text{--}82 \text{ g/l}$) were also noted. Ultrasonography at the primary veterinary practice revealed a large, irregularly shaped heterogeneous mass in the cranial abdomen. Ten months previously the cat had been treated symptomatically with fluid therapy and antibiotics for presumed cholangiohepatitis (raised liver enzymes) and at that

time an eosinophilia was also documented ($1.8 \times 10^9/l$; RI $0.00\text{--}0.79 \times 10^9/l$). The cat had recovered uneventfully and blood tests were not repeated.

One week later, examination at the referral centre revealed the cat had continued to be inappetent and lethargic, and physical examination revealed a very firm, irregular mass in the cranioventral abdomen. Repeat biochemistry revealed a marginal improvement in azotaemia (urea 12.4 mmol/l [RI $6.1\text{--}12.5 \text{ mmol/l}$], creatinine $155 \text{ }\mu\text{mol/l}$ [RI $45\text{--}170 \text{ }\mu\text{mol/l}$]) and a more

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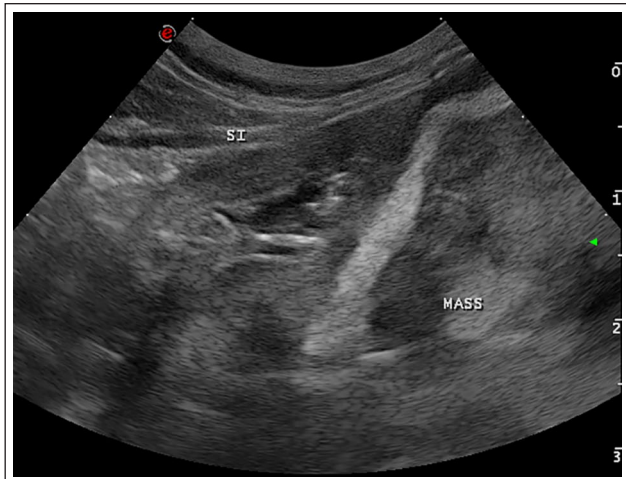


Figure 1 Transverse section of duodenum contiguous with a mass (note hypoechoic duodenal contents with bright strands crossing the lumen, and poorly distinguishable intestinal layering)

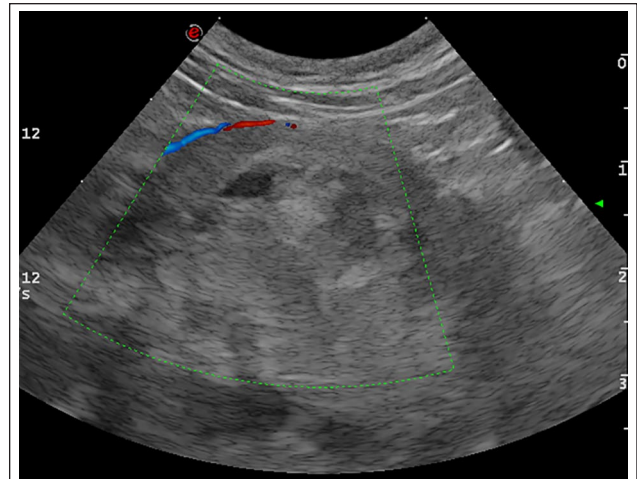


Figure 2 Mass showing peripheral blood flow (cranial is to the left)

marked eosinophilia ($2.6 \times 10^9/l$; RI $0.0\text{--}1.5 \times 10^9/l$). Additionally, a mild non-regenerative anaemia (packed cell volume 21.1%) was documented.

Ultrasound findings

The cat was sedated (butorphanol 0.3 mg/kg IV and dexmedetomidine 3 µg/kg IV). The abdominal ultrasound examination was dominated by a very large (5.5 × 6.7 cm) lobulated mixed-echogenicity cranial abdominal mass apparently contiguous with a short section of descending duodenum (Figure 1). The associated duodenum was mildly dilated with anechoic material and echogenic strands crossing the lumen. The duodenal wall showed abnormal layering, with poorly distinguished layers where the submucosa appeared thicker than usual and had an overall increased echogenicity. The wall contiguous with the mass was separated from it in transverse section by a thick and highly echogenic but non-shadowing wall. This wall surrounded the mass but varied in echogenicity, being quite hypoechoic in many places. The mass had a large volume of mixed echogenicity content with no identifiable blood flow using colour Doppler. Instead, blood flow was identified peripherally (Figure 2).

There was a marked mass effect with a consequentially distorted gastrointestinal tract and pancreas. The pancreas was not clearly identified. No further lesions were identified affecting the stomach wall or the other intestines; however, a dumbbell-shaped small mass, again of mixed echogenicity, was found running between the caudal vena cava and the portal vein at the porta hepatis (Figure 3). This was not the adrenal gland and was thought to be a lymph node or a nodule associated with the mass.

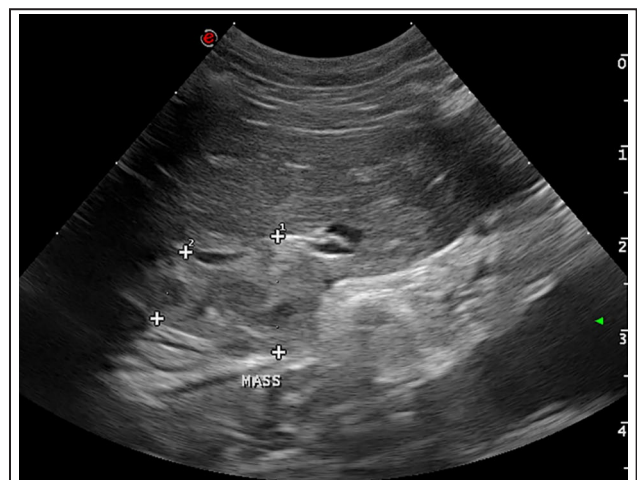


Figure 3 Dumbbell-shaped lymph node/nodule at porta hepatis (cranial is to the left)

Exploratory coeliotomy

A midline coeliotomy from xiphoid to pubis was performed and the abdominal contents examined. Apart from the mass lesion, no other gross abnormalities were identified. A large, firm, pale multilobulated mass of approximately 10 cm × 8 cm was identified in the cranial abdomen, adherent to the pylorus and confluent with the proximal duodenum which entered the wall of the mass dorsally (Figure 4). The pancreas was identifiable, but the left limb was firmly adhered to/entering the wall of the mass. The bile duct was seen to enter the mass in the region where the duodenum was lost to view within the mass.

Local lymph nodes were firm, enlarged and brown. The mass was incised and had a firm, defined 'capsule' of approximately 1–1.5 cm thickness. The contents of the mass were softer pale tissue with pockets of pale-coloured semi-liquid material that felt gritty and

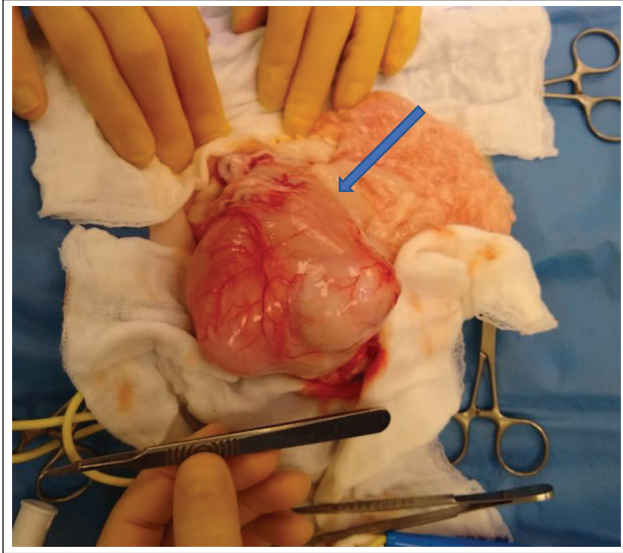


Figure 4 Large mass in the cranial abdomen. Cranial is at the top of the image, and the blue arrow indicates the region of the pylorus with the proximal duodenum confluent with the dorsal portion of the mass (not visible in image)

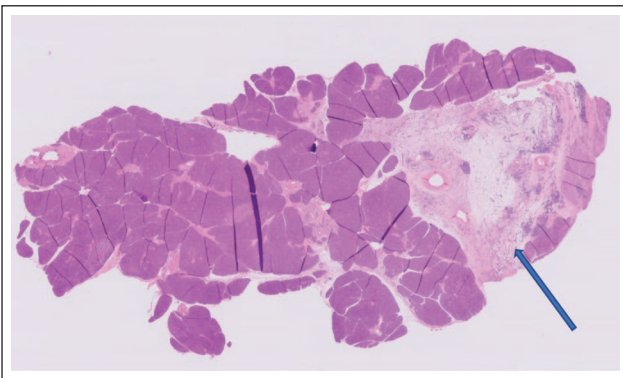


Figure 5 Pancreas. Focal area where there is expansion of the interstitium by oedema, fibrous connective tissue and an eosinophilic and lymphoplasmacytic inflammatory cell infiltrate (blue arrow)

contained more solid concretions. This gross appearance was consistent with a lobulated abscess containing purulent material, but no foreign body was identifiable within the lesion. Samples were obtained from the mass and the owners elected euthanasia due to the complex nature of resection, which would have necessitated a biliary diversion surgery, duodenectomy and partial pancreatectomy.

Histopathological findings

Representative post-mortem samples were submitted from the mass, left limb of the pancreas, stomach, adjacent lymph nodes and a section of tissue suspected to be an abscess with potential foreign body. Expansile lesions that were histologically similar were present in the pancreas



Figure 6 Stomach. The mass lesion (blue arrow) extends from the submucosa through the muscular layers of the stomach. There is focal erosion of the overlying mucosa (yellow arrow)

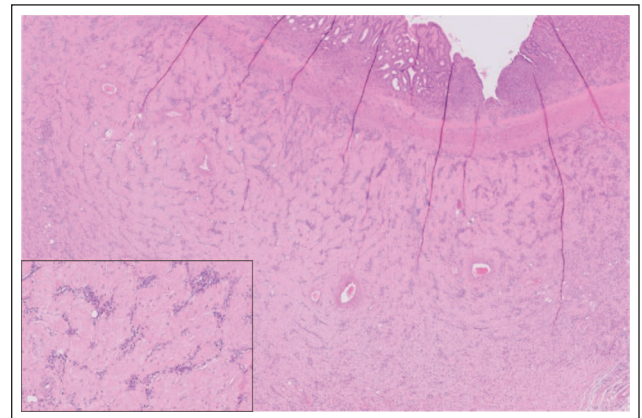


Figure 7 Stomach. Higher magnification image of Figure 6, better demonstrating fibrous connective tissue, fibroblasts and the inflammatory cell population. Inset bottom left: higher magnification view of connective tissue and cellular infiltrate

(Figure 5), the stomach (Figures 6 and 7) and lymph nodes (Figure 8). Tissue consisted of thick trabeculae of mature avascular collagen (Figure 9), which separated moderate numbers of reactive fibroblasts. Fibrous tissue was randomly infiltrated by an inflammatory cell population comprising of mostly lymphocytes, plasma cells, eosinophils and fewer neutrophils, consistent with the previous descriptions of feline gastrointestinal eosinophilic sclerosing fibroplasia (FGESF).¹ There were no histological features of malignancy and no infectious microorganisms were observed on haematoxylin and eosin-stained slides.

Discussion

FGESF is a poorly elucidated condition in cats that is characterised by mature collagen, eosinophils and reactive fibroblasts.¹ Cats with FGESF often present to the

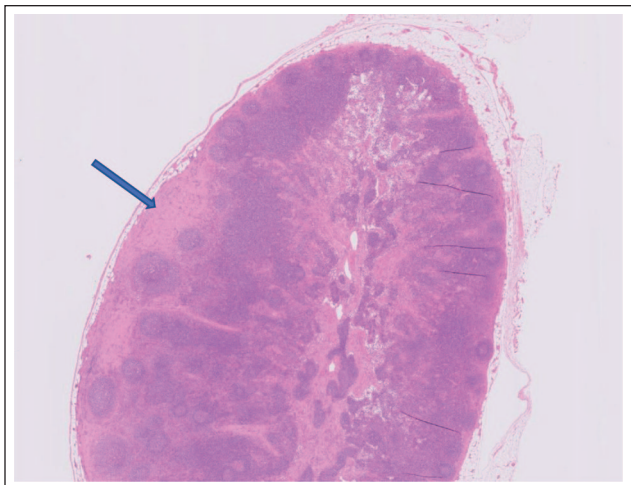


Figure 8 Lymph node. Subcapsular fibroplasia (blue arrow) and infiltration by moderate numbers of mixed inflammatory cells, including eosinophils

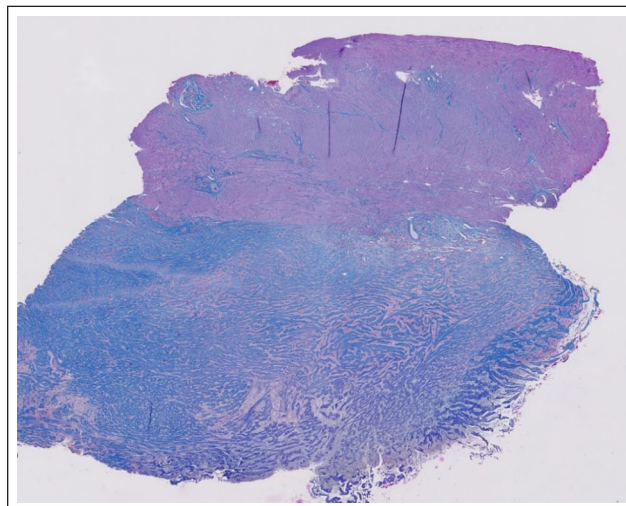


Figure 9 Section of the mass. Masson's trichrome histochemical stain demonstrating mature collagen fibres (blue)

clinician with a history of one or more of the following clinical signs: vomiting, diarrhoea, weight loss and lethargy.¹⁻⁴ Constipation (with progression to obstipation) has been described in one case.⁵ Cases of FGESF are most often characterised by the presence of an abdominal mass, frequently localised to the gastric pylorus, ileocaecocolic junction or colon.¹⁻³ The mass is usually intramural; however, a case with discrete and coalescing, variably cavitated nodules located in the mesentery has been reported as an atypical case of FGESF.⁴ Therefore, to our knowledge, this case report documents the only case consistent with FGESF of an intramural, cavitated cranial abdominal mass that has been described within the alimentary tract.

One case, presenting with constipation, describes an extramural lesion arising from the sublumbar region, consistent with FGESF.⁵ Lymph nodes are commonly enlarged, and the pancreas is the most commonly affected extra-alimentary organ.¹ Gross descriptions of the mass lesions are sparse in the literature, but they are typically pale, firm and, when sampled, the texture is gritty, reminiscent of mineralised material. Further documentation of the gross appearance of the lesions in future cases may help to distinguish them from other mass lesions such as adenocarcinoma or lymphoma. More likely differential diagnoses that must be excluded include nodular fibrosis associated with intestinal parasitism,⁶ a mural abscess secondary to foreign body penetration or pyogenic bacteria⁷, an intestinal mast cell tumour,⁸ intestinal lymphoma (particularly T-cell lymphoma associated with eosinophils), intestinal adenocarcinoma or intestinal metastases from a primary neoplasm. The histopathological assessment of these nodular lesions is fairly specific with all reports describing lesions similar or identical to those observed in this

case (mature collagen, eosinophils, fibroblasts). Densely eosinophilic (sclerotic) bands of collagen may be mistaken for osteoid but tend to resemble granulation tissue at the periphery of the lesion, helping to exclude intestinal osteosarcoma. Usually, the histopathological examination of full-thickness (if intramural) or deep biopsies of the mass, combined with the clinical presentation and haematological findings of hypereosinophilia,^{1,2} are sufficient to reach a definitive diagnosis of FGESF.

The exact aetiology of FGESF is not known, although it is not believed to be neoplastic or arise as a paraneoplastic response. Currently, it is considered to be an inflammatory lesion; however, attempts have been made to determine the exact cause. Bacteria were detected in 56% of 25 cases in one study¹ and 69% of 13 cases in a second study² by various methods, which included tissue PCR, fluorescent in situ hybridisation of formalin-fixed paraffin-embedded tissue on slides or an array of histochemical stains (Gram stain, Ziehl-Neelson). One limitation in this case is that attempts to isolate an infectious agent were not performed. In other studies, various Gram-positive and negative bacteria have been present in the lesion, including *Escherichia coli*, *Staphylococcus* species, *Clostridium* species and *Actinomyces* species. A study by Ozaki et al⁷ demonstrated methicillin-resistant *Staphylococcus* species in a number of cats with intestinal abscesses (one of the major differentials for FGESF). Careful consideration must therefore be given to the history of the animal where nosocomial infection with resistant strains of *Staphylococcus* species are a possibility. Other possible routes of infection include bite wounds or contact with an owner with an aforementioned strain.⁹ Isolation of bacteria in the lesion(s) may be difficult due to previous antimicrobial

therapy or the chronic nature of the lesion, where there has been exuberant inflammation. In one study, fungal organisms were demonstrated in a FGESF lesion; however, there were concurrent bacteria present in the lesion, thus determining the causative organism was not possible.¹⁰ In future, it may be advisable to submit fresh tissues for culture as part of investigations, and possibly consider additional molecular techniques to try to identify microbes associated with FGESF lesions.

In the past, scattered mast cells found during histopathological examination of FGESF lesions may have been suggestive of neoplasia; however, it is not currently thought that the aetiopathogenesis of these lesions is neoplastic in origin. In cats, intestinal mast cell tumours typically have a predilection for the distal small intestine or colon, and frequently metastasise,⁸ which is not consistent with the findings in the cases of FGESF documented in the literature. These mast cells and the predominance of eosinophils in tissues have given rise to the hypothesis that, like eosinophilic granuloma complex, there may be an inherited dysregulation of the inflammatory response.¹¹ Indeed, eosinophils seem to play a role in other documented conditions where there is thickening of the intestinal wall, as well as other sites.¹² In the human and veterinary literature, it is well documented that eosinophils are involved in stimulating fibrosis via the production of mediators such as transforming growth factor beta (β), interleukin (IL)-1 β , major basic protein and IL-6.^{13,14} It is also known that type one hypersensitivity reactions, driven by interactions between IgE binding of previously encountered antigens and mast cells, can lead to chronic eosinophil-dominated inflammation, mainly by the action of IL-5.¹⁵ In the light of this, possible other contributors to FGESF in cats are chronic inflammation or hypersensitivity reactions. Triggers may include dietary or environmental antigens, chronic parasitism or foreign material such as hair fragments in long haired cats, ingested during grooming.

The treatment options have not been explored in great detail and further studies are required to document the efficacy of various modalities. Within these limits it is important to discuss the possible options while considering that the prognosis is likely to be much more favourable than alimentary neoplasia. Surgical resection of discrete mass lesions is achievable and is dependent on the site of the mass. Those with masses associated with the ileocaecocolic junction or colon had longer survival times than those at the pyloric sphincter.¹ Interestingly, cats that were treated with prednisolone alone had far greater survival times than those treated with antibiotics, despite the proposed bacterial aetiology of the lesions. This has been speculated to be due to incomplete tissue penetration or the self-perpetuating nature of chronic

inflammation but requires further investigation. Corticosteroids may be useful as a treatment in non-urgent lesions as they can effectively reduce the size of the mass, and they may reduce the plethora of inflammatory stimuli suspected to be involved in the aetiopathogenesis of these lesions.¹⁶ Other immunomodulatory drugs such as ciclosporin may enhance the effects of glucocorticoids and therefore may be used in conjunction. Evidence on the therapeutic effects of dietary changes and gastro-protectants is lacking. Until further studies have elucidated the aetiology and pathogenesis of this condition a multimodal approach is advised.

Conclusions

This report of a cat with an extensive cavitated, intramural, mass lesion documents a novel presentation of FGESF in a domestic longhair cat and adds to the literature in discussing the typical clinicopathological imaging gross findings in FGESF. FGESF should be considered an increasingly common differential diagnosis for an abdominal mass in cats, including those with areas of fluid content resembling an abscess.

Conflict of interest The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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Ethical approval This work involved the use of non-experimental animals only (including owned or unowned animals and data from prospective or retrospective studies). Established internationally recognised high standards ('best practice') of individual veterinary clinical patient care were followed. Ethical approval from a committee was therefore not necessarily required.

Informed consent Informed consent (either verbal or written) was obtained from the owner or legal custodian of all animal(s) described in this work (either experimental or non-experimental animals) for the procedure(s) undertaken (either prospective or retrospective studies). For any animals or humans individually identifiable within this publication, informed consent (either verbal or written) for their use in the publication was obtained from the people involved.

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