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Management of salt poisoning in an extremely low birth weight infant*

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Abstract. We present the first reported case of severe salt poisoning in an extremely low birth weight neonate. The salt poisoning was managed with the careful use of intravenous fluids, insulin to manage the severe hyperglycemia, and furosemide to induce a saline diuresis. The hypertonicity was normalized slowly over 3 days by following the corrected serum sodium (Na) (serum Na + 2.7 mEq for every 100 mg/dl of glucose over 100). No neurological damage was seen in our patient during the development of the hypertonicity or its correction. This suggests that the premature brain can develop osmoprotective molecules if hypertonicity develops slowly over 2–3 days. Slow correction is therefore recommended to avoid the development of water intoxication during correction. Despite the development of mild reversible renal failure, a large saline diuresis was induced with furosemide, thereby avoiding the need for dialysis in our patient. The only complication was the development of necrotizing enterocolitis, which has not been previously reported in association with salt poisoning.

Key words: Dialysis – Furosemide – Hypernatremia – Necrotizing enterocolitis – Premature infant – Salt poisoning

Introduction

Salt poisoning in children is a serious, life-threatening event. Current recommendations for treatment include the use of peritoneal dialysis in severe cases [1–12]. To our knowledge, no cases of severe salt poisoning have been reported in the extremely low birth weight (ELBW) neonate. We report such an infant with severe salt poisoning who was safely managed without peritoneal dialysis, but with furosemide, insulin, and hypotonic fluids instead. Slow, controlled correction of the hypertonic state was accomplished by following the "corrected" serum sodium (Na) concentration (corrected Na = serum Na + 2.7 mEq for every 100 mg/dl of glucose over 100) [13]. The only complication in our patient was a mild form of necrotizing enterocolitis (NEC) which has not previously been reported in association with salt poisoning.

Case report

The patient was a stable 31-day-old neonatal intensive care unit (NICU) patient who had been born at 27 weeks gestation with a birth weight of 760 g. She was being treated for bronchopulmonary dysplasia with ventilatory support and oral furosemide at 1 mg/kg per day in three divided doses. Formula [0.67 cal/ml, Na 13 mEq/l, potassium (K) 22 mEq/l] was given in a volume of 160 ml/kg per day. Oral sodium chloride (NaCl) supplements were added for hyponatremia (0.5 mEq/feed). On the morning of her presentation, it was noted that, for the previous 2.5 days (19 doses), the patient had been inadvertently given 5 mEq NaCl/feed, resulting in a total Na intake of 41 mEq/day.

Immediate evaluation showed no change in the patient's physical examination. Her weight was 910 g; this represented a 30-g increase over the previous 3 days and a 10-g increase from the previous day. Vital signs were stable, with a pulse rate of 170 and a blood pressure of 54/23 mmHg. She remained intubated with ventilatory settings unchanged over the previous 3 days. The remainder of her examination was normal.

Serum biochemical analysis, performed just prior to the onset of the salt overdose, showed: Na 133 mEq/l, K 4.8 mEq/l, and glucose 118 mg/dl (6.6 mmol/l). At the time the dosing error was discovered her values were: Na 189 mEq/l (corrected to 197 mEq/l for hyperglycemia), K 6.8 mEq/l (hemolyzed sample), chloride 153 mEq/l, bicarbonate 21 mEq/l, blood urea nitrogen 31 mg/dl (11.1 mmol/l), creatinine 0.7 mg/dl (62 μmol/l), and glucose 392 mg/dl (21.8 mmol/l). A spot of urine had a Na of 135 mEq/l. Her urine output for the 24 h prior to the overdose was 2.6 ml/kg per hour, and averaged 3.6 ml/kg per hour during the salt overdose.

The patient was changed to a low Na formula (0.67 col/ml, Na 7 mEq/l, K 15 mEq/l) at 175 ml/kg per day. Intravenous furosemide

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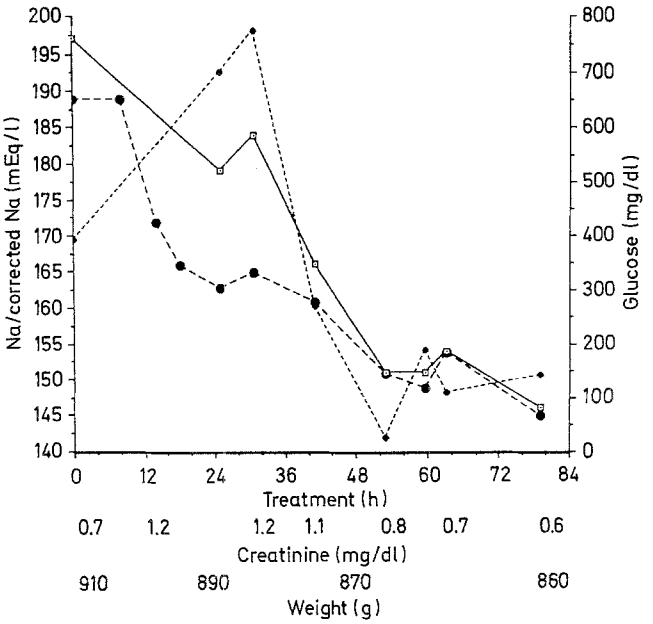

Fig. 1. The course of key clinical and laboratory variables during treatment of the hypernatremia in the salt – poisoned premature infant. ⊡, Corrected soidum (*Na*); ◆, glucose; ●, Na

Table 1. Fluid and sodium (Na) intake of the salt-poisoned premature infant over 78 h of treatment

Fluids[a, b]	Duration (h)	Volume (ml)	Na (mEq)
Formula	11	60.0	0.4
D10 W	10	57.0	0.0
D5 1/2NS	11	82.5	6.4
D5 1/2NS + 15 mEq KCl/l	14	92.5	7.1
D5 1/3NS + 15 mEq KCl/l	6	42.0	2.2
D7.5 1/3NS + 15 mEq KCl/l	21	157.5	8.1
D10 W + 10 mEq NaCl/l + 10 mEq KAc/l +20 mEq NaAc/l	5	30.0	0.9
Total	78	521.5	25.1

KCl, Potassium chloride; KAc, potassium acetate
[a] D5 W, D7.5 W, D10 W = 5,7.5, or 10 g dextrose/dl water
[b] NS = normal saline = Na concentration of 154 mEq/l

was given at a dose of 1 mg/kg per dose every 8 h. Ten hours later, she developed grossly bloody stools, abdominal distention, increased gastric residuals, and radiographic evidence of pneumatosis intestinalis. She was then placed NPO and started on intravenous fluids. During the next 68 h the rate and Na content of her intravenous fluids were adjusted to control the rate of decrease in her corrected serum Na. These fluids are shown in Table 1. When her glucose had risen to 774 mg/dl (43 mmol/l) at 28 h, an insulin drip (0.1 units/kg per hour) was started.

Relevant laboratory data are shown in Fig. 1. Her total urine output during the 78-h correction period was 298 ml, or 4.2 ml/kg per hour with an estimated total urinary Na lost of 41 mEq. This was despite a rise in creatinine to 1.2 mg/dl (106 μmol/l) in the first 24 h which then decreased to 0.6 mg/dl (53 μmol/l) by the end of treatment. Her weight at the end of treatment was 860 g.

The patient fully recovered and was discharged from the NICU at 3 months of age on a regular diet and no medications. Cranial ultrasound examinations performed 1, 2, 4, and 6 weeks after the salt poisoning were normal. At 26 weeks of age (13 weeks adjusted for prematurity) her length, weight, head circumference, and development were progressing normally.

Discussion

The hypertonic state of salt poisoning can adversely affect the central nervous system (CNS), depending on how rapidly it occurs and whether or not the brain cells have had time to generate osmoprotective molecules to preserve their size [14]. Our patient showed no evidence of CNS compromise from the hypernatremia, suggesting that such protective mechanisms are operative and can be effective, even in the extremely premature brain, if the hypernatremia develops over a 2- to 3-day period. We therefore elected to correct the hypernatremia in our patient slowly over 2–3 days, since too rapid a correction of the compensated hypertonic state can result in so-called isotonic water intoxication (brain cell swelling) [4]. Hyperglycemia, which contributes to the hypertonicity of the body fluids, has been reported to occur with hypernatremia [7]. Our patient had severe hyperglycemia, which we elected to control with insulin so we could continue to provide calories to this critically ill patient. We therefore used corrected serum Na values (corrected Na = serum Na + 2.7 mEq for every 100 mg/dl of glucose over 100) [13] rather than the actual serum Na values to adjust our fluid therapy.

Salt poisoning can cause an expansion of the extracellular fluid (ECF) space, resulting in pulmonary edema and congestive heart failure [7]. Our patient did not develop obvious pulmonary edema, congestive heart failure, or increased ventilatory requirements. We can not exclude the possibility that she could have been weaned more rapidly from the ventilator if she had not been salt poisoned. Treatment of the Na excess in salt-poisoned patients requires the net removal of Na from the patient. Peritoneal dialysis is often recommended as a method for removing Na [1–12]. A recent literature review of reported cases of salt poisoning found 20 cases in children less than 12 years of age [15]. Based on these reports, the authors were unable to decide whether peritoneal dialysis was beneficial in salt poisoning.

Our patient shows that the removal of excess Na can be accomplished through the use of loop diuretics. Although furosemide produces more water loss than Na, we were able to replace the water loss (by giving dextrose water and insulin), resulting in a net loss of Na. Our patient lost approximately 15 mEq Na/day in her urine despite the fact that she had mild transient non-oliguric renal failure. This rate of Na loss was sufficient to allow us to completely correct her salt poisoning in a timely manner.

Salt poisoning may cause oliguric acute renal failure through tubular toxicity [14]. If this occurs, or when the expanded ECF space is life threatening (e.g., fulminant pulmonary edema), some form of dialysis may be necessary for rapid Na removal. Even in these cases, other, more reliable, methods of removing Na, such as hemodialysis or arteriovenous ultrafiltration, might be considered. Peritoneal dialysis, depending on the composition of the dialysate, may be little more than an invasive method for administering free water to a patient, because the patient's ECF osmolality may exceed the osmolality of the dialysis fluid and promote water absorption from the peritoneal cavity [8, 12].

In summary, our report shows that it is possible for severe salt poisoning to be successfully treated with a loop diuretic and careful fluid management, even when the patient is an ELBW neonate with mild non-oliguric renal failure. Our case suggests that the brain cells of the ELBW neonate are capable of developing osmoprotective molecules during non-acute salt poisoning. Therefore, to avoid isotonic water intoxication, the hypertonic state should be corrected slowly over 48–72 h. Severe hyperglycemia can be safely treated with insulin if fluid management is adjusted by using the corrected serum Na (serum Na + 2.7 mEq for every 100 mg/dl of glucose over 100). Our patient also demonstrates that the premature kidney can tolerate severe salt poisoning with mild reversible non-oliguric renal failure as the only complication. Furthermore, large amounts of Na can be excreted by the premature kidney under the influence of furosemide, despite the presence of mild renal failure. Our case also suggests that NEC may be a previously unreported complication of salt poisoning in the premature infant and, if this is so, would be another reason for avoiding peritoneal dialysis in such cases.

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Literature abstract

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Failure of high-dose oral acyclovir with or without immune globulin to prevent primary cytomegalovirus disease in recipients of solid organ transplants

Thomas C. Bailey, Neil A. Ettinger, Gregory A. Storch, Elbert P. Trulock, Douglas W. Hanto, W. Claiborne Dunagan, Martin D. Jendrisak, Christopher S. McCullough, Joseph L. Kenzora, and William G. Powderly

Purpose: To assess the efficacy of acyclovir and intravenous immune globulin (IVIG) for cytomegalovirus (CMV) prophylaxis in high-risk recipients of solid organ transplants.

Patients and methods: We randomized 21 CMV-seronegative organ transplant recipients with seropositive donors (D+R–) to receive oral acyclovir, 800 mg four times daily, or, in addition to acyclovir, IVIG, 300 mg/kg, every 2 weeks for six doses. Patients were followed closely for the development of CMV infection and disease.

Results: All but one prophylactically treated patient (95%) developed CMV infection. Fifteen of 21 patients (71%) who received prophylaxis fulfilled criteria for CMV disease. Disease onset was delayed in those who received IVIG, but this did not reach statistical significance. Ganciclovir was used for treatment in 15 of the 21 patients (71%).

Conclusions: Acyclovir, with or without IVIG, did not prevent primary CMV infection or disease in D+R– solid organ transplant recipients at our institution. Moreover, most patients were treated with ganciclovir despite the use of prophylaxis. Given the ready availability of ganciclovir to treat CMV disease, we recommend a reappraisal of the role of CMV prophylaxis by these means in the solid organ transplant population.