

ROTAVIRUS DIARRHEA

**William V. Bernard, DVM
Rood & Riddle Equine Hospital
PO Box 12070; Lexington, KY 40580**

Diarrhea is the most common gastrointestinal disease that is encountered in the foal. Severity ranges from a mild self-limiting condition to a severe life-threatening disease. The severity of the condition varies with the etiology, ensuing degree of dehydration, electrolyte and acid-base abnormalities and the development of endo/exotoxemia and sepsis. All age foals are affected, however, neonates are more susceptible to severe and life-threatening complications. The rapid dehydration, acid-base disturbances, and electrolyte abnormalities can occur before a significant change in fecal character is seen. This may occur as a result of massive fluid accumulation in the lumen of the gastrointestinal tract (third space loss). Infectious diseases can result in severe epidemics with major health and economic impact. Rotavirus usually occurs in young foals less than 2 months of age. Rotaviral particles have been reported in up to 30% of foals with diarrhea. Most adult horses have antibodies to rotavirus. Epidemics of enterocolitis as a result of rotavirus are not uncommon. The disease can be extremely contagious and difficult to control. Group A rotavirus is the usual cause of the older foal rotavirus diarrhea, group B rotavirus has been associated with severe epidemics in neonates and has been frequently called neonatal or 24-hour scours. The source of the virus is likely to be adult shedders. Subclinical infection is thought to be common in foals, subclinical shedding may occur up to 8 months of age. The virus may persist for up to 9 months in the environment. Heavy contamination of the environment occurs when population pressures and maintenance practices favor contamination.

Etiology

Foal diarrhea can be caused by infectious agents (virus, bacteria, protozoa, parasites) nutritional upsets, changes in flora, and antibiotics. The major cause of enteritis / enterocolitis is infectious in nature, either bacterial or viral. The major viral enteritis of foals is rotavirus, although coronavirus and adenovirus have been associated with diarrhea in foals. The pathogenicity of rotavirus in foals is well documented; however, corona and adenovirus infections have not been well established and may in many circumstances be secondary to other pathogens. There are two types of rotavirus infection in foals. Type A results in the more typical older foal diarrhea, whereas type B results in a neonatal diarrhea affecting the foal in the first few days of life. Viral diarrheas can be highly contagious with rapid development of epidemics.

Pathophysiology

The critical and often life-threatening nature of the infectious diarrheas is related to their direct influence on the cells of the gastrointestinal tract. Rotavirus invades the absorptive cells of the villus. These cells are destroyed resulting in a proliferation of crypt cells

(often immature), resulting in a loss of the absorption of substrates and increased secretion. Decreased absorption of substrate exacerbates the diarrhea with increased fluid loss. The combination of decreased absorption and secretion can result in rapid dehydration, electrolyte loss, and acid-base disturbances. If uncomplicated, the diarrhea can be self-limiting; however, if complications such as secondary bacterial involvement and subsequent endo-exotoxemia result, the disease can become severe and life threatening.

Clinical Presentation

The clinical presentation of a foal with diarrhea will vary considerably depending upon severity, age, and secondary complications. The etiology of the diarrhea is difficult to impossible to interpret based off clinical signs. Initial signs of disease may well be non-specific: fever, depression, and inappetence. Physical evidence of diarrhea need not be seen early in the course of the disease, whether mild or severe. Abdominal pain, mild or severe, may be present and can exist before clinical evidence of diarrhea. Other signs of pain, such as tachycardia and/or tachypnea may be present. Gastric reflux may be present. Systemic evidence of shock (endotoxic, septic, exotoxic) is variable, however, can be very severe.

Differential Diagnosis

Differential diagnosis includes a variety of conditions that may cause abdominal pain or non-specific signs of depression and inappetence, plus or minus a fever.

Diagnosis

Diagnosis is based on fecal evaluation and identification of viral particles. Electron microscopy is not practical in the clinical setting. A latex agglutination test and ELISA test are available and do compare well with electron microscopy. Bacterial cultures may rule out other causes.

Management

The basic tenet of therapy for enterocolitis is support of hydration and electrolyte loss, if necessary, and amelioration of toxic influences of bacteria and secondary bacterial infection. Fluid loss and resultant dehydration can be life threatening. The neonate is particularly susceptible to fluid losses and may require therapy for hypovolemic, as well as septic, shock. Significant amount of fluid loss can occur even before physical evidence of diarrhea. Electrolyte loss is variable and dependent upon the severity, degree of secretory component of the diarrhea, and reabsorption of secreted electrolytes. Restriction of oral intake of milk (NPO) can be very useful in therapy of neonates or young foals with diarrhea. Diarrhea in the neonate and young foal can be exacerbated by the osmotic influences of milk intake. "Resting" of the gastrointestinal tract to allow for recovery of damaged enterocytes can be a beneficial part of therapy. Neonates must be

cautiously deprived of milk intake, as glucose energy stores are limited at this age. Parenteral nutrition may be necessary and allows increased periods of time without milk intake. A strong, robust neonate may be cautiously held off milk for 24 hours without parenteral caloric support. A period of 24 hours of gastrointestinal rest may greatly aid the resolution of neonatal diarrhea. Many of the agents that produce diarrhea in the foal produce toxins that may result in systemic influences. Therapy for septic, endotoxic or exotoxic shock may be necessary in patients with enterocolitis. Antimicrobial therapy may well be necessary, particularly in those individuals that are most susceptible to secondary bacterial infection. Bacteremia / septicemia with resultant septic arthritis is a common secondary complication of enterocolitis in the neonate. Broad-spectrum antimicrobial therapy may prevent the development of secondary infectious problems. If Salmonella is suspected then antimicrobials with appropriate sensitivities should be chosen. Systemic antimicrobials do not treat the enteric infection, with the exception of organisms that may locally invade such as Salmonella or Clostridia. Oral aminoglycosides have been used with some anecdotal success when coliforms are suspected.

Polyionic fluids are the initial fluid of choice. The amount of fluid to be administered is determined by the size of the patient and the degree of dehydration. Hydration status is determined by the clinical examination of the patient. The degree of dehydration can be used to determine an “estimate” of fluid requirement. Mild, moderate, and severe dehydration can be correlated with 5%, 7%, and 10% dehydration respectively. Multiplying the percent dehydration, times the bodyweight in kg, results in an estimate of fluid volume needed for rehydration. Continued fluid therapy is dependent upon response to therapy and continued fluid losses. A useful “rule of thumb” to determine maintenance fluid requirements is; maintenance requirement is approximately 1mL/lb/hr. Therefore, a 100lb foal would require 100ml of fluid per hour. If losses continue, then fluid rates of 2-3x maintenance may be necessary. In situations of severe shock with cardiovascular collapse, hypertonic saline or colloids should be considered.

Electrolyte status should be determined initially and then throughout the course of treatment, depending upon response to therapy. Individuals with diarrhea may develop hyponatremia, hypochloremia, hypokalemia, or acidosis. The most frequent abnormalities are hypokalemia and acidosis. Acidosis is often corrected by fluid replacement when persistent or severe bicarbonate requirements can be corrected using the formula $.5 \times \text{BW (kg)} \times \text{base deficit}$. Initially, one-half the deficit can be replaced over 1 hour, with repeat assessment of requirements and replacement of the remainder of the deficit over the next 4-6 hours. Potassium supplementation can be intravenous or oral. Intravenous supplementation should not exceed .02 meq /kg (BW)/hr.

Suggested reading:

Merritt A.M. Pathophysiology of Diarrhea. Colahan PT, Mayhew IG, Merritt AM, Moore JN (eds). *Equine Medicine and Surgery*. American Veterinary Publications, 1991, pp602-635.

Cohen ND. Diarrheal Diseases of Foals. McKay RJ (ed), *Current Therapy in Equine Medicine 4*, 1997, pp 631-636.

Divers TJ. Acute Diarrhea. Mair T, Ducharme N & Divers T (eds), *Manual of Equine Gastroenterology*, WB Saunders, 2002 Manual of Equine Gastroenterology, pp405-425