# Severe Complication of Iatrogenic Sodium Phosphate Enema Overdose: Acute Kidney Injury

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### **ABSTRACT**

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Sodium phosphate-based enema preparations are low-volume, hyperosmotic agents commonly used in bowel preparation for sigmoidoscopy/colonoscopy and surgical operation. Although generally safe and well tolerated in adults, severe metabolic complications have been reported with use. The patient in this case report was admitted to the general surgery department with complaints of nausea, vomiting, and abdominal pain after oral sodium phosphate solution. After clinical evaluation, hemodialysis was started for acute kidney injury (AKI). Clinicians should be alert using sodium phosphate solution for bowel cleansing before sigmoidoscopy/colonoscopy and surgical operation, in terms of acute kidney damage and electrolyte imbalance.

Keywords: Sodium phosphate enema, colonoscopy, acute kidney injury

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### INTRODUCTION

Sodium phosphate-based solution preparations are low-volume, hyperosmotic agents commonly used in bowel preparation for flexible sigmoidoscopy and before a large bowel surgical operation.¹ Although it is generally reliable and well tolerated in patients, severe metabolic complications and deaths have been reported in all age groups.²-5 Acute kidney injury (AKI) caused by oral phosphate solutions may be early symptomatic or late insidious.6

The clinical symptoms occur hours after bowel cleansing, usually as changes in central nervous or cardiovascular systems. Patients are usually admitted for hyperphosphatemia and hypocalcemia and need urgent fluid resuscitation. Some patients may then show a good improvement in renal functions, others may develop chronic renal disease. The latter group may sometimes need dialysis. The main difference between

these 2 patient groups is that the a much higher dose of sodium phosphate-based enema in the early symptomatic AKI group, and mortality is also possible shortly after the onset of symptoms. The symptoms are mostly related to hypocalcemia, and if treatment is managed quickly and aggressively, improvement of renal function is possible.<sup>7,8</sup>

The other pattern of AKI is the onset of insidious chronic kidney disease, days or months after bowel cleansing with enema. Serum phosphorus and calcium levels are generally at almost normal ranges at the time of diagnosis. The opportunity to remove renal calcium phosphate deposits is often lost, and thus chronic renal disease develops in some patients.<sup>9,10</sup>

This case report describes AKI as demonstrated by renal biopsy following use of iatrogenic oral sodium phosphate solution.

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### **CASE PRESENTATION**

A 58-year-old female patient who was followed up with hypertension was admitted to the general surgery department with complaints of nausea, vomiting, and abdominal pain. The patient had taken ACEi as antihypertensive and did not use any other drugs. She had been prepared for colonoscopy 1 week earlier with oral Fleet phospho-soda. Abdominal x-ray revealed bowel gas in the colon. There was no pathology found in the upper and lower endoscopy performed.

Nausea and vomiting increased 2 days after the procedure. There was also muscle weakness on admission to the emergency department. During physical examination, no pathology was detected. No eyeball deviation, ptosis, focal sign of weakness or neurological sign of seizure were noted. In addition, the patient had a normal urine output that was not decreased in amount of urine and not tea-colored. The electrocardiogram (EKG) showed sinus rhythm and her fever was: 36.5°C, pulse: 80 beats/minute, respiratory rate: 24/min, and blood pressure: 130/90 mmHg. No pathology was detected on physical examination. Bilateral kidney size, echo, and parenchymal thickness were found to be normal in urinary ultrasound.

Laboratory studies revealed the blood parameters as follows: blood urea nitrogen: 62 mg/dL, creatinine: 8.3 mg/dL, sodium: 137 mmol/L, potassium: 3.9 mmol/L, total bilirubin: 1.1 mg/dL, direct bilirubin: 1.0 mg/dL, total protein: 6.1 g/dL, albumin: 3.4 g/dL, alkaline phosphatase: 99  $\mu$ /L, lactate dehydrogenase: 327  $\mu$ /L, aspartate aminotransferase: 42  $\mu$ /L, alanine aminotransferase: 45  $\mu$ /L, gamma glutamyl transferase: 56  $\mu$ /L, C-reactive protein: 17 mg/L, calcium: 10.1 mg/dL, phosphorus:7.6 mg/dL, parathormone: 276 pg/dL, hemoglobin: 9.5 g/dL, platelets: 165 000  $\mu$ /L, and white blood cells: 11  $\times$  10°  $\times$ 10° L. The urine tests revealed: 8 erythrocytes per high power field. Proteinuria was found 0.4 gr/day. Urine culture was negative. The parameters for arterial blood gases were: pH:7.24, HCO<sub>3</sub>: 18 mmol/L, PCO<sub>2</sub>:38 mmHg, and PO<sub>2</sub>: 90 mmHg (Table1).

After clinical and laboratory examinations, hemodialysis was started for AKI. During the follow-up of the patient, 3 sessions of hemodialysis were performed, and kidney biopsy was performed for a definitive diagnosis. The renal biopsy of the patient, whose renal functions returned to normal after dialysis, reported acute phosphate nephropathy (Figure 1). The patient, whose kidney functions returned to normal during the follow-up, was discharged.

# **DISCUSSION**

Phosphorus is critical for bone mineralization, cellular structure, and energy metabolism. Phosphate homeostasis is regulated by both the intracellular action of phosphorus and the excretion of the substance by the kidneys. 11 Sodium phosphate-based enemas are generally reliable and well tolerated when used as recommended. However, serious metabolic disorders may occur when used in patients with abnormal

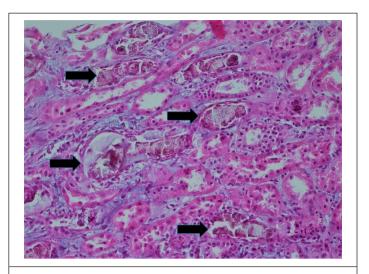


Figure 1. Calcium phosphate crystals appear in the renal tubular lumens.

kidney function or extended intraluminal absorption of the enema.  $^{\mbox{\tiny 3-5,12}}$ 

Phosphate-based enemas are widely used in bowel preparation for flexible sigmoidoscopy/colonoscopy and prior to the large bowel surgical operation. Although enema administration is generally reliable and well tolerated in all age groups, severe metabolic complications and deaths have been shown in infants and young children.<sup>5</sup> In addition, acute phosphate nephropathy has also been found in adults after oral sodium phosphate bowel cleansing.<sup>13</sup>

Martin et al.<sup>2</sup> investigated the effects of sodium phosphate-based enema solution in a swine model and found that a dose of 20-30 mL/kg was fatal in pigs. It has been also shown that hypocalcemia, hyperphosphatemia, hypernatremia, and high anion deficit metabolic acidosis can occur due to the usage of an enema.

Hyperphosphatemia after rectal administration of phosphosoda has been previously described in patients. In addition, metabolic acidosis, decreased serum calcium level, and increased serum phosphate level have also been described in adults with phosphate enema overdose. <sup>14</sup> In addition, hyperphosphatemic hypocalcemic coma caused by enema has also been identified. <sup>15</sup>

These preparations are also frequently used for constipation and are relatively safe and effective for bowel cleansing, except in patients with renal dysfunction, bowel obstruction, small bowel disorders, and poor bowel motility; and in the geriatric group. 16-18

In addition to severe hyperphosphatemia and calcium phosphate accumulation in the renal tubular lumens, AKI and mortality were also observed after using oral sodium phosphate

Table 1. Biological Parameters of Patient Before and After Hemodialysis

Tiemodiatysis	Values	
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Laboratory Markers	Before	After
Blood urea nitrogen (mg/dL)	62	30
Creatinine (mg/dL)	8.3	1.8
Sodium (mmnol/L)	137	135
Potassium (mmol/L)	3.9	3.5
Calcium (mg/dL)	10.1	8.9
Phosphorus (mg/dL)	7.6	4.3
Parathormone (pg/mL)		276
Total protein (g/dL)	5.3	6.5
Albumin (g/dL)	3.2	3.8
Alkaline phosphatase (μ/L)	99	95
Lactate dehydrogenase (μ/L)	327	233
Aspartate aminotransferase (μ/L)	42	44
Alanine aminotransferase (μ/L)	45	40
Gamma glutamyl transferase (μ/L)	56	55
C-reactive protein (mg/L)	13	7
Hemoglobin (g/L)	9.5	11
Platelets (μ/L)	165 000	150 000
White blood cells (/L)	11×10 <sup>9</sup>	8 × 10 <sup>9</sup>

solutions, in the study involving 11 elderly patients. It was concluded that sodium phosphate enemas, even at standard doses, can lead to severe metabolic disorders associated with high mortality and morbidity.19

## CONCLUSION

Clinicians should be alert when using sodium phosphate enemas and oral solutions for bowel cleansing before sigmoidoscopy/colonoscopy and surgical operation, in terms of acute kidney damage and electrolyte imbalance.

**Informed Consent:** Written informed consent was obtained from the patient who participated in this case.

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**Conflict of Interest:** The authors have no conflicts of interest to declare.

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### **REFERENCES**

- 1. Tooson JD, Gates Jr LK. Bowel preparation before colonoscopy. Choosing the best lavage regimen. Postgrad Med. 1996;100(2):203-4, 207, [CrossRef]
- 2. Martin RR, Lisehora GR, Braxton M, Barcia PJ. Fatal poisoning from sodium phosphate enema. Case report and experimental study. JAMA. 1987;257(16):2190-2192. [CrossRef]
- 3. Oxnard SC, O'Bell J, Grupe WE. Severe tetany in an azotemic child related to a sodium phosphate enema. Pediatrics. 1974;53(1):105-106.
- 4. Davis RF, Eichner JM, Bleyer WA, Okamoto G. Hypocalcemia, hyperphosphatemia and dehydration following a single hypertonic phosphate enema. J Pediatr. 1977;90(3):484-485. [CrossRef]
- Reedy JC, Zwiren GT. Enema-induced hypocalcemia and hyperphosphatemia leading to cardiac arrest during induction of 245 anesthesia in an outpatient surgery center. Anesthesiology. 1983;59(6):578-579. [CrossRef].
- Gonlusen G, Akgun H, Ertan A, Olivero J, Truong LD. Renal failure and nephrocalcinosis associated with oral sodium phosphate bowel cleansing: clinical patterns and renal biopsy findings. Arch Pathol Lab Med. 2006;130(1):101-106. [CrossRef]
- 7. Hookey LC, Depew WT, Vanner S. The safety profile of oral sodium phosphate for colonic cleansing before colonoscopy in adults. Gastrointest Endosc. 2002;56(6):895-902. [CrossRef]
- Tan HL, Liew QY, Loo S, Hawkins R. Severe hyperphosphataemia and associated electrolyte and metabolic derangement following the administration of sodium phosphate for bowel preparation. Anaesthesia. 2002;57(5):478-483. [CrossRef]
- Markowitz GS, Nasr SH, Klein P, et al. Renal failure due to acute nephrocalcinosis following oral sodium phosphate bowel cleansing. Hum Pathol. 2004;35(6):675-684. [CrossRef]
- 10. Markowitz GS, Whelan J, D'Agati VD. Renal failure following bowel cleansing with a sodium phosphate purgative. Nephrol Dial Transplant. 2005;20(4):850-851. [CrossRef]
- 11. Gray RW, Wilz DR, Caldas AE, Lemann J Jr. The importance of phosphate in regulating plasma 1,25-(OH)2-vitamin D levels in humans: studies in healthy subjects in calcium-stone formers and in patients with primary hyperparathyroidism. J Clin Endocrinol Metab. 1977;45(2):299-306. [CrossRef]
- 12. Biberstein M, Parker BA. Enema-induced hyperphosphatemia. Am J Med. 1985;79(5):645-646. [CrossRef]
- 13. Markowitz GS, Stokes MB, Radhakrishnan J, D'Agati VD. Acute phosphate nephropathy following oral sodium phosphate bowel purgative: an underrecognized cause of chronic renal failure. J Am Soc Nephrol. 2005;16(11):3389-3396. [CrossRef]
- 14. Pitcher DE, Ford RS, Nelson MT, Dickinson WE. Fatal hypocalcemic, hyperphosphatemic, metabolic acidosis following sequential sodium phosphate-based enema administration. Gastrointest Endosc. 1997;46(3):266-268. [CrossRef]
- 15. Knobel B, Petchenko P. Hyperphosphatemic hypocalcemic coma caused by hypertonic sodium phosphate(Fleet)enema intoxication. J Clin Gastroenterol. 1996;23(3):217-219. [CrossRef]
- 16. Fine A, Patterson J. Severe hyperphosphatemia following phosphate administration for bowel preparation in patients with renal

- failure: two cases and a review of the literature. *Am J Kidney Dis*. 1997;29(1):103-105. [CrossRef]
- 17. Fass R, Do S, Hixson LJ. Fatal hyperphosphatemia following Fleet Phospo-Soda in a patient with colonic ileus. *Am J Gastroenterol*. 1993;88(6):929-932.
- 18. Vukasin P, Weston LA, Beart RW. Oral Fleet Phospho-Soda laxative-induced hyperphosphatemia and hypocalcemic tetany in an
- adult:report of a case. *Dis Colon Rectum*. 1997;40(4):497-499. [CrossRef]
- 19. Ori Y, Rozen-Zvi B, Chagnac A, et al. Fatalities and severe metabolic disorders associated with the use of sodium phosphate enemas: a single center's experience. *Arch Intern Med*. 2012;172(3):263-265. [CrossRef]