Desmopression is an effective adjunct treatment for reversing excessive hyponatremia overcorrection

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Abstract

We report a case of a 50-year-old malnourished African American male with hiccups, nausea and vomiting who was brought to the Emergency Department after repeated seizures at home. Laboratory evaluations revealed sodium (Na⁺) 107 mmol/L, unmeasurably low potassium, chloride < 60 mmol/L, bicarbonate of 38 mmol/L and serum osmolality 217 mOsm/kg. Seizures were controlled with 3% saline IV. Once nausea was controlled with iv antiemetics, he developed large volume free water diuresis with 6 L of dilute urine in 8 h (urine osmolality 40-60 mOsm/kg) and serum sodium rapidly rose to 126 mmol/L in 12 h. Both intravenous desmopressin and 5% dextrose in water was given to achieve a concentrated urine and to temporarily reverse the acute rise of sodium, respectively. Serum Na⁺ was gradually re-corrected in 2-3 mmol/L daily increments from 118 mmol/L until 130 mmol/L. Hypokalemia was slowly corrected with resultant auto-correction of metabolic alkalosis. The patient discharged home with no neurological sequelae on the 11th hospital day. In euvolemic hyponatremic patients controlling nausea may contribute to unpredictable free water diuresis. The addition of an antidiuretic hormone analog, such as desmopressin can limit urine output and prevent an unpredictable rise of the serum sodium.

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Core tip: In euvolemic hyponatremic patients, controlling the underlying reason of excessive vasopressin secretion may lead to sudden, large-volume free water diuresis and rise of serum sodium exceeding 12 mmol/L per day. Polyuria after presentation with symptomatic hyponatremia is a serious warning sign and should not be ignored. These patients need frequent electrolyte monitoring and, in case of excessive rise of serum sodium, pure water replacement with 5% dextrose in water to achieve a targeted reduction in serum sodium levels. Early addition of an antidiuretic hormone analog, such as desmopressin, can limit urine output and improve patient outcome.

INTRODUCTION

Acute hyponatremia can cause death if cerebral edema and seizures are not treated promptly\(^1\). Conversely, osmotic demyelination syndrome (ODS) will occur with rapid correction of severe chronic hyponatremia (serum sodium concentration \(120 \text{ mmol/L or less}\)) that has been present for more than 2 or 3 d, the time required for the cerebral adaptation to occur\(^2\). Excessive correction of chronic hyponatremia triggers a cascade of injury in the brain beginning with breakdown of the blood-brain barrier and culminating in the programmed death of oligodendrocytes, the cells that form myelin sheaths in the central nervous system\(^3\). Known risk factors for ODS are hyponatremia (both in duration and severity), rapid correction of hyponatremia with more than \(12 \text{ mmol/L in less than 24 h, hypokalemia on presentation}\(^4\)\(^5\), low BUN with malnourished state, alcoholism, liver disease and seizures on presentation\(^6\)\(^7\).


For these reasons, administration of vasopressin or synthetic vasopressin analog, such as desmopressin may be a more attractive strategy. Our paper strengthens and confirms the limited published experience to date with the use of desmopressin to prevent or reverse overcorrection of hyponatremia, in face of co-existing complex electrolyte disturbances.  

In conclusion, controlling nausea or any other reversible causes of excessive vasopressin release may lead to unpredictable free water diuresis in euvesmolic hyponatremic patients. Poluria after symptomatic hyponatremia on presentation is a serious warning sign. Early addition of an antidiuretic hormone analog, such as desmopressin, can limit urine output and prevent unpredictable free water losses with sudden rise in serum sodium, simplifying the managements of these complex and high-risk scenarios.

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