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Successful Treatment in the Patient with Serum Sodium Level Greater than 200 mEq/L

Hypernatremia developing in nonhospitalized adults is predominantly a disease of the elderly and mentally handicapped patients, possibly revealing inadequate nursing care of these patients. It has long been claimed that the duration of hypernatremia and its rate of correction are correlated with improvement in patients' neurologic status. Since there are only a handful of cases with serum sodium levels greater than 200 mEq/L until recently, it is not clear at what rate plasma sodium concentration can be safely normalized in severe hypernatremic patients. We report a case of severe hypernatremia with survival. This patient underwent rapid correction of serum sodium concentration during the management of this metabolic derangement using isotonic solution.

Key Words: *Hypernatremia*

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INTRODUCTION

Hypernatremia is most often due to unreplaced water losses from the gastrointestinal or respiratory tracts. A serum sodium of 160 mEq/L is chosen as the cut-off point for severe hypernatremia since this is indisputably abnormal and associated with a high mortality (1). It is known that rapidly lowering plasma sodium concentration causes osmotic water movement into the brain, increasing brain size above normal which can be more dangerous than persistent hypernatremia (2). We present a case of a patient with serum sodium level greater than 200 mEq/L, and in whom hypernatremia was corrected at a rate exceeding 1 mEq/L per hour. The patient survived without any neurologic sequelae. We reviewed the literature on severe hypernatremia and to our best knowledge, we assume that this is the third case with a serum sodium level greater than 200 mEq/L due to dehydration.

CASE REPORT

A 52-year-old woman was brought to the emergency room in a comatose state. She was unresponsive to verbal and deep painful stimuli. History obtained from her family was approximately one to two days of high fever and diminished mental capability, and on the day of admission she was found comatose.

The patient had been hospitalized one year before this admission and a diagnosis of left basal ganglia hemorrhage was made. Since then, she had been unable to care for self and required considerable assistance. Her medical history was otherwise negative, and no drugs such as benzodiazepines, carbamates, or barbiturates had been ingested prior to admission.

Physical examination showed a thin woman without obvious sign of trauma, who was unresponsive but had spontaneous shallow respiration. Blood pressure was 150/100 mmHg and heart rate was 74 beats/min in the supine position. Respiratory rate was 20/min, and temperature was 36.4℃ at axilla. Her pupils were intermediate, symmetric, and reactive. There was no blood or CSF from the ears or nose. The mucous membranes were dry. The chest was clear to percussion and auscultation. Cardiac examination showed a regular rate of 70 beats/min without murmur. Examination of the abdomen showed no tenderness or masses. Bowel sounds were normal. Extremities showed no cyanosis, clubbing, edema, or deformity. The patient had corneal reflex and oculocephalic motion was normal. There was no response to deep pain and Babinski's reflexes were absent bilaterally.

Computerized axial tomographic brain scan showed multiple low density lesions in both side basal ganglia and underlying degenerative atrophic changes.

Initial laboratory evaluation showed the following values: hemoglobin, 12 g/dL; hematocrit, 37%; and WBC

count, 2,300/μL, with a normal different cell count. Serum sodium level was 206 mEq/L, potassium level was 2.2 mEq/L, chloride level was 157 mEq/L, and total CO_2 level was 34 mEq/L. Glucose concentration was 108 mg/dL, BUN level was 28 mg/dL, and creatinine value was 0.7 mg/dL (ratio, 40:1). Serum osmolarity was measured at 427 mOsm/L. The pH was 7.40, PaO_2 was 93 mmHg, $PaCO_2$ was 45 mmHg, and bicarbonate level was 28 mEq/L. Calcium level was 7.1 mg/dL. Total protein level was 5.5 g/dL. Urinalysis showed a specific gravity of 1.015, osmolality of 872 mOsm/L, occasional WBCs, and no RBCs. Urine sodium level was greater than 300 mEq/L. Electrocardiogram showed normal sinus rhythm and nonspecific ST-T wave changes, and chest rentgenogram demonstrated no abnormal findings. The patients was admitted to the medical intensive care unit where intravenous fluid was rapidly administered and serial monitoring of electrolytes and osmolalities was begun. The estimated water deficit was about 8 liters. Two liters of fluid replacement was begun over the first 12 hr through both parenteral and oral routes. Fluid balance was positive for the first 48 hr with output totaling 2000 mL of urine plus insensible loss and input totaling 6500 mL of crystalloid (normal saline). We decided to correct the natremia slowly in order to prevent cerebral edema and brain damage, as recommended by Oh and Carroll (3). Despite our effort not to correct hypernatremia too rapidly, the correction rate exceeded 1 mEq/L per hour eventually (Fig. 1). On the second hospital day, the patient began to have spontaneous movements and respond to verbal stimuli. On the third hospital day, obeying commands was possible. Her serum sodium level has been within normal range since the third hospital day. The BUN level changed to 14 mg/dL and creatinine level to 0.6 mg/dL after hydration and body weight increased to 46 kg. The falling rate of serum sodium was too fast compared to the volume administered. We assume that this was due to very high ADH level and greatest rate of free water reabsorption, and partly due to increased excretion of serum sodium into urine as the circulatory volume was restored. Combined hypokalemia was thought to be the consequence of decreased oral intake, and serum level increased slowly with supply.

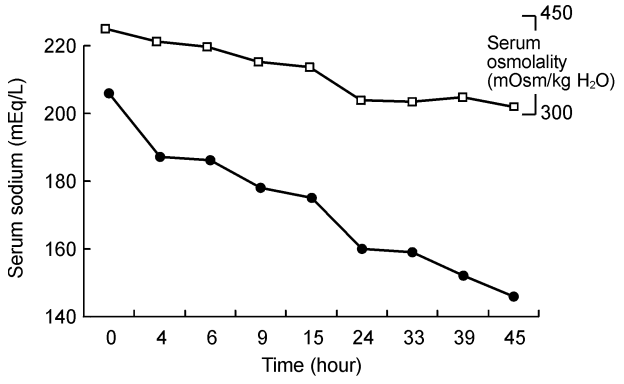

Fig. 1. Serum sodium and osmolality during treatment. Solid circles denote serum sodium values; open squares denote serum osmolality.

DISCUSSION

Hypernatremia, a cardinal disturbance of water homeostasis, is characterized by a decrease in total body water relative to total body electrolyte contents resulting in the increase of sodium concentration of extracellular fluids. Serious hypernatremia has a mortality approaching 60% in adults and almost certainly contributes to the morbidity and mortality of the underlying disease (1). Severe hypernatremia may develop through two pathologic mechanisms: excess water loss or excess solute gain (4-7). For excessive loss of water to lead to dehydration, the individual must either be unable to replace the water loss or have an altered thirst mechanism. This state occurs most commonly as the result of CNS disturbances. Hypernatremia initially causes fluid movement out of the brain and cerebral contraction that is primarily responsible for the associated symptoms. There are two phases of cerebral adaptation to hypermatremia. In response to the acute osmotic shift of water out of the brain, there is rapid uptake of electrolytes that minimizes the decrease in brain volume. This is followed by a slower adaptive phase, during which there is an accumulation of organic osmolytes, a loss of intracellular electrolytes, and the restoration of brain volume. Consequently, brain volume is largely restored due to both the uptake of solutes by the cells (thereby pulling water into the cells and restoring the cell volume) and to water movement from the cerebrospinal fluid into the brain (thereby increasing the interstitial volume) within 1 to 3 days (8, 9).

The adequate rate of correction must be addressed when plasma sodium concentration is corrected, since rapidly lowering the plasma sodium concentration causes osmotic water movement into the brain, increasing brain size above normal. This cerebral edema can then lead to seizures, permanent neurologic damage, or death (2). The rate at which organic osmolytes can be extruded from the brain and at which de-adaptation occurs dictates the rate at which water replacement can be safely administered during treatment (10).

It is known that brain water content increased by 10% when chronic hypernatremia in rabbits was corrected over a period of 8 hr and by 8% when treatment duration was extended to 24 hr. Intracellular electrolytes de-

creased rapidly, whereas organic osmolyte concentrations remained elevated after 24 hr (11). Thus, in this model of chronic hypernatremia, correction of plasma tonicity over periods of 24 hr or less was associated with significant brain edema. In contrast, a rat model of chronic hypernatremia showed that brain water content increased by only 2% despite correction of the serum sodium from 180±4 mmol/L to 155±3 mmol/L over 24 hr (9). Over an additional 24 hr of therapy, despite a further decline in the serum sodium to 143±2 mmol/L, brain water content returned to normal.

In human cases, the sequence of an adverse response to therapy has been primarily described in children in whom the hypernatremia was corrected at a rate exceeding 0.7 mEq/L per hour (12). In comparison no neurologic sequela was induced if the plasma sodium concentration was lowered at 0.5 mEq/L per hour (13).

Oh and Carroll recommended that the rate of correction should not exceed 0.7 mEq/L per hour, or about 10% of the natremia per day (3) and several series reported that improvement in mental status was greatest in patients in whom the hypernatremia was corrected over 2 to 4 days as compared with patients in whom correction occurred within 24 hr (10). At present, the standard recommendation for the treatment of hypernatremia is that, in addition to replacement of ongoing water losses, approximately half of the water deficit be replaced during the first 24 hr and that the remainder of the deficit be replenished during the subsequent 2 to 3 days (14-16). However, it is not clear whether these principles could be applied to adult patients presenting with severe hypernatremia and comatose mental changes.

In our case the correction rate of hypernatremia over the first 24 hr was 1.9 mEq/L per hour, which was far greater than that recommended, and during the first few hours a notable rapid correction rate was achieved unintentionally (4.8 mEq/L per hour).

It is known that a rapid correction can only be made if the hypernatremia has been present less than a few hours (e.g., in some cases of acute salt intoxication) but in our case, we could not find any neurologic signs of rehydration brain edema despite the rapid correction rate.

Since there are some differences between children and adults, and extrapolating from animal studies is difficult, it is not certain that the correction rate of 0.5 mEq/L per hour is preferable in severely hypernatremic adult patients.

In summary, we present here a case of severe hypernatremia corrected at a rate exceeding 1 mEq/L per hour without cerebral edema. This case shows a possibility that the optimal correction rate of serum sodium concentration in severe hypernatremia could be different from that in usual hypernatremia.

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