BMC Nephrology

CASE REPORT Open Access

Acute oxalate nephropathy due to yellow rice wine: a case report

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Abstract

Background Acute oxalate nephropathy is a rare but potentially underrecognized cause of acute kidney injury (AKI). The reports of secondary oxalate nephropathy induced by high doses of vitamin C, portulaca oleracea, Peanut were described, but the cause as yellow wine consumption are rare. We report a 63-year-old man with acute kidney injury due to yellow rice wine. The patient has drunk yellow rice wine 500 mL/day for the past 10 years. He underwent an ultrasound-guided renal biopsy, which showed acute tubular injury and birefringent crystals were observed in the renal tubules under a polarizing microscope. The 24-hour urinary oxalate excretion established and the baseline serum creatinine of 407 μmol/L. Following 3 months therapy, subsequent laboratory evaluation demonstrated significant reduction in urinary oxalate excretion excretion and serum creatinine normal.

Conclusions Acute secondary oxalate nephropathy due to excessive dietary intake of oxalate may lead to AKI. Kidney biopsies in unknown cause AKI patients is important and attention should be payed to food behaviors when reasons for AKI are explored.

Keywords Oxalate nephropathy, AKI, Yellow rice wine

Introduction

Oxalate nephropathy is a rare complication of hyperoxaluria, characterized by the deposition of calcium oxalate crystals in the renal parenchyma [1]. Oxalate is an end product of metabolism excreted via the kidney. Excess urinary oxalate, whether from primary or enteric hyperoxaluria, can lead to oxalate deposition in the kidney. Oxalate crystals are associated with renal inflammation, fibrosis, and progressive renal failure. Elevated oxalate concentrations within the renal tubular lumen promote the aggregation of calcium oxalate crystals, which subsequently induce mechanical obstruction of tubular flow and exert direct cytotoxic effects on tubular epithelial cells through mitochondrial dysfunction and reactive oxygen species (ROS) generation. These pathophysiological processes culminate in progressive tubular injury, interstitial inflammation, and ultimately, decline in glomerular filtration rate (GFR) [2]. Here, we report a biopsy-proven case of acute oxalate nephropathy, caused by excessive ingestion of yellow rice wine that has not been previously reported.

Case report

A 63-year-old man visited our hospital with diarrhea for 5 days and an elevated creatinine level for 1 day. He had diarrhea for 5 days before admission without any obvious causes. His diarrhea occurred 3 times in the form of a yellow watery stool. The night after drinking 500 mL of yellow wine, he experienced unbearable abdominal distension. The patient has no history of chronic kidney

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disease. For the past 10 years, the patient has drunk yellow rice wine 500 mL/day. His blood pressure was 174/79 mmHg, and other findings on physical examination were unremarkable.

The results of initial laboratory examination were as follows: serum creatinine level: 407 umol/L (eGFR, [MDRD] 13.77 mL/min/1.73 m^2), urea: 19.69 mmol/L, uric acid: 515 umol/L, cystatin C: 1.77 mg/L, blood calcium: 2.09mmol/L, and urine protein: 0.99 g/24 h. Urine microscopy: Crystal examination negative. The patient's liver function tests were normal, and the patient was negative for antinuclear antibodies, double-stranded DNA antibody, anti-neutrophil cytoplasmic antibody, anti-neutrophil cytoplasmic antibody, hepatitis B and C virus, HIV, syphilis, tumor markers, and heavy metals (blood lead, blood cadmium, urinary cadmium, and urinary mercury).

Pathological examination

Thirty-six glomeruli were seen in each kidney biopsy specimen, with no lobulation or sclerosis. Glomerular assessment exhibited segmental mild widening of the mesangial region and increased mesangial stroma, but the abundance of mesangial cells did not significantly increase. We observed several small foci and scattered small patches of flattened renal tubular epithelial cells, brush border detachment, tubular lumen dilatation (5-15%), tubular epithelial disintegration, necrosis, cellular regeneration, patchy granular degeneration of renal tubular epithelial cells, vacuolar degeneration, and amorphous crystals in the proximal tubule. The lumen was partly brown in hematoxylin and eosin (H&E) staining. The tubular lumen was segmentally thickened and stratified. H&E staining revealed browning, with a small amount of protein deposition in the tubular lumen. The renal interstitium showed mild swelling, single-nucleated cells spread in small groups, and eosinophilic infiltration of granulocytes. There was no evidence of fibrosis in the interstitium. Segmental hyaline degeneration of small arteries was observed in pathological assessment. The pathological diagnosis was acute tubular necrosis. PASM-Masson and Congo red staining were negative. Immunofluorescencewere negative for immune complex deposition. Several birefringent crystals were observed in the renal tubules under a polarizing microscope (Fig. 1).

Urinary oxalate and genetic testing

24 h urinary oxalate concentration was 109.094 ug/mL (corrected result: 283.934 mg/1.73 m^2/24 h). Genetic testing showed no primary oxalate nephropathy.

Diagnosis

The patient was diagnosed with acute secondary oxalate nephropathy and subsequent acute tubular injury.

Clinical follow-up

The patient was instructed to avoid alcohol drinking and was rehydrated. The patient was given large amounts of water, potassium citrate, probiotics, and vitamin B6, and blood creatinine level gradually decreased (Table 1).

24 h urinary oxalate excretion was 29.408 ug/mL (corrected result: 56.381 mg/1.73 m^2/24 h) after 2 months of treatment. Proteinuria continued with 0.13 g/24 h protein excretion.

Discussion

This case report demonstrates that consuming large amounts of yellow red wine unintentionally leads to massive precipitation of calcium oxalate crystals in renal tubules and acute tubular injury.

Oxalic acid is a dietary anti-nutritional factor, and high levels of dietary oxalic acid can lead to calcium oxalate deposition, acute tubular injury, and nephrolithiasis. Urinary or plasma oxalate levels can be easily measured. In kidney biopsy, calcium oxalate crystals can be observed under polarized light as birefringent aggregating fan or

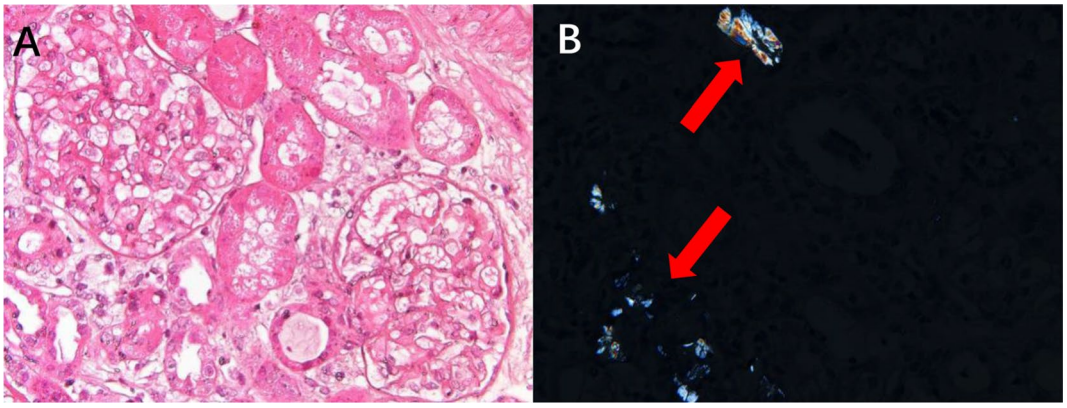
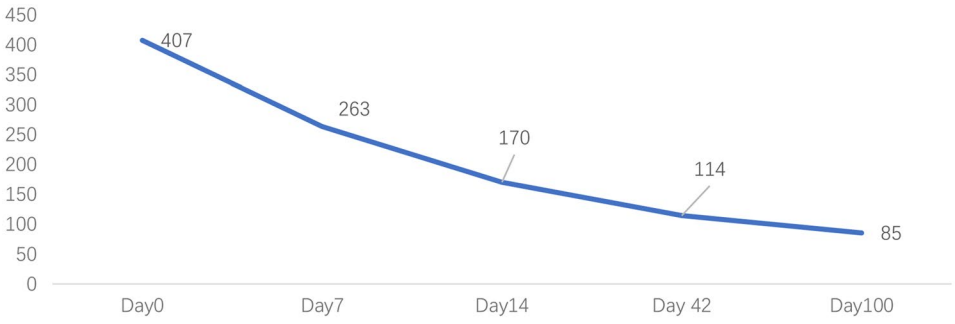


Fig. 1 Renal biopsy specimen with arrow showing a couple of calcium oxalate deposits within renal tubules. **(A)** HE staining, 400× magnification. **(B)** Polarized light, 400× magnification. Red arrow: birefringent crystals

Table 1 Trend in blood creatinine(umol/l)



rosette-shaped structures. They usually deposit in renal epithelial cells or renal tubules [3, 4]. Urinary oxalate excretion in healthy adults is influenced by dietary intake, with 40–45 mg/d (500 µmol/d) urinary oxalate defined as hyperoxaluria [5]. The 24 h urinary oxalate excretion of our patient was above the normal limit (109.094 ug/mL; corrected result: 283.934 mg/1.73 m2/24 h).

The overall prevalence of oxalate nephropathy is unclear. Recent reviews [6] showed that oxalate nephropathy is responsible for kidney disease in 1% of native kidney biopsies, and typically manifests with CKD or AKI. In kidney tissue, calcium oxalate crystals are seen as birefringent aggregating fan or rosette-shaped structures under polarized light. They usually deposit in renal epithelial cells or renal tubules.

Oxalate nephropathy has two broad categories: primary hyperoxaluria and secondary oxalate nephropathy [1]. Primary hyperoxaluria (PH) consists of a group of autosomal recessive disorders causing hepatic overproduction of oxalate due to the accumulation of the oxalate precursor glyoxylate [7]. Secondary hyperoxaluria is a more common type and relies on increased intestinal absorption and excessive intake of oxalate. There are several case reports of oxalate nephropathy associated with high dietary intake of oxalate from different sources, such as nuts, EDTA, Vitamin C, orlistat, and traditional medicinal herbs [3, 8–11]. However, there are no reports of oxalic acid nephropathy due to yellow rice wine.

Some studies have found oxalate precipitation in yellow rice wine. They found that high levels of oxalic acid can be seen at the bottom of finished wine bottles. Oxalic acid levels of 45.32 mg/L to 1046.58 mg/L were found in commercially available yellow rice wine [12]. To our knowledge, this is the first case report suggesting an association between oral yellow rice wine and oxalate nephropathy, highlighting the potential adverse effects of this wine.

Our patient likely experienced chronic intermittent hyperoxaluria due to excessive and prolonged consumption of yellow rice wine. Diarrhea, as a possible contributory factor, accelerated oxalate crystal formation and acute kidney injury.

Treatment of oxalate nephropathy should be started immediately after diagnosis. Treatment of oxalate nephropathy includes a low-oxalate low-fat diet, a high amount of fluid intake, and supplements that can improve the solubility of oxalate, such as citrate and sodium bicarbonate [13].

Recently, probiotics have been thoroughly investigated to reduce urinary oxalate excretion. Wei et al. showed that the probiotic LPN1 can prevent ethylene glycol-induced hyperoxaluria in the rat model by modulating gut microbiota and improving intestinal barrier function [14]. We followed these treatment strategies after confirming the diagnosis of oxalate nephropathy, and the patient's renal function improved after treatment.

In conclusion, oxalate nephropathy is an uncommon cause of AKI that can only be diagnosed in kidney biopsy. We recommend that a thorough history, particularly dietary history, should be taken from all patients with a rapid decline in renal function, especially in the absence of known risk factors of AKI. Furthermore, renal puncture should be recommended for such patients.

Abbreviations

AKI Acute kidney injury
CKD Chronic kidney disease

Acknowledgements

The authors would like to express their gratitude to EditSprings (https://www.editsprings.cn) for the expert linguistic services provided.

Author contributions

CX, RYC and ZR were the clinician responsible for taking care of the patient. CX analyzed the patient clinical data wrote the manuscript. LYX performed pathological analysis. SJ followed up the patient. CX and CMD collected the clinical data. Prof ZR responsible for review and revision of the paper, and for the final approval of the paper. All authors contributed to the writing process and read and approved the final manuscript.

Funding

No funding was needed to report this case.

Data availability

All data generated or analysed during this study are included in this published article [and its supplementary information files].

Declarations

Ethics approval and consent to participate

This paper followed the Helsinki Declaration. This case report was approved by the ethics committee of Yangpu Hospital, Tongji University School of Medicine.

Consent for publication

The patient provided written informed consent for the publication of this case report.

Competing interests

The authors declare no competing interests.

Received: 2 January 2025 / Accepted: 12 March 2025
Published online: 25 March 2025

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