

How do I treat dogs with Gastric Dilatation Volvulus

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Gastric dilatation and volvulus (GDV) is an acute, life-threatening syndrome, requiring immediate medical and surgical treatment, as well as intensive postoperative care.^{1,2} The pathology involves rapid accumulation of gas in the stomach, gastric displacement, increased intragastric pressure and decreased venous return. GDV is characterized principally by relative hypovolemic-distributive and cardiogenic shock, during which the whole body may be subjected to inadequate tissue perfusion and ischemia.^{2,3} The most serious complications of GDV are associated with ischemic-reperfusion injury (IRI) and consequent systemic inflammatory response syndrome (SIRS) and multiple organ dysfunctions (MODS). Complications from organ dysfunctions include hypotension, acute kidney injury (AKI), disseminated intravascular coagulation (DIC), gastric ulceration and cardiac arrhythmias. Despite appropriate medical and surgical treatment, the reported mortality rate in canine GDV is high (10%-28%).^{3,4}

Prognostic Indicators

Gastric necrosis and high serum lactate concentrations have been identified as strong predictors of postoperative complications and mortality in numerous studies of dogs with GDV, indicating the important role of ischemic hypoperfusion in the progression of this disease.⁵ Other reported risk factors for morbidity and mortality include time lag (>5 hours) from onset of clinical signs to admission, body temperature <38°C (<100.4°F) upon admission, hypotension at any time during hospitalization, sepsis, peritonitis and the need for splenectomy and/or partial gastrectomy during surgery.^{3,4} In a retrospective study conducted at our hospital, dogs with GDV who developed AKI had a significantly higher mortality rate.¹

As lactate on presentation and changes in lactate during hospitalization have repeatedly shown to accurately predict complications and outcome in GDV, dogs with a high presenting lactate (>6mmol/L) should be closely monitored and serial lactate measurements in these patients are recommended. Lactate concentrations that remain high post-operatively should raise the suspicion for gastric necrosis.⁶

Preoperative treatment

Standard treatment includes intensive shock therapy and gastric decompression, followed by surgical resection of necrotic tissue, repositioning of the stomach and its fixation to the abdominal wall, with intensive post-operative monitoring. The initial goal of the treatment is reversal of shock by increasing venous return and reperfusion of transiently ischemic tissues. Thus, gastric decompression, its surgical repositioning, and the consequent restoration of blood flow are crucial factors, influencing survival in canine GDV. We believe that these factors are important in improving the prognosis of dogs with GDV and the sooner these dogs are treated, the better.

Goals of post-operative management

Intensive post-operative management of the GDV patient is essential for patient survival. Pain management is imperative for all dogs following surgery. Additional therapy in the post-operative period is focused on maintaining tissue perfusion along with intensive monitoring for prevention and early identification of IRI and consequent potential complications associated with organ dysfunctions such as hypotension, cardiac arrhythmias, AKI, gastric ulceration, and electrolyte imbalances. In addition, early identification of dogs in need for re-exploration due to gastric necrosis, sepsis or splenic thrombosis is crucial.

Serial abdominal focused assessment with sonography for trauma (aFAST) is recommended in the post-operative period for detection and sampling of abdominal effusion.⁷ A

mild to moderate volume of non-septic abdominal effusion is considered normal; however presence of bacteria, large numbers of degenerate neutrophils, and low fluid glucose concentrations (<50 mg/dL) should raise suspicions for gastric necrosis. In some cases, the authors have identified that accumulation of non-septic fluid resulted from splenic thrombi, which can be diagnosed by Doppler ultrasonography. If blood supply to the spleen is severely compromised, immediate splenectomy should be considered.

Post-operative analgesia

Adequate post-operative analgesia is extremely important in dogs with GDV and opioids such as buprenorphine, methadone, meperidine and fentanyl are all acceptable. Synergistic use of continuous infusions of lidocaine and/or ketamine provides good adjunctive analgesia and may allow for opioid dose-reduction. Non-steroidal anti-inflammatory drugs should be avoided to prevent any potential gastrointestinal and renal side effects. (Table 1)

Management of cardiovascular dysfunction

Intravenous fluid therapy is continued post-operatively; however should be tapered down based on perfusion parameters in non-complicated GDV patients. Isotonic crystalloids are the mainstay of intravenous fluid therapy for dogs with GDV. Given the relatively small contribution of hypovolemia to shock in dogs with GDV, and based on our clinical experience, we have taken a conservative fluid approach in GDV patients. We have found that in most GDV cases, initial restoration of perfusion and reversal of shock can be achieved by gastric decompression and moderate fluid therapy rates, i.e. 20-30 mL/kg of Lactated Ringer's Solution as a bolus to minimize the risk of post ischemic reperfusion injuries, followed by 5mL/kg/hr for the next 24 hours. Obviously, monitoring of perfusion parameters such as heart rate, capillary refill time, lactate, blood pressure (especially diastolic as it reflects volume status more accurately), PCV/TS, serum creatinine, and urine output is warranted, in order to address cases in which a more aggressive fluid approach is indicated.

Point-of-care blood tests are usually performed at least every 12-24 hours to monitor PCV/TS, lactate, creatinine, acid-base status, and electrolyte concentrations. Intravenous fluids can be supplemented with potassium as needed.

Management of IRI

Ischemic-reperfusion injury, known sequelae of GDV results in paradoxical tissue damage and destruction, caused by reactive oxygen species (ROS), formed in previously ischemic tissues. During ischemia, two major changes occur in the cells, ATP degrades, resulting in the accumulation of its by-product, hypoxanthine, and the conversion of xanthine dehydrogenase into xanthine oxidase (XO). As oxygen re-enters into previously ischemic tissues, it serves as a substrate for XO, which then transforms excess hypoxanthine into ROS. When ROS interact with cells, they inflict damage to proteins, DNA and RNA, and cause lipid peroxidation of cell membranes, often leading to cell death. Lidocaine, a local anesthetic and antiarrhythmic agent, has traditionally been used for the treatment of ventricular dysrhythmias. The use of intravenous lidocaine to prevent IRI and SIRS was described in human medicine and in laboratory animals.

The author have shown that lidocaine (2 mg/kg given as an IV bolus) was administered before any other medical intervention, followed by constant rate infusion of 0.05 mg/kg/min for 24 h during initial patient management.¹ Lidocaine treatment significantly decreased AKI, cardiac arrhythmias, multiple coagulation disorders and hospitalization period compared to 47 historical control dogs. Mortality rate in the treatment group was lower (10%) compared to the control group (24%), albeit insignificantly.

Conclusion

In conclusion, GDV is an emergency life-threatening syndrome characterized by relative hypovolemic and distributive shock. Early admission and treatment improve survival and decrease complication rate. High lactate concentrations and gastric necrosis are associated with higher complication and mortality rates. Ischemic reperfusion injury plays a major role in this syndrome. Lidocaine treatment combined with conservative fluid therapy seems to be promising treatment to reduce mortality and complication rates.

Table 1. Pharmacologic management of the postoperative GDV patient

Indication	Drug	Drug class / mechanism of action	Recommended dose range	Potential adverse effects / comments
Analgesic drugs	Fentanyl	μ -receptor agonist	5 μ g/kg IV bolus followed by 1-5 μ g/kg/hr CRI	Sedation at higher doses, bradycardia, hypotension
	Meperidine	μ -receptor agonist	2-4 mg/kg q4hrs SC	Do not give IV
	Methadone	μ -receptor agonist		bradycardia
	Buprenorphine	Partial μ -receptor agonist	0.01-0.02 mg/kg IV, IM, SC q6-8 hrs	
	Tramadol	Atypical μ agonist		
Anti-arrhythmics	Lidocaine	Type II Na channel blocker	2 mg/kg slow IV bolus followed by 50 μ g/kg/min CRI	Also provides analgesia and pro-kinetic effects. May cause GI upset
	Procainamide	Type I Na channel blocker	2-4 mg/kg slow IV bolus followed by 10-50 μ g/kg/min CRI	Hypotension following rapid IV injection
Antacids	Famotidine	H ₂ receptor antagonist	0.5-1 mg/kg SIV q12-24 hours	Bradycardia following rapid infusion
	Pantoprazole	Proton pump inhibitor	0.5-1 mg/kg IV q24 hours	
	Omeprazole	Proton pump inhibitor	0.5-1 mg/kg PO q24 hours	
Gastroprotectants	Sucralfate	Local anti-ulcer effect.	0.5-1 g/kg PO per dog q8-12 hours	
Prokinetics	Metoclopramide	Dopamine-receptor antagonist	0.4 mg/kg SC q8hr or 1-2 mg/kg/day CRI	
	Erythromycin	Macrolide antibiotic with prokinetic effects	1 mg/kg q8hr	GI upset
	Ranitidine	H ₂ receptor antagonist	1-2 mg/kg q12 hours	Bradycardia following rapid infusion
Antiemetics	Maropitant	Substance P inhibitor	1 mg/kg SC q24hr	Analgesic effect. Pain at site of injection. High cost

	Dolasetron	Serotonin type 3 (5-HT ₃) receptor antagonist	0.6 mg/kg IV q24hours	High cost
	Ondansetron	Serotonin type 3 (5-HT ₃) receptor antagonist	0.1-1mg/kg IV q8-12 hours	High cost
Treatment of IRI	Lidocaine	Type II Na channel blocker	2 mg/kg slow IV bolus followed by 0.05mg/kg/min CRI	See above
	Deferoxamine	Iron chelating agent	10 mg/kg IM or slow IV q8-12 hours	Not evaluated in naturally-occurring GDV
Treatment of AKI	Furosamide	Loop diuretic	1-4 mg/kg IV bolus followed by 0.25-1 mg/kg/hr CRI	May cause dehydration and mild hypotension
	Mannitol	Osmotic diuretic	0.5-1 g/kg slow IV followed by 1 mg/kg/hr CRI	Caution if cardiac dysfunction
	Dopamine	Dopaminergic antagonist at low doses	3 µg/kg/min	May cause arrhythmias

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