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# Optimizing Survival in Canine Gastric Dilatation-Volvulus: An Innovative Management Protocol

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- Gastric Dilatation and Volvulus (GDV) is an acute, life-threatening disorder in dogs, characterized by abnormal twisting of the stomach on its mesenteric axis, with subsequent gastric gas accumulation and distention
- The disease is typically encountered in large or giant breeds although other breeds can present with the condition

## PHYSIOPATHOLOGY

Once a state of dilation and volvulus is entered this will decrease perfusion to not only the gastric wall but also other organs. Decreased gastric mural perfusion as well as distension place those animals at high risk for gastric rupture and peritonitis. Besides, this might also create the need for partial gastrectomy during surgical treatment.

- Because of the increase in stomach size and its rotation, the **vena cava can be compressed**, which decreases venous return to the heart and thus cardiac output. Patient may then develop hypovolemic shock and might progress to **vasodilatory shock** as the disease worsens or after resuscitation.
- The rotation is usually clockwise (when considering the abdominal cavity in a caudo-cranial direction).

The pylorus is displaced dorsally. Abdominal distension might increase intra-abdominal pressure, which in addition to the state of shock will impair perfusion to abdominal viscera such as the kidneys, liver, gastrointestinal tract.

- Abdominal distension and intra-abdominal hypertension may compress thoracic viscera. Patients may then develop mild to severe respiratory distress.
- During the development of GDV, the spleen can be displaced, thrombosed, or ruptured. Evaluation of the spleen at the time of surgery is therefore important. The short gastric veins might also be torn potentially leading to a hemoabdomen.

Dogs suffering severe problems such as GDV are at risk of SIRS **Systemic inflammatory response syndrome, sepsis and disseminated intravascular coagulation**

## Intrinsic risk factors

- Intrinsic risk factors include breed, body size, thoracoabdominal dimensions, gastric volume, gastric position, gastric ligament laxity, control of eructation and pyloric canal

function. Great Danes, German shepherd dogs, Irish setters and standard poodles have been consistently found to be over-represented in studies of dogs with GDV and are statistically known to be at increased risk.

- A significant predisposition has been correlated with a high thoracic depth to width ratio.
- Increasing age, having a first-degree relative with GDV, thin body condition and a fearful or anxious temperament.

#### Extrinsic risk factors

- Extrinsic risk factors include diet, postprandial GD and accumulation of gastric gas. The precise role of diet is unclear although feeding a small number of large meals and rapid speed of eating are likely to play a part in some dogs.
- It is clear that the development of GDV is influenced by environmental, anatomical, physiological and pathological risk factors, but their specific contributions are likely to vary between individuals.
- In order for GDV to occur there must be a failure of the gastro-oesophageal and pyloric sphincter function, thus preventing normal eructation and pyloric outflow.

#### EARLY STAGES OF GDV

- Pacing, restlessness, salivation panting
- Ineffective Vomiting Attempts (10- 20-minute intervals)
- Abdomen increased in size may appear full
- Deep red gums
- Increased Restlessness dyspnoea
- Increased salivation, panting and severe abdominal distension.
- Increased heart rate (↑100 bpm)

#### TACHYCARDIA

#### NEEDS VETERINARY ATTENTION ASAP!!

#### Key considerations

- Gas in the dilated stomach is due to aerophagia rather than bacterial fermentation
- There is no evidence to suggest that torsion is due to the influence of the spleen. GDV has been reported in splenectomised animals
- Pyloric surgery is not necessary to prevent GDV recurrence
- The ability or inability to pass a stomach tube is not helpful in distinguishing between GDV and GD
- Steroids are not indicated for the management of GDV
- A gastropexy is essential in the management of dogs with GDV
- Presumed gastric necrosis must be treated with partial gastric resection

#### DIAGNOSTICS

- During resuscitation, clinicians might consider measuring lactate and bicarbonate concentrations
- The presence of GDV is confirmed with abdominal radiographs, in particular with the patient in right lateral recumbency. Dorsal displacement of the duodenum gives the stomach the appearance of a “boxing glove” or “Popeye fist”.

#### Prognostic Indicators

- Gastric necrosis and high serum lactate concentrations have been identified as strong predictors of postoperative complications and mortality.

#### PREOPERATIVE TREATMENT AND STABILIZATION

#### Immediate treatment goals

- Immediate goals in treatment of GDV include restoring circulating blood volume and gastric decompression.
- Rapid surgical correction of the volvulus follows initial patient stabilization. Because duration of clinical signs is one of the risk factors of GDV-associated death, it is

imperative to recognize and correct this condition immediately.

## CORRECTION OF HYPOVOLEMIA

- Rapid fluid replacement with one or more large-bore (16- to 18-gauge) IV catheters placed in cranial (jugular, cephalic) veins.
- Shock rate (90 mL/kg/hr) fluid therapy with crystalloids immediately
- Colloids : hetastarch at a rate of 10–20 mL/kg, IV
- hypertonic saline 7% hypertonic saline solution with dextran 70 at a rate of 5 mL/kg over 15 min.

## VASCULAR ACCESS

- Cephalic veins or jugular veins only because lack of blood flow from the caudal half of the body

## FLOW-BY OXYGEN SHOULD BE PROVIDED DURING STABILIZATION

## TROCARISATION

## GASTRIC DECOMPRESSION

### Parameters monitored:

#### Electrocardiography (ECG)

- Ventricular cardiac arrhythmias have been reported in up to 40% of dogs with GDV because of myocardial ischemia.

#### Capnography and Pulse Oximetry

- Respiratory function is affected during GDV so, Capnography is a non-invasive method for the measurement of the respiratory carbon dioxide.

#### Acid-base and electrolyte disturbances

- Increased lactate concentration.s
- Hypokalaemia is the most usual electrolyte disturbance and it can deteriorate cardiac arrhythmias and therefore, it is important to be measured.
- Potassium supplement solutions can be administered IV, separately from the rapid

administration of isotonic crystalloids, at a concentration of 30-80 mEq L<sup>-1</sup>.

- The potassium solutions are administered at a maximum rate of 0.5-1 mEq kg<sup>-1</sup> h<sup>-1</sup> when serum potassium is below 3 mEq L<sup>-1</sup>

## ANAESTHETIC PROTOCOL

### PROTOCOL 1:

#### Premedication:

Fentanyl 2–4 µg/kg IV or Oxymorphone 0.1 mg/kg IV.

Induction: Diazepam/Midazolam 0.25–0.5 mg/kg IV.

If needed, low doses of propofol (1 mg/kg) to help with intubation.

#### Protocol:2 for stable patients

##### For stable patients

- Premedication: Fentanyl 2–10 µg/kg IV.
- Induction: Ketamine 6 µg/kg + Diazepam 0.2 mg/kg.

##### For unstable patients (Without premedication)

- Induction: Fentanyl 2–4 µg/kg, Midazolam 2 mg/kg + ketamine 4 mg/kg.
- Maintenance: Lidocaine and Ketamine CRI (Loading dose of Lidocaine 2 mg/kg and Ketamine 3 mg/kg, then CRI 100 µg/kg/min of both drugs, which can decrease Sevoflurane MAC by 62.8%).

### Protocol 3

- No premedication
- Induction: Fentanyl 2-4 µg kg<sup>-1</sup> & midazolam 0.2- 0.3 mg kg<sup>-1</sup> IV. If needed, low doses of propofol to effect (1 mg kg<sup>-1</sup>) to help with intubation.
- Maintenance: Isoflurane in 100% oxygen.

#### Protocol 4

- Premedication: Midazolam 0.2-0.3 mg kg<sup>-1</sup> IM & pethidine at 3 mg kg<sup>-1</sup> IM.
- Induction: Propofol 1 mg kg<sup>-1</sup> IV until intubation (to effect).
- Maintenance: Isoflurane in 100% oxygen & fentanyl as CRI at 0.1 µg kg<sup>-1</sup> min<sup>-1</sup>.

#### Protocol 5

- Premedication: Midazolam 0.2-0.3 mg kg<sup>-1</sup> IM & pethidine at 3 mg kg<sup>-1</sup> IM.
- Induction: Propofol 1 mg kg<sup>-1</sup> IV until intubation (to effect).
- Maintenance: Propofol as CRI at 0.1 mg kg<sup>-1</sup> min<sup>-1</sup> alone or in combination with fentanyl as CRI at 0.05 µg kg<sup>-1</sup> min<sup>-1</sup>.

#### Anesthetic Complications:

##### Hypotension:

- GDV hypotension is due to the compression of the caudal vena cava, resulting in blood not being able to return to the heart.
- This complication is dealt with by vasoactive therapy. Large amounts of crystalloids can be used in patients.
- Intravenous infusion of inotropic drugs such as Dopamine or Dobutamine are useful (5–7 µg/kg/hr), while Ephedrine (0.06 mg/kg IV bolus) is another, less commonly used option.

#### Antiarrhythmic Therapy in Dogs

##### Intravenous Antiarrhythmic Drugs—Ventricular

##### Lidocaine

- IV bolus (2 mg/kg increments up to total dose of 8 mg/kg) then IV drip at 50 µg/kg/min (500 mg in 500 mL of fluid, administered at maintenance rate [66 mL/kg per

day]). CRI can be increased to 75–100 µg/kg/min if indicated clinically.

**NOTE: If seizures occur, stop the drug and consider using another antiarrhythmic agent. Procainamide (Pronestyl)**

- 5–25 mg/kg slow IV bolus then 25–50 µg/kg/min as CRI

##### Amiodarone

- 1 mg/kg IV repeated up to a total cumulative dose of 3 mg/kg; alternatively 150 µg/kg/min as a CRI.

##### Intravenous Antiarrhythmic Drugs—Supraventricular

##### Diltiazem (Cardizem)

- 1–8 µg/kg/min CRI for acute SVT

##### Oral Antiarrhythmic Drugs—Ventricular Amiodarone

- 10–20 mg/kg PO q24h for 5–7 days, then 5–10 mg/kg PO q24h (may be able to reduce to q48h)

##### Mexiletine (Mexitil)

- 4–8 mg/kg PO q8h

##### Sotalol (Betapace)

- 1–3 mg/kg PO q12h

##### Oral Antiarrhythmic Drugs—Supraventricular

##### Extended-Release Diltiazem (Dilacor XR; Cardizem CD)

- 2–4 mg/kg PO q12h

##### Sotalol (Betapace)

- 1–3 mg/kg PO q12h

##### Amiodarone

- 10–20 mg/kg PO q24h for 5–7 days, then 5–10 mg/kg PO q24h (may be able to reduce to q48h)

##### Digoxin (Lanoxin)

- 0.005–0.01 mg/kg PO q12h

##### Atenolol (Tenormin)

- 0.1–2.0 mg/kg PO q12–24h (gradually up titrate the dose)

Gastric decompression and gastropexy are performed

#### Recommendations for Dogs at High Risk for Gastric Dilatation Volvulus

Feed several small meals a day rather than one large meal.

- Avoid stress during feeding (if necessary, separate dogs in multiple-dog households during feeding).
- Restrict exercise before and after meals (of questionable benefit).
- Do not use an elevated feed bowl.
- Do not breed dogs with a first-degree relative that has a history of gastric dilatation-volvulus.
- For high-risk dogs, consider prophylactic gastropexy.
- Seek veterinary care as soon as signs of bloat are noted

gastric dilatation-volvulus syndrome in 306 dogs. *J Am Anim Hosp Assoc.*, 46: 97-102.

Van Kruiningen HJ, Gargamelli C, Havier J, Frueh S, Jin L and Suib S (2013). Stomach gas analyses in canine acute gastric dilatation with volvulus. *J Vet Intern Med.*, 27: 1260-1261.

### Prognosis

- GDV is a life threatening condition.
- Lactate concentration is thought to be a **prognostic factor** with high reliability.
- Concentrations below 4 mmol L<sup>-1</sup> indicate high survival rate and reduced risk of complications.
- Concentrations of lactate above 6 mmol L<sup>-1</sup> may indicate gastric necrosis and sepsis, and survival rate is reduced significantly

### References:

- Bruchim, Y., & Kelmer, E. (2014). Postoperative management of dogs with gastric dilatation and volvulus. *Topics in Companion Animal Medicine*, 29(3), 81–85.
- Dennler R, Koch D, Hassig M, Howard J, Montavon PM. (2005) Climatic conditions as a risk factor in canine gastric dilatation-volvulus. *Vet J*; 169: 97-101.
- Glickman LT, Glickman NW, Schellenberg DB, Raghavan M, Lee TL. (2000) Incidence of and breed-related risk factors for gastric dilatation-volvulus in dogs. *J Am Vet Med Assoc*; 216: 40-45
- Green TI, Tonozzi CC, Kirby R, Rudloff E. (2011). Evaluation of initial plasma lactate values as a predictor of gastric necrosis and initial and subsequent plasma lactate values as a predictor of survival in dogs with gastric dilatation-volvulus: 84 dogs (2003-2007). *J Vet Emerg Crit Care.*, 21: 36-44.
- Mackenzie G, Barnhart M, Kennedy S, DeHoff W and Schertel E. 2010. A retrospective study of factors influencing survival following surgery for