

## 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 colonoscopic examination because of a recent change in bowel habit. She was ambulatory on admission with normal hemodynamic parameters. Routine laboratory tests revealed normal blood counts, liver function, renal function, and levels of serum electrolytes (including sodium, potassium, calcium, and phosphate).

In the evening before the scheduled colonoscopy, she was given 45 ml of oral Fleet solution (Phospho-soda). Shortly afterwards, 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% dextrose solution was also infused intravenously for rehydration. However, she was found to be unarousable in bed 8 hours later. Initial assessment revealed a Glasgow Coma Scale score of 5/15 without any focal 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 administration of bolus intravenous diazepam. Prompt investigations with electrocardiography, determination of cardiac enzymes, and computed tomography (CT) scan excluded cardiac and cerebrovascular events. The plasma glucose level was normal. Urgent biochemical tests revealed 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, phosphate and magnesium levels were all alarmingly low (see Table 1). Meticulous correction of electrolytes was arranged, in the intensive care unit. Reversal of the acute hyponatremia, hypophosphatemia and hypomagnesemia was accomplished within 48 hours by intravenous replacement. The patient eventually regained full consciousness, and was discharged with no permanent neurological deficits.

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

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

Oral sodium phosphate (Fleet Phospho-soda) is a bowel-cleansing agent commonly 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 osmotically balanced preparation that cleans the bowel by physical washout of the ingested fluid. A meta-analysis comparing these two preparations found that sodium phosphate was superior to polyethylene glycol with respect to cost, compliance, and quality of bowel preparation [1]. However, because of the osmotic activity of the chemical contents, sodium phosphate inevitably carries a possibility of inducing fluid and electrolyte disturbances [2–4]. Patients with impaired renal function, liver cirrhosis, congestive heart failure [5], age more than 65 years, presence of intestinal obstruction, or increased intestinal permeability are more prone to these complications. Our case illustrated that multiple electrolyte disturbances, notably hyponatremia, hypomagnesemia, hypocalcemia and hypophosphatemia, could happen after Phospho-soda was used in a patient who had previously undergone gastrectomy but otherwise lacked the abovementioned contraindications.

We postulated that the severe hyponatremic convulsion and concomitant multiple electrolyte imbalances in our patient occurred because of additive biochemical effects of gastrectomy and sodium phosphate. The rapid emptying of hyperosmolar sodium phosphate into the small intestine 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 attempt to replace the fluid loss had further diluted the sodium concentration and other serum electrolytes (calcium, phosphate, and magnesium; Table 1). The biochemical effects of sodium phosphate in patients with previous gastrectomy can be acute and sudden. Anticipatory symptoms prior to severe clinical manifestations may be absent or nonspecific as in the case of our patient. Hospital admission for close clinical observation and the use of polyethylene glycol are probably more desirable options for bowel preparation in this group of patients.

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