Case Report

Can Profound Hypernatremic Dehydration Ever Be a Good Thing?

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ABSTRACT:
A 3 month-old infant with severe hypernatremia (212 mEq/L) and hyperkalemia (11.7 mEq/L) presented with no electrocardiographic cardiotoxicity. The absence of hyperkalemic electrocardiographic conduction abnormalities raised the hypothesis that the presence of hypernatremia may have been cardioprotective. Then, following large volume normal saline resuscitation asystole occurred. We suspect this caused a rapid decrease in serum sodium concentration that made the heart more susceptible to emergence of conduction disturbance.

Key Words: Hyperkalemia, Hypernatremia, Cardiotoxicity, Lamellar Ichthyosis Continuous renal replacement therapy

Introduction:
Hyperkalemia is known to alter heart depolarization (widened QRS) and repolarization (peaked T waves). Unstable cardiac rhythms that may progress to cardiopulmonary arrest are common especially at serum potassium >10mEq/L. [1] Prior clinical work has suggested a reversal of hyperkalemic cardiotoxicity with intravenous infusion of hypertonic NaCl solutions. [2, 3, 4, 5] Increases in extracellular potassium concentration decrease excitability of the myocyte. [1] This impediment to myocyte depolarization rising velocity appears to be reversed by increases in extracellular sodium concentrations. [2] Though the intravenous administration of high concentration sodium salts to treat hyponatremia has been reported to decrease coincident electrocardiographic hyperkalemic cardiotoxicity it is not known if pre-existing coexistent hypernatremia protects the heart from coexistent hyperkalemic associated cardiotoxicity.

Case Report:
A 3 month old girl with lamellar ichthyosis (LI) presented with “inactivity” after having fed poorly overnight. On admission to the emergency department (ED) her weight was 3 kg, rectal temperature 34.4°C, pulse 86/minute, blood pressure 82/53, shallow breathing at 30/minute, pulse oximeter saturation 100% and bedside blood sugar of 115 mg/dL. Her exam was remarkable for dry, erythematous skin with diffuse brown, plate-like scaling.

Initial treatment in the ED included elective tracheal intubation and intravenous (IV) infusion of 70 mL/kg of normal saline. Then, within twenty five minutes, asystole occurred which prompted 10 minutes of cardiopulmonary resuscitation (CPR) before return of spontaneous circulation. During CPR IV infusion of 4 mEq/kg of sodium bicarbonate (at concentration of 500 mEq Na/liter fluid volume) and three doses of epinephrine were infused.

Initial blood specimen was obtained 2 hours after arrival. The blood was collected from a free flowing arterial blood sample: sodium 212 mEq/L (Na), potassium 11.7 mEq/L (K), chloride 167 mEq/L (Cl), bicarbonate 30 meq/L (HCO3), blood urea nitrogen 198 mg/dL (BUN), creatinine 3.64 mg/dL (Cr), phosphorus 11.8 mmol/L (PO4), lactic acid 11.7 mmol/L. A bedside lead II electrocardiogram (ECG) was absent of peaked T waves, widened QRS or loss of P waves. Four weeks prior to this admission, a serum sodium measured 138 mEq/L, K 5.7 mEq/L, BUN 14 mg/dL, Cr 0.28 mg/dL and during the 7 week newborn hospitalization multiple assays of these values were all reported within normal ranges. After transfer from the ED to the PICU she was moving her extremities and trunk and pupillary light responses were brisk.

Calcium chloride (10 mg/kg IV) and calcium gluconate (100 mg/kg IV) were given at 3 hours into PICU stay. At approximately the same time IV insulin (0.1 units/kg) was administered along with D25 (3 grams/kg dextrose) which was followed by an insulin
drip (0.05-0.15 units/kg/hour) continuing over the next 36 hours. At 9 hours into the PICU stay urine output exceeded 10 mL/kg/hour. Aqueous vasopressin (0.172 milliUnits/kg/min) was infused for blood pressure support in addition to epinephrine (0.12 mcg/kg/min) and dopamine (15 mcg/kg/min); calculated serum and measured urine osmolalities during and after the vasopressin infusion were recorded. (Table 1).

<table>
<thead>
<tr>
<th>Time after arrival to ED (Hours)</th>
<th>Calculated serum osmolality (mOsm/kg)</th>
<th>Measured urine osmolality (mOsm/kg)</th>
<th>Vasopressin rate (milli Units/kg/hour)</th>
</tr>
</thead>
<tbody>
<tr>
<td>12</td>
<td>474</td>
<td>526</td>
<td>10</td>
</tr>
<tr>
<td>34</td>
<td>378</td>
<td>478</td>
<td>6</td>
</tr>
<tr>
<td>54</td>
<td>389</td>
<td>287</td>
<td>0</td>
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Continuous veno-venous hemodialysis (CVVHD) was initiated 14 hours after arrival in the PICU and continued for an additional 34 hours to aggressively reduce the total body potassium burden and promote gentle correction of serum sodium. (Figure 1)

The infant's neurologic status was closely monitored via physical exam and repeat head ultrasounds. She continued to have occasional, minimal movements of the extremities. Pupillary light responses could not be evaluated because of eyelid edema. Cranial ultrasound (US) obtained on hospital day one revealed mild ventriculomegaly with normal diastolic flow in pericallosal vessels. Ventriculomegaly was decreased and diastolic flow reversed on hospital days 2, 4 and 5. A brain MRI on hospital day 6 had changes consistent with diffuse hypoxic/ischemic injury and edema. The mother ultimately decided to withdraw ventilator support on hospital day 9.

**Discussion:**
Even though the history of inactivity and poor feeding spanned one night prior to presentation, it is almost certain that profound hypernatremic dehydration accompanied by severe hyperkalemia developed in our patient over a more prolonged time period. It has been proposed that a slow rise of serum potassium may provide “compensatory changes” that protect against hyperkalemic associated electrocardiographic changes. Profound hyperkalemia (with serum potassium levels as high as 10.3 mEq/L) has been reported without electrocardiographic changes; this is most common to occur in renal failure patients. A potassium level as high as 12.7 mEq/liter has been reported without accompanying ECG changes. In addition, we theorize that the simultaneous development of profound hypernatremia safeguarded the cardiac conduction system from potential fatal dysfunction that might otherwise have been accentuated in the setting of hyponatremia or even euonatremia. The presence of hypernatremia itself may have counterbalanced the detrimental effects of hyperkalemia on cardiac conduction. As extracellular potassium concentration climbs, the transmembrane...
resting potential (TRP) becomes less negative and threshold potential decreases but there is an increase in difference between the RMP and TRP leading to a decrease in excitability. These changes are responsible for a decrement in the action potential upstroke. 

The treatment of hyperkalemia should begin at serum [K] of >6.5 mEq/L. In our patient the initial serum [K] was 11.7 mEq/L. Due to difficulties encountered in establishment of vascular access, treatment of hyperkalemia involving use of calcium infusion (membrane antagonism), insulin-dextrose (redistribution of potassium), and finally CRRT (removal of potassium) was delayed until 5 hours, 5 hours and 14 hours, respectively, after arrival to the ED. During these time periods no ECG evidence of hyperkalemia was present.

Elevated extracellular sodium concentration increases the amplitude and rate of rise of the action potential; usually the sodium induced changes are of little clinical importance because it only occurs at levels incompatible with life. The administration of concentrated sodium has been proposed to foreshorten hyperkalemia associated QRS prolongation by reversing impaired sodium conductance during phase 0 of action potential. It is plausible that in this infant high serum sodium concentration helped to maintain myocyte electrical discharge rates by normalizing the rate of rise of phase 0 that would have been adversely prolonged in the setting of hyperkalemia. On surface ECG multiple readings demonstrated normal QRS and P waves and PR intervals. Serum sodium concentration was not checked before 70 mL/kg of normal saline (sodium concentration of 154 mEq/liter) was infused. It is possible that this relatively hypotonic fluid lowered the serum sodium concentration from a value higher than 212 mEq/L and resulted in hyperkalemia associated asystole.

The order of appearance of ECG changes in the setting of hyperkalemia is tenting of Twaves, widening of QRS, loss of P wave, sine wave configuration, ventricular fibrillation, and asystole. Initial recommended treatment with IV calcium is directed at restoring a diminished resting membrane potential that results from excess intracellular potassium concentration. Similarly, treatment of hyponatremia with hypertonic saline (HTS) in the setting of hyperkalemia is thought a result in the restoration of electrical properties of the myocytes. Treatment of hyperkalemic cardiotoxicity with HTS in the setting of normonatremia is not recommended; rapid increases in serum sodium concentration using IV infusions of HTS may risk development of osmotic demyelination syndrome and/or acute fluid overload.

Excessive skin water losses occur in lamellar ichthyosis and have been implicated in the pathogenesis of hypernatremic dehydration in children with this condition. Reports of hypernatremia and dehydration in this disease process have not included cases with hypernatremia this severe. In this infant the maximum urine osmolality on high dose IV aqueous vasopressin was only 526 (normal kidneys concentrate urine to 800-1400 mOsm/kg.) Though nephrogenic diabetes insipidus could have contributed to water loss and increased serum sodium concentration, urine output irregularity was more likely the manifestation of the high output phase of acute tubular necrosis.

Efforts to gradually correct profound hypernatremic dehydration by using CRRT and careful fluid management did not prevent development of what proved to be fatal brain injuries that were imaged by MRI on hospital day 9. However, implementation of CVVHD did appear to stabilize the rate of decrease of serum sodium concentration after the first 12 hours of treatment. If the sodium concentration in the CRRT bath had been higher, the rate of correction of serum sodium concentration may have been even more gradual. The protective role of CVVHD in treatment of profound hypernatremia in the setting of severe renal dysfunction is unclear.

**Conclusion:** This report describes initial survival in an infant with profound hyperkalemia and concomitant severe hypernatremia. Profound hypernatremia may protect against hyperkalemic cardiotoxicity. Rapid infusion of normal saline in the setting of profound hypernatremia may increase susceptibility of the heart to hyperkalemic cardiotoxicity and result in cardiopulmonary arrest.

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References:

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