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# Common bile duct dilation after bariatric surgery

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## Abstract

**Background** Biliary dilation suggests obstruction and prompts further work up. Our experience with endoscopic ultrasound and endoscopic retrograde cholangiopancreatography in the symptomatic post-bariatric surgery population revealed many patients with radiographically dilated bile ducts, but endoscopically normal studies. It is unclear if this finding is phenomenological or an effect of surgery. Additionally, it is unknown whether the type of bariatric surgery alters biliary pathophysiology. Thus, we studied whether a change occurs in biliary diameter following Roux-en-Y gastric bypass (RYGB) and sleeve gastrectomy (SG).

**Methods** A single-center retrospective study assessing biliary diameter before and after RYGB or SG based on radiographic imaging. All adult patients undergoing RYGB or SG from January 2010 to December 2013 who had imaging studies before and > 3 months after surgery were included. Those with known obstructive etiologies and those without post-operative imaging were excluded. Common bile duct (CBD) diameter was re-read by a radiologist at the same location in the CBD for pre- and post-operative imaging. Baseline clinical factors and cholecystectomy status were collected.

**Results** 269 patients met inclusion criteria (193 RYGB;76 SG). Between the groups, there were no significant differences in pre-operative characteristics. Average time from surgery to repeat imaging was 24.1 months. After adjusting for pre-operative factors, subjects who underwent an RYGB had an increase in CBD diameter of 1.4 mm (95% CI 0.096, 0.18), which was greater than the change following SG 0.5 mm(95% CI −0.007, 0.11). The magnitude of this change did not depend on prior cholecystectomy in the RYGB cohort. Within the SG group, for patients without a prior cholecystectomy, there was a significant increase in post-operative CBD diameter of 0.8 mm(95% CI 0.02, 0.14).

**Conclusion** Bariatric surgery results in CBD dilation, with changes more pronounced after RYGB. Biliary dilation occurs irrespective of cholecystectomy status. Further work is necessary to determine the cause and clinical implications of this phenomenon.

**Keywords** Common bile duct dilation · Bariatric surgery · Roux-en-Y gastric bypass · Sleeve gastrectomy · Interventional endoscopy

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Biliary dilation greater than 6–8 millimeters (mm) in a patient with an intact gallbladder, or over 10 mm in a patient post cholecystectomy suggests possible biliary obstruction and prompts further work-up [1, 2]. Obstructive etiologies include choledocholithiasis, biliary stricture, and masses intrinsic or extrinsic to the biliary tree [3]. Biliary dilation may be the initial presenting sign of a malignancy of the pancreas or bile duct, which are often detected at later disease stages and carry poor prognoses. Magnetic resonance imaging (MRI), which can digitally reconstruct the biliary tree, is a frequently employed non-invasive diagnostic test, but it may miss small masses/stones and pathology of ampulla [4]. The next step in evaluation is endoscopic ultrasound (EUS) and/or endoscopic retrograde cholangiopancreatography (ERCP). Performing a standard EUS or ERCP procedure after Roux-en-Y gastric bypass (RYGB) can be challenging due to the altered anatomy, requiring either a transgastric, balloon enteroscopy ERCP or EUS-guided biliary access.

Obesity has grown to be a worldwide epidemic affecting substantial proportions of the adult population in both developed, and developing countries. In addressing this, the surgical treatment of obesity, and its correspondent obesity-related co-morbid conditions, continues to increase. RYGB and sleeve gastrectomy (SG) are two of the most commonly performed operations worldwide. An RYGB operation involves both restrictive and malabsorptive components. A small gastric pouch is created from the proximal stomach and is anastomosed to a Roux, or alimentary limb. A distal jejuno-jejunostomy joins the Roux limb with the biliopancreatic limb that drains the secretions of excluded stomach, liver, and pancreas. Biochemical digestion and absorption occurs in the common channel, comprising of the small intestine beyond the jejuno-jejunostomy, where pancreatic enzymes and bile mix with ingested food [5–7]. The SG operation on the other hand is predominantly restrictive. The stomach is divided longitudinally, with the fundus and the lateral aspect of the gastric body resected, leaving a tubular stomach with limited ability to dilate to accommodate a food bolus [8]. As more patients undergo these operations, and as follow-up times increase, the long-term effects of these intervention are increasingly understood.

Prior case series from our center suggests that prior RYGB may be associated with biliary dilation. These case series did not include patients undergoing SG [9, 10]. It is unclear if biliary dilation is phenomenological and unrelated, an effect of RYGB. Additionally, it is unknown whether biliary dilation is confined to the post-RYGB population, or is a broad effect of bariatric surgery. Thus, the primary aim of this study was to evaluate the change in common bile duct (CBD) diameter based on non-invasive imaging following both RYGB and SG. The secondary aim was to compare the magnitude of the change in biliary diameter between RYGB and SG.

## Methods

### Study design

This is a single-center retrospective study assessing biliary diameter before and after RYGB or SG based on right upper quadrant (RUQ) ultrasound, computed tomography (CT) or MRI. The protocol was approved by the Institutional Review Board. The study did not receive any funding.

### Patients and procedures

All patients older than 18 who underwent either primary RYBG or SG between January 1, 2010 and December 31, 2013 were retrospectively identified based on Current Procedural Terminology (CPT) code (RYGB 43644, 46645, and SG 43775). Those who had RUQ ultrasound, abdominal CT or abdominal MRI studies before and greater than 3 months after surgery were included in our analysis. We excluded patients who underwent concomitant cholecystectomy at the time of their bariatric operation or during the time of follow-up. Patients with a documented prior history of choledocholithiasis, obstructive jaundice, CBD dilation prior to surgery, concomitant cholecystectomy during bariatric surgery, and prior bariatric operation were excluded. CBD diameter was re-read by a radiologist at the same location in the CBD for pre- and post-operative imaging for all patients included in the study, regardless of imaging modality.

### Study measurements

Baseline and pre-operative clinical factors of age, gender, body mass index (BMI), co-morbid illnesses (hypertension, hyperlipidemia, Type 2 diabetes, obstructive sleep apnea, coronary artery disease, gastroesophageal reflux disease, and osteoarthritis), narcotic use, and cholecystectomy history were collected. Post-operative clinical factors such as prolonged narcotic use (> 1 month of narcotic use after surgery), ursodiol prophylaxis in the post-operative period, and weight loss from time of surgery to post-operative imaging were collected.

Pre- and post-operative liver function tests (within 1 month prior to bariatric operation and at time of repeat imaging postoperatively) including total bilirubin, aspartate aminotransferase (AST), alanine aminotransferase (ALT), and alkaline phosphatase were collected. Post-operatively, data on potential etiologies that could affect bile duct diameter such as choledocholithiasis, pancreatitis, pancreatic mass, or sphincter of Oddi dysfunction were collected.

Finally, data on biliary symptoms, if any, prompting repeat imaging, date of repeat imaging (at least 3 months post-operatively), and time from operation to repeat imaging were collected.

### Statistical analysis

Data are presented as mean ± standard deviation, median [25th, 75th percentiles] or frequency (percent). Univariable analysis was performed to compare baseline characteristics for subjects who underwent RYGB surgery and those who underwent SG; analysis of variance (ANOVA) or the non-parametric Kruskal–Wallis test was used for continuous and Pearson's Chi-square tests or Fisher's exact tests were used for categorical factors. In addition, analysis of covariance (ANCOVA) was used to compare the post-operative change in diameter of the CBD after adjusting for pre-surgery diameter, age, and time from surgery to post-operative imaging.

The prevalence of dilation of the CBD in the absence of biliary pathology in subjects who undergo RYGB or SG was estimated by calculating the percentage of subjects with CBD dilation absent biliary pathology out of all who underwent RYGB or SG, and is reported with the corresponding 95% confidence interval.

ANOVA was used to compare post-surgery CBD diameter between subjects who underwent RYGB versus SG. Adjustment for pre-surgery diameter was done and the change between pre- and post-surgery was reported. In addition, adjustment for potential confounders such as opiate use and age was evaluated. An automated stepwise variable selection method performed on 1000 bootstrap samples was used to choose the model; variables with inclusion rates of at least 50% were included in the final model.

All analyses were performed using SAS (version 9.4, The SAS Institute, Cary, NC) and a $p < 0.05$ was considered statistically significant.

## Results

### Study participants and pre-operative characteristics

A total of 1412 patients underwent RYGB or SG during the study period; 980 underwent RYGB and 432 underwent SG. Of these, 193 patients who underwent RYBG and 76 patients who underwent SG had imaging that included the bile ducts both before, and at least three months after bariatric surgery, which comprised our study cohort.

The total cohort was 75.1% female ($n = 202$). Pre-operative BMI was 46.2 ± 6.9 kg/m$^2$ for RYGB, 47.7 ± 10.0 kg/m$^2$ for SG, and 46.6 ± 7.9 kg/m$^2$ overall. With respect to pre-operative co-morbidities, there was no significant difference between the RYGB and SG groups except that osteoarthritis was more common in the group undergoing SG (22.4% RYBG, 36.8% SG, $p = 0.016$), as seen in Table 1.

**Table 1** Demographics and pre-operative clinical characteristics

| Factor | RYGB (*N* = 193) Statistics | SG (*N* = 76) Statistics | *p* value |
|---|---|---|---|
| Age (years) | 46.2 ± 11.6 | 49.1 ± 11.6 | 0.064[a] |
| Gender | | | |
| Male | 42 (21.8) | 25 (32.9) | 0.057[c] |
| Female | 151 (78.2) | 51 (67.1) | |
| Pre-operative weight (kg) | 127.4 ± 24.1 | 135.9 ± 33.3 | **0.021**[a] |
| Pre-operative BMI (kg/m$^2$) | 46.2 ± 6.9 | 47.7 ± 10.0 | 0.18[a] |
| Hypertension | 123 63.7) | 54 (71.1) | 0.25[c] |
| Hyperlipidemia | 85 (44.0) | 42 (55.3) | 0.097[c] |
| Diabetes mellitus | 85 (44.0) | 27 (35.5) | 0.20[c] |
| Coronary artery disease | 19 (9.8) | 9 (11.8) | 0.63[c] |
| Obstructive sleep apnea | 118 (61.1) | 44 (57.9) | 0.62[c] |
| Osteoarthritis | 43 (22.4) | 28 (36.8) | **0.016**[c] |
| GERD | 67 (34.7) | 27 (35.5) | 0.90[c] |
| Narcotic use before surgery (daily use) | | | |
| Yes | 10 (5.3) | 2 (2.6) | 0.46[d] |
| No | 179 (94.2) | 73 (96.1) | |
| Quit | 1 (0.53) | 1 (1.3) | |
| Prior cholecystectomy | 37 (19.2) | 17 (22.7) | 0.52[c] |
| Imaging modality | | | |
| Ultrasound | 172 (89.6) | 63 (82.9) | 0.13[c] |
| CT | 20 (10.4) | 12 (15.8) | |
| MRI | 0 (0.0) | 1 (1.3) | |

Statistics presented as mean ± SD, median [P25, P75] or *N* (column %)

Bold values indicate statistical significance ($p < 0.05$)

*RYGB* roux-en-gastric bypass, *SG* sleeve gastrectomy, *BMI* body mass index, *GERD* gastroesophageal reflux disease, *CT* computed tomography, *MRI* magnetic resonance image

*p*-values: [a]anova, [b]Kruskal–Wallis test, [c]Pearson's Chi-square test, [d]Fisher's exact test

During the majority of the study period, pre-operative RUQ ultrasound was a routine element of the bariatric surgery care pathway, which is reflected in the large proportion of patients undergoing that modality to evaluate CBD diameter (87.7%). A minority of patients had CT (11.9%), and MRI (0.37%) studies that included the CBD as well. If a patient had consistent biliary symptoms pre-operatively, they would have had a concomitant cholecystectomy during the bariatric operation. Those patients with concomitant cholecystectomy were excluded from the study.

### Post-operative characteristics and repeat imaging

Patient characteristics, findings of non-invasive imaging obtained > 3 months post operatively, and liver function test results appear in Tables 2 and 3. The mean time from

**Table 2** Post-operative clinical characteristics and repeat imaging

| Factor | RYGB ($N = 193$) Statistics | SG ($N = 76$) Statistics | $p$-value |
|---|---|---|---|
| Ursodiol prophylaxis after surgery | 147 (76.6) | 58 (77.3) | 0.89[c] |
| Narcotics use after surgery | 33 (19.1) | 10 (13.3) | 0.27[c] |
| BMI at repeat imaging | 32.0 [27.5, 36.1] | 36.4 [31.5, 42.8] | **< 0.001**[b] |
| Time from bariatric operation to repeat imaging | 24.3 [12.8, 39.0] | 22.1 [10.9, 35.0] | 0.39[b] |
| Biliary or pancreatic pathologies detected on imaging | | | |
| CBD stones | 0 (0.0) | 0 (0.0) | |
| Pancreatitis | 9 (4.7) | 2 (2.6) | 0.45[c] |
| Pancreatic mass | 0 (0.0) | 0 (0.0) | |
| Sphincter of Oddi dysfunction | 2 (1.0) | 0 (0.0) | 0.99[d] |
| Biliary Stricture | 0 (0.0) | 0 (0.0) | |
| Symptoms and laboratory values prompting repeat imaging | | | |
| Abdominal pain | 90 (33.5) | 37 (13.8) | 0.66[c] |
| Nausea | 33 (17.2) | 15 (19.7) | 0.62[c] |
| Vomiting | 17 (8.9) | 9 (11.8) | 0.46[c] |
| Fevers | 2 (1.0) | 3 (3.9) | 0.14[d] |
| Jaundice | 0 (0.0) | 1 (1.3) | 0.28[d] |
| Elevated alkaline phosphatase | 5 (2.6) | 2 (2.6) | 0.99[c] |
| Elevated serum lipase | 5 (2.6) | 0 (0.0) | 0.33[d] |
| Imaging modality | | | |
| Ultrasound | 85 (44.0) | 30 (39.5) | 0.18[c] |
| CT | 97 (50.3) | 45 (59.2) | |
| MRI | 11 (5.7) | 1 (1.3) | |

Statistics presented as mean ± SD, median [P25, P75] or $N$ (column %)

Bold value indicates statistical significance ($p < 0.05$)

*RYGB* roux-en-gastric bypass, *SG* sleeve gastrectomy, *CBD* common bile duct, *CT* computed tomography, *MRI* magnetic resonance image

$p$-values: [a]anova, [b]Kruskal–Wallis test, [c]Pearson's Chi-square test, [d]Fisher's exact test

**Table 3** Pre- and post-operative liver function tests

| Factor | RYGB ($N = 193$) Statistics | SG ($N = 76$) Statistics | $p$-value |
|---|---|---|---|
| Pre-operative LFTs | | | |
| Total bilirubin (mg/dL) | 0.40 [0.30, 0.50] | 0.40 [0.30, 0.60] | 0.13[b] |
| AST (units/L) | 24.0 [18.0, 35.0] | 25.0 [19.0, 37.0] | 0.41[b] |
| ALT (units/L) | 27.0 [17.0, 41.0] | 27.0 [17.0, 41.0] | 0.76[b] |
| Alkaline phosphatase (units/L) | 74.0 [62.0, 90.0] | 66.0 [57.0, 80.0] | **0.009**[b] |
| Post-operative LFTs | | | |
| Total bilirubin (mg/dL) | 0.40 [0.30, 0.50] | 0.45 [0.30, 0.70] | 0.14[b] |
| AST (units/L) | 21.0 [16.0, 27.0] | 19.5 [16.0, 33.0] | 0.95[b] |
| ALT (units/L) | 19.0 [13.0, 29.0] | 15.0 [11.0, 25.0] | **0.030**[b] |
| Alkaline Phosphatase (units/L) | 82.0 [67.0, 102.0] | 70.5 [56.0, 91.0] | **0.003**[b] |

Statistics presented as mean ± SD, median [P25, P75] or $N$ (column %)

Bold values indicate statistical significance ($p < 0.05$)

*RYGB* roux-en-gastric bypass, *SG* sleeve gastrectomy, *LFTs* liver function tests, *AST* aspartate aminotransferase, *ALT* alanine aminotransferase

$p$-values: [a]anova, [b]Kruskal–Wallis test, [c]Pearson's Chi-square test, [d]Fisher's exact test

**Table 4** Pre- and post-operative common bile duct diameter

| Factor | Overall ($N=269$) Statistics | RYGB ($N=193$) Statistics | SG ($N=76$) Statistics | *p*-value |
|---|---|---|---|---|
| Pre-operative CBD diameter (mm) | 4.5 (4.3, 4.7) | 4.5 (4.2, 4.7) | 4.5 (4.1, 4.8) | 0.94[a] |
| Post-operative CBD diameter (mm) | 5.3 (5.0, 5.6) | 5.6 (5.2, 6.0) | 4.6 (4.3, 5.0) | **0.005**[a] |

Statistics presented as mean (95% CI)

Bold value indicates statistical significance ($p < 0.05$)

*RYGB* roux-en-gastric bypass, *SG* sleeve gastrectomy, *CBD* common bile duct

*p*-values: [a]anova, [b]Kruskal–Wallis test, [c]Pearson's Chi-square test, [d]Fisher's exact test

**Table 5** Change in common bile duct diameter by type of surgery

| | RYGB | | SG | |
|---|---|---|---|---|
| | Post–pre CBD diameter (mm)[a] | *p*-value | Post–pre CBD diameter (mm)[a] | *p*-value |
| All subjects | 1.4 (0.96, 1.8) | <0.001 | 0.5 (−0.07, 1.1) | 0.08 |
| Cholecystectomy | 1.5 (0.5, 2.5) | 0.006 | 0.4 (−1.3, 2.0) | 0.65 |
| No cholecystectomy | 1.5 (1.0, 1.9) | <0.001 | 0.8 (0.2, 1.4) | 0.014 |

*RYGB* roux-en-gastric bypass, *SG* sleeve gastrectomy

[a]ANCOVA analysis adjusting for age, pre-operative CBD diameter, time since surgery, post-operative imaging, narcotic use, and cholecystectomy in the case of all subjects. Display as mean (95% confidence interval)

bariatric operation to repeat imaging was 24.1 months. In 219 cases, symptoms or abnormal labs prompted repeat imaging. Thirteen patients (4.8%) had biliary or pancreatic pathology diagnosed at the time of their repeat imaging.

### Change in common bile duct diameter

The mean CBD diameter pre- and postoperatively for each group is shown in Table 4. Mean pre-surgical CBD diameter for all patients was 4.5 ± 1.6 mm, and was consistent for patients undergoing RYGB (4.5 ± 1.7 mm) and SG (4.5 ± 1.4 mm). Mean post-surgical CBD diameter for all patients is 5.3 ± 2.5 mm. In unadjusted analysis, there was a significantly greater CBD diameter following RYGB (5.6 ± 2.7 mm) compared to the SG cohort (4.6 ± 1.5 mm, $p=0.005$).

Table 5 shows the within group change in CBD diameter, adjusting for age, pre-operative CBD diameter, months since surgery, and imaging modality. Results are reported stratified by cholecystectomy status. After adjusting, subjects who underwent RYGB had a mean increase in 1.4 mm (95% CI 0.96–1.8 mm) after RYGB surgery ($p<0.001$ compared to preoperative diameter). The magnitude of this change did not depend on prior cholecystectomy. Within the SG group, after adjustment, there was no difference between pre-and post-operative CBD diameter. However, for patients without a prior cholecystectomy undergoing SG, there was a significant increase in post-operative CBD diameter of 0.8 mm (95% CI 0.2–1.4).

## Discussion

This is the largest study to examine changes in CBD diameter after bariatric surgery and the first to compare changes in CBD diameter after RYGB versus SG. RYGB results in a statistically significant dilation of the CBD by 1.4 mm, regardless of cholecystectomy history. For patients undergoing SG, overall there is no change in CBD diameter post operatively; however, mild dilation of 0.8 mm was noted in the subgroup who still had gall bladder in situ.

### Known causes of biliary dilation

Biliary obstruction from choledocholithiasis, benign/malignant strictures, or pancreatic/biliary masses are the most common explanation for biliary dilation [3]. Patients with known choledocholithiasis or pre-operative biliary dilation were specifically excluded in our study. In fact, in our entire study population, no subjects developed choledocholithiasis, pancreatic masses, or biliary strictures during the study period. Therefore, biliary obstruction did not falsely affect any of our results, leaving bariatric surgery as the cause of biliary dilation.

Biliary dilation is often reported with advancing age [11–13]. However, our cohort of patients was relatively young with a mean age of 47.0 ± 11.7 years, coinciding with the age of most bariatric patients. Age does not seem to be related to biliary dilation after bariatric surgery.

Another well-known cause of biliary dilation is cholecystectomy. In the post-cholecystectomy population, patients can have a normal CBD measuring 6.2 ± 2.5 mm [13]. Putatively, this is an adaptation to the increase in hydrostatic pressure within the bile duct produced once the capacitive reservoir of the gallbladder is removed [13]. We demonstrated a statistically significant CBD diameter dilation of 1.4 mm in patients undergoing RYGB irrespective of cholecystectomy history. In patients undergoing SG with gallbladders, there is also a statistically significant increase in

CBD diameter of 0.8 mm. Finally, in patients undergoing SG with a history of cholecystectomy, there is a non-significant trend toward increase in CBD diameter of 0.4 mm.

Finally, several medications have been associated with biliary dilation, most common of which is opiates [14]. In our study group, only 4.5% (12) patients were on narcotics preoperatively and 17.3% (43) patients were on post-operatively at least 3 months after surgery. While this is a potential limitation to our data, patients not on opiates also demonstrated biliary dilation.

## Potential causes of biliary dilation in the bariatric population

It is unclear what causes biliary dilation after gastric bypass surgery. While the effect size noted in the study is modest (1.5 mm), we have previously identified a subset of patients with massive biliary dilation (> 15 mm) after both cholecystectomy and RYGB, without a clear pathology and endoscopically normal ampullae [9]. Iatrogenic vagal injury causing ampullary denervation is one possible explanation. During a RYGB operation, the gastro–hepatic ligament is mobilized. While variable in location, the hepatic branch of vagus nerve may be divided during this mobilization, removing the afferent neural impulses from the central nervous system to the ampulla. The gastro–hepatic ligament is preserved during a SG operation, which may explain why biliary dilation was not noted after that operation in this study.

Post-operative alteration of gut hormones associated with bariatric surgery may also be explanatory, and differ between RYGB and SG operations. The modulation of gut peptides, specifically peptide YY, GLP-1/2, ghrelin, leptin, and orexin, has been identified as contributors to the improvement of diabetes and associated metabolic changes after bariatric surgery [15, 16]. Although no association between these enzymes and biliary dilation has been made, their altered levels post-surgically could play a role in biliary dilation. Cholecystokinin (CCK) is a gut hormone that contributes to the contraction of the gallbladder, section of pancreatic enzymes, relaxation of the sphincter of Oddi, and promoting satiety [15, 16]. Its release is stimulated by the presence of fatty acids and amino acids in the duodenum. Satake et al. have shown a decrease in CCK levels after bariatric surgery [17]. Other authors have proposed that decreased CCK would increase Sphincter of Oddi tone leading to biliary stasis and subsequent dilation [10]. However, there are newer data suggesting that CCK levels increase after bariatric surgery [15], leaving the effect of bariatric surgery on this hormone unclear. Furthermore, the changes in these hormones differ following RYGB and SG, which may explain the discrepancy in CBD dilation after RYGB and SG.

The gut microbiome is also significantly altered after bariatric surgery [18, 19]. There are also much data that have suggested that biliary disease and chronic liver disease are influenced by gut bacteria [20]. While we are unaware of evidence linking the specific post-operative shifts the makeup of the gut microbiome after RYGB of SG operations, this could be an area of investigation for an underlying etiology of post RYGB biliary dilation [20]. Similar to differential changes in gut hormones following RYGB and SG, the shifts in gut microbiota are more drastic following RYGB compared to SG, and thus could explain the difference observed in CBD dilation in this study.

Finally, we know that both obesity and the rapid weight loss induced by bariatric surgery are risk factors for the development of cholelithiasis. In patients with morbid obesity, the gallstone incidence is three to four times greater than in the general population [21]. Weight loss after RYGB predisposes the patient to the development of gallstones, particularly in the first year. Small, undetected stones may cause intermittent biliary obstruction and then dilation over time. However, there were no patients that developed cholelithiasis in our study population. Postoperative ursodiol prophylaxis appeared to prevent biliary sludge or cholelithiasis from developing in this group, though this study was not designed to evaluate that conclusion.

## Implications

Age, cholecystectomy, and opiate medications have each been documented in prior studies to be associated with benign biliary dilation. This study demonstrates that both RYGB, and in a subset of patients undergoing SG, biliary dilation may be observed after adjusting for those known factors. The underlying mechanisms are not well understood in any case. It is also not known if any other the factors previously identified to relate to CBD dilation operate in an additive fashion. Notably, there was a small percentage of patients (4.8%) in this series that had identified biliary pathology.

This finding is particularly significant given the challenge that RYGB anatomy poses for endoscopists when biliary or pancreatic duct access is required [22]. There are a number of strategies available for endoscopists to perform ERCP in patients with RYGB: duodenoscopes, colonoscopes, or enteroscopes through the anatomic route, ERCP using deep enteroscopy techniques, ERCP through gastrostomy tracts, laparoscopic-assisted transgastric ERCP, and the newer EUS-guided “EDGE” procedure with placement of lumen apposing stent from gastric pouch to restricted stomach [22, 23]. One systematic review showed that the overall endoscopic and ERCP failure rates of over-tube assisted enteroscopy ERCP were highest in patients with RYGB at 30% [24]. Many centers now proceed directly to transgastric ERCP. Given the challenges and risks in undergoing ERCP, the reason for

ERCP should be clear and with a strong indication. There is a high risk of pancreatitis and the definite diagnosis may not even be made [22].

## Limitations and generalizability

There are several limitations to this study. First, this is a retrospective study with its inherent challenges and biases. Second, post-operative imaging was only performed if patients had indications for repeat abdominal imaging. Largely, indications for repeat imaging were symptom based, and may or may not have been for biliary symptoms. As a result, asymptomatic patients may have unintentionally excluded and we cannot make a conclusion about their proclivity to develop biliary dilation. Additionally, the same imaging modality was not always used between patients perhaps resulting in minor discrepancies in assessed biliary diameter. Re-reading biliary diameter at a standard location for all imaging studies included in this analysis was performed to minimize that effect. Finally, all patients included underwent bariatric operations between 2010 and 2013. Longer term follow-up post-intervention may demonstrate progression of biliary ductal dilation. Overall, this study has strong external validity. RYGB and SG are the two most common bariatric surgical procedures performed worldwide. Both of these operations, as well as post-operative care are relatively standardized. Inclusion of 193 RYGB and 76 SG from a large tertiary care referral center should be generalizable to other centers performing bariatric surgery.

## Conclusions

This study demonstrates CBD diameter increases after RYGB, and after SG given the patient has their gallbladder in situ. Further studies aimed at understanding why this phenomenon occurs should be conducted. As physicians continue to treat an increasing number of patients with bariatric anatomy, they should be cautious in consider biliary dilation as a sign of bile duct pathology. This could unnecessarily lead to complex diagnostic procedures with increased risk for patient harm.

**Author Contributions** NM, ATS, TS, KE-H, PC, MR, CAB, JV, JV, MK, AB: role in manuscript preparation: concept and design; acquisition of data; analysis and/or interpretation of data; drafting of the manuscript and critical revision of the manuscript for important intellectual content. AN, AO, AE: role in manuscript preparation: acquisition of data. JM: Role in manuscript preparation: acquisition of data; analysis and/or interpretation of data. RL: role in manuscript preparation: analysis and/or interpretation of data; drafting of the manuscript and critical revision of the manuscript for important intellectual content.

## Compliance with ethical standards

**Disclosures** Dr. Matthew Kroh is a consultant for Medtronic and Levita Magnetics. He also receives research funding from Cook. Dr. Amit Bhatt is a speaker for Aries Pharmaceuticals and a consultant for Medtronic. Neal Mehta, Andrew T. Strong, Tyler Stevens, Kevin El-Hayek, Alfred Nelson, Adeyinka Owoyele, Ahmed Eltelbany, Prabhleen Chahal, Maged Rizk, Carol A. Burke, John McMichael, Rocio Lopez, Joseph Veniero, John Vargo have no conflicts of interest or financial ties to disclose.

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