GDV - PERIOPERATIVE CARE & LITERATURE REVIEW

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October 14th, 2020

Gastric dilatation and volvulus is a rapidly progressive, life-threatening condition. It most commonly occurs in large and giant breed, deep chested, adult dogs, but can effect smaller breeds as well as other species. Male and female dogs are equally represented and the breeds most commonly affected include the German shepherd, great Dane, mixed breed, standard poodle, Labrador retriever, Akita, golden retriever, St. Bernard, and Doberman pinscher. Host factors that have been shown to increase the risk of GDV include increased thoracic-depth-to-width ratio, having an afflicted first-degree relative, and increasing age. Additionally, dogs whose owner's describe them as fearful or nervous have been shown to have an increased risk of GDV when compared to those that are describe as "happy". Management factors that have been associated with an increased risk of GDV include smaller food size particles in great Danes, feeding dry foods containing an oil or fat among the first 4 ingredients, and feeding a large meal once daily. Though many people tout that feeding from an elevated food bowl will decrease the risk of GDV, there is at least one study that indicates feeding from an elevated position may actually be associated with an increase risk. With that being said, no study has actually evaluated the height of the food bowl to a fixed position relative to the height of the dog. Another commonly held belief is that predisposed dogs should be exercise restricted after eating; however, there is no good evidence arguing for or against restricting postprandial activity. Clinical signs are generally acute in onset and progressive in nature. Affected dogs most commonly demonstrate restlessness, non-productive retching, abdominal distension, and lethargy.

The pathophysiology of GDV is not well understood. Gas accumulates in the stomach causing it to distend. Gastric distension not only increases gastric wall pressure but also compresses the caudal vena cava and portal veins. This promotes splanchnic congestion and decreases venous return to the right side of the heart ultimately leading to shock. Increased gastric wall pressure results in ischemia of the gastric mucosa and may result in infarction, ulceration, necrosis, and perforation with peritonitis (especially along the greater curvature). Congestion of the splanchnic vessels leads to breakdown of the gut mucosal barrier, bacterial translocation, sepsis, and disseminated intravascular coagulation (DIC). Volvulus occurs when the distended stomach twists on its long axis and occludes the esophageal hiatus and pylorus. The pylorus and duodenum are displaced ventrally and to the left, across the midline and ending dorsal to the cardia on the left side. A clockwise rotation is most common (when viewed from caudal to cranial with the dog in dorsal recumbency). The spleen is attached to the stomach via the gastrosplenic ligament and the short gastric blood vessels and commonly rotates with the stomach to the right ventral abdomen. This frequently leads to splenic congestion and splenomegaly, splenic infarction and thrombosis, splenic torsion, and significant hemoperitoneum. Reported risk factors for increased mortality associated with GDV include gastric necrosis, splenectomy, requirement for partial gastrectomy and splenectomy, preoperative and post-operative cardiac arrhythmia, and increased duration of clinical signs prior to treatment.

Dogs with GDV are commonly presented in shock and warrant immediate and aggressive stabilization. Before performing diagnostic tests or gastric decompression two large gauge, short length intravenous catheters should be placed. Preference is given to the cephalic or jugular veins as they empty into the cranial vena cava. In GDV the venous return from the caudal portion of the body is impeded; as such, intravenous access in the lateral saphenous vein, which empties into the caudal vena cava, is less optimal. Intravenous fluids are bolused to effect. Three studies evaluating fluid resuscitation using a combination of isotonic crystalloid and hypertonic saline or synthetic colloid compared to isotonic crystalloid alone, have documented reduced hypotension, more rapid cardiovascular stabilization with lesser total fluid volumes, and improved oxygen delivery to tissues (Allen, 1991; Schertel, 1997; Beck 2006). A prospective, unblinded study in 20 dogs with an overall survival rate of 90% compared polymerized stroma-free hemoglobin (HBOC) to 6% hetastarch (HES) in 0.9% saline (Haak, 2012). Each of the enrolled patients initially received 15 mL/kg crystalloid and then were randomized into HBOC and HES groups. Patients then received 5 mL/kg of either HBOC or HES every 10 minutes until resuscitation endpoints were achieved. Patients randomized to the HBOC group reached their resuscitation endpoints faster (12.5 compared to 52.5 minutes) and with less fluid volume (crystalloid – 31.3 compared to 48.1 mL/kg; colloid - 4.2 compared to 18.4 mL/kg) with no difference in adverse effects or mortality. Unfortunately, HBOC's are not currently available. In consideration of these findings, it is not unreasonable to start with 4 mL/kg of 7% hypertonic saline and 20 mL/kg of isotonic crystalloid in GDV patients.

Other recent evidence points to a role for lidocaine administration in GDV patients. A prospective study looked at 83 lidocaine treated dogs compared to 47 untreated, historic controls (Bruchim, 2012). Dogs in the lidocaine treatment group received 2 mg/kg intravenously prior to gastric decompression followed by a 24-hour constant rate infusion (0.05 mg/kg/min). Patients in the lidocaine treatment group had a lower incidence of cardiac arrhythmia (12% compared to

38.3%), lower serum creatinine levels (0 compared to 3.6% of patients with creatinine >2 mg/dL), and shorter hospitalization times (48 compared to 72 hours). While the study only used historic controls, lidocaine is safe and should be considered in these patients.

The extended minimum database should include measurement of packed cell volume, total solids, blood glucose, venous blood gas, electrolytes, plasma lactate, prothrombin time and activated partial thromboplastin time. The packed cell volume and total protein are important to establish baseline values prior to fluid administration. A low total solid measurement may be an early indication of hemoperitoneum. It is important to assess baseline acid base status in these patients as the decreased venous return results in vascular stasis, lactic acid accumulation, and metabolic acidosis. Additionally, as the stomach distends it may also compromise the patient's ventilation and result in a mixed acid base disturbance comprised of both a metabolic and a respiratory acidosis. Correction of acid base and electrolyte abnormalities is important prior to the induction of general anesthesia. In these patients, plasma lactate may provide more objective information with regard to the adequacy of perfusion. While plasma lactate should always be evaluated in conjunction with clinical perfusion parameters such as pulse quality, mucous membrane color, capillary refill time, and temperature (rectal and extremities), it has been shown to have some prognostic value in GDV patients.

A retrospective study evaluating plasma lactate in 102 dogs presented to the emergency room for GDV reported an overall survival rate of 85% (dePapp, 1999). In that study, 99% of dogs with lactate <6.0 mmol/L survived compared with 58% of dogs with lactate >6.0. However, it is important to note that the authors correctly pointed out that a low plasma lactate (<6.0 mmol/L) was a better predictor for survival than a high plasma lactate was a predictor for death. They also suggested that serial measurements of lactate might be more useful than a single measurement drawn at the time of presentation. One decade later, another retrospective study evaluated 64 dogs presented to the emergency room for GDV (Zacher, 2010). Those authors reported an overall survival rate of 77%. This study evaluated plasma lactate clearance by calculating the change in lactate, before and after initial intravenous fluid administration and gastric decompression [(initial plasma lactate – post-resuscitation plasma lactate) / (initial plasma lactate x 100)]. The findings of this study suggest that patients with a plasma lactate clearance >42% are more likely to survive (100%; 30/30 dogs) when compared to patients with a plasma lactate clearance <42% (56%; 19/34 dogs). That being said, a high plasma clearance was again a much better predictor of survival than a low plasma clearance was a predictor of death. A third retrospective study evaluated 84 dogs with GDV and an overall survival rate of 88% (Green, 2011). These authors evaluated the initial lactate as well as lactate clearance in a population of dogs and reported that an initial plasma lactate >6.0 mmol/L was not predictive of gastric wall necrosis or survival to discharge. They also found that 70% of dogs whose plasma lactate decreased by >50% in the initial 12 hours survived to discharge. A more recent retrospective study in 78 dogs presented for GDV with an overall survival rate of 83% reported that an initial plasma lactate >7.4 mmol/L was associated with an increased risk of gastric necrosis and death (Santoro-Beer, 2013). However, the sensitivity and specificity were relatively poor (gastric necrosis Sn=50% and Sp+88%; death Sn=75% and Sp=89%). They also found that the base excess was not useful in predicting either gastric necrosis or outcome. In conclusion the most promising data appears to be evaluation of lactate clearance after initial resuscitation; however, one should exercise caution in applying any of these results to individual patients as there is wide overlap in plasma lactate ranges among survivors and non-survivors.

An older prospective study evaluated coagulation indices in 20 dogs presented for GDV (Millis, 1993). In that study 75% of dogs survived. Coagulation parameters that were measured included platelet count, prothrombin time, activated thromboplastin time (or activated clotting time), fibrinogen, antithrombin activity, and fibrin degradation products or d-dimers. Seven of 10 dogs with 2 or more abnormal hemostatic test results had gastric necrosis as compared to zero of the remaining 10 dogs with less than 2 abnormal hemostatic test results. Given that dogs with GDV are predisposed to DIC and treatment requires surgical intervention, it is reasonable to monitor coagulation indices in these patients. More recently investigators evaluated the usefulness of plasma myoglobin as a prognostic biomarker in dogs presented with GDV (Adamik, 2009). Dogs in this study had an overall survival rate of 79.2%. Plasma myoglobin was measured at the time of diagnosis (prior to resuscitation), and again 24 and 28 hours post-operatively. While a low myoglobin level (<168 ng/mL) at the time of diagnosis was associated with an 88.9% survival rate, the low sensitivity and specificity preclude its usefulness as a prognostic biomarker in these patients.

There are a couple of newer studies looking at inflammatory mediators in dogs with GDV. In one, C-reactive protein and high mobility group box 1 (HMGB1) were evaluated (Uhrikova, 2015). Of these 2 parameters, initial HMGB1 concentration showed more potential as an indicator of poor outcome. HMGB1 is an alarmin, a protein that is released extracellularly during cell necrosis but not apoptosis; it is also a late cytokine with active secretion after a lag phase of 8-18 hours. Outside the cells, it triggers inflammation via stimulation of proinflammatory cytokine production by monocytes/macrophages, increased expression of adhesion molecules, and increased epithelial permeability. Though further research on the uses of HMGB1 assessment in dogs is warranted, it's usefulness in dogs with GDV will be limited until a point-of-care HMGB1

assay becomes available. In a more recent study, cell-free DNA, HMGB1, and procalcitonin (PCT) was evaluated prospectively in dogs with GDV (Troija, 2018). Not surprisingly, dogs with GDV had significantly greater median plasma concentrations of all three biomarkers. Interestingly, the authors also identified a moderate, positive correlation between plasma PCT and blood lactate levels. Though PCT was prognostic for non-survival in the dogs with GDV, like HMGB1, it will not be a useful prognostic test until a point-of-care assay becomes commercially available.

After instituting medical therapy and cardiovascular stabilization, abdominal radiographs are indicated to differentiate between GDV and gastric distension without volvulus or other causes of acute abdomen. The views of choice are a right lateral and an orthogonal view. The right lateral radiograph frequently reveals a double bubble gas pattern with compartmentalization signs. Other radiographic findings might include free air within the abdomen. Free air is most readily seen between the liver and the diaphragm and may indicate gastric necrosis and or stomach rupture. Gastric pneumotosis is thought to represent gas dissection of the gastric mucosa and may represent gastric necrosis. One study reported a 41% chance that a dog with GDV and gastric pneumotosis will require gastric resection as compared to 35% of dogs without gastric pneumotosis (Fischetti, 2004). Given that many dogs with GDV are older, it is not unreasonable to take thoracic radiographs. A retrospective study evaluating the utility of thoracic radiography in 101 dogs with GDV and overall survival rate of 84% supported the notion of taking thoracic radiographs in dogs with GDV prior to surgery (Green, 2012).

Dogs with GDV should have gastric decompression performed after fluid therapy has be instituted. Decompression can be accomplished via passage of an orogastric tube, or gastrocentesis. In a retrospective study evaluating the method of decompression in 116 dogs with GDV and an overall survival rate of 95.7% found no significant difference between the two methods (Goodrich, 2013). In some dogs it may actually be beneficial to decompress via both methods. Ideally, surgery should be performed as soon as the patient is hemodynamically stable. Prompt intervention may minimize gastric necrosis and or splenic congestion or thrombosis. Anesthetics that minimize arrhythmogenic potential and hypotension should be considered.

Post-operative monitoring should include vital signs and blood pressure every 1-4 hours until the patient is completely stable. An extended data base should also be evaluated (packed cell volume, total solids, electrolytes, and blood gas analysis) 1-4 times a day until the animal is stable and on an upward trend. Blood urea nitrogen and creatinine along with blood glucose, platelet count and other coagulation parameters are monitored to permit early detection of complications such as sepsis, oliguria (usually due to inadequate fluid therapy) or DIC. These patients are also monitored closely for post-operative arrhythmia recognizing that ventricular arrhythmias are common especially 12-36 hours postoperatively. These arrhythmias may result from subendocardial necrosis secondary to decreased cardiac output, hypotension, and poor coronary perfusion and usually resolve within 72 hours. These patients require intensive care and frequent examination to optimize rapid identification of life-threatening complications. Post-operative administration of broad spectrum antibiotics is not generally indicated and are reserved for patients that may be septic. Most of these patients will benefit by having oral food and water reintroduced 12-24 hours post-operatively.

Gastric dilatation volvulus is a common surgical emergency and reported mortality rates have decreased significantly over the last 2-3 decades. Improved pre-operative stabilization with aggressive and early goal directed therapy is likely the most significant contributing factor to explain the decrease in mortality.

References:

Adamik, K.N., Burgener, I.A., Kovacevic, J., et al. (2009). Myoglobin as a prognostic indicator for outcome in dogs with gastric dilatation-volvulus. *Journal of Veterinary Emergency and Critical Care*, 19(3):247–53.

Allen, D.A., et al., (1991). Hypertonic saline/dextran resuscitation of dogs with experimentally induced gastric dilatation-volvulus shock. *American Journal of Veterinary Research*, 52(1):92–6.

Beck, J.J., et al., (2006). Risk factors associated with short-term outcome and development of perioperative complications in dogs undergoing surgery because of gastric dilatation-volvulus: 166 cases (1992–2003). *Journal of the American Veterinary Medical Association*, 229(12):1934–1939.

Bell JS. (2014). Inherited and predisposing factors in the development of gastric dilatation volvulus in dogs. *Topics in Compan An Med*. 345:60-63.

Brockman, D.J., Washabau R.J., Drobatz K.J. (1995). Canine gastric dilatation/volvulus syndrome in a veterinary critical care unit: 295 cases (1986-1992). *Journal of the American Veterinary Medical Association* 207(4):460–64.

Brockman, D.J., Holt D.E. (2000). Management protocol for acute gastric dilatation-volvulus syndrome in dogs. *Compendium on Continuing Education for the Practicing Veterinarian* 22(11):1025–28.

Brourman, J.D. et al. (1996). Factors associated with perioperative mortality in dogs with surgically managed gastric dilatation-volvulus: 137 cases (1988-1993). *Journal of the American Veterinary Medical Association*, 208(11):1855–8.

Bruchim Y., Kelmer E. (2014). Postoperative management of dogs with gastric dilatation and volvulus. *Topics in Companion Animal Medicine* 29(3):81–85.

Buber T., Saragusty J., Ranen E., et. al. (2007). Evaluation of lidocaine treatment and risk factors for death associated with gastric dilatation and volvulus in dogs: 112 cases (1997-2005). *Journal of the American Veterinary Medical Association* 230(9):1334–39.

de Papp E.K., Drobatz K.J., Hughes D. (1999). Plasma lactate concentration as a predictor of gastric necrosis and survival among dogs with gastric dilatation-volvulus: 102 cases (1995-1998). *Journal of the American Veterinary Medical Association* 215(1):49–52.

Fischetti, A.J., Saunders H.M., Drobatz K.J. (2004). Pneumatosis in canine gastric dilatation-volvulus syndrome. *Veterinary Radiology and Ultrasound* 45(3):205–9.

Glickman L.T., Glickman N.W., Perez C.M., et al. (1994). Analysis of risk factors for gastric dilatation and dilatation-volvulus in dogs. *Journal of the American Veterinary Medical Association* 204(9):1465–71.

Glickman L.T., Glickman N.W., Schellenberg D.B, et al. (2000). Incidence of and breed-related risk factors for gastric dilatation-volvulus in dogs. *Journal of the American Veterinary Medical Association* 216 (1): 40–45.

Glickman L.T., Glickman N.W., Schellenberg D.B., et al. (2000). Non-dietary risk factors for gastric dilatation-volvulus in large and giant breed dogs. *Journal of the American Veterinary Medical Association* 217 (10): 1492–99.

Goodrich Z.J., Powell L.L. Hulting K.J. (2013). Assessment of two methods of gastric decompression for the initial management of gastric dilatation-volvulus. *Journal of Small Animal Practice*, 54(2):75–79.

Green T.I., Tonozzi C.C., Kirby R., Rudloff E. (2011). Evaluation of initial plasma lactate values as a predictor of gastric necrosis and initial and subsequent plasma lactate values as a predictor of survival in dogs with gastric dilatation-volvulus: 84 dogs (2003-2007). *Journal of Veterinary Emergency and Critical Care* 21(1):36–44.

Haak C.E., Rudloff E., Kirby R. (2012). Comparison of Hb-200 and 6% hetastarch 450/0.7 during initial fluid resuscitation of 20 dogs with gastric dilatation-volvulus. *Journal of Veterinary Emergency and Critical Care*, 22(2):201–210.

Mackenzie G., Barnhart M., Kennedy S., et. al. (2010). A retrospective study of factors influencing survival following surgery for gastric dilatation-volvulus syndrome in 306 dogs. *J Am Anim Hosp Assoc* 46 (2): 97–102.

Millis D.L., Hauptman J.G., Fulton R.B. (1993). Abnormal hemostatic profiles and gastric necrosis in canine gastric dilatation-volvulus. *Veterinary Surgery:*22(2):93–97.

Mooney E., Raw C., Hughes D. (2014). Plasma lactate concentration as a prognostic biomarker in dogs with gastric dilation and volvulus. *Topics in Companion Animal Medicine* 29(3):71–76.

Raghavan M., Glickman N.W., Glickman L.T. (2006). The effect of ingredients in dry dog foods on the risk of gastric dilatation-volvulus in dogs. *Journal of the American Animal Hospital Association* 42(1):28–36.

Raghavan M., Glickman N.W., McCabe G., et al. (2004). diet-related risk factors for gastric dilatation-volvulus in dogs of high-risk breeds. *Journal of the American Animal Hospital Association* 40(3):192–203.

Santoro-Beer K.A., Syring R.S., Drobatz K.J. (2013). Evaluation of plasma lactate concentration and base excess at the time of hospital admission as predictors of gastric necrosis and outcome and correlation between those variables in dogs with gastric dilatation-volvulus: 78 cases (2004-2009). *Journal of the American Veterinary Medical Association* 242(1):54–58.

Schertel, E.R. et al., (1997). Evaluation of a hypertonic saline-dextran solution for treatment of dogs with shock induced by gastric dilatation-volvulus. *Journal of the American Veterinary Medical Association*, 210(2):226–30.

Sharp C.R., Rozanski E.A. (2014). Cardiovascular and systemic effects of gastric dilatation and volvulus in dogs. *Topics in Companion Animal Medicine* 29(3):67–70.

Troija R., Giunti M., Calipa S., Goggs R. (2018). Cell-free DNA, high-mobility group box-1, and procalcitonin concentrations in dogs with gastric dilatation-volvulus syndrome. *Frontiers in Veterinary Science*, 5(67):1-8.

Uhrikova I, et al., (2015). C-reactive protein and high mobility group box 1 in dogs with gastric dilatation and volvulus. *Journal of Veterinary Emergency and Critical Care* 25(4):488–494.

Winkler K.P., Greenfield C.L., Schaeffer D.J. (2003). Bacteremia and bacterial translocation in the naturally occurring canine gastric dilatation-volvulus patient. *Journal of the American Animal Hospital Association* 39(4):361–68.

Zacher L.A., Berg J., Shaw S.P., Kudej R.K. (2010). Association between outcome and changes in plasma lactate concentration during presurgical treatment in dogs with gastric dilatation-volvulus: 64 cases (2002-2008). *Journal of the American Veterinary Medical Association* 236(8):892–97.

Zatloukal J.M., Lexmaulová C.L., Nečas A., Fichten T. (2005). Gastric dilatation-volvulus syndrome: outcome and factors associated with perioperative mortality. *Acta Veterinaria Brno*.