



CASE REPORT

A brief grief over bowel relief [version 1; peer review: 2 approved]

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v1 **First published:** 29 Jan 2013, 2:26 (<https://doi.org/10.12688/f1000research.2-26.v1>)
Latest published: 29 Jan 2013, 2:26 (<https://doi.org/10.12688/f1000research.2-26.v1>)

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.

Open Peer Review

Reviewer Status

	Invited Reviewers	
	1	2
version 1 published 29 Jan 2013	 report	 report

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- 2 **Luan D. Trong**, Houston Methodist Research Institute, Houston, TX, USA

Any reports and responses or comments on the article can be found at the end of the article.

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Competing interests: No competing interests were disclosed.

Grant information: The author(s) declared that no grants were involved in supporting this work.

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How to cite this article: Parmar KS and Parmar MS. **A brief grief over bowel relief [version 1; peer review: 2 approved]** F1000Research 2013, 2:26 (<https://doi.org/10.12688/f1000research.2-26.v1>)

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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 questioned¹⁻⁵ 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 failure³⁻⁵. 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

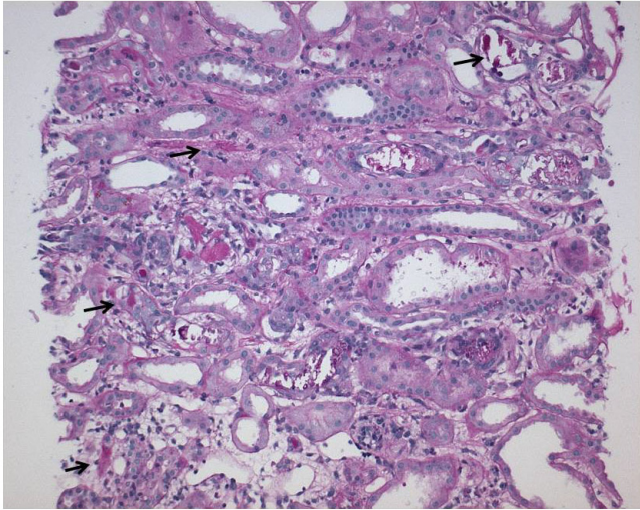


Figure 1. Kidney biopsy – showing numerous tubules with calcification (black arrows) (PAS stain, magnification x100) [Courtesy: Andrew Herzenberg (deceased) and John Rohan, University Health Network, Toronto, Ontario, Canada].

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

The author(s) declared that no grants were involved in supporting this work.

Acknowledgements

I wish to thank Andrew Herzenberg (deceased) and John Rohan of the Department of Pathology, University Health Network, University of Toronto, Ontario, Canada, for providing the photomicrograph of the patient's kidney biopsy.

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Reviewer Report 05 November 2013

<https://doi.org/10.5256/f1000research.1178.r2323>

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Luan D. Trong

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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.

Competing Interests: No competing interests were disclosed.

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.

Reviewer Report 18 March 2013

<https://doi.org/10.5256/f1000research.1178.r844>

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**Christina Yuan**

Walter Reed Army Medical Center, Washington, DC, USA

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.

Competing Interests: No competing interests were disclosed.

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