Hyperoxaluric acute kidney injury and frontotemporal dementia

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A 58-year-old man, who after reporting several days of abdominal pain was found to have acute kidney injury, was transferred to our unit from a regional hospital.

In the preceding 6 months, the patient had been diagnosed with frontotemporal dementia; he had no personal or family history of nephrolithiasis, bowel disease, or weight loss. Notably, the year before this presentation, his kidney function had been found to be normal with the serum creatinine concentration recorded as 71 μmol/L (normal range 60–115).

On examination, the patient was clinically well with a blood pressure of 122/66 mm Hg, heart rate of 95 beats per min, respiratory rate of 18 breaths per min, and an oxygen saturation of 95% on room air.

Laboratory investigations confirmed acute kidney injury—his serum creatinine concentration was 703 μmol/L with preserved urinary output; his remaining serum biochemistry was unremarkable. The urinary protein/creatinine ratio was 39 mg/mmol (normal range 0–12·5).

The patient was admitted to the nephrology ward for a kidney biopsy which showed acute tubular injury with abundant intratubular calcium oxalate crystals; the background kidney was healthy, with little scarring, and only 1/14 obsolete glomeruli (figure).

A detailed dietary history found that the patient had been eating 500–1000 g of almonds daily over the last four years—corresponding to approximately 2–4 g of oxalate per day. He had no evidence of malabsorption; his faecal elastase concentration was >500 ug/g (typical range 200 to less than 500).

A CT of the kidney, ureter, and bladder showed a non-obstructing 1·4 cm calculus in the lower pole of the right kidney and a non-obstructing 0·6 cm calculus in the distal left ureter (figure). We concluded the patient had a diet-related secondary hyperoxaluria; frontotemporal dementia is associated with unusual, compulsive eating habits.

As he was asymptomatic, we decided to manage him conservatively; the patient was started on a low-oxalate, high-calcium diet with a high fluid intake and reviewed regularly. At his last appointment, the patient's laboratory results showed his serum creatinine concentration was improving (478 μmol/L).

Hyperoxaluria causes calcium oxalate stones by raising urinary saturation of calcium oxalate; hyperoxaluria can result from ingestion of oxalate-rich foods, such as rhubarb, spinach, beetroot, almonds—as in our case— and chocolate. Hyperoxaluria is less common than hypercalciuria as a cause of calcium oxalate stones.

Non-obstructive kidney stones of less than 15 mm can be treated conservatively and monitored closely; an increase in the size of the stone or complications, such as infections, may result in a more proactive approach.

Contributors
All the authors provided care for the patient and contributed to writing the paper. Written consent for publication was obtained from the patient.

Declaration of interests
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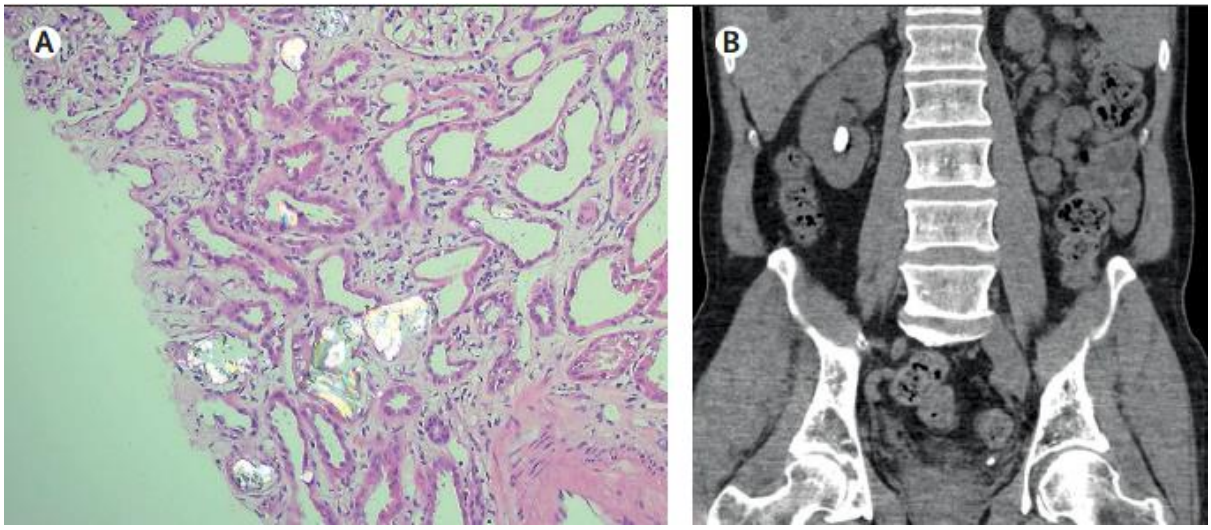
Figure : Almonds and accute kidney injury



(A) Histopathological analysis of sample of biopsy of the kidney shows multiple intratubular calcium oxalate crystals under birefringent light and flattened tubular epithelium (haematoxylin and eosin stain). Magnification X40.

(B) CT of the kidney, ureter and bladder shows a non-obstructing 1.4 cm calculus in the lower pole of the right kidney and a non-obstructing 0.6 cm calculus in the distal left ureter.