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Treatment and Control of Neonatal Diarrhea in Calves

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Abstract

Treatment and control of acute neonatal diarrhea in calves are outlined and discussed. The difficulty in making a definitive etiological diagnosis makes effective treatment and control also difficult and largely empirical. Physiological events in calves with diarrhea are known, and fluid therapy is directed toward treating dehydration and acidosis. Whether affected calves should receive antibacterial agents orally is an open question. Principles of control of diarrhea in calves are outlined and discussed.

Introduction

Neonatal diarrhea of calves is a major cause of economic loss in rearing young calves (13). Treatment and control of the problem are usually difficult and unrewarding because often the cause of the diarrhea is difficult to determine quickly and accurately (7). Biochemical events in newborn calves affected with acute diarrhea are well known, and considerable progress has been made in the last 10 yr in treating physiological effects of the disease with such as fluid and electrolyte therapy for dehydration and acidosis (4, 22). Methods for control of the disease have been empirical

and are usually based on the assumption that the disease is infectious and that the degree of colostral immunity in calves and certain environmental and nutritional factors each play a role in determining whether it will survive effects of diarrhea or die.

The purpose of this paper is to outline and discuss a rational basis for treatment and control of diarrhea in newborn calves based on our current understanding of the disease.

Etiology and Pathogenesis

Effective treatment and control of any disease are dependent on a clear understanding of the cause(s) of the disease and how the causative agent(s) produce the lesions in the animal. One of the major stumbling blocks in the development of effective methods of treatment and control has been the practical difficulty of making a definitive etiological diagnosis with a single calf or a group of calves affected with diarrhea. Diarrhea is only a clinical sign of alimentary tract dysfunction. One of the major mechanisms by which the intestinal tract of a newborn calf reacts to pathogenic bacteria or viruses or indigestible dietary nutrients is hypersecretion and a relative lack of intestinal absorption which results in a loss of fluids, electrolytes, and nutrients, and the net effect is diarrhea. Some of the common causes of diarrhea in newborn calves include enteropathogenic *E. coli* (7) and *Salmonellae spp.* (15), reo-like viruses (11), *Chlamydia spp.*

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(6), milk replacers containing heat denatured whey protein (18), overfeeding and other faulty feeding practices (4).

Biochemical events in calves affected with acute diarrhea have been studied by several workers, and there is agreement on important changes which occur (4, 5, 22). There are major net losses in sodium and bicarbonate ions and water which ultimately lead to a state of metabolic acidosis and dehydration. This becomes progressively worse as diarrhea continues. The time required for severe changes can vary from a few hours to a few days after onset of the diarrhea. Blood chemical analyses of some newborn calves affected with acute diarrhea are shown in Table 1 (22).

In addition to the loss of electrolytes and fluids, calves affected with diarrhea do not consume normal quantities of milk and digestibility of all nutrients is markedly decreased. As the diarrhea continues there is a net loss of all nutrients and a continuous loss in body weight. Blaxter and Wood (1) called this state of negative nutrient balance "physiological starvation".

Treatment

Treatment of calves affected with acute neonatal diarrhea is based on: (1) cause of diarrhea if it can be determined, (2) clinical state of the calf which includes a clinical estimation or an accurate laboratory determination of biochemical changes as a result of diarrhea, and (3) complications such as pneumonia, arthritis, meningitis, peritonitis, and septicemia.

Fluid and electrolyte replacement therapy. Dehydration, acidosis, and electrolyte imbal-

ance are corrected by solutions containing mixtures of electrolytes, water, and glucose. In severe dehydration and acidosis the bicarbonate, sodium, and chloride ions are vital and life saving. During convalescence, solutions containing glucose and potassium may be used but are not essential if the calf begins to consume milk. The amounts of fluids are given according to the following schedule (4).

1) *For severe dehydration:* (dehydrated 8 to 10% of body weight). *Hydration therapy:* 100 ml per kg body weight intravenously in the first 4 to 6 h. *Maintenance therapy:* 140 ml per kg body weight intravenously over the next 20 h.

2) *For moderate dehydration:* (dehydrated 6% of body weight). *Hydration therapy:* 50 ml per kg body weight intravenously in the first 4 to 6 h. *Maintenance therapy:* 140 ml per kg body weight intravenously over the next 20 h. Intravenous therapy is by indwelling intravenous catheter.

Maintenance therapy may be given orally if the calf is well enough to suck from a nipple bottle or drink from a pail, or oral fluids may be given by gravity flow through a plastic stomach tube. When using the oral route for maintenance fluid therapy, the daily dose (24 h) is given in divided doses every 2 to 4 h during the day.

Composition of intravenous fluids. (1) *Isotonic Sodium Bicarbonate:* Dissolve 13 g NaHCO_3 in 1 liter of water. This gives an isotonic solution containing 156 meq/liter HCO_3^- , and 156 meq/liter Na^+ . This solution is used only when calves are severely acidotic, i.e. blood pH 7.00. These calves are usually comatose, 10 to 12% dehydrated, and have

TABLE 1. Blood chemical analysis of neonatal calves with acute enteric infections (22).

Analysis	Observations (no.)	Mean \pm SD	Range	
			Observed	Normal
Packed cell volume	28	45.3 \pm 7.0	31.0 - 60.0	25.0 - 40.0
Plasma protein (g/100 ml)	28	8.6 \pm 1.5	6.8 - 11.3	6.0 - 8.0
Blood pH	11	7.08 \pm .12	6.88 - 7.28	7.35 - 7.40
PCO ₂ (mm Hg)	11	46.8 \pm 6.4	35.8 - 57.3	38.0 - 45.0
CO ₂ (mm/liter)	11	14.8 \pm 4.2	9.3 - 23.8	24.0 - 30.0
Bicarbonate (meq/liter)	11	13.7 \pm 4.2	8.2 - 22.7	23.0 - 28.0
Sodium (meq/liter)	28	138.1 \pm 9.4	119.5 - 165.0	137.0 - 145.0
Potassium (meq/liter)	28	7.4 \pm 1.6	4.8 - 12.3	4.5 - 5.5
Chloride (meq/liter)	27	101.4 \pm 7.5	89.0 - 116.0	97.0 - 105.0
Calcium (meq/liter)	26	6.3 \pm 1.0	4.3 - 8.1	4.5 - 6.0
Magnesium (meq/liter)	26	3.3 \pm .9	2.1 - 5.6	1.0 - 2.9
Phosphorus (meq/liter)	25	9.2 \pm 3.6	3.9 - 15.0	2.9 - 5.8
Blood Urea Nitrogen (mg/100 ml)	28	50.1 \pm 30.5	11.5 - 161.0	10 - 20

cardiac arrhythmias. In these cases, 2 liters (=312 meq HCO_3^-) can be given rapidly to a 45 kg calf before changing to isotonic saline:sodium bicarbonate as outlined below.

(2) Isotonic Saline:Sodium Bicarbonate: Dissolve 26 g NaHCO_3 in 4,000 ml of .5 normal saline (.425%); or mix 1 liter isotonic sodium bicarbonate (13 g NaHCO_3 /liter) and 1 liter isotonic saline (.85% NaCl = 8.5 g NaCl /liter). This gives an isotonic solution containing 78 meq/liter HCO_3^- , 78 meq/liter Cl^- , 156 meq/liter Na^+ . Total HCO_3^- per 3.78 liter = 312 meq.

The mixture of isotonic $\text{NaCl}:\text{NaHCO}_3$ is most commonly used in scouring calves affected with dehydration and acidosis. When administered properly, it supplies an optimal ratio of fluid and bicarbonate to correct these two clinical states which are most dangerous to life. Most scouring calves that are 6% or more dehydrated can be given 4 liters of this solution without making them alkalotic. Depending on the severity of the case, a further 1 to 2 liters may be given before change to one of the balanced electrolyte solutions. Usually after calves have received 4 liters of this solution, they improve considerably and are able to maintain sternal recumbency.

Composition of oral fluids. Formula No. 1 (D. Hamilton, Unpublished observations) Sodium chloride 113.6 g, Potassium chloride 50.3 g, Sodium bicarbonate 108.9 g, Glucose 535.1 g, Glycine 223.0 g, Total 1,030.9 g. For 1,000 ml of solution add 38.2 g of the above powder mixture to 1 liter of water.

Formula No. 2. (5) Sodium chloride 117.0 g, Potassium chloride 150.0 g, Sodium bicarbonate 168.0 g, Potassium phosphate (K_2HPO_4) 135.0 g, Total 570.0 g. For 1,000 ml of solution add 5.7 g of powder to which may also be added 50 g of glucose. With glucose added, this solution can be given subcutaneously.

Calves which respond and recover permanently usually show marked improvement from intravenous and/or oral fluid therapy within 24 to 48 h. Calves which are likely to recover will respond to the hydration therapy in about 12 h; they will begin to urinate (water diuresis), dry matter content of their feces will increase from below 10% to over 16 to 18%, they will maintain hydration and will be well enough to have an appetite for milk.

Calves which do not respond will not hydrate normally, they may not begin to urinate because of irreversible renal failure, their feces remain watery, they remain depressed and not

well enough to suck or drink, and continued intravenous and/or fluid therapy beyond 3 days is usually futile. They continue in a diarrheic state and become emaciated severely. Often the fluids which are administered will collect (sequester) in the lumen of the small intestines which suggests a permanent functional malabsorption syndrome.

Antimicrobial therapy. Calves affected with acute diarrhea are treated routinely with antimicrobial agents by oral and parenteral routes. It has been assumed that enteropathogenic bacteria, especially certain serotypes of *E. coli* and *Salmonellae spp.* are either the initial cause of the diarrhea or at least contribute to the disease initiated by other poorly defined factors.

Broad spectrum antibiotics such as oxytetracycline, neomycin, and chloramphenicol, and chemotherapeutics such as the nitrofurans are used widely for treatment of calf diarrhea. Ideally the drug of choice will depend on the drug sensitivity of the causative bacteria isolated from the feces of the calf. However, it usually is not possible to determine which bacteria isolated from the feces of a diarrheic calf are the real cause of the disease. This is particularly true in single isolated causes of diarrhea and only when the same enteropathogenic serotypes are isolated consistently in problem herds does their presence assume some significance. Until more information becomes available veterinarians will continue to treat diarrheic calves with broad spectrum antibiotics and/or chemotherapeutics by the oral route on the assumption that diarrhea is infectious and because empirically they appear to be beneficial. How the antimicrobial agent affects the spectrum of intestinal microflora in diarrheic calves is not known. Because of the dynamic nature of intestinal microflora, it is a distinct possibility that the particular spectrum of intestinal microflora at the time of treatment may be quite different from that which may have been responsible for the disease only a few hours earlier.

Alteration of diet. Calves with acute neonatal diarrhea should be starved from milk for at least 24 h or until the dry matter content of the feces increases above 12 to 14%. There is little or no scientific evidence to support this practice, but on an empirical basis most herdsmen and veterinarians feel that a period of complete withholding of all milk from scouring calves hastens recovery. On a theoretical basis starvation would; (1) allow time for the gas-

trointestinal tract to return to normal function, (2) reduce the amount of substrate in the lumen of the gut to minimize fermentation, putrefaction, and the end-products of these processes which may be toxic, and (3) remove the dietary factor as a cause of the diarrhea in calves which continue to have diarrhea in spite of all other therapy.

After starvation and if the calf is showing improvement, cow's whole milk is re-introduced to the calf in small amounts frequently. During this convalescent period, milk is given at 6 to 8% of body weight divided into four feedings during the day. This amount is increased to a daily intake of 10% of body weight over 3 days. If diarrhea develops, then starvation and refeeding regime is repeated. During starvation oral electrolytes and fluids are offered or given as described under fluid therapy.

Intestinal tract protectants. Intestinal tract protectants and demulcents such as kaolin and pectin have been recommended as an aid in the treatment of neonatal diarrhea in calves. The rationale has been that an enteritis is present and that these protectants will provide a temporary protective layer over the intestinal mucosa and allow healing. Whether or not this actually occurs is not known. In fact, most calves with diarrhea do not have gross or even microscopic evidence of classical enteritis, and the large quantities of fluid secreted by the gut probably dilute any protective effect which the protectants may provide.

Before diarrhea stops there must be a return to normal of the secretion-absorption function (mechanism) of the intestinal mucosa, and, thus, the original cause of the failure of this homeostasis must be corrected.

Control of Neonatal Diarrhea in Calves

Because of the complex nature of the disease, it is difficult if not impossible to expect or achieve total prevention of neonatal diarrhea in calves. Control of the disease at an economical minimum amount should be the aim of the herdsman. In a well-managed dairy herd mortality in calves under 1 mo of age due to acute undifferentiated diarrhea should not exceed 3% of calves born. Calf mortality due to acute diarrhea ranges from 6 to 25%; the average herd will experience losses up to 6% while other herds experience losses of 25% and even higher.

Reasons for this high mortality are commonly difficult to identify (many herdsmen and owners are reluctant to accept some of the sug-

gested reasons) but include: inadequate colostrum intake, high stocking rate in the calf barn, concentrated calving frequency resulting in a high percentage of susceptible calves in a highly contaminated calf barn, a newly-introduced entero-pathogenic serotype of *E. coli* or *Salmonellae* spp. to which the cows and, hence, the calves have no immunity, the use of inferior milk replacers (18), and inadequate calf management.

Effective control of neonatal diarrhea of calves will be accomplished by applying the four principles in control and prevention of diseases of the newborn (2). These are: (1) Removal of the calf from the contaminated environment, (2) Removal of the infectious agents from the environment of the calf, (3) Increasing the nonspecific resistance of the calf, and (4) Increasing the specific resistance of the calf. The optimum level for these principles will vary from herd to herd; few herds will be able to do all these things at the desirable rate. However, successful calf management will depend on optimal combination of these principles.

1) *Removal of the calf from a contaminated environment.* Dairy calves born indoors should be moved immediately at birth to an individual calf pen which was previously cleaned, disinfected and left vacant for a few days. Newborn calves should not be placed in pens with groups of older calves until they are at least 2 wk of age.

It is now common practice in some herds to transfer the calf immediately after birth to a separate completely isolated housing unit away from all other older cattle in order to minimize the exposure rate of enteropathogens for the calf.

2) *Removal of infectious agents from the environment.* The incidence and occurrence of acute diarrhea in young calves under 3 wk of age is directly proportional to contamination with enteropathogenic bacteria and perhaps viruses. Contamination will be directly proportional to the concentration of calves raised in close confinement and the occupation time of the calf barn (16, 17). Occupation time is the length of time the calf barn has been occupied by calves since the last clean-out, disinfection, and vacancy. As occupation time increases, incidence of diarrhea increases and the build-up of infection increases, and weight gains are significantly reduced. Still further, all of these effects are magnified if inferior milk replacers are used (13, 18). Contamination in a calf

barn can be reduced and maintained at a low level by periodic depopulation, cleaning out, disinfection, and providing a period of a few days vacancy. This kind of regularly scheduled health work for the calf herd has not received widespread acceptance but will in the future considering the ever increasing number of dairy herds which are raising an increasing number of calves in facilities which are not increasing necessarily in size.

3) *Increasing nonspecific resistance.* The maintenance of an optimum nonspecific resistance depends primarily on day to day care and management of the herd and more particularly on calves during their first 3 wk of life. Such factors as nutrition of the dam during pregnancy, temperature and humidity of calf pens, feeding schedule, quality of milk replacers, other diseases such as enzootic viral pneumonia, season of the year (greater incidence of disease during cold winter months), and amount of colostrum ingested and how soon after birth, all influence the nonspecific resistance of the calf especially under 3 wk of age when it is highly susceptible to disease of the digestive tract.

Much has been written about the care and management of young calves, and excellent references are presented elsewhere in this symposium. Most cattle herdsmen are aware of what should be done to minimize calf losses from neonatal diarrhea, but it is only recently that calf survival and calf rearing have become an integral part of comprehensive herd management. Much emphasis has been placed on reproductive performance, nutrition of the dairy cow, and control of bovine mastitis, but only little effort has been given to reducing calf losses. An effective herd health program will include constant surveillance of diseases of the calf and calf mortality on a regular basis (every month at least). In this way disease problems can be identified and measures instituted to resolve them before losses exceed economic limitations.

4) *Increasing specific resistance.* Newborn calves are deficient in immunoglobulin and must ingest colostrum and absorb lactoglobulins, both in sufficient quantities, to obtain passive protection against common neonatal infections such as coliform septicemia and to prevent mortality due to acute neonatal diarrhea (19). High immunoglobulins in the newborn calf protect it against coliform septicemia and, conversely, calves which have little (hypogammaglobulinemia) or none

(agammaglobulinemia) are susceptible to coliform septicemia which is highly fatal. In addition, the death rate from acute diarrhea in calves is significantly higher in calves with low amounts than in calves with adequate amounts (9). The nature of the protection provided by colostral immunoglobulins is not yet well understood but evidence suggests a nonspecific factor (gamma globulin moiety) and a specific factor (specific antibody) are important components of the resistance provided (8).

With available information it is of paramount importance that serum immunoglobulins be maximized in all newborn calves.

Immunoglobulin in newborn calves which have ingested colostrum is extremely variable, which can be explained by: (1) the age at which colostrum is first ingested, (2) the total amount of globulin ultimately absorbed, and (3) the effects of maternal behavior on the calf (19).

Newborn calves should ingest their first feeding of colostrum at 6% of body weight within 6 h of birth in the case of suckling calves left with the cow and within 1 h of birth for calves which are removed from the cow and hand-fed with a nipple-bucket. Absorption of lactoglobulins from colostrum in the calf is maximal only during the first 12 h after life; after this, the ability to absorb macromolecules is lost rapidly. In fact, there is a progressive reduction in the ability to absorb intact macromolecules which is initiated at birth and which proceeds rapidly thereafter (19).

The total amount of colostral immunoglobulins absorbed by the calf is a function of amount of colostrum ingested, concentration of immunoglobulin in the colostrum, efficiency of absorption of macromolecules, and time after birth that colostrum is ingested. There are wide variations in the lactoglobulin concentrations in colostrum; Selman (19) found a range of five-fold difference in the immune lactoglobulin concentrations in 20 different samples of colostrum. This in part accounts for the significant (linear) relationship between concentration of immune lactoglobulin in colostral whey and 48 h serum concentration of absorbed immune lactoglobulin. Kruse (10) also found that total amount of colostrum and concentration of immune lactoglobulin both increased with advancing age of the dam, thus providing a greater total mass of immune globulin for the calf born to a multiparous rather than the primiparous heifer. This raises the question, "Is the incidence of diarrhea

greater in calves born to heifers and young cows than in calves born to mature cows?" Such information is not available.

With respect to the effects of maternal behavior on absorption of globulins by newborn calves "it has been shown both with dairy calves suckling colostrum from their dams and with similar calves obtaining colostrum from teat buckets that the presence of the dam somehow renders a calf more efficient at absorbing immune lactoglobulin" (19). This suggests that all newborn calves should be left with their dams for the first 6 to 12 h after birth. However, merely leaving the calf with the dam does not ensure that the calf will obtain sufficient colostrum voluntarily. To ensure that a calf receives sufficient quantities of colostrum early enough, it should be fed dam's colostrum by nipple bottle or pail within 1 h after birth at 6% of body weight. It could still be left with the dam for 12 to 24 h to obtain the benefits of maternal behavior. The efficiency of the calf attendant in bringing about maximal immune lactoglobulin absorption can be monitored by the zinc-sulfate turbidimetric test on a weekly basis and can constitute part of an "early warning" system that the calves are not absorbing sufficient quantities of globulins for the reasons already discussed above (20).

The Future

Because of relatively high costs of newborn calves and serious economic losses due to acute neonatal diarrhea, emphasis must continue on generating vital information necessary for optimal control of the disease. While progress has been considerable in recent years in developing effective methods of treatment, the average survival rate is not economical in most instances. Some areas of acute neonatal diarrhea of calves in which some research is necessary include: epidemiology of the disease as it occurs in a problem herd, relationship between the presence of pathogens in the feces of a diarrheic calf, the cause of the disease, nature of the protection provided by immune lactoglobulins, and the possibility of immunization of the dam as a means of protecting the newborn against colibacillosis (8).

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