

CJASN

Clinical Journal of the American Society of Nephrology

May 2016 • Vol. 11 • No. 5

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On the Cover

What's the diagnosis? A 56-year-old man with IgG kappa multiple myeloma presented with AKI. Serum free kappa light chains (LCs) were elevated at 326 mg/dL, the kappa/lambda ratio was 200, and urine kappa LCs were elevated at 239 mg/dL. Urinalysis revealed 1+ protein, and 1+ blood. Urine sediment analysis demonstrated aggregates of various shaped and sized crystals (**cover image, panel insert**), which had 25% birefringence with polarization. Kidney biopsy demonstrated LC crystalline casts and many tubular profiles showed LC crystals within the cytoplasm of the tubular epithelial cells and eosinophilic refractile LC crystals of varying shapes in tubular lumens (**cover image**). Electron microscopy revealed multifaceted crystals in the tubular lumens and within the cytoplasm of tubular epithelial cells (**not shown**). These findings were consistent with monoclonal LC-induced crystalline nephropathy as the cause of kidney injury. A 69-year-old man presented with an elevated creatinine of 2.3 mg/dL. Urinalysis revealed 1+ protein, 1+ glucose, pH 6.0 with protein/creatinine ratio 0.73 g/g. Urine microscopy showed casts that contained rhomboidal structures (**left image below**). Serum protein electrophoresis revealed an IgA kappa monoclonal component of 0.46 g/dL, and 6.1% of total protein. Kappa/lambda LC ratio was 314. Kidney biopsy demonstrated diffusely swollen proximal tubules packed with brightly eosinophilic granules. On ultrastructural exam, tubular epithelial cells were swollen due to intracytoplasmic accumulation of electron dense, membrane-bound, rhomboid or needle-shaped crystals some of which exhibited an organized structure (**right image below**). Bone marrow biopsy confirmed multiple myeloma with LC crystal tubulopathy. Monoclonal LCs are a known cause of various forms of kidney injury including cast nephropathy, amyloidosis, LC deposition disease and LC tubulopathy. Electron dense crystalline structures can be seen within the proximal tubular cell cytoplasm, which manifests as a proximal tubulopathy and sometimes a full-blown Fanconi syndrome. Less commonly recognized with these monoclonal LC diseases is the finding of light chain crystals, both free and within casts, in the spun urine sediment. The presence of these same LC crystals within the tubular cell cytoplasm (both cases) as well as within renal tubular lumina (first case) on kidney biopsy confirmed the urinary sediment findings as LC crystals in these 2 cases. Thus, part of the evaluation of patients suspected of a paraprotein-related disease should include thorough evaluation of the spun urine sediment. (Images and text provided by Mark A. Perazella, MD, Yale University School of Medicine, New Haven, Connecticut, and Zita Shiue, MD and Ian deBoer, MD, Division of Nephrology, University of Washington and Roberto Nicosia, MD, Department of Pathology, University of Washington)

