# Case Report Rapport de cas

## Treatment of hypernatremia in neonatal calves with diarrhea

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**Abstract —** Five hypernatremic, diarrheic, neonatal calves were treated mainly by the intravenous administration of 5% dextrose alone or with isotonic sodium bicarbonate. All calves recovered without complications. The average reduction rate of serum sodium concentration was about 4 times that recommended and has not been tried successfully before in hypernatremic scouring calves.

**Résumé — Traitement de l'hypernatrémie chez des veaux nouveau-nés souffrant de diarrhée.** Cinq veaux nouveau-nés hypernatrémiques, souffrant de diarrhée, ont été traités principalement par injection intraveineuse de dextrose à 5 % seul ou avec du bicarbonate de soude isotonique. Tous les veaux ont guéri sans complications. Le taux moyen de réduction de la concentration sérique de sodium était d'à peu près 4 fois celui recommandé et n'avait pas été auparavant essayé avec succès chez des veaux diarrhéiques hypernatrémiques.

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### Case 1

A 15-day-old, 41 kg, Holstein-Friesian heifer was presented to the Large Animal Clinic of the Western College of Veterinary Medicine, University of Saskatchewan, with a history of having had diarrhea for 1 d. The calf had received an unknown amount of oral electrolytes for rehydration.

#### Case description

On presentation, the calf was in lateral recumbency, depressed, and about 6% dehydrated. It had a weak sucking reflex and the perineum was stained with yellow-brown, pasty feces. The navel and the joints felt normal and no signs of septicemia were noted. The rectal temperature was 37.6°C; the heart and respiratory rates were normal. A sample of venous blood was submitted for blood gas analysis and measurement of serum electrolytes. The results revealed a hypernatremic, hyperchloremic metabolic acidosis (Table 1). The calf was given 200 mEq (200 mL) of hypertonic sodium bicarbonate ($NaHCO_3$) (8.4%) by slow IV injection, followed by 2 L of 5% dextrose at a rate of 3 drops/s (10 drops equals 1 mL), IV. The calf was monitored closely for neurological signs. Four hours later, results from a blood gas analysis and serum electrolyte determination, performed on a sample of venous blood, revealed decreased serum sodium and increased bicarbonate concentration (Table 1). The calf was then given isotonic $NaHCO_3$, 1 drop/s, IV, for the next 18 h. On day 2, the calf was bright, alert, and responsive. It had a good sucking reflex. Blood gas analysis and serum electrolyte results revealed continuing metabolic acidosis but a lower sodium concentration (Table 1). The calf was given another 2 L of 5% dextrose at 3 drops/s, followed by isotonic $NaHCO_3$ solution, at 1 drop/s, IV. The calf drank 2 L of milk and 2 L of water on day 2. On day 3, the calf showed marked improvement with blood gas and serum electrolytes in the normal range (Table 1). The feces had started to firm up, the IV fluid was discontinued, and the calf received 4 L of milk. On day 4, the calf was discharged.

### Case 2

A 7-day-old, 40 kg, Charolais heifer was presented with a history of watery diarrhea for 3 d and anorexia for the last 24 h. The calf had received 4 L of commercial oral electrolytes (undetermined trade name) the day before presentation and 2 L on the day of presentation, as a treatment for the diarrhea.

#### Case description

On presentation, the calf was in lateral recumbency, depressed, and about 10% to 12% dehydrated. It had no sucking reflex and the perineum was stained with yellow watery feces. The navel and the joints felt normal and no signs of septicemia were noticed. The rectal temperature and heart and respiratory rates were within the normal ranges, but cardiac arrhythmia was noted. Samples of venous blood were taken for blood gas analysis and serum electrolytes measurement; the results revealed hypernatremia, hyperchloremia, and metabolic acidosis (Table 1). Soon after examination, the calf started agonal

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**Table 1.** Serum electrolytes and venous blood gas analysis of 5 hypernatremic, scouring calves at presentation and after different stages of treatments

| | Normal reference range (1) | Case 1 | | | | Case 2 | | | Case 3 | | Case 4 | | | Case 5 | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | On pres. | 4 h[a] | 24 h[a] | 48 h[a] | On pres. | 9 h[a] | 24 h[a] | On pres. | 4 h[a] | On pres. | 12 h[a] | 72 h[a] | On pres. | 12 h[a] | 72 h[a] |
| Na (mmol/L) | 132–152 | 166 | 154 | 156 | 137 | 195 | 176 | 149 | 170 | 135 | 185 | 155 | 143 | 177 | 150 | 155 |
| K (mmol/L) | 3.9–5.8 | 4.3 | 3.0 | 3.6 | 2.9 | 12 | 6.1 | 4.4 | 4.5 | 3.4 | 5.1 | 4.3 | 3.9 | 6.5 | 5.2 | 4.4 |
| Cl (mmol/L) | 95–110 | 129 | 117 | 123 | 107 | 141 | 132 | 112 | 125 | 101 | 128 | 108 | 107 | 103 | 96 | 104 |
| pH | 7.35–7.50 | 7.0 | 7.1 | 7.2 | 7.3 | 7.1 | 7.3 | 7.3 | 7.0 | 7.1 | 7.0 | 7.1 | 7.3 | 7.3 | 7.3 | 7.3 |
| $pCO_2$ (mm Hg) | 34–45 | 44 | 49 | 45 | 43 | 42 | 46 | 48 | 72 | 59 | 55.3 | 42 | 51 | 77 | 71 | 50 |
| $HCO_3$ (mmol/L) | 20–30 | 11 | 14 | 18 | 22 | 14 | 20 | 23 | 16 | 18 | 14 | 14 | 25 | 38 | 37 | 22 |
| $TCO_2$ (mmol/L) | 20–30 | 12 | 15 | 20 | 23 | 15 | 22 | 24 | 18 | 20 | 15 | 15 | 27 | 40 | 39 | 23 |

[a] Post presentation
Pres. = presentation, Na = sodium, K = potassium, Cl = chloride, $pCO_2$ = carbon dioxide partial pressure, $HCO_3$ = bicarbonate ion, $TCO_2$ = total carbon dioxide content of a plasma sample

breathing and was moribund. She was given 100 mEq (100 mL) of hypertonic $NaHCO_3$ (8.4%) by a slow IV injection, and then 2 L of isotonic $NaHCO_3$, IV, as quickly as possible. The calf had become clinically stable, so the rate of IV fluids was reduced to 1 drop/s. Nine hours later, results from venous blood gas analysis and serum electrolyte determination revealed lower sodium and increased bicarbonate concentrations (Table 1). The IV fluid was changed to 5% dextrose at rate of 1 drop/s and the calf was monitored closely for neurological signs. On day 2, the calf was bright, alert, and responsive and had a good sucking reflex. Blood gas analysis and serum electrolyte results showed a mild metabolic acidosis and normal sodium concentration (Table 1). The calf was continued on isotonic $NaHCO_3$ at a rate of 1 drop/s. The calf was given a total of 2 L milk and 2 L water, PO. On day 3, the calf continued to receive lactated Ringer's solution, 1 drop/ 2s, IV. She was given 2 L of milk and 2 L of electrolytes. On day 4, the calf continued to improve and fluid therapy was discontinued. The calf was discharged on day 5.

## Case 3

A 19-day-old, 35 kg, Holstein-Friesian heifer was presented with a history of having had diarrhea for 7 d. The calf had received an undetermined amount of oral electrolytes on a daily basis.

### Case description

On presentation, the calf was in sternal recumbency and depressed. She was about 8% to 10% dehydrated, had a weak sucking reflex, and her perineum was stained with yellow pasty feces. The navel and the joints were normal and no signs of septicemia were noted. The rectal temperature was 36.7°C; the heart and respiratory rates were normal. The calf was placed under heating lamps. Samples of venous blood were taken for blood gas analysis and serum electrolytes measurement; the results revealed hypernatremia, hyperchloremia, and metabolic acidosis (Table 1). The calf was given 150 mEq (150 mL) of hypertonic $NaHCO_3$ (8.4%) by a slow IV injection, and then 4 L of 5% dextrose, 3 drops/s, IV. Four hours later, results from venous blood gas analysis and serum electrolyte determination revealed normal sodium and increased bicarbonate concentrations, but a continuing metabolic acidosis (Table 1). Then the calf was given 8 L of isotonic $NaHCO_3$ at a rate of 1 drop/s, IV. On day 2, the calf was brighter, alert, and responsive. The sucking reflex had improved. The calf was given 3.6 L of lactated Ringer's solution, 1 drop/s, IV. The IV fluid therapy was discontinued on day 2. The calf was hospitalized for 6 more days to recover from the diarrhea and the acidosis, but it was not able to maintain its hydration status. The owner elected euthanasia. Histopathological examination revealed severe damage to the intestines that appeared to be caused by *Bovine coronavirus*. No gross or microscopic lesions of the brain were found on postmortem examination.

## Case 4

A 12-day-old, 36 kg, mixed beef breed heifer was presented with a history of diarrhea and anorexia for 3 d. The calf had received 2 L of oral nutrient and electrolytes (Revibe; Wyeth Animal Health, Guelph, Ontario), (73 g/pouch, to be dissolved in 2 L of warm water) twice daily for the previous 3 d, as a treatment for the diarrhea.

### Case description

On presentation, the calf was in sternal recumbency, depressed, and about 10% dehydrated. It had a very weak sucking reflex and the perineum was stained with yellow pasty feces. The navel and the joints felt normal and no signs of septicemia were noted. The rectal temperature was 35.6°C; heart and respiratory rates were normal. The calf was placed under heating lamps. Samples of venous blood were taken for blood gas analysis and serum electrolytes measurement; the results revealed hypernatremia, hyperchloremia, and metabolic acidosis (Table 1). When the owner was questioned about the manner in which the oral electrolytes had been prepared, it appeared that an error in the appropriate dilution of the commercial oral electrolyte powder had occurred. The calf was given 150 mEq (150 mL) of hypertonic $NaHCO_3$ (8.4%) by a slow IV injection, followed by 5% dextrose, 1 drop/s, IV. Twelve hours later, results from venous blood gas analysis and serum electrolyte determination revealed lower sodium concentration and metabolic acidosis (Table 1). The IV fluid was changed to isotonic $NaHCO_3$ at a rate of 1 drop/s. On day 2, the calf was brighter, alert, and responsive. The sucking reflex had improved and fluid therapy was discontinued. On day 4, the calf was clinically normal, and results from blood gas analysis and serum electrolyte concentration were within the normal range (Table 1). The calf was discharged.

## Case 5

A 12-day-old, 35 kg, mixed beef breed heifer was presented with a similar history to case 4. They were from the same farm and presented at the same time.

### Case description

On presentation, the calf was in sternal recumbency, depressed, and about 10% to 12% dehydrated. It had a very weak sucking reflex and the perineum was stained with yellow feces. The navel and the joints felt normal and no signs of septicemia were noted. No abnormal lung sounds were heard. The rectal temperature was 37.1°C; heart and respiratory rates were normal. The calf was placed under heating lamps. Blood gas analysis and measurement of serum electrolytes, performed on a sample of venous blood, revealed hypernatremia and metabolic alkalosis (Table 1). When the owner was questioned about the manner in which the commercial oral electrolytes had been prepared, it appeared that an error in the appropriate dilution of the oral electrolyte powder had occurred. The calf was started on 5% dextrose, 1 drop/s, IV. Twelve hours later, results from venous blood gas analysis and serum electrolytes determination revealed a normal sodium concentration and mild metabolic alkalosis (Table 1). The calf was given 3.6 L of lactated Ringer's solution, 2 drops/s, IV. Then fluid therapy was discontinued. On day 2, the calf was brighter, alert, and responsive. The sucking reflex had improved. On day 4, the calf was clinically normal and was discharged (Table 1).

## Discussion

Calves with acute hypernatremia can be safely treated by using a slow drip of 5% dextrose alone or with isotonic $NaHCO_3$ solution with a serum reduction rate of about 4 mmol/L/h.

Hypernatremia is an uncommon complication encountered when treating diarrheic neonatal calves. Hypernatremia in calves is defined as an increase in the serum sodium concentration above 160 mEq/L (2). It can be acute, developing in 24 to 48 h, or chronic, developing over 4 to 7 d (2). In general, hypernatremia develops when excessive water is lost from the animal's body, thereby increasing the sodium concentration, which is common, or when excess sodium-containing salts are ingested or administered therapeutically, which is rare (3–5). The pathogenesis of hypernatremia in neonatal calves involves excessive intake of sodium without an adequate amount of water (oral electrolyte mixing errors), excessive loss of water, or long-term administration of isotonic fluids to diarrheic calves that don't have access to free water (2). The cause of hypernatremia in cases 1, 2, and 3 was not determined, but it was most likely due to water loss associated with diarrhea, although it could have been due to any of other causes. In cases 4 and 5, errors in the appropriate dilution of the oral electrolyte powder appeared to be the cause of hypernatremia.

Clinical signs of hypernatremia, that have been reported before, include lethargy, weakness, depression, seizures, coma, and death. Neurological signs associated with hypernatremia are usually caused by 2 pathogeneses: 1) brain dehydration (shrinkage) as an immediate systemic body response to hypernatremia, in which the water leaves the cells to equilibrate for the osmotic difference between the intracellular and extracellular fluids (5). This can precipitate meningeal vessel damage, subcortical hemorrhage, subdural hematomas, venous thrombosis, and infarction of the cerebral vessels (5); and 2) rapid rehydration and correction of hypernatremia. When hypernatremia develops over several days, the brain generates nondiffusible intracellular solutes, which are referred to as "idiogenic osmoles" (5–7). These help the brain against the dehydrating effect of hypernatremia by increasing the brain intracellular osmolality, which, in turn, decreases water loss from the brain, returns the brain to its normal volume, and protects the brain from the severe effect of extracellular hypertonicity. Idiogenic osmole generation starts soon after hypernatremia. However, final osmotic equilibration is not fully effected for about a week. Therefore, rapid rehydration and correction of hypernatremia results in cerebral edema and elevated intracranial pressure. The risk for developing neurological signs after rehydration is increased when the brain has partially compensated for the extracellular hypernatremia by idiogenic osmoles production, because their removal requires several days (5). In our cases, there were no clinical signs that could be related to hypernatremia alone. Hypernatremic, diarrheic neonatal calves have clinical signs that are similar to those seen in calves with uncomplicated neonatal diarrhea. Therefore, diagnosis can only be made by measuring the serum sodium concentration. However, hypernatremia should be suspected if the history is of excessive sodium intake without an adequate amount of water (oral electrolyte mixing errors), excessive loss of water, or long-term administration of isotonic fluids to diarrheic calves that don't have access to free water and in cases that develop neurological signs after treatment of dehydration by IV fluid therapy.

Treatment of hypernatremia consists of identifying the underlying cause and rectifying it and the hypertonicity (8). Underlying causes in case of neonatal calves with diarrhea include iatrogenic causes and, possibly, gastrointestinal fluid loss (8). Correction of hypertonicity requires a careful and gradual approach (8,9). If the hypernatremia has developed over a period of hours, serum sodium concentration should be reduced by 1 mmol/L/h. Reduction of serum sodium concentration in cases with prolonged or unknown duration of hypernatremia should not exceed 0.5 mmol/L/h, or about 10 mmol/L/d (8,9). The rate of the reduction in the serum sodium concentration in our cases ranged from 1.9 to 8.75 mmol/L/h with an average of 3.67 mmol/L/h. This rate is about 4 times the rate that has been recommended in the literature. However, treatment of hypernatremia as recommended in the literature (8,9) is simply not an economically feasible option in commercial calf operations, because of the treatment duration and close monitoring involved. We consider that the rate of reduction in the serum sodium concentration that we used is safe, based on the fact that treated calves appeared healthy and normal after treatment. They had normal mentation and did not show any evidence of proprioception or cranial nerve deficits; they were sucking, very aware of the surroundings, and ambulating normally. It is also economically feasible. Perhaps the iatrogenic hypernatremia in diarrheic calves develops quickly before idiogenic osmoles are

formed or produced but in a low concentration. Further studies examining cerebrospinal fluid osmolality and sodium concentrations are warranted to better understand the pathogenesis of hypernatremia in neonatal calves with diarrhea.

It is difficult to set a standard protocol for treatment of hypernatremic scouring calves because of the different complications like acidosis, alkalosis, hypoglycemia, hyperkalemia, and dehydration. These complications emphasize the importance of obtaining results from laboratory tests, blood gas analysis, and serum electrolyte determination for the treatment of scouring calves. The calves with severe acidosis and hypernatremia (cases 1,2,3,4) were given a bolus of hypertonic sodium bicarbonate to combat the acidosis and give more time for the slow correction of the hypernatremia. Perhaps this treatment had an influence on the reduction rate of the serum sodium concentration. However, we suspect that this was minimal because of the small volume administered. In case 2, the calf was given isotonic $NaHCO_3$ before the 5% dextrose solution. That resulted in the reduction of the serum sodium concentration and it may be a successful method of treating hypernatremia, but we suspect that the rate of reduction in the serum sodium concentration will be slower if this is used alone. CVJ

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