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CASE REPORT

Findings of Biopsy-Proven Chronicity and End-Stage Renal Failure Associated with Oral Sodium Phosphate Solution

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Abstract

Bowel purgatives containing oral sodium phosphate (OSP) solution are used frequently in general practice and they have the potential of causing acute kidney injury especially in patients with some identified risk factors. Kidney injury may lead to chronicity and end-stage renal disease. Here we present, with renal biopsy findings, an elderly patient suffering from end-stage renal failure due to OSP solution.

Keywords: sodium phosphate solution, renal failure, acute phosphate nephropathy, colonoscopy, hemodialysis

INTRODUCTION

Acute phosphate nephropathy (APN) is a kind of kidney injury that generally occurs after the use of hyperosmotic bowel purgatives containing oral sodium phosphate (OSP) solution. This entity has been referred after multiple cases of renal biopsy-proven acute kidney injury occurred following the use of OSP. It is usually detected days to months following OSP administration.

The incidence of APN remains unknown. There are some identified risk factors for a patient to experience APN. Acute kidney injury associated with OSP can lead to chronic kidney disease especially in the elderly. Here we describe an elderly patient experiencing APN with renal biopsy findings.

CASE REPORT

A 65-year-old man had hypertension and hyperlipidemia and was using an angiotensin receptor blocker (ARB) drug. He underwent colonoscopy to reveal the cause of iron deficiency anemia. The following laboratory values were recorded—urea: 32 mg/dL, creatinine: 1.14 mg/dL, sodium: 142 mmol/L, potassium: 4.39 mmol/L, calcium: 9.44 mg/dL, phosphate: 3.03 mg/dL, uric acid: 5.9 mg/dL; Hb: 15.2 g/L, and WBC: 4900/mm^3. A total of 90 mL of OSP solution was used for bowel preparation. One month after the procedure, he had complaints of nausea and metallic sense of tongue. Renal function tests were elevated as follows—urea: 59 mg/dL, creatinine: 1.82 mg/dL, although postprocedure first-day levels were normal. There was no history of postprocedure blood or water loss or any other way of prerenal failure to cause kidney injury. There was no finding of dehydration. He did not use any nonsteroid anti-inflammatory (NSAI) drug. There was no finding of any infection, rhabdomyolysis, or vasculitic procedure. Postrenal obstruction was excluded with abdominal ultrasonography. Urine sediment revealed granular cylinders but no erythrocyte or leukocyte cylinders. The condition was thought to be caused by hyperphosphatemia due to OSP. Patient was hydrated orally and intravenously. As the renal function tests did not improve, renal biopsy was performed. There was mild focal tubulointerstitial inflammation with mononuclear cells, tubular atrophy and two sclerosed glomerules, of a total glomerule number of 18 on the biopsy specimen. Interlobar artery walls had mild to moderate increase of fibrointimal thickness. He

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came for monthly follow-up kidney function tests. By the fourth month of the follow-up, urea was 52 mg/dL, creatinine was 3.14 mg/dL, and the patient started his hemodialysis treatment.

DISCUSSION

OSP draws water into the gastrointestinal tract. Smaller required volumes, good patient compliance, and good intestinal cleaning makes the agent used frequently. But it has the potential for acute kidney injury called APN.[1] The incidence of APN remains unknown and it can occur even with normal renal function.

The mechanism underlying APN appears to be consisting of volume depletion and increase in serum phosphate levels both of which may occur in a patient given OSP and involves the precipitation of calcium phosphate crystals in the distal tubule. There are some reports about high serum phosphate levels after bowel preparation possibly causing lethal damage to heart (arrhythmias) and kidneys (renal failure).[2] Our patient was normophosphatemic before the colonic cleaning procedure with OSP. There was no finding of dehydration, use of NSAI, vasculitic procedure, nor postrenal obstruction to explain the renal failure which was recognized 1 month after OSP usage.

There are some identified risk factors for a patient to experience APN after the application of OSP. Chronic renal disease, advanced age, female gender, usage of drugs like ACE (angiotensin converting enzyme) inhibitors and ARB, diuretics, NSAI, lithium; comorbid conditions like diabetes mellitus or hypertension and higher doses of phosphate solution are associated with higher APN incidences. Moreover, especially in the elderly,[3] acute kidney injury associated with OSP can lead to chronic kidney disease (including end-stage renal disease) and some reports discourage the usage of the preparation.[4] Our patient was using an ARB for hypertension and this may have worsened the nephropathy induced by phosphate. ACE inhibitors and ARBs worsen the prerenal state caused by OSP-induced volume depletion by promoting the loss of angiotensin II-mediated efferent arteriolar constriction, which reduces glomerular filtration.[5] In addition, they decrease angiotensin II-dependent proximal tubule bicarbonate reabsorption, which leads to an increase in distal tubular bicarbonate concentration. The increase in intraluminal pH promotes precipitation of calcium phosphate crystals.

In previously published cases in the literature, renal biopsy was performed to describe the histologic findings.[5,6] Renal biopsy reveals an acute and chronic tubulointerstitial nephropathy with the distinctive finding of abundant tubular and interstitial deposits of calcium phosphate. In biopsies performed within 3 weeks of OSP exposure, findings of acute tubular injury predominate and resemble changes seen in acute tubular necrosis. Later biopsies exhibit findings of chronicity, including tubular atrophy and interstitial fibrosis.[7] Renal biopsy of our patient showed mild focal tubulointerstitial inflammation with mononuclear cells, tubular atrophy, and two sclerosed glomerules of the total 18 glomerules. Interlobar artery walls had mild to moderate increase of fibrointimal thickness.

Acute kidney injury generally occurs within hours of the administration of OSP.[8] This presentation mainly involves excessive dosing of OSP or other risk factors for hyperphosphatemia. Acute kidney injury occurring in the setting of severe hyperphosphatemia and hypocalcemia leads to tetany, cardiac arrest, and in some cases death. Patients surviving the immediate event typically returns to normal or near-normal renal function, but if not, they usually suffer end-stage renal failure.

CONCLUSION

Oral purgative solutions containing sodium phosphate are used frequently in general practice. They may lead to acute renal injury especially in patients carrying mentioned risk factors and even in normal subjects. In some cases, as in our patient, phosphate may lead to findings of chronicity like sclerosis or tubular atrophy in renal biopsy specimens. We want to emphasize that a careful review of all contraindications to the use of OSP should be done prior to the selection of a bowel purgative for an individual patient.

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