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Case report

Recurrent Cathartic Use and Acute Kidney Injury

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Abstract

Introduction. Oral sodium phosphate containing (OSP) cathartics are used for bowel cleansing in medical practice. They are generally accepted as safe. However, these agents may cause renal injury in some susceptible patients.
Case report. We present 2 patients suffering from acute kidney injury that could have been prevented easily. The first patient had undergone colonoscopy for chronic diarrhea. The second patient had undergone colonoscopy for iron deficiency anemia and colonic polyps. Both patients presented with nausea and vomiting. Fortunately, they recovered with hemodialysis and supportive measures.
Conclusions. Phosphate containing cathartics have the potential to cause prerenal azotemia or acute phosphate nephropathy. However as rarely seen, clinicians frequently neglect this entity. Even so OSP cathartics must be used cautiously in susceptible individuals. Preventive strategies should be implemented in all of these patients instead of management after renal injury occurs.

Keywords: cathartic, colonoscopy, nephropathy, phosphate

Introduction

Various drugs may cause acute kidney injury (AKI). Oral sodium phosphate (OSP) containing cathartics used for bowel cleansing are among these drugs. In addition to the risk of acute prerenal azotemia, transient hyperphosphatemia, volume depletion exacerbated by concurrent renin-angiotensin system blockers and diuretics, and elevated distal tubular phosphate and calcium concentrations may contribute to the renal injury named as acute phosphate nephropathy (APN) [1,2]. Patients with chronic kidney disease are more susceptible for APN, especially if they are using medications like angiotensin-converting enzyme inhibitors, angiotensin-receptor blockers, diuretics, or non-steroidal anti-inflammatory drugs [3]. Some simple measurements can be implemented in order to prevent AKI in these high-risk patients. Proper hydration, stopping risky medications and using safer alternatives to phosphorous-containing formulations are useful. Herein we present two of our recently detected AKI cases in order to increase the awareness about this issue.

Case 1

A 59 year old male admitted to our clinic with nausea and vomiting that started 2 days after a colonoscopy procedure. On physical examination, skin and mucous membranes were dry. No abdominal tenderness was present. Arterial blood pressure was 110/60 mm/Hg. Pulse was 96/min.; Some laboratory parameters were as; urea: 88 mg/dl, creatinine: 3,6 mg/dl, Na: 142 mmol/L, K: 3,1 mmol/L, Ca: 9,8 mg/dl and P: 12,5mg/dl. He was hydrated intravenously for suspected prerenal AKI. But as he was oliguric despite hydration for more than 24 hours, hemodialysis was performed via a jugular catheter. His urine output began to increase at the 3rd day after two dialysis sessions. Hemodynamic parameters and electrolyte levels were followed up closely and corrected rapidly. After 7 days, he was cured completely. Serum phosphate and creatinine levels decreased (Figure 1). Renal biopsy was not performed as healing occurred.
On history, the patient was learnt to be on clinical follow-up for acromegaly after hypophyseal adenectomy 23 years ago. He had also hypertension which was regulated with valsartan and thiazide combination. He had non-bloody, loose diarrhea for a nearly 4 weeks duration. He was scheduled for colonoscopy in part of investigation for his chronic diarrhea. On laboratory before bowel preparation; some parameters were as; urea: 20 mg/dl, creatinine: 0,6 mg/dl, Na: 142 mmol/L, K: 3,1 mmol/L and Ca:10,1 mg/dl and P: 2,6. He was administered a solution including 63.8 g monobasic sodium phosphate monohydrate and 24.3 g dibasic sodium phosphate heptahydrate for bowel preparation. Bowel was accepted as inadequately cleaned after the first cleaning attempt. So he was iven an additinal solution including 900 mg sennosides A and B calcium.?

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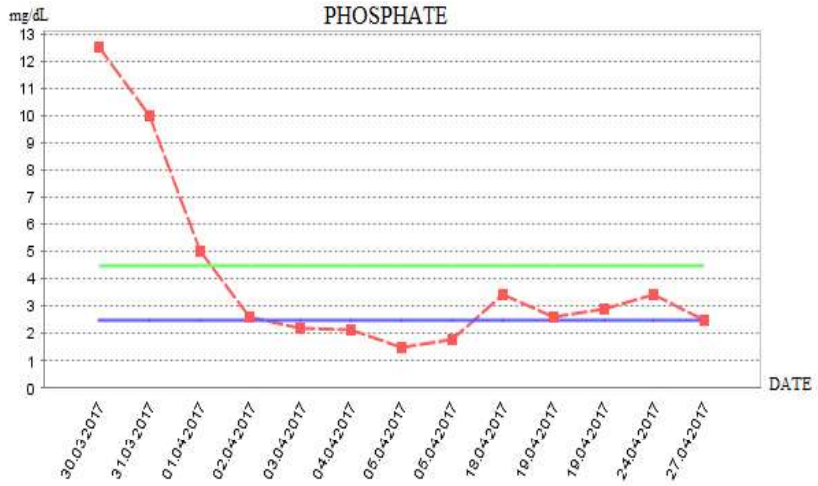

Fig. 1. The serum phosphate and creatinine level graphics of the first patient

Case 2

A 51 year old female consulted to our emergency clinic with nausea, persistent vomiting and fatique one day after a colonoscopy procedure. Arterial blood pressure was 110/60mm/Hg. Pulse was 64/min. On laboratory; some parameters were as; urea: 69 mg/dl, creatinine: 4,4 mg/dl, Na: 140 mmol/L, K: 4,7 mmol/L, Ca: 8,5mg/dl and P: 11,7 mg/dl. She was hydrated intravenously for suspected prerenal AKI but she was oliguric for 12 hours. Hemodialysis was performed via a jugular catheter as she had persistent nausea and vomiting. Her urine output began to increase at the 2nd day after only one dialysis session. Hemodynamic parameters and electrolyte levels were followed up closely and corrected rapidly. After 12 days, she was cured completely. Serum phosphate and creatinine levels decreased. (Figure 2) Renal biopsy was not performed as healing occurred.

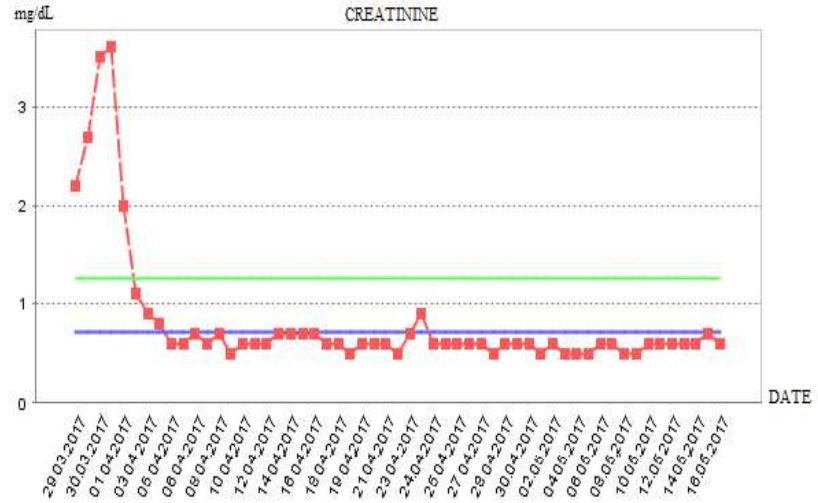

Fig. 2. The serum phosphate and creatinine level graphics of the second patient

On history, this patient was learnt to be under investigation for Cushing's syndrome. She was diagnosed to have bilateral functioning adrenal adenoma. Bilateral adrenalectomy was performed 1 year ago. After the operation, she was on prednisolone 10 mg/day and fludrocortisone 50 mg/day. She was on follow-up for type 2 Diabetes Mellitus and hypertension which was regulated with valsartan/thiazide, amlodipine and nebivolol combination. She was also under investigation for iron-deficiency. Colonoscopy was performed. Colonic polyps were detected. Colonoscopy operator was learnt to fail to finish the procedure because of patient intolerance. Thus biopsy was postponed to another colonoscopic intervention. She was scheduled again for colonoscopy and another preparation with OSP was performed. On laboratory before bowel preparation; some parameters were as; urea: 44 mg/dl, creatinine: 0,5mg/dl, Na: 146 mmol/L, K: 3,2 mmol/L, Ca:

9,1 mg/dl and P: 3,5. She was administered a solution including sodium dihydrogen phosphate and disodium hydrogen phosphate three times for bowel preparation. In addition she was given a solution including 900 mg sennosides A and B calcium. The patient had taken 127.6 g monobasic sodium phosphate monohydrate and 48.6 g dibasic sodium phosphate heptahydrate.

Discussion

Adequate pre-procedural bowel cleansing is essential for colonoscopy. Some oral phosphate containing agents are used effectively for this purpose. Hypovolemia resulting from the preparation procedure may facilitate development of prerenal azotemia especially in patients with chronic kidney disease. Non-steroidal anti-inflammatory drugs (NSAIDs), diuretics and renin-angiotensin system (RAS) blockers that decrease glomerular filtration rate may facilitate renal injury if they are used concurrently with these agents.

In addition to the risk of prerenal azotemia, APN has been reported to occur after exposure to sodium-phosphate (NaP) bowel-cleansing solutions. Intestinal absorption of oral phosphate containing solutions may cause hyperphosphatemia and hypocalcemia [4]. APN is a type of renal injury characterized by tubulointerstitial damage due to deposition of calcium and phosphorus [5]. Clinically, some patients may present with acute kidney injury and very high phosphorus levels. Besides, the renal injury may occur after several weeks or months [6].

The renal injury in APN may recover in some patients but the damage may sometimes become permanent. The most influential marker for prognosis is baseline renal function. Female gender, diabetes mellitus, older age and Caucasian race are other risk factors [2].

The baseline renal filtration function of our first patient was normal. However he was nearly 60 years old and he was taking valsartan/hydrochlorothiazide that may increase the risk of both prerenal azotemia and APN. His medication should be shifted to a safer agent temporarily during bowel-cleansing. A waiting period could be implemented before using the cathartics for the second time in a short time period. Oral hydration should also be motivated. Serum biochemistry tests should also be performed after the first procedure, in order to detect any renal or electrolyte abnormalities.

Our second patient was female. She was diabetic and also on valsartan/hydrochloride treatment. Her baseline renal filtration function was also normal. Her medication was not changed to a safer drug during bowel-cleansing. A waiting period was not implemented before repeating the cathartics for the second time. Oral hydration was not motivated. Serum biochemistry tests were not performed after the first procedure.

They were assessed as euvolemic on physical examination. In addition, despite effective hydration they needed dialysis. So we got away from the preliminary diagnosis of prerenal acute renal injury and acute tubular necrosis due to hypovolemia. Besides all these, they had very high serum phosphate levels on laboratory examination. So we strongly suggested their diagnosis to be APN. Healing occurred. So renal biopsy was not performed as it would be unethical.

Both creatinine and phosphorus improved promptly in the first patient but not in the other patient where creatinine and serum phosphorus remained elevated after 10 days. This may be related to possible different mechanisms of renal injuy acting simultaneously. For example the first patient might have suffered from prerenal injury more, but the second might have suffered from acute phosphate nephropathy more.

Both patients had history of endocrine disease. But their diseases have different impacts on normal human physiology and they were under good control. So a possible coincidence or a tendency to acute renal injury were not suggested to be.

Conclusions

Acute kidney injury due to dehydration or toxic effects of phosphate is a frequently ignored complication of colonoscopy. Clinicians may overlook the diagnosis as a result of variable APN courses. No specific therapy is available, if renal injury occurs once. But prevention is possible with simple measurements. Thus APN must be kept in mind before bowel cleansing procedures.

Conflict of interest statement. None declared.

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