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Reduced risk of de novo Barrett's esophagus after Bariatric Surgery: A National Database Study

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Abstract

Background: Bariatric surgery is an effective treatment for obesity and may decrease the morbidity and mortality of obesity-associated cancers.

Objective(s): We investigated the risk of a new diagnosis of Barrett's esophagus (BE) following bariatric surgery compared to screening colonoscopy controls.

Setting: Large national database including patients who received care in inpatient, outpatient, and specialty care services.

Methods: A national healthcare database (TriNetX, LLC) was used for this analysis. Cases included adults (age ≥ 18 years) who had undergone either sleeve gastrectomy (SG) or Roux-en-Y gastric bypass (RYGB). Controls included adults undergoing screening colonoscopy and an esophagoduodenoscopy on the same day and had never undergone bariatric surgery. Cases and controls were propensity matched for confounders. The risk of de novo diagnosis of BE after at least1 year after bariatric surgery was compared between cases and controls. Secondary analyses examined the effect of bariatric surgery on metabolic outcomes such as weight loss and BMI. The risk of de novo diagnosis of BE in SG was compared to RYGB. Odds ratios (OR) and 95% confidence intervals (CI) were used to report on these associations.

Results: In the propensity matched analysis, patients who had undergone a bariatric procedure showed a significantly reduced risk of de novo BE when compared to screening colonoscopy

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controls (0.67 [0.48, 0.94]). There was substantial reduction in weight and BMI in the bariatric surgery group when compared to baseline. There was no significant difference in de novo BE diagnosis between the propensity matched SG and RYGB groups (0.77 [0.5, 1.2]).

Conclusion: Patients who underwent bariatric surgery (RYGB or SG) had a lower risk of being diagnosed with BE compared to screening colonoscopy controls who did not receive bariatric surgery. This effect appears to be largely mediated by reduction in weight and BMI.

Keywords

Bariatric surgery; Sleeve gastrectomy; Roux-en-Y gastric bypass; Barrett's Esophagus

Introduction:

There has been an exponential rise in obesity in the United States over the last 2 decades, with an estimated current prevalence exceeding 40% [1]. Obesity has been associated with many metabolic and nonmetabolic diseases, with the deadliest among them being cancer. A meta-analysis from 2008 demonstrated that excess bodyweight, described by a high body-mass index (Body Mass Index [BMI] ≥ 30), was associated with several types of cancers, such as esophageal adenocarcinoma (EAC), thyroid, renal, colon, endometrial, and gallbladder cancers [2].

Interventions for reducing obesity include diet and lifestyle changes, pharmacotherapy, endoscopically inserted devices, and surgical interventions [3–5]. Bariatric surgery, with sleeve gastrectomy (SG) and Roux-en-Y gastric bypass (RYGB) being the most popular, has demonstrated to be one of the most effective treatments for sustained weight loss in patients with obesity [6, 7]. Bariatric surgery has been shown to result in a decreased risk for the development of obesity-associated cancers. For instance, the incidence of colorectal cancer has been shown to be lower in patients who undergo bariatric surgery compared to patients with obesity who do not receive bariatric surgery, thereby strengthening the association between colorectal cancer and obesity as well as establishing the protective effect of bariatric surgery [8].

In correlation with an increase in obesity, there has also been a rapid rise in the incidence of EAC and its precursor Barrett's esophagus (BE) in recent years [9]. It has been shown that both BE and EAC are known to be associated with obesity, particularly central adiposity in a reflux independent manner [10]. Therefore, it is reasonable to suggest that bariatric surgery would protect against the development of both diseases. However, a recent meta-analysis suggested an increase in the prevalence of BE three years after sleeve gastrectomy [11]. Interestingly, some recent studies have also found that RYGB may be associated with the development of high grade BE and EAC at variable time periods following surgery [12, 13]. Given the conflicting findings in BE development following bariatric surgery, we felt the need to clarify the association between receipt of bariatric surgery and risk of BE using a large national healthcare database.

Methods:

This was a retrospective observational study using a large commercially available healthcare database. Clinical data were extracted from the TriNetX research network (Cambridge, MA, USA), accessed on June 26, 2023. This globally federated health research network (with waiver from Western IRB) provides clinical information from 57 heath care organizations (HCOs), and about 95 million patients located within the United States. Participating HCOs include inpatient, outpatient, and specialty care services, with a typical participating HCO representing a large academic health center with main and satellite hospitals as well as outpatient clinics. Any data displayed on the TriNetX graphical user interface (GUI) platform only contains de-identified, aggregate data according to the de-identification standard defined by the HIPAA Privacy Rule. Since this study used only de-identified patient records and did not involve the collection or use of individually identifiable data, this study was exempted from Institutional Review Board approval.

We queried the TriNetX database for receipt of bariatric surgery (specifically RYGB and SG) using CPT codes and ICD10 codes for the diagnosis of BE. We excluded ICD10 codes for bariatric surgery procedures, and we also excluded patients with revisions of bariatric surgery, gastric restrictive procedures such as gastric banding and intragastric balloons. We also excluded procedures such as biliopancreatic diversion with or without duodenal switch, as well as single anastomosis duodeno-ileal bypass with sleeve gastrectomy (SADI-S)..[14]. We created two cohorts, namely cases and a control group for comparison. Cases were adults age ≥18 years who had undergone bariatric surgery (either SG or RYGB). For the control group, we selected adults age ≥18 years who were undergoing their first screening colonoscopy as well as their first esophagoduodenoscopic (EGD) examination on the same day, but had never undergone any type of bariatric surgery. For both cases and controls, we only included patients between 2009 and 2019. Exclusion criteria for both cohorts included a prior diagnosis of peptic ulcer disease, and a personal or family history of any gastrointestinal malignancy. We also ensured that patients were not diagnosed with BE prior to their bariatric surgery or screening colonoscopy. A propensity-matched analysis was also performed based on age, gender, race, smoking history, gastroesophageal reflux disease (GERD), diabetes mellitus, proton pump inhibitor (PPI) use, use of commercially available weight loss medications, insulin, metformin, sulfonylureas, pioglitazone, and hiatal hernia. TriNetX uses a 1:1 greedy nearest neighbor matching technique for generating the propensity matched cohorts.

For our primary analysis, we compared the risk of a new diagnosis of BE after bariatric surgery (SG or RYGB) to colonoscopy controls after at least 1 year after the surgery or screening colonoscopy. The primary reason for allowing this 1 year gap was to reduce any bias of diagnosing prevalent cases of BE and also for any potential benefits of bariatric surgery to take effect. For secondary outcomes, we included weight (in lbs) and BMI to determine the effect of bariatric surgery on these outcomes. Additionally, as it is well known that SG increases post-procedural GERD, we performed additional analysis e comparing SG to RYGB to see if the risk of development of BE. Analyses of these matched cohorts are presented in the form of summary statistics for descriptive data, and odds ratio (OR) and

95% confidence intervals (CI) for associations. All analyses are 2-sided with a p-value <0.05 deemed to signify statistical significance.

Results:

Our initial query yielded 25,186 patients who had undergone bariatric surgery (SG or RYGB) and 25,544 patients who had undergone a screening colonoscopy and EGD on the same day. Prior to propensity matching, bariatric cases were younger than screening colonoscopy controls (44±12 vs. 60±8) with a predominance of females (80% vs. 56%%). There were higher number of Whites in colonoscopy controls when compared to bariatric surgery cases (73% vs. 68%).. The average BMI was higher in bariatric cases when compared to screening colonoscopy controls (42.7 ± 5.4 vs. 28.5 ± 6.5). Approximately 60% of bariatric cases in contrast to 41% of screening colonoscopy controls were on a PPI. Interestingly, the prevalence of GERD was lower among bariatric surgery cases when compared to screening colonoscopy controls (49% vs. 60%). Detailed demographic characteristics of the included patient population is shown in Table 1. It was observed that after controlling for potential confounders, the cases and controls were quite evenly matched in attributions as shown in Table 1.

Primary analysis:

We performed a primary analysis by investigating the risk of new diagnosis of BE from 1 year from the time of bariatric surgery to any time after. In the unmatched primary analysis, when compared to screening colonoscopy controls, patients who had undergone bariatric surgery showed a significantly reduced risk of a new diagnosis of BE (OR = 0.33; 95% CI= [0.27, 0.41]); Table 2. The observed beneficial effect persisted, though it was attenuated in the propensity matched cohorts (OR = 0.67; 95% CI = [0.48, 0.93]); Table 2.

Secondary analysis:

We performed secondary analysis to examine the effect of bariatric surgery on metabolic outcomes such as weight and BMI. In the bariatric surgery group, the mean baseline weight was 274 (62.8) prior to surgery, whereas after at least 1 year following bariatric surgery, there was substantial reduction in weight (214.8 [57]). On the contrary, in the screening colonoscopy group, the mean baseline weight was 185 (46.2) which was relatively unchanged 1 year or more following screening colonoscopy (182.8 [47.4]). A similar substantial reduction in BMI was seen in the bariatric surgery group pre and post-surgery (42.7 [5.4] vs. 34.7 [7]), whereas there was minimal change in BMI in the screening colonoscopy group (Table 3).

We also performed additional secondary analysis to examine if there were differences in the risk of new BE diagnosis after SG when compared to RYGB. For the purposes of this analysis, we created two mutually exclusive cohorts comprised of patients who underwent SG only (n = 10,197) or RYGB only (n = 14,747). In the unmatched analysis, there was no significant difference between the SG and RYGB cohorts in the risk of BE (0.36% vs. 0.5%; OR = 0.73; 95% CI = 0.5,1.1). Following propensity matching, the reduction in risk was

consistent with the unmatched analysis albeit slightly attenuated (0.4% vs. 0.5% OR= 0.77; 94% CI = 0.5, 1.2). Detailed results of this analysis are provided in Table 2.

Discussion:

Bariatric surgery, especially SG and RYGB, has proven to be an effective treatment for obesity, leading to long-term weight loss success rates and reductions in obesity-related comorbidities, including obesity-associated cancers [7, 14, 15]. Our study adds to the growing body of evidence on the benefits of bariatric surgery, including reinforcing known concepts such as substantial reductions in weight loss and BMI, but also a reduced risk of diagnosis of BE, which is the only known pre-malignant precursor of EAC. Importantly, the protective effects of bariatric surgery persisted after matching for multiple potential confounders, such as age, gender, race, nicotine dependence, GERD, diabetes mellitus, PPI use, use of weight loss medications, and hiatal hernia.

The risk reduction in de novo BE that was observed in the present study suggests that bariatric surgery may be protective against BE, however, the exact mechanism of protection remains uncertain. Through our secondary analysis, we have shown that bariatric surgery, when compared to screening colonoscopy controls, results in substantial improvements in weight reduction as well as improvement in BMI. The reduced risk of diagnosis of BE in the bariatric surgery group could potentially be attributed to the improvement in these metabolic factors such as weight and BMI. While the major driver of BE risk reduction was weight loss, there are likely other mechanisms in play that could be attributed to bariatric surgery, including but not limited to mechanical, anatomic, and hormonal changes. For instance, by reducing abdominal obesity, bariatric surgery likely reduces mechanical stress on the lower esophageal sphincter thereby reducing reflux events [16]. Furthermore, it is well recognized that abdominal fat associated with central obesity is hormonally active and presents a state of low-grade inflammation. This metabolically active fat is responsible for increased levels of pro-inflammatory adipocytokines which are known to be associated with increased risk of BE [17]. It is possible that a reduction in abdominal obesity mediated through bariatric surgery could have produced hormonal changes such as reduction in circulating pro-inflammatory adipocytokines and indeed, it has been shown that patients have increased adiponectin and decreased leptin levels compared to controls [18].Furthermore, bariatric surgery has been shown to increased expression of hormone secreting cells and their respective hormones, such as glucagon-like peptide-1 (GLP-1), cholecystokinin (CCK), and peptide YY (PYY) [19–21].The induced and combined increase in GLP-1, CCK, and PYY resulting from SG and RYGB may be a contributor to sustained weight loss and the potential protective effect of bariatric surgery we observed in the present study.

In our study, we did not see an increased risk of BE in SG when compared to RYGB. Prior studies have suggested that SG may lead to an increased risk of worsening or even inducing GERD and BE after variable time periods of follow-up. In two studies from Europe, after a follow-up of 5 years, it was shown that between 17 and 19% of patients who underwent SG developed de-novo BE [22, 23]. In the Swiss Multicenter Bypass or Sleeve Study (SM-BOSS) randomized clinical trial, gastric reflux worsened more often after SG (31.8%) than after RYGB (6.3%). Nine patients were converted from SG to RYGB during the 5

years of follow-up, and one patient developed de novo BE [24]. On the other hand, 10-year follow-up results of the Sleeve vs Bypass (SLEEVEPASS) randomized clinical trial found no significant difference in rates of de novo BE (4 of 91 patients (4%) after SG vs. 3 of 85 (4%) after RYGB; p-value = 0.29) [25].. In the present study, we observed that there there was no significant difference in risk of de novo BE after either SG or RYGB. However, there exist a few caveats that need to be mentioned. First, the duration of follow-up of these SG and RYGB patients is unclear from our study. Second, it is unclear if patients in the SG group lost more weight and sustained this weight loss over a period of time when compared to the RYGB group. Third, it is also unclear if patients who underwent SG had a revision to RYGB at a later time due to possible side effects or inadequate weight loss.

It is challenging to find and define an ideal control group for a study such as this which is trying to determine the risk of diagnosis of a rare disease like BE. Population based controls who haven't had bariatric surgery would be preferable, however, when an attempt to use population controls in a large national database such as TriNetX which has over 95 million patients would create a disproportionately large number of controls when compared to cases. As such, we felt that using screening colonoscopy controls who were also undergoing an EGD on the same day would be preferable as these patients would approximate population controls. There is precedence for using screening colonoscopy patients as controls in studies related to Barrett's esophagus [26, 27], though it has to be mentioned that in these studies, patients undergoing screening colonoscopy were recruited to undergo EGD and they were not necessarily symptomatic. On the other hand, due to the de-identified, aggregate nature of the data available to us from TriNetX, it was not possible to identify reasons for these screening colonoscopy patients to be undergoing an endoscopic evaluation at the same time as their screening colonoscopy. Presumably, this was driven by symptoms such as GERD or dyspepsia, however, this is not exactly clear, and as such, is a limitation of the present study.

The study has other limitations that need to be acknowledged, several of which are related to the nature of the database itself. An important limitation is the lack of histopathology data inthe TriNetX database, and the reliance on ICD10 and CPT codes for diagnoses and procedures. However, we assume that ICD10 and CPT codes were assigned accurately and agreed with patients' actual state of health and disease status. Other limitations may include the potential duplication of patient data when a patient moves from one HCO to another, however, TriNetX does have mechanisms in place to remove duplicates. Lastly, there was a lack of genetic information and inability to perform additional analyses looking at contributions from possible genetic susceptibility to BE [28]. However, any such impact of genetic risk factors for BE was controlled by the exclusion of a personal and family history of gastrointestinal malignancies.

Conclusion:

The present study demonstrated a reduced risk of new diagnosis of BE following the two commonest types of bariatric surgery when compared to screening colonoscopy controls. The protective effect of bariatric surgery on BE is likely multifactorial, but appears to be mediated by substantial reductions in weight and BMI.

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Table 1:

Baseline information about Bariatric surgery cases and Screening colonoscopy controls

Demographics				
	Before Matching		**After Matching**	
	Bariatric Surgery Cases	**Screening Colonoscopy Controls**	**Bariatric Surgery Cases**	**Bariatric Control Controls**
Sample (N)	25,102	25,247	8,080	8,080
Age at index	43.9 ± 12.0	60.2 ± 8.4	54.9 ± 8.5	55.4 ± 6.6
Female	20,121 (80%)	14,186 (56.2%)	5,628 (69.7%)	5,820 (72%)
Male	4,980 (20%)	11,060 (43.8%)	2,451 (30.3%)	2,260 (28%)
White Race	17,143 (68.3%)	18,528 (73.4%)	5,924 (73.3%)	5,913 (73.2%)
Black Race	3,201 (12.7%)	2,949 (11.7%)	959 (12%)	1,036 (12.8%)
BMI	42.7 ± 5.4	28.5 ± 6.5	41.8 ± 6.1	29.6 ± 7.0
Weight (lbs)	274 ± 62.8	185 ± 46.2	267.2 ± 65.1	186.3 ± 49.6
Diagnoses				
GERD	12,349 (49.2%)	15,126 (60%)	4,479 (55.4%)	4,432 (55%)
Nicotine dependence	2,552 (10.2%)	3,626 (14.4%)	962 (12%)	991 (12.3%)
Diabetes mellitus	8,081 (32.2%)	5,458 (21.6%)	2,439 (30.2%)	2,646 (32.7%)
Diaphragmatic hernia	4,374 (17.4%)	7,674 (30.4%)	1,911 (23.7%)	1,831 (22.7%)
Medications				
Proton pump inhibitors (PPI)	15,240 (60.7%)	10,505 (41.6%)	4,262 (52.7%)	4,562 (56.5%)
Metformin	4,831 (19.2%)	2,440 (9.6%)	1,189 (14.7%)	1,239 (15.3%)
Sulfonylureas	1,597 (6.3%)	1,093 (4.3%)	538 (6.7%)	528 (6.5%)
Insulin	7,525 (30%)	2,047 (8.1%)	1,469 (18.2%	1,461 (18.1%)

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Table 2.

New diagnosis of BE after 1 year following bariatric surgery

Before Matching					After Matching				
Bariatric surgery vs. screening colonoscopy									
Bariatric surgery cases	**BE**	**Screening colonoscopy controls**	**BE**	**OR (95% CI)**	**Bariatric surgery cases**	**BE**	**Screening colonoscopy controls**	**BE**	**OR (95% CI)**
25,186	112 (0.44%)	24,952	332 (1.33%)	0.33 (0.27,0.41)	8,080	60 (0.74%)	8,080	85 (1.1%)	0.67 (0.48, 0.94)
Sleeve Gastrectomy (SG) vs. Roux-en-Y gastric bypass (RYGB)									
SG	**BE**	**RYGB**	**BE**	**OR (95% CI)**	**SG**	**BE**	**RYGB**	**BE**	**OR (95% CI)**
10,197	37 (0.36%)	14,747	73 (0.5%)	0.73 (0.5, 1.1)	8,924	35 (0.4%)	8,924	45 (0.5%)	0.77 (0.5, 1.2)

Table 3.

Metabolic outcomes after 1 year following bariatric surgery or screening colonoscopy

Outcome	Before Matching					After Matching				
	Cases	Mean (SD)	Controls	Mean (SD)	p-value	Cases	Mean (SD)	Controls	Mean (SD)	p-value
Weight (lbs)	25,186	214.8 (57)	26,544	182.8 (47.4)	< 0.0001	8,080	211.7 (55.5)	8,080	187.1 (50)	< 0.0001
BMI	25,186	34.7 (7)	26,544	29.0 (6.4)	< 0.0001	8,080	34.3 (6.9)	8,080	30.2 (7)	< 0.0001