GASTRIC DILATION INTHE HORSE

Etiology

Gastric outflow obstruction. Idiopathic. Ingestion of excess fluid or feedstuffs.

Chronic gastric dilatation can be caused by the following:

• Outflow obstruction, such as cicatricial constriction of the pylorus secondary to gastroduodenal ulceration or pressure by a tumor

• Gastric atony in older horses or wind-sucking (aerophagic) horses

Acute gastric dilatation is associated with:

• Reflux of intestinal contents secondary to acute intestinal obstruction, e.g., anterior enteritis, small-intestinal strangulation, or ileus

• Ingestion of excess fluid or feedstuffs such as whey or grain

• Acute idiopathic dilatation after racing

Epidemiology

Sporadic. No age, breed, or sex predilection

Clinical signs

Colic. Reflux from nasogastric tube. Gastric rupture, acute severe peritonitis, and death are representing the mean clinical signs.

The clinical findings in gastric distension depend in large part on the underlying disease. However, horses with primary gastric distension have abdominal pain, often of 12 to 36 hours' duration, that progressively worsens. The heart and respiratory rates increase progressively as the distension worsens, and the horse

may sweat and exhibit signs of increasingly severe abdominal pain. Paradoxically, some horses with gastric distension, especially the type that develops over several days or in horses recovering from intestinal surgery and being treated with analgesics, may not exhibit any but the most subtle signs until rupture of the stomach occurs.

Vomition in horses is very rare, In **grain engorgement dilatation** abdominal pain is usually severe. Dehydration and shock develop rapidly, often within 6 to 8 hours of ingestion of the grain, and can be severe. Death from gastric rupture can occur within 18 hours.

Acute postrace dilatation occurring immediately after racing is accompanied by more serious and acute signs. There is abdominal distension, coughing, and dyspnea. Tympany is also detectable on percussion of the anterior abdomen and large amounts of foul-smelling gas, and usually fluid, are passed via the stomach tube. This immediately relieves the animal's distress. In **chronic dilatation** there is anorexia; mild pain, which is either continuous or recurrent; scanty feces; and gradual loss of BW persisting for a period of months. Vomiting and bouts of pain may occur after feeding, but they are not usually severe. Dehydration may be present but is usually only of moderate degree.

Clinical pathology

Nondiagnostic. Inflammatory cells and ingesta in peritoneal fluid of horses with gastric rupture.

Diagnostic confirmation

Nasogastric reflux without other identifiable cause

Lesions

Gastric dilatation. Gastric rupture with hemorrhage at margins of rupture

Treatment

Gastric decompression. Treat underlying disease.

Control

Prevent overeating. Control inciting diseases.

Equine gastric ulcer

Gastric ulcers (equine gastric ulcer syndrome) are common in horses and foals. This syndrome is most closely associated with horses. Neonatal foals are at significant risk for development of perforating peptic ulcers until they are several weeks old, because their gastric mucosa is not developed to full thickness at birth. Although spontaneous healing of peptic ulcer lesions has been noted.

Etiology and Epidemiology

Ulcers in the nonglandular squamous mucosa are associated with repeated direct insult from ultra-low pH fluid normally found in the glandular region of the stomach. Pressure increases inside the abdomen (associated with exercise), collapsing the stomach and

forcing the acid gastric contents upward. The more fluid (and highly acidic) contents of the lower stomach come in contact with the nonglandular squamous mucosa, causing inflammation and, potentially, erosions to varying degrees.

The causes of ulcers in the glandular mucosa of the stomach are less well defined. Use of nonselective NSAIDs are known to reduce blood flow to the GI tract, causing decreased production of the mucobicarbonate matrix by the gastric glandular mucosa and resulting in ulceration.

Epidemiology Foals from 1 day of age; 50% of normal foals have gastric mucosal ulceration. Clinical disease in 0.5% of foals. More severe ulceration in stressed foals or foals with other diseases.

Clinical signs

None in most foals. Teeth grinding, excessive salivation, colic, diarrhea, inappetence, and weight loss. Ulcers in glandular mucosa are considered most clinically important. Sudden death with perforation. Ulcers present on gastroduodenoscopy.

Clinical pathology:

Non diagnostic

Lesions

Gastric mucosal ulceration, duodenal ulceration and stenosis, and esophagitis. Peracute septic peritonitis

Diagnostic confirmation

Gastroscopic demonstration of ulcers in foals with appropriate clinical signs.

Treatment

Treatment should be reserved for foals with clinically important disease. Ranitidine 6.6 mg/kg, orally every 8 to 12 h, *or* cimetidine 6.6 to 20 mg/kg orally or intravenously every 6 h, *or* omeprazole 1 to 4 mg/kg orally or intravenously every 24 h

Control

Minimize occurrence of inciting or exacerbating diseases. Do not administer antiulcer medications as prophylaxis.

Equine Colic

The term “colic” is defined as a general manifestation of abdominal discomfort in the horse, regardless of the cause. While most cases of colic are associated with gastrointestinal disturbances, the nature of some abdominal discomfort may be non-gastrointestinal in origin, such as those resulting from other abdominal organs (including but not limited to the liver, spleen, ovaries, or kidneys). This discomfort can be anything from mild belly pain, causing the horse to paw, bite, and kick at its sides or seem restless.

Etiology

Colic have a variety of causes therefore, colic can be caused by /Types

1- Gas - Excessive accumulation of gas stretches the intestines, causing pain.

2- Obstruction or impaction - Fecal material becomes hard and difficult to pass due to dehydration, the presence of large numbers of worms, ingestion of sand, etc.

3- Strangulation - The intestines rotate or become entrapped, which prevents the flow of food and feces and blocks blood flow.

4- Infarction - Poor blood supply to the intestine, which leads to tissuedeath.

5- Inflammatory - Infectious diseases or other conditions can cause gastroenteritis or colitis (inflammation of the gastrointestinal tract) or peritonitis (inflammation of the abdominal cavity).

6- Ulcers - Erosions of the lining of the gastrointestinal tract can lead to pain and poor gastrointestinal function.

Pathogenesis

The pathogenesis of equine colic is variable depending on the cause and severity of the inciting disease. A horse with a strangulating lesion involving 50% of its small intestine has a much more rapidly evolving disease, with severe abnormalities, than does a horse affected with mild spasmodic colic or impaction of the pelvic flexure of the large colon. Although equine colic often involves changes in many body systems, notably the gastrointestinal, cardiovascular, metabolic, and endocrine systems, there are several features and mechanisms that are common to most causes of colic that depend only on the severity

of the disease for the magnitude of their change. The features common to severe colic, and often present to a lesser degree in milder colic, are pain, gastrointestinal dysfunction, intestinal ischemia, endotoxemia or toxemia, compromised cardiovascular function (shock), and metabolic abnormalities.

Clinical signs

A horse with mild colic will paw the ground with its front feet, be restless, lie down, roll frequently, and look at its abdomen. It may also kick at its abdomen in an attempt to relieve the pain. A horse with more severe colic will roll and may become cast. Horses with very severe colic will throw themselves to the ground and roll violently. These horses can be dangerous to work with until properly sedated. Pulse and respiratory rates will be higher than normal. The absence of abdominal sounds is characteristic of a horse with colic. Mucos as (eye conjunctiva, vulva, gum, etc.) can be very pale or may be dark red, depending on the cause of colic.

Diagnosis

• General condition and behavior (calm, restless, alert, dull, apathetic)

• Frequency of abdominal pain (none, intermittent, or continuous)

• Frequency of abdominal sounds (normal, increased, decreased, or absent)

• Abdominal size (normal, reduced, distended)

• Nature of peripheral pulse (normal or weak)

• The length of time it takesfor gumsto return to normal color after pressure is applied (capillary refill time) • Other signs (sweating, wounds, etc.) • Water intake • Presence of and consistency and regularity of feces

Differential diagnostic

The following diseases may be mistaken for colic:

• Laminitis
• Pleuritis
• Enterocolitis
• Rhabdomyolysis
• Obstructive urolithiasis
• Uroperitoneum
• Foaling and dystocia
• Uterine torsion
• Peritonitis
• Cholelithiasis
• Ovulation and ovarian pain
• Esophageal obstruction
• Duodenitis-proximal jejunitis
• Gastric ulceration
• Testicular torsion
• Tetanus
• Rabies
• Botulism

Medical Treatment

The specific treatment of each case of colic varies and depends on the nature of the lesion and the severity of the disease. However, several principles are common to the treatment of most colic:

• Provision of analgesia and Nonsteroidal Antiinflammatory Drugs such as **Flunixin meglumine** is a potent, long-acting analgesic.

• Correction of fluid, electrolyte, acidbase and hemostatic abnormalities

• Gastrointestinal lubrication or administration of fecal softeners

• Treatment of underlying disease

Prevention

Here are some preventative measures you can take:

1- Always make sure that your horse has access to fresh, clean water. In the winter, horses are more susceptible to impaction colic. In cold climates, regularly check to make sure there is no cold water, or install water heaters.

2- Ensure that your horse has enough access to roughage in his diet, such as pasture or hay. This part of a horse's provides the bulk needed for proper gut motility.

3- Make sure your horse has regular dental checkups to ensure there are no sharp points or missing teeth that prevent him from grinding his food properly.

4- Talk to your veterinarian about the best way to control intestinal parasites.

5- In the spring, slowly allow your horse to pasture. Do not let him out to graze full-time on new spring grass all at once.