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On the Cover

Oxalate nephropathy has many etiologies. Primary hyperoxaluria (PO) is a group of autosomal recessive disorders causing overproduction of oxalate. Primary hyperoxaluria type I accounts for the majority (70–80%) of cases, affecting approximately 1 in 120,000 live births in central Europe. Secondary forms may be due to increased intestinal oxalate absorption, endogenous production or dietary intake. Patients with PO type 1 develop urolithiasis and nephrocalcinosis, which can cause progressive renal damage and chronic kidney disease. Oxalate deposition is also seen in bone, skin, retina, myocardium, blood vessel walls and the central nervous system. The biopsy on the cover is from a 6-month-old male baby evaluated for failure to thrive. His creatinine was 8.1mg/dl and he had 1+ proteinuria. Ultrasound examination revealed bilateral markedly hyperechoic “bright” kidneys. Histologically, calcium oxalate crystals appear colorless on hematoxylin and eosin stain (see cover image) and are fan shaped and radially arranged. Under polarized light the crystals are strongly birefringent (see image below, which is the same area of the biopsy on the cover image). The reaction to the intraparenchymal crystals can lead to fibrosis. A diagnosis of primary hyperoxaluria was made based on the clinical, radiological and histopathological findings. Supportive treatment with adequate hydration, oral potassium citrate and pyridoxine supplementation can improve the prognosis and slow the progression to ESRD. Combined liver and kidney transplantation is the treatment of choice. (*Image and text provided by Anila Kurien, Center for Renal and Urological Pathology, Chennai, Tamil Nadu, India*)

