

Acute nephrocalcinosis following oral sodium phosphate bowel cleansing

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■ ABSTRACT

Acute nephrocalcinosis following oral sodium phosphate colonoscopy preparation is an under-reported cause of acute renal failure. Potential coadjutant factors are inadequate hydration, older age, hypertension, concomitant angiotensin-converting enzyme inhibitor or angiotensin receptor blocker, diuretics and nonsteroid anti-inflammatory use. We report a case of a 45 year-old male with dermatological and digestive vasculitis who developed acute renal failure following oral sodium phosphate bowel purgative. Kidney biopsy showed intratubular and interstitial deposits of calcium phosphate with no sign of renal vasculitis.

Key-Words:

Acute nephrocalcinosis; acute renal failure; oral sodium phosphate; osmotic purgative.

■ INTRODUCTION

Nephrocalcinosis is characterised histologically by abundant renal parenchymal deposits of calcium phosphate associated with chronic tubulointerstitial injury. Clinically patients with nephrocalcinosis have renal insufficiency, typically of gradual onset, and proteinuria <1g/day. Nephrocalcinosis is associated with hypercalcaemia-related conditions, including

hyperparathyroidism, increased bone turnover, excessive vitamin D intake, hypercalcaemia of malignancy, sarcoidosis, and distal renal tubular acidosis. While a few reports have focused on phosphate intake as a trigger factor for nephrocalcinosis¹, several recent isolated reports have described acute nephrocalcinosis following oral sodium phosphate (OSP) bowel cleansing (Fosfosoda®)^{2,3}.

We report a 45 year-old patient who developed acute renal failure two days after taking Fosfosoda®.

■ CASE REPORT

A 45 year-old Caucasian male was admitted to the emergency room with subocclusive bowel obstruction, abdominal pain and rectal bleeding of five days history. Patient's vital signs were normal on admission and laboratory results, including serum creatinine (0.7 mg/dL), urea (29 mg/dL), Ca (9.4 mg/dL), P (2.3 mg/dL) were unremarkable, as was patient's medical history. An abdominal CT scan with contrast dye and a colonoscopy were performed following OSP (two 45 ml doses, taken before each procedure) on days two and four following admission.

Neither procedure confirmed chronic intestinal inflammatory disease. Patient's hospital course

was complicated by the onset of generalised arthralgias with no sign of inflammation and with palpable purple in lower limbs. These appeared the day following colonoscopy and lasted for two days. A skin biopsy of the purple revealed a leukocytoclastic vasculitis. The day following the last imaging procedure (colonoscopy) serum creatinine rose to 4 mg/dL, urea to 119mg/dL, P to 5.9 mg/dL and Ca was 8.7 mg/dL. This meant that the calcium-phosphate (CaxP) product had increased from 21 at admission to 51 within five days. Urine tests revealed proteinuria 0.3 g/day with no eosinophils. A renal biopsy was performed at this point and empiric treatment with 1g methylprednisolone i.v. for three consecutive days initiated. This was followed by oral administration of 1mg/kg/body weight for the next five days at which point the renal biopsy results became available. Findings showed intratubular and interstitial deposits of calcium phosphate (Figure 1). There were no signs of vasculitis and all immunology studies were negative.

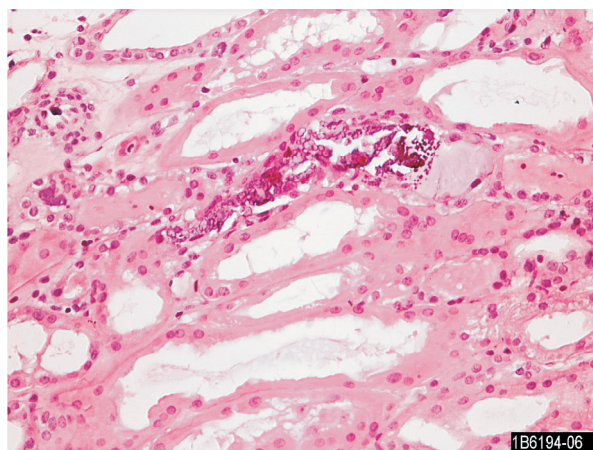


Figure 1

Kidney biopsy. Haematoxylin-Eosin stain showing intratubular and interstitial deposits of calcium phosphate.

Treatment was discontinued, with supportive hydration maintained. Patient was discharged on day 15, asymptomatic, with normal electrolyte balance and serum creatinine 2.3 mg/dL. After six months, patient's serum creatinine was 2.2 mg/dL with no digestive or cutaneous symptoms.

■ DISCUSSION

We describe a 45 year-old man who developed non-oliguric acute renal failure with intratubular and interstitial calcium phosphate deposits following colonoscopy and CT scan preceded by an osmotic purgative (Fosfosoda®). Patient developed simultaneously cutaneous purpura and arthralgias with no vasculitis visible on kidney biopsy.

OSP is used both as a laxative and as a purgative for bowel cleansing before colonoscopy and/or abdominal CT scan. The recommended regimen is two 45 mL doses taken 12h apart, the night before and the morning of the procedure. Each 45 mL dose contains 24.4g of monobasic sodium phosphate and 10.8g of dibasic sodium phosphate. In general, OSP is well tolerated. The most serious adverse events are related to inappropriate dosing or use in patients with preexisting conditions, such as renal insufficiency and/or intestinal disease with the majority of phosphate absorbed in the duodenum and jejunum³. Serum electrolyte monitoring over the initial 24h after administration of OSP reveals minor increases in phosphorus, sodium, haematocrit, serum osmolality and other disturbances might include hypocalcaemia⁴.

Transitory hyperphosphataemia is frequent following OSP use. A study including 143 patients found a mean increase in serum phosphate of 3 mg/dL, and a mean fall of 0.3 mg/dL in serum calcium levels⁵. An earlier study found that older patients attain greater levels of hyperphosphataemia after OSP⁶. CxP is an indicator of risk of calcium phosphate precipitation in the kidney. Vanner *et al.*⁷ found CxP rose to 71.2.

In our case, serum phosphate level rose from 2.3 to 5.9 mg/dL, Ca fell from 9.4 to 8.7 mg/dL and CxP rose from 21.6 to 51.3. Acute renal failure onset within one day of both procedures (CT scan and colonoscopy). Each Fosfosoda® dose before CT scan and colonoscopy was 90 ml, exceeding recommended doses of 45 ml.

Acute nephrocalcinosis is an underdiagnosed condition. Calcium phosphate renal deposits and acute renal failure following OSP administration are the main lines of evidence strongly pointing to OSP as the aetiological agent in acute nephrocalcinosis.

In our case both high Fosfosoda[®] doses and inflammatory colitis (leading to increased phosphate intestinal absorption) contributed to acute nephrocalcinosis.

Acute nephrocalcinosis should always be considered in the differential diagnosis of acute renal failure following administration of OSP.

Conflict of interest statement. None declared.

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