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What is the diagnosis? A 48-year-old woman presented to the Emergency Department with altered mental status. Laboratory work revealed: sodium of 138 mEq/L, potassium 5.1 mEq/L, chloride 102 mEq/L, bicarbonate of < 5 mEq/L, blood urea nitrogen (BUN) 28 mg/dL, and serum creatinine of 2.3 mg/dL. Serum osmolar gap was calculated at 52 mOsm. Arterial blood gas confirmed severe acidemia and metabolic acidosis. 4-methylpyrazole (fomepizole) was administered for presumed toxic alcohol ingestion and hemodialysis was initiated. Urine sediment examination revealed numerous monohydrated calcium oxalate crystals (left picture), which were birefringent with polarization, raising concern for ethylene glycol intoxication. Ethylene glycol level subsequently returned at 302 mg/dL. Kidney biopsy was undertaken after the patient remained dialysis dependent for 3 weeks. The biopsy demonstrated significant calcium oxalate crystal deposition within tubules (middle picture) that were positively birefringent (right picture). These findings were consistent with acute calcium oxalate nephropathy from ethylene glycol ingestion. Ingestion of ethylene glycol is classically complicated by an increased osmolar gap and anion gap metabolic acidosis as well as multiple organ dysfunction from glycolic acid and oxalic acid. Acute kidney injury is primarily the result of calcium oxalate deposition within the renal tubules, which leads to tubular obstruction and an inflammatory response within the renal interstitium. Visualization of calcium oxalate crystals at the time of the presentation can help confirm ethylene glycol intoxication. *(Images and text provided by Mark A. Perazella, MD, Yale University School of Medicine, New Haven, Connecticut)*