



CASE REPORT

A brief grief over bowel relief [v1; ref status: indexed, <http://f1000r.es/wq>]

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Abstract

Oral sodium phosphate (OSP) solution is commonly used as bowel purgative before colonoscopy. Its safety has recently been questioned with several reports of acute renal failure and chronic kidney disease following its use. All of the cases reported are following bowel preparation for colonoscopy. I present a case of acute renal failure following OSP solution given to relieve constipation. This report further highlights the dangers of OSP and the importance of caution and careful monitoring when OSP solution is used as a cathartic, or for bowel preparation before colonoscopy.

Article Status Summary

Referee Responses

| Referees | 1 | 2 |
|--------------------------------|------------|------------|
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1 Christina Yuan, Walter Reed Army Medical Center USA

2 Luan D. Trong, Houston Methodist Research Institute USA

Latest Comments

No Comments Yet

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Case report

A 72-year-old woman with essentially unremarkable past medical history fell and sustained back injury and was noted to have a T₁₁ compression fracture without any neurovascular compromise. The patient received Tylenol#3 for pain relief and was sent home. A few days later, she returned with ongoing vague lower back and abdominal discomfort and was noted to be constipated. Tylenol#3 was stopped and she was given a laxative - oral sodium phosphate solution (OSP, 45 ml, *Pharmascience, Montreal, Canada*) to treat constipation.

Three days later, she returned to the local emergency department with feeling of generalized weakness, numbness around her lips, ongoing vague abdominal discomfort and nausea, but denied vomiting or diarrhea. Her intake had been poor since the fall and she noted decreased urine output. There is no history of diabetes or hypertension. Her medication was rabeprazole 20 mg a day and acetaminophen as required.

Investigations at the local emergency department revealed low hemoglobin of 109 g/L, normal white blood cell count WBC of 4.5, elevated blood urea nitrogen BUN of 9.4 mmol/L with serum creatinine of 345 µmol/L, and serum potassium of 3.4 mmol/L. The old records showed that her BUN was 6.1 mmol/L with serum creatinine of 74 µmol/L in December 2007. She was transferred for further management of acute renal failure.

Physical examination was remarkable for a woman of stated age with mild decreased skin turgor, blood pressure of 106/60 mmHg without orthostatic changes and regular rate of 72 beats per minute. Lungs were clear and heart sounds were normal. Abdominal examination revealed a soft abdomen with mild diffuse tenderness without rebound. There were no masses, renal angle fullness or tenderness. There was mild tenderness in the lower thoracic area. There was no pedal edema and neurological examination was non-focal. She had a Foley catheter with small amount of concentrated urine in the bag.

Investigations in our emergency department revealed low hemoglobin of 112 g/L, normal WBC of 4.9, elevated BUN of 9.4 mmol/L with serum creatinine of 419 µmol/L, serum potassium of 3.4 mmol/L, low serum calcium of 1.85 mmol/L (2.02–2.60 mmol/L) with serum albumin of 36 g/L, low ionized calcium of 0.85 mmol/L (1.15–1.29 mmol/L) and

elevated phosphate of 3.68 mmol/L (0.87–1.45 mmol/L) and creatine kinase [CK] of 349. A urinalysis showed a concentrated urine with specific gravity of >1.030, 1+ protein and trace of blood with few white and red blood cells with few hyaline casts. Random urine sodium was 64 mmol/L with urine creatinine of 5330 µmol/L. A urine culture was negative. An abdominal ultrasound showed normal size kidneys without obstruction. The hospital course is shown in [Table 1](#).

This patient presented with acute kidney injury (AKI) and the differential diagnosis included ischemic acute tubular necrosis (poor intake, decreased skin turgor, FE_{Na} of 3.78%), rhabdomyolysis (history of fall, elevated phosphate). The likelihood of vasculitic process was low in view of bland urine sediment and negative antinuclear and anti-neutrophilic cytoplasmic antibodies. However, significant hyperphosphatemia and hypocalcemia within 72-hours of standard dose (45 ml) of OSP [21.6 gm of monobasic sodium phosphate monohydrate and 8.1 gm of dibasic sodium phosphate heptahydrate] suggests the high probability of acute phosphate nephropathy (APN) that results from deposition of calcium-phosphate crystals in renal tubules and parenchyma (nephrocalcinosis)². A kidney biopsy confirmed findings of acute phosphate nephropathy with acute tubular necrosis ([Figure 1](#)). She required supportive dialysis treatment twice.

Discussion

OSP solution is commonly used as a bowel purgative before colonoscopy³. Its safety has recently been questioned^{1–5} with several reports of AKI and chronic kidney disease following its use. All of the cases reported occurred after bowel preparation with OSP for colonoscopy but AKI in this woman occurred after its use to relieve constipation. Decreased kidney function, use of renin angiotension aldosterone system (RAAS) blockers and older age and female gender are the most probable risk factors for APN, but other contributing factors are - use of non-steroidal anti-inflammatory agents, diuretics, history of hypertension, diabetes or heart failure^{3–5}. Women, because of their smaller body mass, are more sensitive to fluid loss. Adequate fluid intake⁴ is important to prevent AKI when OSP is used as a bowel purgative. Poor oral intake since the fall in this woman likely contributed to both ATN and APN after OSP use. Although most patients recover renal function, some may have persistent chronic kidney disease⁴.

Table 1. Showing the hospital course of the patient.

| | Normal range | 9-months before | Day 1 | Day 2 | Day 5 | Hemodialysis × 2 | Day 10 | Day 21 |
|------------------|------------------|-----------------|-------|-------|-------|------------------|--------|--------|
| BUN | 2.6–7.7 mmol/L | 6.1 | 9.9 | 11.3 | | | 6.4 | |
| Serum creatinine | 35–97 µmol/L | 74 | 419 | 486 | 675 | | 200 | 98 |
| Serum potassium | 3.6–5.0 mmol/L | | 3.6 | | | | | 3.8 |
| Serum calcium | 2.02–2.60 mmol/L | | 1.85 | 1.66 | 1.92 | | 2.20 | 2.24 |
| Ionized calcium | 1.15–1.29 mmol/L | | 0.85 | 0.94 | | | 1.07 | 1.16 |
| Serum albumin | 38–46 g/L | | 33 | | | | | 38 |
| Serum Phosphate | 0.87–1.45 mmol/L | | 3.68 | 3.59 | 3.39 | | 1.12 | 1.18 |

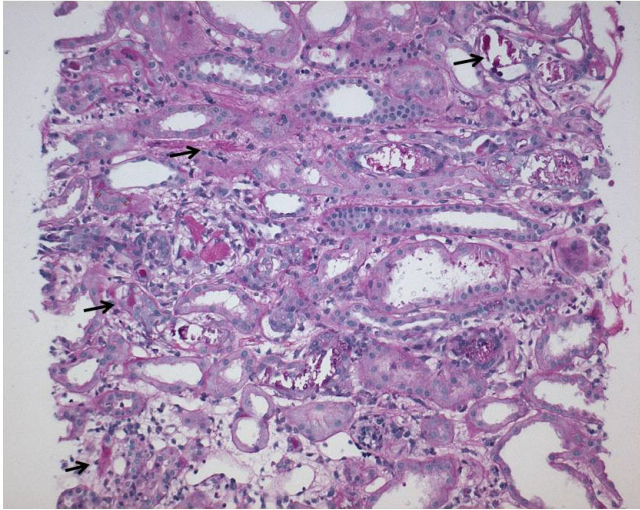


Figure 1. Kidney biopsy – showing numerous tubules with calcification (black arrows) (PAS stain, magnification x100) [Courtesy: Andrew Herzenberg (deceased) and John Rohan, University Health Network, Toronto, Ontario, Canada].

This report further highlights the need for vigilance when using OSP solutions for bowel preparation or to relieve constipation. Alternative solutions should be considered, especially in elderly and high-risk individuals⁵.

Key Points

1. Consider acute phosphate nephropathy in the presence of acute renal dysfunction, hyperphosphatemia and hypocalcemia.
2. Consider alternate agents to oral phosphate solutions for bowel preparation or for relief of constipation, especially in elderly patients and in patients with chronic kidney disease.
3. Ensure adequate hydration, if and when these agents (OSP) are used.

Author contributions

MSP was involved in the care of this patient and obtained consent from patient for publication. KSP did literature search and prepared the initial draft and both authors approved the final draft.

Competing interests

No competing interests were disclosed.

Grant information

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[PubMed Abstract](#) | [Publisher Full Text](#)
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[PubMed Abstract](#) | [Publisher Full Text](#)

Current Referee Status:

Referee Responses for Version 1



Luan D. Trong

Houston Methodist Research Institute, Houston, TX, USA

Approved: 05 November 2013

Referee Report: 05 November 2013

In this manuscript entitled "A Brief Grief over Bowel Relief," by K.S. Parmar and M.S. Parmar, the authors describe a patient with acute renal failure after oral sodium phosphate administration for constipation.

As the authors mentioned, this type of renal failure is almost always secondary to administration of sodium phosphate for colonoscopy. This report expands the possible complications of oral sodium phosphate in a new clinical context, i.e. treatment for constipation. It is an important finding both clinically and pathologically.

A few minor suggestions:

1. The quality of the renal biopsy illustration can be improved. The picture should be sharper with better contrast and the areas of tubular calcification should be better illustrated.
2. In the Discussion, the dose as well as the drug schedule of oral sodium phosphate for colonoscopy should be reviewed and compared with those in this case. This should highlight better the clinical context in which oral sodium phosphate as a treatment for constipation can cause acute renal failure.

I have read this submission. I believe that I have an appropriate level of expertise to confirm that it is of an acceptable scientific standard.

Competing Interests: No competing interests were disclosed.



Christina Yuan

Walter Reed Army Medical Center, Washington, DC, USA

Approved: 18 March 2013

Referee Report: 18 March 2013

This is a case report of acute kidney injury (requiring dialysis) after use of OSP for constipation in an elderly woman. The patient had a kidney biopsy demonstrating findings consistent with acute phosphate nephropathy. This is a valuable reminder that AKI secondary to OSP may occur independently of the indication for OSP prescription.

I have read this submission. I believe that I have an appropriate level of expertise to confirm that it is of an acceptable scientific standard.

Competing Interests: No competing interests were disclosed.
