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Case report

Acute phosphate nephropathy due to use of phosphorous enema: Case report

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Acute phosphate nephropathy is a rare but serious type of kidney injury that commonly occurs after the use of bowel purgatives that contain oral sodium phosphate. Bowel purgatives are widely used to prepare patients for colonoscopy, but their use can cause acute or chronic kidney disease. Acute phosphate nephropathy is a type of crystal nephropathy characterized by tubular and interstitial deposition of calcium phosphate. Here we presented a case of acute kidney injury following the use of a sodium phosphate-containing enema who required a renal biopsy for diagnosis.

Key words: acute phosphate nephropathy, acute kidney injury, oral sodium phosphate, crystal nephropathy, colon cleansing.

INTRODUCTION

Acute phosphate nephropathy, or nephrocalcinosis, is a type of tubulointerstitial nephropathy characterized by deposition of calcium phosphate crystals within renal tubules and the interstitium (Lochy et al., 2013). Oral sodium phosphate solutions are widely used for bowel cleansing before colonoscopy, for surgical procedures, and for treatment of severe constipation (Lochy et al., 2013; Weiss and Thop, 2011; Santos et al., 2010). These drugs must be used with caution among the elderly and among patients with renal dysfunction, small intestinal disorders, or poor gut motility.

Acute phosphate nephropathy is a rare but serious condition which can cause acute kidney injury and progress to chronic kidney disease (CKD) (Weiss and Thop, 2011). Here, we presented a case of acute kidney injury following the use of sodium phosphate-containing enema.

Case

Our 67-year-old female patient was admitted to the hospital with complaints of nausea, vomiting, loss of appetite, and dysuria. She has a history of hypertension and type 2 diabetes mellitus. Two months before her admission to our clinic, she had gone to another hospital because of weakness. At that time her laboratory examinations revealed creatinin: 1.03 mg/dL, ESR: 89mm/h, CRP: 5.4mg/dl, leucocytes: 6,500/mm3, hemoglobin: 10.1 gr/dL, and hematocrit: 30%. A colonoscopy had been performed to investigate the patient's anemia. Phosphorous enema had been administered to prepare the patient for colonoscopy. The colonoscopy was reported to be normal.

At her presentation to our unit, the patient's physical examination was unremarkable. Laboratory examination revealed glucose: 122 mg/dL, creatinin: 11.8 mg/dL, urea: 226 mg/dl, BUN: 105 mg/dl, sodium: 131 mmol/L, potassium: 4.5 mmol/L, Ca: 9.1 mg/dl, P: 7.3 mg/dl, Mg: 1.38 mg/dl, uric acid: 5.4 mg/dl, ESR: 66 mm/h, CRP: 1.2 mg/dl, leucocytes: 6.800 /mm3, hemoglobin: 9.3 gr/dL, hematocrit: 28.2%, thrombocyte: 324.000/mm^3, MCV: 80 fL, Fe/TIBC: 62/294 ug/dL, ferritin: 329 ug/dL, and PTH: 338 pg/ml. Her venous blood gas results were pH: 7.25, PO_2: 61 kpa, PCO_2: 27 kpa, and HCO_3: 19 mmol/l. Her urinalysis revealed a pH of 5, 18 white blood cells, a specific gravity of 1.010 (normal is 1.002-1.030), and 2+ glucose without ketones.

During hospitalization the patient was rehydrated and three sessions of hemodialysis were performed before

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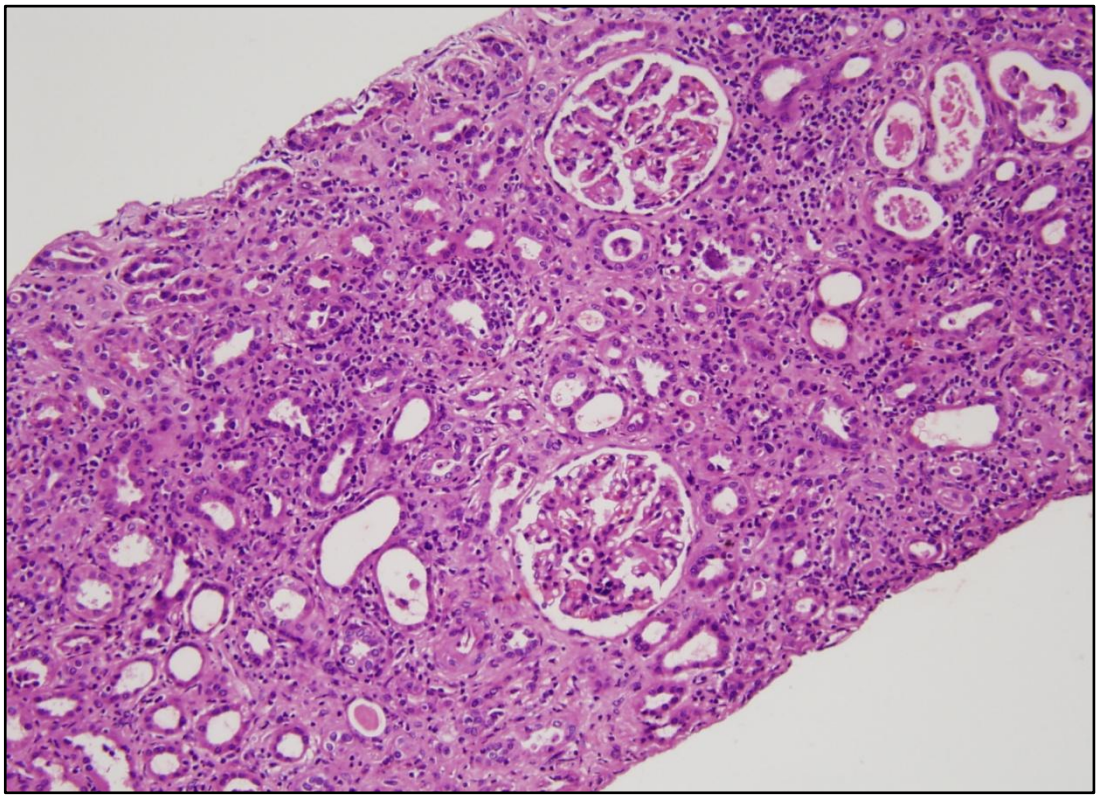

Figure-1. Tubular damage characterized by flattening and desquamation of tubular epithelial cells. Tubulointerstitial inflammation was seen. Glomeruli were unremarkable (HE 100x).

her kidney biopsy. The patient's creatinine decreased to 5 mg/dl and stayed constant at this level without dialysis. No complication ensued after the biopsy. The biopsy revealed acute tubular damage such as flattening and desquamation of tubular epithelial cells, as well as interstitial mixed inflammatory cell infiltration (Figure 1). Calcification with foreign body type multinucleated giant cells was detected focally in the interstitium (Figure 2). Five out of 19 globally sclerotic glomeruli were detected. No glomerular pathology was seen. Immunofluorescence microscopy did not show any immune deposits. Hence, acute phosphate nephropathy was highly suggested.

Oral steroid (0.5 mg/kg/day) was initially administered to the patient. Since she weighed 64 kilos, she received 32 mg/day methylprednisolone for a month, which dosage was gradually tapered for one month. After being discharged, the patient was followed up on day 53 at which time she was on 16 mg of methylprednisolone and her creatinine had dropped to 2.1 mg/dl.

DISCUSSION

The number of colonoscopies is rising worldwide due to national screening programs (Rex and Vanner, 2009). In the USA, for example, approximately 14.2 million colonoscopies are performed every year (Seeff et al., 2004). Adequate cleansing of the bowel before colonoscopy is important in order to obtain good results, and many preparations are used as purgatives. One such kind of purgative is an enema containing sodium phosphate. In one study, 28% of the patients who underwent elective colonoscopy had serum phosphate concentrations greater than 8.0 mg/dL (2.6 mmol/L) (normal range 2.5 to 4.5 mg/dL) (0.8 to 1.5 mmol/L) following administration of oral sodium phosphate (Lieberman et al., 1996). Hyperphosphatemia is more likely to occur in patients with renal insufficiency who have decreased excretion of phosphate (Gumurdulu et al., 2004). This study demonstrated a strong correlation between patient age and the degree of hyperphosphatemia, suggesting that hyperphosphatemia may be promoted by age-related factors unrelated to kidney function including decreased total body water, abnormal bowel motility, and enhanced GI absorption. Our patient was elderly and had a history of diabetes which may have made her susceptible to kidney injury.

Acute phosphate nephropathy, which may be detected days to months after administration of oral sodium phosphate, was in our case evident 2 months after the colonoscopy. In renal biopsies performed within three weeks of oral sodium phosphate exposure, findings of acute tubular injury predominate and resemble changes seen in acute tubular necrosis. Later biopsies may dis-

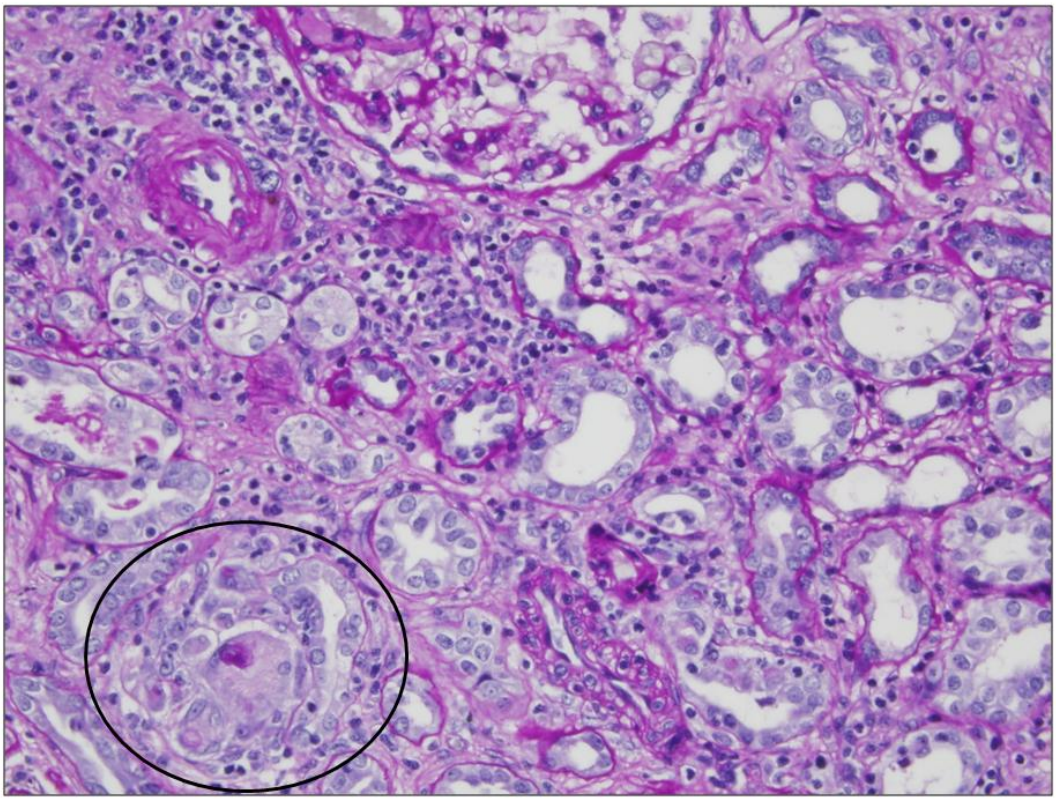

Figure- **2.** İnterstitial aggregate of foreign body type giant cells showing calcifications in their cytoplasms (PAS 200x)

disclose findings of chronicity, including tubular atrophy and interstitial fibrosis. In our patient, glomerular sclerosis, tubular atrophy, inflammatory cells infiltration in the interstitium, and tubular calcification were detected.

No specific treatment has been established for acute phosphate nephropathy although acute hemodialysis can be beneficial in patients diagnosed within 12 to 24 hours after administration of oral sodium phosphate. Deposition of calcium phosphate crystals within tubules and interstitium stimulate the immune system and cause inflammation for which steroids may be useful although complete recovery of renal function is rare. The presentation of our patient was late and because of the pathology findings we administered oral steroid (0.5 mg/kg/day) for a month of gradually tapered dosage. Upon discharge, her renal function had not recovered fully but she was stable and did not require dialysis support.

To conclude, sodium phosphate can be harmful and cause acute kidney injury, especially in elderly patients and patients with chronic kidney disease. It should therefore be used with caution and physicians must be aware of its harmful effects. In susceptible patients, other preparations (preparations without sodium phosphate) are preferable to sodium phosphate.

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