



Antibiotic Associated Kidney Injury in a Roux-en-Y Gastric Bypass Surgery Patient

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Introduction

Oxalate nephropathy has a devastating effect on renal function. Kidney is the main target for oxalate deposition causing oxalate nephropathy and kidney failure. Precipitation of calcium oxalate in renal tubules and interstitium leads to direct tubular injury and atrophy, and excites chronic interstitial inflammation leading to fibrosis, and ultimately nephron loss resulting in decrease in renal function. Under normal conditions, oxalate is rendered relatively insoluble by binding to calcium in the colon, forming a calcium oxalate complex. Causes of secondary oxalate nephropathy are increased intake of oxalate precursor, decreased degradation of oxalate in the intestine, increased permeability of oxalate in the colon. After Roux-en-Y gastric bypass surgery, there is increased bioavailability of oxalate in the colon due to decreased intestinal calcium availability from fat malabsorption.

Case Presentation

This is a case of ATN from oxalate nephropathy secondary to antibiotic use for osteomyelitis in a patient with history of gastric bypass surgery. A 73-year-old Caucasian female with HTN, hx of Roux-en-Y gastric bypass surgery, hx of right foot osteomyelitis treated with Vancomycin and Unasyn for proteus mirabilis presented with worsening renal function. She finished her antibiotic course for osteomyelitis and was found to have creatinine of 3.1 post antibiotic treatment.

Case Presentation

Kidney biopsy revealed ATN with many calcium oxalate crystals and tubular atrophy and interstitial fibrosis. Patient underwent hemodialysis. However, her creatinine did not go back to her baseline of 0.6-0.8.

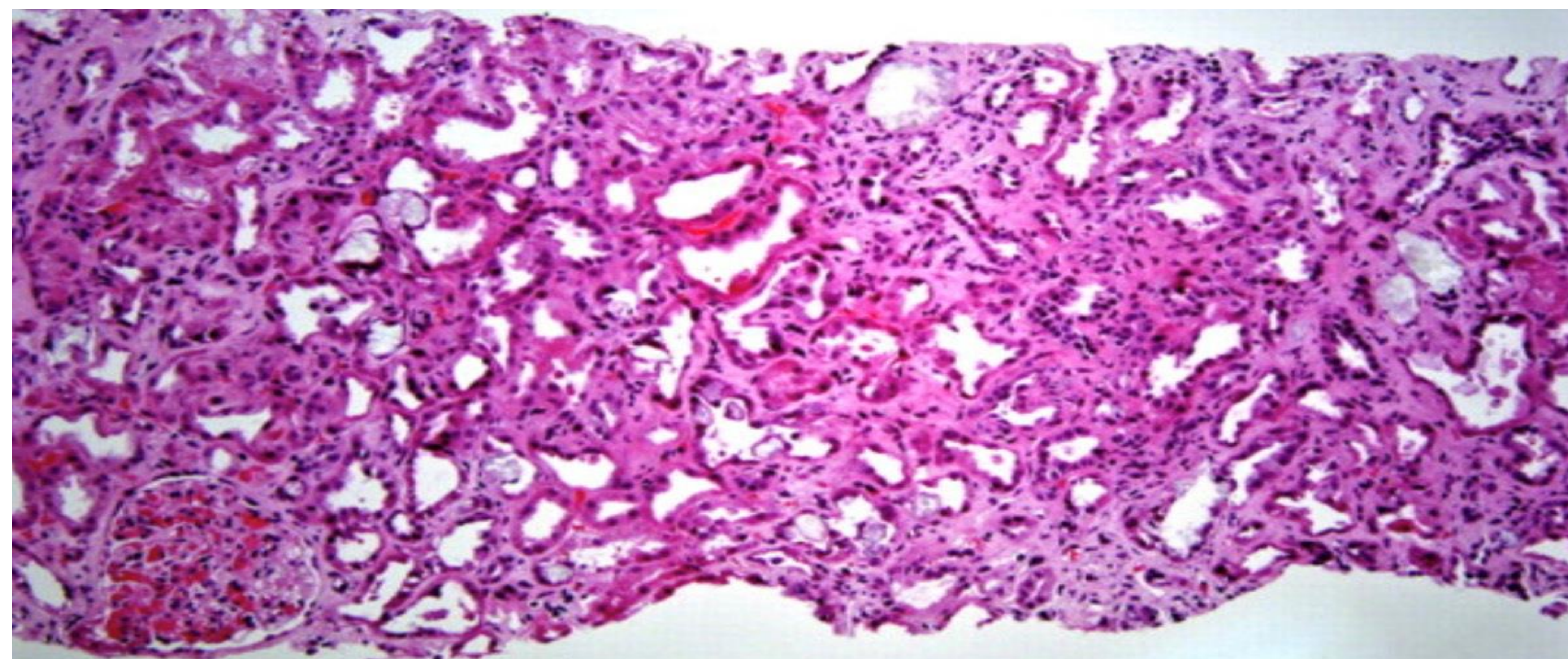


Figure 1: calcium oxalate deposits in the tubules resulting in degenerative changes. (source: cjasn)

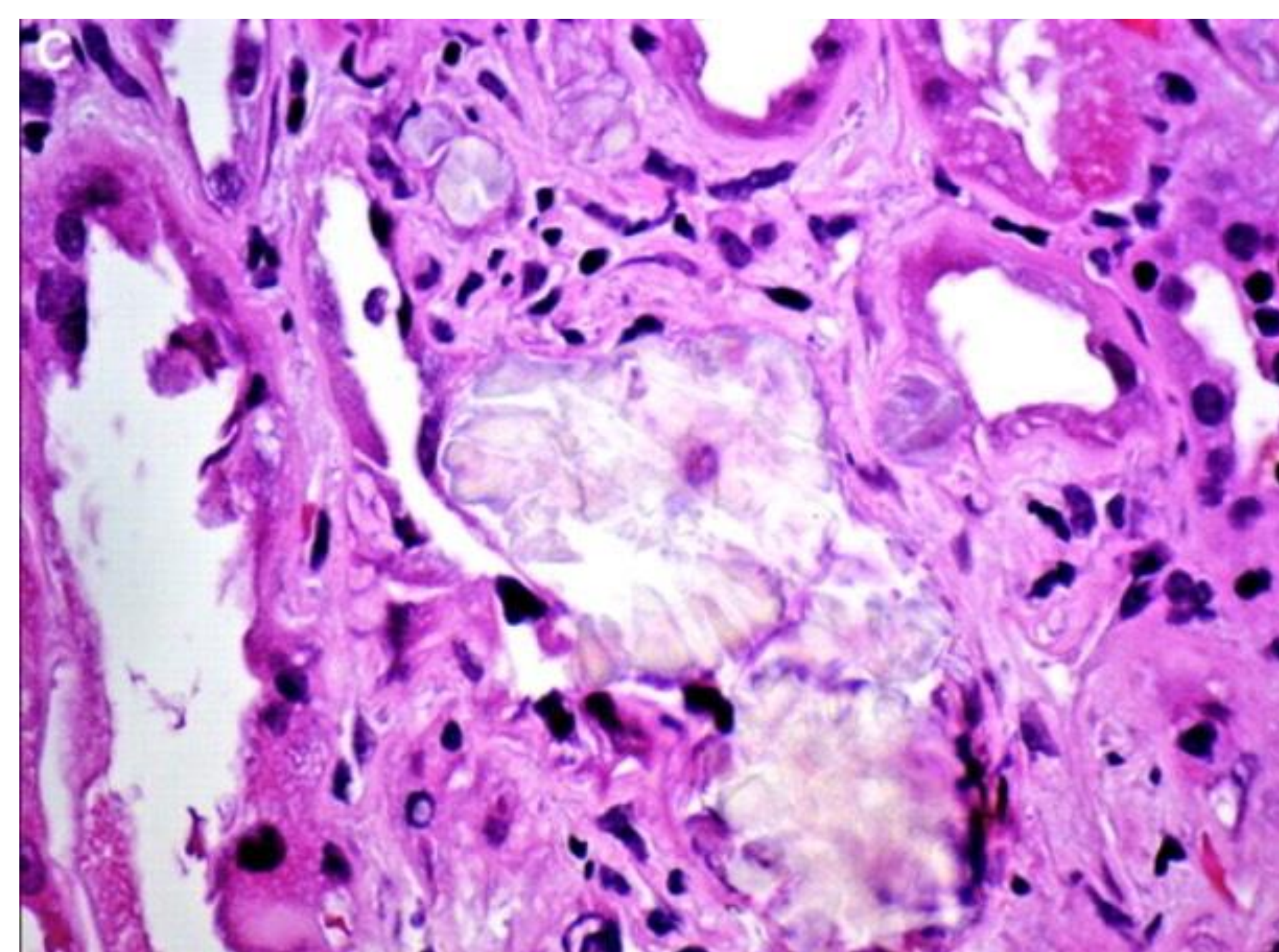


Figure 2: Intraluminal calcium oxalate crystals (Source: cjasn)



Figure 3: Calcium oxalate crystals under polarized light (Source: cjasn)

Discussion

Secondary oxalate nephropathy is a devastating condition that might require renal replacement therapy. Like our patient, most of them remain dialysis dependent. Patients are at increased risk if multiple etiologies for the hyperoxaluria enabling conditions are involved. Most likely etiology for the oxalate nephropathy is the antibiotic use in this patient, which caused decreased rate of colonization of *Oxalobacter Formigenes* resulting in the increased colonic absorption of oxalate and favored the development of calcium oxalate crystals. This patient's history of of Roux-en-Y gastric bypass surgery itself is a risk factor of oxalate nephropathy and exacerbated the formation of calcium oxalate stones. Further researches are required for effective preventative measures for patients who are at high risk for oxalate nephropathy.

References

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