

Salt Poisoning as a Cause of Morbidity and Mortality in Neonatal Dairy Calves

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An investigation was performed to determine the cause of increased morbidity and mortality in calves <21 days of age on a large commercial dairy farm. Historically, affected calves suffered from anorexia, diarrhea, abnormal gait, seizures, and death between 3 and 14 days of age. Serum chemistry determinations and measurement of the sodium content of milk replacer and water used on the farm identified salt poisoning as the cause of the clinical signs. The use of high salinity water to mix milk replacer, mixing of oral electrolyte powder directly into liquid feed, adding additional milk replacer powder to the same volume of water for daily cold weather feedings, and limited access to water contributed to the salt poisoning. Morbidity and mortality rates decreased after switching to an entirely whole-milk diet, mixing oral electrolyte powder in water according to label instructions, and providing ad libitum access to normal salinity water.

Sodium is the major cation in extracellular fluid, and under normal conditions serves as a marker of total body water. Its osmotic effect is important in establishing concentration gradients that drive fluid shifts between the extracellular and intracellular fluid compartments. Active and passive transport mechanisms in the body facilitate these processes. Maintenance of sodium homeostasis is critical to maintaining cellular hydration and preventing rapid fluxes of water between compartments.^{1,2}

Sodium imbalances may be caused by dysfunction in either sodium or water regulation. In dairy calves, milk replacer or milk sodium content and water consumption are the major dietary contributors to serum sodium concentration.³ Less common contributors may be high salinity water, oral electrolyte powders, and sodium-containing feed additives or medications such as lasalocid that also might provide a substantial amount of sodium to the calf.⁴ Inappropriately high concentration of the milk replacer solution, milk replacer mixing errors, lack of access to water, dehydration

Abbreviations:

BVD	bovine virus diarrhea
CNS	central nervous system
ECF	extracellular fluid
ICF	interstitial cell fluid
SUN	serum urea nitrogen

from disease, or a combination of these factors also can result in hypernatremia.^{3–5} Regardless of the cause, hypernatremia causes inhibition of neuronal cell glycolysis and results in central nervous system (CNS) depression.^{1,3} Hypernatremia also causes plasma osmolality to increase relative to tissue osmolality. This is particularly important with nervous tissue in which neurons effectively shrink as water is lost to the extracellular space in an attempt to equilibrate the osmotic gradient between the intracellular and extracellular space. A partially protective mechanism occurs with accumulation of amino acids, sugars, and alcohols in neuronal cells in an attempt to balance the intra- and extracellular osmolality.^{6,7} Correcting hypernatremia can be difficult because the animal is predisposed to additional neuronal damage as fluid shifts into the intracellular space creating cellular edema and possible brain herniation through the foramen magnum.^{1,3} The current report describes acute and chronic salt toxicity in neonatal dairy calves on a Wisconsin dairy from November 2010 to February 2011 in which abnormally high concentrations of sodium were detected in sera collected from these animals and in the water in which a commercial milk replacer powder was being mixed.

The dairy that is the subject of this report milks approximately 500 Holstein, Jersey, and Holstein-Jersey mixed breed cows. The calves are born at the dairy, separated from the dam, moved to an individual pen in the calf barn, and fed colostrum. Calves are fed whatever volume of colostrum that they voluntarily suckle, up to a maximum volume of 2 quarts for the Jersey and 4 quarts for the Holstein calves. Colostrum force-feeding occurs only when the calf does not suckle at all. Water by itself is not provided until 1 month of age. Whole milk is fed for the first 3–5 days of life followed by milk replacer, which is fed until 8–12 weeks of age, when weaning occurs.

The primary complaint by the farm owner that led to this herd investigation was high mortality in calves between 3 and 14 days of age, starting in November 2010. The farm had lost 90% of its newborn calves in the interim period (approximately 4 months) for a total of 85 calves. Calves that did not die had a tendency to develop diarrhea at 3–5 days of age with recovery approximately 14 days later. Abnormal gaits

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and seizures had occurred in several of the calves. Since the onset of the problem, attempts at treatment included addition of electrolyte powder to the liquid feed, administration of sodium lasalocid to the liquid feed (which already contains lasalocid from the manufacturer) and with multiple antibiotics to no avail. Upon further questioning, it was found that the owner began increasing the amount of milk replacer powder provided to the calves in late fall to provide additional energy during the winter months. Additional history indicated that water quality had been a concern. The depth of the well had been increased and 400 gallons of sodium hypochlorite had been added to the well water supplying the calf barn to improve water quality.

The clinical signs reported by the owner included scours, ataxia, seizures, and lethargy before death. In the week before the farm visit, calf health seemed to have improved. The only management change at that time was switching the calves to an entirely whole-milk diet. Upon investigation, 3 of 29 calves were showing signs of decreased mentation, base-wide stance, and mild ataxia. Seventeen of 29 (40%) calves had respiratory disease, seven of which had 3 or more signs of respiratory disease (fever, cough, abnormal nasal discharge, ocular discharge, or otitis media) and were under no treatment. Thirteen of 29 (31%) calves had scours. Based upon the history and clinical signs observed, hypernatremia was considered the primary differential diagnosis. Other considerations were meningoencephalitis, lasalocid toxicity, bovine virus diarrhea (BVD) infection, enteric *colibacillosis*, *Salmonellosis* or other causes of diarrhea, and associated metabolic derangements or toxemia. Blood samples were collected for measurement of serum electrolyte concentrations from 3 lethargic animals that stood with abnormal extension of the head and neck and were reluctant to move. Fecal samples were obtained for *Salmonella enterica* culture and acid fast stain for *Cryptosporidium* spp. from each of 7 untreated calves, five of which had diarrhea at the time of sampling. Two milk replacer samples from the first and last calves fed for each of 6 successive feedings after the farm visit were analyzed for total solids and sodium concentration. Milk replacer total solids were determined by a Brix refractometer^a after construction of a standard curve, with known milk replacer concentrations. Milk replacer sodium concentrations were determined by a VITROS[®] 5,1 FS Chemistry System^b after the milk replacer solution was agitated, centrifuged, and an aliquot of the supernatant obtained for analysis. Milk replacer powder^c also was collected, to be mixed with university water for sodium analysis and to serve as quality control standards for total solids calculations by a Brix refractometer. The farm protocol was to mix 10 ounces (283 g) of milk replacer in 2 quarts (1.9 L) of water. Results of analyses are presented in the tables as follows: milk replacer (Table 1) and well water (Table 2). Postmortem reports from 4 calves that died previously were reviewed.

Table 1. Liquid feed analysis.

Sample ID	Sodium Concentration (mmol/L)	Total Solids (%)
Milk replacer mix 1 – farm water	185	18.9
Milk replacer mix 2 – farm water	171	20.8
Milk replacer mix 3 – farm water	176	19.6
Milk replacer – university water	80	Not done
Whole milk 1	21	11.2
Whole milk 2	31	11.8

Antemortem testing disclosed severe hypernatremia in the calves with neurologic signs that were sampled. Serum sodium concentrations were 179, 187, and 193 mEq/L (reference range: 133–141 mEq/L), respectively. Chloride concentrations also were increased at 145, 134, and 149 mEq/L (reference range: 95–103), respectively. Only 1 of the 3 calves had an increased anion gap of 21 mmol/L (reference range: 12–16 mmol/L). Extremely high salinity well water (farm water, 7,100 ppm; safe level, <1,000 ppm⁸) used to mix the milk replacer on the farm resulted in extremely high milk replacer sodium concentrations, compared with considerably lower milk replacer sodium concentrations in milk replacer mixed according to farm protocol but by university water (Table 1). Fecal diagnostic testing showed no evidence of pathogenic or toxin-producing *E. coli* or *Salmonella enterica*. One of 7 (14%), 4 of 7 (57%), and 2 of 7 (29%) of the calves were shedding rotavirus, coronavirus and *Cryptosporidium* spp., respectively. No postmortem testing was performed on the day of the farm visit but the reports from 4 previously examined calves indicated no gross lesions and no evidence of meningoencephalitis, BVD infection, or *Salmonellosis*. No lung or intestinal pathogens were identified, including toxin-producing *E. coli*.

Salt toxicity outbreaks have been reported in cattle on pasture^{9–13} and in a few calves,^{3,4} but none have involved a herd problem associated with dairy calves. Access to a salt lick without free-choice water created signs of salt toxicity, including diarrhea, abdominal pain, anorexia, convulsions, muscle twitching, paresis, and death in a group of 6- to 10-month-old Holstein beef cattle.¹¹ Clinical signs seen in cattle grazing on coastal pastures with high salinity water (15,744 ppm) and marsh grass (10,400 ppm) included diarrhea and excitability.¹³ In another report, mature beef cows showed signs of recumbency, seizures, opisthotonus, caudal ataxia, and hypersensitivity after excessive consumption of a protein supplement containing 5% sodium chloride after breaking into a salt storage shed.¹⁰ Severe neurologic signs including mentation changes (excitability or lethargy), head pressing, ataxia, blindness, recumbency, and death were noted in several adult steers that had been deprived of water after allowing access to salt after a period of salt restriction.¹² The calves in this report exhibited many of these signs, including diarrhea, convulsions, anorexia,

Table 2. Water analysis.

Sample ID	Salinity (TDS)		Sodium Concentration		Chloride Concentration	
	ppm	mmol/L	ppm	mmol/L	ppm	mmol/L
Milk room tap water	7,100	122.4	2,793	121.4	4,307	123.1
Recommended level ¹⁶	<1,000	<17.2	<100	<4.3	<250	<7.1

ataxia, and death. Lastly, the calves of this report were given approximately 2 mg/kg lasalocid between the liquid medicated suspension and the medicated milk replacer, which is less than reported toxic dosage of 5 mg/kg in neonatal calves.¹⁴ Blood, liquid feed, and water analysis confirmed the primary differential diagnosis of severe hypernatremia secondary to high salinity water, lack of water availability, unusually high milk replacer total solids concentration (Table 1), and history of adding additional sodium lasalocid to the milk replacer.

The volume of the extracellular fluid (ECF) is determined by the total body sodium content, whereas the osmolality and sodium concentration of ECF are determined by sodium and water balance.² Sodium, glucose, and serum urea nitrogen (SUN) are solutes that affect plasma osmolality. All conditions associated with hypernatremia reflect hyperosmolality and hypertonicity of the ECF.² These conditions are caused by a deficit of pure water, such as lack of water access; loss of hypotonic fluid, such as occurs during osmotic diuresis; or gain of sodium, such as during salt poisoning which may result in either relative or absolute dehydration. Hypernatremia caused by the gain of sodium results in expansion of the ECF at the expense of the intracellular fluid (ICF), that is, relative dehydration in an attempt to return plasma osmolality to normal. Hypernatremia because of deficient free-water intake or hypotonic fluid loss is associated with absolute dehydration and reduction in total body water content. Characterization of dehydration is important when determining treatment options. It is likely that the 3 calves tested in this report suffered predominately from relative dehydration because gain of salt was the primary factor predisposing to hypernatremia. Specifically in cattle, the pathogenesis of hypernatremia involves excessive intake of sodium as a result of mixing errors, excessive water loss, or prolonged administration of IV isotonic crystalloids without access to free-choice water.¹⁵ Water salinity of 7,100 ppm on this farm contributed 122.4 mEq/L of sodium from water added to the milk replacer powder. Additional sodium from the milk replacer powder (approximately 60 mEq/L), electrolyte powder (3% sodium), and 2 times the recommended lasalocid concentration fed to calves with no access to free-choice water likely contributed to the early onset of clinical signs and death. No deaths occurred in calves fed colostrum and whole milk exclusively.

Multiple factors contributed to salt poisoning in the neonatal dairy calves on this farm. The importance of the availability of free-choice water, appropriate

mixing of milk replacer and electrolyte solutions, as well as water salinity testing are evident. Two of the 3 calves with hypernatremia died. The surviving calf was given no access to water. Instead, it was fed whole milk and offered a progressively more dilute oral electrolyte solution over a course of 7 days. Water was offered on day eight after the farm visit.

Footnotes

^a Reichert r²mini Digital refractometer, Reichert Technologies, Inc, Buffalo, NY

^b Ortho-Clinical Diagnostics, Inc, Rochester, NY

^c Nutrena Dairyway 22-20, Minneapolis, MN

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References

1. Angelos SM, Van Metre DC. Treatment of sodium balance disorders. Water intoxication and salt toxicity. *Vet Clin North Am Food Anim Pract* 1999;15:587–607.
2. Bagshaw SM, Townsend DR, McDermid RC. Disorders of sodium and water balance in hospitalized patients. *Can J Anesth/J Can Anesth* 2009;56:151–167.
3. Pearson EG, Kallfelz FA. A case of presumptive salt poisoning (water deprivation) in veal calves. *Cornell Vet* 1982;72:142–149.
4. Pringle JK, Berthiaume LM. Hypernatremia in calves. *J Vet Intern Med* 1988;2:66–70.13.
5. Carmalt JL, Baptiste KE, Naylor JM. Hypernatremia in neonatal elk calves: 30 cases (1988–1998). *J Am Vet Med Assoc* 2000;216:68–70.
6. Angelos SM, Smith BP, George LW, et al. Treatment of hypernatremia in an acidotic neonatal calf. *J Am Vet Med Assoc* 1999;214:1364–1367.
7. Lien YH, Shapiro JI, Chan L. Effects of hypernatremia on organic brain osmoles. *J Clin Invest* 1990;85:1427–1435.
8. Mount ME, Feldman BF. Practical toxicologic diagnosis. *Mod Vet Prac* 1984;65(8):589–595.
9. Osweiler GD, Carr TP, Sanderson TL, Kinker JA. Water deprivation-sodium ion toxicosis in cattle. *J Vet Diagn Invest* 1995;7:583–585.
10. Sandals WC. Acute salt poisoning in cattle. *Can Vet J* 1978;19:136–137.
11. Senturk S, Cihan H. Salt poisoning in beef cattle. *Vet Human Toxicol* 2004;46:26–27.
12. Trueman KF, Clague DC. Sodium chloride poisoning in cattle. *Aust Vet J* 1978;54:89–91.

13. Van Leeuwen JA. Salt poisoning in beef cattle on coastal pasture on Prince Edward Island. *Can Vet J* 1999; 40:347-348.

14. Benson JE, Ensley SM, Carson TL, et al. Lasalocid toxicosis in neonatal calves. *J Vet Diagn Invest* 1998;10:210-214.

15. Abutarbush SM, Petrie L. Treatment of hypernatremia in neonatal calves with diarrhea. *Can Vet J* 2007;48:184-187.

16. National Research Council. *Nutrient Requirements of Dairy Cattle*, Seventh Revised Edition, 2001. Washington, DC: The National Academies Press; 2001.