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Renal Failure due to Excessive Intake of Almonds in the Absence of *Oxalobacter formigenes*

To the Editor:

A previously healthy 49-year-old man was admitted with a serum creatinine level of 1265 umol/L (normal, 60-105 umol/L) following several weeks of tiredness and loss of appetite. He was in reduced general condition but alert and normotensive. Urine output and urine analyses were normal without proteinuria or hematuria. Renal ultrasound examination revealed kidneys of normal size with high echogenicity and small calculi in the renal papillae. These findings could be consistent with nephrocalcinosis. Blood analysis for monoclonal gammopathies was negative. A renal biopsy revealed oxalate nephropathy with excessive depositions of birefringent oxalate crystals associated with severe tubular damage, interstitial edema, and inflammation (**Figure**).

Oxalate nephropathy is characterized by the depositions of calcium-oxalate complexes in the kidneys. The underlying cause might be primary or secondary hyperoxaluria. Primary hyperoxaluria is an extremely rare genetic disorder usually detected in infants, but there have also been some cases reported among adults. Secondary hyperoxaluria is much more frequent and is related to a broad spectrum of medical conditions. This disorder is a known complication of malabsorption seen in cystic fibrosis, inflammatory bowel diseases, and secondary to bariatric surgery.[1] Several cases of oxalate nephropathy due to customized diets, excessive intake of ascorbic acid, and consumption of ethylene glycol have also been reported.[2] Primary and secondary hyperoxaluria can both lead to systemic oxalosis with oxalate depositions in the myocardium, retina, and joints.

Blood analysis in our patient showed elevated plasma oxalate at 270 umol/L (normal, 3-11 umol/L); respective values in the 24-hour urine could not be conclusively interpreted due to the advanced kidney failure. The patient did not have a family history of kidney disease or a personal history of kidney stones, abdominal surgery, or consumption of ethylene glycol.[2] Subsequent investigations did not detect malabsorption or evidence of systemic oxalosis, as investigated by echocardiography, skeletal radiography, and retinal examination. In addition, the respective genetic tests for primary hyperoxaluria remained negative.

The dietary history of the patient revealed an unusually restricted meal pattern, with minced meat and almonds as primary staple foods. These restrictions were based on a subjective intolerance to most foodstuffs typical for the Western diet. He consumed 150-200 grams of almonds and 50-100 grams of almond-containing marzipan daily. Almonds have a high oxalate content. We calculated a daily oxalate intake of 735-1060 mg, compared with 100-150 mg among the general population.

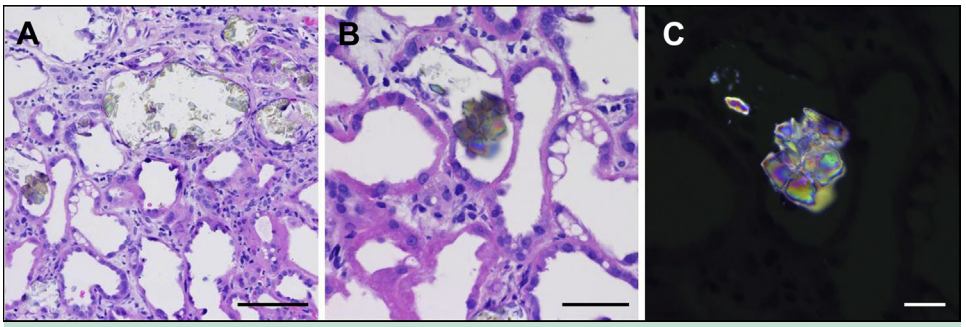

Figure Oxalate nephropathy. Kidney biopsy with multiple crystals in the tubular lumina (**A**,**B**). Crystals were birefringent under polarized light (**C**), consistent with oxalate crystals (hematoxylin-eosin stain, range A: 100 µm, B: 50 µm, and C: 20 µm).

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Requests for reprints should be addressed to Yngvar Lunde Haaskjold, MD, Department of Medicine, Haukeland University Hospital, Post Box 1400, Bergen 5021, Norway.

E-mail address: yngvarhaaskjold@gmail.com

Several studies indicate that intestinal dysbiosis with absence of the oxalate-degrading bacteria *Oxalobacter formigenes* could contribute to increased intestinal oxalate absorption.[3,4] This dysbiosis is seen after systemic treatment with antibiotics. A fecal polymerase chain reaction examination was performed, and *O. formigenes* was not detectable. This result suggests the absence of this bacterium in the patient's intestinal microbiota.

We believe that this patient's renal failure is caused by excessive dietary intake of oxalate-rich foods combined with an intestinal dysbiosis. Almonds are rich in oxalate, and studies indicate that oxalate from almonds has a very high bioavailability.[5]

When faced with uncertain causes of renal failure, it is of great importance to thoroughly examine dietary habits and nutritional supplements.[2] The patient in this case remains hemodialysis dependent, but needs to change his dietary habits for future kidney transplantation.

Yngvar Lunde Haaskjold, MD[a]
Aslaug Drotningsvik, MSc[b]
Sabine Leh, PhD[b,c]
Hans-Peter Marti, PhD[a,b]
Einar Svarstad, PhD[a,b]

[a]*Department of Medicine*
Haukeland University Hospital
Bergen, Norway
[b]*Department of Clinical Medicine*
University of Bergen
Bergen, Norway
[c]*Department of Pathology*
Haukeland University Hospital
Bergen, Norway

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