Gastric Dilatation-Volvulus in Dogs

Acute gastric dilatation-volvulus (GDV) is a life-threatening condition, with fatality rates ranging from 10% to 60%. The animals most commonly affected by GDV include older, large or giant breed, deep-chested dogs, including Great Danes, German Shepherds, Standard Poodles, and large mixed breed dogs. Early diagnosis, medical stabilization, surgical intervention and post-operative monitoring are important factors in reducing the mortality rate.

Gastric dilatation-volvulus is the result of accumulation of gas, fluid, or a combination of the two in the stomach. Factors responsible for causing dilatation include aerophagia, exercise after ingesting a meal, and overeating. The stomach distends with gas or fluid, and rotation along the axis of the esophagus and cardia follows. The rotation is generally in the clockwise direction (when viewed in dorsal recumbency), and can be up to a maximum

of 360°. A less common fate is a counter-clockwise rotation, to a maximum of 90°. In addition to the accumulation of gas and/or fluid, there is often an outflow obstruction due to a mechanical or functional abnormality.

The clinical signs associated with GDV are restlessness, anxiousness, respiratory dysfunction, hypersalivation, retching, abdominal distension and frequent attempts to vomit. The animal may present weak, collapsed, or comatose, depending on the degree of shock. Signs related to hypovolemic shock are pale mucous membranes, prolonged capillary refill time, rapid, weak, thready pulses, and tachypnea. Diagnosis of GDV is based upon clinical signs, inability to pass a gastric or nasogastric tube effectively, and consistent radiographic findings.

The major life threatening abnormality associated with GDV is shock. Shock is due to compression of the caudal vena cava, from distension of the stomach, and the portal vein, from distension and rotation of the stomach. As a result, there is decreased tissue perfusion, which leads to hypoxia and ischemia of tissues. Ischemia of cardiac muscles can result in arrhythmias, and ischemia of abdominal organs can lead to necrosis/death of affected organs/tissues.
The first priority for treatment of GDV is cardiovascular stabilization. Dogs that have persistent circulatory collapse are thought to be at greater risk of dying than those dogs that are stabilized.¹ Hosgood, et al, suggests that intravenous therapy with 7% sodium chloride plus 6% dextran initially, followed by 9% sodium chloride, is superior to 9% sodium chloride alone. Once the animal is stabilized the stomach is decompressed using orogastric intubation or needle gastrocentesis. Radiographs are taken prior to surgery to determine if a volvulus is present. Broad spectrum antibiotics are administered prophylactically. The use of corticosteroids remains controversial, but have shown to be beneficial in instances of septic or endotoxic shock.

The timing of surgery also remains controversial, as there are advantages and disadvantages to early and late surgical intervention. The surgical techniques used for repair of GDV include tube gastrostomy, circumcostal gastropexy, belt-loop gastropexy, and permanent incisional gastropexy. Complications associated with tube gastrostomy are peritonitis (due to premature removal or loosening of the tube), cellulitis (due to gastric content leakage), and alteration of gastric myoelectric activity. There is some evidence suggesting gastropexy procedures lead to chronic bloaters by altering gastric emptying.³

Perfusion of the tissues is maintained perioperatively and post-operatively, using an intravenously administered, balanced electrolyte solution. Perioperative and post-operative monitoring of the patient for perfusion, as well as abdominal distension, are important. The parameters used to assess tissue perfusion include capillary refill time, blood pressure, peripheral pulse pressures, arterial blood gas, urine output, PCV, and total protein. Abdominal distension is monitored due to the potential of re-bloating following surgery.

There are many complications that can occur postoperatively, most of which are secondary to the initial problems associated with GDV. Cardiac arrhythmias, usually of ventricular origin, tend to occur in the first 12-36 hours following surgery. A continuous ECG is recommended to monitor for arrhythmias, and anti-arrhythmic drugs (lidocaine, procainamide) are used when the arrhythmia is responsible for poor tissue perfusion. Additional complications include disseminated intravascular coagulation, sepsis caused by gastric leak or aspiration pneumonia, protein loss, gastric ischemia and esophagitis.
Additional medical options available for the treatment of GDV include: a lipid peroxidase inhibitor to prevent lipid peroxidation secondary to reperfusion injury; cisapride, metoclopramide, erythromycin, and ranitidine to facilitate gastric emptying; and vasoactive intestinal peptide to facilitate eructation and lower esophageal sphincter tone in animals which are chronic bloaters. The effect of metoclopramide on gastric emptying in dogs with GDV has been studied, and the results suggest there is no improvement of gastric emptying.4

Left untreated, GDV can lead to multiple organ failure, circulatory shock and death.4 Factors which contribute to a higher mortality rate include gastric necrosis, gastric resection, splenectomy and pre-operative cardiac arrhythmias.5 It is therefore important to be familiar with the clinical signs of GDV in order to arrive at an early diagnosis, stabilize the patient as soon as possible, surgically correct the volvulus, and medically manage any additional complications.

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**Post Publication Correction**

The section discussing fluid therapy for gastric dilatation-volvulus in dogs should have read “… intravenous therapy with 7% NaCl (5ml/kg) in 6% Dextran 70 (HS/D70) initially followed by 0.9% NaCl is superior to 0.9% NaCl alone.” Resource is Section 7/Gastrointestinal Disorders, Chapter 4/Disease of the Stomach, pp. 675 of Saunders Manual of Small Animal Practice, edited by Stephen J. Birchard and Robert G. Sherding, W.B. Saunders Company, 1994.