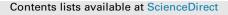
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# Letter to Editor

# High-dose vitamin C-induced acute oxalate nephropathy in a renal transplant recipient: A case report and literature review



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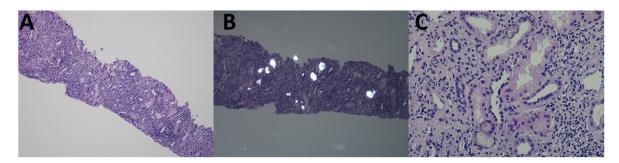
To the editor,

We would like to provide new information concerning highdose vitamin C-induced acute oxalate nephropathy in a renal transplant recipient. Intravenous vitamin C has been reported to have benefits in patients with sepsis, burn injury, and malignancy.<sup>1</sup> Intravenous vitamin C is used in a contentious adjunctive cancer therapy. However, some side effects had been mentioned, including acute renal failure.<sup>2</sup> Ascorbic acid is converted to oxalate endogenously, which leads to hyperoxaluria and oxalate crystals deposition in renal tubules and interstitium, results in acute interstitial nephritis and tubular necrosis.<sup>3</sup> We herein present a complication of high-dose vitamin-C in a patient after kidney transplantation and literature review.

A 41-year-old woman visited our emergency room due to fever, nausea, progressive decreased urine output. She received living donor kidney transplantation in 2019. She also had a history of left renal pelvis urothelial carcinoma status post left nephroureterectomy and bladder cuff resection in 2011. Four days prior to this presentation, she received 7 g intravenous vitamin C administered by a local medical doctor for recurrent urothelial carcinoma. Her baseline serum creatinine level was 1.7 mg/dL and rose to 7.3 mg/ dL 4 days after high-dose vitamin C therapy. Urine sediment examination revealed numerous monohydrated calcium oxalate crystals. Hemodialysis was initiated on the third day after admission for persistent anuria. Renal biopsy revealed significant calcium oxalate deposition within both tubular epithelial cytoplasm and tubular lumen and acute tubular necrosis, and also marked tubular atrophy and interstitial fibrosis (Fig. 1). The diagnosis of acute oxalate nephropathy was confirmed. The patient did not recover renal function upon discharge.

We also reviewed the literatures about vitamin C induced acute oxalate nephropathy during 1976-2021 in PubMed. A total of 31 cases, including our case, with biopsy-proven vitamin C-induced oxalate nephropathy were reported (supplementary data). Considering the rationale and administration route, 21 patients received oral vitamin C supplement for nutrition supply or cancer, and the other 10 patients received parenteral vitamin C supplement for cancer, sepsis, burn injury, renal amyloidosis. Unfortunately, most patients (28 cases) developed acute kidney injury requiring dialysis therapy and 15 patients remained dialysis-dependent. The doses varied greatly, while not reported sometimes, with a maximal dose up to 60,000 mg intravenously. Furthermore, we investigated the risk factors associated with worse outcome of vitamin Cinduced oxalate nephropathy (supplementary data Table 2). We found some potential risk factor of vitamin C-induced oxalate nephropathy, including baseline impaired renal function (eGFR <60), history of kidney transplantation, and parenteral route administration.

We confirmed that high-dose intravenous vitamin C led to the acute oxalate nephropathy in this renal transplant recipient. Considering the efficacy and relevant clinical safety of intravenous vitamin C therapy for malignancy, we suggest that high-dose vitamin C therapy should be avoided in renal transplant recipients.



**Fig. 1.** Renal biopsy showing diffuse translucent crystals deposition in a background of marked tubular atrophy, interstitial fibrosis and interstitial inflammation on hematoxylin and eosin stain (100X) (A). These crystals showing birefringent on polarized light (B) and distributing within both tubular epithelial cytoplasm and tubular lumen, accompanied with tubular dilatation (400X) (C), consistent with calcium oxalate crystal deposits.

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Further carefully designed studies are still needed to confirm the risk factors for vitamin C-induced oxalate nephropathy.

# Declaration of competing interest

We have no conflicts of interest to disclose.

## Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.asjsur.2022.11.112.

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