Case report

UNINTENDED OVERCORRECTION OF HYponatremia IN A PATIENT WITH SUSPECTED SMA SYNDROME

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Keyword

Hyponatremia, SMA syndrome, overcorrection

Abstract

\textbf{Introduction} Hyponatremia (Sodium level less than 135 mmol/l) (1) is one of the most important and common electrolyte abnormalities with prevalence of about 15–20\% of emergency admissions. It will increase mortality, morbidity and length of hospital stay. And also management of these patients remains problematic.\textsuperscript{(2)}

A number of conditions impair water excretion temporarily. When the cause of water retention ends, excretion of dilute urine increases the plasma sodium concentration more than what the clinician expects\textsuperscript{(3)}.

\[\text{Continued...}\]

1. \textbf{Introduction}

Hyponatremia (Sodium level less than 135 mmol/l) (1) is one of the most important and common electrolyte abnormalities with prevalence of about 15–20\% of emergency admissions. It will increase mortality, morbidity and length of hospital stay. And also management of these patients remains problematic.\textsuperscript{(2)}
A number of conditions impair water excretion temporarily. When the cause of water retention ends, excretion of dilute urine increases the plasma sodium concentration more than what the clinician expects (3). There are several medical settings that can result in unintended overcorrection of hyponatremia (4). In these settings, the urine may become dilute greatly, so it can increase the plasma sodium concentration about 2 mEq/L/h or more (3); some examples include: Discontinuing of thiazide diuretics; Volume resuscitation in patients with high vasopressin level due to hypovolemia or low solute intake like beer potomania; adrenal insufficiency after corticosteroid replacement; Spontaneous resolution of reversible causes of the syndrome of inappropriate anti-diuresis (SIADH), such as nausea and hypoxia; Discontinuation of medications that cause SIADH such as the selective serotonin reuptake inhibitors (5).

Osmotic demyelination syndrome (ODS) is a rare but dramatic complication of hyponatremia treatment that happens when the sodium level corrects too rapid. (2, 12)

[Abstract Continued…]

Case report A recent case report describes a patient suspected to SMA syndrome (Superior Mesenteric Artery syndrome) with plasma sodium of 114 mEq/L and plasma potassium of 2.3 mEq/L at presentation that developed ODS following overcorrection of hyponatremia.

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1.1. Case Presentation

A 22 year old girl attended the emergency department with decreased level of consciousness, nausea and vomiting and history of tonic colonic seizure. Her symptoms were started from 2 months before her admission, after her divorce with nausea, vomiting and weight loss. Clinical history included depression due to PTSD (Post Traumatic Stress Disorder) & anorexia nervosa. Her drug history included Chlorpromazine from two months ago, Biperiden and Trifluoperazine from a few days ago. She does not use any cigarettes or alcohol.

At the admission time she was ill, disoriented and unable to communicate. She had general weakness. Her blood pressure was 90/60 mmHg, Pulse Rate was 110 b/min, Respiratory Rate was 20/min and axillary temperature was 36 c. She was anuric. On physical examination there was no pathological finding except abdominal distention & reduction in bowel sounds. Her lab tests are summarized in table 1.

<table>
<thead>
<tr>
<th>Na: 114 meq/l</th>
<th>WBC : 8000</th>
<th>PH : 7.53</th>
<th>BHCG : Negative</th>
</tr>
</thead>
<tbody>
<tr>
<td>K: 2.3 meq/l</td>
<td>Hg : 9.8</td>
<td>Pco2 : 55</td>
<td></td>
</tr>
<tr>
<td>Cr : 3.6 mg/dl</td>
<td>PLT : 146000</td>
<td>Hco3 : 54</td>
<td></td>
</tr>
</tbody>
</table>

Table1. Lab tests at the admission time

According to her symptomatic hypovolemia and hyponatremia she was treated with 4 vials of hypertonic saline (50cc saline 3%) and 2 liters Normal Saline and 40 meq potassium. Then she had an increase in her serum sodium to 136 meq/L over approximately 10 hours. At this time her urea was 36 mg/dl, her Cr became 2 mg/dl and her K became 3 meq/l. Her blood pressure became stable (110/70mmHg).and anuria resolved. Her urine output was 1500 cc.

On the third day of admission her nausea and vomiting continued and she became confused again. At this time, her blood pressure was 120/70 mmHg, Pulse Rate was100 b/min, Respiratory Rate was20 /min and axillary temperature was 37 c. She was unable to follow the commands and her neurological examination revealed horizontal nystagmus, dysarthria and extremities weakness. NG Tube was also inserted for her and about 4 liters biliary discharge was collected. Her hyponatremia and neurological statue had improved. Brain CT scan was done which had no pathological finding. Abdominal x. ray
showed small bowel loops dilation. So a surgery consultation was done for her; functional obstruction due to hypokalemia was suggested.

Her lab tests revealed Na: 144 meq/l, Cr: 1.8 mg/dl and K: 3 meq/l. Her urine output was 2000 cc/24h. She admitted in ICU. A nephrology consultation performed and overly rapid correction of hyponatremia suggested that caused ODS (Osmotic Demyelination Syndrome) and relowering therapy recommended as follow. So Dextrose Water 5% 1000 cc+ 50 cc kcl 15% 150cc/h and Half Saline 1000 cc +30 cc kcl 15% 100cc/h infused. And Desmopressin spray 2 PUFF was administered.

On day 5 the patient’s mental status had improved slightly and she was alert and oriented. At this time Na level was 133 meq/l, Cr: 0.7 mg/dl and her K: 5 meq/l. Na level diagrams have shown in picture1. MRI study of the brain showed no abnormality. Feeding with NG tube was started and Supportive care was continued but at the end of the day her nausea and vomiting was started again. Her NG Tube content was about 5 liter biliary discharge and undigested food. Barium study was done for her; that stomach had relocated in pelvis. (picture2) Surgery consult excluded surgical etiologies for her nausea and vomiting. Neurologic consult excluded neurological etiologies for her symptoms. Psychiatric consult: started Trifluoprazine 1mg. Gastroenterology consult candidate her for EGD (Esophago Gastro Deodenoscopy) according to small bowel loops dilation. EGD showed gastric, fundus and D1, D2 severe dilatation and there was a pulsatile pressure on the third part of duodenum so Superior Mesenteric Artery syndrome highly suggested; so total parenteral nutrition was started for her as a part of management of SMA syndrome for 2 weeks before surgery.

Fig 1. Na level diagram
During her TPN therapy she was complicated with common femoral vein and Inferior Vena Cava thrombosis, pulmonary emboli and aspiration pneumonia, so treatment with antibiotics and anticoagulant was done for her. Her signs and symptoms had improved except nausea and vomiting. EGD was done and a nasojejunal feeding tube was placed by trans nasal endoscopic technique. She did not tolerate NJ Tube and her vomiting was continued. A barium study was done for her. (picture2) Surgery consult was done and gastrojejunostomy was planned, so she underwent surgery. In operation a jejunal obstructive tumor was found and resected. Pathological exam revealed intestinal Adenocarcinoma, so chemotherapy prescribed by oncologist. She discharged the hospital and continued chemotherapy. One year later in her follow up a rise in CEA & CA19-9 was observed. Abdominal ct scan showed multiple hypo echo lesions in liver and chemotherapy started again, unfortunately she died finally because of nonresponse.

Fig 2: barium study
2. Discussion

Water balance is the basis in regulation of sodium concentration. Management of hyponatremia in some cases could be very challenging and problematic. Symptoms of this electrolyte abnormality vary from mild to severe, even life threatening. Severe symptoms caused by brain edema and raised intracranial pressure (2). These symptoms include lethargy, confusion, agitation, seizures, stupor, and coma. Despite known signs and symptoms of severe hyponatremia, chronic hyponatremia could have mild clinical symptoms like nausea, headache, gait disturbances, falling, concentration and cognitive deficits. Chronic hyponatremia could cause osteoporosis and bone fractures. Although it could increase the mortality risk.

Mild hyponatremia defined as serum sodium level between 130-135; sodium level between 125-129 known as moderate hyponatremia; and levels lower than 125 classified as severe hyponatremia. Acute hyponatremia is when it occurs less than 24-48 hours ago and chronic is when it lasts more than 24-48 hours. After correction of sodium level with serum glucose level, if hyponatremia was severe or acute, emergent treatment is mandatory. The important point in the management of hyponatremia, is when there is a combined severe hyponatremia and hypokalemia, correction of hypokalemia is the first step. Every 1 mEq correction of potassium can affect on serum sodium level as much as 1 mEq, even partial correction of potassium depletion leads to an excessive rise in serum sodium without sodium administration. The other important point is to know that the management of hypokalemia is more emergent because of cardiac arrhythmogenicity effect. These above mention key points were missed in the management of this patient. In the history review of the patient retrospectively we found out that the seizure and loss of consciousness of the patient is under question, there was no tonic colonic seizure and it was extra pyramidal drug side effects. And after the correction of hypokalemia muscle weakness had improved partially and there was no any degrees of loss of consciousness. So administration of hypertonic saline before correction of hypokalemia was not reasonable treatment and maybe dangerous.

Correction of sodium level was previously calculated with the Adrogue-Madias formula:

\[
\text{Change in serum sodium} = \frac{[\text{infusate Na}^+ + \text{infusate K}^+] - \text{serum Na}^+}{\text{Total body water (liters)} + 1}
\]

But this formula is not acceptable now because it does not include the effect of free water clearance and solute clearance and the AVP secretion. It should be kept in mind that alterations in water excretion have direct effect on serum sodium and also it depends on the nature of the solute that is urinary excreted. So the electrolyte free water clearance is now usable which better predicts directional changes in serum sodium concentration. The electrolyte-free water clearance (Ce water), is defined by the following equation:

\[
\text{Ce water} = V \times (1 - \text{urin Na}^+ \text{ urine K/plasma Na})
\]

It's important to know that to prevention of overcorrection the sodium level should check every four hours and the maximum of the correction rate is 10 mmol/24h for the first day and 8 mmol/24h for the
next days, until the sodium level reaches 130mmol/l. If it corrects more rapidly (more than 12mmol/24h or 18mmol/18h, life threatening complications will occur. Patients whose hyponatremia is caused by volume depletion, cortisol deficiency, Desmopressin or thiazide diuretics are more susceptible to overcorrection.(19) Osmotic demyelination syndrome (ODS) is a rare but dramatic complication of hyponatremia treatment that happens when the sodium level corrects too rapid. (2, 12) High risk patients for ODS complication are: serum sodium level lower than 105mmol/l, hypokalemia, alcoholism, malnutrition, advanced liver failure.(20) If overcorrection occurred, hyponatremia treatment should discontinue promptly and dextrose water should infuse 10cc/kg over one hour in combination with Desmopressin 2-4 µg until the serum sodium level has been returned to a level below the therapeutic limit for the patient. Some authors suggest the use of corticosteroids in the acute faze of ODS because of its anti-inflammatory effect on cellular shrinkage.(19, 21)

In our patient we had an unintended overcorrection of hyponatremia that caused ODS and reversed after relowering of overcorrection.

REFERENCES