

CANINE GASTRIC DILATATION-VOLVULUS

Part 1 – Pathophysiology and Fluid Resuscitation

Fax Continuing Education Series – Animal Emergency Center

Elke Rudloff, DVM, DACVECC and Rebecca Kirby, DVM, DACVIM, DACVECC

Gastric dilatation-volvulus (GDV) is a malignant rotation of the stomach which commonly results in a variety of life-threatening conditions. Common presenting complaints include non-productive retching, restlessness, increased salivation, and abdominal distension. Although most commonly seen in large breed dogs, GDV can occur in any breed or species of animal.

The most common life-threatening problem associated with GDV is circulatory shock. Severe gastric distention causes significant intra-peritoneal pressure elevation. This can lead to obstruction of the compliant caudal vena cava, decreased venous return and cardiac output. Poor blood flow to the periphery leads to microcirculatory injury and maldistribution of flow to the splanchnic and genitourinary organs. During rotation, rupture of the short gastric and epiploic vessels can cause significant hemorrhage contributing to hypovolemia.

Other life-threatening problems that can occur include upper airway obstruction from regurgitation and aspiration of swallowed saliva. Hypoxemia, electrolyte disturbances, acid/base imbalances and release of myocardial depressant factors can exacerbate myocardial arrhythmias and decreased cardiac output. Cytokine release from traumatized and suffocated tissues can induce the systemic inflammatory response syndrome (SIRS). Circulatory collapse can induce disseminated intravascular coagulation (DIC). Bacterial and endotoxin translocation through disabled gastrointestinal mucosal barrier into the bloodstream is expected. Because the liver may be compromised from venous congestion and circulatory shock, bacteria may not be removed from the portal system resulting in septicemia.

Cells that have been deprived of oxygen develop an elevation of cytosolic calcium and hypoxanthine. Reperfusion of hypoxic cells with oxygen during fluid resuscitation and decompression of the stomach fuels oxygen derived toxic radical production, overwhelming the protective antioxidative enzymes. Oxygen-derived free-radical species initiate lipoperoxidation of cell and organelle membranes, destroy enzyme systems, and cleave DNA strands resulting in cellular dysfunction and apoptosis.

These consequences can culminate in multiorgan failure. The key to successful treatment of complications associated with GDV is the anticipation of these complications, institution of aggressive monitoring and intervention early. Blood samples are collected prior to fluid administration for immediate evaluation of the packed cell volume (PCV), total solids (TS), electrolyte, venous gas, lactate, azostick and dextrostick, platelet count, activated clotting time, and samples are saved for coagulation profile, serum chemistries, complete blood count and urinalysis. Values obtained prior to fluids provide a baseline from which subsequent values are compared. Significant abnormalities are addressed during resuscitation and prior to surgical intervention. Initial lactate levels > 6 mmol/L in GDV can be associated with increased incidence of gastric necrosis. Clinical or laboratory evidence of coagulation abnormalities requires appropriate treatment be initiated prior to surgery. Frozen plasma may be indicated if DIC or coagulation factor defect is suspected.

Fluid Resuscitation (Pre-Op)

Flow-by oxygen is administered during resuscitation if there is poor perfusion. Immediate fluid resuscitation using multiple large-bore peripheral catheters is performed to reach end-point parameters of improved perfusion, normal heart rate and normal to slightly increased blood pressure. Most large breed dogs without evidence of hemorrhage will benefit from large volume resuscitation to supranormal end-points. This means normal heart rate, improved physical perfusion parameters (CRT, mucous membranes, pulses) and a CVP (when monitored) between 8-10 cm H₂O. To reach these end-points of resuscitation, isotonic replacement crystalloids (such as Plasmalyte-A®, Normosol-R®) are administered in doses of 20-50 ml/kg. Synthetic colloids (dextran and hetastarch: 15-20 ml/kg) or Oxyglobin™ (Biopure, Boston, MA: 5-10 ml/kg) are administered to preserve colloid osmotic pressure during fluid resuscitation. A benefit of Oxyglobin™ is that it carries oxygen to tissues with compromised blood flow and causes mild vasoconstriction which might be desired in the GDV patient. If replacement crystalloids are used alone, the crystalloid volume required for resuscitation may increase by 40-60% over what was needed when combined with colloids.

The rapidly deteriorating hypovolemic patient that shows no signs of significant hemorrhage may be resuscitated slightly faster when hypertonic saline (4 ml/kg 7% solution) is administered with the initial infusion of synthetic colloid and buffered isotonic crystalloids. The high concentration of sodium administered will rapidly bring water from the interstitium into the intravascular space. The concurrent administration of crystalloids and colloids will prevent the extravasation of this sodium into the interstitium and replace the interstitial fluid volume. Large volume resuscitation with any fluid product can induce a dilutional coagulopathy necessitating plasma transfusions to prevent hemorrhage.

If significant hemorrhage and/or DIC are present, small volume resuscitation techniques to reach hypotensive end-points may be warranted until surgical exploration can provide hemostasis. "Hypotensive" resuscitation is really a misnomer, with resuscitation end-points truly being the low range of normal (mean arterial pressure of 60 mmHg, systolic blood pressure of 90 mmHg, CVP 5 cmH₂O). The goal is to re-establish perfusion without disturbing any clots. This is accomplished by administering incremental boluses of crystalloids (10-30 ml/kg) and colloids/Oxyglobin™ (5 –10 ml/kg), titrated to administer the least volume possible to reach the desired end-points.

When the desired resuscitation end-point for blood pressure can not be reached with adequate fluid administration (CVP 5-10 cm H₂O), dopamine infusion (5-15 mcg/kg/min) may be required and underlying causes of nonresponsive shock investigated (see Table 1). Dobutamine infusion (5-10 mcg/kg/min) may be necessary instead if cardiomyopathy or poor cardiac contractility is suspected.

Normal to increased blood pressures are evaluated in conjunction with heart rate. The heart rate is a reflection of sympathetic stimulation reflecting intravascular volume and pain control. Adequate or increased blood pressure may be a result of a compensatory sympathetic response to hypovolemia, and aggressive fluid resuscitation may still be indicated, as described above. A constant rate infusion (CRI) of hetastarch or Oxyglobin™ is commonly administered with crystalloids at a rate of 0.8 ml/kg/hr to help maintain intravascular COP and blood pressure.