Colic in the neonatal foal

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Colic in the foal differs from colic in several ways. Foals can have almost all the same causes of colic as adults but there are a few which are specific to foals. A list of the main differential is below. Colic in the neonate is not uncommon and it can be difficult to distinguish surgical from non-surgical causes. The signalment, history and physical examination can help along with imaging and laboratory aids.

DIFFERENTIAL DIAGNOSES

Category	Considerations	Common causes	Less common causes
Congenital	First few days of life, pain without pyrexia	None	12-24 hours: Atresia ani/coli/recti Lethal White foal Chyloperitoneum Any age: hernia
Obstruction	Usually without pyrexia	Meconium impaction	Colon displacement/impaction Caecal impaction Small colon obstruction Older foals: ascarids Faecoliths (minis) Duodenal stricture (>1 mo) Sand enteropathy Ileal impaction
Strangulation	Severe pain no pyrexia	Small intestinal volvulus Intussusception	Large colon volvulus Mesodiverticular band Meckel's diverticulum Strangulating hernia
Inflammatory	Pain varies, often pyrexic/diarrhoea/sepsis	Necrotizing enterocolitis Clostridium Rotavirus Salmonella	Cryptosporidia Giardia Aeromonas NSAIDs Abscess

			Adhesions Antibiotic induced peritonitis Older foals: Rhodococcus abscess or colitis Lawsonia intracellularis
Other	Mild to moderate pain, no pyrexia; ulcers freq have diarrhoea	Gastric ulcers Ruptured bladder Ileus secondary to other disease	Ovarian torsion Haemoperitoneum

Table adapted from Michelle Henry Barton, Colic in the newborn foal, Equine Neonatal Medicine edited by MR Paradis

Clinical signs of colic:

- Attempting the defecate (legs usually camped forward under the foal) or urinate (legs stretched out behind the foal), restlessness, walking the box, tail swishing
- More severe signs are rolling, getting up and down, lying on the back
- +/- Abdominal distention (often best monitored by measuring the abdominal circumference)
- Pyrexia suggests an infectious cause
- Gastric ulceration cases can show diarrhoea, bruxism, ptyalism and abruptly stop nursing

History:

- Breed: minis (faecoliths), Overo-overo Paint horse → Lethal White foal (aganglionosis)
- Sex: colts predisposed to meconium impaction
- Age: see differential list above
- Urination and defecation patterns
- Diet: on mare's milk or supplement
- Foaling and farm histories (enteritis, infectious diseases)

Physical examination: As for adults but palpation for the bladder and impactions, and ballotment of the abdomen is possible. Gastric dilation can distend the ribcage which colonic causes can cause severe abdominal distention. No gut sounds suggest ileus and high pitched pings indicate gas. A careful rectal palation can be used to detect meconium. Passage of a stomach tube is always indicated but getting

reflux from a foal can be difficult so use the largest tube you can safely pass. Make sure to complete the physical exam of the whole foal looking for systemic disease.

Laboratory aids:

- Haematology can be variable. Remember a total white blood cell count is only the balance of production and consumption so a normal white blood cell count is possible. A differential is very important as a result. Enteritis, which should be suspected in pyrexic foals, will often have a leukopenia with the presence of band cells, toxic neutrophils and an elevated fibrinogen and SAA.
- Biochemistry: particularly important in ruptured bladder cases in which electrolyte derangements occur, the most important of which is hyperkalemia which can be fatal. Diarrhoea can cause electrolyte disturbances. Any cause of abdominal distention can cause thoracic compression and decrease oxygenation.

Imaging:

- While we can't rectal foals, we can radiograph and ultrasound their abdomens (and chests). Contrast studies, either orally or rectally can be useful to image obstructions (meconium impactions or delayed gastric emptying)
- Radiographs can help diagnose small intestinal volvulus, large colon torsion, meconium impactions, diaphragmatic hernias, pyloric stenosis, and enteritis.

Abdominocentesis:

- This has a higher rate of complications than in adult horse due to the delicate nature of the foal's intestines so should be performed carefully, ideally using ultrasound to localise free fluid. If no fluid is found on ultrasound, consider NOT doing a tap. Place a skin bleb and sedate the foal as needed and perform the tap sterilely. If only a small sample is obtained, collect into an EDTA pot first.
- The WBCC in a foal's peritoneal fluid is lower than in adult horses. Cell counts greater than $1.5x10^9$/litre and total protein greater than 18 g/l are considered abnormal. Cytology should look for free bacteria, food or degenerate white blood cells. Culture the fluid if peritonitis is suspected. If uroperitoneum is suspected, measure the creatinine in the blood and the peritoneal fluid; if the creatinine in the peritoneal fluid is more than twice that of the serum, uroperitoneum is highly likely.

Faecal testing:

- Floatation: parasites (Strongyloides, ascarids)
- Culture: salmonella, Clostridium
- PCR testing : *Lawsonia, Rhodococcus*

Gastroscopy

- Do this with caution and remove as much air after the procedure as possible; mainly used for diagnosis of gastric ulcers (see below)

Exploratory laparotomy: see surgical notes

When should you consider surgery?

1. Pain that is unresponsive to analgesics
2. Deterioration despite medical management, especially if no firm diagnosis has been reached
3. Peristent high heart rate (>120 bpm)
4. Abnormal abdominal tap
    a. increase in TP or WBCC
    b. sanguinous peritoneal fluid
    c. evidence of sepsis
5. Evidence of an obstruction on radiographs or ultrasonography

Special considerations for foal surgery

1. Correct fluid, electrolyte and blood gas abnormalities before surgery if possible
2. If surgery can be done without removing the umbilical remnants, then avoid damaging them. However, if the umbilical structures are suspect, or not removing them diminishes visibility, then remove them.
3. Foals lose more heat via an open abdomen than adults so use warm air blankets and warm fluids for abdominal lavage.
4. Foals are more likely to get adhesions so minimise tissue handling and copiously lavage the abdomen.

SPECIFIC CONDITIONS

1. Enteritis
    a. Use laboratory aids
    b. Peritoneal fluid is normal unless there is a rupture
    c. Enteritis affect the large and small intestine. Ultrasonography shows fluid filled hypermotile large and small intestine.
    d. Clostridial enteritis is often fatal and requires immediate treatment. High mortality from *C. Perfringens* in the first two days of life. Cause a necrotising haemorrhagic

diarrhoea seen as colic, depression, shock and death. Some peracute cases are found dead. *C. difficile* can present two ways: as above, or as watery diarrhoea. Diagnosed by faecal PCR. Treatment is supportive plus IV antibiotics as gut is compromised.

e. There is a condition called necrotising enterocolitis which is frequently fatal. Below is a summary of the condition **from Professor Pam Wilkins**:

NECROTIZING ENTEROCOLITIS

Necrotizing enterocolitis is considered the most common acquired gastrointestinal emergency of human infants. The 1500 to 2000 infants that die every year from this disease in the United States and the large number of infants who develop short gut syndrome from this disease only represent the tip of the iceberg of the problems necrotizing enterocolitis causes. The widespread fear of necrotizing enterocolitis among neonatologists and pediatric surgeons has contributed in large part to the use of the intravenous route rather than the gastrointestinal tract for nourishing these infants for long periods. The pathogenesis of necrotizing enterocolitis is unknown but may result from a disturbance of the delicate balance among gastrointestinal perfusion, enteric organisms, and enteral feeding. Risk factors for necrotizing enterocolitis in human infants include prematurity, hypoxic-ischemic insult, and formula or breast milk feedings. The clinical spectrum of necrotizing enterocolitis is multifactoral and ranges from temperature instability, apnea, lethargy, abdominal distention, bilious residuals, septic shock, disseminated intravascular coagulation, and death. Medical management is usually adequate treatment for necrotizing enterocolitis although surgical resection and anastamosis may be required in the most severe cases. In the neonatal foal, necrotizing enterocolitis is probably one of the most underrecognized causes of gastrointestinal dysfunction and in the past has been attributed only to infection with anaerobic organisms including Clostridium perfringens type C and C. difficile. Although a specific form of enteritis in the foal is associated with intestinal infection by these organisms, most necrotizing enterocolitis is associated with prematurity or PAS in the infant and the foal. One should suspect necrotizing enterocolitis in any foal that is having difficulty tolerating oral feeding, demonstrating signs of ileus, or having episodes of colic and in any foal with occult blood, digested blood or frank blood in the stool or reflux. Foals exhibiting any of these clinical signs should not be fed orally if possible and should receive parenteral nutrition until gastrointestinal function returns to near normal. The mucosal barrier of the intestine is unlikely to be fully intact, and these foals are at risk for sepsis from bacterial translocation. One should institute broadspectrum antimicrobial therapy in these foals and, if any evidence of coordinated gastrointestinal motility is apparent, should administer sucralfate orally as a protectant.

2. Strangulation of the large or small intestine

a. Clinical signs: depression, anorexia, face covered in milk. Elevated respiratory rate, heart rate +/- pyrexia. Abdominal distention. Systemic deterioration and persistent pain despite analgesics.
b. Diagnosis: small intestinal strangulation often have a faster clinical course. Abdominal radiography very useful as shows loops of gas filled intestine. Abdominal US can also help and usually shows non motile, distended intestine. Remember to look for inguinal and umbilical hernias which have become strangulating.

3. Meconium impaction
    a. CS: meconium are dark brown pellets. Once the foal is digesting milk the foal passes pasty light brown faeces, but occasionally a foal can still have a meconium impaction and be passing the pasty faeces. Foals often strain to defecate and most impactions can be felt digitally (be careful as it is possible to rupture a foal's rectum) although there are also "high meconium impactions" which are not palpable per rectum, but visible on a barium study. Barium should clear a foal's stomach by two hours and reach the transverse colon by three hours.
    b. Treatment: a soapy enema is usually effective. I avoid phosphate enemas as hyperphosphatemia can result so I use very dilute chlorhexidine administered by a Foley catheter and a funnel using gravity flow. Analgesics are not always necessary but butorphanol can be effective. If this is not effective, mineral oil can be given by stomach tube (60-120 mls). Analgesia is not always required but if needed butorphanol (3-5mg) can be given IM. If the impaction does not respond quickly, the foal can be muzzled to avoid adding to the GI load and giving IV fluids with 5% dextrose. Acetylcysteine enemas can be used if soap enemas fail; these cause secretion into the gut.
    c. Make sure to examine the foal for other conditions which may have led to ileus/dysmotility as a meconium impaction can be secondary to another condition.
4. Gastric **ulcers (Notes courtesy of Professor Pam Wilkins)**

Gastric ulcer disease has been recognized in foals, and lesions vary in anatomic distribution, severity, and cause. In clinically normal neonatal foals (<30 days of age), gastric ulcers and mucosal desquamation have been documented. Because of these reports and other early reports of death following ruptured clinically silent ulcers in neonatal foals, for years many clinicians felt it necessary to treat critically ill neonates with antiulcer medication prophylactically. Recently, this paradigm has been challenged. The pathophysiology of gastric ulcer disease is described most reasonably as an imbalance in protective and aggressive factors. These protective factors are responsible for maintaining a healthy gastrointestinal tract by promoting adequate mucosal blood flow, adequate mucus and bicarbonate production, prostaglandin E2 production, epithelial growth

factor production, gastric afferent innervation, epithelial cell restitution, and gastroduodenal motility. Probably the most important factor is maintenance of mucosal blood flow. Hypoxia, NO, prostaglandins, and gastric afferent innervation influence mucosal blood flow. The aggressive factors include gastric acid, bile salts, pepsin, and enzymes. Few specific causes have been found for gastric ulcer disease in foals. Excessive administration of nonsteroidal anti-inflammatory drugs can result in ulceration of the glandular and squamous epithelium because of an inhibition of prostaglandin production, which leads to a decrease in mucosal blood flow and an increase in acid production. Nonsteroidal anti-inflammatory drugs also can impair the healing of lesions and rarely are indicated in neonatal equine medicine. In the critically ill neonate the suspected cause of gastric ulcers has shifted away from an excessive amount of intraluminal gastric acid toward gastric mucosal ischemia caused by hypoxia, low blood flow conditions, or both. Perforating gastric ulcers are more likely a manifestation of necrotizing enterocolitis than of excessive gastric acid. Shock, sepsis, or trauma can result in gastric mucosal ischemia, allowing for the disruption of epithelial cell integrity and permitting damage by aggressive factors or providing an environment suitable for the establishment of bacteria colonization. Impairment of mucosal blood flow also may result in reperfusion injury, allowing the formation of gastric ulcers. In the sick neonatal foal (<7 days of age) a wide variability in the intragastric pH has been documented depending on the type of disease, severity, and milk intake frequency and volume, suggesting that in the critically ill equine neonate, ulcer prophylaxis using histamine antagonists or proton pump inhibitors is not only unnecessary but unlikely to work. Clinically significant gastric ulcers can occur in the squamous, glandular, or both portions of the stomach as a primary problem or resulting from another problem. Clinical signs include diarrhea, abdominal pain, restlessness, rolling, lying in dorsal recumbency, excessive salivation, and bruxism. In the neonatal foal the only clinical signs present may be depression or partial anorexia until a more catastrophic event, such as perforation, occurs. Some lesions in the gastric mucosa extend from the pylorus into the proximal duodenum and can result in stricture of the pylorus and proximal duodenum. These foals are usually older (>1 month of age) and have a greater volume of reflux. Bruxism and ptyalism are also more prominent in these older foals. The most sensitive and specific method for diagnosing gastric ulcers is visualization by endoscopic examination. Unfortunately, the use of gastric endoscopy has led to recognition of relative nonlesions and ulcers resulting from other problems and of clinically significant disease states. The clinician should not stop simply when ulceration of the stomach is recognized with endoscopy but should examine that patient fully for other potential sources of the clinical signs. Other diagnostic tests may help in determining the severity of the ulcers, including fecal occult blood or gastric blood assessments, contrast radiography, abdominal ultrasound, and abdominocentesis. Endoscopy of the foal stomach carries an additional risk of exacerbating colic in the short term, unless the examiner ensures that as much introduced air as possible is evacuated from the stomach at the end of the procedure. The presence of a brown gastric reflux fluid may indicate the presence of bleeding ulcers

or necrotizing enterocolitis. Blood in the feces of the neonate is more consistent with a diagnosis of necrotizing enterocolitis, which can be associated with gastric ulcers. Contrast radiography is useful if one suspects delayed gastric emptying or pyloric or duodenal stricture in older foals. If a stricture has occurred, one will note a delay in complete emptying of barium from the stomach (>2 hours). Abdominal ultrasound may be useful to visualize free abdominal fluid and gastric or small intestinal distention if one suspects a perforation. One can visualize portions of the descending duodenum, and a thickened duodenum should increase the index of suspicion for duondenal stricture. Abdominocentesis also may confirm perforation. Traditional therapy for gastric ulceration includes mucosal adherents, histamine type 2 receptor antagonists, proton pump inhibitors, and antacids. The most widely used mucosal adherent is sucralfate, which is a hydroxy aluminum salt of sucrose. The main therapeutic action of sucralfate is to bind to the negatively charged particles in the ulcer crater. At a pH less than 2, sucralfate is converted to a sticky viscous gel, which adheres to the ulcer crater and remains adhered for 6 hours, but at a higher pH, sucralfate remains in a suspension. Sucralfate is still effective because it inhibits pepsin and buffers hydrogen ions. Other important actions of sucralfate include stimulating production of prostaglandin E, which maintains mucosal blood flow; increasing bicarbonate secretion; stimulating mucous secretion; decreasing peptic activity; and binding epidermal growth factor. The histamine type 2 receptor antagonists include cimetidine, ranitidine, and famotidine. These compounds block the interaction of histamine with the histamine type 2 receptor on the parietal cell, resulting in inhibition of gastric acid secretion. Clinically normal neonatal foals have a highly acidic gastric fluid that is influenced by sucking. Intravenous and oral administration of ranitidine increases intragastric pH in normal foals but critically ill neonatal foals have a blunted response to ranitidine administration. One possible conclusion reached from these studies is that in critically ill neonatal foals, gastric ulcers may not be caused by an increased intraluminal gastric acidity. The most commonly used proton pump inhibitor is omeprazole. Omeprazole inhibits the secretion of hydrogen ions at the parietal cell by irreversibly binding to the H_2,K_2-ATPase proton pump of the cell. Most of the lesions in older foals were healed after daily administration of omeprazole for 28 days according to one report. Omeprazole effectively increases intragastric pH in normal neonatal foals and in ill neonatal foals that have decreased pH prior to treatment, suggesting a functionally intact gastric mucosa in those foals. The efficacy of omeprazole in treating very ill foals without the ability to generate gastric acid has not been demonstrated, but is likely similar to the findings of ranitidine in critically ill foals. Table 1 summarizes the therapeutic agents for treating gastric ulcers in foals. Prophylactic treatment of critically ill neonates for gastric ulcers has been standard therapy for years because of the evidence of clinically silent ulcers. This approach may not be appropriate for several reasons. An increased incidence of nosocomial pneumonia and systemic sepsis is associated with high gastric pH in human patients in intensive care. Patients in intensive care units treated prophylactically with histamine type 2 receptor antagonists are more likely to develop pneumonia

during ventilation therapy and gastric colonization with potentially pathogenic bacteria or yeast. An acidic environment appears to protect against airway colonization by bacteria of intestinal origin and bacteria translocated across the gastrointestinal tract. Pathogenesis of ulcers in the neonatal foal most likely does not involve increased intraluminal gastric acid but instead may be caused by decreased mucosal perfusion associated with shock, hypoxia, and hypoxic/ischemic insult to the gastric mucosa. Several reports have now revealed that gastric ulcer disease in equine NICU patients is independent of pharmacologic prophylaxis. In one study, despite decreased prophylactic treatment, the incidence of gastric ulcers found in these foals at necropsy had decreased significantly with time. The decrease was attributed to overall improvement in management of these cases. Similarly, in a human intensive care unit, the incidence of stress ulcers decreased independent of the use of prophylaxis. Early treatment of sepsis, sufficient oxygenation, improved monitoring, institution of enteral feedings, and improved nursing care may contribute to the reduction in gastric ulcers in the neonatal patient. Use of histamine type 2 receptor antagonist and proton pump inhibitors apparently may not be necessary; however, in some instances sucralfate may be useful. Sucralfate reduced the rate of bacterial translocation in a rat model during hemorrhagic shock and also may prohibit the generation of acute gastric mucosal injury and progression to ulcer formation induced by ischemia-reperfusion. In a human medical intensive care unit, airway colonization by new pathogens occurred more frequently in patients receiving agents that increased gastric pH than in those receiving sucralfate. In the critically ill neonatal foal, risk factors for gastric ulceration have not been identified clearly, although foals treated routinely with nonsteroidal anti-inflammatory drugs may be at increased risk for gastric lesions. Prophylactic treatment for gastric ulcers in critically ill neonates may not be necessary, and one should consider carefully the pros and cons of their use before their administration. Some recent work has identified an association between acid inhibition and the development of diarrhea in neonatal foals so prophylactic treatment may not simply be of economic cost but potentially harmful if not necessary for clinical ulcer disease.

TABLE 1

Therapeutic Agents for Treating Gastric Ulcers in Foals

DRUG CATEGORY	DRUG	DOSE	ROUTE	FREQUENCY
Mucosal protectant	Sucralfate	10-20 mg/kg	p.o.	t.i.d. to q.i.d.
Histamine type 2 receptor antagonist	Cimetidine	10-20 mg/kg	p.o.	q4h
	Ranitidine	6.6 mg/kg	IV*	q4h
		5-10 mg/kg	p.o.	b.i.d. to q.i.d.
		0.8-2.2 mg/kg	IV	q.i.d.
Proton pump inhibitor	Omeprazole	4 mg/kg	p.o.	s.i.d.
		1-2 mg/kg	p.o.	s.i.d. (prophylaxis)
Antacids	Milk of Magnesia	2-4 oz	p.o.	s.i.d. to b.i.d.
	Maalox	240 ml	p.o.	q4h

Adapted from Barr B: Gastric ulcer prophylaxis in the critically ill equine neonate. In Wilkins PA, Palmer JE, editors: *Recent advances in equine neonatal care*, Ithaca, NY, 2001, International Veterinary Information Service (A0413.1101).
**IV*, intravenous.