Oxalate Nephropathy after Bariatric Surgery: Incidence and Case Report of Successful Kidney Transplant after Conversion from Roux-en-Y Gastric Bypass to Gastric Sleeve

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Abstract

Oxalate nephropathy is characterized by tubular crystalline deposits of calcium oxalate leading to acute and chronic tubular injury, interstitial fibrosis, and progressive renal insufficiency. Conditions characterized by fat malabsorption can result in binding of fatty acids to calcium, leading to hyperabsorption of oxalate and sometimes kidney injury since calcium normally binds to oxalate. Oxalate nephropathy has been reported after bariatric surgery although the incidence is unclear. Reported cases have been in the setting of malabsorptive bariatric surgeries such as jejunoileal bypass, and to a lesser extent, Roux-en-Y gastric bypass (RYGB). No cases in the literature have been reported after gastric sleeve. Out of 4,554 patients in our bariatric surgery research registry, we identified 2 cases of biopsy-confirmed oxalate nephropathy in patients who underwent RYGB surgery. We report a unique case of a patient who had oxalate nephropathy associated with RYGB, then underwent reversal of RYGB and conversion to gastric sleeve, and eventually received a kidney transplant.

Introduction

Oxalate is naturally found in certain foods and is also a metabolic end-product that is excreted in the urine after absorption in the gastrointestinal tract. In most individuals, the impact of dietary oxalate is on urinary oxalate excretion is small (1). However, in the presence of fat malabsorption, excessive oxalate absorption may increase the risk for oxalate nephropathy (2). Oxalate nephropathy is characterized by tubular crystalline deposits of calcium oxalate leading to acute and chronic tubular injury, interstitial fibrosis, and progressive renal insufficiency (3). In the normal state, calcium and oxalate within the lumen of the intestine combine to form insoluble calcium oxalate complexes that are excreted in feces. In the setting of fat malabsorption and enteric hyperoxaluria, excessive intraluminal free fatty acids bind to and saponify calcium within the intestine, thereby inhibiting the formation of calcium oxalate. As a result, greater quantities of soluble free oxalate are absorbed by the colonic mucosa, which can lead to kidney injury.

Initial cases were reported after jejunoileal bypass surgery, a purely malabsorptive surgery that is no longer performed due to the high incidence of oxalosis (4). More recently, cases of oxalate nephropathy have been reported after malabsorptive bariatric surgery Roux-en-Y gastric bypass (RYGB), although the incidence is unclear (5). In contrast, no case reports of oxalate nephropathy after restrictive bariatric surgery procedures such as gastric sleeve or gastric banding have been reported. Proximal RYGB is often referred to as a combination restriction–malabsorption procedure. It involves stapling of the stomach to create a small (≤30.0 ml) upper gastric pouch. The small intestine is then divided at the mid jejunum, and the distal portion (Roux limb) is anastomosed to the gastric pouch.

Incidence of oxalate nephropathy

We examined data from 4,554 patients in Geisinger's bariatric surgery research registry to determine the incidence of biopsy-confirmed oxalate nephropathy after bariatric surgery. We employed the following strategy. First, we identified 123 patients who had either "oxalate nephropathy" in EHR progress notes using a text mining tool, a diagnosis of ESKD, or eGFR <15 ml/min/1.73m2. Second, we searched our kidney biopsy pathology databases for "oxalate nephropathy" and did not identify any additional patients in the bariatric surgery research registry. Lastly, chart review on the 123 patients meeting the above criteria were reviewed by an attending nephrologist to determine if patients had evidence of a kidney biopsy or biopsy-confirmed oxalate nephropathy. Out of a total of 3 patients in the bariatric surgery cohort who had kidney biopsy data, 2 patients had biopsy-confirmed oxalate nephropathy.

Case Presentation

Case presentation of a patient who had oxalate nephropathy associated with RYGB, then underwent reversal of RYGB and conversion to gastric sleeve, and eventually received a kidney transplant.

A 58-year-old female with hypertension, Type 2 diabetes, chronic kidney disease stage 3, underwent RYGB (150 cm or less) for morbid obesity in 2012. She had a long-standing history of diabetes (2003) requiring insulin (2008), diabetic retinopathy, diabetic neuropathy, and proteinuria. At the time of surgery, she had stage 4 chronic kidney disease.

About 11 months after RYGB, she was hospitalized for acute kidney injury, which was initially thought to be due to dehydration. She received intravenous fluid resuscitation. However, her kidney function continued to worsen. Other workup was negative, including complement C4, anti-nuclear antibody, anti-neutrophil cytoplasmic antibody, cryoglobulin panel, rheumatic fever, hepatitis profile, and renal panel. A kidney biopsy was performed, demonstrating oxalate

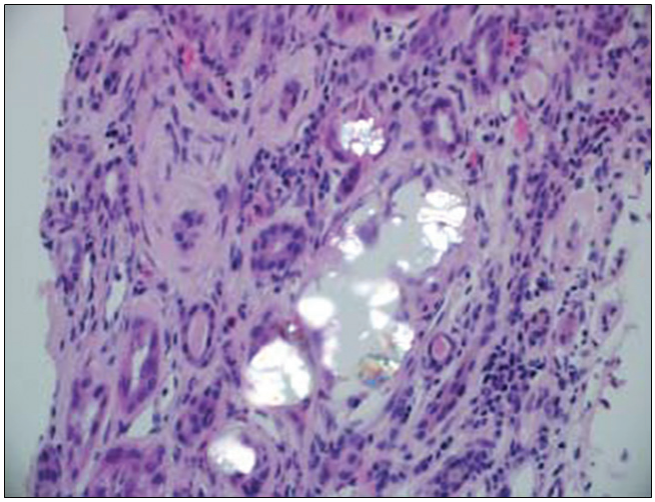

Figure 1. Left kidney, renal biopsy confirmed renal oxalosis with significant tubulointerstitial disease and interstitial fibrosis. Microscopic examination of the tubules demonstrates large crystalline deposits in tubular lumina with reactive tubular epithelial changes.

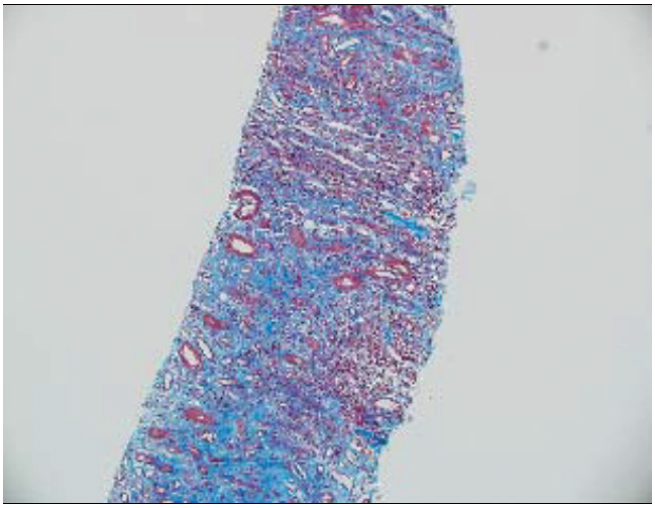

Figure 2. Kidney biopsy, trichrome stain. Examination of the interstitium with trichrome stain shows moderate interstitial fibrosis of 26 to 50% of cortical area, mild edema and interstitial inflammation of 26% to 50% of the cortical parenchyma consisting predominantly of mononuclear inflammatory cells and a remarkable number (>10% of inflammation) of eosinophils and neutrophils.

nephropathy (Figure 1). In an attempt to reverse the acute kidney injury, maintain weight loss to preserve candidacy for the kidney transplant list, the patient had laparoscopic reversal of RYGB and conversion to gastric sleeve approximately 4 months later. Despite the operation, her kidney function did not recover, and she went on to develop end-stage kidney disease and started continuous cyclic peritoneal dialysis. She eventually received a deceased donor kidney transplant. No evidence of oxalate nephropathy was seen on transplant kidney biopsies that were done for delayed graft function (Figure 2 and Figure 3).

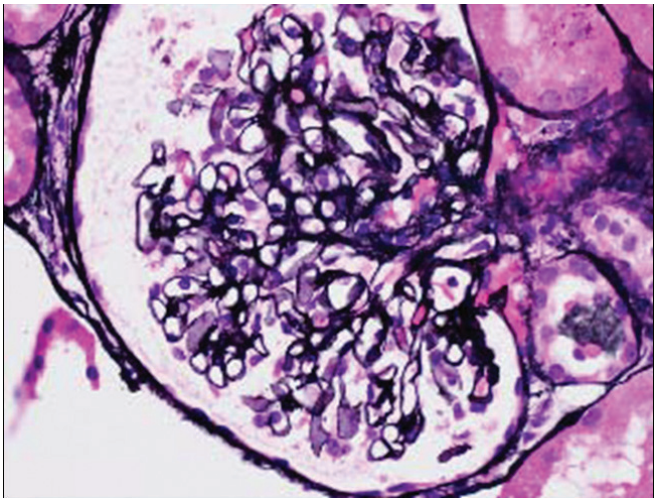

Figure 3. Allograft kidney, needle biopsy, light microscopy. There was no evidence of oxalate nephropathy on transplant kidney biopsy.

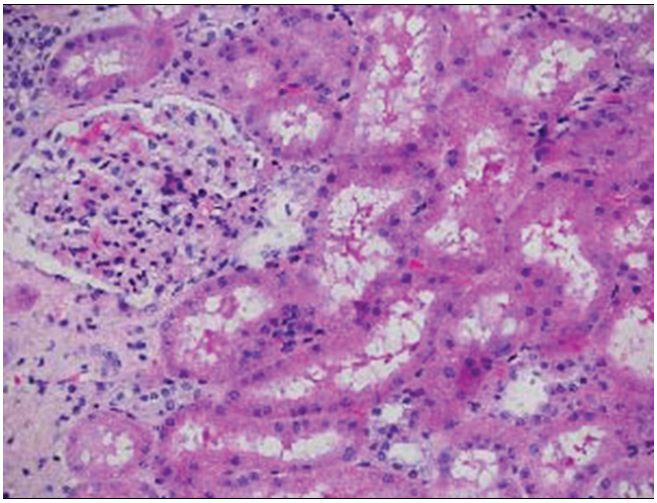

Figure 4. Allograft kidney, needle biopsy, light microscopy. There was no evidence of oxalate nephropathy on transplant kidney biopsy.

Discussion

Medical management of oxalate nephropathy after RYGB focuses on lowering intake of dietary oxalate and fat and measures to bind oxalate such as calcium supplementation (3). However, there is little evidence demonstrating whether interventions to reduce enteric hyperoxaluria after bariatric surgery can stabilize kidney function. Unlike a prior case report, (6) her chronic kidney disease continued to progress despite reversal of the RYGB and conversion to gastric sleeve (Figure 4). In our patient's case, the continued progression may have been a result of her underlying diabetic kidney disease and significant renal fibrosis seen on kidney biopsy (Figure 5).

However, she was able to maintain her weight so that she was able to qualify for eligibility on the transplant waitlist and eventually receive a kidney transplant. Interestingly, on kidney biopsies of her transplanted kidney, no oxalate

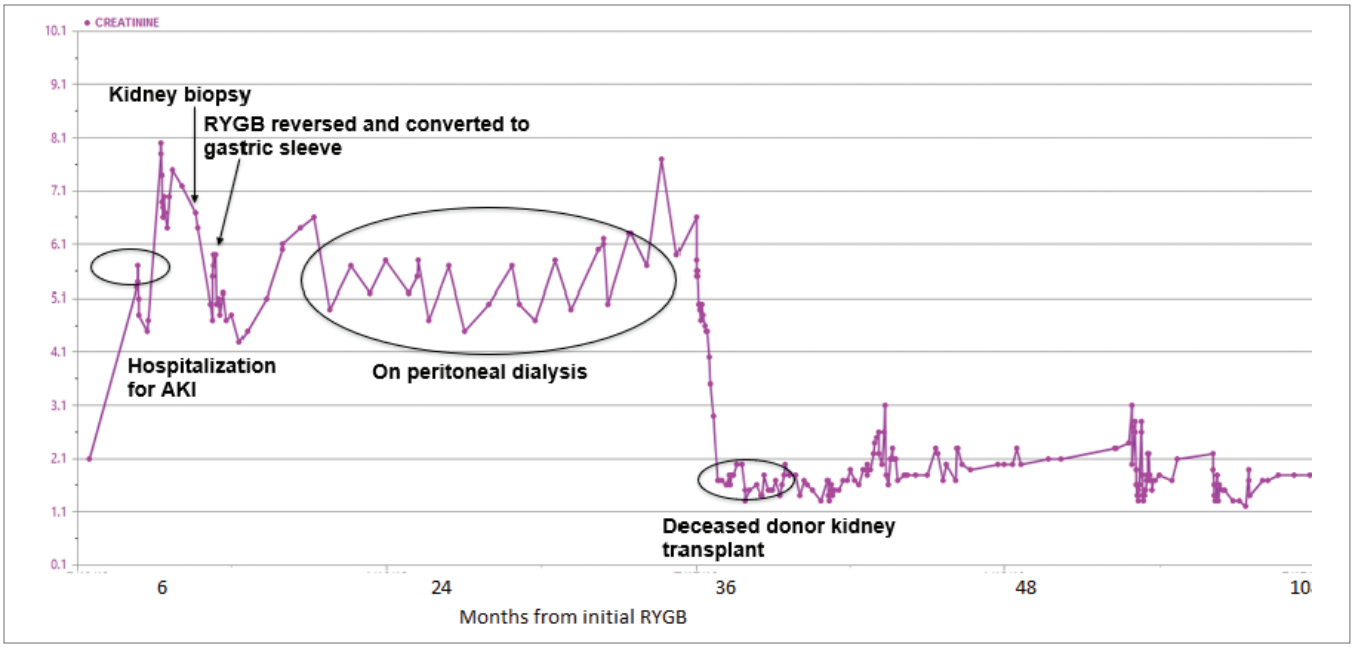

Figure 5. Changes in serum creatinine (mg/dL) in the patient

crystals were observed. There are some limitations to note. First, kidney biopsy data was only available on 3 patients after bariatric surgery, and it is possible that oxalate nephropathy is underrecognized. In addition, we had incomplete data available on 24-hour urine oxalate levels post-RYGB surgery when kidney function was worsening.

Conclusion

In conclusion, we identified 2 cases of biopsy-confirmed oxalate nephropathy in a cohort of 4,554 bariatric surgery patients. However, additional studies are needed to determine the exact incidence of oxalate nephropathy since kidney biopsies and 24-hour urine oxalate are not completed routinely after bariatric surgery. Prompt measurement of 24-hour urine oxalate levels and kidney biopsy should be considered in the event of unexplained acute kidney injury or chronic kidney disease progression in patients post-RYGB surgery. Conversion to gastric sleeve surgery to maintain weight loss after oxalate nephropathy may be helpful for patients with oxalate nephropathy to preserve kidney transplant candidacy.

Acknowledgments

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