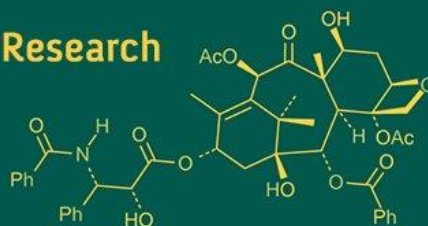


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Case presentation: Gastric dilatation-volvulus (GDV) in dogs: A comprehensive review and case presentation

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Abstract

Gastric Dilatation-Volvulus (GDV) is an acute, life-threatening syndrome primarily affecting large and giant breed dogs. Characterized by rapid gastric distention and rotation, its precise etiology is complex, involving risk factors like breed, age, diet, and temperament. Despite aggressive interventions, GDV carries a substantial 15-24% mortality rate. Clinically, GDV presents abruptly with abdominal distension and unproductive retching, often post-feeding/exercise. Systemic consequences include hypovolemic shock, cardiac arrhythmias, gastric necrosis, and multi-organ hypoperfusion. Prognosis is significantly impacted by gastric necrosis and presentation time. A case in an four-year-old female Labrador, presenting with sudden death and abdominal distension, revealed characteristic post-mortem findings. Necropsy showed severe gastric dilatation with serosal congestion and mucosal necrosis which showed progression to gangrene, enlarged spleen and liver (nutmeg appearance), pale kidneys, and cardiac hypertrophy/dilatation. These findings confirmed multi-systemic dysfunction, including gastrointestinal, renal, and respiratory compromise, consistent with fatal GDV. Treatment necessitates immediate stabilization, rapid decompression, and surgical correction with gastropexy. Anesthetic protocols prioritize cardiovascular stability. Challenges remain due to GDV's rapid progression. Continued research in pathophysiology, diagnostics, and prophylactic strategies is crucial to mitigate GDV's devastating impact on canine health and welfare.

Keywords: Gastric dilatation-volvulus, GDV, large breed dogs, gastric distention

Introduction

Gastric dilatation-volvulus (GDV) is a critical and frequently encountered syndrome in veterinary medicine, primarily affecting large and giant breed dogs, although it can also manifest in smaller canines and felines. This life-threatening condition is characterized by acute gastric distention and malposition, often involving a rotation of the stomach along its mesenteric axis. Despite its significant clinical impact, the precise etiology and pathogenesis of GDV remain incompletely understood, though various risk factors have been identified (Monnet, 2003) [18]. The rapid onset and severe systemic consequences of GDV necessitate prompt and aggressive medical and surgical intervention.

Epidemiological studies indicate that GDV is most commonly observed in large and giant, deep-chested breeds (Brockman *et al.*, 1995; Tivers *et al.*, 2009) [4, 22]. While no sex predisposition has been definitively established, affected dogs typically range from 10 months to 14 years of age (Brockman *et al.*, 1995) [4]. Despite concerted efforts in treatment, the fatality rate for GDV remains substantial, reportedly ranging from 15% to 24% (Millis *et al.*, 1995) [17]. Identified risk factors extend beyond breed and age to include familial predisposition, specific dietary characteristics like small food particle size, rapid eating, and certain temperamental traits (Glickman *et al.*, 1997; Theyse *et al.*, 1998; Glickman *et al.*, 2000) [10, 11, 21]. Furthermore, conditions such as splenectomy have been associated with an increased risk of GDV development (Millis *et al.*, 1995; Bell and Jerold S 2014) [1, 17].

Etiology

Etiology and pathogenesis of GDV are not well understood but specific risk factors have been identified.

Epidemiology & Risk factors

Gastric dilatation-volvulus (GDV) is a life-threatening condition primarily affecting large and giant-breed dogs, although it has also been documented in small dogs and cats (Brockman *et al.*, 1995; Tivers *et al.*, 2009; Rozanski *et al.*, 2014) [4, 19, 22]. The age range for diagnosis is broad, spanning from 10 months to 14 years (Brockman *et al.*, 1995) [4], with some studies indicating that age is the most significant risk factor, with older dogs being at a higher risk of developing GDV (Theyse *et al.*, 1998; Betts *et al.*, 1974) [2, 21].

Despite aggressive medical and surgical interventions, the fatality rate for GDV remains high, with reported ranges from 15% to 24% (Millis *et al.*, 1995) [17].

Several factors have been investigated for their potential association with GDV. While no sex predilection has been demonstrated (Brockman *et al.*, 1995) [4], dietary factors have been identified as potential risk factors. Specifically, a diet containing food particles larger than 30 mm was found to be the second most important risk factor after age (Theyse *et al.*, 1998) [21]. Other variables, such as gender, neuter status, feeding frequency, food intake time, interval between feeding and exercise, duration of exercise, and overall physical activity, have not been conclusively identified as risk factors in some studies (Theyse *et al.*, 1998) [21]. Additionally, a history of splenectomy has been associated with the development of GDV (Millis *et al.*, 1995) [17].

Gastric dilatation-volvulus (GDV) is a serious condition primarily affecting certain dog breeds. A 2000 study by Glickman and colleagues found the Great Dane had the highest incidence of GDV among seven large and four giant breeds. Other breeds with a high occurrence included the Bloodhound, Irish Wolfhound, Akita, Irish Setter, Standard Poodle, Collie, Weimaraner, Newfoundland, Saint Bernard, and Rottweiler.

Beyond breed, genetics play a role. An increased risk of GDV is linked to a family history of GDV in a first-degree relative, such as a sibling or offspring. Additionally, increasing age is associated with a higher GDV risk in large and giant breed dogs (Glickman *et al.*, 2000) [11].

Several factors impact a dog's chance of survival from GDV. While the age, time from the last feeding to symptom onset, and body weight didn't differ much between dogs that survived and those that didn't, the time from when clinical signs appeared until the dog was presented at the clinic is a significant factor in survival rates. The sooner a dog receives veterinary care, the better its chances (Zatloukal *et al.*, 2005; Hendriks *et al.*, 2012) [14, 24].

The presence of gastric necrosis (tissue death in the stomach) dramatically increases mortality, with a rate of 26.3%—significantly higher than in dogs without necrosis ($p < 0.01$). Dogs with gastric necrosis are 6.5 times more likely to die. Furthermore, dogs undergoing a splenectomy (removal of the spleen) also face a higher mortality rate ($p < 0.05$) (Zatloukal *et al.*, 2005) [24]. Certain lifestyle and personality traits can influence a dog's risk of developing GDV (Glickman *et al.*, 1997; Monnet *et al.*, 2003) [10, 18]. Factors that significantly increase a dog's risk ($p < 0.10$) include: Eating only one meal daily, Being male, Eating very quickly, Having a fearful temperament, Being underweight

Conversely, some factors appear to significantly reduce the risk of GDV: Including table foods in a diet primarily consisting of dry dog food, Having a "happy" temperament.

It's also worth noting that stress seems to be a trigger for acute GDV episodes (Glickman *et al.*, 1997; Monnet *et al.*, 2003) [10, 18].

Research has indicated a potential anatomical difference in dogs affected by gastric dilatation-volvulus (GDV). Hall and colleagues (1995) [13] observed that the hepatogastric ligaments in dogs diagnosed with GDV were notably longer when compared to those in control animals. The precise cause-and-effect relationship remains uncertain; it is unclear whether this increased ligament length is a consequence of GDV or if such elongated ligaments contribute to a predisposition for the condition. This observation might suggest that an increased looseness or laxity of the supporting hepatogastric ligament, located in the right upper abdomen, plays a role. A ligament of greater length could potentially allow for enhanced stomach mobility, thereby increasing a dog's susceptibility to either partial or complete gastric volvulus (Hall *et al.*, 1995) [13].

The incidence of Gastric Dilatation-Volvulus (GDV) is consistently correlated with both breed and age. Great Danes demonstrate the highest occurrence of GDV among giant breeds, whereas Newfoundlands exhibit the lowest prevalence (Glickman *et al.*, 2000; Bhatia *et al.*, 2010) [3, 11]. Similarly, within large breeds, Bloodhounds show the highest GDV incidence, while Rottweilers have the lowest. Notably, Great Danes experience a significantly higher prevalence of GDV compared to other giant or large-breed dogs. However, research has not established a direct relationship between a breed's average height or weight and its susceptibility to GDV (Glickman *et al.*, 2000) [11].

The onset of clinical manifestations associated with Gastric Dilatation-Volvulus (GDV) is typically abrupt, frequently reported to occur subsequent to the ingestion of a large meal and/or a period of strenuous physical activity (Tivers *et al.*, 2009) [22]. Canines afflicted with this condition commonly exhibit a spectrum of signs, including behavioral indicators such as restlessness and agitation, a generalized reduction in activity and responsiveness termed lethargy, and a progressive increase in abdominal girth due to gas accumulation, known as abdominal distension. A particularly characteristic sign is unproductive vomiting or retching, where the animal makes efforts to vomit but expels little to no gastric content, indicative of a mechanical obstruction (Tivers *et al.*, 2009) [22]. In severe instances, the condition can culminate in collapse, signifying profound physiological compromise.

Clinically, affected dogs frequently present with evidence of hypovolemic shock, a critical state resulting from reduced circulating blood volume. The signs of hypovolemic shock observed include tachycardia (an elevated heart rate), poor peripheral pulses (indicating diminished peripheral perfusion), tachypnea (increased respiratory rate), and pale mucous membranes (reflecting inadequate tissue oxygenation and perfusion). The degree of hypovolemia can vary considerably among individual cases, and consequently, the severity of the associated clinical signs will fluctuate accordingly (Tivers *et al.*, 2009) [22].

Regarding prognostic factors and outcomes, studies have consistently demonstrated that the presence of gastric necrosis (tissue death of the stomach wall) is associated with a significantly elevated mortality rate ($p < 0.01$) in dogs diagnosed with GDV (Zatloukal *et al.*, 2005; Gazzola *et al.*, 2014) [8, 24]. This highlights the critical importance of assessing gastric viability during surgical intervention.

However, it is noteworthy that when gastric necrosis is identified and surgically managed through gastrectomy (resection of the necrotic gastric tissue), this complication is no longer independently linked to increased mortality. This underscores the positive impact of timely and appropriate surgical intervention in mitigating the severe consequences of gastric tissue compromise. Furthermore, a heightened mortality rate ($p < 0.05$) has also been observed in dogs undergoing splenectomy in conjunction with GDV correction (Zatloukal *et al.*, 2005; Gazzola *et al.*, 2014) [8, 24]. This could be attributed to various factors, including the systemic impact of splenic injury or the increased surgical complexity in cases where the spleen is also affected and requires removal.

Clinico pathological findings

Gastric dilatation and volvulus (GDV) represents a prevalent and critical emergency in veterinary medicine, carrying substantial rates of morbidity and mortality. This condition instigates widespread systemic derangements, impacting multiple organ systems including the respiratory, cardiovascular, gastrointestinal, and renal systems, in addition to inducing coagulation abnormalities (Wingfield, *et al.*, 1974; Humm *et al.*, 2014) [15, 23].

From a cardiovascular standpoint, GDV can lead to various manifestations, such as cardiac arrhythmias, myocardial dysfunction, and ultimately, shock. Respiratory compromise in GDV is primarily a consequence of the mechanical effects of gastric dilatation, which impedes diaphragmatic movement and leads to decreased pulmonary perfusion. Within the gastrointestinal tract itself, a critical and life-threatening consequence is gastric necrosis, resulting from compromised blood flow to the stomach wall. Beyond these, dogs afflicted with GDV frequently exhibit acute kidney injury, as well as significant acid-base and electrolyte disturbances (Wingfield, *et al.*, 1974; Humm *et al.*, 2014) [15, 23].

The pathophysiology of the gastric dilatation-torsion complex in canines involves a cascade of interconnected changes that, if not addressed with prompt therapeutic intervention, can rapidly culminate in fatality (Wingfield, *et al.*, 1974; Humm *et al.*, 2014) [15, 23]. The initial event in this complex is typically gastric dilation, which may or may not progress to gastric torsion (volvulus). A crucial systemic consequence of significant gastric dilation is the mechanical compression of major abdominal vessels, specifically the caudal vena cava and the portal vein. This compression profoundly impedes venous return of blood to the heart, leading to a direct reduction in cardiac output and subsequent systemic arterial hypotension.

The onset of this systemic hypotension initiates a cascade of detrimental effects on cellular and organ function. Diminished arterial pressure results in widespread impairment of tissue perfusion, leading to cellular hypoxia and a shift towards anaerobic metabolism, which in turn promotes cellular catabolism and the accumulation of metabolic byproducts. Concurrently, there is a marked decrease in renal function due to reduced renal blood flow. Furthermore, the severe physiological stress and compromised organ perfusion associated with GDV predispose affected animals to the development of endotoxic shock (often due to bacterial translocation from compromised gut mucosa) and disseminated intravascular coagulation (DIC). These two profound systemic processes

are significant contributors to the high mortality rate observed in untreated or severely affected animals (Sharp *et al.*, 2014) [20]. The interplay of these multi-organ dysfunctions underscores the critical nature of GDV and the necessity for rapid, aggressive medical and surgical management.

In dogs presenting with Gastric Dilatation-Volvulus (GDV), cardiac arrhythmias are a frequently observed complication, with studies reporting their detection in approximately 40% of affected animals (Brockman *et al.*, 1995; DeHoff *et al.*, 1974) [4, 7]. These arrhythmias represent a significant cardiovascular manifestation of the systemic derangements induced by GDV, which, as previously discussed, include hypovolemic shock, acid-base imbalances, and myocardial compromise. The specific types of arrhythmias can vary, but they often include ventricular premature complexes (VPCs) and ventricular tachycardia, which are thought to arise from myocardial hypoxia, electrolyte disturbances (such as hypokalemia), and increased sympathetic tone.

Despite their common occurrence, interestingly, no statistically significant correlation has been established between the development of a cardiac arrhythmia and the ultimate outcome (prognosis or mortality) in dogs afflicted with GDV (Brockman *et al.*, 1995; DeHoff *et al.*, 1974) [4, 7]. This finding might initially seem counterintuitive given the critical nature of cardiac function. However, from a pathology perspective, this suggests that while arrhythmias are a common sign of underlying systemic stress, they may not always be the primary or direct cause of death in GDV cases. Instead, the overall prognosis is likely more heavily influenced by the severity of the multi-organ dysfunction, the extent of gastric necrosis, the degree of shock, and the timeliness and efficacy of medical and surgical intervention. Indeed, the underlying causes of death in dogs suffering from GDV are multifactorial and highly variable (Brockman *et al.*, 1995; DeHoff *et al.*, 1974) [4, 7]. As discussed, these can include irreversible shock, profound metabolic acidosis, severe gastric necrosis leading to peritonitis and sepsis, acute kidney injury, disseminated intravascular coagulation (DIC), and refractory arrhythmias that do lead to severe myocardial dysfunction or cardiac arrest. Therefore, while arrhythmias require careful monitoring and management to stabilize the patient, their presence alone may not be the sole determinant of survival in a disease as complex and systemically devastating as GDV. The overall picture of organ system failure and the body's compensatory mechanisms against overwhelming physiological insult ultimately dictate the patient's fate.

Treatment

Managing Gastric Dilatation-Volvulus (GDV) critically depends on appropriate anesthetic protocols and timely surgical intervention. For pre-anesthetic medication, a combination of an opioid and a benzodiazepine is generally recommended (Broome *et al.*, 2003) [5]. The choice of induction agents is crucial, with a preference for those that induce minimal cardiovascular alterations. Recommended options include opioids, neuroactive steroidal agents, and etomidate (Broome *et al.*, 2003) [5]. Anesthesia should subsequently be maintained using an inhalational agent.

The cornerstone of GDV treatment is surgical therapy, which encompasses several vital steps:

Decompression: Relieving the gas accumulation within the stomach. Correction of Gastric Malpositioning: Manually

repositioning the stomach to its anatomical location. Debridement of Necrotic Tissue: Excising any non-viable (necrotic) portions of the gastric wall, which is critical for preventing further systemic compromise and peritonitis. Gastropexy: A surgical procedure to permanently adhere the stomach to the abdominal wall, thereby preventing future episodes of volvulus (Broome *et al.*, 2003)^[5].

Various techniques for gastropexy exist, including but not limited to: Incisional gastropexy

Circumcostal gastropexy, Tube gastropexy, Incorporating gastropexy, Belt-loop gastropexy, Laparoscopic gastropexy (Broome *et al.*, 2003)^[5].

Despite surgical intervention, the expected mortality rate associated with GDV remains significant, typically ranging from 15% to 24% (Broome *et al.*, 2003)^[5]. Several factors have been identified as prognostic indicators influencing the outcome for dogs with GDV. These include the animal's mental status upon presentation, which reflects the severity of shock and central nervous system perfusion deficits; the presence of gastric necrosis, a direct indicator of severe gastric tissue damage; the presence of cardiac arrhythmia, signifying significant cardiovascular instability; and plasma lactate levels, which serve as a marker of tissue hypoperfusion and anaerobic metabolism (Broome *et al.*, 2003)^[5]. Given the high risk of recurrence, prophylactic gastropexy should be strongly considered for dogs identified as being at high risk for GDV (Broome *et al.*, 2003)^[5].

In terms of adjunctive medical management, early administration of intravenous lidocaine, followed by a continuous rate infusion (CRI) for 24 hours post-presentation, has shown promising results. In a study comparing lidocaine-treated dogs with GDV to historical controls, this protocol significantly reduced the occurrence of cardiac arrhythmias, lowered the incidence of acute kidney injury (AKI), and decreased the overall hospitalization time period (Bruchim *et al.*, 2012; Meyer-Lindenberg *et al.*, 1993)^[6, 16]. However, it is important to acknowledge that this particular study had limitations, being non-blinded, placebo-uncontrolled, and non-randomized. Consequently, further rigorous evaluation of lidocaine's efficacy in dogs with GDV is warranted to confirm these beneficial effects and establish definitive treatment protocols.

Materials and methods

1. **Animal:** A four years old Labrador female carcasses was presented to department of veterinary pathology, Veterinary college, Hebbal. for post mortem examination in the month of September 2023.
2. **History:** history of sudden death and distended abdomen.
3. **Postmortem examination of animal:** Thorough post mortem examination of carcasses was done.

Results

The postmortem examination of the carcass revealed several significant post-mortem changes indicative of Gastric Dilatation and Volvulus (GDV).

Gross External Examination

The general body condition of the carcass was good, suggesting that the animal was previously healthy. Externally, the most striking finding was a markedly

distended abdomen, a classic sign of severe gastric tympany. The visible mucous membranes were pale, a finding consistent with hypovolemic shock and poor peripheral perfusion that typically accompanies GDV. Rigor mortis set in.

Abdominal Cavity and Organs

Upon opening the abdominal cavity, a distended stomach was immediately apparent. The observation of blood flowing from the incision site suggests significant vascular compromise and possibly splenic or gastric vessel rupture, or severe congestion. The spleen was enlarged, and its cut surface revealed dark-colored blood, characteristic of splenic congestion. This is a common secondary finding in GDV, as the spleen often becomes entrapped and congested within the twisting stomach. The liver was enlarged with rounded edges, and its cut surface exhibited a "nutmeg" appearance with multifocal pale areas. The "nutmeg" pattern is indicative of chronic passive congestion (likely due to impaired venous return via the caudal vena cava), while the pale areas could represent areas of hepatocellular degeneration or necrosis due to ischemia. The kidneys showed cortical pale areas, suggestive of renal ischemia, a consequence of reduced blood flow during the hypotensive state associated with GDV.

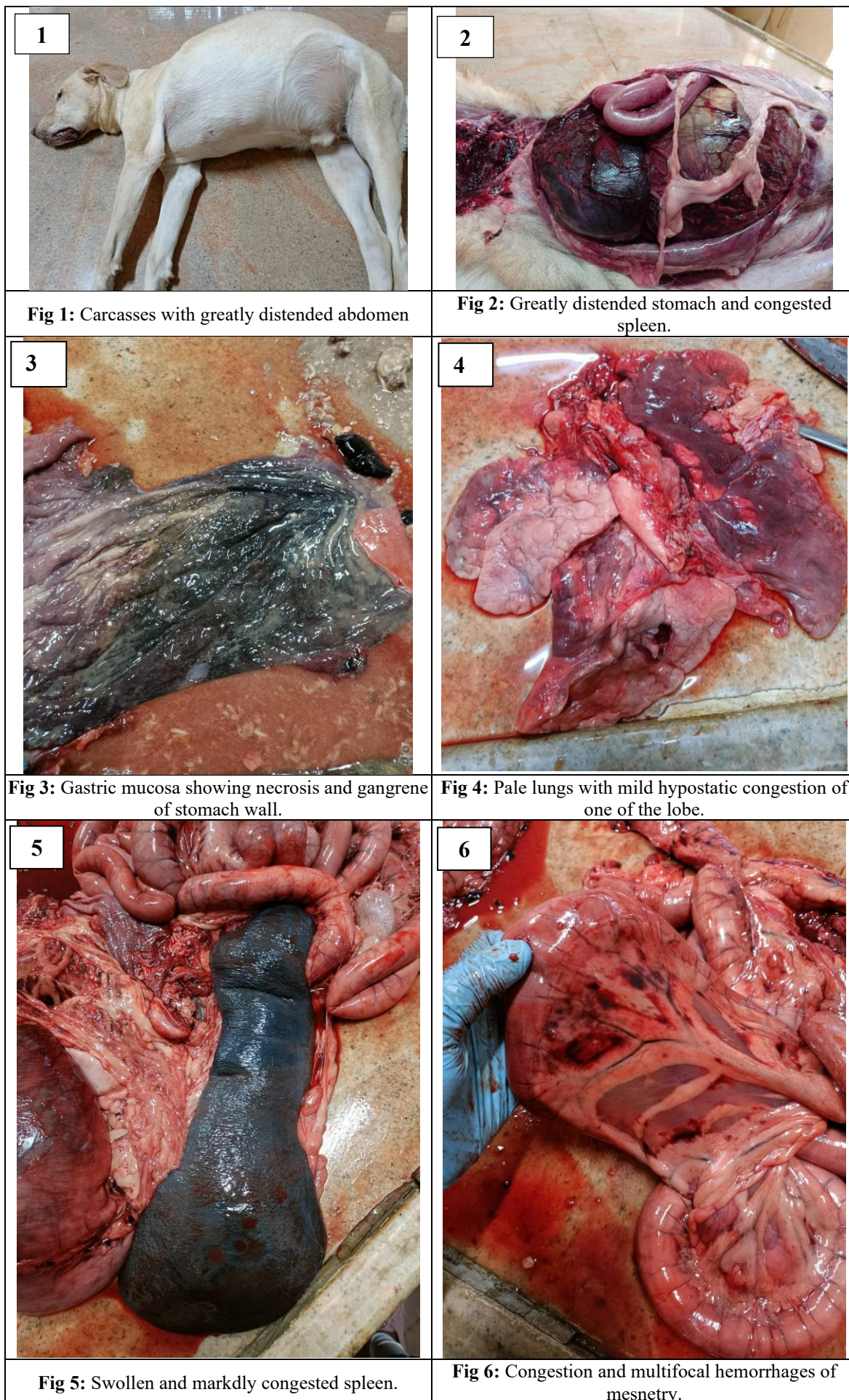
Thoracic Cavity and Organs

The lungs exhibited hypostatic congestion, which is post-mortem settling of blood in dependent areas of the lungs. While not specific to GDV, severe respiratory compromise and hypovolemia could exacerbate this. The heart presented with left ventricular hypertrophy and dilatation of the right ventricle. Left ventricular hypertrophy might indicate pre-existing cardiac disease. Right ventricular dilatation is a significant finding in GDV, often due to increased afterload resulting from compression of the caudal vena cava, which impedes venous return to the right side of the heart. This can lead to right-sided heart failure and subsequent systemic venous congestion.

Gastrointestinal Tract

The stomach displayed severe dilatation and severe serosal congestion, indicative of profound gastric distension and compromised venous drainage. The gastric mucosa showed blackish discoloration, which is a hallmark of gastric necrosis resulting from severe ischemia due to twisting of the organ on its mesentery. Congestion and emphysematous areas (gas within the gastric wall, often due to bacterial proliferation in ischemic tissue) were also observed within the gastric mucosa. The serosal surface of the intestines exhibited severe congestion and hemorrhages, reflecting generalized splanchnic vascular compromise and possibly early disseminated intravascular coagulation (DIC).

Based on the constellation of these gross pathological findings – particularly the gastric distension and signs of volvulus (splenic displacement, severe gastric serosal congestion and mucosal necrosis), and widespread evidence of systemic shock and hypoperfusion affecting the spleen, liver, kidneys, and intestines – the diagnosis of Gastric Dilatation and Volvulus (GDV) is strongly supported. These findings paint a clear picture of the multi-organ system failure that characterizes this acute and often fatal condition.



Discussion

Gastric dilatation and volvulus (GDV) constitutes an acute and life-threatening emergency in canines, frequently leading to fatal outcomes. Given that the precise etiology of GDV remains incompletely understood, a proactive approach by pet owners focusing on known risk factors is paramount. These predispositions include, but are not limited to:

Physiological Factors: Decreased stomach motility: Reduced efficiency of gastric muscle contractions. Delayed gastric emptying: Food remaining in the stomach for prolonged periods.

Dietary and Feeding Practices: Diet composition: Certain diets may be implicated, though specific links are still debated. Amount of food ingested: Large meal volumes are a significant risk. Frequency of feeding: Less frequent, larger meals tend to increase risk. Feeding behavior: Rapid eating habits ("fast eating style") can lead to aerophagia (swallowing air). Activity and Stress: Exercise after a meal: Physical exertion immediately following food intake. Stress after a meal: Environmental or psychological stress following feeding. Breed and Conformation: Large-breed dogs: A well-established breed predisposition, with giant breeds particularly affected. Dogs with a large thoracic depth-to-width ratio: Deep-chested breeds are anatomically predisposed. Body Condition and Age:

Underweight dogs: Surprisingly, some studies suggest a higher risk. Old age: Advanced age is also considered a risk factor. Given its rapid progression and severe systemic consequences, GDV mandates immediate and aggressive veterinary intervention. The primary contributors to mortality in GDV cases stem from a complex interplay of physiological disturbances:

Hemodynamic Derangements: Profound cardiovascular shock, characterized by decreased venous return, reduced cardiac output, and severe hypotension, leading to widespread tissue hypoperfusion. Gastric Disturbances: Primarily, the mechanical obstruction and subsequent gastric necrosis resulting from ischemia to the stomach wall. Renal Disturbances: Acute kidney injury, often secondary to hypoperfusion, coupled with severe acid-base and electrolyte imbalances (e.g., metabolic acidosis, hypokalemia) that further destabilize cellular function and organ systems.

In the present case, the necropsy findings provide clear evidence of these critical contributors to the animal's demise. The presence of severe gastric necrosis, observed as blackish discoloration of the gastric mucosa, directly aligns with the established literature, which reports a significantly higher mortality rate ($p < 0.01$) in dogs presenting with this specific lesion (Zatloukal *et al.*, 2005)^[24]. Furthermore, the gross findings of pale mucous membranes, splenic congestion, "nutmeg" liver, and pale renal cortices are all consistent with the profound hemodynamic derangements and multi-organ hypoperfusion that characterize severe GDV and were highly significant contributors to the fatal outcome in this animal. The evidence from the post-mortem examination therefore strongly corroborates the clinical and pathological understanding of GDV as a rapidly progressive

and often lethal condition driven by both mechanical and systemic complications.

Conclusion

Gastric Dilatation-Volvulus represents a formidable emergency in canine veterinary practice, characterized by a complex pathophysiology that extends beyond the gastrointestinal system to induce severe cardiovascular, respiratory, renal, and coagulation dysfunctions. The rapid progression of the syndrome, coupled with the systemic derangements it precipitates, underscores the absolute necessity for immediate diagnosis and aggressive stabilization to mitigate the high associated morbidity and mortality. Understanding the intricate interplay of gastric distention, circulatory compromise, and subsequent organ damage is paramount for effective critical care management in affected patients.

While advancements in emergency medicine and surgical techniques have improved outcomes, GDV continues to pose significant challenges. Given the diverse and sometimes subtle risk factors, client education on breed predispositions, feeding practices, and early recognition of clinical signs remains crucial for prevention. Furthermore, the consideration of prophylactic gastropexy in at-risk breeds stands as a vital preventive strategy. Ongoing research is essential to further unravel the precise mechanisms underlying GDV, refine diagnostic indicators, and develop more targeted therapeutic approaches, ultimately aiming to reduce the devastating impact of this condition on canine health and welfare.

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