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*Case report*

# FELINE GASTRIC ADENOCARCINOMA – A CLINICAL CASE

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**Summary**

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Gastric adenocarcinomas are encountered exceptionally infrequently in cats. Some studies have claimed a predisposition in Siameses, while others affirm no relation to the breed. In veterinary literature, only few cases of feline gastric adenocarcinoma (FGA) are reported, which accounts for about 1% of all feline gastrointestinal adenocarcinomas. The present case report describes a clinical case of FGA. The clinical signs, blood morphology and biochemistry, ultrasound and histopathological findings are presented. This is the first report of FGA from Bulgaria.

**Key words**: cat, clinical case, gastric adenocarcinoma

Although gastric carcinomas constitute a considerable part of neoplasms in this body region, they are relatively rarely encountered in domestic animals (Sullivan *et al.*, 1987; Meuten, 2017; Hardas *et al.*, 2021). Unlike animals, these tumours are diagnosed in 5.7% of all human cancer patients and rank second as cause of cancer deaths (Bray *et al.*, 2018). Gastric adenocarcinomas are exceptionally rare in cats, which is not the case with dogs (Araújo, 2022). Some studies have reported a predisposition in Siameses, while others affirm no relation to the breed (Cribb, 1988; Denis *et al.*, 2006; Dickens *et al.*, 2006). In veterinary literature, only few cases of feline gastric adenocarcinoma (FGA) are reported, which accounts for about 1% of all feline gastrointestinal adenocarcinomas (Turk *et al.*, 1981; Sullivan *et al.*, 1987; Rossmeisl *et al.*, 2002; Dennis *et al.*, 2006; Esmans *et al.*, 2014). The aim of the presented case report was to describe clinical and morphological findings in a case of feline gastric carcinoma. This is the first report of FGA from Bulgaria.

*Case history*

A 8-year-old intact female calico cat, 2.2 kg body weight, was referred to the Small Animal Clinic, Faculty of Veterinary Medicine, Trakia University – Stara Zagora. According to owners, the animal had

decreased appetite and vomiting from several weeks, and during the last few days its condition has been significantly worsened. The cat was treated with medications, but without improvement.

The clinical examination of the patient demonstrated cachexia. A firm mass was palpated in the cranial abdominal part. Blood was sampled from the cephalic vein in EDTA- and heparin-anticoagulated tubes. Complete blood counts were determined with automated haematological analyzer Exigo EOS-VET (Boule Medical AB, Sweden). Blood plasma biochemistry profile was analysed on automated biochemical analyzer Mindray BS-120 (China). The results from blood analyses are presented in Table 1. All parameters were within the respective reference ranges except for the slightly increased blood glucose concentration. This was a manifestation of stress hyperglycaemia, commonly seen in cats.

Ultrasonography was performed in dorsal recumbency with Mindray DC-6 VET ultrasounc equipped with 5.0 - 8.0 MHz convex transducer. The examination revealed a mass associated to the stomach and located dorsally and at the right side of the organ. The formation was of mixed echogenicity, with distinct borders and irregular contours (Fig. 1)

Diagnostic cranial medial laparotomy was done. The anaesthesia protocol included premedication by intramuscular injection of of 7.5 µg/kg medetomidine hydrochloride (Dorbene vet®, 1 mg/mL, Syva, Spain) with 7.5 mg/kg ketamine hydrochloride (Anaket®, 100 mg/ml, Richter Pharma, Austria). Endotracheal intubation was performed after induction of general anaesthesia with propofol (Propofol Fresenius®, Fresenius Kabi GmbH Germany) at a dose of 1 mg/kg intravenously to effect. After intubation, general inhalational anaesthesia was maintained with isoflurane (Forane®, Abbott Labora-

**Table 1.** Results from blood morphological and biochemical analysis.

| Parameters | Result | Reference range* |
|---|---|---|
| Haemoglobin, g/L | 109 | 98–154 |
| Red blood cells, T/L | 7.6 | 5.0–10.0 |
| Haematocrit, % | 38.3 | 30–45 |
| White blood cells, G/L | 17.2 | 5.5–19.5 |
| Platelets, G/L | 432 | 300–800 |
| Glucose, mmol/L | 8.4 | 3.3–6.7 |
| Urea, mmol/L | 10.5 | 6.8–12.1 |
| Creatinine, µmol/L | 137.8 | 80–194 |
| Total protein, g/L | 62.7 | 60–79 |
| Albumin, g/L | 29.4. | 28–39 |
| ASAT, U/L | 42 | 7–38 |
| ALAT, U/L | 58 | 25–97 |
| Alkaline phosphatase, U/L | 43 | 0–45 |
| Ca, mmol/L | 2.6 | 2.2–2.9 |
| P, mmol/L | 1.7 | 1–2.0 |

*Merck Veterinary Manual (https://www.msdvetmanual.com/special-subjects/reference-guides/serum-biochemical-reference-ranges).

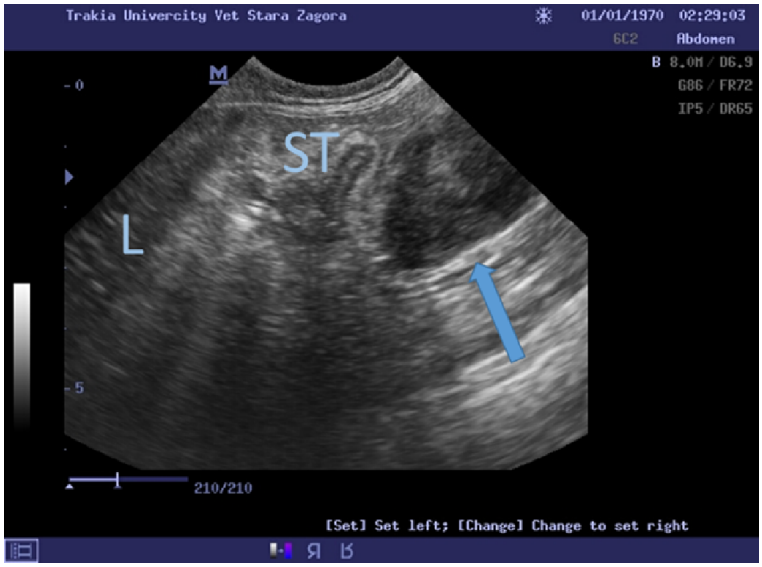


**Fig. 1.** Ultrasonogram of the stomach, longitudinal view. A large mass attached to the stomach, located dorsally and at the right side of the stomach is seen (arrow). The mass is of mixed echogenicity, sharp borders and irregular contours. L – liver, ST – stomach.

tories Limited, United Kingdom) in 100% $O_2$. Fluid therapy (Ringer lactate; Ringer Braun, B. Braun Melsungen AG, Germany) was applied at 10 mL/kg/h.

Intraoperatively, a substantial diffuse thickening of the gastric wall was found out. The neoplastic mass was non-encapsulated and affected the omentum (Fig. 2).

The formation was dense, with granulated surface and non-elastic structure, with haemorrhages in some areas. Lymph nodes around the pancreas were enlarged and of firm consistency. Due to the size of the neoplastic formation, it was not removed. Biopsy of the mass was performed at the boundary between intact and neoplastic tissue with regard to ultimate diagnosis. The specimen was fixed in 10% neutral formalin and send to the histopathological laboratory. Specimens were embedded in paraffin, 4 µm sections were cut on a microtome and stained with haematoxylin-eosin. The cross-sections were transferred on adhesion slides (Su-

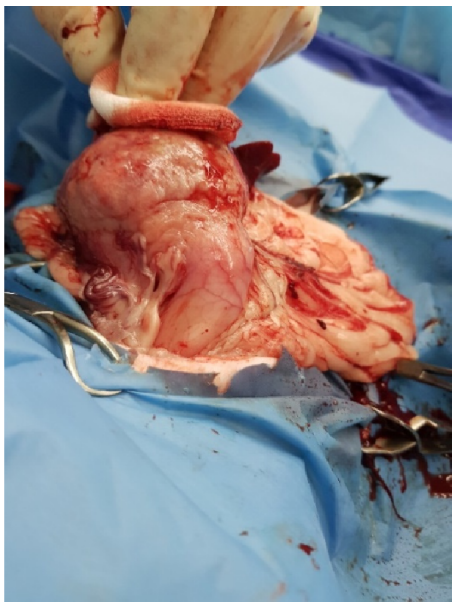

**Fig. 2**. Gross appearance of the neoplastic formation.

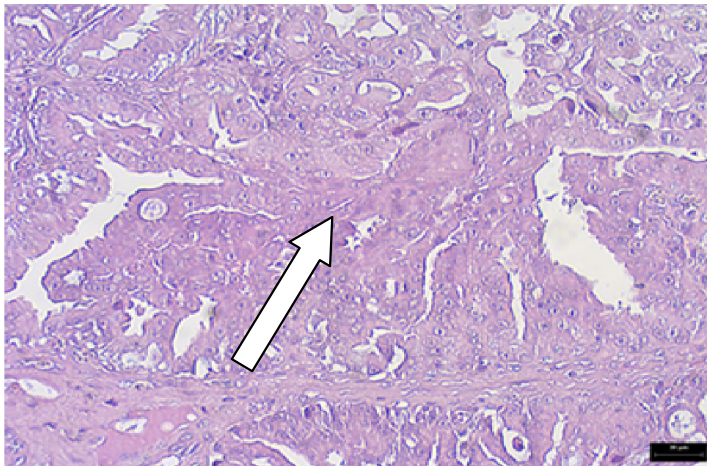

**Fig. 3.** Feline gastric carcinoma. Neoplastic cell proliferation, demonstrating signs of atypia and pleomorphism (arrow). Haematoxylin/eosin staining, bar = 30 µm.

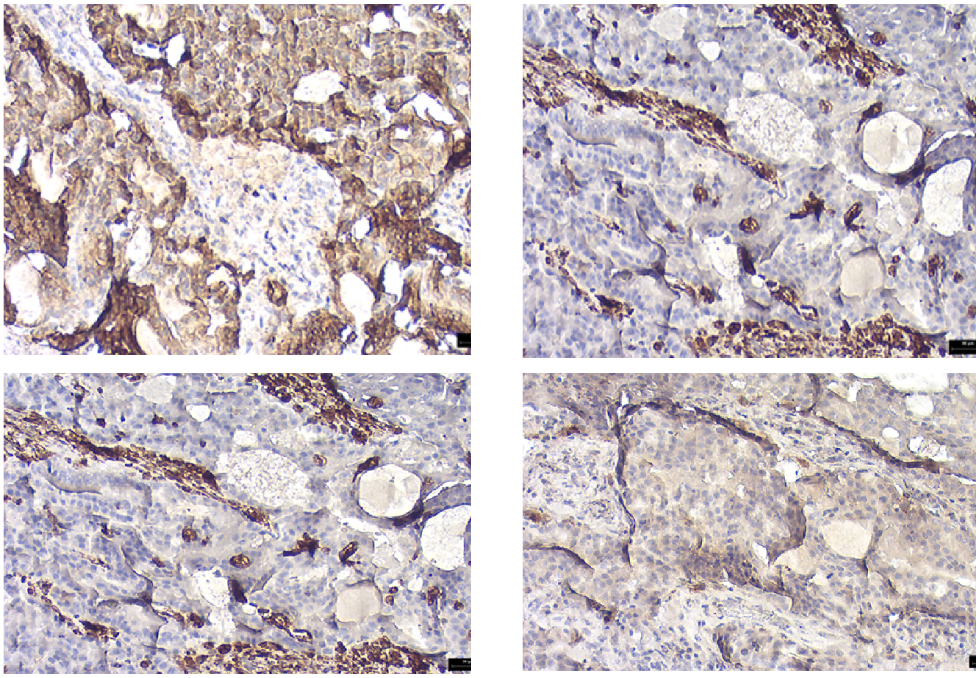

**Fig. 4.** Feline gastric carcinoma - immunohistochemistry. Cytokeratin (+) neoplastic cells (arrow, left upper), Desmin (–) neoplastic cells (arrow, right upper), Vimentin (–) neoplastic cells (arrow, left lower), and Actin (–) neoplastic cells (arrow, right lower); bar = 30 µm.

perfrost® Plus, Thermo Fisher Scientific, Inc.) and placed in a thermostat at 60 °C for 24 hours. For confirmation of the formation' genesis, the Monoclonal Mouse

Anti-Human Cytokeratin, Clone AE1/AE3 (Code № IS053, Dako, Denmark - ready to use), Monoclonal Mouse Anti-Human Desmin (D33) (Code № 1526, Dako, Denmark – ready to use), Monoclonal Mouse Anti-Human Vimentin, Clone V9 (Code № 1521, Dako, Denmark - ready to use) and Monoclonal Mouse Anti-Human Smooth Muscle Actin, Clone 1A4 (Code № M0851, Dako, Denmark – dilution 1:100 antibodies were used. Paraffin cross sections were submitted to immunohistochemistry as described by Gulubova (1998), based on the method of De Vos *et al.* (1985).

Histopathologically, a neoplastic cell proliferation with signs of atypia and pleomorphism was observed. In some areas, cells were densely arranged while in others they produced tubulo-papillary formations (Fig. 3). Cells' nuclei were large, irregularly shaped and with convex nucleoli. In the surrounding healthy tissue, inflammatory response with predominating lymphocytes was observed. The stroma was scarce. Immunohistochemically, neoplastic cells were Cytokeratin (+) Desmin (–), Vimentin (–) and Actin (–) (Fig. 4).

The commonest clinical signs associated with gastric tumours are vomiting, partial anorexia and weight loss (Hart *et al.*, 2018; Barker *et al.*, 2019; Pires *et al.*, 2022). This was confirmed in our patient, although it should be noted that the signs were non-specific and could be observed in different diseases. In this cat, due to advanced disease, the tumour formation was not surgically excised. The animal has died two weeks after the laparotomy. According to literature data, the average life span in cats with untreated gastric adenocarcinoma is from several weeks to several months depending on tumour size, presence or not of metastases and differentiation of neoplastic cells (Crib, 1988). In men and rodents, atrophic gastritis and intestinal metaplasia are considered precancerous states leading to stomach cancer (Sipponen & Marshal; 2000; Peek & Blaser, 2002; Nambiar *et al.*, 2005). In the veterinary literature, only few cases have reported the ultrasound findings of feline gastric carcinoma (Rivers *et al.*, 1997). According to the authors, this tumour may be mistaken for gastric lymphoma. With this imaging modality, lymphoma is accompanied with thinned gastric wall, impossibility for visualisation of the different layers and proliferation in tunica muscularis (Daniaux *et al.*, 2014; Griffin, 2019). Routine histopathological examination in most cases is sufficient for the ultimate diagnosis. If this is not the case, histochemical and immunohistochemical analyses are done. Tubular formations were reported to have a better prognosis compared to the other histological types, especially if no metastasis is present at the time of diagnosis (Crib, 1988).

In conclusion, this is the first report from Bulgaria describing feline gastric adenocarcinoma. Clinical and morphological findings were similar to those seen in gastric adenocarcinomas in other animal species. The differential diagnosis of FGA should include also gastric lesions and gastric lymphoma.

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