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Case Report

Peanut-induced acute oxalate nephropathy with acute kidney injury

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Abstract

Oxalate nephropathy is commonly caused by ethylene glycol, vitamin C, and foods like star fruit that contain a lot of oxalate. Peanuts also have high oxalate contents. However, case reports of peanut-induced oxalate nephropathy are not common. Here, we describe a case of peanut-induced acute oxalate nephropathy with acute kidney injury and intend to demonstrate the conditions under which peanut-induced oxalate nephropathy is likely to occur.

Introduction

Acute oxalate nephropathy is caused by deposition of oxalate crystals in the kidney that induce renal epithelial cell injury and inflammation. In severe cases, oxalate nephropathy can progress to acute renal failure. Ethylene glycol, vitamin C, and star fruit are considered possible causes of acute oxalate nephropathy along with other foods that contain significant levels of oxalate [1–4].

Peanuts have high oxalate contents (187.0 mg/100 g roasted peanuts) [5]. Therefore, peanuts could also cause oxalate nephropathy. Furthermore, peanuts are common foods that are easily consumed in everyday life. A few studies have described peanut-induced oxalate nephropathy [6,7]. Here, we also describe a case of peanut-induced acute oxalate nephropathy with acute kidney injury and intend to confirm whether peanuts can be a significant etiology for oxalate nephropathy.

Case report

A 44-year-old man visited the emergency room for persistent vomiting and diarrhea. He was diagnosed with diabetes mellitus, hypertension, and coronary artery disease several years ago, but his renal function was normal (serum creatinine, 0.82 mg/dL) until 2 months ago. He took 100 mg aspirin, 160 mg valsartan, 5 mg amlodipine, 10 mg rosuvastatin, and 60 mg nitrate once daily. He sometimes drank too much, and at the time of 20 days prior to the visit, he consumed five bottles of Korean distilled spirits and 20 bottles of beer daily without meals for 5 days (daily alcohol consumption 140 g, total alcohol consumption 700 g). During this period he only consumed peanuts ($>$ 130 g/day). Since then, he started to vomit and had diarrhea. The symptoms worsened for 15 days until the visit.

Initially, he complained of heat sensation, gross hematuria, and edema. Physical examination showed blood pressure 152/92 mm Hg, pulse rate 87 beats/minute, body temperature 36.6°C, respiration rate 20/minute, O_2 saturation 97%, and central venous pressure 8 cmH_2O. In addition, pretibial pitting edema was observed, whereas costovertebral tenderness was not. In blood tests, he had a white blood cell count of 8,220/mm^3, hemoglobin 12.9 g/dL, and platelet count of 139,000/mm^3, and in arterial blood gas analysis, pH 7.38, pO_2 (oxygen partial pressure) 90.3 mmHg, pCO_2 (carbon dioxide partial pressure) 21.9 mmHg, and HCO_3 12.9 mmol/L. Calculated serum anion gap was 24.3 mEq/L and osmolal gap was 4 mOsm/kg, which represent high anion gap metabolic acidosis. Blood urea nitrogen and creatinine were elevated to 106.4 mg/dL and 20.9 mg/dL, respectively, demonstrating acute renal failure. Creatinine kinase and myoglobin were elevated to 616 U/L and 591.6 ng/mL, respectively, whereas urine myoglobin

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was negative, suggesting mild rhabdomyolysis. Amylase of 89 U/L and glucose of 89 mg/dL showed no evidence of acute pancreatitis. Albumin was 3.4 g/dL, C-reactive protein was 0.63 mg/dL, aspartate aminotransferase and alanine aminotransferase were 14 U/L and 119 U/L, respectively, and total bilirubin was 0.87 mg/dL. Serum sodium was decreased to 107mmol/L and serum osmolality was 260 mOsm/kg, indicating severe hyponatremia. We believed that hyponatremia occurred because of acute renal failure, as he had edema and all relevant hormone levels (renin, aldosterone, adrenocorticotropic hormone, cortisol, and thyroid hormone) were normal. Although hyponatremia was severe, no central nervous system symptoms were observed. Potassium was 4.4 mmol/L, and chloride was 70 mmol/L. Calcium level was as low as 5.3 mg/dL, and phosphorus was as high as 12.9 mg/dL, which are common findings in renal failure. Dipstick urine test showed diluted urine (specific gravity 1.008), proteinuria, hematuria, and pyuria. In addition, urine electrolytes showed that sodium was 38 mmol/L, potassium 12.0 mmol/L, and chloride 24 mmol/L. Fractional excretion of sodium was 7.62%. Chest radiography showed neither pulmonary edema nor pneumonia. Abdominal computed tomography showed that both kidneys were swollen, indicating acute kidney injury, and there was no finding of ureteral stone.

On hospital Day 2, hemodialysis was initiated due to high urea nitrogen level and oliguria with a urine output of 10 mL/hour. Additional tests, serum antinuclear antibody and antineutrophil cytoplasmic antibody, were negative. Serum complement component-3 and antistreptolysin O titer were normal. Abdominal sonography showed ascites, kidney size 11 cm, increased cortical echogenicity, and no obstructive uropathy. We treated the patient conservatively with intermittent hemodialysis. On hospital Day 13 renal biopsy was done to investigate the exact cause of renal failure.

Biopsy results showed that glomeruli appeared normal under light microscopy (Fig. 1A), whereas the interstitium showed mild edema, fibrosis, and multifocal lymphoplasmacytic infiltration (Fig. 1B). Some tubular epithelial cells showed degeneration, loss of nuclei, and regenerative atypia with intraluminal acellular deposits (Fig. 2A). In polarizing microscopy, these deposits showed crystal features (Fig. 2B). Immunofluorescent microscopy showed no specific findings. A semithin electron microscopy section showed the shape of the crystals more clearly (Fig. 3A). The crystals had the shape of a prism, which is the characteristic of oxalate crystals [8]. In addition, a high-power view showed a hollow area, which was caused by washing out oxalate crystals (Fig. 3B). Finally, the pathological diagnosis was acute tubular necrosis with oxalate nephropathy.

On hospital Day 15 the patient's urine volume increased to 1,400 mL/day and serum creatinine decreased to 3.4 mg/dL. The patient had received a total of six hemodialysis sessions. He was discharged from hospital, along with renal recovery. Because a 24-hour urine test was not done initially due to oliguria, the test was performed after recovery. A 24-hour urine test showed total protein 132 mg/day, microalbumin 12 mg/day, creatinine clearance 92.7 mL/minute, calcium 124 mg/day (reference range, $<$ 150 mg/day), phosphorus 796 mg/day (reference range, 400–1,300 mg/day), uric acid 672 mg/day (reference range, 250–750 mg/day), citrate 80.2 mg/day (reference range, $>$ 150 mg/day), and oxalate 35.9 mg/day (reference range, 16.2–53.3 mg/day), which shows that there was no significant hyperoxaluria after recovery. After that, the patient had an improved renal function with a serum creatinine level of 1.0 mg/dL.

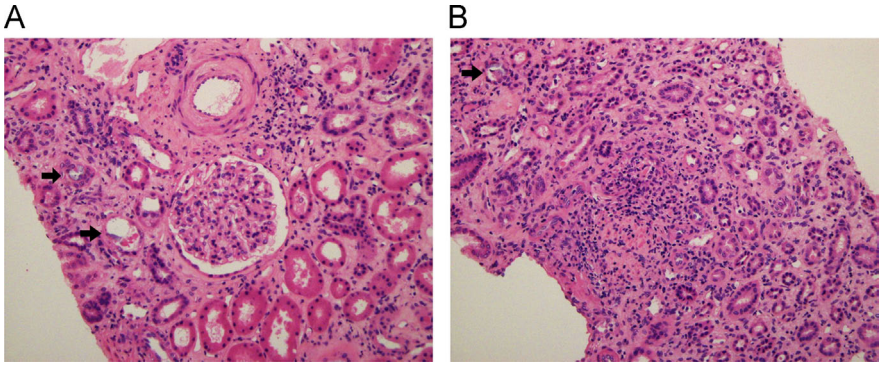

Figure 1. Light microscopic findings of kidney biopsy tissue. (A) Light microscopy shows the normal appearance of glomerulus. (B) On the contrary, interstitium is infiltrated by inflammatory cells, and tubular epithelial cells are damaged. A few tubular lumens are filled with acellular materials (black arrows) (hematoxylin and eosin, 200 ×).

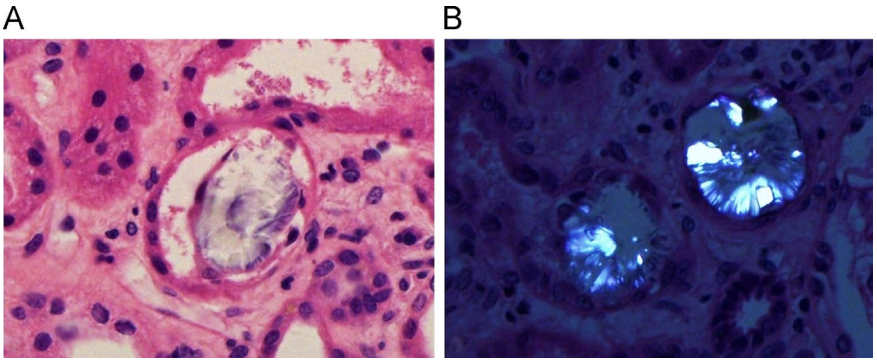

Figure 2. Light and polarizing microscopic findings of intra-tubular deposits. (A) Light microscopy shows acellular deposits in tubular lumens and damaged tubular epithelial cells (hematoxylin and eosin, 400 ×). (B) Polarizing microscopy shows two refractile crystals in tubular lumens.

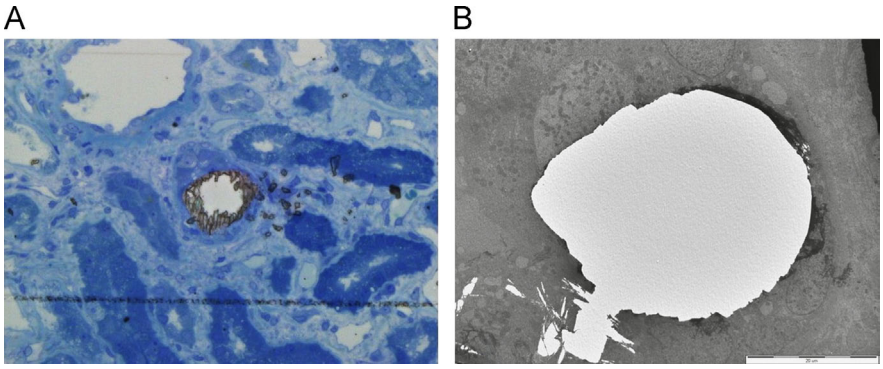

Figure 3. Electron microscopic findings of intra-tubular deposit. (A) Semithin electron microscopy shows the deposit of prism-shaped crystals in tubular lumen. (B) High-power view shows hollow space that was caused by washing out deposited crystals (2,000 ×).

Discussion

Oxalate nephropathy is a crystal-associated kidney disease. Previous studies have demonstrated that oxalate crystals could induce an injury to renal tubular cells. When urinary oxalate becomes supersaturated, crystal nucleation starts to occur, and then crystals grow and are aggregated. Formed crystals can attach to the apical membrane of renal tubular cells and are transported into the intracellular space by endocytosis. In particular, oxalate crystals have the characteristics of attaching to the membrane of tubular cells more easily than other types of crystals. Intracellular oxalate crystals can induce apoptosis and necrosis of renal tubular cells by oxidative stress and other processes [9].

The common sources of oxalate crystals are ethylene glycol, vitamin C, and foods that contain a lot of oxalate. Peanuts are also considerable sources of oxalate nephropathy. A few studies have described peanut-induced oxalate nephropathy. To the best of our knowledge, two cases were described by Getting et al [6] and Sasaki et al [7]. Getting et al [6] described a case of acute oxalate nephropathy in a review of patients with biopsy-proven renal calcium oxalate crystals, whereas Sasaki et al [7] described the case of chronic oxalate nephropathy. Our case was similar to that of Getting et al [6].

In our case, the cause of acute renal failure other than oxalate nephropathy was uncertain. In particular, the probability of alcoholic ketoacidosis was low because initial tests showed no urinary ketones, and his last drinking was done 15 days prior to the visit. Dehydration was also excluded due to the finding of edema. Furthermore, there was no history of nephrotoxic drug use. Therefore, we believe that oxalate nephropathy induced acute renal failure in this case.

His daily intake of oxalate was >243 mg/day during the period of binge drinking. Such an amount of oxalate was more than the average intake of a usual western diet (100–150 mg/day) [6]. However, although peanuts are considerable risk factors for oxalate nephropathy and the patient had above-average consumption of peanuts, we believe that there is little chance that peanuts consumption alone caused acute oxalate nephropathy with renal failure. A notable fact is that the patient consumed large amounts of alcohol at the same time. Alcohol generally has a low oxalate content (draft beer: 1.0 mg/100 g, bottled beer: 0.0 mg/100 g, and distilled liquor: 0 mg/100 g) [5]. Therefore, it is believed that alcohol alone does not cause significant oxalate nephropathy. Instead, we believe that alcohol might have an additive role in inducing peanut-associated oxalate nephropathy. Sasaki et al [7] have indicated that alcohol could be synergistic in the development of oxalate nephropathy, depending on body fluid balance. In our opinion, alcohol could prevent crystal aggregation through induction of diuresis. If alcohol induces marked diuresis without dehydration, it is helpful for prevention of oxalate crystal formation in the tubular lumen. On the contrary, if alcohol induces heavy diuresis sufficient to cause volume depletion, it may rather aggravate oxalate crystal formation. We believe that our case corresponds with the latter.

In conclusion, our case indicates that excess intake of alcohol and peanuts simultaneously could result in development of acute oxalate nephropathy with renal failure.

Conflicts of interest

The authors have no financial conflicts of interest.

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