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Gastroduodenal Perforation in a Cat: Clinical Findings, Diagnosis, and Treatment Management

Abstract

Gastrointestinal perforations, although rarely reported in cats, are life-threatening conditions that can result in septic peritonitis and death if left untreated. Contributing factors include long-term or high-dose use of non-steroidal anti-inflammatory drugs (NSAIDs), gastrointestinal neoplasms, inflammation, and foreign bodies. This report describes the clinical, diagnostic, and therapeutic management of a feline case of gastroduodenal perforation complicated by septic peritonitis. A 5-year-old male domestic shorthair cat was referred with anuria and diagnosed with urolithiasis. Following unsuccessful medical therapy, the cat underwent cystotomy. Postoperatively, meloxicam and a cephalosporin antibiotic were administered. By the third day post-surgery, the cat exhibited anorexia and systemic deterioration. Radiographs showed decreased abdominal detail and a ground-glass appearance. Ultrasonography revealed free abdominal fluid, intact urinary bladder, and fluid-filled stomach. Bilateral acute nephritis was noted. Abdominocentesis confirmed septic peritonitis cytologically. During exploratory laparotomy, brown free fluid consistent with gastric content was identified in the abdomen. Extensive peritonitis, circulatory compromise, and adhesions were present. Due to poor prognosis, intraoperative euthanasia was performed. While gastroduodenal perforation in cats is rarely documented, this case suggests a possible link between NSAID use, renal dysfunction, and gastrointestinal rupture. The cat had no prior gastrointestinal symptoms and had undergone surgery for an unrelated condition. This highlights the importance of cautious NSAID use in patients with compromised renal function and suggests the need for further studies to better understand risk factors and outcomes associated with feline gastroduodenal perforation.

Keywords: Feline, non-steroids, renal dysfunction, rupture, septic peritonitis

Bir Kedide Gastroduodenal Perforasyon: Klinik Bulgular, Tanı ve Sağaltım Yönetimi

Öz

Kedilerde nadir olarak bildirilen gastrointestinal perforasyonlar, hayvanın yaşamını tehdit eden ve sağaltılmadığında septik peritonitis ve ölümle sonuçlanabilen durumlardır. Etiyolojisinde anti-inflamatuar ilaçların uzun süreli ve yüksek dozda kullanımları, intestinal neoplaziler, gastrointestinal yangılar, yabancı cisimler gibi pek çok faktör yer alır. Bu çalışmada amaç, sistotomi yapılan bir kedide postoperatif dönemde şekillenen gastroduodenal perforasyon ve buna bağlı olduğu düşünülen septik peritonitisin klinik, tanı ve sağaltım süreçlerinin değerlendirilmesidir. Cerrahi Kliniğine anüri şikayeti ile getirilen 5 yaşlı, erkek, tekir bir kediye ürolitiazis tanısı konularak, medikal sağaltıma yanıt alınamaması üzerine cerrahi müdahale olarak sistotomi uygulandı. Postoperatif meloksikam ve sefalosporin başlanan kedide üçüncü gün iştahsızlık ve genel durumunda bozulma gözlenen hastada radyografik

muayenede abdominal organlardaki detay kaybı ve buzlu cam görünümü dikkati çekti. Ultrasonografik muayenede abdomende serbest sıvı tespit edilirken, idrar kesesinin bütünlüğünü koruduğu, midenin ise yoğun bir sıvı içerik ile dolduğu görüldü. Bilateral akut nefritis dikkati çekti. Abdominosentez ile alınan sıvının sitolojik incelemesinde septik peritonitis tespit edildi. Laparotomi ile abdomene girildiğinde kahverengi serbest sıvının varlığı görüldü. Bu sıvının gastrik içerik olduğu tespit edildi. Şiddetli peritonitis, organ genelinde dolaşım bozuklukları ve şiddetli adezyonlar değerlendirilerek, prognozun kötü olduğu sonucuna varıldı ve hastaya intraoperatif ötanazi işlemi gerçekleştirildi. Kedilerde gastrik perforasyonların etiyolojisi, prevalansı ve predispoze faktörler ile ilgili oldukça az sayıda çalışma bulunmaktadır. Yapılan çalışma ile sindirim sistemi dışında bir şikayetle opere edilen bir kedide şekillenen mide rupturuna ilişkin etiyoloji ve sağaltım süreçleri farklı açılardan değerlendirilerek tartışılmıştır.

Anahtar kelimeler: Böbrek disfonksiyonu, felin, gastritis, non-steroidler, ruptur

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Introduction

Gastrointestinal perforations, if left untreated, can lead to severe complications such as organ adhesions and septic peritonitis, potentially resulting in mortality (Bernardin et al., 2015). The etiology of these perforations includes gastrointestinal foreign bodies, inflammatory reactions, tissue necrosis, and trauma. In addition, non-steroidal anti-inflammatory drugs (NSAIDs) can damage the gastric epithelium either directly or indirectly by reducing epithelial cell regeneration, which consequently leads to gastrointestinal ulceration (Saunders and Tobias, 2003). The most clinically significant adverse effects include upper gastrointestinal peptic ulcers, bleeding, and perforation, any of which may result in mortality. Another albeit rare, cause of gastric ulcers is renal dysfunction (McLeland et al., 2014). Gastritis and gastric ulcers are interrelated inflammatory processes that significantly compromise the quality of life.

Gastritis is defined as an inflammatory reaction of varying severity in the gastric mucosa, resulting from factors such as foreign bodies, improper diet, viral or bacterial infections, systemic illnesses, and medication administration (Kollannur et al., 2024). During the inflammatory phase, continuous exposure of the gastric epithelium and the underlying mucosal layer to various inflammatory mediators exacerbates the condition. These mediators not only directly affect the gastric mucosal cells, but also increase mucosal permeability and blood flow, further deteriorating the gastric environment (Tolbert and Gould, 2020). Chronic gastritis may eventually lead to ulcer formation. Gastric ulcers are defined as mucosal defects extending into the muscular layer, resulting from the disruption of the mucosal barrier and compromise of gastric defense mechanisms dueto sustained inflammation (Liptak et al., 2003). When these ulcers perforate, the leakage of gastric contents into the abdominal cavity may precipitate septic peritonitis. Gastric ulceration is more commonly observed in dogs, whereas it remains relatively rare in cats (Bernardin et al., 2015; Mate et al., 2020). The increased mortality risk associated with gastrointestinal ruptures is primarily due to the development of septic peritonitis. Although gastroduodenal perforation and subsequent septic peritonitis are the most commonly reported complications of gastroduodenal ulceration, they are infrequently encountered in the veterinary literature (Cariou et al., 2009).

This study aims to evaluate the clinical, diagnostic, and therapeutic management of gastroduodenal perforation and the associated septic peritonitis observed in a cat during the postoperative period following a cystotomy.

Case Description

This case involves a 5-year-old male domestic shorthair cat, presented to Ankara University Faculty of Veterinary Medicine Animal Hospital Surgery Clinic with a complaint of anuria. Clinical, radiological (Figure 1), and ultrasonographic examinations confirmed a diagnosis of urolithiasis, with stones identified in the renal pelvis, bladder, and urethra.

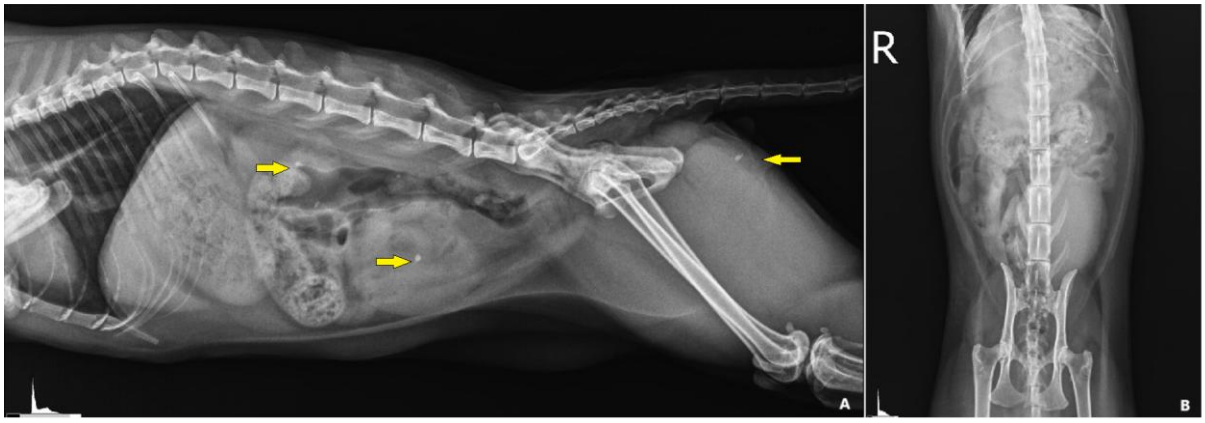

Figure 1. (A) Lateral (L/L) view. (B) Ventrodorsal (V/D) view. Radiographs reveal the presence of radiopaque uroliths located in the urinary bladder, urethra, and renal regions, consistent with urolithiasis involving both the lower and upper urinary tract (arrow).

The animal's history revealed that, over a 14-day period in the internal medicine department, he had been treated with Vetexpert Urinovet Cat Dilution (q12h) and Piyeloseptyl (Biofarma; 50 mg q12h). Additionally, a urinary diet (Hill's c/d) was provided, and meloxicam (Meloxicam, 0.2 mg/kg, SC, q24h, Bavet, Türkiye) was administered for 5 days. After further evaluation, enrofloxacin (Baytril-K, 5 mg/kg q24h, SC, Bayer, Türkiye) was initiated. Based on these clinical findings, a cystotomy was indicated, and the cat was referred to the surgical clinic for the procedure.

General anesthesia was induced with propofol (Propofol-PF 1%, 6 mg/kg, IV, Polifarma, Türkiye) and maintained with isoflurane (Isoflurane, 100% oxygen, Adeka İlaç, Türkiye). Preoperative analgesia was provided with butorphanol (Butomidor, 0.2 mg/kg, SC, Richter Pharma, Austria). Additionally, a continuous infusion of butorphanol (4 µg/kg/h) and ketamine (10 µg/kg/h, IM, Richter Pharma, Austria) was prepared in 100 mL of 0.9% NaCl (Polifarma İlaç, Türkiye) solution and administered at a fixed rate of 5 mL/kg/h. Intraoperative cephalosporin (Eqizolin 25 mg/kg, IV, q12h, Tümekip İlaç, Türkiye) was administered, and fluid therapy was maintained with balanced electrolyte solutions throughout the surgery. A ventral laparotomy was performed to access the abdominal cavity, where the bladder wall was observed to be thickened and hyperemic. Following the cystotomy, both normograde and retrograde urethral catheterization were performed, and the bladder was irrigated with sterile physiological saline. After complete removal of the stones from the bladder and urethra, the bladder was closed in separate mucosal and seromuscular layers using monofilament absorbable sutures (3/0 Polydioxanone, Boz, Türkiye). The abdominal muscles, subcutaneous tissue, and skin were subsequently closed with appropriate suture materials. Postoperatively, meloxicam (Meloxicam, 0.2 mg/kg, SC, q24h, Bavet, Türkiye) was administered for 3 days, and amoxicillin (Amoklavin-BID, 25 mg/kg, PO, Deva, Türkiye) was given for 7 days. Despite regular monitoring, on the 3rd postoperative day the cat exhibited signs of lethargy, weakness, and loss of appetite, although urination remained normal; defecation was absent. Laboratory analyses revealed elevated blood urea nitrogen (BUN: 129.3 mg/dL; reference range: 17.6-32.8 mg/dL) and creatinine (CRE: 5.40 mg/dL; reference range: 0.8-1.8 mg/dL). On the 5th postoperative day, a direct abdominal radiograph demonstrated a loss of organ detail with a "ground-glass" appearance detected (Figure 2).

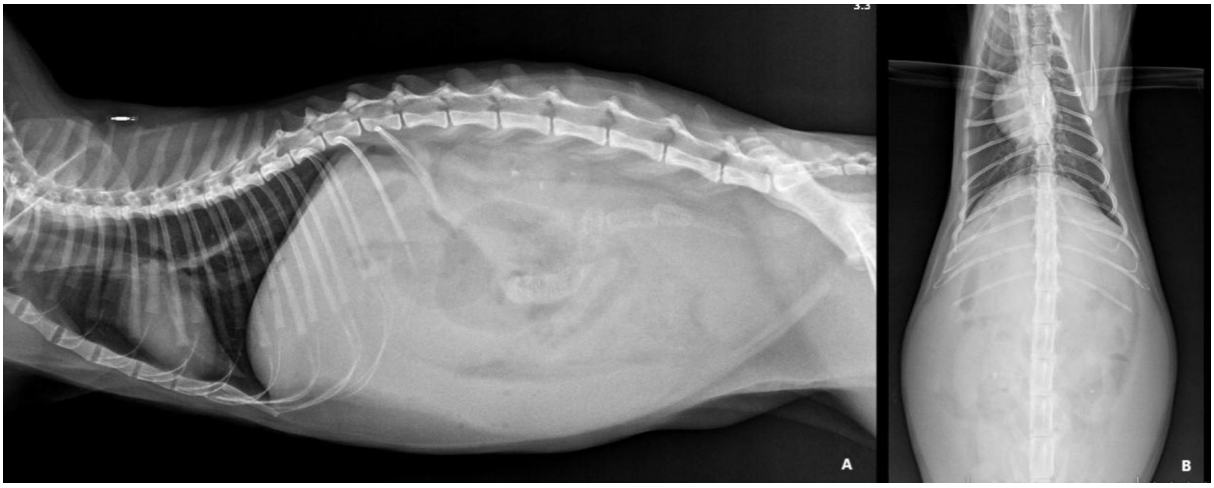

Figure 2. Follow-up abdominal radiographic evaluation performed five days post-cystotomy. Right lateral radiographic projection (A). Ventrodorsal radiographic projection. The abdominal radiographs demonstrate a diffuse loss of serosal detail and a characteristic "ground-glass" appearance, consistent with the presence of free peritoneal fluid (B).

Ultrasonographic examination (Figure 3) revealed free fluid with a hypoechoic appearance within the abdominal cavity, consistent with peritoneal effusion. The presence of hyperechoic mesenteric fat further raised suspicion for peritonitis. While the bladder remained intact, the stomach was filled with dense fluid content (Figure 3A), and the colon wall was diffusely thickened with an inflammatory appearance (Figure 3B). Additionally, chronic kidney failure was diagnosed in the left kidney, with compensatory hypertrophy of the right kidney. Abdominocentesis was performed, and cytological examination of the aspirated fluid showed numerous degenerated neutrophils and macrophages, along with lymphocytes, thereby confirming a diagnosis of septic peritonitis.

Due to the diagnosis of septic peritonitis, an exploratory laparotomy was performed using the same anesthesia protocol as for the cystotomy. Upon entering the abdominal cavity, dark brown free fluid was encountered, which upon complete aspiration was identified as being of gastrointestinal origin. The abdominal cavity was thoroughly irrigated with sterile warm saline. Detailed examination of the gastrointestinal organs revealed that, while the serosa of the small intestine maintained its integrity, it appeared markedly pale. Hypertrophy of the mesenteric lymph nodes was also noted. Importantly, the increased fluid on the gastric serosa suggested a possible perforation; further inspection identified a 1 cm rupture in the gastroduodenal area (Figure 4). The perforated site was repaired using 3/0 monofilament absorbable sutures (Polydioxanone, Boz, Türkiye). However, due to the severity of the peritonitis, widespread circulatory disturbances, and extensive adhesions, the prognosis was deemed poor, and intraoperative euthanasia was performed.

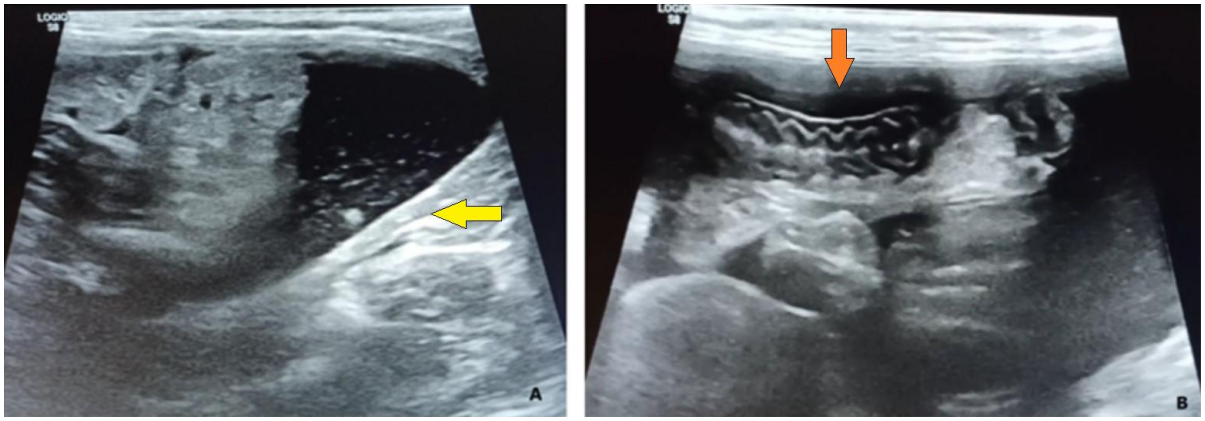

Figure 3. Abdominal ultrasonographic findings. Ultrasonographic image demonstrating a markedly distended stomach containing anechoic fluid with minimal echogenic particulate material, consistent with gastric fluid retention (arrow) (A). Ultrasonographic image showing a segment of the colon with diffuse mural thickening and a hypoechoic appearance of the colonic wall layers. The presence of anechoic free fluid in the adjacent peritoneal space may indicate an underlying inflammatory process (arrow) (B).

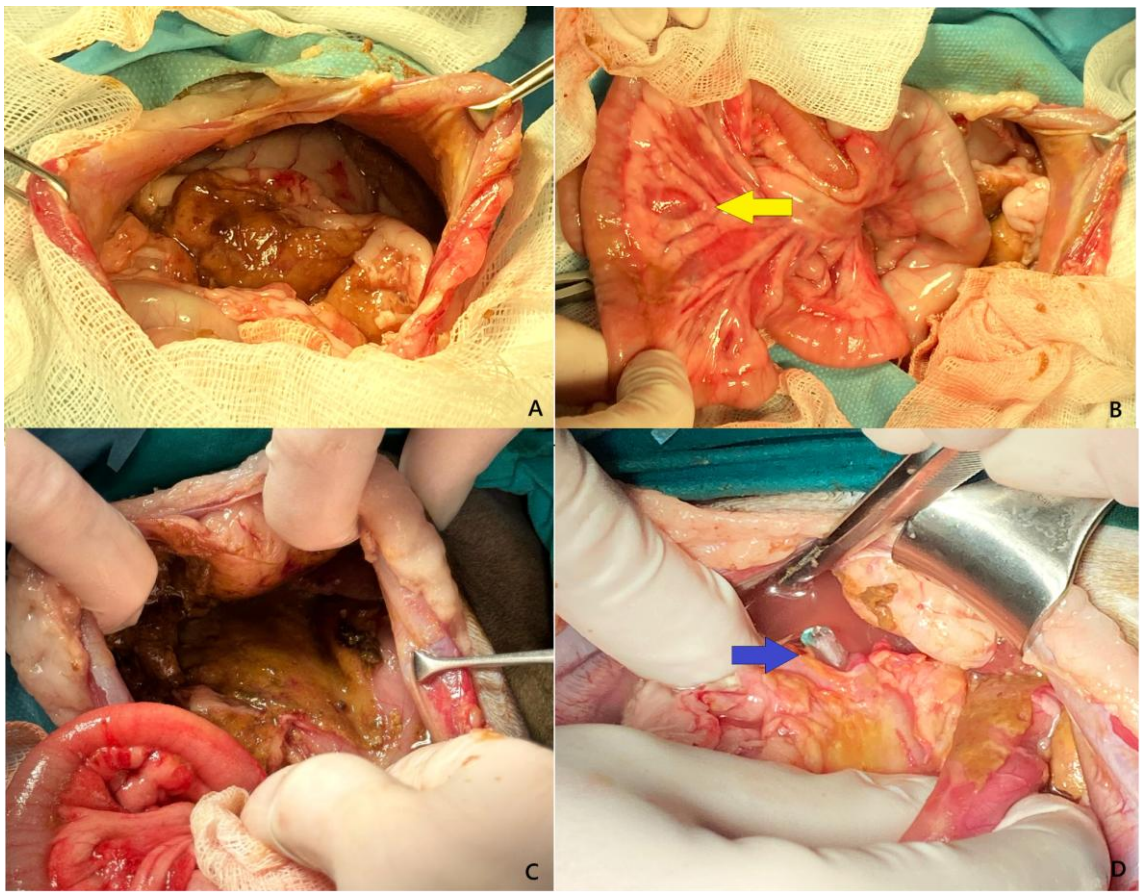

Figure 4. Intraoperative evaluation of the abdominal cavity. Free gastric content is observed within the abdominal cavity, consistent with gastric leakage (A–C). Marked enlargement of the mesenteric lymph nodes is evident, suggestive of a reactive or inflammatory response. The intestinal wall appears pale yet remains structurally intact (yellow arrow) (B). A focal rupture at the gastroduodenal junction is identified during surgical exploration, facilitated by the placement of an orogastric tube (blue arrow), confirming gastrointestinal perforation (D).

Discussion and Conclusion

One of the most significant complications arising from gastrointestinal foreign bodies in veterinary medicine is septic peritonitis, a condition that can lead to severe consequences and even be life-threatening. Although septic peritonitis is reported to be rare in cats (Bernardin et al., 2015), gastrointestinal perforations may occur due to a variety of factors, including foreign bodies, severe inflammatory reactions, tissue necrosis, trauma, chronic gastritis, improper dietary practices, anti-inflammatory drugs, intestinal neoplasms, acute kidney failure, and liver failure. In instances where no definitive underlying cause can be identified, the condition is deemed idiopathic (Cariou et al., 2009; Tolbert and Gould, 2020; Barandun et al., 2021; Weston et al., 2022).

Non-steroidal anti-inflammatory drugs (NSAIDs) are among the most critical clinical contributors to gastrointestinal perforations in pets, primarily affecting the upper gastrointestinal tract. Numerous studies have demonstrated that NSAIDs increase the risk of peptic ulcer complications by three to five times. By inhibiting cyclooxygenase -an enzyme essential for both protective and deleterious prostaglandin synthesis- NSAIDs reduce overall prostaglandin production. This inhibition diminishes the mucosal defenses against gastric acid, predisposing the gastric epithelium to damage (Dobberstein et al., 2022; Drini et al., 2017; Eskafian et al., 2017; Tolbert and Gould, 2020; Wun et al., 2023). Studies have reported that in both cats and dogs, NSAID-related ulcers and perforations most commonly occur in the stomach and duodenum (Bernabe et. al., 2021). In the present case, meloxicam was administered during both preoperative and postoperative periods. Although the animal did not display overt clinical signs during NSAID therapy, the rupture's location in the gastroduodenal area – consistent with the literature- raises suspicion regarding its contributory role.

Another potential factor in the development of gastrointestinal ulcers in small animals is renal dysfunction. The kidneys and the gastrointestinal system are interconnected, with kidneys playing a key role in gastrin metabolism (Yavuz and Ankaralı, 2021). Gastrin, secreted by G cells in the gastric antrum, stimulates parietal cells to secrete gastric acid and is normally excreted by the kidneys. In renal failure, diminished kidney function leads to hypergastrinemia and increased gastric acidity, thereby elevating the risk of peptic ulcer formation (McLeland et al., 2014; Tolbert and Gould, 2020). Although the clinical manifestations of renal failure are often nonspecific, alterations in blood urea levels are commonly observed in both acute and chronic conditions (Lew and Radhakrishnan, 2020). In cats, chronic kidney failure is frequently associated with gastrointestinal signs related to uremic gastropathy (McLeland et al., 2014). While uremic ulceration is more typically observed on the tongue and palate, recent studies have occasionally reported erosive-ulcerative gastritis in the context of uremia. However, the pathogenesis and incidence of uremic gastropathy in cats remain unclear (Nelson and Couto, 2019; Tolbert and Gould, 2020).

In the present case, laboratory findings revealed markedly elevated kidney parameters, a result that was consistent with ultrasonographic evidence of acute kidney failure. Although it cannot be definitively determined whether NSAID use directly contributed to the deterioration of renal function, this finding supports the multifactorial etiology underlying the gastric rupture.

Clinical signs and laboratory tests in cats with gastric rupture or perforation are generally nonspecific, and imaging findings may become conclusive only in advanced cases. This diagnostic challenge can pose a life-threatening risk during early stages of the disease, thereby underscoring the critical importance of vigilant monitoring and prompt intervention in suspected gastroduodenal ruptures (Bernabe et. al., 2021). In our case, regular postoperative monitoring with radiography and ultrasonography allowed for the early detection of complications. Nevertheless, due to severe circulatory disturbances in the abdominal organs, the rapid progression of septic peritonitis, and extensive tissue damage, the decision was ultimately made to perform intraoperative euthanasia.

In conclusion, both preoperative and postoperative evaluations of the cat revealed two significant predisposing factors for gastric ulceration and perforation: NSAID administration and kidney failure. Although the precise cause of the renal dysfunction remains unclear, it is likely that NSAID use accelerated the pathological process. This case underscores the importance of judicious NSAID use and close postoperative monitoring, serving as a valuable example for clinical colleagues regarding the potential multi-systemic impacts in similar cases.

Peer-review: External, Independent.

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This work is original.

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Concept: AU; ***Conceptualization:*** DU,AU,OOS; ***Literature Search:*** DU; ***Data Collection:*** DU; ***Data Processing:*** DU; ***Analysis:*** DU,AU; ***Writing – original draft:*** DU,AU,AY; ***Writing – review & editing:*** DU,AU,AY,OOS.

3. Ethics approval:
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The authors declare no competing interests.

6. GenAI Usage Statement:
No GenAI tools were used at any stage of the study.

7. Sustainable Development Goals:

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