On-farm calf assessment and practical fluid therapy

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Abstract

Large animal practitioners are faced with varying levels of dehydration in neonatal cattle. The goal of this talk will be to describe and formulate fluid therapy plans using intravenous catheter placement in varying circumstances in an ambulatory setting. Determining route of fluid administration and intravenous catherization techniques will be reviewed. Following this presentation, the practitioner will be able to identify the proper use intravenous catherization and fluid therapy plans in clinical cases to be administered on farm.

Key words: calf, neonatal scours, fluid therapy, catherization

Introduction

Ill neonatal calves can be challenging yet rewarding patients to effectively treat in ambulatory practice. Large animal practitioners are presented with varying levels of dehydration in neonatal cattle. The clinician is required to think on their feet, with a standing knowledge of pathophysiology to formulate the treatment plan needed. The purpose of this lecture is to review the clinical information needed to formulate fluid therapy plans for neonatal calves in an ambulatory setting. Determining route of fluid administration and intravenous catherization techniques will be reviewed.

Fluid therapy generalities

Many of these calves are acidotic as well as dehydrated. In addition, diarrhea does not always have to be present in order for metabolic acidosis to develop. There are 3 mechanisms behind the development of acidosis in calves:

1. Severe dehydration (> 8%) causes hypovolemia and poor tissue perfusion. Anerobic metabolism produces overwhelming concentrations of L-lactic acid, leading to metabolic acidosis.
2. Bacterial fermentation in the gut (regardless of the etiologic agent) produces an over production of D-lactic acid. D-lactic acid is not easily metabolized by the liver and accumulates to very high concentrations, leading to metabolic acidosis.
3. Loss of HCO3- rich fluid in diarrhea results in a low systemic HCO3- concentration, leading to metabolic acidosis.[1]

Regardless of which mechanism is occurring, metabolic acidosis results in decreased mentation and central nervous depression in neonates. Acidosis can be directly assessed with biochemical blood analysis or indirectly assessed through a change in behavior.[4]

Determining hydration status

Clinical signs of dehydration are generally not detectable until the animal is at least 3-5% dehydrated. Prolonged capillary refill time over 3 seconds, is an indication of poor peripheral perfusion. A skin tent test can be used to qualify dehydration by pinching the skin along the lateral neck or thorax and twist 90 degrees, holding for 1 second and then release. The time (seconds) it takes for the skin to return to normal is used with the following equation: percent dehydration ≈ (2 × seconds)-4. However, the usefulness of this technique varies by species, age and gender due to differences in skin thickness and consistency, working best in animals with thin, elastic skin such as horses, dogs and cats. Eyeball recession can be a more quantitative method to assess dehydration in livestock. The eyeball recedes into the bony orbit when the retrobulbar tissues become dehydrated resulting in enophthalmos. At the level of the medial canthas, estimate the eyeball recession in millimeters and multiply by 2 to get the estimated dehydration (i.e. percent dehydration = eyeball recession (mm) × 2).

Practical fluid therapy

The severity of dehydration in the patient will determine route of fluid administration for initial therapy. Dehydration over 8 percent is the upper limit for choosing oral vs. IV fluid therapy. Thus, less clinically dehydrated patients, including calves that are maintaining a suckle reflex, are good candidates of oral fluid resuscitation. Oral electrolytes are the backbone of fluid therapy in diarrheic calves. Not all electrolyte solutions are created equal. There are 4 key ingredients for good oral electrolyte solutions: 1) enough Na to replace the losses in the extracellular fluid space, 2) co-transport molecules (glucose, acetate, glycine), 3) an alkalinizing agent (acetate, proprionate, or bicarbonate), 4) energy source (glucose or acetate).[3] Oral electrolytes should be fed as an extra meal to calves with diarrhea. If the calves are fed milk twice a day, then oral fluids can be fed in the middle of the day. No electrolyte solution can maintain blood glucose and a positive energy balance as well as milk. There is little evidence to substantiate that milk feeding exacerbates diarrhea or slows intestinal healing, but lots of evidence to show calves gain more weight and avoid severe negative energy balance when milk is fed.[5]

For more severely dehydrated calves, including patients that are recumbent, absent suckle, and decreased mentation, intravenous fluid therapy is indicated.[1] There are several IV electrolyte solutions commercially available for the treatment of calves. All these products are effective, but the best ones contain 3 key ingredients: **1)** an alkalinizing agent (HCO3, acetate or lactate), **2)** an energy source (glucose, gluconate or acetate) and **3)** a pinch of potassium. A great all-around solution can be made by combining 1.3% bicarb solution (more bicarb can be added if base deficit warrants), 1 mL 20 mEq/mL KCl, 100 ml 50% dextrose (5% final solution) per 1 liter of fluid.

Sick, neonatal calves are commonly suffering from some degree of metabolic acidosis. Some clinicians recommend giving only half of the total deficit rapidly or over the first 2-6 hours of fluids and then reassess the patient.[1] There are no standard rules on when to use sodium bicarbonate for the correction of

acidosis. The clinician should always consider the blood pH, base deficit and electrolyte concentration (i.e. strong ion difference).[2] In some cases, optimizing tissue perfusion with volume expansion may resolve the acidosis without the need for the use of additional bicarbonate. However, in cases of D-lactic acidosis, additional bicarbonate is needed since the D-lactate cannot be metabolized and renal excretion is more efficient if we provide additional sodium.[4]

Ideally, fluid administration would occur over a 24-hour period and not exceed 90 mL/kg/hr, but rapid administration of up to 8 L within 4 hours has been achieved without complications. Another approach is to deliver 3-4 L intravenously (takes about 1 hour) then follow up with 2 L oral electrolyte solution in 6-12 hours when the calf has responded to treatment.

There are situations where jugular catheters and large volumes of IV fluids cannot be effectively administered on the farm. In these cases, a single administration of hypertonic sodium bicarbonate (HSB) (10 mL/kg over 10 min) can act as a rapid alkalinizing agent and osmotically draw extracellular fluid into vascular for rapid vascular expansion. Oral fluids must be administered via esophageal feeder at the same time in order to replace the fluid loss from the extracellular space.

Intravenous catheter placement

Once the practitioner has decided to place an IV catheter, supplies must be gathered and procedure performed aseptically in the field. Supply list includes sterile gloves, clippers, surgical scrub (e.g., betadine and isopropyl alcohol), lidocaine – 3 mL for local block, 15 blade – to incise skin, suture, 14-18-gauge indwelling catheters for jugular vein, and 22-gauge indwelling catheters for ear vein catheterization, 2-inch catheters are used as more long term use for up to 3 days, extension set and catheter endcap, heparinized saline, and adhesive tape. There are multiple options of venous catheter placement, the author prefers jugular or auricular vein catherization. Both have challenges and benefits including distension of the vessel and securing the catheter appropriately. Basics of catherization includes: clip and aseptically prepare the area, hold off vein, insert the catheter and secure it in place.

Treatment response

Following effective replacement fluid therapy, several clinical events should occur including urination within 30-60 min, improvement in mental and hydration status, restoration of suckle reflex, recumbent calves should stand within a couple hours.[5] If these signs are not observed within hours of treatment, suspect more severe disease processes such as septicemia, meningitis or pneumonia.

Additional treatments

Mild cases of diarrhea where the calf is not dehydrated and remains bright and alert should not be treated with antibiotics. Calves with diarrhea regardless of the cause have small intestinal overgrowth of *E. coli*. Twenty to 30% of systemically ill calves have *E. coli* bacteremia. Bacteremia should also be considered in calves showing clinical signs consistent with salmonellosis. Antibiotic therapy may eliminate D-lactate-producing bacteria and improve the outcome of calves with metabolic acidosis.

Diarrhea is an inflammatory condition and appears to induce painful behavior in calves (hunching, straining and bruxism). Experimental studies have demonstrated several benefits in calves following non-steroidal anti-inflammatory drug administration including improved appetite, hydration status and activity with decreased days of morbidity. Several other ancillary treatments for calf diarrhea have been proposed. These include bismuth subsalicylate, aspirin, kaopectate, psyillium, oligosaccharides, B vitamins, intestinal acidifiying agents, prokinetics and probiotics. To date there is very little supporting evidence to justify their recommendation for use.

Conclusions

Understanding the common presentations and metabolic derangements in ill patients is critical for the successful treatment in ambulatory practice. The use of practical fluid therapy, allowing the correction of dehydration and acidosis, is needed when dealing with neonatal calves. When response to treatment is poor, other conditions such as sepsis, hypoglycemia and shock should be considered.

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