Improve Veterinary Education

Gastric Ulcerations in Dogs and Cats

Introduction

This lecture addresses gastric ulcerations in dogs and cats, with particular emphasis on the clinical indications for instituting gastroprotectant therapy. The discussion reviews contemporary treatment options and considers a major consensus statement that supports the rational, evidence-based use of antacids and other gastrointestinal protectants in small animal practice.

Aetiology and Predisposing Conditions

Non-steroidal anti-inflammatory drug (NSAID) toxicosis represents one of the most frequent causes of gastric ulceration in companion animals. A common scenario involves ingestion of palatable, flavoured formulations, such as carprofen tablets, in which dogs access and consume an entire supply, resulting in overdose and mucosal injury. Ulceration may also occur at prescribed therapeutic doses in both dogs and cats. Glucocorticoid-associated ulceration is most often reported when steroids are combined with NSAIDs; however, ulcerative lesions may also develop with high-dose steroid therapy alone. Endoscopic studies evaluating patients receiving high-dose corticosteroids have identified mucosal erosions. Clinically, lesions may be encountered incidentally during endoscopic removal of gastric foreign bodies, particularly in patients receiving prednisone for oncological or internal medicine indications. Although concurrent foreign material within the stomach can confound interpretation, patients receiving prednisone that exhibit compatible clinical signs should be considered at meaningful risk of gastric ulceration.

Gastrointestinal neoplasia is another important cause. Ulceration may arise directly from ulcerated gastric tumours, with gastric adenocarcinomas being highly invasive and frequently ulcerative. Ulceration is also associated with mast cell tumours through degranulation, which can promote gastrointestinal mucosal injury and ulcer formation. Gastric foreign bodies constitute a common and often readily reversible cause; following removal of the offending object, associated ulceration frequently resolves rapidly, sometimes with minimal pharmacological support.

A complex and clinically significant subset involves advanced liver disease with portal hypertension. Although the pathophysiology remains poorly understood, impaired hepatic metabolism and the haemodynamic consequences of portal hypertension are associated with severe gastrointestinal ulcerations. Importantly, this risk appears concentrated in those patients with portal hypertension rather than being a universal feature of hepatic disease in dogs and cats. Severe systemic illness and shock states, including hypovolaemic shock, can also precipitate mucosal injury through compromised perfusion and damage to gastrointestinal mucosal barriers. Addisonian dogs, owing to deficient endogenous steroid production, are likewise predisposed and should be considered at increased risk of ulceration.

Differential Diagnosis and the Importance of Excluding Coagulopathies

When an animal presents with haematemesis or melaena, exclusion of coagulopathy is essential, as haemostatic disorders can closely mimic primary gastrointestinal ulceration. Severe thrombocytopenia may manifest with petechiae and ecchymoses, emphasising the importance of meticulous physical examination. Petechiae are often most readily identified on the oral mucosa, particularly the inner lip, and may also be evident on the pinnae, ventral abdomen, and inguinal regions where hair is sparse. Some coagulopathies present with striking bruising. A young cat with haemophilia may display extensive ecchymosis, which can become apparent following clipping for abdominal ultrasonography in the investigation of haematemesis; diagnosis is confirmed via coagulation testing.

Toxin exposures must also be considered. Amanita mushroom intoxication is associated with severe hepatic failure. Anticoagulant rodenticide exposure can lead to markedly prolonged PT and PTT, and bleeding may present primarily as haematemesis or bloody diarrhoea. These considerations represent critical rule-outs in any presumptive diagnosis of gastric ulceration, particularly in patients presenting with significant gastrointestinal haemorrhage.

Clinical Presentation and Physical Examination Findings

Clinical signs vary with the duration and severity of ulceration. Haematemesis may present as fresh liquid blood or as “coffee-ground” material, reflecting partially digested blood with granular particulate content. Vomitus may also contain brown blood. In some cases, such as gastric adenocarcinoma, patients may vomit frank clots. Melaena classically indicates upper gastrointestinal bleeding originating from the stomach or proximal small intestine, with the dark, tarry appearance resulting from digestion of blood during intestinal transit. Additional

signs may include ptyalism and abdominal pain, particularly in patients with more severe ulcerative disease.

On physical examination, pallor or light pink mucous membranes may indicate anaemia. Rectal examination is essential to evaluate for melaena. Assessment of abdominal discomfort should be incorporated routinely. Because coagulopathies can masquerade as ulcerative disease, the examination must also include directed assessment for petechiae and bruising, particularly in the oral cavity, pinnae, ventrum, and inguinal regions.

Laboratory Findings and Diagnostic Testing

Haematological findings depend strongly on the chronicity of haemorrhage. In acute bleeding, haematocrit may remain within reference limits or only mildly decreased, as regeneration requires time to become evident. If bleeding has persisted for approximately five to seven days, a regenerative anaemia consistent with haemorrhage may develop. In more chronic cases, a microcytic, hypochromic anaemia compatible with iron deficiency may be observed. A normal haematocrit can still occur in some cases. Mild neutrophilia is commonly associated with ulceration. On serum biochemistry, severe or prolonged bleeding can produce hypoalbuminaemia, and an increased blood urea nitrogen concentration is also commonly identified. Thrombocytosis may develop, with markedly increased platelet counts in some patients. In cases of substantial haematemesis or where haemostatic disease is suspected, PT and PTT should be performed.

An illustrative case involved a dog, Caly, with chronic anaemia associated with severe mast cell tumours. Initial haematocrit was 25% with no evidence of regeneration. Because the disease had been present for a prolonged period, iron deficiency was suspected, and the dog received iron supplementation alongside omeprazole administered twice daily. On re-evaluation one week later, haematocrit had increased to 32% and a pronounced reticulocytosis was present; thrombocytosis with increased platelet numbers was also evident. This case reinforces the capacity of aggressive or metastatic mast cell tumours to cause clinically significant ulcerative disease within the gastrointestinal tract.

History, Imaging, and the Role of Endoscopy

Clinical history is highly informative, particularly regarding exposure to NSAIDs, steroids, and potential toxins. Diagnostic imaging must be interpreted within its limitations. Radiography is

generally unhelpful for identifying gastric or intestinal ulcers because ulceration is primarily a mucosal disorder and typically does not produce radiographically visible changes.

Ultrasonography can be valuable and may detect ulcerative lesions in approximately 60% of cases; however, a normal ultrasound does not exclude ulceration, as up to 40% of affected patients may have no ultrasonographic abnormalities. Ultrasonographic features can include marked segmental thickening, gas-related shadowing, apparent mucosal irregularity, and mesenteric changes suggestive of focal inflammation. Ultrasound can also support decisions regarding the need for surgical intervention or further diagnostics. Endoscopy remains the gold standard for definitive diagnosis through direct visualisation of erosions or ulcers. Nevertheless, endoscopy is not invariably required, as empirical medical therapy may be initiated when the clinical suspicion is strong.

Therapeutic Approach and Indications for Surgical Intervention

Treatment depends on the underlying cause and the severity of mucosal injury. In the case of gastric foreign bodies that have caused ulceration through mechanical irritation, removal of the object often allows the ulcer to heal rapidly, sometimes even without pharmacological intervention, because the primary inciting factor has been eliminated. In contrast, deep ulcerative lesions, lesions with imaging findings concerning for perforation, and bleeding neoplasms may necessitate surgical resection. An exception may occur with lymphoma, for which chemotherapy may be the preferred approach; such cases are comparatively uncommon.

Medical management is centred on acid suppression and mucosal protection, selected according to aetiology and clinical context. Proton pump inhibitors (PPIs), including omeprazole and pantoprazole, are superior to famotidine for clinically important acid suppression. Misoprostol is indicated specifically for NSAID-induced ulceration and is not recommended as a routine addition for all ulcerative disease. Sucralfate can provide effective local mucosal protection but does not significantly alter gastric pH and is therefore best considered adjunctive rather than primary therapy.

H2-Receptor Antagonism with Famotidine

Famotidine reduces gastric acid secretion by blocking H2 receptors on parietal cells. Oral famotidine is available in 10 mg tablets and can be obtained without prescription; however, the oral formulation is comparatively less potent than PPIs. A key limitation is tachyphylaxis, in

which efficacy diminishes rapidly. Data evaluating intragastric pH demonstrate that on the first day of administration, a high proportion of dogs achieve meaningful acid suppression, with pH maintained above 4 for a substantial proportion of time. By the second day, this effect declines, and by approximately day 12, there is little difference between famotidine and placebo. For this reason, oral famotidine may be acceptable when short-term acid suppression is required, such as during a brief period following foreign body irritation, but it is not an optimal first-line option for most ulcerative conditions requiring sustained control.

Famotidine can be highly effective when administered as a continuous rate infusion in severe cases, such as acute, substantial gastrointestinal bleeding. In this setting, a protocol of 1 mg/kg intravenous bolus followed by 8 mg/kg/day as a continuous rate infusion may provide clinically useful acid suppression.

Proton Pump Inhibition: Omeprazole and Pantoprazole

Proton pump inhibitors provide profound gastric acid suppression by inhibiting the gastric proton pump and can sustain suppression over a prolonged period. They represent the preferred therapy for gastroduodenal ulcers and for severe oesophagitis when robust and durable acid control is required. Omeprazole is commonly administered orally as a 20 mg tablet, whereas pantoprazole is typically used as an injectable formulation. Other agents, such as esomeprazole and lansoprazole, are available but are used less frequently in routine veterinary practice.

Appropriate dosing and timing are important. Administration ideally occurs 30 to 45 minutes before a meal to maximise absorption, and twice-daily dosing is more effective than once-daily dosing. Even enteric-coated tablets formulated for human use should generally be administered twice daily in dogs and cats to achieve optimal acid suppression. Comparative studies using telemetric pH monitoring capsules demonstrate that omeprazole produces a substantial increase in the percentage of time intragastric pH is maintained above clinically relevant thresholds compared with placebo and famotidine. In dogs, both intact 20 mg tablets and compounded formulations, including paste preparations, have shown comparable efficacy in maintaining intragastric pH above 3 and above 4. Similar findings have been demonstrated in cats, including efficacy with fractionated tablets and paste formulations. These studies support the conclusion that omeprazole is superior to famotidine for acid suppression in both species.

Despite efficacy, PPIs should be used judiciously. Some dogs develop diarrhoea during omeprazole therapy, plausibly due to substantial elevation of gastric pH altering digestion and

potentially promoting bacterial overgrowth. Consequently, PPIs are most appropriate when ulceration is suspected or confirmed, rather than being prescribed routinely in the absence of an acid-mediated condition.

Sucralfate as Adjunctive Mucosal Protection

Sucralfate acts as a local protectant by binding to exposed ulcerated mucosa, forming a protective barrier. Clinically, it can be particularly beneficial in painful oesophagitis, where patients may show rapid symptomatic improvement when an active lesion is present. Sucralfate has important practical limitations, notably its capacity to bind other medications, which necessitates careful timing of administration. It should be given one to two hours separated from meals and other drugs. The intact tablet form is less effective because it does not dissolve adequately to coat mucosa; tablets should therefore be crushed, dissolved in water, and administered as a suspension. A commercial suspension preparation, containing 1 g in 10 mL, can also be used, but must be shaken thoroughly prior to dosing because it is a true suspension and phase separation may otherwise occur. Sucralfate should be regarded as an adjunct, as it does not modify gastric pH sufficiently to function as sole therapy, but it remains valuable in selected cases, particularly oesophagitis.

Misoprostol for NSAID-Induced Ulceration

Misoprostol is a prostaglandin E1 analogue indicated for patients with NSAID-associated ulceration, whether due to overdose or ulceration developing during prescribed therapy. Diarrhoea is a recognised adverse effect and should be anticipated. Misoprostol is also an abortifacient; therefore, appropriate precautions are required for people handling the medication, and it should not be administered to pregnant animals.

Rational Use of Gastrointestinal Protectants: ACVIM Consensus Guidance

The lecture's therapeutic recommendations align with an ACVIM consensus statement published in the Journal of Veterinary Internal Medicine, developed by a panel of experts in response to widespread overprescribing of gastroprotectants. The consensus emphasises that these medications should be reserved for patients in whom an acid-mediated disease is likely, given that gastroprotectants are not devoid of adverse effects and should not be used indiscriminately.

Indications for gastroprotectant therapy include cases in which gastric ulceration is highly suspected based on physical examination, history, laboratory findings, and imaging where available. Oesophagitis is also a clear indication, as effective acid suppression may reduce the risk of oesophageal stricture formation. In selected high-risk patients predisposed to reflux oesophagitis, antacid therapy may be considered pre-anaesthesia, such as in animals with significant hiatal hernia or brachycephalic breeds including French Bulldogs that already regurgitate frequently. These recommendations do not extend to routine administration in standard anaesthetic patients. Gastroprotectants are also indicated in true gastrointestinal bleeding, including patients with melaena or substantial haematemesis, those bleeding secondary to gastric tumours or mast cell tumour-associated disease, and in patients with haemostatic disorders such as immune-mediated thrombocytopenia that are experiencing significant gastrointestinal haemorrhage. Additionally, acid suppression may be warranted following shock or critical illness when mucosal damage is expected.

Conversely, gastroprotectants are not indicated for uncomplicated vomiting or uncomplicated gastroenteritis, nor for dietary indiscretion. They are often unnecessary in pancreatitis, in which ulcerative disease is frequently absent and therapy may not confer benefit. They are similarly not indicated for chronic hepatopathies unless there is evidence of oesophagitis, ulceration, or gastrointestinal bleeding. In dogs with inflammatory bowel disease, omeprazole should not be prescribed solely on the basis of diagnosis or steroid administration unless clinical signs or laboratory findings suggest an acid-mediated disorder such as oesophagitis or gastrointestinal haemorrhage.

Chronic kidney disease warrants specific consideration. Historically, patients with chronic kidney disease have been presumed to develop gastrointestinal ulceration, and they may experience uraemic gastritis or gastropathy. However, studies in dogs and cats across chronic kidney disease stages 1, 2, and 3, in which endoscopy was performed, have not demonstrated ulcerative lesions. Findings may include glandular mineralisation and other mucosal changes, but not active ulceration. These patients frequently have reduced appetite and are often subject to polypharmacy; adding omeprazole unnecessarily may therefore impose additional burden and potential adverse effects, including effects on bone and mineralisation. Stage 4 disease is less clear, and when clinical signs suggest oesophagitis or ulceration, treatment may be appropriate; nevertheless, prophylactic use in stages 1 to 3 is not recommended.

Similarly, prophylactic administration of antacids when initiating steroids, meloxicam, or carprofen is not supported. These agents do not reliably prevent ulcers; rather, they are used to

treat ulcers when present, and there is no evidence that routine acid suppression at treatment initiation meaningfully alters ulcer risk.

Conclusion and Recommended Further Reading

The lecture has reviewed major causes of gastric ulceration in dogs and cats, the typical clinical signs and diagnostic findings, and a rational approach to therapy emphasising PPIs as first-line acid suppression where ulceration is suspected or confirmed, famotidine as a limited oral option with rapid tachyphylaxis but potential utility as a continuous infusion in severe bleeding, sucralfate as adjunctive mucosal protection, and misoprostol as a targeted therapy for NSAID-associated ulceration. A central theme is the need to avoid routine use of gastroprotectants in patients who are unlikely to have an acid-mediated disease, particularly given their adverse effects and the prevalence of overprescription in hospitalised animals. For deeper understanding, the ACVIM consensus statement in the Journal of Veterinary Internal Medicine provides a detailed review of mechanisms, indications, and the supporting evidence base, and is strongly recommended as follow-up reading.