Case report

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Role of vasopressin in hyponatremic dehydration

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Abstract

Excessive correction of hyponatremia frequently occurs in the management of hyponatremic dehydration. This is more common in infants when correction of dehydration rapidly covers the hyponatremia faster than desired. This is the case of a 2½-month-old male infant with hyponatremic dehydration, which was overcorrected during the initial phase of treatment and was later managed by subcutaneous vasopressin. Vasopressin has got a dual role in the management of hyponatremic dehydration as it helps in dehydration correction as well as slow correction of sodium.

Key words: Vasopressin, hyponatremia, dehydration, sodium.

Case presentation

A 2½-month-old male infant with known case of hirschsprung disease, status post ileostomy on pediatric surgical follow-up was brought to outpatient department with complaints of poor feeding for 2 days duration and occasional non bilious vomiting. History of high stoma losses from ileostomy was present. No history of decreased urine output. On examination, the baby was found to be irritable, severely dehydrated with sunken eyes and depressed anterior fontanelle. He was tachycardic with stable other vitals. Systemic examination was within normal limits. Blood investigations revealed a serum sodium value of 107.3 mmol/l and he was admitted in pediatric intensive care unit for further management. The baby had no symptoms of acute hyponatremia like seizures or altered sensorium. Urine sodium level was 9.7 mmol/l, hence the possibility of increased ileostomy loss was considered as the cause of hyponatremic dehydration. Since there were no recent documented sodium values and the ileostomy losses were chronic, the hyponatremia was assumed to be chronic. Other electrolytes were within the normal limits. Initially, baby was administered normal saline (NS) bolus and 0.9% NS with 5% dextrose was started as the maintenance fluid. Repeat sodium value after 6 hours was 122.1 mmol/l, which was more than the desired rise. Therefore, we decided to introduce injection vasopressin 0.5 U treatment, subcutaneously. Subsequent sodium values showed gradual increase and came up to 130 mmol/l in the next 48 hours. Total 6 doses of subcutaneous vasopressin were administered at 4 hours interval. As the serum sodium improved, the baby was commenced on feeds, which he well tolerated. Gradually, the baby was put on direct breastfeeds alternating with neocate feeds. The baby improved symptomatically with documented weight gain. At discharge the baby was afebrile and hemodynamically stable.

Discussion

Electrolyte disturbances constitute a routine complication in most critical care units. (1,2) Hypo-

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natremia is the most commonly observed electrolyte abnormality seen in approximately 25% of hospitalized children. (2) Hyponatremia, defined as the serum sodium concentration <135 mmol/l, occurs in up to 8% of the population and up to 60% of the inpatients. (1,3) Hyponatremia can be classified into acute (<24-48 hours) or chronic (>48 hours) based on the time taken by cells to adapt. Therefore, to allow time for the dissipation of these adaptive mechanisms, one need to correct the serum sodium levels gradually, not exceeding the rate of 8-12 mmol/l during the initial 24 hours and <18 mmol/l during the first 2 days. (2,4) Hyponatremia with dehydration is recognized as hypovolemic hyponatremia. (2) Infants, especially neonates, are vulnerable to dehydration due to the lack of their ability to access fluids themselves without depending on their caregivers. In moderate to severe cases, children with hyponatremic dehydration have serum osmolarity <270 mOsm/kg and very likely require prompt circulatory support. Maintenance and deficit volumes can be infused cautiously, not exceeding 12-15 mmol/l over the 24 hour time period to avoid overcorrection. In case of hyponatremic dehydration, antidiuretic hormone (ADH) is often released further diluting the intravascular solute by the reabsorption of water. (5) Vasopressin is a hormone that acts on vasopressin 1 (V1) and vasopressin 2 (V2) receptors. (6) However, they function as an antidiuretic hormone, predominately at the V2 receptors of the collecting ducts in the kidneys by activating protein kinase A and aquaporin 2 water channels. (7) Although few studies on desmopressin (synthetic analogue of vasopressin) administration for the overcorrection of serum sodium exists, administration of vasopressin remains novel for the management of rapid increase in serum sodium levels in hypovolemic hyponatremic dehydration. (8)

Treatment of symptomatic hyponatremia is complicated because over correction of hyponatremia carries a risk of development of osmotic demyelination syndrome (ODS). Therefore, it is advised not to correct the serum sodium level in excess of 0.5 mmol/l/h. In this particular case, the baby was hyponatremic with serum sodium level of 107.3 mmol/l and severely dehydrated. Initial treatment involved NS bolus and 0.9% NS with 5% dextrose at a maintenance rate. Repeat sodium after 6 hours was 122.1 mmol/l (i.e.14.8 mmol/l increase). Accordingly, we recommended prompt intervention to facilitate gradual rise in serum sodium levels. Intravenous dextrose, parenteral desmopressin or both given simultaneously report to have reversed the overcorrection of sodium levels according to some studies. (7) Here, injection of vasopressin 0.5 U subcutaneous was considered and appeared to be safe and effective. Subsequent sodium levels revealed a gradual increase to 138 mmol/l over the next 48-72 hours. The baby referred in this case was hyponatremic and dehydrated, necessitating dehydration correction and simultaneously slow correction of sodium. Vasopressin assisted us in both aspects. Vasopressin act as an antidiuretic at the V2 receptor. The release of vasopressin from the posterior pituitary primarily depends on the effective osmolality of the serum i.e. under normal circumstances ADH is released in response to decreased plasma volume and increased serum osmolality, which exists in dehydration. (4) When rapid dehydration correction is given, it leads to volume expansion and suppression of ADH, which leads to diluted urine and rapid rise of sodium. Also, the sodium content of isotonic fluids used for correcting dehydration overcorrects the sodium. When we supplement with vasopressin, we can prevent the rapid rise of sodium. This proves that the administration of vasopressin can be a successful strategy to treat inadvertent overcorrection of hyponatremia.

In this case, we observed that vasopressin had a significant effect on gradual correction of sodium levels. Except for a few studies that suggest to end the current treatment and consider re-lowering of serum sodium by administration of hypotonic infusions and desmopressin, there are no protocol-based studies available for the overcorrection of hyponatremia. Here, vasopressin injection appeared to be safe and effective. However, more studies are vital to assess the clinical efficacy of vasopressin for the treatment of overly rapid correction of serum sodium. It is probably beneficial to start vasopressin prophylactically along with dehydration correction in scenarios with chronic hyponatremic dehydration, where rapid correction of sodium is anticipated, and the risks of osmotic demyelination are high.

Conflict of interest
All authors have none to declare.

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