CJASN
Clinical Journal of the American Society of Nephrology

March 2012 • Vol. 7 • No. 3

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On the Cover

What's the diagnosis? Conventional light microscopy of a urine sample from an untreated patient with adenine phosphoribosyltransferase (APRT) deficiency. The photomicrograph shows typical 2,8-dihydroxyadenine (DHA) crystal aggregates, which are round shaped and brown or yellow colored with a dark outline and central spicules. (Original magnification ×400). Small- and medium-sized crystal aggregates are positively birefringent and produce a central Maltese cross pattern when viewed with polarized light microscopy. APRT deficiency is a rare autosomal recessive disorder of purine metabolism that is estimated to affect 1:50,000–1:100,000 individuals. The absence of APRT enzyme activity prevents recycling of adenine, which is instead converted to DHA by xanthine dehydrogenase. DHA is excreted in the urine where it is poorly soluble, causing recurrent kidney stones and/or chronic kidney disease due to DHA crystalline nephropathy. A significant proportion of patients progress to kidney failure, even in the absence of a history of kidney stone disease. Lack of awareness of this disorder among clinicians and pathologists is a major concern because many patients experience symptomatic disease for years or decades before the correct diagnosis is made. A number of patients with recurrence of DHA crystalline nephropathy in kidney transplants have been reported, usually due to missed diagnosis and lack of proper treatment. Treatment with allopurinol, a xanthine dehydrogenase inhibitor, effectively prevents stone formation and kidney failure in patients with APRT deficiency. (Image and text provided by Vidar O. Edvardsson, MD, and Runolfur Palsson, MD, Landspitali–The National University Hospital of Iceland, Reykjavik)