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Topical Review

Postoperative Management of Dogs With Gastric Dilatation and Volvulus

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The objective of the study was to review the veterinary literature for evidence-based and common clinical practice supporting the postoperative management of dogs with gastric dilatation and volvulus (GDV). GDV involves rapid accumulation of gas in the stomach, gastric volvulus, increased intragastric pressure, and decreased venous return. GDV is characterized by relative hypovolemic-distributive and cardiogenic shock, during which the whole body may be subjected to inadequate tissue perfusion and ischemia. Intensive postoperative management of the patients with GDV is essential for survival. 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. In addition, early identification of patients in need for re-exploration owing to gastric necrosis, abdominal sepsis, or splenic thrombosis is crucial. Therapy with intravenous lidocaine may play a central role in combating IRI and cardiac arrhythmias. The most serious complications of GDV are associated with IRI and consequent systemic inflammatory response syndrome and multiple organ dysfunction syndrome. Other reported complications include hypotension, AKI, disseminated intravascular coagulation, gastric ulceration, and cardiac arrhythmias. Despite appropriate medical and surgical treatment, the reported mortality rate in dogs with GDV is high (10%-28%). Dogs with GDV that are affected with gastric necrosis or develop AKI have higher mortality rates.

Introduction

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 volvulus, 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.[3] The most serious complications of GDV are associated with ischemic-reperfusion injury (IRI) and consequent systemic inflammatory response syndrome and multiple organ dysfunction.[4-6] Complications from organ dysfunctions include hypotension, acute kidney injury (AKI), disseminated intravascular coagulation (DIC), gastric ulceration, and cardiac arrhythmias.[3,7] Despite appropriate medical and surgical treatment, the reported mortality rate in dogs with GDV is high (10%-28%).[4-6,8,9]

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.[4-6,8,10] Other reported risk factors for morbidity and mortality include time lag (>5 hours) from onset of clinical signs to admission, body temperature $<38^{\circ}C$ (<100.4 F) on admission, hypotension at any time during hospitalization, sepsis, peritonitis, and the need for splenectomy or partial gastrectomy or both during surgery.[4,8] In a retrospective study conducted at our hospital, dogs with GDV that developed AKI also had a significantly higher mortality rate.[8]

As blood lactate concentration 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 level (>6 mmol/L) should be closely monitored, and serial lactate measurements are recommended in these patients.[6,9,10] Lactate concentrations that remain high postoperatively should raise suspicion for gastric necrosis.[9]

Goals of Postoperative Management

Intensive postoperative management of dogs with GDV is essential for patient survival. Pain management is imperative for all dogs following surgery. Additional therapy in the postoperative 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 owing to gastric necrosis, sepsis, or splenic thrombosis is crucial.

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Serial abdominal focused assessment with sonography for trauma (aFAST)[11] is recommended in the postoperative period for detection and sampling of abdominal effusion. A mild to moderate volume of nonseptic 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.[12,13] In some cases, the authors have identified that accumulation of nonseptic fluid associated with splenic thrombosis, which can be diagnosed by Doppler ultrasonography. If blood supply to the spleen is severely compromised, immediate splenectomy should be considered drugs commonly used for post-operative management of patients with GDV are listed in Table 1.

Postoperative Analgesia

Adequate postoperative analgesia is extremely important in dogs with GDV, and opioids such as morphine, buprenorphine, methadone, meperidine, hydromorphone, and fentanyl are all acceptable. Synergistic use of continuous infusions of lidocaine or ketamine or both provides good adjunctive analgesia and may allow for opioid dose reduction. Nonsteroidal anti-inflammatory drugs should be avoided to prevent any potential gastrointestinal and renal side effects.

Management of Cardiovascular Dysfunction

Intravenous (IV) fluid therapy is continued postoperatively; however, it should be tapered down based on perfusion parameters in patients with noncomplicated GDV. Isotonic crystalloids are the mainstay of IV 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 dogs with GDV. 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, that is, 20-30 mL/kg of lactated Ringer solution as a bolus to minimize the risk of post–ischemic-reperfusion injuries, followed by 5 mL/kg/h 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), packed cell volume and total solids (PCV/TS), serum creatinine, and urine output is warranted to identify 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. IV fluids can be supplemented with potassium as needed.

The use of synthetic colloids in critically ill human patients has been called into question owing to a higher incidence of AKI and increased mortality associated with their use.[14-19] In some European countries, their use in human medicine has been banned and as such they are no longer available. Additionally, a black box warning has been instituted by the Food and Drug Administration in the United States cautioning against their use in critically ill people. Until clear data regarding the safety and efficacy of synthetic colloids is published in the veterinary literature, it is our opinion that these fluids should be used cautiously, and when necessary and financially feasible, the use of natural colloids should be considered. Severely hypoalbuminemic dogs (albumin level <1.5 g/dL) with hypovolemic shock will likely benefit from canine or human albumin solutions; however, if human albumin is to be used, owners should be made aware of potential serious adverse effects.[20-22] Modest data are available regarding the safety and efficacy of canine albumin; however, its use increased albumin concentrations in a small group of septic dogs with no apparent significant adverse effects or change in outcome.[23]

Postoperative cardiac arrhythmias, generally of ventricular origin, have been reported in 40%-70% of dogs with GDV, and their development has been associated with a worse prognosis in some studies.[24,25] As such, electrocardiogram monitoring of cardiac arrhythmias is important in the first 24-48 hours such that treatment can be instituted when indicated. Although some of the potential causes of cardiac arrhythmias in dogs with GDV are not able to be prevented (such as myocardial ischemia), it is important to rule out potentially modifiable causes of cardiac arrhythmias such as hypoxemia and electrolyte abnormalities. The potential for hypoxemia owing to aspiration pneumonia or the development of acute respiratory distress syndrome should be evaluated by pulse oximetry, arterial blood-gas analysis, and thoracic radiographs if clinical signs supporting respiratory failure are present (e.g., dyspnea, tachypnea, and cyanosis). Electrolyte and venous blood-gas analysis should be performed to evaluate potassium and magnesium disturbances and acid-base status. Treatment with lidocaine (2 mg/kg slow IV bolus followed by a constant rate infusion of 50 μg/kg/min) or procainamide (2-4 mg/kg slow IV bolus followed by 10-40 μg/kg/min) is indicated if sustained ventricular tachycardia (heart-rate >180) is present in the face of compromised perfusion despite appropriate fluid resuscitation. In addition, if “R on T phenomena” or multiform ventricular premature complexes are present, lidocaine should be administered.[2,26,27]

Management of Respiratory Dysfunction

Respiratory rate and effort should be monitored closely in the postoperative period in dogs with GDV. Dogs with evidence of aspiration pneumonia on preoperative thoracic radiographs should receive appropriate IV antimicrobial therapy and be monitored particularly closely in the postoperative period. Pulse oximetry or arterial blood-gas analysis or both are warranted in dogs with signs of respiratory difficulty to assess the severity of their impairment and guide therapy. Oxygen therapy may be indicated in dogs with hypoxemia and can be provided in a variety of ways. Nasal oxygen insufflation is perhaps the most practical way of providing oxygen supplementation to large and giant breed dogs, although oxygen cages can also be used when available. Dogs with severe hypoxemia, hypercapnia, or impending respiratory fatigue may require mechanical ventilation.

Management of Gastrointestinal Dysfunction

Given the propensity for gastric ulceration, nausea, anorexia, vomiting, and regurgitation in dogs following surgery for GDV, pharmacotherapy often involves drugs targeting the gastrointestinal system.

Antacid medications are administered routinely in the postoperative period and may include either a H_2 receptor antagonist or a proton pump inhibitor or both. Sucralfate may be administered as a gastroprotectant. In dogs with evidence of postoperative ileus (identified by diagnostic imaging) or regurgitation, prokinetics are also indicated. Metoclopramide, as a continuous rate infusion, is generally the first-line prokinetic agent used in dogs with GDV, but erythromycin or ranitidine or both can also be used. The use of antiemetics in dogs with postoperative vomiting is recommended. Maropitant is the only antiemetic specifically labeled for use in dogs, although serotonin receptor antagonists

such as dolasetron and ondansetron are also commonly used in veterinary medicine.

There are no specific postoperative nutrition recommendations for dogs with GDV; however, it is generally accepted that early, enteral, postoperative feeding is essential for recovering gastrointestinal motility and maintaining nutritional status in both human and veterinary patients.[28-32] Uncomplicated GDV cases will usually resume voluntary food consumption within 1-3 days postoperatively. In complicated cases, nutritional support in the form of nasogastric or nasoesophageal tube feeding or peripheral or central parenteral nutrition is necessary. Enteral nutrition is preferred over parenteral nutrition unless protracted vomiting is present. In dogs with prolonged anorexia, placement of an esophageal feeding tube is indicated.

Management of Coagulation Dysfunction

The occurrence of multiple hemostatic abnormalities in dogs with GDV is reportedly 16%.[3,23] Interestingly, the presence of multiple hemostatic abnormalities was not found to be a risk factor for mortality in our recent study,[33] in contrast to previous studies in which it was associated with gastric necrosis and a higher mortality rate.[4,34] The difference can perhaps be explained by the more abundant use of fresh frozen plasma and aggressive postoperative treatment in recent years in emergency centers reducing DIC as a cause of death in dogs with GDV. In many cases, the coagulation dysfunction resolves without specific treatment; however, use of FFP (10-20 mL/kg) should be considered when clinical signs of hypocoagulable DIC are present along with an abnormal coagulation profile.

Management of AKI

As AKI may develop in dogs with GDV, hydration status, weight, and urine output should be assessed frequently in the postoperative period. In addition, small increases in creatinine level ($>$ 0.3 mg/dL) may be significant even if creatinine concentration is still within its normal range.[35] If AKI is suspected, an indwelling urinary catheter should be placed for objective quantification of urine output and better assessment of fluid balance. In oliguric patients with signs of overhydration (e.g., "jelly"-like skin turgor and chemosis), an "ins-and-outs" fluid regimen should be employed, and diuretics should be initiated. Drugs that can be considered to stimulate urine output include fenoldopam (0.8 μg/kg/min),[36] furosemide 1-2 mg/kg, IV, followed by a CRI, and mannitol 1 g/kg IV over 20 minutes. If feasible, hemodialysis may benefit patients with acute anuric AKI.

Low-dose dopamine had historically been suggested as a preventative treatment for AKI in people. Although previous studies yielded conflicting results, a recent prospective randomized clinical trial concluded that low-dose dopamine had no role in prevention and management of AKI.[37-40] Fenoldopam is a selective dopamine-1 receptor agonist that induces vasodilation and selectively increases both renal-cortical and outer-medullary blood flow.[41] Similar to dopamine, fenoldopam increases renal blood flow and GFR.[42,43] However, unlike dopamine, fenoldopam is not associated with the undesired side effects resulting from α- and β-adrenergic receptor activation. A recent large-scale study that evaluated fenoldopam as a preventive therapy for contrast-induced nephropathy in human patients failed to show advantage of its use.[44] Conversely, a study of AKI in intensive care unit human patients demonstrated that fenoldopam (0.09 μg/kg/min IV) led to a significantly decreased incidence of AKI. Fenoldopam pharmacokinetics has been evaluated in dogs.[36] Recently, we have had promising results with the use of fenoldopam in dogs with heatstroke, with treated dogs demonstrating increased GFR compared with the placebo group.

Management of IRI

IRI, a known sequelae of GDV, results in paradoxical tissue damage and destruction, caused by reactive oxygen species (ROS), formed in previously ischemic tissues. During ischemia, 2 major changes occur in the cells, adenosine triphosphate degrades, resulting in the accumulation of its by-product, hypoxanthine, and the conversion of xanthine dehydrogenase into xanthine oxidase. As oxygen re-enters into previously ischemic tissues, it serves as a substrate for xanthine oxidase, 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[45,46] (Fig).

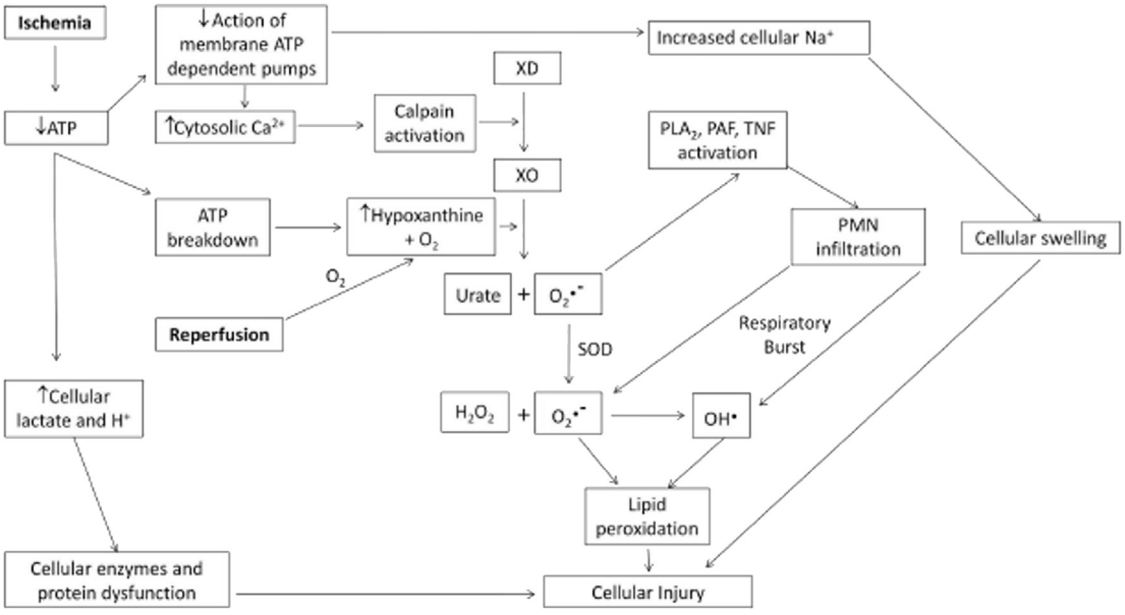

Fig. Schematic representation of the cascade of molecular events occurring following ischemia and reperfusion injury.

Treatment of IRI with deferoxamine (an iron chelator) and to a lesser extent dimethyl sulfoxide (Me_2SO; a free radical scavenger) in experimentally induced GDV in dogs was reported to reduce mortality but to our knowledge has never been evaluated for safety and efficacy in the clinical settings.[3] Lidocaine, a local anesthetic and antiarrhythmic agent, has traditionally been used for the treatment of ventricular dysrhythmias. The use of IV lidocaine to prevent IRI and systemic inflammatory response syndrome has been described in human medicine and in laboratory animals.[45-49] Previous studies in experimentally induced GDV in dog and rat models showed that preischemic lidocaine administration reduced gastric and cardiac histopathologic and ultrastructural tissue damage and cardiac arrhythmias.[50]

Lidocaine treatment was evaluated in a retrospective study of 112 dogs diagnosed with GDV in our hospital.[8] IV lidocaine was initiated in all treated dogs before surgical repositioning of the stomach but after commencement of medical treatment and gastric decompression. We found no significant differences in mortality and complication rates between lidocaine-treated or nontreated dogs. However, we suspected that by the time lidocaine was administered, reperfusion injury had already commenced. Therefore, in a follow-up prospective study, lidocaine treatment was initiated before gastric decompression and fluid therapy. 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 hours during initial patient management.[33] Lidocaine treatment significantly decreased AKI, cardiac arrhythmias, multiple coagulation disorders, and hospitalization period compared with 47 historical control dogs. Mortality rate in the treatment group was lower (10%) compared with the control group (24%), albeit insignificantly so.[25]

Postoperative Antimicrobial Therapy

No data regarding the use of prophylactic antibiotics in GDV cases are available. In simple GDV cases, that is, with no splenectomy or partial gastrectomy, the authors use a single agent antibiotic such as a first-generation cephalosporin for up to 3-5 days postoperatively. If gastric perforation or necrosis was present during surgery, if spillage of gastric contents occurred, or if evidence of aspiration pneumonia is evident on preoperative thoracic radiographs, broad-spectrum antibiotic coverage, such as amoxicillin-clavulanic acid and fluoroquinolones are used for 5-7 days.

Table
Pharmacologic Management of Postoperative Patients With GDV

Indication	Drug	Drug Class or Mechanism of Action	Recommended Dose Range	Potential Adverse Effects or Comments
Analgesic drugs	Fentanyl	μ Receptor agonist	3-5 μg/kg IV bolus followed by 1-5 μg/kg/h CRI	Sedation at higher doses, bradycardia, and hypotension
	Morphine	μ Receptor agonist	0.2-0.5 mg/kg IV, IM or SC every 6-8 h or 0.1-0.2 mg/kg/hr CRI	Side effects same as per fentanyl
	Hydromorphone	μ Receptor agonist	0.05-0.1 mg/kg every 4 h IV	As per fentanyl. Panting.
	Meperidine	μ Receptor agonist	2-4 mg/kg every 4 h SC	Do not give IV
	Methadone	μ Receptor agonist	IV	Bradycardia
	Buprenorphine	Partial μ receptor agonist	0.01-0.02 mg/kg IV, IM, or SC every 6-8 h	
	Tramadol	Atypical μ agonist	3 mg/kg IV, SC, or PO every 8-12 h	Injectable form not available in USA
Antiarrhythmics	Lidocaine	Type II Na channel blocker	2 mg/kg slow IV bolus followed by 50 μg/kg/min CRI	Also provides analgesia and prokinetic effects. May cause GI upset
	Procainamide	Type I Na channel blocker	2-4 mg/kg slow IV bolus followed by 10-50 μg/kg/min CRI	Hypotension following rapid IV injection
Antacids	Famotidine	H_2 receptor antagonist	0.5-1 mg/kg slow IV every 12-24 h	Bradycardia following rapid IV injection
	Pantoprazole	Proton pump inhibitor	0.5-1 mg/kg IV every 24 h	
	Omeprazole	Proton pump inhibitor	0.5-1 mg/kg orally every 24 h	
Gastroprotectants	Sucralfate	Local antiulcer effect	0.5-1 gram per dog every 8-12 h	
Prokinetics	Metoclopramide	Dopamine receptor antagonist	0.4 mg/kg SC every 8 h or 1-2 mg/kg/day CRI	
	Erythromycin	Macrolide antibiotic with prokinetic effects	1 mg/kg every 8 h	GI upset
	Ranitidine	H_2 receptor antagonist with prokinetic effects	1-2 mg/kg every 12 h	Bradycardia following rapid infusion
Antiemetics	Maropitant	Substance P inhibitor	1 mg/kg SC every 24 h	Analgesic effect. Pain at site of injection. High cost
	Dolasetron	Serotonin type 3 (5-HT_3) receptor antagonist	0.6 mg/kg IV every 24 h	High cost
	Ondansetron	Serotonin type 3 (5-HT_3) receptor antagonist	0.1-1 mg/kg IV every 8-12 h	High cost
Treatment of IRI	Lidocaine	Type II Na channel blocker	2 mg/kg slow IV bolus followed by 50 μg/kg/min CRI	Mentioned previously
	Deferoxamine	Iron-chelating agent	10 mg/kg IM or slow IV every 8-12 h	Not evaluated in naturally occurring GDV
Treatment of AKI	Fenoldopam	Selective dopamine-1 receptor agonist	0.8 μg/kg/min CRI	High cost. Not evaluated in GDV patients
	Furosemide	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 agonist at low doses	3 μg/kg/min	May cause arrhythmias

CRI, constant rate infusion; IM, intramuscular; PO, oral; SC, subcutaneous; AKI, acute renal failure; IRI, ischemic reperfusion injury.

Postoperative Management Following Hospital Discharge

Discharge recommendations include restricted activity, along with short-term antibiotics where appropriate, H_2 blockers, and analgesia. To prevent recurrence of gastric dilation, small, frequent meals are recommended over a single large meal per day.[51]

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 rates. High lactate concentrations and gastric necrosis are associated with higher complication and mortality rates. IRI plays a major role in this syndrome. Lidocaine treatment combined with conservative fluid therapy seems to be a promising approach to reduce mortality and complication rates following surgical correction.

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