AKI in a Patient Engaged in Vegetable Juicing

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Clinical Images in Nephrology and Dialysis
Case Description
A 68-year-old man with a past medical history of diabetes mellitus, hypertension, and achalasia presented to the emergency department with nausea and vomiting about 6–8 times daily, poor appetite, and 10-pound weight loss for >6 weeks. He had become a vegetarian 3 years before to lose weight and to better control his blood glucose. For the last 6 months, he had blended a vegetable juice and drunk 1 quart daily. This blend included unmeasured amounts of raw spinach, broccoli, cauliflower, green beans, asparagus, kale, beets, cilantro, parsnips, mushrooms, onions, and yams. On physical examination, his BP was 142/90 mm Hg and pulse rate was 83. He appeared ill with nausea and vomiting. Laboratory results revealed serum creatinine 9.6 mg/dl and BUN 92 mg/dl, increased from 1.4 mg/dl to 24 mg/dl 2 months earlier. Bicarbonate was 18 mmol/L, phosphorus 11.2 mg/dl, and albumin 3.6 g/L. Urine sediment showed ten red cells per high-power field and a moderate number of muddy brown casts, but no crystals. He was aggressively hydrated, but his kidney function failed to improve and he was started on hemodialysis. On day 10 he underwent a kidney biopsy, which showed severe tubular injury with diffuse intratubular oxalate crystal deposition (Figure 1A). The biopsy demonstrated characteristic birefringence when viewed under polarized light (Figure 1B). Intratubular oxalate crystals were also identified on electron microscopy (Figure 1C). He denied ingesting any vitamin C or ethylene glycol.

Previous reports have shown that excess oxalate intake, whether from primary or enteric hyperoxaluria, can lead to calcium oxalate deposition in the kidney (1) and a small percentage of patients with oxalate nephropathy are attributed to diets rich in oxalate content. In a review by (2), a retrospective study done at the Mayo clinic looked at 65 patients with kidney biopsy specimens showing calcium oxalate crystals between 1985 and 2010. These patients were reviewed to identify the underlying causes of calcium oxalate nephropathy. High dietary intake of oxalate-containing foods was identified as the likely cause of hyperoxaluria in three patients. Two or more factors were identified as the cause of oxalate nephropathy in 29 of the patients. In our patient, the biopsy findings clearly diagnosed acute oxalate nephropathy as the cause of his AKI due to his diet, and volume depletion as a second factor (3).

Teaching Points
- Vegetable juicing with fruits and vegetables that contain a high oxalate content is a rare cause of acute oxalate nephropathy.
- Microscopy with polarization is useful to identify calcium oxalate crystals.
- Acute oxalate nephropathy can lead to ESKD.

Figure 1. | Biopsy images. (A) Light microscopy demonstrates diffuse intratubular oxalate crystals. (B) Immunofluorescence demonstrates birefringence when viewed under polarized light. (C) Electron microscopy demonstrates intratubular oxalate crystals.

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O. Obadan was responsible for investigation and writing the original draft of the manuscript; and M. Yudd was responsible for supervision.

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References

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