

Recurrent feline gastrointestinal eosinophilic sclerosing fibroplasia and presumptive eosinophilic cystitis in a domestic short-haired cat: a case report

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ABSTRACT: This report describes recurrent feline gastrointestinal eosinophilic sclerosing fibroplasia (FGESF) and eosinophilic cystitis in an 8-year-old domestic short-haired cat. The cat presented with incontinence, pollakiuria, vomiting and weight loss. An abdominal mass was palpated and ultrasonography revealed severe segmental thickening of the jejunal wall with loss of layering. Complete resection of the jejunal mass was performed. The resected mass was composed of branching trabeculae of dense collagen, intermixed with bundles of large fibroblasts and inflammatory cells, which is consistent with FGESF. One year later, a new mass reoccurred in another intestinal segment. Between both FGESF occurrences, mycoplasmas were cultured from urine and fine needle aspiration of the urinary bladder wall showed numerous eosinophils only; therefore, a presumptive diagnosis of eosinophilic cystitis was made. All haematological analyses performed within the one-year period revealed eosinophilia.

Keywords: *Felis catus*; FGESF; gastrointestinal tract; haematology; urinalysis; ultrasonography; mycoplasma

Feline gastrointestinal eosinophilic sclerosing fibroplasia (FGESF) has recently been classified as a feline nodular non-neoplastic, fibroproliferative eosinophil and mast cell inflammatory response in cats (Craig et al. 2009; Weissman et al. 2013). The lesion is typically limited to the gastrointestinal tract and associated lymph nodes although in a single case a liver lesion has been reported (Weissman et al. 2013). Grossly, FGESF appears most commonly as an ulcerated mural nodule at the pyloric sphincter or ileocecolic junction (Craig et al. 2009; Sihvo et al. 2011; Weissman et al. 2013). Histopathological characteristics of FGESF are a branching trabecular pattern of dense collagen resembling osteoid, numerous eosinophils and varying numbers of mast cells (Craig et al. 2009; Weissman et al. 2013; Linton et al. 2015).

The pathogenesis of FGESF is still unclear. It is presumed that eosinophils cause the specific fibrosis of FGESF and play a key role in the patho-

genesis of FGESF (Craig et al. 2009; Suzuki et al. 2013; Weissman et al. 2013). Previously, bacterial or fungal infection has been considered to be involved in the pathogenesis of FGESF (Ozaki et al. 2003; Grau-Roma et al. 2014), but in subsequent cases, no pathogens were detected (Craig et al. 2009; Suzuki et al. 2013; Weissman et al. 2013). In the most recent study bacteria were found in nine of 13 FGESF cases (Linton et al. 2015). Several mechanisms, such as penetrating wounds from a migrating foreign body, genetic eosinophil dysregulation, herpesvirus infection, fungal infection and food hypersensitivity, have also been proposed to be involved in the pathogenesis of FGESF (Craig et al. 2009; Weissman et al. 2013; Grau-Roma et al. 2014).

Eosinophilic cystitis is a rare manifestation of urinary bladder inflammation with an unascertained aetiology that has only been reported in dogs and humans (Fuentealba and Illanes 2000; Evason and

Carr 2007; Ozaki et al. 2008; Gelberg 2010; Koyima et al. 2013; Jiang et al. 2014).

In this article, we describe the clinicopathological features of recurrent FGESF and eosinophilic cystitis, the latter diagnosed between the two occurrences of FGESF. To our knowledge, this is the first report of eosinophilic cystitis in a cat.

Case description

An 8-year-old domestic short-haired neutered female cat presented with incontinence, pollakiuria, vomiting and weight loss. An abdominal mass, distended, fluid-filled intestinal loops and dehydration were found during clinical examination. Ultrasonography revealed severe segmental thickening of the jejunal wall (25 mm) with loss of layering (Figure 1), a thickened urinary bladder wall (9 mm), distended, fluid-filled intestinal loops, and severely enlarged mesenteric lymph nodes. Thoracic and abdominal radiography was unremarkable. Urinalysis showed hyposthenuria and haematuria, and haematology and biochemistry of blood serum revealed eosinophilia, azotaemia, hyperphosphataemia and hyperglobulinaemia (Table 1, column I). The cat was treated with fluids, antibiotics enrofloxacin (5 mg/kg *s.c.*) and amoxicillin with clavulanic acid (8.75 mg/kg *s.c.*), ranitidine (2 mg/kg *s.c.*) and vitamin B 12 (cyanocobalamin, 0.5 mg). The cat's condition improved immediately, the kidney values decreased in one week and subsequently, complete resection of the

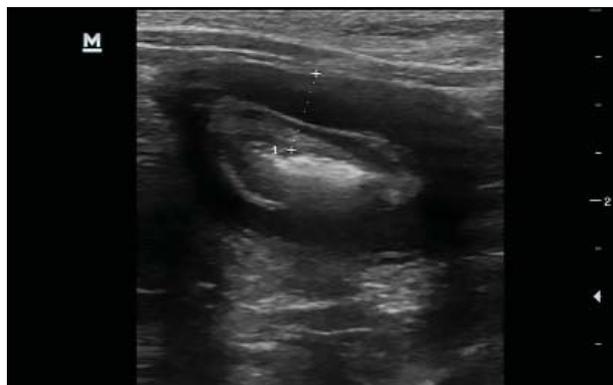


Figure 1. Ultrasonographic image of a longitudinal view of the jejunal wall. There is segmental, concentric and mildly asymmetrical severe thickening of the jejunal wall (up to 10 mm) with marked muscular thickening and partial loss of layering

jejunal mass was performed. The resected jejunum and mesenteric lymph node were sampled for histopathological examination. Samples were fixed in 10% buffered formalin and embedded in paraffin. Four- μ m-thick tissue sections were deparaffinised, stained with haematoxylin and eosin (HE), Gram, Periodic acid-Schiff (PAS) and Ziehl-Neelsen, and examined under a light microscope.

Histopathological lesions were consistent with FGESF. Histopathology revealed severe thickening of the jejunal wall due to a poorly demarcated mass, present in the submucosa and muscular layer and focally invading the mucosa. The mass was composed of branching and anastomosing trabeculae of dense collagen, intermixed with bundles of large fibroblasts and mildly infiltrated with eosinophils, plasma cells, neutrophils, lymphocytes and mast cells. The mesenteric lymph node showed marked reactive hyperplasia. The results of the special stains were negative for bacteria, including mycobacteria, and fungi.

Clinical signs resolved after surgery. Haematology and biochemistry of blood serum were unremarkable (Table 1, column II). The urinary bladder wall was thinner than at initial presentation (5 mm). Urine culture was negative.

Two months after intestinal mass resection, the cat presented with inappetence, polydipsia, incontinence and vomiting. Ultrasonographically, severely thickened urinary bladder wall (15 mm) of heterogeneous structure (Figure 2), bilateral hydronephrosis and hydroureters were identified (Figure 3). Urinalysis showed haematuria and bacteriuria. Biochemistry of blood serum showed hyperglobulinaemia and severely increased values of urea, creatinine and phosphorus (Table 1, column III).



Figure 2. Ultrasonographic image of a longitudinal view of the urinary bladder. There is severe focal extensive thickening of the bladder wall up to 15 mm with mild heterogeneous echogenicity

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Table 1. Haematology, biochemistry of blood serum and urinalysis at different times of presentation

	I	II	III	IV	V	Reference interval
Leukocytes	$10.73 \times 10^9/l$	$15.55 \times 10^9/l$	$11.27 \times 10^9/l$	$9.88 \times 10^9/l$	$29.76 \times 10^9/l \uparrow$	$5.50-19.50 \times 10^9/l$
Monocytes	$0.49 \times 10^9/l$	$0.52 \times 10^9/l$	$0.78 \times 10^9/l$	$0.93 \times 10^9/l$	$0.84 \times 10^9/l$	$0.15-1.70 \times 10^9/l$
Lymphocytes	$1.54 \times 10^9/l$	$1.41 \times 10^9/l$	$1.2 \times 10^9/l$	$1.70 \times 10^9/l$	$0.92 \times 10^9/l$	$0.40-6.80 \times 10^9/l$
Neutrophils	$6.35 \times 10^9/l$	$12.56 \times 10^9/l \uparrow$	$7.39 \times 10^9/l$	$6.41 \times 10^9/l$	$22.41 \times 10^9/l \uparrow$	$2.50-12.50 \times 10^9/l$
Eosinophils	$2.30 \times 10^9/l \uparrow$	$1.04 \times 10^9/l \uparrow$	$1.87 \times 10^9/l \uparrow$	$0.83 \times 10^9/l \uparrow$	$5.49 \times 10^9/l \uparrow$	$0.10-0.79 \times 10^9/l$
Basophils	$0.05 \times 10^9/l$	$0.02 \times 10^9/l$	$0.03 \times 10^9/l$	$0.01 \times 10^9/l$	$0.10 \times 10^9/l$	$0.00-0.10 \times 10^9/l$
Erythrocytes	$7.96 \times 10^{12}/l$	$5.54 \times 10^{12}/l$	$5.72 \times 10^{12}/l$	$5.61 \times 10^{12}/l$	$6.26 \times 10^{12}/l$	$5.00-10.00 \times 10^{12}/l$
Thrombocytes	$368 \times 10^9/l$	$540 \times 10^9/l$	$581 \times 10^9/l$	$564 \times 10^9/l$	$523 \times 10^9/l$	$175-600 \times 10^9/l$
ALP	$0.71 \mu\text{kat}/l$	$0.80 \mu\text{kat}/l$	$0.44 \mu\text{kat}/l$	$0.97 \mu\text{kat}/l$	$0.44 \mu\text{kat}/l$	$0.24-1.89 \mu\text{kat}/l$
ALT	$0.68 \mu\text{kat}/l$	$0.95 \mu\text{kat}/l$	$1.05 \mu\text{kat}/l$	$0.91 \mu\text{kat}/l$	$0.37 \mu\text{kat}/l$	$0.2-2.16 \mu\text{kat}/l$
Glucose	$7.15 \text{ mmol}/l$	$7.25 \text{ mmol}/l$	$7.23 \text{ mmol}/l$	$7.19 \text{ mmol}/l$	$6.90 \text{ mmol}/l$	$3.94-8.83 \text{ mmol}/l$
Urea	$> 46.4 \text{ mmol}/l \uparrow$	$7 \text{ mmol}/l$	$> 48.5 \text{ mmol}/l \uparrow$	$18.7 \text{ mmol}/l \uparrow$	$9.3 \text{ mmol}/l$	$5.7-12.9 \text{ mmol}/l$
Creatinine	$> 1202 \mu\text{mol}/l \uparrow$	$139 \mu\text{mol}/l$	$1188 \mu\text{mol}/l \uparrow$	$358 \mu\text{mol}/l \uparrow$	$131 \mu\text{mol}/l$	$71-212 \mu\text{mol}/l$
Albumin	$28 \text{ g}/l$	$32 \text{ g}/l$	$31 \text{ g}/l$	$24 \text{ g}/l$	$25 \text{ g}/l$	$23-39 \text{ g}/l$
Globulin	$55 \text{ g}/l \uparrow$	$51 \text{ g}/l \uparrow$	$57 \text{ g}/l \uparrow$	$52 \text{ g}/l \uparrow$	$51 \text{ g}/l \uparrow$	$28-51 \text{ g}/l$
Total protein	$81 \text{ g}/l$	$83 \text{ g}/l$	$83 \text{ g}/l$	$80 \text{ g}/l$	$76 \text{ g}/l$	$57-89 \text{ g}/l$
Phosphorous	$4.61 \text{ mmol}/l \uparrow$	$2.32 \text{ mmol}/l$	$> 5.19 \text{ mmol}/l \uparrow$	$1.57 \text{ mmol}/l$	$1.07 \text{ mmol}/l$	$1.00-2.42 \text{ mmol}/l$
Urine						
-specific gravity	1.019	1.028	1.012	1.014	1.034	
-stick analysis	pH6, prot1+, B4+	pH6, prot1+	pH6, prot2+, B4+	pH6, prot2+, B4+	pH7, prot1+	
-sediment	erythr4+	unremarkable	erythr4+, leuk3+, bact3+	erythr4+, leuk3+	unremarkable	

1+, 2+, 3+, 4+ = semi-quantitative; ALP = alkaline phosphatase; ALT = alanine aminotransferase; B = blood; bact = bacteria; erythr = erythrocytes; I = 1st presentation; II = two weeks after 1st presentation (jejunal mass resection); III = two months after jejunal mass resection (hydro-nephrosis); IV = two and a half months after jejunal mass resection (eosinophilic cystitis); V = one year after 1st presentation (recurrence of intestinal mass); leuk = leukocytes; prot = proteins
 \uparrow Increased

Despite treatment with amoxicillin with clavulanic acid (8.75 mg/kg/24h *s.c.*), ranitidine (2 mg/kg/12h *s.c.*) and fluids the condition did not improve for the first three days, and kidney values increased even further. At the beginning of the second week, the kidney values decreased but urinalysis still showed haematuria and isosthenuria (Table 1, column IV) and the urinary tract did not improve ultrasonographically. Fine needle aspiration of the urinary bladder wall showed numerous eosinophils only; therefore, a presumptive diagnosis of eosinophilic cystitis was made. Urine culture of the sample collected by cystocentesis revealed mycoplasmas and therefore the cat was treated with enrofloxacin for three weeks. After one week of therapy the upper urinary tract resolved ultrasonographically; however, the bladder wall had thickened further (up to 18 mm). Urinalysis revealed hyposthenuria and

haematuria. The therapy was discontinued after four days. Urine culture was negative one week after cessation of the therapy. The owner rejected therapy with corticosteroids. The thickened urinary bladder wall and haematuria resolved two months following the end of antimicrobial therapy.

One year after initial presentation the cat presented with anorexia and vomiting again. Clinical examination revealed fever (40.4 °C) and a mass in the caudal abdomen. Ultrasonographically, there was a large heterogenous mass in the distal jejunal segment, just before the ileum (Figure 4). The jejunal wall was diffusely thickened and the mesenteric lymph nodes were enlarged. Haematology revealed leukocytosis with neutrophilia and eosinophilia. Biochemistry of blood serum was within reference ranges (Table 1, column V). The cat was treated with amoxicillin with clavu-

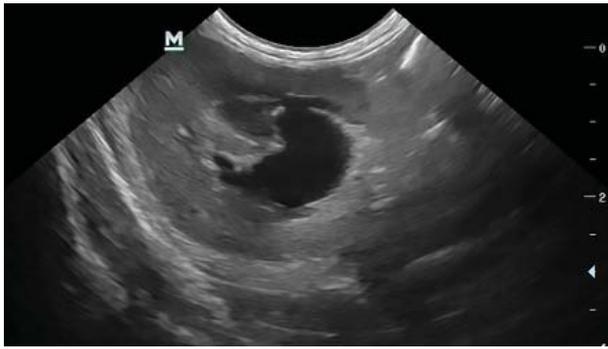


Figure 3. Ultrasonographic image of a kidney with hydronephrosis. The transverse view of the kidney shows severe dilation of the renal pelvis (up to 12 mm)

lanic acid (8.75 mg/kg/24h *s.c.*) and enrofloxacin (5 mg/kg/24h *s.c.*), ranitidine (2 mg/kg/12h *s.c.*) and fluids. The mass was surgically removed and sent for histopathological examination. Grossly, a segmental transmural mass, measuring 3.5 cm in diameter, with severe luminal stenosis was found (Figure 5). Histopathological examination of the mass revealed results, which were similar to the previously resected mass, and FGESF was diagnosed (Figure 6). The results of special stains were again negative. The cat's condition deteriorated after three days due to dehiscence of the intestinal wall sutures and septic peritonitis. The owner opted for euthanasia, but declined autopsy.

DISCUSSION AND CONCLUSIONS

FGESF is a rare, recently described inflammatory lesion in cats. The disease has characteristic micro-



Figure 4. Ultrasonographic image of a recurrent jejunal wall mass. On transverse view there is segmental, severe concentric wall thickening (up to 17 mm), mainly involving the muscular layer with almost complete loss of layering



Figure 5. Cross section of the resected recurrent jejunal mass

scopic lesions (Craig et al. 2009; Sihvo et al. 2011; Suzuki et al. 2013; Weissman et al. 2013; Munday et al. 2014; Linton et al. 2015), which were also confirmed in both resected intestinal masses in this case, thus confirming the diagnosis of FGESF.

Recurrence of the FGESF was here diagnosed one year after initial presentation, which is much later than in two previously reported cases, where FGESF recurred five and seven months after surgery (Weissman et al. 2013). This indicates that the lesion may reoccur on another intestinal segment up to one year after the first occurrence. In

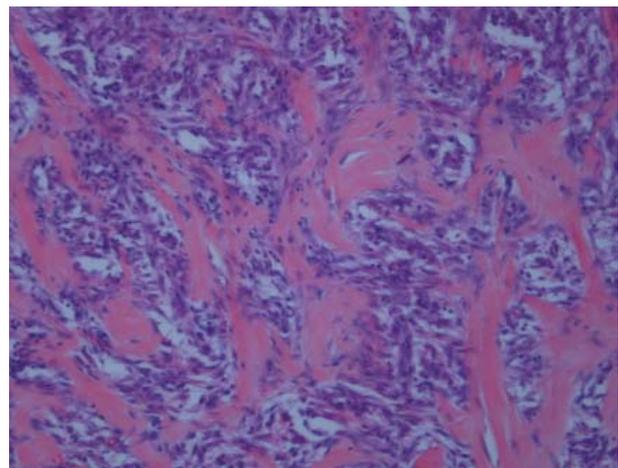


Figure 6. Characteristic histopathological lesions of FGESF. The mass was composed of branching and anastomosing trabeculae of dense collagen, intermixed with bundles of large fibroblasts and mildly infiltrated with eosinophils, plasma cells, neutrophils, lymphocytes and mast cells. HE. Final magnification $\times 100$

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our case, the survival time after first diagnosis of FGESF was much longer than the mean survival time of cats treated with surgery alone in other studies (Craig et al. 2009; Weissman et al. 2013). The prognosis for FGESF is guarded because of the wide range of survival times, varying from less than one week (Craig et al. 2009), to more than 43 months after surgery (Weissman et al. 2013). FGESF seems to respond better to corticosteroid administration than to antimicrobial treatment despite the presence of intralesional bacteria (Craig et al. 2009; Sihvo et al. 2011). It has been reported that cats undergoing surgery only had a significantly shorter survival time than cats treated with surgery and corticosteroids (Craig et al. 2009). In our case, the owner rejected corticosteroids due to recurrent urinary tract infections.

The pathogenesis of FGESF is still not clearly understood. Several aetiological factors, such as bacterial infection, penetrating wounds from a migrating foreign body, herpesvirus infection, fungal infection, food hypersensitivity and genetic eosinophil dysregulation have been proposed as possible triggers for the eosinophilic inflammatory response (Craig et al. 2009; Weissman et al. 2013; Grau-Roma et al. 2014). However, immunohistochemistry was negative for coronavirus and herpesvirus – type 1 in 12 FGESF cases (Linton et al. 2015). It has generally been suggested that cats, which develop eosinophilic reactions, have inherited eosinophil dysregulation, leading to an inappropriate eosinophilic inflammatory response to a variety of stimuli (Bloom 2006). However, eosinophilia was noted only in five of 10 FGESF cases (Linton et al. 2015). We believe that the cat in our case may have had a form of eosinophilic disorder, since in a period of one year the cat presented with two intestinal masses, diagnosed as FGESF, as well as eosinophilic cystitis and eosinophilia in all haematological analyses, which were performed in that period.

Reports of eosinophilic cystitis in dogs and humans are very rare (Fuentealba and Illanes 2000; Evason and Carr 2007; Ozaki et al. 2008; Gelberg 2010; Koyima et al. 2013; Jiang et al. 2014) and to the best of our knowledge the condition has yet to be described in cats. In this case a biopsy of the urinary bladder wall would be necessary to confirm the presumptive diagnosis of eosinophilic cystitis, since eosinophils in FNAB could also represent peripheral eosinophilia and/or eosinophil infiltration in the bladder wall as part of another inflammatory

process. The cause of eosinophilic cystitis remains elusive (Evason and Carr 2007). In humans, eosinophilic cystitis is described as a rare manifestation of hypereosinophilic syndrome, and in most patients, has a benign course with spontaneous resolution (Kojima et al. 2013; Jiang et al. 2014). In dogs, meanwhile, it is associated with urolithiasis and infections (Fuentealba and Illanes 2000; Evason and Carr 2007); in some cases, the exact cause cannot be determined (Ozaki et al. 2008). Hypereosinophilic syndrome, characterised by hypereosinophilia, infiltration of eosinophils into multiple tissues and subsequent organ damage, which is apparently not secondary to another disease process, has also been described in cats (McEwen et al. 1985; Takeuchi et al. 2008). In our case, the eosinophil values were much lower than in the above-cited cases and no clinicopathological findings suggestive of the involvement of parenchymatous organs were found.

Mycoplasmas were isolated from the urine in this case. Infection with urinary mycoplasmas has been very rarely described in the literature, and to our knowledge only in dogs (Ulgen et al. 2006; L'Abée-Lund et al. 2013). Moreover, according to the literature these bacteria have been ruled out as the possible etiological agent in feline lower urinary tract disease (Brown et al. 1991; Senior and Brown 1996; Abou et al. 2006). Despite these data, we believe that mycoplasmas cannot be completely excluded from playing a role in the aetiology of cystitis, since in this case they were isolated from the urine collected by cystocentesis, and because treatment with enrofloxacin for three weeks resulted in clinical improvement.

FGESF is a rare and unique disease with a characteristic histological appearance. The occurrence of a second FGESF lesion in another intestinal segment is possible even one year after resection of the first lesion. In cats with FGESF the possibility of other eosinophilic lesions such as eosinophilic cystitis should also be taken into consideration, even though a unifying pathogenesis has not been established. The case described here also demonstrates that eosinophilic cystitis should be on the list of differential diagnoses in cats with clinical signs of cystitis and diffusely thickened bladder wall.

Although it has been reported that cats treated with surgery and corticosteroids live longer than cats treated only with surgery (Craig et al. 2009; Linton et al. 2015), future studies are needed to determine optimal therapeutic recommendations.

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