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Diseases of Neonatal Calves: An Update

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The major enteric diseases of neonatal calves are diarrhea, ruminal drinking, abomasal ulceration, and the acidosis without dehydration syndrome. New developments have improved our ability to understand, diagnose, treat, and prevent these conditions. In addition, nutritional muscular dystrophy has recently been described in newborn calves. This issue of *Large Animal Veterinary Rounds* reviews these new developments.

Diarrhea

Diarrhea in calves can be caused by enteropathogenic *Escherichia coli*, which is most common in 1- to 4-day-old calves. In older calves, rotaviruses, coronaviruses, *Salmonella sp.*, *Cryptosporidium sp.*, and *Eimeria sp.* are important pathogens. Recent work indicates that *Giardia sp.* can frequently be isolated from calf feces by sucrose flotation and this protozoan may be pathogenic.¹⁻³

Recent developments in the understanding of neonatal calf diarrhea have highlighted the importance of intestinal bacterial overgrowth and D-lactic acidosis in the pathogenesis of neonatal scours. These two factors are probably interrelated, since D-lactic acid is produced primarily in the gastrointestinal tract by bacterial fermentation. A recent review documented evidence for bacterial overgrowth in the distal small intestine and ileum of diarrheic calves. Small intestinal bacterial counts can increase 5- to 10,000-fold when the primary intestinal pathogen is viral.⁴ The immediate causes for this increased bacterial count are almost certainly diminished digestion, malabsorption of food, and an increase in intestinal nutrient availability. Bacterial overgrowth is a source for bacteremia, a finding in 30% of calves with diarrhea and systemic signs.^{5,6}

Intestinal bacterial overgrowth may also allow the fermentation of nutrients into compounds that are difficult for the calf to metabolize eg, lactose may be converted to D-lactic acid. Another source of D-lactic acid in diarrheic calves is the rumen, presumably as the result of esophageal dysfunction with ruminal drinking and fermentation.⁷ Recent studies document that D-lactic acid is a major component of acidemia in diarrheic calves, accounting for 55% of the increase in anion gap. In contrast, L-lactic acid explained only about 5% of the variation.⁸ D-lactic acidosis is significant because most of the systemic signs associated with neonatal calf diarrhea, including depression of the central nervous system (CNS), weakness, ataxia, and hypothermia can be produced by experimental D-lactic acid infusion. Other acids are much less effective at producing these signs (Naylor et al, unpublished).

A new method of potentially controlling bacterial overgrowth and limiting D-lactic acidosis is to seed the gut with beneficial organisms. In humans, probiotics such as *Lactobacillus rhamnosus* strain GG (LGG) have successfully treated infectious diarrhea, antibiotic-associated diarrhea, traveler's diarrhea, and *Clostridium difficile* diarrhea in randomized placebo-controlled trials.⁹ Studies in calves indicate that LGG can temporarily colonize the gastrointestinal tract.¹⁰ A randomized controlled trial in diarrheic calves with severe systemic signs indicated that administration of LGG (10 capsules, BID) at the start of fluid therapy reduced the water content of the diarrhea, but did not affect D-lactic acidosis.

Antibiotic use in the treatment of neonatal diarrhea has a long history. Studies have demonstrated that some orally administered antibiotics (eg, potassium and procaine penicillin, neomycin sulfate, ampicillin trihydrate, tetracycline hydrochloride, and chloramphenicol) can increase the incidence of diarrhea, produce malabsorption, and reduce growth.⁴ Use of antibiotics with high oral bioavailability, parenteral administration, or lower dosages may result in less frequent adverse effects. A recent review concluded that there was evidence for using amoxicillin trihydrate (10 mg/kg PO q 12h), amoxicillin trihydrate-clavulanate



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potassium (12.5 mg combined drug/kg PO q12h), amoxicillin or ampicillin (10 mg/kg IM q 12h), or potentiated sulfonamides (25 mg/kg IV or IM q 24h) to control bacterial overgrowth in diarrheic calves with signs of systemic illness (reduced appetite for milk and fever). There is also evidence that ceftiofur sodium or hydrochloride is effective in reducing mortality and the severity of diarrhea from enteric colibacillosis in other species. At a dosage of 2.2 mg/kg IM q12h in 7-day old calves, free plasma β -lactam antimicrobial concentrations are maintained at the desired level of at least 4 times the MIC₉₀ (minimum inhibitory concentration) for *E. coli*. In addition, 30% of the antibiotic active metabolite is excreted into the gastrointestinal tract.⁴ Parenteral administration of potentiated sulfonamides has demonstrated effectiveness in treating acute salmonellosis in calves when treatment was administered at the earliest signs of malaise, but before signs of diarrhea were seen.¹¹ Many of the preceding recommendations constitute extra-label usage. Judicious use of antibiotics is best restricted to calves with diarrhea and systemic illness.

Prevention

Scours is the major cause of preweaning mortality in calves that survive birth. For beef herds, the median mortality from live birth to weaning is < 1%.¹² Mean mortality for dairy heifer calves can be nearly 10%, although the goal is for < 5% mortality from diarrhea.¹³ The infectious agents are similar in the two groups, but the factors that predispose to scours and mortality are different. The principles of prevention are:

- Ensure adequate colostrum intake¹⁴
- Boost specific and non-specific immunity
- Reduce the possibility of introduction/spread of infectious agents

Boosting immunity

In general, vaccination against enteropathogens, particularly viral pathogens, is not a reliable strategy for reducing the risk of calf scours. Several studies, including one recent large-scale beef survey, have revealed that farms administering scours vaccinations have a higher incidence of diarrhea.¹² Presumably, this is because poor management favours the survival and propagation of a wide variety of enteropathogens that either swamp or circumvent vaccination.

Vaccination against enterotoxigenic *E. coli* is very effective in preventing this single cause of diarrhea when the vaccine is based on the F5/K99 antigen. These fimbrial antigens enable enterotoxigenic *E. coli* to adhere to the mucosa. In unvaccinated cows, colostrum antibody titers against F5/K99 antigens are frequently low and enterotoxigenic diarrhea often strikes in the first few days of life. Experimental studies confirm that colostrum antibody titers increase when the dam is vaccinated with bacterins containing F5/K99-positive *E. coli*. Vaccination has also been effective in field trials. Pregnant cows should be vaccinated 6 and 3 weeks prior to calving, but *E. coli* vaccination can still be effective if a priming dose is given 18 months before calving and boosted in the second half of gestation. Clinical experience in farms with unvaccinated beef and with severe outbreaks of *E. coli* diarrhea also indicates that vaccinating cows >10 days from

parturition can protect against death from enterotoxigenic *E. coli* infection.

Passive protection with oral products containing preformed antibodies against F5/K99 are often effective in reducing the severity of diarrhea in experimental challenge models, but can be less successful in the field. In experimental models, calves are typically dosed with antibodies and then challenged with enterotoxigenic *E. coli* a few hours later. A newer product containing a mixture of *E. coli* and coronavirus antibodies has also been effective in reducing the severity of experimental coronavirus-induced diarrhea.¹⁵ However, in field situations some products have a lower efficacy. This is presumably because a single dose only provides a short period of enteric protection and in the field, challenge is continuous. Antibody supplements are expensive and vaccination of the dam to boost colostrum immunity for enterotoxigenic *E. coli* will usually be more cost-effective. On farms experiencing an outbreak of neonatal diarrhea caused by enterotoxigenic *E. coli*, antibody-containing products may be useful until vaccinated cows begin to calve. An alternative, highly effective, short-term approach to control is to give an antibiotic effective against *E. coli* for the first 3 days of life. In hand-rearing situations, a possible method of passive protection is to store immune colostrum and feed small amounts (200 ml) daily throughout the first few weeks of the calf's life.

Vaccination against rota and coronavirus diarrhea continues to have a checkered history. Early research indicated that vaccinating calves orally with a rotavirus vaccine would protect against rotavirus diarrhea. However, in many calves in the field, the vaccine is inactivated by antirotaviral antibodies from colostrum. Field studies with beef herds failed to show any benefit from this vaccine. Vaccines can also be given to the dam to boost colostrum immunity to higher levels. Studies with some early vaccines demonstrated no significant seroconversion in the cows, no boost in colostrum antirotavirus antibody titers, and no protection against viral scours. However, field trials of vaccines developed in Europe have demonstrated seroconversion, increased colostrum antibody titers, and reduced incidence of rotavirus diarrhea. Some North American vaccines boost colostrum antirotavirus antibody titers, but these decline rapidly and it is uncertain if they provide protection. Further, the strain differences in viral vaccines are sometimes sufficient to prevent vaccination with one strain from protecting against challenge with other strains.

Some producers routinely administer vitamin A to neonatal calves. Many, but not all, studies in children indicate that supplementation can reduce the incidence of diarrhea in areas where clinical and subclinical vitamin A deficiency is endemic. In cattle, vitamin A deficiency is most likely with a diet of unsupplemented straw and grain. Since epidemiologic studies indicate that administering vitamins or minerals by injection is associated with a higher risk of scours, providing a balanced food ration is probably the best mode of supplementation.¹² Calves should not require supplementation if born to cows fed good quality, green forage or receiving a vitamin A supplement, particularly if the calves receive adequate colostrum high in vitamin A. Vitamin C supplementation is reported to offer benefits at an initial oral dose of 1 g per day TID for the first week of life.¹⁶⁻¹⁸

Reducing microbial challenge

Most enteropathogens survive for long periods of time in the environment. *Salmonella* and *E. coli* can survive in a damp environment for months. In one instance, *Salmonella* persisted in a calf-rearing unit despite the fact that the unit was depopulated, cleaned, disinfected, and kept free of calves for 6 months. Rotaviruses survive for about 2 weeks in fresh water and survival is enhanced by cool temperatures, as well as by the presence of organic material to stabilize viral particles. Under favorable conditions, rotaviruses can persist for months. *Cryptosporidium* is also very resistant and can survive for 2–6 months at -4°C to 4°C ,¹⁹ but it dies off more rapidly in airy or very cold locations.

Dairy calves

Raising calves in groups of ≥ 7 increases the risk of mortality.¹³ In recent years, the use of calf hutches has gained widespread acceptance and provides individual isolated housing for each calf. Cleaning is facilitated because the hutches can be moved to new sites between calves. Calves should be born in newly cleaned calving boxes and the udder and perineum of the cow should be cleansed.

Cleanliness is also essential to successful hand rearing of calves. All equipment should be clean and cleanable (a recent study showed that cleaning with soap gave a lower scours risk than disinfecting). Milk replacer should be prepared with very high standards of hygiene. Nipple buckets and other feeding utensils should be cleaned thoroughly between each feed. Old cracked pails are very difficult to clean effectively and can result in a milk replacer solution containing a very high bacterial load. A paper presented at the World Buiatrics Congress in Quebec recommended that milk or milk replacer fed to a calf have a total bacterial count of $<10,000$ cfu/mL (colony forming units) with no fecal coliforms.²⁰

Cleaning followed by disinfection after each batch of calves is important in reducing contamination in housed calves; all movable equipment should be removed and scrubbed down. After the electricity has been turned off, the building can be soaked to trap dust and then thoroughly cleaned; this removes about 90% of bacteria. High-pressure hosing is very effective and can remove 99.98% of contamination even when no disinfectants are used. Since aerosolization can potentially occur, it is good practice to wear a face mask and to remove all animals prior to cleaning. The most readily cleaned surfaces are made of smooth impervious materials such as plastic and varnished wood. Many disinfectants are inactivated by organic matter. Viruses, coccidia, and particularly, cryptosporidia may be resistant. Disinfectants may be toxic and are best applied with the use of rubber gloves and respirators (if indoors). In general, potent phenolics such as cresol (cresylic acid) are very useful for disinfecting dirty surfaces (eg, boots) because they are not inactivated by organic matter and are effective against Gram-negative bacteria and viruses. The phenolics are highly toxic and leave lingering odours. Hypochlorite solutions (5 g available chlorine/L) have a broad spectrum of action, but are rapidly inactivated by organic matter. However, they are inexpensive and useful as a final disinfectant on previously cleaned

surfaces. Because hypochlorite is unstable, it is unlikely to leave toxic residues. For previously cleaned surfaces, another effective agent against many bacterial and viral enteric pathogens of calves is potassium monopersulfate. It has the advantage that accidental spills will not bleach clothing. Iodophors are not very effective against rotaviruses, particularly if organic matter is present. Formaldehyde is one of the few effective agents against cryptosporidia, but it requires a long contact time and is highly toxic. Formaldehyde is usually used for terminal fumigation in buildings that can be tightly sealed. Following disinfection, the building should be ventilated and allowed to rest before re-introducing calves.

Adequate ventilation may also be important because some enteric pathogens infect the respiratory tract and there is a related incidence between respiratory and enteric disease in calf units. Attention should also be given to rodent control, since these can be reservoirs for *Salmonella*.

Beef herds

Diarrhea is more likely to be a problem in large herds. Viral agents can be excreted in low numbers by adult cows and can introduce infection into the calf population. Bacterial agents such as *Salmonella* and *E. coli*, and protozoa are also often brought into, or maintained, in a herd by asymptomatic carrier cows. The organisms multiply in calves and the infection is magnified until it reaches epidemic proportions in neonates.

The major risk factors for diarrhea include a high percentage of heifers in the herd, intensive stocking, poor drainage, and dirty or wet calving and rearing areas. Beef cattle are healthier if overwintered in separate groups for cows and heifers. This provides an opportunity to improve the feeding for heifers and to slow disease transmission to the rest of the herd from susceptible calves born to heifers. Large herds are best split into groups of 50 to 75. Prior to calving, the cows or heifers should be moved to a clean, well-drained, calving area with a minimum of 100 m^2 (1000 square feet) per cow (ideally, 200 m^2). Ample clean bedding and extensive wind breaks should be provided. During this period, the cow can be carefully observed for dystocia. Once the calf is born, it should be checked frequently to make sure it is up, sucking, and has received colostrum. When cow and calf show successful mothering, they can be moved to a separate nursing area and that area should be clean, well-drained, and spacious. If $> 5\%$ of the area is poorly drained (prone to standing water) or if cows have mud above their hocks (and thus likely on their udders) the risk of diarrhea is increased. Early cases of diarrhea should be isolated from the main group to reduce contamination. Purchasing replacements for calves lost at birth should be discouraged; it is easy to bring in diseased calves.

Diet

Usually, it is suggested that calves do best when fed whole cows' milk and that milk replacers using milk-derived protein are better than those using plant-based protein. Feeding mastitic milk or milk containing antibiotic residues is associated with increased calf mortality. Although many calves are not fed roughage until later in life, one study showed that introducing roughage after 21 days of life increased the risk of mortality.¹³

Table 1: Clinical syndromes produced by abomasal ulceration in calves^{34,41-44}

Syndrome	Pathology	Clinical Signs
Subclinical	Multiple non-perforating ulcers, usually in pyloric area.	None
Hemorrhagic abomasal ulceration	Perforation of major abomasal blood vessel	Melena, anemia, weakness, shock.
Abomasal ulceration with perforation	Perforating ulcer, usually single, pyloric area or body, towards greater curvature. Ulcer often perforates into the omental bursa. Increased probability of abomasal hairball. Concurrent left displacement of the abomasum (LDA) in some cases.	Peracute form. Often better grown calves with a very rapid course or found dead with no premonitory signs. Acute form. Ill for several days. Depression, arched back, abdominal distension, tense abdominal wall and/or pain on palpation, expiratory grunt, bruxism, colic, standing with mouth in water without swallowing.

Case-based evidence suggests that milk replacers can have problems with hyperosmolarity and excessive sodium content. Cheese makers use sodium salts to neutralize acids in whey and high sodium content milk replacers can result if cheese whey is used to manufacture milk replacers. Another source of salt is the addition of oral electrolyte powders or other sodium-containing medications when milk replacers are mixed. As a result, the final milk-replacer solution can contain high sodium concentrations that may induce salt poisoning. In addition, high sodium content has been anecdotally linked to digestive upsets and a higher incidence of diarrhea. It is recommended that milk-replacer solutions contain a maximum of 120 mmoles/L of sodium on an “as fed” basis and that fresh water is always available.²⁰

Other factors

In one large survey, a factor that made a systematic difference in calf survival was the sex and age of the caregiver.¹³ Women and children perform better than men.

Operations that bring in calves for rearing purposes should be encouraged to buy from as few sources as possible. Direct purchase from one farm is best; assembling collections of calves through auction markets should be avoided. In some cases, calves may be infected with *Salmonella* on the farm of origin from carrier cows. Cultures taken from calves and cows can help detect this problem.

Acidosis without dehydration syndrome

This condition of young calves is characterized by clinical signs of ataxia or, in more severe cases, involuntary recumbency and coma. There are either no, or minimal, clinical signs of dehydration, although clinical chemistry may show a mild uremia. The feces are never profuse or watery, but they may be slightly loose. Early reports documented that the severity of CNS depression, measured by a scoring system based on the strength of the suck, menace and panniculus reflexes, and an assessment of ataxia, was very highly correlated with the degree of metabolic acidosis.²¹⁻²³ Recently, it has been shown that D-lactic acid is the cause of the acidemia,²⁴ a finding consistent with the ability of this acid to produce similar

clinical signs when given intravenously. Acidosis without dehydration responds very well to acidemia correction with intravenous sodium bicarbonate. The amounts of sodium bicarbonate required can be initially assessed, in practice, by using the scoring sheet developed for calves.²⁵ However, supplemental sodium bicarbonate may be needed because base deficits as high as 30 mmol/L are found in severe cases of acidosis without dehydration. The syndrome is not very common, but it is important because it can be confused with meningitis. Meningitis tends to affect calves in the first few days of life, whereas acidosis without dehydration is seen in calves between 7 and 30 days of age. Meningitis carries an extremely poor prognosis, but acidosis without dehydration has an excellent prognosis if diagnosed and treated.

Abomasal ulcers

Abomasal ulcers remain an important problem in young calves, particularly those between 1 and 12 weeks of age. At least 3 different clinical syndromes have been recognized in calves (Table 1). The pathogenesis is not completely understood, but prolonged periods of severe abomasal acidity are thought to be an important predisposing factor. Calves bottle-fed whole cows' milk twice daily, have lower abomasal pH than those fed milk replacer.²⁶ Abomasal pH is also lower when there are long intervals between milk replacer feedings, particularly feeding <3 times-a-day.²⁷ In veal calves, some,^{28,29} but not all studies,³⁰ show an increased incidence in calves fed roughage in addition to milk replacer. Stress does not appear to be a major predisposing cause. Calves with a nervous temperament, as measured by the degree of anxiety a calf shows in response to a standard stimulus, did not have an increased incidence of abomasal ulceration.³¹ Calves stressed by frequent mixing and regrouping had evidence of adrenal hyperplasia, but did not have an increased frequency of abomasal ulceration.³² *Helicobacter pylori* has been implicated in the development of human gastric ulceration, but it does not appear to be involved in calves.³³

The potential predisposing effects of abomasal hairballs, copper deficiency, and *Clostridium perfringens* type A

are controversial. Hairballs are found with an increased frequency in calves < 30 days of age dead from abomasal ulcers as opposed to other causes. However, this may be due to decreased haircoat licking in calves dying from diseases other than perforating ulcers.³⁴ One study found low copper concentrations in the liver of calves dying from abomasal ulceration as opposed to other causes,³⁵ but in another study calves dying of abomasal ulceration were not deficient in copper.³⁶ *C. perfringens* type A was isolated from the abomasum of an almost identical percentage of calves dying with perforating or hemorrhagic abomasal ulcers as those dying of other causes.³³ On the other hand, experimental inoculation of *C. perfringens* type A into the rumen of calves produced anorexia, depression, bloat, diarrhea, and in some calves, death. Necropsy examination revealed variable degrees of abomasitis, petechial and ecchymotic hemorrhages, and ulcers (ranging from pinpoint to nearly perforate) in the abomasum.³⁷

Calves with perforating ulcers often die before treatment can be instigated or die in spite of therapy. In one series, 4 of 10 calves treated by surgically resecting the ulcer survived; mortality usually occurred within 48 hours due to diffuse peritonitis and shock.³⁸ Clinical signs due to non-perforating ulcers are much less common in calves. Calves with severe blood loss due to hemorrhagic ulceration should be treated with intravenous fluids and anticoagulant-treated blood collected from the calf's dam.

A variety of antacids and histamine H₂ antagonists have been shown to increase abomasal pH and may also be helpful. An oral antacid for human use containing a mixture of aluminium and magnesium hydroxide administered at the rate of 50 mL TID for one day increases abomasal pH by approximately 2 units.³⁹ Oral cimetidine, oral ranitidine, and intramuscular ranitidine have all been shown effective in raising abomasal pH. Cimetidine and ranitidine have been administered orally to calves in milk replacer (fed from a nipple pail at 60 mL/kg body weight) at dosages of 50 or 100 mg/kg for cimetidine and 10 or 50 mg/kg for ranitidine, given 3 times-a-day for one day. At these dosages, cimetidine was more effective (Table 2). Injectable ranitidine is expensive, but at a dose of 6.6 mg/kg IM, a single dose raised abomasal pH in young steers and might be useful as a 1 time initial treatment in valuable calves unwilling to suck.

White muscle disease

Nutritional muscular dystrophy has often been recognized as a cause of stiff gait, weakness, tachypnea, reddish discolored urine, and involuntary recumbency in younger calves. Some calves suffer from other concomitant diseases, such as enteritis. In a small percentage of diarrheic calves, co-existing white muscle disease is a reason for poor response to standard therapies for diarrhea.

Case

A case of congenital nutritional muscular dystrophy has been described in a Canadian beef calf; the affected calf was recumbent and unable to move. Additional signs

Table 2: Effect of various antacid treatments on abomasal pH of young calves^{39,45}

Treatment	Abomasal pH, mean +/- 1 SD
Experiment A	
None / control	2.87 +/- 0.58
Oral proprietary magnesium and aluminum hydroxide solution, 50 mL TID	4.0 +/- 0.67
Oral proprietary magnesium and aluminum hydroxide solution, 25 mL TID	3.65 +/- 0.47
Experiment B	
None / control	3.08 +/- 2.01
Cimetidine 100 mg/kg, q 8 h, PO	4.61 +/- 1.52
Ranitidine 50 mg/kg, q 8 h, PO	4.5 +/- 1.52
Cimetidine 50 mg/kg q 8 h, PO	4.29 +/- 1.66
Ranitidine 10 mg/kg q 8 h, PO	3.91 +/- 1.77

found on examination at 13 hours of age included a diminished suck reflex, mild dehydration, hypothermia, and a comatose appearance. Pretreatment creatinine kinase (CK) was about 30,000 U/L (reference range 35–280 U/L). Serum concentrations of both vitamin E and selenium were subnormal. The calf was successfully treated with a mixture of warming, intravenous fluids, tube feeding, and subcutaneous injections of selenium and vitamin E.⁴⁰

Conclusion

Neonatal calves suffer from a varied and interesting collection of diseases. Although disease can be severe and the progression rapid, treatment is often very rewarding. Recent advances should further improve our ability to treat and prevent these conditions.

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