Gastric Dilatation-Volvulus Syndrome – A Practical Approach

Philip R Judge BVSc, MVS, MACVSc*
Terry Bassett BPhil, BVSc, MACVSc†

* Senior Veterinarian, Animal Emergency Centre, Melbourne
† Staff Veterinarian, Bundoora Veterinary Clinic/Northern Suburbs Animal Emergency Centre, Melbourne

Introduction

Gastric Dilatation-Volvulus (GDV) Syndrome – from an early age in veterinary medicine, we are taught that a GDV is a major medical and surgical emergency, involving significant stress to both the patient and clinician alike. The aim of this presentation is to make the GDV a delight to deal with – both medically and surgically.

Etiology

A discussion of the etiology and predisposing causes is beyond the scope of this presentation, however, a brief outline of known or suspected predisposing causes is given below:

- Diet – low fiber, low protein, high carbohydrate diets delay gastric emptying, and may lead to a chronically enlarged, distended stomach. If fed over a long period of time, this may lead to stretching of the ligaments that support the stomach.
- Conformation – large breed, deep chested dogs have a higher incidence of the disease. However, GDV has been reported in dogs as small as miniature Daschunds!
- Post-prandial exercise.
- Delayed gastric emptying – is affected by diet as outlined above. In addition, infection, ileus, trauma, and pyloric outflow obstruction may also predispose the patient to developing GDV.
- Recent weight loss.
- Nervous temperament and agitation, rapid ingestion of food, and neurological disease affecting eructation may all predispose the patient to developing GDV.

Pathophysiology

The pathophysiology of metabolic changes in GDV has been well documented. Clinically, we need to be aware of the pathophysiology of GDV, because it helps us to determine the most appropriate medical and surgical priorities in treatment, and helps us determine a prognosis for the patient. The pathophysiology of GDV should be considered in terms of gastric ischemia and edema, gastric motility, gastric ulceration, and cardiovascular events that occur because of gastric distension, gastric decompression, and reperfusion. These are briefly outlined as follows:

- Following gastric torsion, blood flow and oxygen tension dramatically decrease to the stomach, pancreas, duodenum and other abdominal viscera. Why does this happen? A volvulus decreases gastric and intestinal blood flow by obstructing venous outflow, resulting in capillary stasis, and thrombosis of the microvasculature within the organ. As we know, decreasing blood flow through an organ depletes normal stores of ATP – when this happens, cells cannot function normally, they show signs of cell swelling, dysfunction and rupture. This results in a clogging of the microvasculature with cellular debris, and blood clots. In addition, increased sympathetic tone during the stress associated with gastric torsion further decrease blood flow to the stomach and intestines. Following gastric decompression and treatment of the patient with intravenous fluid therapy, the blood flow through the gastrointestinal microvasculature may not become normal until regeneration of the capillary networks has occurred. This results in continued poor perfusion of intestinal capillary networks, and causes loss of protein, red cells, and fluid from the intravascular space into the extracellular space, into the intestinal tract lumen. A damaged gastric mucosa may allow gastric acid to penetrate normal mucosal defense mechanisms and contribute to gastric necrosis and ulceration. If gastric dilatation and volvulus is not corrected within 4 hours, damage to the gastric nervous system may become permanent. This results in permanently disordered gastric motility and/or gastric atony.
- Circulatory changes that occur in gastric dilatation-volvulus syndrome result from:
  - Increased intra-abdominal pressure, resulting in compression of splanchnic blood vessels. This results in a decrease in blood flow to many abdominal organs including the liver, pancreas, duodenum, jejunum, and colon. The resultant tissue hypoxia results in disordered intestinal motility and function following correction of the gastric dilatation or volvulus.
  - Direct compression of the vena cava at the level of T12-L2. The immediate effect of this compression is to decrease venous return to the heart. This results in the patient frequently showing symptoms of sympathetic and adrenal gland stimulation – elevated heart rate, respiratory rate, and peripheral blood pressure elevation. Sympatho-adrenal stimulation results in a further decrease in blood supply to the abdominal viscera, and further compounds circulatory disturbances within the abdominal organs.
  - Cardiac arrhythmias may develop prior to, or following decompression of GDV, and result from increasing cardiac workload in the face of decreasing venous return to the heart, the release of toxic mediators of inflammation from ischemic tissue, and inadequate coronary blood flow and myocardial hypoxia. In addition, thrombi released from microvasculature in the intestines and gastric tissues following reperfusion may lodge within the coronary vasculature, resulting in myocardial hypoxia and subsequent dysfunction.
  - Coagulopathy – Disseminated intravascular coagulopathy may occur in GDV patients due to a combination of vascular stasis within the abdominal organs prior to gastric decompression, release of inflammatory mediators from damaged blood vessels following re-perfusion of hypoxic tissues, and inadequate tissue perfusion.

Clinical Signs

Typically, dogs with GDV present to the clinic with a short history of having non-productive retching, or having frequent attempts to vomit. Occasionally, small amounts of food may be present in the vomitus. The presence of food in vomitus does not preclude the presence of

GDV. In addition, some dogs may have an episode of abdominal discomfort, abdominal bloating and ineffectual vomiting while at home, but may present with no obvious symptoms!

Common signs observed on presentation include abdominal tympani, excessive salivation, cranial abdominal discomfort, abnormal splenic position on abdominal palpation, and clinical signs of sympathetic and adrenal gland stimulation (elevated heart rate, poor femoral pulses, panting etc).

It is advised to take a right lateral abdominal radiograph in patients suspected of having GDV, or in patients with a suspicious history, but equivocal clinical signs.

Initial Medical Management

Traditionally, administration of large volumes of isotonic crystalloid fluids has been recommended for the initial treatment of GDV. However, it is important to stress that fluid therapy should be tailored to the individual patients' needs. Some patients may present in severe decompensating hypovolemic shock, and require administration of shock rates of fluid (see below). Other patients are hemodynamically stable on presentation, and may require more modest fluid administration rates presentation.

A Suggested Algorithm for the Management of Gastric Dilatation-Volvulus

1. Assess:
    (a) Heart rate.
    (b) Pulse rate.
    (c) Cardiac rhythm.
    (d) Temperature.
    (e) Capillary refill time.
    (f) Abdominal cavity.
2. Needle decompress the stomach with one or two 18G needles placed in the stomach percutaneously at the area of maximum tympani to improve patient comfort while the condition is discussed with the owner (disease, treatment, prognosis, and costs).
3. Place a 20 or 18G IV catheter in a peripheral vein in the forelimb (not hindlimb). Obtain blood for the following:
    (a) PCV/TP.
    (b) ACT – a prolonged ACT on presentation is associated with a worse prognosis – however, patients with a coagulopathy on presentation may still have good survivability if managed appropriately.
    (c) Lactate – blood lactate below 6 mmol/l is associated with improved outcome (up to 99% survival rate). Patients with gastric necrosis have been found to have a mean blood lactate level of 6.6 mmol/l.

4. Fluid therapy – begin an infusion of lactated Ringers' solution (LRS) at 10 ml/kg/hr. If the patient is showing signs of shock, begin LRS at 40-60 ml/kg/hr in combination with dextran 70 bolus at 3-5 ml/kg given IV over 10 minutes. Alternatively, hypertonic saline may be used in place of the initial bolus of LRS. HTS may be given as a 7% solution at a dose rate of 3.5 ml/kg IV over 10 minutes, and followed with LRS at 10 ml/kg/hr.
5. Analgesia – Butorphanol 0.1-0.2 mg/kg IV or Fentanyl 4-8 μg/kg IV are the initial analgesics of choice, as they provide rapid analgesia, and reduce doses of induction anesthetic agents. Both drugs should be given as a constant rate infusion within 1 hour of administration.
6. Anesthesia – induce anesthesia with a drug familiar to the clinician – we commonly use diazepam/ketamine; however propofol is an acceptable alternative. Inhalation anesthesia is provided with isoflurane.
7. Gastric Decompression – following anesthetic induction, pass a large bore stomach tube (2-inch diameter) to allow decompression of the stomach. Following decompression, perform a thorough gastric lavage with warm water until the stomach is empty. This procedure may take up to 30 minutes to complete, and has the advantage that when the stomach is investigated at surgery, any necrotic areas of the stomach are easily identified, as gastric decompression and lavage will allow return of blood to viable areas of the stomach prior to surgery. If you have difficulty passing a large bore stomach tube initially, passing a smaller diameter tube may allow removal of the gas-cap, and will facilitate passing of a larger tube for gastric lavage. In addition, re-positioning of the patient may also allow passage of a stomach tube.
8. Surgery – At surgery, gastric torsion should be corrected. The stomach should be thoroughly inspected for areas of necrosis or poor viability. Areas of necrosis should be resected by partial gastrectomy. Invagination of necrotic areas should be avoided as the invaginated are may act as a focus for infection, ongoing release of inflammatory mediators, and sepsis. The spleen should be evaluated thoroughly – if the spleen is torsed – DO NOT UNTWIST IT – remove the spleen if it is torsed or has poor or compromised vascular supply.
9. The Gastropexy – Our preferred technique is the incisional gastropexy. Other techniques such as belt loop gastropexy and circum-costal gastropexy are more time-consuming, and technically more difficult, but offer little or no advantage over incisional gastropexy in terms of stability or longevity of the pexy.

Post-Surgical Care

Following surgical correction of GDV, the patient must be supported appropriately in order to achieve a successful recovery. Failure to identify and act on potential complications can affect the patients' recovery. The key points in post-operative care are:

1. Fluid therapy – continue infusion of LRS at 1.5-2 x maintenance rates (3.5-5 ml/kg/hr). Infusion with dextran 70 at 10-20 ml/kg/day if required to minimize fluid loss into the gastro-intestinal tract lumen.
2. Analgesia – butorphanol or Fentanyl CRI is preferred. Patients with good analgesia have reduced sympathetic tone, and improved blood supply to the gastro-intestinal tract i.e. they recover faster.

3. Gastric motility – begin a CRI of metoclopramide following a subcutaneous dose following surgery of 0.5-1 mg/kg SC. Place a nasogastric tube and suction gastric contents every 3-6 hours if the patient remains nauseous, or has persistent retching or regurgitation.
4. Electrolyte status – hypokalemia contributes to the presence of cardiac arrhythmias post-surgery, and disordered gastric motility and gastric and intestinal atony. Electrolytes should be monitored every 12-24 hours if available. If electrolyte measurement is not readily available, place 26.8 mEq KCL in each litre of maintenance intravenous fluids (LRS).
5. PCV/TP – monitor PCV/TP and ACT every 12 hours. Manage hypoproteinemia using either dextran 70 or pentaspan at 10-20 mf/kg/day or plasma transfusion at 10-20 ml/kg/day as required. A coagulopathy indicated by prolonged ACT should be treated with whole blood or fresh frozen plasma.
6. Gastric mucosal health – many cases of GDV do not require administration of gastric protectants once torsion is corrected. However, ranitidine may be used at a dose of 0.5 mg/kg IV q 8 hrs to reduce gastric acid secretion and reduce the likelihood of gastric ulceration. Sucralfate may be used as an alternative. Begin oral administration of lectade or gastrolyte at 1 ml/kg PO q 4 hrs as soon as the patient is able to tolerate oral medications to aid in maintaining enterocyte health and gastric motility. Begin feeding the patient i/d or boiled chicken once the patient is able to tolerate oral liquids.
7. Cardiac arrhythmias – ventricular tachyarrhythmias are not uncommon in patients following GDV. However, only clinically significant arrhythmias require treatment with anti-arrhythmic medications. Tachyarrhythmias requiring treatment include those that cause a reduction in cardiac output significant to the patient (i.e., signs of weakness, panting, collapse, evidence of poor perfusion etc). Prior to administration of anti-arrhythmic medications, ensure the patient is euvolemic (has adequate fluid therapy) assess pain and manage appropriately – this may involve an alteration of analgesic drug dose or medication; check electrolyte levels and treat accordingly (especially hypokalemia and magnesium), and characterize the arrhythmia. Guidelines for ventricular arrhythmias that require treatment include: sustained heart rate above 140/min; systolic blood pressure less than 100 mm Hg, pre-existing or concurrent cardiac disease, or when R- on T-phenomenon is observed. Treatment is usually commenced with lignocaine at 2 mg/kg IV given over 2 minutes, and repeated every 5-10 minutes up to a cumulative total dose of 8 g/kg. A constant rate infusion may be required.
8. Antibiotic therapy – is usually required due to the compromise of the gastric and intestinal mucosa during the gastric dilatation. Cephalexin, cephazolin or cephalothin, or amoxicillin given at 22 mg/kg IV q 8 hrs are good initial choices.

Conclusion

In summary, the patient with GDV can be successfully managed in the majority of cases. Mortality rates vary depending on a number of factors. Patients with gastric necrosis, requiring gastrectomy, splenectomy, and patients with cardiac arrhythmias prior to surgery all have increased risk of mortality. However, appropriate and timely management of pre-surgical and post-surgical abnormalities can greatly minimize the risk of mortality. In all cases, adherence to the basics of fluid therapy, timely gastric decompression, support of gut health, and cardiovascular and clotting abnormalities is essential to improving patient outcome.

References and Suggested Reading

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