

Review Article

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Hepatic-Origin Secondary Gastritis in Dogs: A Review

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ABSTRACT

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Canine gastritis is a frequently encountered yet often underdiagnosed gastrointestinal disorder characterized by inflammation of the gastric mucosa. Among the various etiologies, hepatic dysfunction and portal hypertension represent important but frequently overlooked causes of secondary gastritis and gastroduodenal ulceration in dogs. Hepatic-origin secondary gastritis develops due to altered gastric blood flow, impaired mucosal defense mechanisms, oxidative stress, metabolic derangements, and hormonal imbalances associated with liver disease. This review compiles and synthesizes available literature on the etiology, epidemiology, pathophysiology, diagnosis, and therapeutic management of hepatic-origin secondary gastritis in dogs, emphasizing the complex liver–stomach axis and its clinical relevance in veterinary practice.

Introduction

Canine gastric disorders are commonly associated with inflammatory processes and may manifest as gastritis, peptic ulceration, obstruction, or neoplasia. Gastritis refers to inflammation of the gastric mucosa and is frequently observed in dogs presenting with anorexia and vomiting. Despite its clinical prevalence, gastritis is rarely definitively diagnosed, particularly in acute cases where histopathological confirmation is seldom pursued. Chronic gastritis is similarly under-recognized and often not differentiated from generalized gastrointestinal disorders (Day et al., 2008; Dixon et al., 1996).

Gastritis in dogs is histologically classified based on the predominant inflammatory cell infiltrate and the presence of architectural alterations in the gastric

mucosa (Day et al., 2008; Dixon et al., 1996). In contrast, gastropathy describes nonspecific gastric mucosal injury with minimal or absent inflammation. Etiological factors include primary gastrointestinal disease, administration of non-steroidal anti-inflammatory drugs (NSAIDs) or glucocorticoids, systemic disorders such as hepatic or renal disease, endocrinopathies, shock, neurological disease, and neoplasia (Bolton, 1985).

Hepatic disorders are considered major risk factors for gastroduodenal ulceration in dogs (Doige and Furneaux, 1975; Dowdle et al., 2003). Altered gastric blood flow, portal hypertension, impaired epithelial turnover, accumulation of metabolic toxins, and oxidative stress collectively contribute to gastric mucosal injury. This review focuses on hepatic-origin secondary gastritis,

highlighting current understanding of its pathogenesis, diagnosis, and management.

Hepatic-Origin Secondary Gastritis

The liver plays a central role in the metabolism and clearance of endogenous compounds such as histamine, ammonia, bile acids, and gastrointestinal hormones (Bunch et al., 2006). In hepatic disease, particularly cirrhosis and portal hypertension, diversion of portal blood through collateral circulation or portosystemic shunts leads to systemic accumulation of these substances (Buob et al., 2011; Bosch et al., 2008). The stomach becomes a major target organ, predisposing affected dogs to erosions, ulceration, and gastrointestinal hemorrhage, commonly referred to as hepatogenic ulcers. Portal hypertension impairs gastric mucosal defense and alters gastric motility (Auroux et al., 2003; Buob et al., 2011). In dogs with chronic liver disease, gastric ulceration represents a significant cause of morbidity and mortality, with potential complications including perforation and septic peritonitis (Doige and Furneaux, 1975).

Etiology

Hepatic disease, both acute and chronic, is a major predisposing factor for gastroduodenal ulceration (Doige and Furneaux, 1975). Chronic liver disease results in reduced gastric mucosal perfusion due to portal hypertension and vascular thrombosis, while acute liver failure may involve disseminated intravascular coagulation and ischemia (Bosch et al., 2008; Buob et al., 2011). Portal hypertension may be prehepatic, intrahepatic, or posthepatic in origin, with intrahepatic causes such as fibrosis, chronic hepatitis, cirrhosis, and portal vein hypoplasia being most common in dogs (Buob et al., 2011).

Epidemiology

No consistent breed, age, or sex predisposition for gastritis has been conclusively identified; however, middle-aged large-breed dogs appear overrepresented in several reports (Dowdle et al., 2003). Liver dysfunction has been identified as an etiological factor in a proportion of dogs with gastritis, and increased incidence of gastric ulceration has been documented in dogs with congenital intrahepatic portosystemic shunts (Buob et al., 2011).

Pathophysiology

The gastric mucosa is protected by mucus-bicarbonate secretion, tight epithelial junctions, prostaglandins, nitric oxide, adequate mucosal blood flow, and rapid epithelial regeneration (Eastwood, 1991). In portal hypertension, these protective mechanisms are compromised (Bosch et al., 2008).

Altered gastric microcirculation, reduced mucus production, impaired epithelial renewal, decreased prostaglandin synthesis, excessive nitric oxide production, and oxidative stress collectively increase mucosal susceptibility to injury (Eastwood, 1991). Overproduction of reactive oxygen species and lipid peroxidation further amplify epithelial damage and delay healing (Drake et al., 1998; Chaudhuri et al., 2008; Crnogaj et al., 2010).

Diagnosis

Diagnosis of hepatic-origin secondary gastritis relies on clinical history, physical examination, laboratory testing, and imaging. Clinical signs include vomiting, anorexia, weight loss, hematemesis, melena, abdominal pain, and anemia (Bolton, 1985). Laboratory evaluation may reveal anemia, electrolyte imbalances, elevated liver enzymes, altered bile acid concentrations, and increased oxidative stress markers (Chaudhuri et al., 2008). Endoscopy remains the most sensitive diagnostic tool for definitive diagnosis and biopsy collection (Day et al., 2008).

Therapeutic Management

Management focuses on elimination of inciting factors, correction of systemic abnormalities, and protection of the gastric mucosa (Bexfield and Watson, 2009). Therapeutic measures include gastric rest, dietary modification, acid suppression with H₂-receptor antagonists or proton pump inhibitors, cytoprotective agents, fluid therapy, and correction of anemia and hypoproteinemia.

Adjunctive antioxidant therapy plays an important role in mitigating oxidative injury. Silymarin has demonstrated hepatoprotective, antioxidant, and anti-ulcer properties (Ajay et al., 2009; Alarcon de la Lastra et al., 1992). Its anti-ulcer activity is partly attributed to inhibition of lipid peroxidation and enhancement of

mucosal defense mechanisms (Alarcon de la Lastra et al., 1992). Carvedilol may reduce portal pressure through combined beta- and alpha-adrenergic blockade, although dose adjustment is required in advanced liver disease (Bosch et al., 2008).

In conclusion, Hepatic-origin secondary gastritis is a clinically important yet underdiagnosed condition in dogs with liver disease and portal hypertension. Impaired gastric mucosal defense, altered hemodynamics, and oxidative stress play central roles in its pathogenesis. Early recognition, comprehensive diagnostic evaluation, and integrated therapeutic approaches targeting both hepatic dysfunction and gastric protection are essential for improving clinical outcomes. Further research is needed to clarify disease mechanisms and optimize evidence-based management strategies.

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Author Contributions

Shivani Khare: Investigation, formal analysis, writing—original draft, data curation, reviewing the final version of the manuscript.

Data Availability

The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethical Approval Not applicable.

Consent to Participate Not applicable.

Consent to Publish Not applicable.

Conflict of Interest The authors declare no competing interests.

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