Daisy's Big Twist

Canine Gastric Dilatation and Volvulus

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Introduction

Gastric dilation and volvulus or GDV is an acute, life-threatening condition that can be fatal without emergent intervention. It is commonly referred to as "bloat" and is characterized by a gas-distended stomach that rotates anywhere from 90 degrees to 360 degrees on its mesenteric axis. This phenomenon leads to a large, tympanic stomach that causes compression and occlusion of major abdominal vessels resulting in respiratory and cardiovascular compromise; other common sequelae often seen include fluid, electrolyte, and acid-base imbalances, as well as, cardiac arrhythmias[1].

GDV is primarily known as an inherited disorder seen in dogs with certain genetic predisposing factors such as type of breed, temperament, and anatomical characteristics[2]. However, there are also many environmental and pathogenic risk factors such as stress, diet, physiologic condition, and trauma that are suspected to play a role in the development of GDV[1]. Generally, middle-aged to older large and giant breed dogs, such as German Shepherds, Great Danes, Standard Poodles, Golden Retrievers, and some mixed breeds are the most frequent patients that present to emergency centers for treatment of GDV[2]. Other non-hereditary factors that have been correlated, although not entirely proven, to predispose GDV episodes include nervous and fearful temperaments, stressors such as kennels and car rides, aerophagia, gastric foreign bodies, inflammatory bowel disease, anesthesia and surgery, and parturition. Dietary components such as rapid ingestion of food, eating fewer meals per day, consuming large volume meals, elevating food bowls, ingesting food with high fat concentrations, and exercising after eating are commonly proposed associated factors but remain controversial[1].

Dogs with GDV often present with a progressively distending, painful abdomen, non-productive retching, signs associated with shock, and hypersalivation. Diagnosis is always

confirmed with abdominal radiographs, particularly a right lateral view. Once confirmed, treatment consists of medical stabilization, gastric decompression, and surgical correction. In general, prognosis is typically guarded with a high mortality rate around 45%. Early diagnosis and timely treatment may improve a patient's prognosis, but ultimately remains only fair. Complications such as gastric necrosis, perforation, sepsis, or disseminated intravascular coagulation often result in a much poorer prognosis[3].

History and Presentation

Daisy is a thirteen-year-old, female, Tree Walker Coonhound that presented to the Animal Emergency and Referral Center (AERC) on May 18, 2019, for an acute onset of lethargy with abnormal mentation and behavior. The owner noted Daisy to first appear abnormal shortly after dinner time, when she found her standing stiff, not responding to visual or verbal cues, and "staring into space." She appeared very uncomfortable and stood with a hunched posture, but no vomiting or retching was noted. Having no previous medical concerns or incidents, Daisy's owners immediately rushed her to the referral center, thinking she may have had a stroke.

On presentation, Daisy was alert but extremely depressed and very slow to respond. Her status was deemed unstable, and primary triage was immediately initiated. Daisy's rectal temperature and respiratory rate were within normal limits, and her respiratory effort was slightly increased. She was severely tachycardic (188 bpm) with weak femoral pulses. She was estimated to be 7-8% dehydrated with light pink mucous membranes and a capillary refill time greater than two. Noninvasive blood pressure measurement was low-normal (101/83 (89) mmHg), and electrocardiogram assessment indicated sinus tachycardia with no arrhythmias noted. Daisy's abdomen appeared mildly distended with palpation revealing a large spleen and suspected mass effect in the cranial abdomen. Abdominal FAST scan revealed no free fluid but was able to

identify a distended, gas-filled stomach and an enlarged spleen with rounded margins and stagnant blood supply within the splenic vessels, as revealed by the color flow doppler. A right lateral abdominal radiograph revealed moderate to severe gastric distention with dorsal displacement of the pylorus, confirming an acute gastric dilation and volvulus.

A peripheral blood sample was collected for hematological diagnostics. Hematocrit tubes were used to gather ancillary results such as packed cell volume (52%) and total solids (6.4 g/dL), and a lactate meter was used to measure lactate concentration (5.8 mmol/L). A complete blood count (CBC) identified severe thrombocytopenia (26 K/uL) with no other remarkable findings. To confirm the quality of the sample and findings previously reported, a second blood sample was collected and analyzed. Daisy's platelet count was moderately higher (72 K/uL) than the previous platelet count, and her hematocrit was mildly decreased (36.8%). With Daisy's crucial need for surgical correction, a manual platelet count was performed which confirmed severe thrombocytopenia (60 K/uL). Remarkable findings on the chemistry panel included the following: mild hypocalcemia (7.3 mg/dL), moderate hypoproteinemia (3.8 g/dL), moderate hypoalbuminemia (1.9 g/dL), moderate hypoglobulinemia (1.9 g/dL), moderately decreased alkaline phosphatase (<10 U/L), mild cholesterolemia (94 mg/dL), moderately decreased amylase (296 U/L), and potassium was low-normal (3.6 mmol/L).

Given these diagnostic findings and assessment, it was confirmed that Daisy had a gastric dilatation and volvulus. Stabilization and surgical correction were discussed with the owner, and it was elected that Daisy would undergo surgery once she was stabilized. Concurrently, an 18-gauge intravenous catheter was placed in her left cephalic vein, as well as, an additional catheter placed in her right saphenous vein. She was bolused one liter of Plasmalyte fluids with an additional 600 milliliter bolus administered just prior to anesthetic induction. While receiving

fluids, ultrasound-guided trocarization of the stomach was performed with a 14-gauge catheter to decompress her stomach. Prepping of the surgical site was also initiated, in order to limit time under general anesthesia. Daisy's cardiovascular system was deemed stable, and she was rushed to the operating room where she was induced under general anesthesia.

Pathophysiology

GDV has a wide array of effects on body systems and is often associated with many secondary complications; therefore, it is very crucial to understand the pathophysiology involved in this syndrome. Many factors are involved in causing GDV, but gastric distention is the primary initiating factor that leads to volvulus and many other consequences[4]. Most gas within the stomach accumulates due to aerophagia that can occur with rapid eating, hyperventilation, and esophageal motility disorders[5]. It is postulated that swallowed air can pass through the twisted gastroesophageal junction into the stomach but cannot escape from the stomach[1]. Gastric hypomotility, ingesting large volumes of food and water, and pyloric outflow obstruction have been implicated as causes of gastric distention as well[5]. The accumulated air is unable to be adequately relieved through eructation, vomiting, or passing into intestines which leads to gastric distention[4]. Impaired eructation can be seen in deep-chested dogs due to anatomical or functional abnormalities of the gastroesophageal junction[5]. As air continues to accumulate, the gastric walls begin to stretch, and dilatation pursues frequently leading to volvulus of the distended stomach. Gastric distention creates tension within the gastric wall, which predisposes ischemia and necrosis, though infarction is most common along the greater curvature of the stomach where the short gastric artery provides blood supply [4].

The interrelationship between gastric distention and volvulus is poorly understood, but fundamentally, volvulus is understood to be caused by increased mobility of the stomach within

the abdomen. The most commonly associated predisposing factor to increased mobility of the stomach is from stretching or laxity of the hepatogastric and gastroduodenal ligaments, as seen with middle to older aged large and giant breed dogs[1]. The gastric ligaments are also known to stretch with gastric distention caused by delayed gastric emptying. Generally, the stomach rotates in a clockwise direction, as viewed caudal to cranial with the dog in dorsal recumbency. The rotation is most often 220 to 270 degrees, but it can range anywhere from 90 to 360 degrees. As twisting of the stomach occurs, the pylorus and duodenum move ventrally under the stomach and to the left of midline, eventually stopping dorsal to the cardia on the left aspect of the abdomen. Splenic displacement and torsion are often seen with volvulus of the stomach. Because the spleen is attached to the greater curvature of the stomach, rotation of the stomach causes stretching of the gastrosplenic ligament leading to right dorsal displacement of the spleen. Once displaced, splenomegaly, congestion, and possible ischemia can occur due to occluded or infarcted blood supply. Along with ischemia, endotoxins from the GI tract begin to accumulate and result in endotoxemia which can lead to activation of the coagulation cascade and disseminated intravascular coagulation[1].

Gastric dilatation and volvulus has detrimental systemic effects on the body in addition to gastrointestinal compromise. Cardiovascular dysfunctions such as shock, cardiac arrhythmias, and myocardial dysfunction are the primary systemic side effects seen, and they are thought to contribute to the early morbidity and mortality associated with GDV. Obstructive shock is ultimately caused by severe gastric dilatation which decreases venous blood return to the heart leading to low stroke volume and cardiac output. Intravascular fluid volume can potentially be lost due to abdominal hemorrhage, third spacing, and loss into the GI tract, all of which create hypovolemic shock. Myocardial dysfunction and ventricular arrhythmias are manifested as

sequalae of global shock in which the patient may be experiencing. Ventricular arrhythmias are the most common cardiovascular derangement and occur in approximately 40% of GDV cases. Dogs with GDV present most frequently for experiencing signs associated with respiratory and cardiovascular compromise such as tachycardia, weak pulses, and depressed mentation while in decompensated shock. Depending on the state of compensation, capillary refill time may be rapid to prolonged and mucous membranes may be pale or injected. Gastric dilatation often causes respiratory compromise due to inhibiting normal respiratory excursion. The dilated stomach and increased intraabdominal pressure limit thoracic volume and range of movement for the diaphragm which leads to hypoventilation and ventilation-perfusion mismatching; in addition, decreased cardiac output also leads to ventilation-perfusion mismatching[6].

GDV can cause a wide range of metabolic derangements which can be severe if prolonged. Metabolic acidosis results from decreased circulating volume, arterial hypoxemia, and lactic acidosis. As fluid becomes sequestered within the stomach, along with hydrogen ion, chloride and potassium loss, metabolic alkalosis can occur. As mentioned before, gastric distention can cause hypoventilation or hyperventilation which leads to respiratory acidosis and respiratory alkalosis, respectively. Hypokalemia is most common electrolyte abnormality, which results from potassium pooling within the lumen of the stomach[5].

Diagnostic Approach

Signalment, history, and physical exam findings can be enough for GDV to be diagnosed in the exam room. Clinical signs that are characteristic of GDV such as acute onset of retching with a rapidly distending abdomen and signs associated with hypovolemic shock in a large breed, deep-chested dog should raise suspicions of GDV. However, simple gastric dilatation without volvulus can present similar to gastric dilatation with volvulus, and radiographs allow

for definitive diagnosis between the two syndromes. Prior to performing radiographs, the stomach should be decompressed to prevent further cardiovascular compromise. Right lateral and dorsoventral radiographic views are preferred because positioning allows air to fill the abnormally displaced pylorus which facilitates identification. In dogs with GDV, the right lateral shows the pylorus cranial to the body of the stomach and separated from the rest of the stomach by a band soft tissue, and it is often referred to as "reverse C sign", "double bubble", "boxing glove", or "Popeye's arm." Normally, the right lateral view shows the pylorus positioned ventral to the fundus. When viewed with dorsoventral positioning, radiographs show a gas-filled pylorus displaced to the left of midline, while normally it is positioned to the right of midline. Other radiographic findings may include free abdominal air suggesting gastric rupture, air within the wall of the stomach suggesting gastric necrosis, abdominal fluid if peritonitis or hemorrhage are present, and sometimes, megaesophagus[3].

Laboratory findings can be used to help support pathology caused by the GDV. A complete blood count is seldom helpful but can help when DIC is suspected, as it can reveal thrombocytopenia, if present[3]. Bloodwork often shows an increased hematocrit and a wide range of acid-base and electrolyte abnormalities. Metabolic acidosis and hypokalemia are the most common derangements seen; however, metabolic alkalosis can be seen as gastric acid sequesters within the stomach, along with vomiting[3]. A biomarker such as lactate can be used as a prognostic indicator and also used to assess response to treatment. Initial lactate concentrations were once thought to be predictive of gastric necrosis and further complications when elevated (>6 mmol/L), but changes in lactate concentrations in response to fluid therapy and stabilization are now a better indicator of outcome. Lactate may decrease in response to fluid therapy, but if it does not continue to remain within reference range, there is an underlying processing causing

necrosis leading to lactate accumulation within circulation. Prolonged hyperlactatemia that does not decrease within the first 24-48 hours with fluid therapy and appropriate treatment is associated with poor survival rates. Lactate is often increased in GDV cases due to variable degrees of global hypoperfusion and hypovolemia. It can also be increased with more severe cases GDV with secondary sepsis, septic shock, and SIRS. The magnitude of hyperlactatemia is a balance between tissue production, metabolism, and excretion; therefore, it is reversible and serial lactate concentration monitoring is the best way to use lactate as a prognostic indicator. One study found that survival was significantly lower in dogs with an ending lactate concentration >6.4 mmol/L, whereas, a fall in lactate concentration greater than 40% after fluid resuscitation likely indicates better survival. Overall, if the initial lactate concentration is moderately to severely increased (5-10 mmol/L) and does not decrease after fluid therapy, death is likely if the underlying cause is not diagnosed and treated appropriately [7].

Treatment and Management

On emergency presentation, fluid support and gastric decompression are the most important emergency treatments. Stabilization of the patient should be performed first, and one or more large bore catheters should be placed in a jugular vein and/or cephalic veins. High volume of balanced isotonic fluids (90 ml/kg/hr) such as lactated Ringer's solution should be given intravenously within the first fifteen minutes to reestablish cardiac output, with an additional bolus over the next thirty to forty-five minutes. If needed, low volumes of hypertonic saline (4-5 mL/kg or Hetastarch 5-10 mL/kg) can be administered. The patient must be monitored closely, and fluid administration is decreased or increased based on clinical response[1]. Once it is noted that the patient's shock has been resolved, as indicated by an improvement in perfusion parameters such as pulse quality, blood pressure, mucous membrane color and

capillary refill time, maintenance crystalloid fluid therapy should be continued until the patient is taken to surgery. During fluid therapy, packed cell volume and total protein should be monitored, and if clinically significant decreases in PCV/TS are noted, whole blood or plasma should be administered[1]. In addition, significant electrolyte imbalances should be corrected[3].

Gastric decompression is typically performed as soon as fluid therapy is initiated. It is typically accomplished by passage of an orogastric tube or by gastric trocarization, which is the easier and more well-tolerated method. With gastric trocarization, a 14 or 16-gauge, 2-inch needle is used to trocar the stomach on the left side of the abdomen at the site of maximum distention. If an orogastric tube is passed, gas should be relieved first, and then gastric contents should be removed[3].

Once fluid resuscitation and gastric decompression have been performed, caudal vena cava and portal vein occlusion should be improved, and therefore, cardiac output and arterial blood pressure should improve. Once the patient is deemed stable, surgical correction should be performed to reposition and stabilize the stomach[5]. Choices for anesthetic induction can include hydromorphone (0.1 mg/kg IV) plus diazepam (0.2 mg/kg IV) given as incremental doses until intubation is possible. If intubation is not possible with pre-medications alone, etomidate (0.5-1.5 mg/kg IV) or propofol (at reduced dosages) may be used, and once intubated, the patient is maintained on isoflurane inhalant. The patient is placed in dorsal recumbency, and the abdomen is prepped for a midline celiotomy. The surgery typically has three goals. These goals include inspecting the stomach and spleen to identify and remove any damaged or necrotic tissue, decompressing the stomach and correct any malpositioning, and surgically attaching the stomach to the body wall to prevent subsequent malpositioning. When entering the abdominal cavity of a GDV patient, the first structure noted is the greater omentum covering the dilated stomach. The

stomach is decompressed with a large-bore needle attached to suction, and if the needle become occluded, an assistant passes an orogastric stomach tube to perform lavage. If the stomach is rotated clockwise, it is de-rotated counterclockwise by grasping the pylorus with the right hand and also grasping the greater curvature with the left hand. The greater curvature, or fundus, is pushed toward the table while simultaneously elevating the pylorus towards the incision. Necrotic gastric tissue is invaginated or removed without entering the gastric lumen. The spleen is then inspected and rotated back to the left abdominal quadrant, and a partial or complete splenectomy is performed if there is necrosis or significant infarction present. Next, the gastrosplenic ligament and intraabdominal esophagus are located and assessed to ensure they are de-rotated. Before closure, a gastropexy is then performed once all tissues are deemed to be viable and healthy. The gastropexy adheres the stomach permanently to the body wall to prevent recurrence of GDV[3].

Postoperative care and management include monitoring electrolyte, fluid, and acid-base status. Hypokalemia is common postoperatively and may require potassium supplementation. Food is typically withheld 12 to 24 hours after surgery, and when food is offered, most choose to offer small amounts of soft, low-fat food. If vomiting occurs post-operatively, a centrally acting antiemetic such as maropitant or ondansetron may be given. Secondary gastric ulcers can occur and may require treatment with H_2- receptor blockers or proton-pump inhibitors. Intravenous fluids should be continued until the patient's oral fluid intake is adequate. Approximately 12 to 24 hours after surgery, ventricular arrhythmias are quite common and occur in 40-70% of dogs with GDV. Treatment for these arrhythmias includes maintaining normal hydration and correcting electrolyte imbalances, such as hypokalemia. A test bolus of lidocaine (2 mg/kg up to 8 mg/kg IV) is used for the treatment of ventricular arrhythmias if the arrhythmia interferes with

cardiac output, is multiform, or has a sustained ventricular rate above 160 beats per minute. If the arrhythmias diminish or stop in response to the test bolus, a constant rate infusion of lidocaine (50 to 75 ug/kg/min) should be started; however, it is important to monitor for signs of lidocaine toxicity, such as muscle tremors, vomiting and seizures[3].

Post-operative analgesia is extremely important in patients with GDV. Opioids such as morphine, buprenorphine, methadone, meperidine, hydromorphone and fentanyl are all acceptable. Use of synergistic constant rate infusions of lidocaine or ketamine provide good adjunctive analgesia and allow opioid dose reduction. Non-steroidal anti-inflammatory drugs should be avoided due to the potential for any gastrointestinal and renal side effects. Antibiotics are considered necessary in GDV patients due to shock, likely gastrointestinal mucosal damage and portal hypertension which predispose sepsis. Antibiotics that are effective against gram-positive, gram-negative, and anaerobic organisms are best. A good combination of antibiotics includes ampicillin and enrofloxacin, and alternatively, a second-generation cephalosporin (cefoxitin) or trimethoprim-sulfa are also reasonable choices[1].

Case Outcome

Daisy underwent surgical correction of GDV. Upon opening the abdomen, the greater omentum was the first anatomical structure seen, as it was wrapping around the torsed stomach. The spleen was also displaced to the right aspect of the abdomen. Her stomach was de-rotated and assessed for vitality and necrosis. At this time, there was significant hyperemia noted on the gastric serosa, but no necrosis or infarction was seen. The spleen was slightly congested, but once returned to the left aspect of the abdomen, it returned to a normal, healthy color. The rest of the abdomen was assessed for necrosis and infarctions but was unremarkable. Before closure, a belt-loop gastropexy was performed to adhere her stomach to the right aspect of the abdominal

body wall. Daisy's procedure was uneventful, and she made a full recovery with no complications noted.

Once recovered from surgery, Daisy's PCV (39%), TP (5.0 g/dL), and blood lactate (1.3 mmol/L) began to show improvement. She remained quiet, slightly depressed but alert and responsive with normal vital signs. However, approximately eight to twelve hours after surgery, sporadic single ventricular premature contractions with a normal heart rate were noted. She was maintained on intravenous medications and fluids such as Plasmalyte (100 ml/hr), Cerenia (1 mg/kg), hydromorphone (0.1 mg/kg), Entyce (3 mg/kg), and pantoprazole (1 mg/kg). Daisy's PCV (48%) continued to improve, while total protein remained static (5.0 g/dL). She began to have small amounts of intermittent diarrhea 24-hours after surgery, but appetite and thirst were appropriate. A manual platelet count was performed which revealed the thrombocytopenia had improved (72.5 K/uL). Electrocardiogram revealed that VPCs remained intermittent with no tachycardia. Forty-eight hours after surgery, Daisy's vitals remained within normal limits, and laboratory findings (PCV, TP, Glucose, and Lactate) were all within normal limits. Since her appetite and thirst remained appropriate with no diarrhea or vomiting, intravenous fluids were discontinued, and she was transitioned to oral medications, such as Tylenol 4, Pantoprazole, Cerenia, and Ondansetron. By 72-hours post-op, Daisy was bright, alert, and responsive. No additional VPCs were noted, and she was discharged at four days post-op, on May 21,2019.

Conclusion

Daisy's case is testimonial to how rapid diagnosis and treatment of GDV can lead to a more favorable outcome and improved prognosis. Daisy's owner did an excellent job by noticing the acute change in her behavior and bringing her in immediately for assessment. Although Daisy's acute change in behavior and expression of clinical signs did not perfectly match the

textbook description of a typical GDV presentation, her signalment alone was enough to place GDV on the differential diagnosis list. In addition, her triage and physical exams were very supportive of GDV with signs of bloat and cardiovascular compromise. To definitively diagnose GDV, Daisy's right lateral abdominal radiograph was pathognomonic for the syndrome. With rapid diagnosis, stabilization, and treatment, Daisy's stomach, spleen, and other abdominal organs did not suffer any secondary, catastrophic injuries, thus reducing morbidity and mortality rates.

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