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Gastric Bypass Procedures and Renal Calculi—How Should We Counsel Patients and Bariatric Surgeons?

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OBESITY (body mass index greater than 30 kg/m²) is clearly an epidemic in the United States, affecting more than 20% of the population.¹ Furthermore, as many as 5 million of these Americans have medically complicated obesity with weight related comorbidities such as diabetes mellitus, hypertension and sleep apnea. For the majority attempts at diet and lifestyle change have been a disappointing strategy to achieve lasting weight loss. Therefore, an increasing number are turning to bariatric surgery. The large randomized Swedish Obese Subjects study, with more than 4,047 subjects, has confirmed that bariatric surgery is associated with sustained long-term weight loss and overall decreased mortality.² Furthermore, the perioperative morbidity and mortality of this procedure appear to be acceptably low.³ Given these considerations the number of bariatric procedures has increased dramatically from approximately 13,000 in 1998 to more than 120,000 in 2004.⁴ Roux-en-Y gastric bypass (RYGB), often performed laparoscopically, is the most common procedure and appears to be associated with the greatest likelihood of durable weight loss. However, laparoscopic adjustable gastric banding is increasingly popular as an alternative and less invasive approach.

The risks of fat malabsorption, enteric hyperoxaluria, kidney stones and even kidney failure were well recognized with the jejunoileal bypass procedure, and were reasons why the procedure was abandoned in the early 1980s. Modern bariatric procedures were initially viewed as unlikely to have these complications. However, in 2005 a retrospective review at our institution identified 23 cases of enteric hyperoxaluria after RYGB including 2 cases of biopsy proven renal failure secondary to oxalosis.⁵ Cases appeared more common among those who had more extensive or distal procedures. During the next year we identified a larger group of 60 patients with kidney stones after RYGB including 31 with complete metabolic data.⁶ Hyperoxaluria, low urinary citrate and low urine volumes were common features. In a small random subset recruited from the endocrinology clinic before or after RYGB the prevalence of hyperoxaluria appeared to be alarmingly high since it was present in approximately half of those individuals 12 or more months postoperatively. Furthermore, some of these patients had rather severe hyperoxaluria (eg greater than 100 mg per day). Other investigators identified the same trends. For example, in 132 patients in whom kidney stones developed after RYGB and who were evaluated through a large referral laboratory common features included hyperoxaluria (mean 77 mg), reduced citrate (mean 423 mg) and lower volume (1.56 l). These changes were all in the same direction but were less severe than those seen in an older cohort of patients after undergoing jejunoileal bypass.⁷ Although such urinary changes would be expected to lead to an increased risk of calcium oxalate stones, this was not proven until Matlaga et al completed a case-control study of 4,639 patients who had undergone RYGB surgery a mean of 4.6 years earlier and appropriately matched to an obese, nonoperated control group.⁸ They demonstrated that stone events occurred in 7.65% of the RYGB group vs 4.63% of the obese controls, or almost a doubling of risk, with a mean time from RYGB to stone diagnosis of 559 days. Urological procedures for stones were also more common in the RYGB group.

In this issue of The Journal 2 important articles add to our knowledge regarding this potential complication of bariatric surgery. Park et al (page 2334) followed a cohort of 45 morbidly obese patients before RYGB and then obtained followup urine chemistry studies in the same subjects 6 to 12 months postoperatively. Mean urinary oxalate increased 8 mg while citrate decreased 183 mg and urinary volume decreased close to 500 ml daily. The net effect was a clinically significant increase in urinary calcium oxalate supersaturation from 1.27 to 2.23. Looked at another way, after surgery 42% of patients had definite hyperoxaluria while another third had low urinary citrate and 60% had an increased calcium oxalate supersaturation. This information gives us important insights into the course and prevalence of urinary changes in a relatively large number of patients followed through RYGB surgery.

Penniston et al (page 2340) identified 27 patients after RYGB and another 12 after gastric banding. Urinary oxalate was higher and urinary citrate was lower in the RYGB group, while urinary volume was similarly reduced in both groups. The net effect was that urinary calcium oxalate supersaturation was higher, although not significantly so, in the gastric banding group. It has been speculated that gastric banding should not result in fat malabsorption and enteric hyperoxaluria, and this is one of the first studies to support that commonly held belief. On the other hand, urinary supersaturation was slightly higher in the gastric banding group. Therefore, the risk of kidney stones may be increased even in patients who undergo the relatively noninvasive laparoscopic adjustable banding procedure.

Based on this information what do we know, and what can we tell our colleagues about kidney stones and bariatric surgical procedures? It seems clear that hyperoxaluria, reduced urinary citrate and increased calcium oxalate supersaturation are common side effects of bariatric surgery. Therefore, kidney stone risk may be increased by approximately 2-fold in the first 5 years after RYGB. On the other hand, the Swedish Obese Subjects study clearly demonstrates that the overall net effect of RYGB is positive with decreased patient comorbidities and improved overall mortality. Therefore, it does not seem reasonable to suggest that this increased risk of kidney stone disease is a contraindication to these procedures. Rather, at this point it seems that increased awareness of the problem is needed. All patients in whom a kidney stone develops after RYGB merit a prompt metabolic evaluation and consultation with someone knowledgeable in this area. We also need to pay attention to the apparent risk of kidney damage and possible kidney failure, and an important research question is to identify those patients at risk for this more extreme complication. Anecdotal observation suggests that individuals with underlying chronic kidney disease may be at higher risk for renal failure after RYGB but this remains to be clarified. Another unknown is whether patients with a preexisting history of kidney stone disease are at greater risk for this complication after RYGB, and if they merit closer followup and perhaps prophylactic treatment. Finally and most importantly we urgently need improved strategies for the treatment of enteric hyperoxaluria and the associated risk of kidney stone disease. After RYGB patients often have relatively strict dietary regimens, and adding a complicated program that includes avoiding almost all oxalate containing foods and high doses of calcium supplements can, quite literally, be difficult for patients to swallow. It is important to remember that complications of jejunoileal bypass were progressive for up to 20 years after the procedure and that the volume of modern RYGB procedures (more than 120,000 per year) thoroughly dwarfs the entire number of jejunoileal bypass operations ever performed (approximately 25,000 between 1961 and 1983).⁹ Therefore, even if the percentage of affected patients is much less for RYGB than after the notorious jejunoileal bypass, it would appear that issues related to the diagnosis and management of enteric hyperoxaluria after RYGB will be an important area of clinical research for many years to come.

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REFERENCES

1. Demaria EJ, Jamal MK. Surgical options for obesity. *Gastroenterol Clin North Am.* 2005; 34:127. [PubMed: 15823443]
2. Sjöström L, Narbro K, Sjöström CD, et al. Effects of bariatric surgery on mortality in Swedish obese subjects. *N Engl J Med.* 2007; 357:741. [PubMed: 17715408]
3. Flum DR, Belle SH, King WC, et al. Perioperative safety in the longitudinal assessment of bariatric surgery. *N Engl J Med.* 2009; 361:445. [PubMed: 19641201]
4. Elder KA, Wolfe BM. Bariatric surgery: a review of procedures and outcomes. *Gastroenterology.* 2007; 132:2253. [PubMed: 17498516]
5. Nelson WK, Houghton SG, Milliner DS, et al. Enteric hyperoxaluria, nephrolithiasis, and oxalate nephropathy: potentially serious and un-appreciated complications of Roux-en-Y gastric bypass. *Surg Obes Relat Dis.* 2005; 1:481. [PubMed: 16925274]
6. Sinha MK, Collazo-Clavell ML, Rule A, et al. Hyperoxaluric nephrolithiasis is a complication of Roux-en-Y gastric bypass surgery. *Kidney Int.* 2007; 72:100. [PubMed: 17377509]
7. Asplin JR, Coe FL. Hyperoxaluria in kidney stone formers treated with modern bariatric surgery. *J Urol.* 2007; 177:565. [PubMed: 17222634]
8. Matlaga BR, Shore AD, Magnuson T, et al. Effect of gastric bypass surgery on kidney stone disease. *J Urol.* 2009; 181:2573. [PubMed: 19375090]
9. Requarth JA, Burchard KW, Colacchio TA, et al. Long-term morbidity following jejunoileal bypass. The continuing potential need for surgical reversal. *Arch Surg.* 1995; 130:318. [PubMed: 7887801]