

Feline Gastrointestinal Eosinophilic Sclerosing Fibroplasia in a Bengal Cat

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Abstract : A 2-year-old neutered male Bengal cat presented with a 6-month history of weight loss and chronic vomiting. An abdominal ultrasound revealed increased thickness of the pylorus and ascending duodenum with concurrent enlargement of the mesenteric lymph nodes. Histologically, branching and anastomosing trabecular pattern of dense collagen was the characteristic feature, and large populations of spindle cells were also observed. These large spindle-shaped cells were positive for smooth muscle actin and vimentin on immunohistochemical examination. Based on these findings, the cat was diagnosed with feline gastrointestinal eosinophilic sclerosing fibroplasia (FGESF), and immunosuppressive therapy was initiated immediately. Unfortunately, the cat's condition deteriorated despite treatment; the cat died 56 days after initiation of therapy. This is the first report of FGESF in South Korea.

Key words : FGESF, gastric mass, eosinophilic inflammation, cat.

Introduction

Feline gastrointestinal eosinophilic sclerosing fibroplasia (FGESF) is a newly recognized clinical disease characterized by the presence of eosinophilic masses, usually limited to the gastrointestinal (GI) tract and associated lymph nodes (4). Although the pathogenesis of FGESF is largely unknown, several mechanisms have been proposed, including an eosinophilic inflammatory response to certain antigens such as bacteria or parasites, which results in a breach of the intestinal mucosa (4).

As this disease has been recognized only recently, FGESF is not commonly considered as a major differential diagnosis in cats with intra-abdominal masses. However, as FGESF is a clinically distinctive disease in cats and has a better prognosis than other common GI neoplastic diseases such as lymphoma or adenocarcinoma (2,9), early diagnosis with appropriate treatment increases the likelihood of a successful outcome. Although FGESF has been reported in a few countries including the USA, Japan, Europe, and New Zealand (4,5,8,10,11), this is the first case report confirming the diagnosis of FGESF in South Korea. This report also emphasizes the clinical importance of FGESF as a major differential diagnosis of gastrointestinal mass lesions in cats.

Case

A 2-year-old neutered male Bengal cat presented with a 6-month history of decreased appetite, weight loss, and chronic vomiting. At presentation, the patient had a thin body condition (body condition score 3/9); mild dehydration (5%) and

hyperthermia (39.6°C) were noted on physical examination. A complete blood count showed mild lymphocytosis ($7.52 \times 10^3/\mu\text{l}$, reference interval (RI) $0.92\text{-}6.88 \times 10^3/\mu\text{l}$) with a normal white blood cell count ($12.08 \times 10^3/\mu\text{l}$, RI $2.87\text{-}17.02 \times 10^3/\mu\text{l}$).

Thoracic radiographs were within normal limits. An abdominal ultrasound revealed increased thickness of the pyloric wall (9.3 mm; RI pyloric wall < 3 mm) with loss of normal wall layering (Fig 1-A). Increased wall thickness of the ascending duodenum, with a prominent muscular and submucosal layer, was also noted (Fig 1-B). Concurrent enlargement of the adjacent mesenteric lymph node was observed (12.4×8.2 mm).

A biopsy specimen of the pyloric antrum region was obtained, and fine-needle aspiration of a mesenteric lymph node was performed on exploratory laparotomy. Histologic examination of the pylorus revealed a transmurally expanded muscular wall with a branching and anastomosing trabecular pattern of dense collagen separated by a dense cellular population of large spindle-shaped cells with high mitotic figure; the findings were consistent with the characteristic features of FGESF. Multiple numbers of inflammatory cells, including eosinophils, lymphocytes, and macrophages, were observed on hematoxylin and eosin staining (Fig 2). The anastomosing sclerotic trabeculae stained uniformly blue with Masson's trichrome (Fig 3), confirming the evidence of a collagenous matrix. The large spindle-shaped cells were positive for smooth muscle actin and vimentin, which is a characteristic immunohistochemical feature of spindle cells (Fig 4). The cytologic features of the pyloric region were similar to those observed in adjacent lymph nodes, which implies the involvement of the regional lymph nodes. There was no evidence of intralesional bacteria or fungal infection. These findings were consistent with the characteristic features of FGESF and confirmed the diagnosis of FGESF in the patient.

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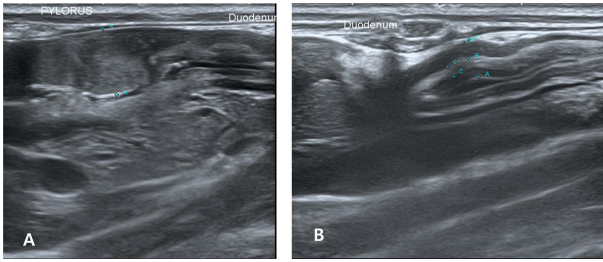


Fig 1. Ultrasound images of the gastric pylorus. (A) Ultrasonographic image showing increased pyloric wall thickness (9.3 mm; RI pyloric wall < 3 mm) with loss of normal wall layering. (B) Ultrasonographic image showing increased wall thickness (4.5 mm; RI: < 3 mm) with a prominent muscular and submucosal layer.

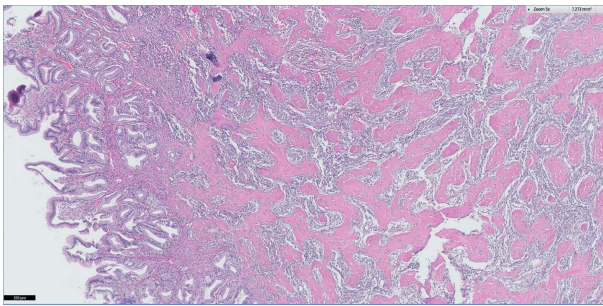


Fig 2. Photomicrograph of the stomach after hematoxylin and eosin staining. A transmurally expanded muscular wall with branching and anastomosing trabeculae of dense collagen was noted. The trabeculae of dense collagen are separated by a densely cellular population of large spindle-shaped cells with high mitotic figures.

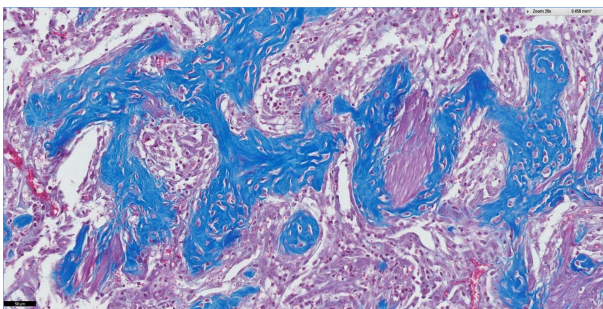


Fig 3. Photomicrograph of the stomach stained with Masson's trichrome stain. The anastomosing sclerotic trabeculae stained uniformly blue with Masson's trichrome, indicating an increased amount of fibrous connective tissue.

Treatment was initiated with a combination of prednisolone (2 mg/kg/day) and chlorambucil (20 mg/m² every 2 weeks); antibiotics, including metronidazole and marbofloxacin, were also administered. The clinical signs, including chronic vomiting, began to improve within 2 weeks of treatment, and the pyloric wall thickness was decreased on subsequent abdominal ultrasounds. However, 6 weeks after initiation of the treatment, the vomiting recurred with notably decreased activity. Increased thickness of the pyloric wall was observed on

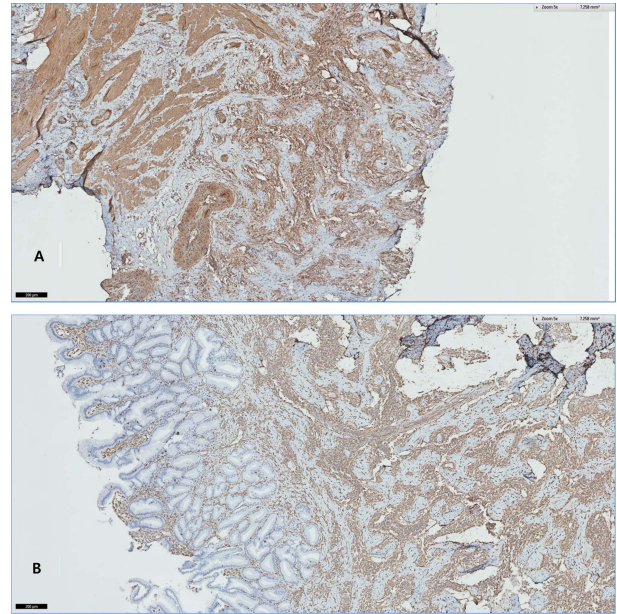


Fig 4. Immunohistochemical photomicrography of the stomach visualized using anti-smooth muscle actin and anti-vimentin antibodies. (A) The smooth muscle area was diffusely immunoreactive to smooth muscle actin. (B) The mesenchymal area was immunoreactive to vimentin.

abdominal ultrasound, and the patient died 56 days after initiation of treatment.

Discussion

FGESF is a recently described eosinophilic inflammatory condition of the gastrointestinal tract, histologically characterized by a branching trabecular pattern of dense sclerotic collagen separated by large populations of spindle cells (11). Due to its gross resemblance to other common GI neoplasias and histological features similar to osteosarcoma or mast cell tumors, it is often mistaken for a GI neoplastic disease. However, as FGESF is a clinically distinctive disease entity, with characteristic features that confirm the diagnosis, it should always be considered one of the primary differential diagnoses for mass-like lesions in the gastrointestinal tract.

The cat in this case was 2 years old, which is less than the mean age of FGESF cats (8.8 ± 4.4 years) reported in a previous study, although a few cases have occurred in younger animals (4). Although age is not a definite criterion used to differentiate FGESF from other GI neoplasias in cats, the relatively young age of this patient, compared to the reported median ages of those with either lymphoma (median 12 years) or intestinal adenocarcinoma (median 11 years) (1,4,7), makes the diagnosis of neoplasia less likely.

Although eosinophilia is one of the characteristic findings of FGESF and was reported in over half of the cat with FGESF in a previous report (4), peripheral eosinophilia was not observed in the current patient. Despite the relatively high occurrences of marked eosinophilia in reported FGESF cats, this should not be used as a criterion to diagnose FGESF, as peripheral eosinophilia has been reported as a conse-

quence of other intestinal neoplasias, including lymphoma (3).

An abdominal ultrasound of this patient revealed increased thickness of the pylorus region with the loss of normal wall layering, which was consistent with the most common site of FGESF lesions reported previously (4).

Histopathologic examination revealed a transmurally expanded muscular wall with branching and an anastomosing trabecular pattern of dense collagen, separated by a densely cellular population of large spindle-shaped cells with high mitotic figure. Although the branching trabecular pattern of dense collagen is very characteristic of the histopathologic features of FGESF, this feature should be interpreted cautiously, since this pattern may also resemble osteoid, often misleading the clinician to make a diagnosis of osteosarcoma (4). However, the anastomosing sclerotic trabeculae stained uniformly blue with Masson's trichrome, and the immunohistochemical features of the spindle cells confirmed the diagnosis of FGESF in this patient.

Current evidence suggests that FGESF could be treated effectively with immunosuppressive therapy and/or surgical excision (4,11). One retrospective study of 25 FGESF cases revealed that corticosteroids significantly prolonged the survival time compared to antibiotic treatments (4). In this study, 17 cats were followed for up to 2 years after diagnosis. This is a better prognosis than the reported survival times of either intestinal adenocarcinoma or GI lymphoma (2,9). Additional immunosuppressive agents such as ciclosporin A, chlorambucil, hydroxyurea, or lomustine can be administered to enhance the effect of the glucocorticoids. However, the clinical advantage of this combination protocol is unknown (6). In this case, due to the severity of clinical signs at presentation, a combination of prednisolone and chlorambucil was initiated immediately. Improvement in clinical signs and reduced pyloric wall thickness were observed within 2 weeks of treatment. Unfortunately, despite the transient improvement of clinical signs and ultrasonographic findings, patient's condition deteriorated, and he died 6 weeks after the initiation of treatment; the owner declined a postmortem examination.

In conclusion, we report FGESF in a 2-year-old Bengal cat, primarily localized to the pyloric regions. To the authors' knowledge, this is the first case report of FGESF in South Korea. Although the patient failed to survive for an extended period of time, FGESF is known to have a more favorable prognosis than other intestinal neoplastic diseases (8). This is important, as earlier diagnosis and timely treatment would

improve the prognosis of cats with FGESF. Furthermore, this case emphasizes the fact that FGESF should be considered as a primary differential diagnosis, especially in younger cats with palpable intestinal masses. Further studies are necessary to determine the optimal therapeutic regimens and prognosis, as well as the pathogenesis of this unique feline disease.

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