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What's the diagnosis? Renal biopsy reveals many tubules which appear to be obstructed by abundant clear needle-shaped crystals, some of which have dissolved with tissue processing. The crystals are surrounded by intra-tubular macrophages and multinucleated giant cells. There is evidence of acute tubular injury, interstitial chronic inflammation, and tubulointerstitial scarring. The patient presented with sterile pyuria and acute kidney injury; the biopsy is diagnostic of a "crystal nephropathy". In this case, the etiology of crystal nephropathy is the protease inhibitor, indinavir. Crystal deposition within tubular lumina of the kidney can cause both acute and chronic kidney injury. While a number of endogenous processes are associated with crystal-induced kidney injury (tumor lysis syndrome with acute uric acid nephropathy, gastric bypass with acute oxalate nephropathy), several medications can cause this clinical renal syndrome. Common examples include agents such as acyclovir, sulfadiazine, ciprofloxacin, methotrexate, indinavir, atazanavir, sodium phosphate bowel purgatives, ascorbic acid, ethylene glycol, and triamterene. With these agents, either the drug or its metabolites crystallize within the renal tubules, often due to one or more factors such as inherent drug insolubility, urine pH (high or low), sluggish urine flow rates (increased urinary drug concentration), and underlying kidney disease. For example, ciprofloxacin, indinavir and atazanavir can precipitate in tubular lumens in alkaline urine. In contrast, drugs such as sulfadiazine and methotrexate (and their metabolites) are less soluble in acid urine and more likely to precipitate in the kidney at pH < 6.0. Drugs such as orlistat can induce malabsorption with enteric hyperoxaluria and acute oxalate nephropathy. Clinically, patients may develop asymptomatic crystalluria, crystal-induced AKI, or nephrolithiasis. Prevention and therapy are directed at improving urinary flow rates, stopping or dose reducing the culprit drug, and altering urine pH (when feasible) to enhance drug/metabolite solubility. Most patients recover kidney function with drug discontinuation; however, some patients develop chronic kidney disease. (Image and text provided by Mark A. Perazella, Yale University School of Medicine; and Glen S. Markowitz, Columbia University College of Physicians and Surgeons)