Hyponatremic Convulsion after Oral Sodium Phosphate for Bowel Preparation in a Patient with Previous Gastrectomy

A 64-year-old lady with a previous history of subtotal gastrectomy for stomach mal-toma was scheduled to undergo colono-scopic examination because of a recent change in bowel habit. She was ambula-tory on admission with normal hemody-namic parameters. Routine laboratory tests revealed normal blood counts, liver function, renal function, and levels of se-rum electrolytes (including sodium, po-tassium, calcium, and phosphate).

In the evening before the scheduled colonoscopy, she was given 45 ml of oral Fleet solution (Phospho-soda). Shortly after-wards, she developed profuse diarrhea and vomited a small amount of yellowish fluid. Further intake of Phospho-soda was therefore stopped, and 500 ml of 5% dex-trose solution was also infused intrave-nously for rehydration. However, she was found to be unarousable in bed 8 hours lat-er. Initial assessment revealed a Glasgow Coma Scale score of 5/15 without any fo-cal neurological signs. Oxygen saturation, blood pressure, and pulse remained at normal levels. She suddenly developed generalized tonic-clonic convulsion, which was stopped 2 minutes later by ad-ministration of bolus intravenous diazepam. Prompt investigations with electro-cardiography, determination of cardiac enzymes, and computed tomography (CT) scan excluded cardiac and cerebro-vascular events. The plasma glucose level was normal. Urgent biochemical tests re-vealed an extremely low plasma sodium level of 114 mmol/l (normal range 135–145 mmol/l) and a serum osmolality of 248 mOsm/kg. Plasma calcium, phos-phate and magnesium levels were all alar-mingly low (see Table 1). Meticulous cor-rection of electrolytes was arranged, in the intensive care unit. Reversal of the acute hyponatremia, hypophosphatemia and hypomagnesemia was accomplished within 48 hours by intravenous replace-ment. The patient eventually regained full consciousness, and was discharged with no permanent neurological deficits.

Oral sodium phosphate (Fleet Phospho-soda) is a bowel-cleansing agent com-monly used prior to colonoscopy. It works by inducing an osmotic influx of fluid from the intravascular compartment to the bowel lumen. In contrast to sodium phosphate, polyethylene glycol is an osmo-tically balanced preparation that cleans the bowel by physical washout of the ingested fluid. A meta-analysis compar-ing these two preparations found that sodium phosphate was superior to polyethyleneglycol with respect to cost, com-pliance, and quality of bowel preparation [1]. However, because of the osmotic ac-tivity of the chemical contents, sodium phosphate inevitably carries a possibility of inducing fluid and electrolyte disturb-ances [2–4]. Patients with impaired renal function, liver cirrhosis, congestive heart failure [5], age more than 65 years, presence of intestinal obstruction, or in-creased intestinal permeability are more prone to these complications. Our case illustrated that multiple electrolyte disturbances, notably hyponatremia, hy-pomagnesemia, hypocalcemia and hypo-phosphatemia, could happen after Phos-pho-soda was used in a patient who had previously undergone gastrectomy but otherwise lacked the abovementioned contraindications.

We postulated that the severe hyponatreme-mic convulsion and concomitant multiple electrolyte imbalances in our patient oc-curred because of additive biochemical effects of gastrectomy and sodium phos-phate. The rapid emptying of hyperosmo-lar sodium phosphate into the small in-testine with the addition of the accelerated intestinal transit after gastrectomy had probably resulted in an exaggerated intravascular volume contraction, leading to acute hypovolemic hyponatremia. The infusion of dextrose solution in an at-tempt to replace the fluid loss had further diluted the sodium concentration and other serum electrolytes (calcium, phos-phate, and magnesium; Table 1). The bio-chemical effects of sodium phosphate in patients with previous gastrectomy can be acute and sudden. Anticipatory symp-toms prior to severe clinical manifesta-tions may be absent or nonspecific as in the case of our patient. Hospital admis-sion for close clinical observation and the use of polyethylene glycol are probably more desirable options for bowel pre-pparation in this group of patients.

Table 1  Change in and subsequent correction of serum electrolyte levels, after administration of oral sodium phosphate in a 64-year-old woman with a previous gastrectomy

<table>
<thead>
<tr>
<th>Serum electrolyte (Normal range)</th>
<th>Serum electrolyte levels</th>
<th>Day before administration of sodium phosphate</th>
<th>9 hours after sodium phosphate intake (patient in unconscious state)</th>
<th>24 hours of correction</th>
<th>48 hours of correction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium (135 – 145 mmol/l)</td>
<td>143</td>
<td>114</td>
<td>136</td>
<td>134</td>
<td></td>
</tr>
<tr>
<td>Potassium (3.5 – 5.0 mmol/l)</td>
<td>3.7</td>
<td>3.5</td>
<td>3.7</td>
<td>3.8</td>
<td></td>
</tr>
<tr>
<td>Creatinine (44 – 80 umol/l)</td>
<td>72</td>
<td>48</td>
<td>60</td>
<td>48</td>
<td></td>
</tr>
<tr>
<td>Total calcium (2.15 – 2.55 mmol/l)</td>
<td>2.37</td>
<td>1.95</td>
<td>2.14</td>
<td>2.10</td>
<td></td>
</tr>
<tr>
<td>Adjusted calcium (2.15 – 2.55 mmol/l)</td>
<td>2.31</td>
<td>2.06</td>
<td>2.16</td>
<td>2.20</td>
<td></td>
</tr>
<tr>
<td>Phosphate (0.82 – 1.40 mmol/l)</td>
<td>1.12</td>
<td>0.66</td>
<td>0.61</td>
<td>1.01</td>
<td></td>
</tr>
<tr>
<td>Magnesium (0.67 – 1.01 mmol/l)</td>
<td>Not available</td>
<td>0.53</td>
<td>1.01</td>
<td>0.81</td>
<td></td>
</tr>
<tr>
<td>Osmolarity (275 – 295 mOsm/kg)</td>
<td>Not available</td>
<td>248</td>
<td>282</td>
<td>273</td>
<td></td>
</tr>
</tbody>
</table>

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S. Y. W. Liu, F. K. Y. Cheung, W. T. Siu, E. K. W. Ng
Department of Surgery, The Chinese University of Hong Kong, Hong Kong.

References


Corresponding Author

E. K. W. Ng
Department of Surgery
Prince of Wales Hospital
The Chinese University of Hong Kong
Shatin, New Territories
Hong Kong
Fax: +852-2637-7974
E-mail: endersng@surgery.cuhk.edu.hk