CASE REPORT

HYPEROSMOLAR NONKETOTIC SYNDROME WITH HYPERNATREMIA: HOW CAN WE MONITOR TREATMENT?

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SUMMARY - We report the case of a 62 year-old symptomatic patient with severe hyperglycemic hyperosmolality associated with hypernatremia. During treatment, the progressive decrease in serum tonicity, which resulted in the amelioration of the neurological symptoms, followed the decrease in serum glucose and mainly the corrected serum sodium levels rather than the decrease in the uncorrected serum sodium levels. The case illustrates the usefulness of glucose — corrected serum sodium levels to monitor treatment in such conditions in order to avoid neurological consequences caused by the decrease in serum osmolality.

Key-words: hyperosmolality, hyperglycemia, hypernatremia, glucose-corrected serum sodium levels.

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any patients with nonketotic hyperglycemia exhibit hypernatremia due to the free water loss induced by the osmotic diuresis. In such cases, the profound water loss would counteract the hyperglycemia-induced decrease in serum sodium levels [1, 2]. In these patients fluid and insulin administration may be followed by cerebral edema from an overly rapid decrement in plasma osmolality related to a precipitous fall in both serum glucose and sodium levels [3]. Thus, it is suggested that the abrupt decrease in serum glucose should be avoided, while insulin administration should either be stopped or reduced when the plasma glucose levels decline to approximately 250-300 mg/dl, while a glucose-containing solution is usually begun at this time [1]. At the same time, although the optimal rate of correction of hypernatremia remains to be defined precisely, the general rule not to lower the serum sodium level more rapidly than 0.5 mmol/L/h seems reasonable [4]. However, in these cases the decrease in serum sodium induced by fluid administration may be counterbalanced by an increase in serum sodium levels due to the progressive decrease in serum glucose and eventually in serum osmolality leading to water entry into cells. Thus, it is pointed out that the corrected serum sodium levels after adjustment for the dilutional effect of hyperglycemia should be closely monitored during treatment. The corrected serum sodium is calculated by increasing the serum sodium concentration by 1 mmol/L for every 62 mg/dl increment in the serum glucose levels [2]. This statement is illustrated in the case reported here.

■ CASE REPORT

A patient aged 62 with type II diabetes of 5 years' duration was recently admitted with severe hyperglycemia associated with hyperosmolar nonketotic syndrome, precipitated by the patient not taking his recommended antidiabetic treatment (combination of glyburide plus metformin). Except from polyuria, polydipsia and weight loss the patient exhibited neurological abnormalities, including lethargy and obtundation, and findings indicative of profound volume depletion [decreased skin turgor, tachycardia with 120 beats/min, and hypotension (blood pressure 100/70 mmHg)]. Biochemical investigations summarized in the table revealed the presence of prerenal azotemia, hypernatremia, and hypertonicity. A loading dose of 20 units of regular insulin was initially given, followed by 8 units/h, which was then appropriately adjusted, along with careful rehydration with isotonic saline to replace the fluid losses and correct the extracellular volume depletion. The rate of fluid administration was 500 ml/h for the first 4 h followed by 250 ml/h for the next 8 h. On the second day of hospitalization, half-isotonic saline was given to replace the free-water loss induced by the osmotic diuresis as well as dextrose—containing saline solutions when serum glucose concentration fell below 300 mg/dl to avoid hypoglycemia and cerebral edema. Additionally, potassium chloride solutions were also added in the intravenously administered fluids. A progressive improvement in neurological symptoms followed the amelioration of the metabolic abnormalities throughout treatment.

■ DISCUSSION

Our data suggest that the progressive decrease in serum tonicity resulting in the improvement of neurological symptoms followed the decrease in serum glucose as well as the corrected serum sodium levels (at a rate lower than 0.5 mmol/L/h) rather than the decrease in the uncorrected serum sodium levels (Table I). These findings clearly demonstrate the dissociation between uncorrected and corrected serum sodium levels within the first 48 hours of treatment, while serum glucose levels remain above 300 mg/dl, a period which is critical in terms of the risk of cellular dehydration and neurological damage. The importance of the careful follow-up of the corrected serum sodium levels is further strengthened by the recently proposed suggestion that the serum sodium level corrected for the concomitant serum glucose value may be a useful index of the state of cerebral cellular hydration in hyperosmolar syndromes, since hyperglycemic patients with hypertonicity (tonicity > 310 mosmol/Kg) are symptomatic only if the serum sodium levels corrected for the dilutional effect of hyperglycemia are increased, as it was the case in our patient [5, 6]. In other words, hyperglycemic patients without hypernatremia are asymptomatic. The absence of neurologic symptoms in cases of severe hyperglycemia is due to the capacity of the brain tissues to restore intracellular water by accumulating electrolytes, and the so called “idiogenic osmoles”. Moreover, the brain tissue is relatively permeable to glucose even in the absence of insulin. So, hyperglycemia does not induce severe hypertonicity in the CNS [7, 8].

Thus, it is imperative that in cases with extreme hyperglycemia, not only should the serum glucose and osmolality levels be taken into account but particularly the sodium concentration after adjusting it for the dilutional effect of hyperglycemia. Our report emphasizes the fact that this procedure is especially helpful during treatment monitoring.

We conclude that the determination of glucose—corrected serum sodium levels is extremely useful for the monitoring of treatment in cases of hyperosmolar nonketotic syndrome.
REFERENCES


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**TABLE I.** Patient’s laboratory parameters.

<table>
<thead>
<tr>
<th>Days of hospitalization</th>
<th>On admission</th>
<th>after 12h</th>
<th>2nd</th>
<th>3rd</th>
<th>4th</th>
<th>5th</th>
<th>6th</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum glucose (mg/dl)</td>
<td>1 067</td>
<td>456</td>
<td>323</td>
<td>230</td>
<td>193</td>
<td>194</td>
<td>132</td>
</tr>
<tr>
<td>BUN (mg/dl)</td>
<td>68</td>
<td>62</td>
<td>58</td>
<td>37.5</td>
<td>24</td>
<td>17</td>
<td>14</td>
</tr>
<tr>
<td>Serum creatinine (mg/dl)</td>
<td>2.6</td>
<td>2.3</td>
<td>2</td>
<td>1.3</td>
<td>1.2</td>
<td>1.2</td>
<td>1</td>
</tr>
<tr>
<td>Serum sodium (mmol/L)</td>
<td>160</td>
<td>166</td>
<td>161</td>
<td>151</td>
<td>149</td>
<td>142</td>
<td>140</td>
</tr>
<tr>
<td>Corrected serum sodium (mmol/L)</td>
<td>176</td>
<td>172</td>
<td>164.5</td>
<td>153</td>
<td>151.5</td>
<td>143.5</td>
<td>140</td>
</tr>
<tr>
<td>Serum potassium (mmol/L)</td>
<td>4.7</td>
<td>3.92</td>
<td>3.67</td>
<td>4.37</td>
<td>4.8</td>
<td>4.4</td>
<td>4.8</td>
</tr>
<tr>
<td>Posm (mosmol/Kg)</td>
<td>404</td>
<td>379</td>
<td>361</td>
<td>329</td>
<td>318</td>
<td>301</td>
<td>292</td>
</tr>
<tr>
<td>Tonicity (mosmol/Kg)</td>
<td>380</td>
<td>357</td>
<td>340</td>
<td>315</td>
<td>309</td>
<td>295</td>
<td>287</td>
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