DIARRHEA, COLIC, AND NASTY GI DISEASES OF THE FOAL

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ASSESSMENT OF COLIC IN THE NEONATE
Signalment, history, and physical exam

The age, breed and sex of a foal can help categorize the most likely diagnosis. The most common cause of colic in the neonate that is a few hours old is meconium retention. Enterocolitis can occur shortly after birth as well. Uroperitoneum is a common cause of colic in the foal less than a week of age (usually less than 72 hours of age). Congenital abnormalities are usually revealed within the first few days of life. Important facts to ascertain include the duration of colic, the degree of pain and progression, passage of feces, straining to urinate and/or defecate, appetite and response to treatment. A review of any medications the foal has received for an unrelated problem such as antimicrobials or NSAIDs may be valuable.

Straining is common with small colon obstruction and uroperitoneum, and differentiation between straining to urinate or defecate can be difficult. With the former, the back is typically ventroflexed, whereas with the latter, it is typically dorsoflexed. The evaluation of a colicky foal can often be guided initially by the presence or absence of gross abdominal distention. The most frequent cause of an enlarged abdomen is gas accumulation within the cecum and/or large colon. Abdominal distension can also be caused by fluid or free gas within the peritoneum. In general, foals with strangulating lesions have uncontrollable pain initially, which will eventually lead to depression and systemic shock. One should always look for obvious causes of colic, such as inguinal or umbilical herniation.

Diagnostic techniques

The duration and severity of signs, together with evaluation of response to chemical analgesia, will often dictate surgical versus non-surgical management. Severe, unrelenting pain that is poorly or unresponsive to analgesics is often unto itself a valid criterion for surgical exploration. Foals are less tolerant of abdominal pain and the degree of pain exhibited does not always correlate with the severity of the lesion. Foals appear to be more susceptible to adhesion formation post-operatively.

Rectal palpation is limited to digital palpation in foals. This is most useful for meconium retention (palpation of impaction and presence of normal feces vs. meconium) and to rule out complete atresia.

Nasogastric intubation is as important in the evaluation of foals as it is for adults with colic. Obtaining nasogastric reflux from some foals can be difficult, especially if the stomach is significantly distended with resulting compression of the lower esophageal sphincter. A stallion catheter with stylet combines ease of passage, ease of identification within the esophagus, and appropriate luminal size for reflux of neonatal and early suckling foals. Lack of nasogastric reflux does not rule out small intestinal obstruction. Assessment of gastric size via ultrasound can be extremely useful.

Abdominal ultrasound in the colicky foal has essentially become an extension of the physical examination and can be performed with the foal standing or in lateral recumbency. Knowledge of normal anatomy is critical to accurate interpretation of findings. During a basic exam, one should evaluate small intestinal size, character of intestinal contents (fluid, gas, meconium in the small colon), thickness and character of the intestinal walls, amount and character of free peritoneal fluid or gas, bladder size and integrity, gastric size and contents, duodenal size and wall thickness, and umbilical structures. The stomach is easily imaged from the cranial aspect of the ventral abdomen in neonatal foals or more commonly from the left craniolateral abdomen caudal to the liver. Additional evaluation of the kidneys, liver, and spleen should be pursued if indicated. If a hernia is suspected, the hernial sac should be examined. Peritoneal fluid should be examined for echogenicity and volume. Increased echodensity is consistent with a highly cellular fluid, whereas anehoic fluid is consistent with uroperitoneum.

Plain radiography can provide more information in foals with abdominal pain, especially for diagnosis of large colon intraluminal obstructions, such as with meconium, bedding or sand, and to identify the presence of a large gas cap in cases of gastric outflow obstruction. Contrast radiography is helpful for evaluation of gastric outflow or confirmation of atresia. Gastroscopy and duodenoscopy are used to evaluate foals with suspected gastric or duodenal ulceration or gastric outflow obstruction. Peritoneal fluid evaluation is a useful adjunct when the distinction between medical and surgical disease is unclear. Many consider the incidence

and severity of complications associated with this procedure in foals greater than that in adults and ultrasound guidance is recommended.

SPECIFIC CAUSES OF COLIC IN THE NEONATAL FOAL
Congenital anomalies

Congenital anomalies should be considered in all colicky newborn foals. Common abnormalities include atresia coli, atresia recti and atresia ani. Foals affected with Overo lethal white syndrome are white, although some may have small darker markings. They are typically normal at birth but become colicky within the first 24 hours. Genetic testing is available to identify carriers of the mutation (www.animalgenetics.us or www.vgl.ucdavis.edu).

Uroperitoneum

Newborn foals with uroperitoneum most typically present with abdominal distention and low-grade pain. Occasionally, affected foals can also present with extreme lethargy or obtundation due to electrolyte abnormalities. Differentiation between gas and fluid distention can generally be made by careful physical examination and confirmed by ultrasound examination. Uroperitoneum is a medical emergency which requires surgical therapy. Volume depletion, acid-base and electrolyte status should be addressed prior to anesthesia and surgical repair.

Meconium retention

Retention of meconium within the intestinal tract represents the most common gastrointestinal anomaly in newborn foals. Meconium is a sterile concretion of intestinal secretions and sloughed cells that accumulate in the tract during gestation. Foals with longer gestational periods (>345 days) may be at greater risk for developing meconium retention. Many horse owners and farm managers routinely administer soapy water enemas to all newborn foals. Colicky signs can vary from very mild to severe, and often include straining. Appetite can remain normal to slightly decreased until severe impaction and bloat, but for some foals, decreased appetite is the first observable abnormality. Digital rectal palpation can identify caudal obstructions and presence of meconium-type feces, and ultrasound examination will often provide a definitive diagnosis. Meconium impaction is often observed as a homogenous intraluminal mass of variable echogenicity. Typically, most intraluminal obstructions will result in fluid accumulation and distention of the intestinal segments orad to the site of obstruction. Enemas, IV fluid therapy, laxatives and pain management are the basis of treatment. Surgical correction is rarely (but occasionally) necessary. Repeated enemas with caustic substances such as dioctyl sodium succinate (DSS) can cause rectal edema making passage difficult. Foals that have had repeated enemas or long-standing impactions are often leukopenic due to mucosal irritation and subsequent bacterial translocation; thus, systemic antimicrobial therapy in such cases is warranted. Warm soapy water enemas should be the first therapeutic option. Use a small, well-lubricated urinary catheter and 200-500 mL of soapy (gentle soap such as Ivory or Dove) water via gravity flow only. If that is not successful, retention enemas using acetylcysteine are often effective. If using Mucomyst (liquid), add 40 ml of 20% solution to 160 ml of water to make a 4% solution. If using the power, add 8 grams of powder and 1½ tablespoons of baking soda to 200 ml of water. In a sedated, recumbent foal, place a lubricated Foley urinary catheter approximately 2-4 inches into the rectum and inflate the balloon, using care not to over-inflate the rectum. Infuse 120-200 mL of the acetylcysteine solution, clamp the foley, and allow at least 15 minutes of retention time before removing the catheter. Oral laxatives such as mineral oil (100-200 mL) administered via nasogastric tube are also commonly used.

Strangulating small intestinal lesions

The diagnosis of small intestinal intussusception is commonly made via ultrasonography. A series of concentric circles are seen (target lesions or "bull's eye" appearance). Other strangulating lesions include volvulus and entrapment within a mesenteric rent. The differentiation between non-strangulating intestinal ileus and strangulated bowel can be difficult if the strangulation is early, but compromised intestine will develop mural thickening and progressive luminal distention over time.

Gastroduodenal Ulcer Disease (GDUD)

Gastroduodenal ulcer disease is more commonly seen in the late suckling foal. The most common clinical signs include bruxism, excessive salivation, colic after suckling or eating hay, weight loss and diarrhea. Ultrasound may identify a duodenal lesion, (distention and/or mural thickening) or gastric distention. Plain films will often revealed gastric distention with a large gas cap. The diagnosis of GDUD is best made using a combination of signalment, history, physical examination findings, gastroscopy and imaging.

Foals with GDUD can develop an ascending cholangitis or cholangiohepatitis if the ulceration leads to stricture formation distal to the opening of the common bile duct within the small intestine. Similar changes can also occur in animals with intestinal stasis due to enteritis of the duodenum. Characteristic changes seen ultrasonographically include increased liver mass, increased echogenicity of the parenchyma, and dilation of the biliary tree. If a gastric outflow obstruction has been documented, therapy is surgical. Depending upon stricture location, either a gastrojejunostomy or gastroduodenostomy is performed. Otherwise, medical therapy can be attempted with omeprazole and supportive care. But, stricture formation may occur subsequently. Prognosis is fair with medical therapy and fair-good but expensive with surgical therapy.

FOAL DIARRHEA

Diarrhea is one of the most common problems of newborn foals. Diarrhea can be a primary problem or secondary to sepsis. Searching for a specific pathogen in the feces of these foals is often unrewarding. Taking a thorough history is important to rule in/out age linked causes of diarrhea such as foal heat diarrhea, as well as to rule out other less common causes such as sand ingestion. Frequency of the diarrhea, suckling activity of the foal, and whether or not any other foals are affected on the premises are all very pertinent questions. Complications of diarrhea include severe dehydration, metabolic acidosis, electrolyte derangements, hypoproteinemia and bacteremia.

Noninfectious diarrhea in the neonate

Foal heat diarrhea is so named due to its occurrence during the first heat cycle post-partum (5-15 days). One study suggests that the cause of foal heat diarrhea is inoculation of microflora and maturational changes of the gastrointestinal tract. Foals typically do not demonstrate the signs of systemic illness observed with infectious causes. They are usually bright, alert and suckling with normal vigor. Nutritional causes of diarrhea in neonates are infrequent, with excessive milk intake that may occur when feeding orphaned foals and consumption of sand occurring most commonly. Asphyxia related gut injury (also called neonatal gastroenteropathy) can occur with severe oxygen deprivation at birth. Affected foals typically display other signs of neonatal encephalopathy and develop ileus. Miscellaneous causes of noninfectious diarrhea include gastric ulceration, ingestion of sand and irritants. Digital examination of the rectum may reveal sandy material in foals that have consumed excessive quantities of sand. Abdominal radiographs can confirm the presence of sand. Treatment for sand includes small amounts of mineral oil and psyllium via nasogastric tube.

Infectious diarrhea in the neonate

Up to 50% of diarrheic foals less than 30 days of age are bacteremic, based upon two reports.[2,3] As will be discussed in the sepsis lecture, diarrhea is often one of the primary presenting complaints for septic foals. The most common specific aerobic intestinal pathogen is *Salmonella* sp. Although outbreaks of Salmonellosis are possible in horses of any age, most occurrences in foals are isolated cases. Mares appear to be the primary source of infection and are often positive for fecal shedding when their foals are diagnosed with salmonellosis even though they rarely have clinical signs of disease. Most affected foals have moderate to severe clinical signs that include fever, diarrhea, dehydration, profound depression and reduced appetite. Diarrhea can vary in both consistency and volume, and may contain blood. Colic is common in the early stages of the disease. A complete blood count usually reveals neutropenia, with a left shift and toxicity, but is replaced by a rebound neutrophillia as the disease becomes chronic. Extra-intestinal disease is common in foals less than 2 months of age and includes uveitis, infective synovitis, osteomyelitis, pneumonia, and meningitis. A diagnosis is typically achieved using blood culture, fecal culture, fecal PCR, or a combination. *Escherichia coli* is the most common cause of bacteremia in newborn foals, but is an uncommon primary enteric pathogen. Intestinal manifestations of *Rhodococcus equi* are common.[4]

Intestinal clostridiosis can be caused by *Clostridium perfringens* biotypes types A and C and *Clostridium difficile.* Disease induced by *C. perfringens* biotype C is associated with hemorrhagic diarrhea, abdominal distention, colic, circulatory shock and high mortality. [1] The disease often occurs within the first 48 hours of life and is most commonly seen in vigorous foals with adequate passive transfer. In recent years, there has been an emergence of enteric disease in newborn foals associated with *C. perfringens* biotype A. Clinical signs are more variable, but may include transient bloody stool, colic and fever. Mortality is reduced when compared with disease induced by *C. perfringens* biotype C. This biotype also appears to be commonly present in the feces of healthy young foals. The role of *C. difficile* in foal diarrhea is not clear. A well-defined cause of enterocolitis in foals less than 4 days of age, but appears rare in older foals. Prevalence varies with geographic location. A diagnosis is typically achieved using toxin analysis, fecal Gram stain, and/or blood culture.

Group A rotavirus is the most common cause of infectious diarrhea in foals. Typically, several foals are affected over a short period of time. The disease is highly contagious and has a very short incubation period.

The diagnosis of rotavirus is often made on the basis of epidemiological findings, physical examination findings (diarrhea, depression, reduced appetite, ± fever), and fecal antigen tests. Fecal antigen tests are sensitive and provide rapid confirmation. Treatment is supportive and a maternal vaccine is available and may confer modest protection. There are recent reports of coronavirus acting as a primary pathogen in young immunocompetent foals. It is unlikely that coronavirus infection is responsible for outbreaks of foal diarrhea. Diagnosis is possible with fecal ELISA or EM.

The role of cryptosporidium in foal diarrhea remains controversial. Infection rates have been reported between 15 and 31% in suckling foals. Cryptosporidium has been associated with fatal outcomes in foals and should be considered as a cause of diarrhea in compromised, hospitalized neonates. One can detect oocysts in fecal samples via acid-fast staining, immunofluorescence assay, or flow cytometry. Treatment is generally supportive. Prevention includes environmental disinfection and isolation of infected foals. *Giardia* infection rates in foals have been reported to be as high as 35%, but data to associate shedding with disease is lacking. *Strongyloides westeri* may cause diarrhea in neonates if a large amount of larvae are acquired from the mare's milk after birth.

Treatment

Regardless of cause, therapy for most foals with diarrhea is predominantly supportive, includes crystalloid and/or colloid fluid therapy, and correction of acid-base and electrolyte derangements. Neonates with diarrhea should be treated early and aggressively with broad-spectrum antibiotics due to the likelihood of bacteremia. Please refer to my sepsis notes for additional information on antimicrobial selection. Older foals (>1 month) that appear systemically healthy or 7-10 day old foals with suspected foal heat diarrhea that appear otherwise normal likely do not need antimicrobials if the diarrhea is self-limiting and uncomplicated. Di-tri-octahedral smectite (Biosponge, Platinum Performance) neutralizes *C difficile* toxins A and B and *C perfringens* enterotoxins in vitro. *Saccharomyces boulardii* (SynerGI, Stellar Mark) has been shown to help decrease the severity and duration of clinical signs in adult horses. The use of Lactobacillus pentousus did not prevent diarrhea (it actually worsened it) in foals and is not recommended[5].

Bismuth subsalicylate is commonly used in foals with diarrhea. Its anti-diarrheal action is through stimulation of fluid and electrolyte absorption and by inhibiting the synthesis of prostaglandins (when hydrolyzed to salicylic acid) involved in intestinal inflammation. The use of motility-modifying agents, such as atropine or loperamide, is contraindicated in foals with enteric infections where bacteria or bacterial toxins may invade or damage the intestinal mucosa (e.g., clostridial infections, *Salmonella*). Loperamide may be useful in other forms of diarrhea, but prolonged use is not recommended. If clinical improvement is not apparent by 48 – 72 hours then further use is unlikely to be helpful.

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