Fatal acute sodium phosphate enemas intoxication

Raymond Farah

Department of Internal Medicine Nahariya Hospital, B. Rappaport Faculty of Medicine, Technion, Nahariya, Israel.

Abstract

We describe a patient who died as a result of severe hypocalcaemia and hyperphosphatemia after treatment with a sodium-phosphate enema.

Physicians should be aware of the risk when using these enemas, even in normal doses, especially in elderly patients without signs of renal failure, as in our patient. (Acta gastroenterol. belg., **2005**, 68, **392-393**).

Key words : hypocalcemia, hyperphosphatemia, fleet enema, hypernatremia.

Introduction

A variety of sodium phosphate preparations have become popular as bowel-cleansing regimens and are commonly used to relieve constipation in both children and adults.

They are apparently effective although randomized clinical trials comparing them with other treatment for constipation do not exist. Their widespread use, however, is not without risk (1). A number of deaths and serious electrolyte abnormalities have been reported from the use of sodium phosphate enemas. The phosphorus load given with these preparations is high, these enemas contain a hypertonic solution of mono and dibasic sodium phosphate and the plasma phosphate concentration can rise significantly after administration of these products (1). Both the phosphate and the sodium can be absorbed resulting in hypernatremia and hyperphosphatemia, manifested usually by hypocalcaemia tetany and acute renal failure with high anion gap metabolic acidosis (2).

Most of the serious complications have been reported in situations where enemas have been given to very young children (3), in higher than the recommended dose or in situations where the patient has retained the enema due to neurological abnormality or debilitation or renal failure. In our case none of these risk factors were detected.

Case presentation

A 70-year-old male was admitted in the emergency department with mild confusion, diffuse abdominal pain and weakness. There was no fever, vomiting or diarrhoea. Medical history was limited to chronic back pain due to spondylarthropathy and chronic constipation. There was no history of chronic medications. At his initial evaluation in the emergency room, the patient was normotensive, with a pulse rate of 82, temperature of 36.4°C, respiratory rate of 14., and no sign of dyspnoea or dehydration. Lungs were clear at auscultation and heart sounds were normal. Abdominal examination revealed soft abdomen, mild tenderness, no organomegaly and normal bowel sounds. Peripheral signs such as leg oedema, spider nevi, and palmar erythema, were not noted. Laboratory findings were as follows : haemoglobin 12.5 g /dl ; hematocrit 36.8% ; WBC 8800 / mm^3 ; platelets 392000 / mm^3 ; serum sodium 140 mEq / L ; potassium 3.6 mEq / L ; chloride 101 mEq / L ; BUN 16 mg / dl ; serum creatinin 1.0 mg / dl ; uric acid 7.0 mg / dl ; calcium 9.5 mg / dl ; phosphate 3.5 mg / dl ; ALT 22 U / L ; AST 12 U / L ; total protein 8.1 g /dl ; albumin 3.9 g / dl ; glucose 98 mg /dl and ammonia 12 UMOL /L (normal ranges : 11-35). Chest and abdominal X-ray were normal. The patient was admitted to the geriatric department for further evaluation. The following day, his condition deteriorated, severe weakness, gait disorders and confusion appeared, and after revaluation of the patient, it was decided to run laboratory tests for electrolytes and blood gases. Urgently drawn blood revealed that sodium was 168 mEq /L ; potassium 5.3 mEq /L ; calcium 2.5 mg /dl ; phosphate 70.5 mg /dl ; blood pH was 7,10 ; bicarbonate 8.0 mEq/L ; serum creatinin 2.0 mg / dl, chloride 95 mEq/L, anion gap 65. Urinary sodium was 7.3 mmol /L while urine output decreased to 25 ml / hour. Electrocardiography showed normal Q-T interval. Further investigation found that the patient had been treated with two Fleet enemas on the previous evening and two more on that morning (a total of four enemas, within 12 hours, 133 ml each, every 100 ml contain 16 gr monobasic sodium phosphate and 6 gr dibasic sodium phosphate). An emergency haemodialysis was planned, but the patient was found to be unsuitable for haemodialysis due to severe hypotension which is resistant to vasopressor agents. The patient then had cardiac arrest and did not respond to resuscitation measures.

Correspondence : Raymond Farah, M.D., Internal Medicine F Department, Western Galilee Hospital, Nahariya 22100, P.O.B.21-Nahariya-Israel.
E-mail : raymondfarah@hotmail.com, Raymond.Farah@naharia.health.gov.il.

Discussion

Phosphate homeostasis is regulated by both intracellular movement of phosphorus as well as excretion of this substance by the kidneys. Normal dietary intake of phosphate ranges from 800 to 1,800 mg/day. Soluble dietary phosphates, such as those found in meats and dairy products, are nearly completely absorbed from the ileum and jejunum. Vegetable phosphate, which is often complexes in fibre, is less absorbable. Phosphorus balance is maintained predominantly by renal excretion of ingested phosphate. Phosphate in plasma exists as organic and inorganic compounds. Approximately 85% of the circulating phosphate is filtered in the glomerulus (2). The portion of plasma phosphate that is not filterable is complexed to proteins, lipoproteins, and other large molecules. Approximately 80% of filtered phosphate is actively reabsorbed in the proximal tubule, while the distal nephron passively reabsorbs 10-12%. Fine regulation of phosphate reabsorption is performed in the proximal nephron under the influence of parathyroid hormone. The occurrence of hyperphosphatemia inhibits parathyroid hormone secretion and this facilitates excretion of excess phosphate. As a result, phosphate ingestion above the usual range of intake (> 1,000 mg/day) will produce only a small rise in plasma phosphate concentration if performed over a 24 hour period in a person with normal renal function (4). However, excessive exogenous loads of phosphate or massive endogenous cellular release of phosphate can result in potentially severe hyperphosphatemia with the associated complications. Thus the diagnosis approach to hyperphosphatemia involves identification of the reason that phosphate entry into the extracellular fluid exceeds the rate at which it can be excreted. There are three general circumstances in which this occurs : massive acute phosphate load ; chronic renal failure ; and a primary increase in proximal phosphate reabsorption. Several cases of hypocalcaemia and hypernatremia as complications of sodium phosphate enemas have been described in the literature especially in infants (5,6). Additionally fatal hyperphosphatemia and electrolyte disturbances have been reported particularly in patients with risk factors such as renal failure, severe dehydration, congestive heart failure, colonic ileus or gastrointestinal bleeding (7,8,9). No similar cases of severe hypocalcaemia and hyperphosphatemia secondary to sodium laxative use in patients with normal renal function tests and without other risk factors and with the highest serum phosphorus level have been described. We believe that our patient developed hyperphosphatemia from the excessive phosphate administration and the increased absorption of phosphate due to faecal impaction that caused partial bowel obstruction. It is possible that hyperphosphatemia itself caused renal failure through a direct tubular toxic effect (10) aggravated by calcium-phosphate deposition, and the volume depletion caused by iatrogenic diarrhoea, all may contributed to the mild renal failure presented in our patient. Unfortunately dialysis is not effective in removing this amount of phosphorus and these patients quickly develop cardiac arrest secondary to the severe hypocalcaemia (11). Clinicians should be careful using these enemas in elderly patients and using only one fleet in 24 hours (as recommended), avoiding them completely in patients with known risk factors. Assessment of serum creatinin, electrolytes, calcium and phosphorus prior to sodium phosphate preparation in elderly patients should be considered, and in selected cases post procedural electrolytes assessment and correction may be required, since this simple treatment can sometime be fatal.

Summary

Sodium phosphate is widely used for colonoscopy preparation and constipation treatment, it is generally safe and easer to use, but sometime can cause severe complications and even death. We report a case of a patient without known risk factors who died as a result of severe hyperphosphatemia and hypocalcaemia after more than a single dose of fleet's Phospho-Soda. Caution is suggested in the use of this preparation in elderly patients even without renal failure and other risk factors.

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