Practical fluid therapy in ambulatory practice

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Abstract

Large animal practitioners are faced with varying levels of dehydration in both mature and neonatal cattle. The goal of this talk will be to describe and formulate fluid therapy plans in varying circumstances in an ambulatory setting. Determining dehydration, acid-base status, and electrolyte abnormalities will be reviewed. Following this presentation, the practitioner will be able to identify the proper use of oral and intravenous fluid in clinical cases and appropriate volume to be administered. Homemade fluid recipes will be discussed including pros and cons compared to commercially available products.

Key words: cattle, neonatal scours, fluid therapy

Introduction

Large animal practitioners are presented with varying levels of dehydration in 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 both adult cattle and neonatal calves in an ambulatory setting. Determining dehydration, acid-base status, and electrolyte abnormalities will be reviewed.

Fluid therapy generalities

Most adult livestock with gastrointestinal disease suffers from metabolic alkalosis. In adults, metabolic acidosis is more common in the following conditions: grain overload (ruminal acidosis), urinary tract disease, intestinal strangulation, and chronic enteritis/diarrhea. Outside of these aforementioned conditions, the large animal veterinary can assume that the most common metabolic derangement in adult ruminants is hypochloridemia, hypokalemia, and hypocalcemia (dairy cattle) frequently accompany acid-base imbalances. Since laboratory analysis is not always possible, assumptions of acid-base balance must be made based on presumptive diagnosis. In anorexic, lactating or gestating livestock, glucose supplementation is critical.

Many of 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 HCO_3- rich fluid in diarrhea results in a low systemic HCO_3- 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 x 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 canthus, estimate the eyeball recession in millimeters and multiply by 2 to get the estimated dehydration (i.e. percent dehydration = eyeball recession [mm] x 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 upper limit for choosing oral versus IV fluid therapy. Thus, less clinically dehydrated patients, including calves that are maintain a suckle reflex, are good candidates for oral fluid resuscitation. In adult cattle, 5-10 gallons of oral solution can be administered at one time. Oral fluids are contraindicated if the animal already has abdominal distension, vagal indigestion or a mechanical gastrointestinal obstruction is suspected.

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) cotransport molecules (glucose, acetate, glycine); 3) an alkalinizing agent (acetate, propionate, 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 a lot of evidence to show calves gain more weight and avoid severe negative energy balance when milk is fed.[5]

In more several dehydrated adult cattle combination therapy of oral and IV fluid resuscitation can be used. Since adults are are more likely to have metabolic alkalosis, use of an acidifying solution such as hypertonic saline, 5-7% NaCl, is beneficial. Hypertonic saline can be used as resuscitation in cases of shock including hypovolemic shock from acute blood loss or in septic or severely dehydrated adult cattle. It is administered at a 4ml/kg as a rapid bolus, this solution must be given as a rapid bolus to observe all of the physiological benefits although short acting. Benefits of hypertonic saline administration include: drawing fluids from intracellular and interstitial space to the vascular space, improving cardiac output, improving renal perfusion, increasing blood pressure, and possibly being cardioprotective during times of sepsis. Ensure that the animal has access to water or is given additional oral or IV fluids after administration to assist fluid equilibration.

For more severely dehydrated calves, including patients that are recumbent, absent suckle and have decreased mentation, intravenous fluid therapy is indicated.[1] There are several IV electrolyte solutions commercially available for the treatment of calves. All of these products are effective, but the best ones contain 3 key ingredients: 1) an alkalinizing agent (HCO_3, 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 and 100 ml 50% dextrose (5% final solution) per one 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] When correcting acidosis with sodium bicarbonate, 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 hr period and not exceed 90 ml/kg/hr, but rapid administration of up to 8 L within 4 hr 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 hr 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.

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 and 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 neonatal 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-thirty percent 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, psyllium, 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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