Published in final edited form as:

Curr Urol Rep. 2014 May ; 15(5): 401. doi:10.1007/s11934-014-0401-x.

Kidney Stone Risk Following Modern Bariatric Surgery

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Abstract

Over the past 10 years, a variety of reports have linked bariatric surgery to metabolic changes that alter kidney stone risk. Most of these studies were retrospective, lacked appropriate controls, or involved bariatric patients with a variety of inclusion criteria. Despite these limitations, recent clinical and experimental research has contributed to our understanding of the pathophysiology of stone disease in this high-risk population. This review summarizes the urinary chemistry profiles that may be responsible for the increased kidney stone incidence seen in contemporary epidemiological bariatric studies, outlines the mechanisms of hyperoxaluria and potential therapies through a newly described experimental bariatric animal model, and provides a focused appraisal of recommendations for reducing stone risk in bariatric stone formers.

Keywords

Morbid obesity; Hyperoxaluria; Bariatric surgery; Gastric bypass surgery; Hypocitraturia; Nephrolithiasis; Calcium oxalate stones

Introduction

Obesity and its related complications are becoming increasingly common and more costly. Recent estimates suggest over one-third of American adults are obese, with a body mass index (BMI) >30 kg/m², including more than 15 million who are considered morbidly obese (BMI >40 kg/m²), and that this trend on the rise [1–3]. To date, the most effective weight loss therapy for these patients is bariatric surgery, curing obesity-related diabetes and hypertension as well as lowering cardiovascular and overall mortality risk [4]. Due to its weight loss successes, the rates of bariatric surgery have increased an astonishing six-fold

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Conflict of Interest Dr. Ricardo D. Gonzalez declares no potential conflicts of interest relevant to this article.

Compliance with Ethics Guidelines

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

This article is part of the Topical Collection on *Minimally Invasive Surgery*

over the last 10 years, from 36,700 procedures in the year 2000 to approximately 220,000 in the year 2010 (Fig. 1) [3, 5].

In 2005, Nelson et al. first described the renal complications of hyperoxaluria, calcium oxalate stones, and oxalate nephropathy following modern bariatric surgery in a select group of patients [6]. Since that report, over 30 different publications have attempted to examine the potential metabolic derangements that alter kidney stone risk following bariatric surgery. In this review, we will summarize published data that detail urinary chemistry profiles and the incidence of kidney stones following bariatric surgery. Additionally, we will focus on recently published clinical and experimental literature that gives insight into both pathophysiology of stone risk as well as techniques that can be used to reduce this risk in bariatric patients who are stone formers.

Types of Bariatric Surgery

Modern bariatric surgery induces weight loss by restricting the amount of food that can be ingested (restrictive), by decreasing the amount of intestine available for nutrient absorption (malabsorptive), or by a combination of both procedures. The most commonly used restrictive procedures are the adjustable gastric band (GB) and the vertical sleeve gastrectomy (Sl Gx). The laparoscopic gastric band procedure, commonly referred to as “lap band,” involves placing an inflatable silicon band around the upper portion of the stomach to create a small pouch. The band is attached to a subcutaneous saline reservoir, which can be adjusted in clinic depending on tolerability and the amount of satiety the bariatric surgeon wants to create. Alternatively, instead of banding the stomach, 70 % of the lateral stomach can be surgically excised, creating a long tubular gastric remnant, termed sleeve gastrectomy. Long-term weight loss for both of these procedures are patient-and surgeon-dependent but are considered to be traditionally less (approximately 10 % total body weight) than a malabsorptive procedure (25–30 % total body weight) [4].

Modern malabsorptive procedures have evolved from the 1950s to include both a restrictive and a malabsorptive component. Biliopancreatic diversion with duodenal switch (BPD) reduces the stomach pouch by Sl Gx. The distal small bowel is then divided 250 cm proximal to the ileocecal valve, and the duodenum is then transected. This distal ileal limb is anastomosed 2 cm distal to the pylorus while the biliary-pancreatic limb is reanastomosed approximately 100 cm proximal to the ileocecal valve. Roux-en-Y gastric bypass (RYGB) surgery is the most common bariatric procedure in the U.S., again combining the principles of restrictive and malabsorptive procedures. Unlike BPD, the lesser curvature of the stomach is stapled into a 20–30 ml pouch for RYGB. The jejunum is then transected 60–70 cm distal to the ligament of Treitz, and the portion distal to this is sewn to the pouch. The transected jejunum is then anastomosed anywhere from 80–120 cm down the jejunum, creating two limbs in the shape of a “Y” that effectively separate ingested food until they meet at a distal jejunal common channel [7, 8].

Changes in Urinary Chemistry After Modern Bariatric Surgery

Over the past 10 years, a variety of reports have linked modern bariatric surgery to metabolic changes that alter the urinary milieu and kidney stone risk. Table 1 summarizes

the studies that involve 9 or more patients and report urine chemistry profiles before and/or after bariatric procedure. Of the studies reported, 9/13 contain retrospective components with highly variable inclusion criteria, use of controls, timing of 24-hour urine collection, non-standardized diets or dietary supplements, and methodological quality. Some studies include bariatric non-stone formers, bariatric stone formers both pre- and post-op, or bariatric patients who develop stones de novo postop. Despite these limitations, a review of these studies gives the practicing urologist a broad sense of urinary changes that may be seen with each procedure.

Nelson et al. (2005) first proposed the link between hyperoxaluria and stone risk in a group of 23 patients who developed either calcium oxalate stones (n=21) or oxalate nephropathy (n=2) following RYGB, spurring a multitude of studies on postoperative urinary changes after bariatric surgery [6]. In a prospective longitudinal study (n=45), Park et al. (2009) described significant increases in urinary oxalate excretion, calcium oxalate supersaturations (CaOx SS), and decreases in urinary citrate, calcium, and total urine volume after RYGB when compared to preoperative urine samples [9]. Although there were no symptomatic stone events after a mean of 9.6 study months, the authors contended that chronic acidosis in these patients may lead to decreases in urinary citrate (a known stone inhibitor), further increasing stone risk, in addition to hyperoxaluria [9]. Similarly, Duffey et al. (2010) described a doubling of urinary oxalate excretion and significant decreases in urinary citrate excretion in a 2-year prospective study in RYGB non-stone-forming patients [10]. Importantly, this study showed that risk of postoperative hyperoxaluria appears to increase over time, not decrease or remain stable [10]. Wu et al. recently (2013) noted urinary changes 6 months after RYGB (n=38) from baseline, including significant increases in urinary oxalate excretion, calcium, and calcium oxalate supersaturation, and decreases in total urine volume [11•]. The lack of hypocitrituria and presence of hypercalciuria in this cohort as compared to previous studies was thought to be due to increased utilization of calcium citrate supplementation in these patients postoperatively [11•].

Interventional diet or dietary supplement studies in the post-bariatric surgery setting have also provided insight into the mechanisms involved in recurrent stone disease. Pang et al. (2012) looked at the effect of changing diet in a small number of recurrent stone formers who were an average 11 years out from RYGB (n=6), jejunioileal bypass (n=2), or BPD (n=1) [12]. The authors performed 24-hour urines on these patients on baseline diets and then placed them on a metabolic diet consisting of 1000 mg calcium, reduced oxalate (70–80 mg), 20 % protein, <25 % fat, and 3000 mg sodium. Results showed that, although urine oxalate levels remained high, the metabolic diet positively affected urinary CaOx SS by increasing urine volume and raising urine pH and urinary citrate [12]. Although not supported by their data, the authors also recommended additional strategies such as oral calcium supplements, oxalate binders, and lower-fat meals to reduce future stone risk.

These recommendations were further investigated by Froeder et al. (2012), who observed similar decreases in urine citrate and total urinary volume in RYGB and BPD patients compared to morbidly obese controls [13]. Interesting, they found that bariatric patients had similar rates of hyperoxaluria (~13–20 %) compared to controls at baseline. However, when a group of patients (n=43) were given a 375 mg oxalate load (spinach juice), bariatric

patients had a twofold elevation in urinary oxalate at all urine time points compared to controls, suggesting that excessive oxalate absorption was occurring [13]. The authors went on to test a subset of these patients (n=21) for *Oxalobacter formigenes* (a commensal pathogen in the GI tract that uses oxalate as an energy source) colonization and found no difference in rates, again suggesting that the differences in hyperoxaluria between these two populations was due to oxalate over-absorption, not lack of bacterial consumption. Lastly, Sakhaee and colleagues (2012, not listed in Table 1) performed a short 2-phase placebo crossover study using effervescent potassium citrate and calcium citrate (40 meq potassium, 800 mg calcium, 100 meq citrate/day) in 24 patients at a mean 4.7 years after RYGB [14]. From a bone standpoint, the more soluble delivery of calcium lowered markers of bone resorption but had no effect on serum parathyroid hormone. The potassium and citrate raised urine pH and lowered calcium oxalate agglomeration by direct crystal testing. Although this product is not commercially available, this placebo-based interventional trial convincingly contends that calcium citrate reduces stone risk in bariatric stone formers.

Changes in Stone Incidence After Modern Bariatric Surgery

Table 2 focuses on incidence of stone events after bariatric surgery. Durrani et al. (2006) were the first to report an increased prevalence of stones (3.2 % de novo stones from chart review) in a cohort of RYGB patients (n=972) compared with expected rates derived from a control population [15]. Matlaga et al. (2009) then reported claims data in a case-control study of 4,639 post-RYGB surgery patients compared to obese controls, demonstrating stone diagnosis in 7.65 % of RYGB versus 4.63 % of controls [16•]. To determine whether this phenomenon remained true in non-obese patients, Shimizu et al. (2012) reviewed CTscans from gastric cancer patients who had either distal gastrectomy with Billroth I/Roux-en-Y or total gastrectomy with Roux-en-Y reconstruction [17]. In this population, patients with total gastrectomy were more likely to have renal stones by CT (21/85, 25 %) than patients with some portion of the stomach remaining (10/141, 7 %). Although no urinary chemistries were available, the authors hypothesized that total gastrectomy may lead to more fat malabsorption than partial gastrectomy, perhaps furthering hyperoxaluria. Furthermore, they did not find a significant difference in stone incidence between distal gastrectomy patients with either Billroth I or Roux-en-Y reconstruction, again suggesting that extent of stomach resection may be more important from an absorptive standpoint than length of the common channel. This study does not address potential confounders such as differences in fluid intake.

For restrictive procedures, Semins et al. (2009) reported from claims data that morbidly obese controls experienced higher incidence of stone disease (6 %) over 2.5 years than a population of patients after gastric banding (1.5 %) [7]. This study was the first to suggest that restrictive surgeries may minimize urinary metabolic changes compared to a malabsorptive procedure. Likewise, Chen et al. (2013) reported 1 % stone incidence rates by chart review of over 400 GB (mean 43 months post-op) and SI Gx (mean 27 months postop) patients, estimating person-time stone incidence rate of 3.40 stones per 1000 person-years for GB and 5.25 stones per 1000 person-years for SI Gx [18•]. This is in contrast to the Matlaga study [16•], which estimated person-time stone incidence rate of 16.62 stones per 1000 person-years for RYGB and 11.3 stones per 1000 person-years for routine obesity

[16••]. Based on this epidemiologic data, it appears that restrictive bariatric procedures have very little impact on stone risk. Further long-term studies are needed to determine if these results are conclusive, as the nature of the reduced gastric reservoir may potentially limit fluid intake, reduce urine volume, and increase CaOx SS and stone risk [7].

Pathophysiology in Experimental Models of Bariatric Surgery

To date, clinical data suggest that malabsorptive bariatric procedures have higher incident kidney stone risk due to changes in urinary volume, oxalate, and citrate, and therefore these procedures fall within the spectrum of gastrointestinal disorders characterized by malabsorption of bile salts and/or fatty acids, termed “enteric hyperoxaluria.”

Mechanistically, as fat is malabsorbed, fat-soluble vitamins and calcium ions are saponified by intraluminal free fatty acids, leading to steatorrhea with subsequent nutrient loss. As a consequence of this reduced calcium availability in the intestinal lumen, there is decreased calcium oxalate binding and increased unbound oxalate within the small and large intestine. This increased free oxalate load, otherwise destined for fecal excretion, has a higher chance of either passive or active gut absorption. Once absorbed, oxalate is an end-product of metabolism and must be filtered and excreted by the kidney [13, 19, 20, 21••]. Concurrently, permeability of the GI tract to oxalate can be increased dramatically by exposure to unconjugated bile salts and long-chain fatty acids, both of which have been shown to be increased in the GI tract of RYGB patients [12, 21••, 22–24]. While this hypothesis conforms well for hyperoxaluria and malabsorption, only a small number of human studies have been published in this area, with limited results [13, 21••, 25]. In an attempt to better understand these mechanisms, our group established a diet-induced obese rodent model of RYGB surgery and tested the long-term effect of high dietary fat and oxalate on fecal fat excretion and 24-hour urine parameters [26•].

In a cohort of 19 experimental animals and 16 sham-operated controls, we tested two different diets (40 % vs. 10 % fat) with high (1.5 %) and no oxalate content. In all age-matched sham-operated control animals, we saw no appreciable change in urinary oxalate or fecal fat on any of these regimens [26•]. However, RYGB animals on high-fat and oxalate diets had eight-fold higher fecal fat excretion and heavier stools, a fivefold increase in urine oxalate excretion, and fourfold increase in calculate oxalate supersaturation. In this setting, simply lowering dietary fat resulted in a 40 % decrease in oxalate excretion. For RYGB animals on a no-oxalate-added diet, urine oxalate was consistently higher than controls, suggesting that other mechanisms may have resulted in excess oxalate excretion. These include increased systemic oxalate production, enhanced renal oxalate excretion, slow GI transit times, causing increased GI uptake, and perhaps even changes in gut flora as a direct result of the bypass.

For years, the anaerobic bacterium *Oxalobacter formigenes*, a gut commensal capable of metabolizing oxalic acid, has been implicated as a possible etiology for increased urinary oxalate levels in calcium oxalate stone formers [27]. In 2005, subjects with fat malabsorption, hyperoxaluria, and calcium oxalate stones caused by a variety of GI diseases (including 6 RYGB patients) were given the probiotic Oxadrop® (*Lactobacillus acidophilus* and *brevis*, *Streptococcus*, and *Bifidobacterium*) [28]. Over a period of 3 months, mean

urinary oxalate levels decreased by 20 % in 10 patients [28]. In the RYGB population, it has been suggested that higher rates of *O. formigenes* colonization could also result in decreased levels of oxalate in the gut and, subsequently, the urine [11••, 13]. To test this hypothesis, our group has begun colonizing hyperoxaluric RYGB animals with this bacterium, and preliminary data in 4 animals has shown that persistent colonization results in a 75 % reduction in urinary oxalate levels (unpublished). These exciting results require further validation with larger experimental numbers, but may offer a future potential therapy for RYGB patients who suffer from hyperoxaluria and recurrent oxalate stones.

In addition to hyperoxaluria, the other commonly cited urinary abnormalities in bariatric surgical patients include low urinary volume and low urine citrate, both of which can significantly increase calcium oxalate risk and supersaturation [10, 11••, 21••, 29–32]. After full review of all existing literature in this area, we agree with the dietary recommendations that have been advocated by so many other authors: reduction in oxalate-rich and fatty foods to minimize enteric absorption, increased hydration to increase total urine volume, calcium supplementation using calcium citrate instead of calcium carbonate, and citric salts (potassium citrate) to correct metabolic acidosis and hypocitrituria [10, 16••, 17, 32]. Probiotics may also be beneficial, although existing data is limited [28]. Future directions for study or therapy in these patients include a more critical appraisal of vitamin deficiencies, including pyridoxine (vitamin B6, an important co-factor in liver metabolism of glyoxylate); clarification of the role of gut hormones in renal and GI oxalate transport; and publication of results from investigators using cholestyramine to bind bile acids in this population.

Conclusion

Malabsorptive bariatric procedures and calcium oxalate stone disease appear to be irrevocably linked through mechanisms of hyperoxaluria, low urine volume, and hypocitrituria. Further investigations in the RYGB animal model as well as interventional human trials are needed to better define the etiology of and the best means to prevent this renal complication, especially in light of growing obesity and bariatric surgical trends.

Acknowledgments

Dr. Benjamin K. Canales received funding from National Institutes of Health (NIH K08 DK089000-04), AUA Foundation Rising Star in Urology Research Award, Astellas Global Development, Inc., and Ethicon Endo-Surgery.

References

Papers of particular interest, published recently, have been highlighted as:

- Of importance
- Of outstanding importance

1. Ogden CL, Carroll MD, Curtin LR, et al. Prevalence of overweight and obesity in the United States, 1999–2004. *JAMA*. 2006; 295:1549–55. [PubMed: 16595758]
2. Mokdad AH, Serdula MK, Dietz WH, et al. The spread of the obesity epidemic in the United States, 1991–1998. *JAMA*. 1999; 282:1519–22. [PubMed: 10546690]

3. Demaria EJ, Jamal MK. Surgical options for obesity. *Gastroenterol Clin N*. 2005; 34:127–42.
4. Sjostrom L, Narbro K, Sjostrom CD, et al. Effects of bariatric surgery on mortality in Swedish obese subjects. *N Engl J Med*. 2007; 357:741–52. [PubMed: 17715408]
5. Elder KA, Wolfe BM. Bariatric surgery: a review of procedures and outcomes. *Gastroenterology*. 2007; 132:2253–71. [PubMed: 17498516]
6. Nelson WK, Houghton SG, Milliner DS, et al. 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. [PubMed: 16925274]
7. Semins MJ, Matlaga BR, Shore AD, et al. The effect of gastric banding on kidney stone disease. *Urology*. 2009; 74:746–9. [PubMed: 19683804]
8. Santry HP, Gillen DL, Lauderdale DS. Trends in bariatric surgical procedures. *JAMA*. 2005; 294:1909–17. [PubMed: 16234497]
9. Park AM, Storm DW, Fulmer BR, et al. A prospective study of risk factors for nephrolithiasis after Roux-en-Y gastric bypass surgery. *J Urol*. 2009; 182:2334–9. [PubMed: 19762043]
10. Duffey BG, Alanee S, Pedro RN, et al. Hyperoxaluria is a long-term consequence of Roux-en-Y Gastric bypass: a 2-year prospective longitudinal study. *J Am Coll Surg*. 2010; 211:8–15. [PubMed: 20610243]
- 11••. Wu JN, Craig J, Chamie K, et al. Urolithiasis risk factors in the bariatric population undergoing gastric bypass surgery. *Surg Obes Relat Dis*. 2013; 9:83–7. Recent prospective series showing increased urinary oxalate excretion after RYGB compared to preoperative samples. [PubMed: 22100051]
12. Pang R, Linnes MP, O'Connor HM, et al. Controlled metabolic diet reduces calcium oxalate supersaturation but not oxalate excretion after bariatric surgery. *Urology*. 2012; 80:250–4. [PubMed: 22554593]
13. Froeder L, Arasaki CH, Malheiros CA, et al. Response to dietary oxalate after bariatric surgery. *Clin J Am Soc Nephrol*. 2012; 7:2033–40. [PubMed: 23024163]
14. Sakhaee K, Griffith C, Pak CY. Biochemical control of bone loss and stone-forming propensity by potassium-calcium citrate after bariatric surgery. *Surg Obes Relat Dis*. 2012; 8:67–72. [PubMed: 21703942]
15. Durrani O, Morrisroe S, Jackman S, Averch T. Analysis of stone disease in morbidly obese patients undergoing gastric bypass surgery. *J Endourol*. 2006; 20:749–52. [PubMed: 17094749]
- 16••. Matlaga BR, Shore AD, Magnuson T, et al. Effect of gastric bypass surgery on kidney stone disease. *J Urol*. 2009; 181:2573–7. Claims data from bariatric surgery and obese controls patients (n=4,639 respectively) showing near doubling of stone incidence post-RYGB. [PubMed: 19375090]
17. Shimizu H, Ichikawa D, Tamagaki K, et al. Evaluation of postoperative nephrolithiasis and renal dysfunction in gastric cancer patients. *Gastric Cancer*. 2013; 16:338–44. [PubMed: 22948316]
- 18•. Chen T, Godebu E, Horgan S, et al. The effect of restrictive bariatric surgery on urolithiasis. *J Endourol*. 2013; 27:242–4. Retrospective series highlighting low incidence (1%) of stone disease after restrictive bariatric surgery. [PubMed: 23006039]
19. Hofmann AF, Laker MF, Dharmasathaphorn K, et al. Complex pathogenesis of hyperoxaluria after jejunoileal bypass surgery. Oxalogenic substances in diet contribute to urinary oxalate. *Gastroenterology*. 1983; 84:293–300. [PubMed: 6848409]
20. Andersson H, Jagenburg R. Fat-reduced diet in the treatment of hyperoxaluria in patients with ileopathy. *Gut*. 1974; 15:360–6. [PubMed: 18668844]
- 21••. Kumar R, Lieske JC, Collazo-Clavell ML, et al. Fat malabsorption and increased intestinal oxalate absorption are common after Roux-en-Y gastric bypass surgery. *Surgery*. 2011; 149:654–61. One-year clinical and experimental investigation of plasma and urinary oxalate levels in 9 patients following RYGB, including evaluation of oxalate loading and fecal fat testing. [PubMed: 21295813]
22. Dobbins JW, Binder HJ. Effect of bile salts and fatty acids on the colonic absorption of oxalate. *Gastroenterology*. 1976; 70:1096–100. [PubMed: 1269869]
23. Siener R, Petzold J, Bitterlich N, et al. Determinants of urolithiasis in patients with intestinal fat malabsorption. *Urology*. 2013; 81:17–24. [PubMed: 23200965]

24. Freel RW, Hatch M, Earnest DL, Goldner AM. Oxalate transport across the isolated rat colon. A re-examination. *Biochim Biophys Acta*. 1980; 600:838–43. [PubMed: 7407147]
25. Odstrcil EA, Martinez JG, Santa Ana CA, et al. The contribution of malabsorption to the reduction in net energy absorption after long-limb Roux-en-Y gastric bypass. *Am J Clin Nutr*. 2010; 92:704–13. [PubMed: 20739420]
26. Canales BK, Ellen J, Khan SR, Hatch M. Steatorrhea and hyperoxaluria occur after gastric bypass surgery in obese rats regardless of dietary fat or oxalate. *J Urol*. 2013; 190:1102–9. New description of hyperoxaluria and fecal fat malabsorption in an RYGB model that holds promise for future investigations and therapies. [PubMed: 23499748]
27. Allison MJ, Dawson KA, Mayberry WR, Foss JG. *Oxalobacter formigenes* gen. nov., sp. nov.: oxalate-degrading anaerobes that inhabit the gastrointestinal tract. *Arch Microbiol*. 1985; 141:1–7. [PubMed: 3994481]
28. Lieske JC, Goldfarb DS, De Simone C, Regnier C. Use of a probiotic to decrease enteric hyperoxaluria. *Kidney Int*. 2005; 68:1244–9. [PubMed: 16105057]
29. Asplin JR, Coe FL. Hyperoxaluria in kidney stone formers treated with modern bariatric surgery. *J Urol*. 2007; 177:565–9. [PubMed: 17222634]
30. Patel BN, Passman CM, Fernandez A, et al. Prevalence of hyperoxaluria after bariatric surgery. *J Urol*. 2009; 181:161–6. [PubMed: 19013593]
31. Maalouf NM, Tondapu P, Guth ES, et al. Hypocitraturia and hyperoxaluria after Roux-en-Y gastric bypass surgery. *J Urol*. 2010; 183:1026–30. [PubMed: 20096421]
32. Sinha MK, Collazo-Clavell ML, Rule A, et al. Hyperoxaluric nephrolithiasis is a complication of Roux-en-Y gastric bypass surgery. *Kidney Int*. 2007; 72:100–7. [PubMed: 17377509]
33. Buchwald H, Oien DM. Metabolic/bariatric surgery worldwide 2008. *Obes Surg*. 2009; 19:1605–11. [PubMed: 19885707]
34. Penniston KL, Kaplon DM, Gould JC, Nakada SY. Gastric band placement for obesity is not associated with increased urinary risk of urolithiasis compared to bypass. *J Urol*. 2009; 182:2340–6. [PubMed: 19762051]
35. Semins MJ, Asplin JR, Steele K, et al. The effect of restrictive bariatric surgery on urinary stone risk factors. *Urology*. 2010; 76:826–9. [PubMed: 20381135]
36. Costa-Matos A, Guidoni LR, Carvalho KA, et al. Is there an association between urolithiasis and Roux-en-y gastric bypass surgery? *Int Braz J Urol*. 2009; 35:432–5. [PubMed: 19719858]
37. Marceau P, Marceau S, Biron S, et al. Long-term experience with duodenal switch in adolescents. *Obes Surg*. 2010; 20:1609–16. [PubMed: 20835899]

Fig. 1.
Number of bariatric surgeries performed in the United States from 1992–2010. Estimates taken from Buchwald and Oien [33] and the American Society of Metabolic and Bariatric Surgery (ASMBS) website: <http://asmbs.org>

Table 1

Urinary profiles following bariatric surgery, grouped by stone history and study design

Article	Procedure (n)	F/U (mo.)	Urinary Oxalate (CaOx SS)	Other urinary changes and study comments
Non-stone formers, prospectively collected				
Park 2009 [9]	RYGB (45)	9.6	Pre-op: 32 (1.27) Post-op: 40 (2.23)	De novo hyperoxaluria occurred in 90 %. No symptomatic stone events during study.
Duffey 2010 [10]	RYGB (21)	24	Pre-op: 33 (1.73) Post-op: 63 (2.2)	De novo hyperoxaluria occurred in 52 %. Hypocitraturia increased from 10 % at baseline to 48 %, but relative CaOx SS was unchanged.
Kumar 2011 [21••]	RYGB (9) BPD (2)	6, 12	Pre-op: 26 (1.0) Post-op: 32 (1.8)	Decreased total urine volume and higher fecal fat excretion at 6 and 12 months. Oral oxalate loading test at 6 and 12 months resulted in higher urine oxalate excretion.
Wu 2013 [11••]	RYGB (38)	6	Pre-op: 38 (NR) Post-op: 48 (NR)	Urine calcium increased by 43 mg/day (perhaps due to higher supplementation), while urine volume decreased by 0.5 liter/day. Stone formation or passage events were not recorded.
Non-stone formers, retrospectively collected				
Nelson 2005[6]	RYGB (13) LL-RYGB (9)	NR	RYGB=88 (2.38) LL-RYGB=95 (2.69)	CaOx SS was reported in $\mu\text{mol/L}$ (normal range <1.77). Long-limb RYGB results in a shorter common channel, resulting in more malabsorption.
Patel 2009 [30]	RYGB (52) BPD (6)	14.2	RYGB=62 (NR) BPD=90 (NR)	Comparisons made to healthy and stone-forming adults from a commercial database.
Penniston 2009 [34]	RYGB (27) GB (12)	32	RYGB=48 (1.89) GB=41 (2.78)	Urine calcium decreased ~50 % in RYGB versus GB, and 52 % RYGB had urinary citrate <370 mg/day vs. 9 % in GB. Urine volume decreased in both groups.
Semins 2010 [35]	GB (14) SI Gx (4)	12.4	Restrictive=35.4 (5.22)	GB and SI Gx (termed restrictive cohort) was compared to normal and stone-forming adults as well as RYGB patients (mean urinary oxalate=61 mg/day and CaOx SS=7.76.)
Maalouf 2010 [31]	RYGB (19) Con (19)	42	RYGB=45 (7.0) Control=30 (5.0)	~50 % reduction in urinary citrate level compared to controls (mean 358 mg/day vs. 767 mg/day).
Froeder 2012 [13]	RYGB (58) BPD (3) Con (30)	48	RYGB/BPD=26 (NR) Control=29 (NR)	Oxalate loading (RYGB=22, Con=21) showed higher urine oxalate in RYGB. No difference in <i>O. formigenes</i> colonization between subgroups (RYGB=10, Con=13). 6 patients had stones pre-op. No difference in CaOx SS, reported as Tiselius index.
Stone formers, retrospectively or prospectively collected				
Sinha 2007 [32]	RYGB (31)	N/A	Post-op: 60 (2.23)	Post-RYGB data compared to normal population references for urinary oxalate excretion.
Asplin 2007 [29]	JIB (27) GB/RYGB (132) Con (2210)	N/A	JIB=102 (NR) GB/RYGB=83 (NR) Control=34 (NR)	GB and RYGB surgeries were not separated for analysis. Mean time from procedure to stone event of 3.6 years. Stone formers identified in a corporate stone database.
Pang 2012[12]	JIB (1) RYGB (6) BPD (2)	N/A	Entire Cohort Free Diet=65 (1.97) Met Diet=62 (1.13)	Recurrent stone-forming bariatric patients mean 11 years after surgery had increased pH, urine volume, and citrate on Met (controlled metabolic) diet. No significant changes in urine oxalate excretion were noted, even on low oxalate diet.

Key: F/U=Follow-up in months (some means are number of months post-procedure), CaOx SS=Calcium oxalate supersaturation, RYGB=Roux-en-Y gastric bypass; LL-RYGB=Long-limb Roux-en-Y gastric bypass, BPD=Biliopancreatic diversion with duodenal switch, JIB=Jejunioileal bypass, GB= Gastric banding; SI Gx=Sleeve gastrectomy

Table 2

Kidney stone incidence following bariatric surgery, grouped by stone history and study design

Article	Procedure (n)	F/U (mo.)	Post-Procedural Stone Incidence	Comments
Durrani 2006 [15]	RYGB=972	NR	26/85 (31 %): recurrent 32/887 (3.6 %): de novo	Stones identified by patient chart review. Mean time to stone formation was 2.8 years (de novo) and 1.9 years (recurrent).
Semins 2009 [7]	GB (201) Con (201)	28*	GB=3/201 (1.5 %) Con=12/201 (6 %)	Stones identified by CPT code within claims data versus matched obese controls. 1 patient in each cohort underwent surgical treatment for symptomatic stones.
Matlaga 2009 [16••]	RYGB (4,639) Con (4,639)	18.6	RYGB=355/4639 (7.7 %) Con=215 (4.6 %)	Stones identified by CPT code within claims data versus matched obese controls.
Costa-Matos 2009 [36]	RYGB(58)	42*	RYGB=0/58 (0 %)	Stones identified by RUS. 1 patient had a stone pre-RYGB which remained unchanged post-op.
Marceau 2010 [37]	BPD (13)	126	BPD=1/13 (7.7 %)	Stone identification method not reported. Patients 15–17 years of age with BMI >40 kg/m ² .
Shimizu 2012 [17]	DTGx (226)	NR	31/226 (13.7 %)	Stones identified by CT in gastric cancer patients. Incident stones occurred in 25 % (21/85) of total vs. 7 % (10/141) distal gastrectomy. Mean time to first stone 17.6 months post-surgery.
Chen 2013 [18•]	GB(332) SI Gx (85)	38	GB=4/332 (1.2 %) SI Gx=1/85 (1.2 %)	Stones identified by patient chart review.

Key: F/U=Follow-up in months (some means are number of months post-procedure),

* Follow-up time reported as median, RYGB=Roux-en-y gastric bypass, GB=Gastric banding, Con=Control, CPT=Common procedural terminology, RUS=Renal ultrasound, BPD=Biliopancreatic diversion with duodenal switch, DTGx=Distal or total gastrectomy with Billroth I or Roux-en-y gastric bypass, SI Gx=Sleeve gastrectomy