Oxalate Nephropathy Complicating Roux-en-Y Gastric Bypass: An Underrecognized Cause of Irreversible Renal Failure

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Background and objectives: The most common bariatric surgery is Roux-en-Y gastric bypass (RYGB), which has been associated with hyperoxaluria and nephrolithiasis. We report a novel association of RYGB with renal insufficiency as a result of oxalate nephropathy.

Design, setting, participants, & measurements: Eleven cases of oxalate nephropathy after RYGB were identified from the Renal Pathology Laboratory of Columbia University. The clinical features, pathologic findings, and outcomes are described.

Results: Patients were predominantly white (72.7%) with a mean age of 61.3 yr. Indications for RYGB included morbid obesity (eight patients) and reconstruction after total gastrectomy for gastric cancer (three patients). All 11 patients had a history of hypertension, and 9 were diabetic. Patients presented with acute renal failure, often superimposed on mild chronic renal insufficiency (\(n = 11\)), at a median of 12 mo after RYGB. The mean creatinine at baseline, at discovery of acute renal failure, and at biopsy was 1.5, 5.0, and 6.5 mg/dl, respectively. Renal biopsies revealed diffuse tubular degenerative changes, abundant tubular calcium oxalate deposits, and varying degrees of tubulointerstitial scarring. In addition, seven biopsies had underlying diabetic glomerulosclerosis and two had glomerulosclerosis attributable to obesity and hypertension. Eight of 11 patients rapidly progressed to ESRD and required hemodialysis at a mean of 3.2 wk after renal biopsy. The remaining three patients were left with significant chronic kidney disease.

Conclusions: Oxalate nephropathy is an underrecognized complication of RYGB and typically results in rapid progression to ESRD. Patients with pre-existing renal disease may be at higher risk for this complication.


Oxalate nephropathy (oxalosis) is characterized by tubular crystalline deposits of calcium oxalate leading to acute and chronic tubular injury, interstitial fibrosis, and progressive renal insufficiency. Established causes of oxalate nephropathy include primary (hereditary) hyperoxaluria; ethylene glycol (antifreeze) intoxication; enteric hyperoxaluria; exposure to the anesthetic agent methoxyflurane; pyridoxine (vitamin B6) deficiency; and excessive ingestion of vitamin C or dietary substances rich in oxalic acid, such as rhubarb, cocoa, parsley, and nuts.

Enteric hyperoxaluria, the most common cause of moderate hyperoxaluria, occurs in conditions that are associated with fat or bile acid malabsorption, such as inflammatory bowel disease, pancreatic insufficiency, bowel resection, blind loop syndrome, and jejunoileal (JI) bypass. Fat malabsorption leading to steatorrhea plays a central role in all forms of enteric hyperoxaluria (1,2). 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 (1). In addition, free fatty acids and bile salts enhance the colonic mucosa’s permeability to oxalate (3).

Nephrolithiasis and oxalate nephropathy are widely recognized complications of JI bypass, one of the first surgical approaches to morbid obesity (4–6). Mole et al. (4) described eight cases of renal failure after JI bypass and reviewed 18 previously reported cases. Histologic evaluation was available for 17 of the 26 patients and revealed findings of tubular calcium oxalate crystals, frequently accompanied by “chronic interstitial nephritis.” Urine oxalate levels, available for 11 patients, were elevated (4). These results are not surprising given that hyperabsorption of dietary oxalate (assessed by increased urinary excretion of \(^{14}\)C oxalate given orally) and marked hyperoxaluria were known to occur after JI bypass (7). As a result of the high rate of serious complications, including liver failure, electrolyte imbalances, diarrhea, nephrolithiasis, oxalate nephrop-
athy, and death, JI bypass was largely abandoned in 1979 (6). Several newer bariatric operations have been introduced over the years, including Roux-en-Y gastric bypass (RYGB) surgery, biliopancreatic diversion with duodenal switch, vertical banded gastroplasty, and laparoscopic adjustable gastric banding.

RYGB is currently the most common bariatric surgery in the United States because of its high success rate in weight reduction (8). In one study with >5 yr of follow-up, successful outcomes (weight reduction to body mass index [BMI] ≤35 kg/m² or loss of excess weight >50%) were achieved in 93% of obese (BMI ≥36 kg/m²) or morbidly obese (BMI 40 to 49 kg/m²) patients and in 57% of superobese (BMI >50 kg/m²) patients (9). Recent data indicate that RYGB is a potential risk factor for hyperoxaluria and nephrolithiasis (10–13). Asplin and Coe (10) found that stone formers who had undergone modern bariatric surgery had a higher mean urine oxalate excretion (83 mg/d) than routine stone formers (39 mg/d) and normal individuals (34 mg/d) but lower than stone formers who underwent JI bypass (102 mg/d). Sinha et al. (11) recently reported 60 patients who were seen at Mayo Clinic-Rochester and developed nephrolithiasis after RYGB. Hyperoxaluria was present in 17 of 31 patients for whom metabolic analysis was performed. Importantly, two of the 60 patients presented with renal failure and received a diagnosis of oxalate nephropathy by renal biopsy. Both patients progressed to ESRD and had high plasma oxalate levels (12,13). No other cases of oxalate nephropathy after RYGB have been reported in the literature.

Herein, we describe 11 cases of oxalate nephropathy after RYGB. RYGB was performed for treatment of morbid obesity in eight patients and for reconstruction (Roux-en-Y esophagojejunostomy) after total gastrectomy for gastric cancer in the remaining three patients. The demographic, clinical, pathologic, and outcome data are detailed.

Materials and Methods

We identified 11 cases of oxalate nephropathy after RYGB surgery from the archives of the Renal Pathology Laboratory of Columbia University. All 11 biopsies were received during a 6-yr period from 2002 through 2007 and showed acute and chronic tubulointerstitial injury accompanied by abundant tubular oxalate crystals. All 11 renal biopsies were processed according to standard techniques for light microscopy (LM), immunofluorescence (IF), and electron microscopy (EM). For each case, 11 glass slides were prepared and stained with hematoxylin and eosin, periodic acid-Schiff, trichrome, and Jones methenamine silver. IF was performed on 3-μm cryostat sections using polyclonal FITC-conjugated antibodies to IgG, IgM, IgA, C3, C1q, κ, λ, fibrinogen, and albumin (Dako Corp., Carpinteria, CA). Ultrastructural evaluation was performed using a JEOL 100S or 1010 electron microscope (Tokyo, Japan).

Patients’ medical charts were reviewed for demographics, medical history, indication for RYGB surgery, interval from RYGB to renal impairment, parameters of renal function, treatment, and outcome. The following clinical definitions were applied: Obesity, BMI >30 kg/m²; morbid obesity, BMI >40 kg/m²; acute renal failure (ARF), doubling of the serum creatinine; renal insufficiency, serum creatinine >1.2 mg/dl; leukocyturia, >5 white blood cells per high-power field on microscopic examination of the urinary sediment; and hematuria, >5 red blood cells per high-power field on microscopic examination of the urinary sediment. Hypertension was defined as a systolic BP >140 mmHg, diastolic BP >90 mmHg, or use of antihypertensive medications. None of the patients had a history of excessive consumption of vitamin C, chronic pancreatitis, inflammatory bowel disease, or familial renal disease. Individual dietary histories were not available to evaluate dietary oxalate content.

The number of tubules that contained intraluminal or intracellular calcium oxalate deposits was recorded for each biopsy. Tubular atrophy and interstitial fibrosis and interstitial inflammation were graded on a semiquantitative scale on the basis of the percentage of renal cortex affected and recorded as 1 to 25% (mild), 26 to 50% (moderate), or >50% (severe). The degree of diabetic glomerulosclerosis (DGS) was graded as mild, moderate, or severe on the basis of the degree of mesangial sclerosis. The study was approved by the institutional review board of Columbia University Medical Center.

Results

Clinical Features

The clinical features are summarized in Table 1. The cohort consisted of six women and five men with a mean age of 61.3 yr (range 45 to 79 yr). Eight (72.7%) patients were white, and the remaining three (27.3%) were black. The indication for RYGB was morbid obesity for eight (72.7%) patients and gastric adenocarcinoma for three (27.3%) patients. The mean pre-RYGB BMI for patients who underwent bariatric surgery was available for six of eight patients and was 55.8 kg/m². After surgery, all patients achieved significant weight loss with a mean BMI of 33.2 kg/m² at the time of renal biopsy. For all eight patients, RYGB was successful based on a definition of loss of >50% of excess weight. The three patients (patients 9 through 11) with gastric adenocarcinoma underwent total gastrectomy and Roux-en-Y esophageal jejunostomy. Six patients had documented histories of chronic diarrhea after RYGB. Urine and serum oxalate levels at time of biopsy were available for only two patients (patients 5 and 7) and one patient (patient 11), respectively, and were markedly elevated. Oxalate crystals were noted in the urine of two patients (patients 5 and 8), and three patients (patients 5, 9, and 10) also developed post-RYGB nephrolithiasis. Nine (81.8%) of the 11 patients had longstanding diabetes with a mean duration of 16 yr, including three who were known to have diabetic retinopathy. All 11 patients had a history of hypertension. At the time of biopsy, three of 10 patients with available data were receiving a diuretic (furosemide), two patients an angiotensin-converting enzyme inhibitor, and one patient an angiotensin receptor blocker.

Patients presented with ARF, often superimposed on chronic renal insufficiency (Table 1). The mean and median times from surgery to discovery of ARF were 33 and 12 mo, respectively (range 4 to 96 mo). The mean baseline serum creatinine for the 10 patients with available data was 1.5 mg/dl (range 0.9 to 2.5 mg/dl). The baseline serum creatinine was obtained at the time of surgery for six patients and at 13, 36, 92, and 6 mo after surgery for the remaining four patients (patients 4, 5, 8, and 11). Baseline serum creatinine was elevated in seven patients (patients 3, 4, 5, 7, 8, 10, and 11), five of whom had evidence of DGS. Mean serum creatinine at time of discovery of ARF and at time of biopsy was 5.0 mg/dl (range 2.4 to 9.2 mg/dl) and 6.5 mg/dl (range 2.4 to 10.6 mg/dl), respectively.
**Table 1. Clinical data**

<table>
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<tr>
<th>Patient</th>
<th>Age</th>
<th>Gender</th>
<th>Race</th>
<th>Indication for Surgery</th>
<th>Diabetes (Duration; yr)</th>
<th>BMI at Time of Surgery</th>
<th>BMI at Time of RB</th>
<th>Baseline Cr (mg/dl)</th>
<th>Time from Surgery to ARF (mo)</th>
<th>Time from ARF to RB (mo)</th>
<th>Cr upon Discovery of ARF (mg/dl)</th>
<th>Cr at Time of RB (mg/dl)</th>
<th>24-H Urine Protein (g)</th>
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<td>F</td>
<td>W</td>
<td>Morbid obesity</td>
<td>Yes (10)</td>
<td>41.4</td>
<td>34.2</td>
<td>1.2 at RYGB</td>
<td>9.00</td>
<td>3.00</td>
<td>4.0</td>
<td>6.5</td>
<td>1.00</td>
</tr>
<tr>
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<td>Yes (20)</td>
<td>57.0</td>
<td>36.6</td>
<td>NA</td>
<td>59.75</td>
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<td>31.7</td>
<td>1.4 at RYGB</td>
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<td>26.7</td>
<td>1.5 at 13 mo after RYGB</td>
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<td>41.3</td>
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<td></td>
<td>6.00</td>
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<td>3+ on UA</td>
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<td>79</td>
<td>M</td>
<td>W</td>
<td>Gastric adenocarcinoma</td>
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<td>2.5 at RYGB</td>
<td></td>
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<tr>
<td>11</td>
<td>57</td>
<td>F</td>
<td>W</td>
<td>Gastric adenocarcinoma</td>
<td>Yes (10)</td>
<td>1.6 at 6 mo after RYGB</td>
<td></td>
<td></td>
<td>12.00</td>
<td>2.00</td>
<td>4.5</td>
<td>7.5</td>
<td>1.90</td>
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<sup>a</sup>ARF, acute renal failure; B, black; BMI, body mass index (kg/m²); Cr, serum creatinine; F, female; M, male; NA, not available; R Bx, renal biopsy; RYGB, Roux-en-Y gastric bypass; W, white; UA, urinalysis.

<sup>b</sup>Longstanding.

<sup>c</sup>1.6 mg/dl at 86 mo after RYGB.
mg/dl (range 3.4 to 10.0 mg/dl), respectively. The mean time from discovery of ARF to renal biopsy was 1.5 mo (range 0.0 to 5.0 mo) and was ≤1 wk for five patients. Patient 11 was on dialysis for 1 mo at the time of biopsy, whereas patient 5 had received dialysis for 1 mo before biopsy but had discontinued dialysis at the time of biopsy. Proteinuria was documented for all 10 patients with available data. The mean 24-h urine protein collection, available for eight patients, was 1.40 g/d (range 0.37 to 6.00 g/d). Only one patient (patient 4) had proteinuria >2 g/24 h. Six (54.5%) patients had leukocyturia, and three (27.3%) patients had microhematuria, although none had evidence of cellular casts in the urine sediment.

Post-biopsy clinical follow-up was available for all 11 patients (Table 2), with a mean and median follow-up of 19.4 and 11.0 mo, respectively (range 2.5 to 58.0 mo). Seven (63.6%) patients were instructed to follow low-fat, low-oxalate diets, including four patients who were also instructed to take calcium supplements with meals to bind dietary oxalate in the intestinal tract. The remaining four patients did not receive treatment for enteric hyperoxaluria, and no patient underwent revision of RYGB. Within the period of follow-up, eight (72.7%) patients, including the single patient who was on dialysis at time of biopsy (patient 11), rapidly progressed to ESRD at a mean of 3.2 wk (range 0.0 to 12.0 wk). Four of the eight patients subsequently died, one from septicemia and three from undetermined causes. Among the remaining three patients, one had a decline in creatinine from 3.5 to 2.0 mg/dl over 22 mo (patient 5), one had a decline in creatinine from 3.4 to 3.0 mg/dl over 11 mo (patient 7), and one had a minimal increase in creatinine from 4.2 to 4.4 mg/dl over 2.5 mo (patient 8).

Pathologic Findings
Renal biopsy findings for patients with oxalate nephropathy after RYGB are summarized in Table 3. Sampling for LM included a mean of 13.8 glomeruli (range 4.0 to 31.0 glomeruli), and a mean of 33.7% of glomeruli were globally sclerotic. Seven of the nine patients with diabetes had evidence of DGS, including mesangial sclerosis and thickening of glomerular and tubular basement membranes. The patterns of DGS were nodular in five patients and diffuse in two. The degree of DGS ranged from mild (two cases) to moderate (three cases) to severe (two cases). The two obese patients without DGS had biopsy findings of glomerulosclerosis, glomerulomegaly, and arteriosclerosis which were interpreted as changes secondary to the combined effects of obesity and hypertension.

The distinctive histologic feature was an acute and chronic tubulointerstitial nephropathy with acute tubular injury diffusely involving non-atrophic tubules, varying degrees of tubular atrophy and interstitial fibrosis, and abundant tubular calcium oxalate deposits (Figure 1, A and B). The tubular calcium oxalate deposits formed translucent crystals with an intraluminal, intracellular, and focally interstitial distribution (Figure 1, C and D). Under polarized light, the deposits appeared strongly birefringent forming fan-like, sheaf-like, or irregular shapes. They were seen predominantly in the cortex and involved both proximal and distal tubules, with fewer deposits in the medulla. The mean and median number of calcium oxalate deposits present in each renal biopsy was 42.9 and 31.5, respectively (range 15.0 to 118.0). Only a single biopsy exhibited <20 calcium oxalate deposits, and this occurred in a biopsy that contained only four glomeruli (patient 4). In an attempt to correct for the size of each biopsy sample, a ratio of calcium oxalate deposits per glomerulus was calculated. The mean and median number of calcium oxalate deposits per glomerulus was 3.5 and 3.1 (range 1.5 to 7.9). In general, intraluminal crystals predominated over intracellular and interstitial crystals. In many instances, the calcium oxalate crystals were associated with peritubular chronic inflammation, including four cases in which foreign-body giant cell reaction was seen.

In all biopsies, the oxalate crystalline deposits were accompanied by diffuse acute tubular injury in non-atrophic tubules characterized by luminal ectasia, epithelial simplification, loss

<table>
<thead>
<tr>
<th>Table 2. Treatment and outcomea</th>
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<tr>
<td>Patient</td>
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<tr>
<td>1</td>
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aCalcium, calcium supplementation; LFLOx, low-fat, low-oxalate diet; RRT, renal replacement therapy.
of proximal tubular brush border, enlarged reparative nuclei with prominent nuclei, and coarse cytoplasmic vacuolization (Figure 1D). In addition to the tubular degenerative changes, all biopsies showed irreversible tubular atrophy and interstitial fibrosis, which ranged from mild (two cases) to moderate (three cases) to severe (six cases). The nine patients with moderate or severe tubular atrophy and interstitial fibrosis had coexistent DGS or glomerulosclerosis attributed to obesity and hypertension. In areas of interstitial fibrosis, there was mild (six cases) to moderate (five cases) interstitial inflammation composed of mononuclear leukocytes with rare neutrophils, eosinophils, and plasma cells; however, no significant tubulitis was seen in any case. Arteriosclerosis and arteriolar hyalinosis were present in all but one case and ranged from mild in two cases to moderate in nine.

EM was performed in 10 of 11 cases and revealed tubular degenerative changes including loss of brush border, cytoplasmic simplification, dilation of the endoplasmic reticulum, enlarged reparative nuclei, and shedding of cellular debris into the tubular lumina. The intraluminal and intracytoplasmic oxalate deposits appeared as single or radially-oriented clusters of clear, needle-shaped crystals that indent the tubular epithelium. No glomerular or tubulointerstitial immune-type electron-dense deposits were identified. All cases of DGS showed mesangial matrix expansion and thickening of glomerular and tubular basement membranes. The mean degree of foot process effacement was 35.5% (range 0 to 90%). Immunofluorescence was performed in all cases and confirmed the absence of immune-type deposits.

Discussion

The incidence of obesity has reached epidemic proportions in the United States and worldwide. In the United States, the percentage of adults with obesity (defined as BMI \( \geq 30 \text{ kg/m}^2 \)) has increased from 15.3% in 1995 to 23.9% in 2005 (14), and approximately 5% of patients are morbidly obese (BMI \( \geq 40 \text{ kg/m}^2 \)) (15). Obesity is associated with multiple health risks, including type 2 diabetes, hypertension, heart disease, secondary FSGS, hyperlipidemia, fatty liver, and sleep apnea, as well as with increased mortality and reduced life expectancy (16–18). Bariatric surgery is the most effective treatment for morbid obesity, whereas lifestyle changes and pharmacotherapy alone have not shown long-term efficacy in the majority of patients. Bariatric surgery not only leads to significant long-term weight reduction but also results in decreased mortality and improvement or even resolution of several comorbidities such as diabetes, hyperlipidemia, hypertension, and obstructive sleep apnea (19,20). Consequently, the number of bariatric surgeries performed in the United States has increased dramatically (21).

RYGB is currently the most common bariatric procedure in the United States. In a multi-institutional consecutive cohort study of 1144 bariatric surgeries, RYGB was performed in 1049 (91.7%) cases (8). RYGB is both a gastric restrictive and a malabsorptive operation. The gastric restrictive procedure involves stapling the stomach to create a small (15 to 30 ml in capacity) proximal stomach pouch. The small bowel is then divided at a distance of 15 to 100 cm distal to the ligament of
Treitz, and the distal portion (alimentary or Roux limb) is anastomosed to the gastric pouch. The distal segment of stomach, duodenum, and proximal portion of jejunum (biliopancreatic limb) is anastomosed end to side to the jejunum at a point 75 to 150 cm distal to the gastrojejunostomy (22). The separation of food, which passes through the Roux limb, from the biliopancreatic secretions of the biliopancreatic limb results in malabsorption.

RYGB is also the preferred mode of reconstruction after total gastrectomy for gastric carcinoma because of its simplicity and its ability to prevent biliary and pancreatic secretions from reaching the esophageal mucosa (23). The separation of food, which passes through the Roux limb, from the biliopancreatic secretions of the biliopancreatic limb results in malabsorption.

RYGB is also the preferred mode of reconstruction after total gastrectomy for gastric carcinoma because of its simplicity and its ability to prevent biliary and pancreatic secretions from reaching the esophageal mucosa (23). The small bowel is usually divided at a distance of 25 cm distal to the ligament of Treitz (23). Postoperatively, some patients develop steatorrhea, weight loss, and diarrhea (24). The causes of fat malabsorption in these patients include rapid intestinal transit, bacterial overgrowth, and pancreatic understimulation (25). Unfortunately, the three patients who underwent total gastrectomy with RYGB in our series were not tested for hyperoxaluria, and no studies in the literature addressed the prevalence of hyperoxaluria after total gastrectomy and Roux-en-Y reconstruction. Such studies are difficult to perform due to the short life expectancy of these patients. Of note, hyperoxaluria has been reported after Roux-en-Y gastrojejunostomy for Crohn’s disease (26).

In the series reported herein, patients with oxalate nephropathy after RYGB presented with ARF, often superimposed on chronic renal insufficiency, at a mean of 33 mo and a median of 12 mo (range 4 to 96 mo) after surgery. In comparison, for patients with oxalate nephropathy after JI bypass, the mean time from surgery to renal failure was 44.5 mo (range 6.0 mo to 25 yr) (4). Baseline serum creatinine was elevated in seven of 10 patients in our study, five of whom had evidence of DGS. The main risk factor for oxalate nephropathy and nephrolithiasis after RYGB seems to be hyperoxaluria, which is likely due to fat malabsorption (10,13). The urinary profile of patients with RYGB resembles those with JI bypass, and, in fact, 23% of patients who have undergone modern bariatric surgery have urinary oxalate levels of >100 mg/d (levels at which renal

Figure 1. Pathologic findings in oxalate nephropathy. (A) A low-power view shows diffuse tubular degenerative changes with numerous intracellular and intraluminal tubular calcium oxalate deposits. A normal-appearing glomerulus also is present. (hematoxylin and eosin [H&E]). (B) The same field as A is shown under polarized light. The calcium oxalate crystals are more easily identified (H&E). (C) At high magnification, the calcium oxalate deposits form intraluminal translucent crystals (H&E). (D) In this field, the calcium oxalate crystals are smaller and lie within the cytoplasm of tubular epithelium. Tubules exhibit prominent degenerative changes including luminal ectasia, cytoplasmic simplification, and loss of brush border (H&E). Magnifications: ×40 in A and B; ×400 in C and D.
damage has been described) (10). More than half of the patients in our cohort had a history of chronic diarrhea, and all three patients for whom measurements were performed had hyperoxaluria and/or hyperoxalemia. Volume depletion associated with chronic diarrhea may play a role by leading to higher intratubular concentrations of oxalate and calcium. Importantly, the majority of patients in this report had longstanding obesity, diabetes, hypertension, and, in some cases, underlying chronic kidney disease. These findings suggest that pre-existing renal disease may create a ripe environment for calcium oxalate crystal formation and the acute and chronic tubulointerstitial injury that follows.

There are multiple limitations to our study that relate to its retrospective nature and the obvious lack of a control group. In addition, incomplete data are available on serum and urine oxalate levels, and we were unable to obtain detailed dietary information. For instance, we are aware of a single patient who had been instructed to curtail or discontinue eating star fruit (carambola), but we are unable to assess the patient’s compliance with these instructions. Unfortunately, we were unable to assess the incidence of oxalate nephropathy after RYGB because all surgeries were performed at outside institutions.

The prognosis of oxalate nephropathy after RYGB seems to be dismal, with progression to ESRD within 3 mo in 72.7% of patients in this study. Coexistent DGS or changes secondary to obesity and hypertension, present in 81.8% of patients, likely contributed to the poor outcomes. Of the two patients without underlying DGS or glomerulosclerosis related to obesity and hypertension, one started on permanent dialysis 1 mo after biopsy and the other had partial improvement in renal function. The majority of patients were placed on low-fat, low-oxalate diets, and four received calcium supplements, which did not appear to be beneficial when instituted so late in the process after significant irreversible injury had occurred. Earlier institution of these dietary interventions may have been more successful. Similar poor outcomes were reported for patients with oxalate nephropathy after JI bypass (4), except for a few patients in whom surgical reversal of bypass was performed for morbid obesity: A clinical and pathological (EM) study of a case. Surgery 82: 629–634, 1977


Conclusions
Oxalate nephropathy is a seemingly rare but underrecognized complication of RYGB. Considering the substantial increase in the number of RYGB surgeries performed annually in the United States (from 14,000 in 1998 to 108,000 in 2002) (21), the incidence of oxalate nephropathy likely will increase. On the basis of our data, patients who undergo RYGB should have long-term follow-up of renal function and metabolic parameters with the hope of instituting dietary modifications or even surgical reversal at an early time point at which these interventions may be beneficial. In patients with acute renal functional deterioration and a history of RYGB, the differential diagnosis should include oxalate nephropathy, and renal biopsy should be considered to establish the diagnosis.

Disclosures
None.