Diagnosis and Treatment of Infectious Enteritis in Neonatal and Juvenile Ruminants

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KEYWORDS

• Ruminant • Enteritis • Infection • Juvenile • Neonate • Diarrhea

KEY POINTS

- Common causes of infectious enteritis in neonate and juvenile ruminants include viral, bacterial, and protozoal pathogens.
- The most common presenting sign in ruminants with infectious enteritis is diarrhea.
- Diagnosis of the cause of enteritis has important zoonotic and herd health implications.
- Severity of clinical signs with similar pathogens may differ between calves and small ruminants.
- Treatment of enteritis involves supportive care to correct fluid and electrolyte imbalances, provision of nutritional support for the neonate, prevention and treatment of endotoxemia or sepsis, and pathogen-specific treatments when relevant and available.

PATHOPHYSIOLOGY

Several mechanisms of diarrhea are possible in ruminant neonates. This article summarizes the various mechanisms:

- Malabsorption
  - It is important to remember that, under physiologic conditions, more fluid is secreted into the intestinal lumen, and reabsorbed, compared with the ingested amount. Therefore, impaired reabsorption of fluids has a major impact on the fluid balance of the patient. Several diarrheal pathogens interfere with digestion and absorption by blunting intestinal villi, as observed with rotavirus and coronavirus infections.
- Osmotic
  - Increased solutes within the intestinal lumen osmotically pull more water into the lumen, thereby resulting in dehydration of the patient. Osmotic particles

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include maldigested disaccharides, and increased D-lactate levels from bacteria fermentation of unabsorbed nutrients that enter the colon.

- Secretory
  - Specific pathogens, such as enterotoxigenic *Escherichia coli* (ETEC), stimulate cyclic AMP, thus increasing secretion of chloride (Cl), sodium (Na), and potassium (K) into the intestinal lumen, thereby drawing water into the intestinal lumen.
  - In addition, some pathogens denude the intestinal surface and cause villous blunting, resulting in maldigestion and malabsorption. This damage to the villous leads to proliferation of secretory crypt cells and increased secretory capacity of the intestinal wall.
- Abnormal intestinal motility
  - Decreasing intestinal transit time may lead to maldigestion and malabsorption because of inadequate time for digestion and absorption of the ingested feed material. This process further contributes to osmotic retention of fluid in the intestinal tract.
- Increased hydrostatic pressure
  - Disease conditions, including heart failure, renal disease, and liver disease, may result in increased hydrostatic pressure within the intestinal tract causing movement of water from extracellular tissues into the intestinal lumen, resulting in diarrhea.
- Gastrointestinal (GI) inflammation
  - Inflammation of the GI tract or the peritoneum (peritonitis) can exacerbate all of the above mechanisms of diarrhea. Increasing intestinal permeability or increasing hydrostatic pressure within the intestinal wall can increase fluid loss into the lumen. In addition, prostaglandin production stimulates fluid secretion into the lumen. Infiltration of the intestinal wall by inflammatory cells can also disrupt intestinal motility, increase intestinal secretion, and decrease absorptive function.

Diarrhea often results in fluid and electrolyte losses for the patient. As long as the ruminant neonate can compensate for losses, it will remain hemodynamically stable, and continue to nurse. However, if losses exceed intake, systemic effects will be observed on clinical examination. Fluid loss from the vascular compartment leads to hypovolemia (dehydration), hypotension, and shock. Metabolic acidosis develops as a result of intestinal and fecal loss of sodium bicarbonate, increased L-lactate from hypoperfused tissues, and increased absorption of L-lactate and D-lactate produced by bacterial fermentation in the intestinal tract.[1] As dehydration and acidosis worsen, clinical signs progress, leading to weakness, loss of suckle reflex, and recumbency. Vascular collapse and electrolyte imbalances can lead to heart failure, whereas death can also result from malnutrition and hypoglycemia in neonates. In addition, endotoxemia from gram-negative bacterial infection, such as *Salmonella* or *E coli*, can directly cause circulatory failure.

PATIENT HISTORY

Patient history should include information regarding the age and use of the animal (eg, dairy, beef, show animal), history of colostrum ingestion, duration and progression of diarrhea, age, and number of animals affected or dead in the herd. Assessment of housing, management, feeding, sanitation practices, and preventive health measures is also important. On-farm standard operating procedures regarding treatment protocols are important to obtain and review, especially when approaching outbreaks of