# #1969 - Renal Failure Secondary to Oxalate Nephropathy following Roux-en-Y gastric bypass surgery


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Body

**Introduction** Oxalate nephropathy after RYGB, albeit rare, has a poor prognosis, with a rapid progression to kidney failure1. Oxalate nephropathy has many etiologies and remains a rare cause of renal failure. It is characterized by tubular crystalline deposits of calcium oxalate leading to acute and chronic tubular injury, interstitial fibrosis, and progressive renal insufficiency.

**Case presentation**

A 39 years old diabetic woman came to our nephrology clinic for progressive increase in serum creatinine from 1 to 4.2 with nausea, dizziness and malase during two months . there was no history of fever ,diarrhea , steatorrhoea ,urinary tract symptom and use of nephrotoxic agent .her past medical history included diabetes mellitus from 15 years ago , diabetic retinopathy and neuropathy from 5 years ago, renal stone two month ago and gastric bypass surgery of stomach (RYGB) 6 month ago and treated diabetic foot ulcer and two episode of cesarean.n.she had undergone a surgery for morbid obesity 6 months earlier subsequently she had lost more than 30 Kg of body weight after surgery during 6 months and there was significant improvement in the control of blood glucose . there was no prior of renal disease .her serum creatinine was 1 mg/dl and urinalysis was normal at the time of surgery.

There was no family history of kidney disease ,Her vital signs was stable and clinical examination was unremarkable .Laboratory investigation showed a normocytic

normochromic anemia with a hemoglobin 7.5 g/dl and renal insufficiency with a serum Bun 33mg/dl ,creatinine 4.2mg/dl.urinalysis revealed PH 5 , specific gravity 1.025 , 6-8 white cells /hpf without any casts ,proteinuria or hematuria.

A 24 hours urine collection revealed hypocalciuria and hypo phosphaturia urinary .uric acid was within normal range and urinary oxalate wasn't determined . immunological test were negative .anti GBM Ab ,Anti ds DNA antibody, AncA-MP and AncA PR3 antibodies completment levels were within the normal range .

Ultrasound of kidney showed normal size kidneys without any evidence of obstruction. Color doppler sonography of renal arteries was normal, abdominal and pelvis CT scan was normal In view of progressive unexplained increase in creatinine a kidney biopsy was done. Histological examination of the biopsy specimen under light microscopy revealedthat glomeruli appeared normal . There was an mild interstitial inflammation with moderate interstitial fibrosis and tubular atrophy,and bright intra-tubular calcium oxalate crystals.

The immunofluorescence was negative and electron microscopy did not show any electron dense deposits .

Despite medical management of hyperoxaluria her acute kidney injury worsen

Subsequently no recovery of renal function was observed so the patient is currently undergoing regular hemodialysis

**Discussion**

The prevalence of severe obesity in both the general and the chronic kidney disease (CKD) populations continues to rise,. Severe obesity has significant renal consequences, including increased risk of end-stage renal disease (ESRD) and nephrolithiasis. Bariatric surgery represents an effective method for achieving sustained weight loss, and bariatric surgery is also effective in improving (1)

in hypertension control diabetes, dyslipidemia, urinary albumin excretion, markers of atherosclerosis, and progression of chronic kidney disease (CKD) after weight reduction surgeries.[2,3,4] There is also evidence for reduction in mortality and morbidity after bariatric surgery.[5]

The most common bariatric surgery is Roux-en-Y gastric bypass (RYGB)(,6 )which has been associated with hyperoxaluria and nephrolithiasis. 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) (7), the incidence of oxalate nephropathy likely will increase. Oxalate nephropathy is progressive in nature and has a poor prognosis.[8,9] with progression to ESRD within 3 mo in 72.7% of patients in this study(10)

In the setting of fat malabsorption/enteric hyperoxaluria, enteric free fatty acids are elevated and bind calcium within the intestinal lumen, inhibiting the formation of calcium oxalate. The increased amount of soluble free oxalate is absorbed by the colonic mucosa. The absorbed oxalate is excreted by the kidney and then deposited in the renal parenchyma forming stones (11).Oxalate nephropathy is associated with surgery, ethylene glycol poisoning, inflammatory bowel disease, or food ingestion(1 ). The most common clinical presentations Nephrolithiasis, nephrocalcinosis, progressive chronic kidney disease, and end stage kidney disease (11). Oxalate nephropathy is the most severe renal complication of bariatric surgery and has been reported in patients after jejunoileal bypass and RYGB surgery. A case series of 11 patients with oxalate nephropathy after RYGB demonstrated Time from surgery to AKI ranged from 4 to 96 months, and the majority of patients progressed to ESRD (10)

In 2005, Nelson *et al*. first described the presence of hyperoxaluria in patients after a standard RYGB.[12] Until now, only a few case reports of oxalate nephropathy have been reported after bariatric surgery. In a Mayo clinic series of 60 cases of post-RYGB, only two had oxalate nephropathy

and both reached end-stage renal disease (ESRD).[13] Nasr *et al*. have reported the largest series of oxalate nephropathy with 11 cases diagnosed post-RYGB of which eight reached ESRD.[10] ]

Definitive diagnosis of oxalate nephropathy is made by kidney biopsy and the characteristic finding of oxalate crystals in renal tubules with associated inflammatory injury. 14 Oxalate nephropathy is an entity characterised by the presence of tubular crystalline deposits of calcium oxalate,which can lead to both acute and chronic tubular injury(10)

Treatment of oxalate nephropathy post-bariatric surgery targets reduction of enteric hyperoxaluria. A low-fat diet is recommended to reduce the binding of calcium by free fatty acids. Low oxalate, high-fluid intake and citric salts, such as potassium citrate are also recommended. The use of calcium salts to bind oxalate is the mainstay of therapy, though there is little published on the efficacy of this approach.[15]. Cholestyramine may also be helpful as the presence of bile salts in the colon may increase colonic permeability to organic acids such as oxalate.[16 )

Reversal of RYGB be considered in patients with progressive loss of kidney function due to proven oxalate nephropathy, especially if medical therapies fail to reduce hyperoxaluria. (14).. Reversal of RYGB may be considered to reduce hyperoxaluria, although it is unclear whether this improves long-term outcomes.(17)

In one case report in 2016 Laparoscopic reversal of RYGB was performed and within 1 month hyperoxaluria resolved (urine oxalate 20 mg/day) and AKI improved(14)..Bariatric surgeons report reversal of RYGB to be safe and postoperative complications are minimal [18].

However,there are also renal risks in bariatric surgery, namely, acute kidney injury, nephrolithiasis, and, in rare cases, oxalate nephropathy,Although bariatric surgery may improve long-term kidney outcomes, this potential benefit remains unproved and must be balanced with potential adverse events(1)

Our case, oxalate nephropathy and kidney failure occurred six months after RYGB surgery. Our case demonstrates that a patient presenting with AKI and a history of RYGB should be evaluated for oxalate nephropathy and nephrolithiasis. We recommend prompt initiation of measures to reduce 24-hour urine oxalate and addressing other lithogenic factors (such as decreased urine volume and citrate) [19]. In this case incomplete data are available on serum and urine oxalate levels, and we were unable to obtain detailed dietary information . 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 . (10)

outcome of oxalate nephropathy is usually disappointing because of its rapid progression to end-stage kidney disease. Ultimately, because no effective treatments are currently available, substantial further research is necessary to find efficacious ways to extend the lives of patients with oxalate nephropathy (11)

**Conclusion**

Oxalate nephropathy is a rare and poor prognosis complication of RYGB. In patients with a history of RYGB presenting with acute or chronic renal injury of unclear etiology, Renal biopsy should be considered for definitive diagnosis. Considering the rapid

progression of oxalate nephropathy to kidney failure,patients who undergo Roux-en-Y gastric bypass surgery should have regular follow up of renal function

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References

References:

1. Alex R. Chang1,2, Morgan E. Grams3,4 and Sankar D. Navaneethan5,6 .Bariatric

Surgery and Kidney-Related Outcomes .KI reports

2. Navaneethan SD, Kelly KR, Sabbagh F, Schauer PR, Kirwan JP, Kashyap SR. Urinary albumin excretion, HMW adiponectin, and insulin sensitivity in type 2 diabetic patients undergoing bariatric surgery. Obes Surg. 2010;20:308–15.]

3. Shah SS, Todkar JS, Shah PS, Cummings DE. Diabetes remission and reduced cardiovascular risk after gastric bypass in Asian Indians with body mass index <35 kg/$m^2$. Surg Obes Relat Dis. 2010;6:332–8.

4. Navaneethan SD, Yehnert H. Bariatric surgery and progression of chronic kidney disease. Surg Obes Relat Dis. 2009;5:662–5. [PubMed]

5. Sjöström L, Narbro K, Sjöström CD, Karason K, Larsson B, Wedel H, et al. Effects of bariatric surgery on mortality in Swedish obese subjects. N Engl J Med. 2007;357:741–52. [PubMed]

6 . Nguyen NT, Silver M, Robinson M, Needleman B, Hartley G, Cooney R, Catalano R, Dostal J, Sama

Result of a national audit of bariatric surgery performed at academic centers: A 2004 University HealthSystem Consortium Benchmarking Project. *Arch Surg* 141: 445–450, 2006

7. Shinogle JA, Owings MF, Kozak LJ: Gastric bypass as treatment for obesity: Trends, characteristics, and

complications. *Obes Res* 13: 2202–2209, 2005

8 .Park AM, Storm DW, Fulmer BR, Still CD, Wood GC, Hartle JE., 2nd A prospective study of risk factors for nephrolithiasis after Roux-en-Y gastric bypass surgery. J Urol. 2009;182:2334–9.

9. Matlaga BR, Shore AD, Magnuson T, Clark JM, Johns R, Makary MA. Effect of gastric bypass surgery on kidney stone disease. J Urol. 2009;181:2573–7.

1. Samih H. Nasr,* Vivette D. D'Agati,* Samar M. Said,* Michael B. Stokes.Oxalate Nephropathy Complicating Roux-en-Y Gastric Bypass: An Underrecognized Cause of rreversible Renal Failure.Clin J Am Soc Nephrol 3: 1676–1683, 2008

11. Cheng-Wei Chou a, Chieh-Lin J. Teng a, b, c, *, Wen-Li Hwang. Oxalate nephropathy related end-stage renal disease in a gallbladder cancer patient after Roux-en-Y surgery.Journal of Cancer Research and Practice 2016 94-95

12. Nelson WK, Houghton SG, Milliner DS, Lieske JC, Sarr MG. Enteric hyperoxaluria, nephrolithiasis, and oxalate nephropathy: Potentially serious and unappreciated complications of Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2005;1:481–5.

13. Sinha MK, Collazo-Clavell ML, Rule A, Milliner DS, Nelson W, Sarr MG, et al. Hyperoxaluric nephrolithiasis is a complication of Roux-en-Y gastric bypass surgery. Kidney Int. 2007;72:100–7.

14. Varun Agrawala Jonathan B. Wilfongb Christopher E. Richc Pamela C. Gibson Reversal of Gastric Bypass Resolves Hyperoxaluria and Improves Oxalate Nephropathy Secondary to Roux-en-Y Gastric Bypass . Case Rep Nephrol

Dial 2016;6:114–119

15 .Harper J, Mansell MA. Treatment of enteric hyperoxaluria. Postgrad Med J. 1991;67:219–22.

16. Canales BK, Gonzalez RD. Kidney stone risk following Roux-en-Y.gastric bypass surgeryTransl Androl Urol. 2014 Sep; 3(3):242–249.

17. Agrawal V, Khan I, Rai B, et al. The effect of weight loss after bariatric surgery on albuminuria. Clin Nephrol. 2008;70:194–202

18. Campos GM, Ziemelis M, Paparodis R, Ahmed M, Davis DB: Laparoscopic reversal of Roux-en-Y gastric bypass: technique and utility for treatment of endocrine complications. Surg Obes Relat Dis 2014;10:36–43.

19. Agrawal V, et al: Calcium oxalate supersaturation increases early after Roux-en-Y gastric bypass. Surg Obes Relat Dis 2014;10:88–94.