“Zero’s Twisted Times”

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Introduction:

Gastric dilatation-volvulus (GDV) is a rapidly progressive disease that is considered a medical and surgical emergency in dogs.[1, 2] This disease occurs most commonly in large and giant breed, deep chested dogs (i.e. Labrador retrievers, German Shepherd Dogs, Great Danes, Huskies, Standard Poodles, etc.).[2] GDV is defined as gas accumulation in the stomach and rotation around the short axis of the stomach. Gastric rotation causes occlusion of gastric vasculature and gastric outflow, compression of the great vessels which often leads to several systemic changes, including but not limited to hypovolemic shock, endotoxic shock, metabolic acidosis, and respiratory abnormalities, which can all lead to a rapid decline if not treated promptly.[3] There are several risk factors associated with GDV development, however the etiology continues to be unclear at this time.[4] Some of the risk factors associated with GDV include prolonged gastric emptying times, gastric motility abnormalities, eating large meals once daily, and consuming meals too quickly. The following case report will review the presentation, clinical signs, pathophysiology, diagnostic approach, treatment, management strategies, and prognosis of a classic GDV case.

History and Presentation:

Zero was an approximately 5-year-old male intact German Shepherd who presented to Mississippi State University Small Animal Emergency Services on June 27, 2020 for non-productive retching and abdominal distension. The day before presentation, Zero had been pursuing the family's intact female dog who was in heat and he was eventually trapped in a pen with her. The morning of June 27, 2020, Zero rapidly consumed a large amount of water after being let out of the pen. Zero's owner found him under the deck later that day. He noticed that

Zero was not able to keep water down and was panting. The dog was not on any medications at that time, and he was up to date on vaccinations.

On physical examination, Zero was depressed, but responsive. He weighed 32.4 kg (71.3 pounds) and had a body condition score of 5/9. He was tachycardic with a heart rate of 176 beats per minute, tachypneic with respiratory rate of 64 breaths per minute, and had a temperature of 100.3˚F. On cardiothoracic auscultation, there were no murmurs, arrhythmias, wheezes, or crackles appreciated. His femoral pulses were snappy but synchronous with his heartbeat. His mucous membranes were pink and slightly tacky with a capillary refill time of less than 2 seconds. His peripheral lymph nodes palpated small, soft, and symmetrical. His abdomen was moderately distended and painful on palpation. His hair coat was covered in red clay/mud and fleas were found near the tail base. A brief neurological exam was performed and was within normal limits.

Pathophysiology:

As stated earlier, gastric dilatation-volvulus is a common and severe disease that occurs in dogs. At this time, it is unclear whether the dilation or volvulus happens first in this disease process.[3] One theory is that dilation of the stomach happens first and is worsened by aerophagia, leading to volvulus. Another theory is that volvulus happens first due to gastric motility abnormalities, causing pyloric obstruction and subsequent dilation. Generally, when volvulus occurs, the pylorus moves from the right cranial abdomen ventrally around the fundus of the stomach and rests in the left abdomen beside the esophagus.[5] The rotation of the stomach and subsequent displacement from its normal anatomical position causes obstruction of the esophagus and pylorus, resulting in prevention of gastric emptying. Gas production continues due to carbohydrate metabolism and continued production of gastric products, which leads to retention

of H^+ ions and subsequent metabolic alkalosis.[5] The spleen can be involved in the volvulus due to the stomach pulling the spleen via the gastrosplenic ligament and short gastric arteries as it rotates. If the spleen is involved, it can become partially obstructed and undergo venous congestion, leading to splenic enlargement.

As the pressure in the stomach begins to increase due to dilation, several severe systemic effects can occur. The respiratory impairments due to increased pressure on the diaphragm include decreased respiratory output and decreased perfusion to the lungs via pulmonary vasculature, leading to hypoxia and hypercapnia. Respiratory acidosis is often present but can be disguised by the mixed acid-base disorder created by other aspects of GDV (metabolic acidosis created by increased lactate vs. metabolic alkalosis created by sequestration of H^+ ions in the gastric lumen).[5] The cardiovascular effects include decreased venous return to the heart, decreased cardiac output, and decreased perfusion to abdominal organs due to compression of abdominal vasculature. These cardiovascular effects lead to hypovolemic shock and cardiac arrhythmias such as ventricular premature contractions (VPCs) and ventricular tachycardia. When the stomach rotates, it can cause overall decreased perfusion to the gastrointestinal tract, and other vital organs including kidneys, spleen, and brain if severe enough to affect overall perfusion. As previously stated, the metabolic effects include metabolic alkalosis due to H^+ ion retention, and metabolic acidosis due to hypercapnia, cellular hypoxia, and increased blood lactate levels. Since lactic acid is a product of anaerobic metabolism, lactate levels rise and exceed the rate at which lactic acid can be cleared by the liver and kidneys.[5] Endotoxemia can occur due to ischemic injury to the gastrointestinal tract, causing translocation of bacteria and endotoxin.[4] Reperfusion injury occurs when oxygenation and perfusion of tissue is restored following an ischemic insult.[4] Once the GDV is corrected via decompression and/or surgery, oxygen and adequate blood flow

return to the gastrointestinal tract. The reintroduction of oxygen to the ischemic areas of the gastrointestinal tract causes release of free radicals, leading to cellular damage, microvascular dysfunction, and apoptosis.[4] It is imperative to understand these potential systemic effects in GDV cases in order to accurately detect early complications, initiate early intervention, therefore maximizing the chance of survival in these patients.

Diagnostic Approach:

Often times, the diagnosis of GDV is based on signalment/history, presentation, and clinical signs. Initial diagnostics include obtaining a quick history, vital parameters, and a physical exam including abdominal palpation. Due to the likelihood that the patient is in hypovolemic shock when it presents, it is important to address the shock first by placing two large bore intravenous cephalic catheters, collecting a blood sample for packed cell volume/total protein (PCV/TP), lactate, and glucose level checks, and administering shock doses of intravenous crystalloid and/or colloid fluids. The goals of fluid resuscitation are rapid expansion of the intravascular volume, maintenance of intravascular volume over time, and provision of adequate hydration at the cellular level. [6] During the administration of fluids, it is important to monitor the patient's heart rate, blood pressure, and pulse strength to ensure that the fluids are correcting the hypotension and not causing fluid overload, which can lead to detrimental effects including extravasation of fluids into interstitium and vital organs including the brain, heart, and lungs. While attempting to stabilize the patient, it is recommended to perform additional diagnostics, including general bloodwork (CBC/chemistry) and radiographs, to help improve the prognosis. When preforming radiographs, the only view needed for an accurate diagnosis of GDV is a right lateral radiograph. This is due to the pathognomonic appearance GDV has on a radiograph in this position. Radiographic findings include severe gas/fluid distension of the stomach, dorsal

displacement of the pylorus, and/or compartmentalization of the stomach. If these radiographic findings are seen, treatment should be initiated immediately. An exception to the one radiographic view rule, however, is a 360-degree gastric volvulus in which the pylorus and fundus are in their normal positions, causing it to appear as gastric dilation without volvulus.[7] If this is the case, a left lateral projection to identify the location and effect on the pylorus is indicated, as well as potentially a ventral/dorsal (VD) or dorsal/ventral (DV) projection. Normally, the pylorus fills with fluid and the fundus or body of the stomach fills with gas on a right lateral projection, however in a GDV case, the opposite occurs.

Upon Zero's arrival and after a history was obtained, two cephalic intravenous large bore catheters were placed. Zero received 30 mL/kg bolus of Lactated Ringer's Solution and a 0.2 mg/kg intravenous injection of methadone for sedation and analgesia. Right lateral radiographs were obtained and confirmed the diagnosis of GDV based on severe gastric distension with dorsal displacement of the pylorus. Zero's stomach was decompressed using an 18-gauge catheter immediately following radiographs. A CBC was preformed and revealed leukocytosis of 24,000/ul (5,000-14,200/ul). A chemistry panel was preformed and revealed mild azotemia due to an elevated blood urea nitrogen (BUN) of 42 mg/dl (8-24.0 mg/dl) and an elevated creatinine of 2.26 mg/dl (0.5-1.4 mg/dl). A blood lactate was performed and an revealed hyperlactatemia due to an elevated lactate of 1.5 mmol/L (0.5-1 mmol/L). Zero received another 30 mL/kg bolus of PlasmaLyte and a 1 mg/kg intravenous injection of maropitant citrate to prevent vomiting and was then prepped for surgery.

Treatment and Management:

Due to the severity of most GDV cases, diagnostic testing and treatment protocols generally occur simultaneously. Once an accurate diagnosis has been made, gastric decompression followed by surgical correction of the volvulus should be pursued. Gastric decompression can occur via an orogastric tube, which requires heavy sedation and analgesia, or general anesthesia. Another method of gastric decompression is by gastric trocarisation with a 16–18-gauge needle or catheter. When determining the appropriate location to perform the trocarization, it is recommended to either use an ultrasound to find the location of the spleen and avoid it or find the most tympanic region of the stomach and trocarize there. The skin will need to be clipped and prepped appropriately before the trocarization occurs. Gastric decompression improves chances of survival by alleviating compression of the great vessels, which improves systemic and gastric perfusion, and aids in improving the patient's status until surgery occurs. Definitive surgical management of gastric dilatation volvulus involves gastric repositioning, gastric resection when indicated, surgical formation of a permanent adhesion to prevent recurrence of the problem, and assessment of the spleen with the splenectomy if indicated.[8] Surgical correction consists of completely decompressing the stomach, rotating the stomach back into the normal anatomical position, and suturing the stomach to the body wall as a prevention measure for reoccurrence. In one study, median survival times in dogs that experienced GDV and were treated without gastropexy is 188 days compared to 547 days for dogs treated with gastropexy at the time of GDV.[9] The entire gastrointestinal tract should be assessed for any permanent damage or evidence of disease, which will need to be addressed accordingly. The spleen and associated vessels are assessed for adequate pulses and perfusion. If no pulses are detected or the short gastric arteries are ruptured, the spleen is removed.

Once surgery is complete, the patient will need to receive proper post-operative management and continuous monitoring for any adverse complications. Therapy in the postoperative period is focused on maintaining tissue perfusion along with intensive monitoring for prevention and early identification of ischemia-reperfusion injury (IRI) and consequent potential complications such as hypotension, cardiac arrhythmias, acute kidney injury (AKI), gastric ulceration, electrolyte imbalances, and pain.[10] If cardiac arrhythmias (most commonly VPCs) occur post-operatively, intravenous lidocaine is indicated as it plays a central role in treating VPCs, however when used to treat ischemic reperfusion injury, no significant differences were detected in the mortality rate or postoperative complications between treated and untreated dogs, and hospital stays were significantly longer in treated dogs.[6] The most serious complications of GDV are associated with IRI and consequent systemic inflammatory response syndrome, multiple organ dysfunction syndrome, and death.[10]

Once Zero was diagnosed with GDV, he was promptly prepped for surgery. He was anesthetized and positioned in dorsal recumbency. The abdomen was clipped, aseptically prepared with 4% chlorhexidine solution, followed by alcohol and duraprep. The abdomen was aseptically draped. An approximately 50 cm ventral midline skin incision was made extending from the xyphoid to several centimeters cranial to the prepuce using a #10 scalpel blade. The subcutaneous tissue was dissected with Metzenbaum scissors and the linea alba visualized. Monopolar electrocautery was employed for hemostasis. The linea alba was tented and incised with a #10 scalpel blade. The incision was then extended with a #10 scalpel blade and debakey forceps to match the length of the skin incision. The falciform fat was excised with monopolar cautery.

The stomach was distended and covered by omentum, and gastric volvulus in addition to the dilation was confirmed (180-degree clockwise twist). An 18 gauge needle was inserted into the

body of the stomach and attached to the suction to deflate the stomach and allow for derotation. Once sufficiently deflated, the stomach was derotated by locating and grasping the pylorus on the left side of the abdomen and gently pulling it ventrally and to the right of midline, while the gastric body was gently pushed dorsally, to return it to its normal anatomical position. An orogastric tube was passed into the stomach and ingesta and gas were expelled from the tube, facilitating additional gastric decompression. The abdominal wall was lined with moistened laparotomy sponges and a balfour retractor was placed to improve visualization. The orogastric tube was kinked and removed. A complete abdominal explore was performed. The stomach was pink and had adequate pulses. The spleen was diffusely and mildly enlarged, of normal color and consistency, and had good pulses throughout. The remainder of abdominal contents were unremarkable.

An incisional gastropexy was then performed. An approximately 4 cm incision was made in the seromuscular layer in the pyloric region of the stomach with a #15 blade. Additionally, an incision was made in the right ventrolateral abdominal wall caudal to the last rib through the peritoneum and the transversus abdominus muscle. The abdominal incision was sutured to the edge of the gastric incision using two lines of simple continuous sutures with 2-0 PDS. The abdomen was copiously lavaged and adequate hemostasis was confirmed.

The linea alba was closed in a simple continuous pattern with 0 PDS. Subcutaneous tissue was apposed in a simple continuous pattern with 2-0 Monocryl, and the skin was apposed with 2-0 Monocryl using an intradermal suture pattern. The anesthetic event and immediate recovery were unremarkable. Zero was continued on a fentanyl CRI following surgery for pain management. He was monitored via ECG overnight for ventricular premature contractions (VPCs).

Case Outcome:

Zero initially recovered well following surgery. He was transitioned from intravenous pain management to oral pain management the day following his surgery. He was receiving a fentanyl constant rate infusion (CRI) of 3 mcg/kg/hr and was transitioned to oral acetaminophen/codeine 300mg/30mg (Tylenol #4) at 1.6 mg/kg every eight hours. Zero was doing well after surgery for a day or so, but he was not returning to his usual self despite supportive care. Two days postoperatively, he developed hemoglobinuria, hemoglobinemia, anemia, thrombocytopenia, and ascites. An abdominal ultrasound was performed and suggested that the spleen had multiple microthrombi and was not receiving adequate blood flow.

Zero was brought back into surgery on July 1, 2020 to remove his spleen. A Jackson Pratt drain and an esophagostomy tube were placed at this time to facilitate recovery. Zero recovered uneventfully following this second procedure. He continued to improve postoperatively and at the time of discharge was eating readily, with normal vitals, and was comfortable on oral medications. Zero was discharged 4 days following his second procedure.

Conclusion:

While GDV is considered a medical and surgical emergency, it can be manageable if identified early. It is important to educate clients ahead of time of risk factors associated with GDV, such as breed predispositions and appropriate feeding frequency and amount, and prevention strategies for GDV. Long term, dietary management will likely include multiple small meals (2-3) per day rather than a single large meal and continued monitoring for recurrence of clinical signs.[2] The most effective prevention strategy is performing a prophylactic gastropexy. In one study, of the dogs at risk for development of gastric dilatation volvulus, dogs that underwent prophylactic gastropexy had a 29-fold decrease in the mortality rate compared to dogs that had not had gastropexy.[6] There are several different methods to performing a gastropexy, including

incisional, circumcostal, belt-loop, incorporating, and gastrocolopexy. In one study, the reoccurrence rate of GDV after circumcostal gastropexy is as high as 9%.[9] In another study, belt-loop gastropexy was evaluated, and recurrence was not reported in any dogs.[6] In a study by Przywara et al., no occurrences of gastric dilatation volvulus were noted in dogs after therapeutic or prophylactic incisional gastropexy; however, there are individual reports of gastric dilatation volvulus is dogs that have previously undergone incisional gastropexy.[6]

When a patient presents with a history and clinical signs similar to those of a GDV, it is important to recognize them and act promptly in confirming the diagnosis to increase the chances of survival in these cases. The morbidity and mortality rates associated with GDV increase with the severity of disease and the amount of time from initial onset to treatment.[3] The mortality rates for GDV are reported to be as high as 10-25%.[3] Factors that have been associated with increased mortality include the duration of clinical signs for more than 6 hours, concurrent gastrectomy or splenectomy, and the presence of hypotension, gastric necrosis, preoperative cardiac arrhythmias, peritonitis, sepsis, or disseminated intravascular coagulation.[11] Prognostic indicators such as recumbency, depression or coma on presentation, gastric necrosis, and arrhythmia all increase mortality; thus, the most effective means of reducing mortality is by prophylactic gastropexy.[3] The decrease in mortality presumably reflects the time spent stabilizing the patient with aggressive fluid support and gastric decompression. This is critical and positively impacts survival.[5] A key element to successful treatment of this syndrome appears to be prevention or early treatment of hypotension because hypotension at any time was a significant risk factor for death.[11] Therefore, the prognosis for dogs with GDV appears to be favorable if medical and surgical treatment is instituted as soon as possible.[11]

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