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**On the Cover**

*What's the diagnosis?* A 51-year-old woman with a longstanding history of alcohol abuse and end stage liver disease presented with increased abdominal girth and pain as well as lower extremity swelling. She was taking 1-2 ibuprofen tablets (200 mg) per day for the past 10 days to 2 weeks for low back pain. Vitals signs revealed: BP- 86/44mmHg, P- 80/min, RR- 24/min, and T- 100 degrees F. Exam was notable for a distended abdomen with rebound tenderness and 2+ pitting edema to knees. Labs revealed: AST 30 IU/L, ALT 48 IU/L, alkaline phosphatase 245 IU/L, and total bilirubin 29 mg/dl. Serum creatinine was 3.1 mg/dl. Urine sodium was 16 mEq/L. Urine microscopy revealed free bilirubin crystals (upper left panel) and bilirubin crystals within a renal tubular epithelial cell (upper right panel) as well as numerous bile stained renal tubular epithelial cell (RTEC) casts (lower panel). The patient had spontaneous bacterial peritonitis with E coli, which was successfully treated with intravenous ceftriaxone and albumin. Acute kidney injury (AKI) was thought due to acute tubular injury in the setting of SBP and ibuprofen use. The patient’s kidney function began to improve on day 8 of hospitalization and kidney function returned to baseline (serum creatinine 0.8 mg/dl). The finding of bilirubin crystals within the urine is simply a reflection of hyperbilirubinemia. They are needle-like crystals that can be seen alone are but more often clumped together as seen in the upper left panel. Sometimes, these crystals are engulfed by renal tubular epithelial cells or macrophages (upper right panel). The finding of numerous bile-stained RTEC casts within the urine of a cirrhotic with AKI reflects tubular injury and strongly suggests acute tubular injury (rather than prerenal AKI or hepatorenal syndrome). However, this is not a perfect test as small numbers of RTEC casts and granular casts can be seen in patients with hepatorenal syndrome (HRS). In this case, the combination of numerous RTEC casts, relatively high urine sodium, and the clinical AKI course are consistent with acute tubular injury / necrosis rather than prerenal AKI or HRS. *Image and text provided by Jose Antonio Tesser Poloni, Irmunda de Santa Casa de Misericordia de Porto Alegre, Porto Alegre, Brazil, and Mark A. Perazella, Yale University School of Medicine*