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

In the evening before the scheduled colono-
scopy, 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 diaze-
pam. 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-
tate and magnesium 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 Pharso-
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 os-
motically balanced preparation that
cleans the bowel by physical washout of
the ingested fluid. A meta-analysis com-
paring these two preparations found that
sodium phosphate was superior to polye-
thyleneglycol 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 distur-
bances, notably hyponatremia, hy-
ponagnesemia, 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 con-
traindications.

We postulated that the severe hyponatre-
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 acceler-
ated 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-
temt 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-
paration in this group of patients.

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

<table>
<thead>
<tr>
<th>Serum electrolyte (Normal range)</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>
</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>
</tr>
<tr>
<td>Creatinine (44 – 80 umol/l)</td>
<td>72</td>
<td>48</td>
<td>60</td>
<td>48</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>
</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>
</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>
</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>
</tr>
<tr>
<td>Osmolarity (275 – 295 mOsm/kg)</td>
<td>Not available</td>
<td>248</td>
<td>282</td>
<td>273</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.

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