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Topical Review

Cardiovascular and Systemic Effects of Gastric Dilatation and Volvulus in Dogs

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Gastric dilatation and volvulus (GDV) is a common emergency condition in large and giant breed dogs that is associated with high morbidity and mortality. Dogs with GDV classically fulfill the criteria for the systemic inflammatory response syndrome (SIRS) and can go on to develop multiple organ dysfunction syndrome (MODS). Previously reported organ dysfunctions in dogs with GDV include cardiovascular, respiratory, gastrointestinal, coagulation and renal dysfunction. Cardiovascular manifestations of GDV include shock, cardiac arrhythmias and myocardial dysfunction. Respiratory dysfunction is also multifactorial, with contributory factors including decreased respiratory excursion due to gastric dilatation, decreased pulmonary perfusion and aspiration pneumonia. Gastrointestinal dysfunction includes gastric necrosis and post-operative gastrointestinal upset such as regurgitation, vomiting, and ileus. Coagulation dysfunction is another common feature of MODS in dogs with GDV. Disseminated intravascular coagulation can occur, putting them at risk of complications associated with thrombosis in the early hypercoagulable state and hemorrhage in the subsequent hypocoagulable state. Acute kidney injury, acid-base and electrolyte disturbances are also reported in dogs with GDV. Understanding the potential for systemic effects of GDV allows the clinician to monitor patients astutely and detect such complications early, facilitating early intervention to maximize the chance of successful management.

Introduction

Gastric dilatation volvulus (GDV), commonly referred to as bloat, is a common condition in large and giant breed dogs presenting for emergency care that is associated with high morbidity and the potential for mortality. Although the pathogenesis of GDV is incompletely understood, a lot is known about the potential cardiovascular and systemic side effects of the condition. GDV results in development of the systemic inflammatory response syndrome (SIRS) and potentially multiple organ dysfunction syndrome (MODS). Organ dysfunctions that have been documented as part of MODS in dogs with GDV include cardiovascular, respiratory, gastrointestinal (GI), coagulation, and renal dysfunction. Hepatic, central nervous system, and endocrine system dysfunction have not been specifically evaluated in dogs with GDV, although are considered other important organ systems involved in MODS.[1] The multisystemic effects of GDV will be discussed here with a focus on pathophysiology. The final article in this special issue discusses management of the postoperative GDV patient, with a special focus on management of complications and organ dysfunctions.[2]

Pathophysiology

The pathogenesis of GDV is complex and multifactorial, with apparent genetic and environmental influences. The first article in this special issue discusses the genetic predispositions to GDV in dogs,[3] whereas the second article discusses the role of GI motility disturbances in the development of GDV.[4] It is unknown whether dilatation or volvulus occurs first in dogs with GDV, but either is possible as dilatation in the absence of volvulus and volvulus in the absence of dilatation are both reported.

Dogs with GDV classically fulfill criteria for SIRS, although this has not been formally evaluated to the knowledge of the authors.[5,6] Although no consensus definition exists, in general, in veterinary studies, dogs are considered to have SIRS, if they have an underlying disease that could predispose to SIRS in addition to having abnormalities of a least 2 of the following 4 abnormalities: body temperature, heart rate (HR), respiratory rate (RR), and white blood cell count or differential.

There are many potential triggers for SIRS and subsequently MODS in dogs with GDV, including global tissue hypoperfusion and cellular ischemia, gastric ischemia and reperfusion, gastric necrosis, and GI translocation of bacteria and bacterial products. Ischemia reperfusion injury (IRI) have been discussed extensively in the context of GDV.[5,7,8] Tissue injury and cell death leading to the release of damage-associated molecular patterns into circulation, in addition to the potential for translocation of microbial pathogen-associated molecular patterns (PAMPS), trigger the innate immune response, resulting in the production of proinflammatory cytokines, activation of the complement cascade, activation of coagulation, and associated downstream effects. Inflammation is also exacerbated by the reactive oxygen species produced as a consequence of IRI. In addition, general anesthesia required for surgical correction of GDV may be a "second hit" that exacerbates SIRS and contributes to MODS in these patients. The potentially deleterious components or effects of general anesthesia are thought to include administration of 100% oxygen (albeit for a relatively short period of time), positive pressure ventilation, vasodilation and hypotension, and the immunomodulatory effects of opioid analgesics.[9] Dysregulation of the immune system and mitochondrial dysfunction are also thought to be key to the initiation of the MODS in people with sepsis and SIRS[1]; however, these pathologies have not yet been explored in dogs with GDV.

Cardiovascular Dysfunction

Cardiovascular manifestations of GDV include shock, cardiac arrhythmias, and myocardial dysfunction. Cardiovascular compromise is thought to be the primary systemic side effect that contributes to early morbidity and mortality in dogs with GDV.

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The end result of cardiovascular dysfunction is decreased tissue oxygen delivery (DO_2) and the clinical manifestations of shock. Decreased DO_2 contributes to the development of MODS, with consequences on the cardiovascular, respiratory, renal, GI, and central nervous systems. Dogs with GDV likely have a combination of obstructive, distributive, hypovolemic, and cardiogenic shock.

Obstructive Shock

Marked gastric dilatation results in the compression of the low-pressure intra-abdominal veins, including the portal vein, splenic veins, and caudal vena cava. As a consequence, venous return to the heart, and subsequently stroke volume, is reduced. HR will increase to compensate for reduced stroke volume; however, usually this compensation is inadequate and cardiac output too is reduced.

Distributive Shock

Decreased venous return and increased venous pressure result in splanchnic pooling of blood. Compromise of splenic blood flow tends to result in splenic congestion and splenomegaly. Splanchnic vasodilation is likely also exacerbated by production of the potent endogenous vasodilator nitric oxide via inducible nitric oxide synthase, further exacerbating peripheral vasodilation and splanchnic pooling of blood.

Hypovolemic Shock

Absolute hypovolemia is likely a relatively small component of the shock syndrome in dogs with GDV, although they may well have a relative hypovolemia. In one study, 34% of dogs with GDV had microcardia on thoracic radiographs suggestive of hypovolemia, although obstructive and distributive shock could also account for this.[10]

Loss of intravascular volume can occur via abdominal hemorrhage, third spacing of fluids, and GI secretions. Concurrently gastric inflow obstruction prevents oral intake of water. Blood loss associated with GDV is generally relatively minor and typically occurs as a result of the rupture of small gastric vessels as gastric volvulus occurs. For this reason, a small volume hemoabdomen is often noted at the time of abdominal surgery. Splanchnic pooling of blood and portal hypertension can contribute to third spacing of fluid and interstitial edema particularly in the abdominal viscera.

Cardiogenic Shock

Cardiogenic shock can occur as a result of myocardial dysfunction and cardiac arrhythmias.

Cardiac arrhythmias, predominantly ventricular arrhythmias, occur in approximately 40% of dogs with GDV, likely as a result of myocardial ischemia.[11,12] Significant reductions in coronary blood flow have been documented in experimentally induced GDV in dogs, and histopathologic evidence of myocardial ischemia has been seen in both experimental and naturally occurring GDV.[13] It is well known that ischemic myocardium is likely to establish ectopic foci of electrical activity. Dogs with GDV also commonly have elevated circulating concentrations of cardiac troponins, and concentrations of these biomarkers correlate with the severity of electrocardiogram abnormalities and patient outcome.[14] Increased circulating concentrations of cardiostimulatory substances such as the catecholamines, and cardioinhibitory substances such as proinflammatory cytokines (e.g., tumor necrosis factor α), have also been implicated in the generation of arrhythmias.

Physical examination abnormalities in dogs presenting with GDV are generally manifestations of the circulatory and respiratory compromise they experience. Dogs often present in early decompensated shock, with tachycardia, weak pulses, and depressed mentation. Depending on the state of compensation mucus membranes may be pale or injected, and capillary refill time may be rapid or prolonged. Irregular cardiac rhythms and pulse deficits may be present. In one study, 18 of 20 dogs (90%) had tachycardia (HR $\geq$ 120), and all dogs had abnormal perfusion parameters (mucus membranes, capillary refill time, and pulses).[15] Tachypnea, dyspnea, or both may be associated with discomfort and respiratory compromise as documented later.

Hyperlactatemia is a common finding in dogs with GDV. Its origin is likely multifactorial, stemming from both global and local (gastric) hypoperfusion. Although multiple studies report the use of lactate as a prognostic biomarker in dogs with GDV,[16-19] the true prevalence of hyperlactatemia at presentation is not always reported. One recent study documented that 58.5% of dogs (38/66) with GDV were hyperlactatemic at presentation,[20] whereas another reported that 30% of dogs (30/101) had an admitting lactate level $>$ 6 mmol/L.[10]

Respiratory Dysfunction

Respiratory compromise may occur in dogs with GDV as a result of gastric dilatation adversely affecting normal respiratory excursion, reduced pulmonary perfusion, and aspiration pneumonia.

Gastric dilatation and increased intra-abdominal pressure will decrease the total thoracic volume and prevent the normal caudal excursion of the diaphragm to initiate inspiration, resulting in hypoventilation and ventilation-perfusion mismatching. In severe cases, lung lobe collapse can also occur. Increased RR and effort ensue so as to compensate, although these efforts may become inadequate and hypercapnea and hypoxemia can occur. Pulmonary blood flow may also be decreased in dogs with GDV due to decreased cardiac output, further contributing to ventilation-perfusion mismatch.

Dogs with GDV are at risk of developing both preoperative and postoperative aspiration pneumonia. In addition, multiple studies identified pneumonia in dogs with GDV as a poor prognostic indicator.[11,12] As dogs with GDV often fulfill SIRS criteria and have tachypnea and dyspnea on presentation, the presence of pneumonia can be difficult to discern based on clinical signs alone. As such, preoperative thoracic radiographs are recommended to identify evidence of aspiration pneumonia and guide antimicrobial therapy.[10]

Of a population of 101 dogs with GDV, Green et al.[10] documented that at presentation 27% were tachypneic (RR $>$ 34 breaths/min), 20% had increased respiratory effort, 2% had respiratory distress, and 14% had evidence of aspiration pneumonia on preoperative thoracic radiographs. The development of the acute respiratory distress syndrome has also been reported in dogs with GDV.[19]

GI Dysfunction

Gastric necrosis is a feared complication of GDV, as it contributes to morbidity and mortality. In dogs with GDV, gastric blood flow is likely decreased owing to a combination of factors including compression, thrombosis, or avulsion of the splenic or short gastric arteries or both, elevated intragastric pressure, and reduced cardiac output. The degree of dilatation, and degree and duration of volvulus likely contribute to the risk of gastric and severity of necrosis. Susceptibility of the gastric mucosa to damage by