Gastrointestinal Disorders in Equine Neonates

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Abstract

Foals can suffer from a variety of gastrointestinal (GI) disorders that can be challenging to diagnose and treat and that many times differ to the typical presentation of an adult horse. In the field, there are limitation of diagnostic tools, therefore, it is important to recognize clinical signs that could help the practitioner direct the treatment, and advise referral to a hospital. This review intends to point out the most common causes of gastrointestinal disorders in equine neonates, as well as some rare conditions that may come across in the daily equine ambulatory practice.

History and Physical Examination

History is of utmost importance when dealing with equine neonates. Is the foal at term? And was the foaling and placenta observed? Colostrum intake and quality plays an important role, since failure of transfer of immunity can lead to a number of gastrointestinal disorders. The age of the foal can help orient the diagnosis, for example if the problem is congenital, or acquired after birth.

The initial examination should include observation of the foal's interaction with the mare, including its ability and desire to find the udder, appropriate suckle reflex, and ability to swallow

ingested milk. It's very important to observe any evidence of prolonged recumbency like pressure sores, as well as fecal staining in the hind end. Medication history is important since neonates are more prompt to GI disorders due to prolonged use of NSAIDs, and they are not exempt to antibiotic associate diarrhea.

Disorders of the oral cavity

While assessing the suckle reflex, thoroughly palpate the foal's palate for any evidence of malformations. Cleft palate is a rare disorder that affects about 0.02% of foals. Mutation has been identified in some cases, hence the possibility of being a hereditary disorder. In equids, it is restricted to the hard or soft palates, therefore classified as secondary. Clinical evidence includes nasal discharge of milk during nursing, and coughing because of aspiration of milk. Signs are usually noticed shortly after birth, but in smaller defects, it can be seen a few days after. Diagnosis is made by palpation or endoscopic observation; small defects of the soft palate might be evident just after careful oral endoscopy. Differential diagnoses for nasal regurgitation of milk includes immature nasopharyngeal function, which usually resolves over time. Potential complications include aspiration pneumonia, failure of passive transfer and rhinitis. If treatment is to be attempted, surgical repair is recommended. The prognosis is generally poor, and depends on the location and size of the defect. Prior to treatment, the presence of concurrent congenital conditions should be ruled out, as well as the presence of secondary complications. Medical management is usually unrewarding

Dysphagia in neonates can be due to morphologic or functional abnormalities. Morphological are uncommon, and besides cleft palate, cases of laryngeal masses, cysts, and dorsal displacement of the soft palate have been reported. Functional include generalized weakness and neurologic deficits of cranial nerves affecting normal deglutition. Pharyngeal dysfunction is well

documented in foals and can be predisposed by prematurity or hypoxic events. The dysfunction can resolve but may require days to weeks of supportive care, with a favorable prognosis for life and performance.

Disorders of the esophagus

Disorders of the esophagus can include intra- and extraluminal obstructions. Most of the extramuminal obstructions will be due to congenital abnormalities such as vascular ring or estenosis. Vascular ring is usually secondary to a persistent right aortic arch. Clinical signs often consist of milk regurgitation.

Disorders of the stomach

Gastric ulceration

Gastric ulceration has a reported prevalence of above 25%, and it is most often noticed in the squamous mucosa adjacent to the margo plicatus. In one study, squamous ulceration was noted more commonly in older asymptomatic foals, whereas glandular ulceration, especially in the cardiac region, occurred more commonly in neonates with another primary clinical disorder. Gastric ulceration is a concern in neonatal foals because clinical signs are often not evident until ulceration is severe, and in some cases until gastric rupture has occurred. Clinical signs of gastric ulceration in foals often includes decreased appetite or signs of colic; with bruxism or ptyalism less commonly observed. Abdominal pain may be worse after nursing

Confirmation of gastric ulcers require endoscopic examination. A 1 meter endoscope may allow at least for evaluation of the squamous and glandular mucosa along the greater curvature. At least a 2 meter may be necessary for evaluation of the antrum or duodenum; and some foals can present antral lesions without ulceration elsewhere in the stomach. Due to the liquid diet, neonates do not require a prolong fasting for gastroscopy. The squamous mucosa in neonates is

thinner, with more of a light pink appearance compared with adults. Care must be taken when inflating the stomach, and insufflated air should be removed.

The pathophysiology of ulcer disease is likely complex and multifactorial, involving both intrinsic (acid production) and extrinsic factors such as NSAIDs, stress or disorders leading to delayed gastric emptying. Intrinsic gastric protective factors such as mucosal blood flow can be affected in ischemic conditions typical of neonates. Hypovolemia and hypotension commonly accompany many of the primary conditions for which neonatal foals require intensive care. Thus, perfusion of the GI tract and other vital organs can become compromised.

A retrospective study found no correlation between the incidence of gastric ulcer disease and the administration of pharmacologic prophylaxis. Alterations in gastric perfusion play a more important role than hydrochloric acid in the development of ulcers in severely compromised foals. Prolonged periods of gastric alkalinity potentially could allow for alteration of bacterial flora within the GI tract, increasing the propensity for bacterial translocation across a compromised GI mucosal barrier. Supportive care likely provides more protection against ulcer formation than would any medication directed to the suppression of hydrochloric acid. In ambulatory foals receiving repeated administration of NSAIDs, or those with diarrheal disorders may warrant prophylactic treatment. Duodenal ulceration can occur in neonates, but the syndrome of gastroduodenal ulceration typically occurs in foals between 2 and 6 months of age.

Small Intestine

Ulcerative duodenitis usually affects foals less than 4 months old, and can lead to duodenal strictures if left untreated. Enterogastric reflux, increase in gastric silhouette, and delayed gastric emptying can be indication of duodenal stricture. Gastroscopy and duodenoscopy are valuable diagnostic tools. Using a 3-m endoscope, severe gastric ulceration and distal (reflux) esophagitis

with linear erosions are often observed. Ulcers are also encounter in the glandular gastric mucosa and duodenum, and strictures can be observed. If gastric outflow obstruction is suspected, contrast gastrointestinal radiography is recommended. A normal foal should have evidence of barium in the duodenum by 5 to 10 minutes after barium administration. Medical treatment can be attempted by gastric decompression, use of antiulcer medication, and supportive care, however, if the ulceration has progressed to fibrosis, and medical management does not result in improvement, surgical intervention is needed.

Intestinal stages of *Parascaris equorum* can cause intestinal obstruction, intussusception, abscessation and even rupture in foals, normally after 6 months of age. Ultrasound examination may demonstrate the parasites in the small intestine. Distal jejunum and ileum are the most common site. Heavily parasitized foals should be treated with a slow-acting drug such as a benzimidazole, which reduces the numbers gradually. Ivermectin, which is also slow acting but highly effective against this parasite, can be given 3 weeks later.

Intussusception usually cause chronic intermittent colic signs, however it can also be acute and difficult to distinguish from volvulus. Ultrasonography can be helpful in diagnosis by demonstrating the typical target or "bull's eye" lesion formed by the intussusceptum and intussuscipiens. Many foals have asymptomatic intussusceptions, that are dynamic and diagnosed as an incidental finding.

Ileus is a common secondary complication in foals suffering from prematurity, sepsis, or ischemic-hypoxic syndrome, or a any condition causing intestinal inflammatory response. The newborn gut is particularly sensitive to systemic hypotension and hypoxemia. Clinical signs include gastric reflux or abdominal distension. Often, signs of colic are not seen in severely ill

foals due to the obtunded mentation. It is crucial to monitor the foal for reflux frequently and to routinely monitor abdominal circumference.

Colon, rectum and anus

Meconium impaction is the most common cause of colic in neonatal foals. It is normally passed beginning within the first few hours of birth and should be passed completely within 48 hours. Colic signs usually begin from 12 to 24 hours of age. Colic signs range from decreased suckling, depression, straining to defecate and tail flagging to severe signs of pain such as rolling and abdominal distension. Diagnosis is bases on history, clinical signs, and per rectum palpation with a gloved, well lubricated finger. Contrast radiography may help to identify the impaction or to distinguish from conditions such as colonic atresia or aganglionosis.

Enemas are often the first treatment attempted. Warm soapy enemas with a nonirritating soap such as Ivory are often effective, but some cases may necessitate phosphate-containing products (Fleet), however repeated use may cause electrolyte disturbances, including hyperphosphatemia. If unsuccessful, a retention enema with acetylcysteine (40 mL, final concentration of 4%) is recommended, using a Foley catheter, left in place for 30-45 minutes

In cases of intestinal atresia, foals typically present within the first 2 to 48 hours of life with progressive signs of colic and abdominal distension. Foals do not pass meconium but may have mucus in the rectum. Survival is not possible without surgical correction, and the prognosis is guarded even with surgical intervention.

Aganglionosis is most commonly seen in foals with the lethal white syndrome. This is an autosomal recessive mutation that leads to absence of submucosal and myenteric ganglia from the distal small intestine and large colon. The condition is most commonly seen in American Paint Horses born to overo-over cross-mating. These foals present with signs of colic within 12-

24 hours of life. Therapy is not available and euthanasia is recommended. Genetic testing is available and recommended for at-risk horses.

Septic Peritonitis

When present it is often associated with gastric rupture secondary to gastric ulceration, as well as due to contamination secondary to septic omphalitis. Severe enterocolitis with bacterial translocation can develop in septic peritonitis.

Diarrhea

Diarrhea is a common GI disorder in neonate foals, and may or may not cause colic signs. Foall heat diarrhea, in ages 5-15 days should not cause clinical signs of pain, and foal's mentation is unchanged; it is believed that this occurs in response to establishment of normal flora. In foal's infectious causes of diarrhea include bacteria (Clostridium spp., Salmonella spp.) and viruses (rotavirus, coronavirus). Asphyxia-associated diarrhea is probably the most common cause in severely ill foals with ischemic encephalopathy. Diarrhea caused by infectious agents can cause secondary lactase deficiency, requiring supplementation with the enzyme as part of the treatment. Parasitic and protozoal agents such as *Strongyloides westeri* and *Cryptosporidium parvum* respectively can cause diarrhea in foals, however less commonly than bacteria or viruses.

Feeding foals with GI disorders

Neonatal foals do not handle fasting as well as adults, hence continuing feeding is important. In recumbent foals, enteral fluids is often not well tolerated. Since the activity level is decreased, these foals required less calories for maintenance than a normal foal (20% of body weight). It is better to start at no more than 5% of the foal's body weight, spaced at 1 to 2 hour interval. Since

5% is probably not enough, supplementation with dextrose or parenteral nutrition might be needed.

If the foal develops reflux (volume of >50 mL), feedings should be discontinued until reflux is no longer obtained and there is evidence of increased motility.

In general, it should be attempted to continue some degree of enteral feeding, even in small volumes. Although the foal can be maintained with parenteral nutrition, trophic feeding of enterocytes has been proven to be beneficial to the health and development of the intestinal tract.

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