EQUINE COLIC

General principle: Adult horse

Abdominal pain in horses, evident as a constellation of clinical and behavioral signs is commonly referred to as colic. Most often caused by gastrointestinal disease, although it can manifest as a result of disease in any intraabdominal organ.

Colic is a frequent and important cause of death and is considered the most important disease of horses encountered by practicing veterinarians.

Etiology:

A-Physical colic

1- Low grade roughage, Bad teeth , Debility, Exhaustive, Excessive inspissation (retained meconium , impaction of lower intestine)
2- Lush green feed , Clostridium Perfringens A, secondary to acute intestinal obstruction(distention of intestine)
3- Engorgement on grain or other palatable food (distention of stomach).
4- Engorgement on whey, Pyloric obstruction , Reflex from intestinal obstruction, spontaneous after racing (distention of stomach)
5- Impaction of ileocecal valve, fiber-ball, foreign bodies, volvulus ,torsion, intussusception ,strangulation in occlusive hernias , diaphragmatic hernia , hematoma of gut wall (distention of intestine ,accumulation of fluid)
6- Verminous mesenteric arteritis (adhesion causing cicatricle constriction of lumen ,terminal ileus hypertrophy (distention of intestine ,accumulation of fluid) subacute intestinal obstruction

B- Functional colic

1- Parasitism (strongylosis) , Bacteria (Salmonlosis), Viral (Equine viral arteritis), Physical sand colic, enteriolith and chemical poisoning.
2- Excitement , Cold drinking or chilling , Verminous mesenteric arteritis and Reflex from other viscera.

EPIDEMIOLOGY

Occurrence

Equine colic occurs worldwide, although there are regional differences in the types of colic

Risk Factors

Risk factors for colic can be categorized as

(1) Horse Characteristics

a –Age: Horses 2 to 10 years of age are 2.8 times more likely to develop colic that horses less than 2 years.

b- *Sex:* There is no overall effect of gender on risk of colic, but certain diseases are restricted by gender. For instance, inguinal hernias occur only in males, whereas entrapment of intestine in the mesometrium is restricted to mares.

(2) Diet and Feeding Practices :Horses on pasture are at a lower risk of developing colic than are **stabled horses** fed concentrate feeds. The risk of colic increases with the amount of concentrate fed

(3) MANAGEMENT:

a- *Watering:* Horses without constant **access to water** are at increased risk of developing colic.

b- *Housing:* Increased duration of stabling per day is associated with an increased risk of colic.

c- *Exercise*: Overall, there appears to be an increased risk of colic among horses undertaking physical activity or that have a recent change in the amount of physical activity.

(4) Medical History: Horses with a history of colic are more likely to have another episode, and horses that have had colic surgery are approximately five times more likely to have another episode of colic

(5) Parasite Control: Inadequate parasite control programs have increased risk of developing colic such as(presence of tapeworms, roundworms).

(6) SEASON : The seasonal pattern might represent changes in management and use of horses rather than a direct effect of weather.

Pathogenesis

The pathogenesis of equine colic is variable depending on the cause and severity of the inciting disease. 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. 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.

Pain

Pain is the **hallmark of gastrointestinal disease** in horses and is attributable to distension of the gastrointestinal tract and stimulation of stretch receptors in the bowel wall and mesentery. Gastrointestinal pain has an inhibitory effect on normal gastrointestinal function, causing a feedback loop in which the pain inhibits normal gut motility and function, allowing accumulation of ingesta and fluid, resulting in distension and further pain.

Gastrointestinal Dysfunction

Colic is almost invariably associated with impaired gastrointestinal function, usually alterations to motility or absorptive function. Gastrointestinal motility may be increased, as is presumed to be the case in spasmodic colic, altered in its character or coordination, as in some cases of impaction colic, or absent, such as in ileus secondary to inflammation or ischemia of the bowel or to the presence of endotoxemia.

The absorptive function of the intestine may be decreased by inflammation or ischemia, which results in distension of the small intestine or large colon, pain, and potentially rupture of the stomach or colon.

Ischemia of the Intestinal Wall

Ischemia may be the result of impaired blood flow to or from the intestine because of torsion or volvulus of the intestine. Ultimately, most forms of lethal colic involve some degree of ischemia of the intestine, with subsequent loss of barrier function, evident in its most extreme form as rupture of the viscus, endotoxemia, bacteremia, cardiovascular collapse, and death.

Endotoxemia

Death in fatal cases of colic in affected viscus ruptures secondary to distension, or when ischemia and/or infarction damages a segment of bowel wall, is caused by the absorption of endotoxins and other compounds from the gut lumen into the systemic circulation.

Shock

The usual cause of death in severe colic is cardiovascular collapse secondary to endotoxemia/ toxemia and hypovolemia.

Hypovolemia is caused by the loss of fluid and electrolytes into the lumen of the gastrointestinal tract or loss of protein from the vascular space with subsequent reduction in the circulating blood volume.

Cardiorespiratory function is impaired if there is severe distension of gut, such as in large-colon torsion, because of restricted respiration by pressure on the diaphragm and reduced venous return to the heart because of pressure on the caudal vena cava.

- **Small intestine**

The hypermotility and spasmodic colic , volvulus , strangulation and intussusception these account for almost the colic case , internal parasitism special strongylosis is credited heaving this effect. The poor blood supply also has effects on the vascular endothelium, leading to an increased permeability which first leaks plasma and eventually blood into the intestinal lumen. In the opposite fashion, gram-negative bacteria and endotoxins can enter the bloodstream, leading to further systemic effects

- **Large intestine**

Colic from disease of the large intestine , is mostly due to the impaction with undigested food fiber or infraction as a result of verminous arteritis .

- **Inflammatory**

Inflammation of the intestine or peritoneum alters gastrointestinal motility and absorptive function leading to accumulation of fluid and ingesta, distension and abdominal pain.

Clinical signs

Visual examination

- Behavior:
  - Restlessness manifested by pawing, stamping or kicking at the belly or pacing in small circles and repeatedly getting up and lying down, often with exaggerated care.
  - The pain in severe abdominal manifested by sweating, looking or nipping at the flank, rolling, and lying on the back. Often the penis is protruded without urinating or with frequent urination of small volumes.
  - The common signs is continuous playing with water without actually drinking **(sham drinking).**
  - Pain may be continuous or, more commonly, intermittent with bouts of pain lasting as long as 10 minutes interspersed with similar periods of relaxation.

- The posture

Is abnormal, with standing stretched out with the forefeet more cranial and the hind feet more caudal than normal - the so- called **'saw horse' stance**. lie down on their backs with their legs in the air in some horses.

- Vomiting

Projectile or regurgitation of intestinal contents through the nose is very unusual in the horse and is a serious sign suggesting severe gastric distension and impending rupture.

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- Abdomen size

The important diagnostic sign is distension of the abdomen .**Symmetrical,** when the distention is sever as distension of the colon, , cecum, secondary to colon torsion, or impaction of the large or small colon and subsequent fluid and gas accumulation. **Or asymmetrical** when the cecum is distended and the abdomen is an enlargement in the right sub lumbar fossa.

Physical examination

- Heart and respiratory rates
  - Mild disease when the heart rates is less than 40/min
  - Severe disease when the heart rates is above 120/min in the terminal stages.
  - The respiratory rate is variable and may be as high as 80/min during periods of severe pain.
  - Sobbing dyspnea is feature in the terminal stage when shock and dehydration are at their peak.
- Mucous membranes and extremities
  - Dry mucous membranes, normal capillary refill time and pink color.
  - Dry mucous membranes , pale color and delayed capillary refill(> 2 s) in impaired cardiovascular function.
  - Bluish tint and capillary refill is longer than 3 seconds in severe cases.
  - Cool extremities and signs of, cardiovascular collapse, is indicative of a poor prognosis.
- Auscultation of the abdomen

  All four quadrants (dorsal and ventral, left and right sides) of the abdomen should be examined for at least 1 minute at each site.
  - Continuous, loud borborygmi distributed in all or most quadrants are indicative of intestinal hyper motility and consistent with spasmodic colic, impending diarrhea or the very early stages of both enteritis and peritonitis.
  - Absence of sounds indicated paralytic ileus or impaction
  - Auscultation is high up in the right flank, over the ileocecal valve if it is functioning there will be aperiodic squirt of fluid contents into agas-filled cavity with fluid at the bottom the resulting sound squelching, gassy rush.

Note: When laboratory tests are not available, dehydration can be crudely assessed by **tenting the skin of the neck or eyelid**, **looking for sunken eyes**, **depression, high heart rate, and feeling for tackiness of the gums**. **Jugular filling** and quality of the peripheral pulses can be used to approximate blood pressure. **Capillary refill time (CRT)** may be decreased early in the colic, but generally prolongs as the disease progresses and cardiovascular status worsens

Percent Dehydration	Heart rate	Mucous membrane quality	CRT	Time skin tent holds	Other
5%	Normal	Moist to slightly tacky	< 2 seconds	1–3 seconds	Decrease in urine production
8%	40-60 bpm	Tacky	Usually 2–3 seconds	3–5 seconds	Decrease in blood pressure
10-12%	60+ bpm	Dry	Usually > 4 seconds	5+ seconds	Decrease in jugular fill and quality of peripheral pulses; sunken eyes present

Clinical pathology

Few changes have diagnostic significance but many are used to monitor the severity of the disease. Hemoconcentration, azotemia and metabolic acidosis are frequent findings. Peritoneal fluid may have increased protein and leukocyte concentration

Differential diagnostic : The following diseases may be mistaken for colic:

• Laminitis • Pleuritis • Enterocolitis • Obstructive urolithiasis • Foaling and dystocia • Uterine torsion • Peritonitis • Ovulation and ovarian pain • Esophageal obstruction • Duodenitis-proximal jejunitis • Gastric ulceration • Anthrax • Testicular torsion • Lactation tetany • Tetanus • Rabies • Botulism • Grass sickness • Purpura hemorrhagica • Clostridial myonecrosis (gas gangrene)

Treatment

Specific causes of colic are best managed with certain drugs. These include:

- Spasmolytic agents, most commonly Buscopan, especially in the case of gas colic.
- Pro-motility agents: metoclopramide, lidocaine, bethanechol, and erythromycin are used in cases of ileus.
- Anti-inflammatories are often used in the case of enteritis or colitis.
- Anti-microbials may be administered if an infectious agent is suspected to be the underlying cause of colic.
- Phenylephrine: used in cases of nephrosplenic entrapment to contract the spleen, and is followed by light exercise to try to shift the displaced colon back into its normal position.
- Psyllium may be given via nasogastric tube to treat sand colic.
- Anthelminthics for parasitic causes of colic.
- Analgesia
- Correction of fluid, acid-base and electrolyte abnormalities ,
- Gastric decompression via nasogastric intubation.
- Administration of fecal softeners or lubricants (mineral oil 10-15 ml/kg, via nasogastric tube, every 12-24 hrs , purgative and dioctylsodium, sulfosuccinate1 2-25 mg/kg, via nasogastric tube, every 24 h .
- surgical correction of the lesion

Trocarization

Occasionally in severe cases of flatulent (gas) colic or in cases of colon torsion in which the abdominal distension is impairing respiration, it may be necessary to relieve the gas distension of the colon or cecum by trocarization, performed through the **right paralumbar fossa** immediately caudal to the last rib. The exact place for trocarization can be located by simultaneously flicking the body wall with a finger and listening with a stethoscope.

Prevention and Control

- Good management
- Good care of the teeth
- Good care of the feeding that horses intestine can digest.
- Control of intestinal laminitis (Parasitism as strongyles spp.) .
- Ensure adequate roughage in the diet (avoidance of very mature coarse hay or straw).

Table(1).Analgesics and Spasmolytic for used in equine colic

Drugs class	Drug	Dose	Comments
NSAIDs	Flunixin meglumine	0.25-1 .0 mg/kg, IV or 1M every 8-24 h 0.25-1 .0 mg/kg, IV or 1M every 8-24 h	Potent analgesic for up to 1 2 h May mask signs of surgical disease.
	Ketoprofen	2.2 mg/kg, IV every 1 2 h	Potent analgesic for up to 1 2 h Potent analgesic for up to 1 2 h.
	Phenylbutazone	2 .2-4.4 mg/kg, IV or PO every 1 2 h	May mask signs of surgical disease Potent analgesic for up to 1 2 h.
			Weak analgesic for gastrointestinal pain. Minimal effect on motility.
Opiates	Dipyrone	1 0 mg/kg, IV or 1M every 4--6 h	Weak analgesic. Often combined with hyoscine in commercial preparations (Buscopan compositum).
	Butorphanol	0.025-0 . 1 mg/kg, IV or 1M as required	Potent analgesia for 30-90 min. Safe. Often combined with an alpha-2 agonist. May cause ataxia.
	Meperidine (pethidine)	0.2-2.0 mg/kg, slowly IV or 1M as required	Moderate analgesia for 0. 5-4 h. Can cause excitement and/or ataxia
	Pentazocine	0.5-1 .0 mg/kg, IV or 1M as required	Moderate analgesia. May cause ataxia
	Morphine sulfate	0.05-0.01 mg/kg slowly IV or 1M as required	Potent analgesia. Can cause excitement
Alpha-2 agonists	Xylazine	0 . 1 -1 .0 mg/kg, IV or 1M, as needed	Potent analgesia and sedation for up to 30 min. Decreases intestinal motility. Often combined with butorphanol.
	Detomidine	1 0-40 μg/kg, IV or 1M as needed	Potent analgesia and sedation for up to 1 20 min
	Romifidine	0.04-0.08 mg/kg, IV or 1 M	Potent analgesia and sedation
	Medetomidine	0.01-0.02 mg/kg, I V or 1 M	Potent analgesia for up to 1 20 min. Sedation
Spasmolytics	Atropine	0.0 1 -0.04 mg/kg I V or 1M	Do not use because of induction of ileus
	Hyoscine butylbromide	0.1 -0.4 mg/kg, IV or 1M every 6-1 2 h	Reduces gastrointestinal motility. Mild analgesic. Often combined with dipyrone
Other	Acetylpromazine	0.02-0.04 mg/kg, IV or 1M every 6-24 h	No analgesia but marked sedation. Potent hypotensive agent. Do not use
	Lidocaine	1 .5 mg/kg IV loading dose followed by 0.05 (mg/kg)/min IV infusion	Substance P inhibitor. Analgesic, anti-inflammatory, promotility agent.

1M intramuscularly, IV intravenously; NSAIDs, Nonsteroidal anti-inflammatory drugs; PO, orally.