

Case Report

Severe hypocalcaemia and hyperphosphataemia caused by oral sodium phosphate fleet solution in a haemodialysis patient after parathyroidectomy

Maggie Ming Yee Mok¹, Terence Yip¹, Sing Leung Lui², Daniel Tak Mao Chan¹, Kar Neng Lai¹ and Wai Kei Lo²

¹Department of Medicine, Queen Mary Hospital, Hong Kong, China and ²Dr Lee Iu Cheung Memorial Renal Research Centre, Tung Wah Hospital, Hong Kong, China

Correspondence and offprint requests to: Maggie Ming Yee Mok; E-mail: maggiemymok@gmail.com

Abstract

We report a case with severe electrolyte disturbance after the use of oral sodium phosphate solution (OSPS). A 69-year-old patient on haemodialysis received 45 mL of OSPS for bowel preparation. He had symptomatic hypocalcaemia with a serum calcium level of 0.95 mmol/L and serum phosphate level of 4.73 mmol/L. He was treated with haemodialysis and intravenous calcium supplementation. This patient had total parathyroidectomy recently leading to the absence of parathyroid hormone response. OSPS has been reported to cause life-threatening electrolyte disturbance especially in patients with renal failure. We suggest the use of safer alternatives for bowel preparations in renal failure patients.

Keywords: haemodialysis; hyperphosphataemia; hypocalcaemia; OSPS

Background

Oral sodium phosphate solution (OSPS) is a commonly used agent for bowel preparation before colonoscopy and surgical procedures. It is favoured by its good patient tolerability and high bowel cleansing effect [1]. However, OSPS may cause renal failure and its use is not uncommonly associated with electrolyte disturbances that may lead to cardiac arrhythmia and even death.

Case report

A 69-year-old man who had end-stage renal failure due to unknown cause and had been on haemodialysis for 8 years underwent a surveillance colonoscopy for history of colonic adenoma. Two months before the procedure, the patient received total parathyroidectomy with reimplantation of part of the parathyroid tissue for severe hyperparathyroidism. Parathyroid hormone level was lowered to 8 pg/mL after operation. He was put on calcitriol 1 µg twice daily and calcium carbonate

1250 mg daily afterwards. On admission, his serum-adjusted calcium level was 2.4 mmol/L and his phosphate level was 0.80 mmol/L. He was given 45 mL of OSPS a day before the colonoscopy procedure for bowel cleansing.

Four benign looking colonic polyps were found and they were all snared for biopsy. Later after the procedure, the patient complained of generalized weakness with muscle cramps and twitching. Blood results revealed severe hypocalcaemia of 0.95 mmol/L and hyperphosphataemia of 4.73 mmol/L (Figure 1). He urgently received a session of haemodialysis and was supplemented with intravenous calcium. The electrolyte disturbance normalized and the patient made an uneventful recovery.

Discussion

Bowel cleansing for colonoscopy is essential for proper procedure. There are two types of commonly used bowel-cleansing agents, OSPS and polyethylene glycol-electrolyte solution (PEG-ELS). In our unit, the routine agent was PEG-ELS. For this gentleman, OSPS was prescribed by a non-nephrologist and had led to serious hypocalcaemia and hyperphosphataemia in this patient. OSPS contains sodium phosphate. If defaecation does not occur, a substantial amount of phosphate and sodium will be absorbed. Patel *et al.* has done a quantitative experiment to measure the amount of phosphate absorption and excretion after a standard regime of two doses of 45 mL OSPS ingestion in five healthy subjects. Fifty-seven per cent of phosphate was excreted in the stool and 14.7% in urine. The gut absorbs 43% of the ingested phosphate and 28.3% (3.28 g phosphorus) is still retained in the body 6 h afterwards [2]. For patients with renal failure, the absorbed phosphate produces a significant rise in serum phosphate.

Normally, phosphate load stimulates parathyroid hormone secretion to increase its renal excretion and inhibits renal synthesis of calcitriol. Hyperphosphataemia also induces hypocalcaemia. With parathyroid hormones, the body aims

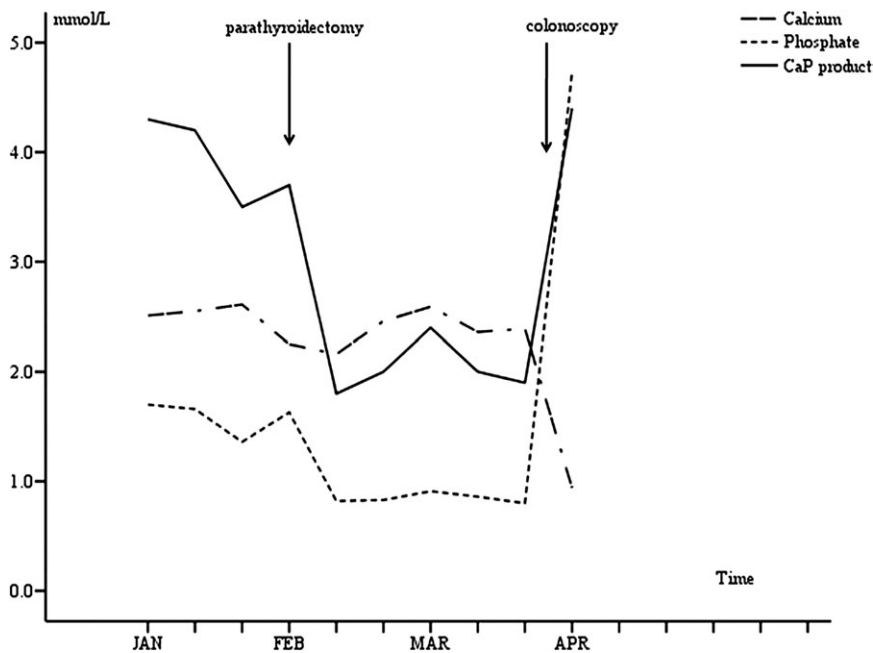


Fig. 1. Calcium and phosphate levels in our patient before and after the use of OSPS.

to maintain a normal plasma calcium level through immediate mobilization of calcium from the bones and subsequently reduces renal calcium excretion and increases calcium absorption from the intestine. However, in patients with renal failure, the impairment of phosphate excretion by the kidneys will lead to severe hyperphosphataemia from the heavy phosphate load contained in sodium phosphate-based laxatives (oral or rectal).

There had been some case reports on severe hyperphosphataemia and hypocalcaemia following the use of OSPS in patients with a background of renal impairment in recent years. The major adverse clinical effects include cardiac and neurotoxicity of hypocalcaemia and calcium phosphate precipitation. Several risk factors have been identified for electrolyte disturbance after OSPS use [3]. Renal failure is an obvious risk factor. Patients with intestinal obstruction and inflammatory intestinal disorders may have increased gastrointestinal phosphate absorption. This also applies to those with a higher dose of OSPS use. Patients aged >65 years old are also at a higher risk. Elderly patients may have a mild degree of renal impairment despite normal creatinine level. They are also more prone to develop vitamin D deficiency and have diminished intestinal motility. Patients with systemic diseases such as congestive heart failure and liver cirrhosis are also at a higher risk. Patients taking angiotensin-converting enzyme inhibitors, AT2-receptor antagonists or diuretics are also found to have a greater rise in serum phosphate [4].

Although commonly available over-the-counter for relief of constipation, sodium phosphate-based enema preparations may have the potential of causing metabolic derangement if overdosed or retained in the bowels for a prolonged period. Pitcher *et al.* [5] described a case who received 11 sodium phosphate-based enema preparations (~1100 mL) for bowel clearance before revision of his

stenotic colostomy site. The patient developed severe hypocalcaemia of 1.1 mmol/L, hyperphosphataemia of 3.36 mmol/L, which led to marked cardiovascular collapse with metabolic acidosis and ultimately death. Therefore, the risk of use of the apparently safe sodium phosphate-based fleet enema should not be overlooked, especially in those with the risk factors mentioned above.

In our patient, the recent parathyroidectomy had severely impaired the parathyroid hormone response to hyperphosphataemia induced by OSPS use. Our patient has had almost the lowest calcium level that has ever been reported. Niemeijer *et al.* [6] has also reported a similar case of severe hypocalcaemia in a patient with chronic renal disease and asymptomatic hypoparathyroidism.

In view of the potentially life-threatening electrolyte disturbance of OSPS in renal disease patients, we recommend avoidance with substitution by safer alternatives. A possible choice would be PEG-ELS. It appears to have similar efficacy to OSPS as a bowel-cleansing agent. In patients with serum creatinine level <133 mmol/L, it appears to cause no renal toxicity [7]. There is currently no study on the risk of adverse effects in patients using PEG-ELS with renal impairment. There have been several reported cases of disruption of sodium balance leading to convulsion and even death, but the number is much less than the reported electrolyte disturbance caused by OSPS [1]. For patients with risk of fluid overload, an alternative with 2L of PEG-ELS could be used with acceptable bowel cleansing efficacy [8].

In summary, OSPS contains phosphate which may be absorbed systemically. In patients with impaired renal function, there is impairment in phosphate excretion that may lead to hyperphosphataemia and subsequent hypocalcaemia. The hypocalcaemia is even more severe in cases where there is absence of the counteraction by parathyroid hormones. We

recommend alternative bowel-cleansing agents such as PEG-ELS instead of OSPS.

Conflict of interest statement. None declared.

References

1. Belsey J, Epstein O, Heresbach D. Systematic review: adverse event reports for oral sodium phosphate and polyethylene glycol. *Aliment Pharmacol Ther* 2009; 29: 15–28
2. Patel V, Emmett M, Santa Ana CA *et al.* Pathogenesis of nephrocalcinosis after sodium phosphate catharsis to prepare for colonoscopy: Intestinal phosphate absorption and its effect on urine mineral and electrolyte excretion. *Hum Pathol* 2007; 38: 193–194; author reply 4–5
3. Azzam I, Kovalev Y, Storch S *et al.* Life threatening hyperphosphataemia after administration of sodium phosphate in preparation for colonoscopy. *Postgrad Med J* 2004; 80: 487–488
4. Ainley EJ. Hyperphosphataemia after bowel preparation with oral sodium phosphate. *Endoscopy* 2006; 38: 759
5. Pitcher DE, Ford RS, Nelson MT *et al.* Fatal hypocalcemic, hyperphosphatemic, metabolic acidosis following sequential sodium phosphate-based enema administration. *Gastrointest Endosc* 1997; 46: 266–268
6. Niemeijer ND, Rijk MC, van Guldener C. Symptomatic hypocalcemia after sodium phosphate preparation in an adult with asymptomatic hypoparathyroidism. *Eur J Gastroenterol Hepatol* 2008; 20: 356–358
7. Singal AK, Rosman AS, Post JB *et al.* The renal safety of bowel preparations for colonoscopy: a comparative study of oral sodium phosphate solution and polyethylene glycol. *Aliment Pharmacol Ther* 2008; 27: 41–47
8. Poon CM, Lee DW, Mak SK *et al.* Two liters of polyethylene glycol-electrolyte lavage solution versus sodium phosphate as bowel cleansing regimen for colonoscopy: a prospective randomized controlled trial. *Endoscopy* 2002; 34: 560–563

Received for publication: 30.3.11; Accepted in revised form: 9.5.11