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American Society for Metabolic and Bariatric Surgery literature review on risk factors, screening recommendations, and prophylaxis for marginal ulcers after metabolic and bariatric surgery

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Abstract

Background: Marginal ulcers (MU) are a significant postoperative complication following anastomotic metabolic and bariatric surgeries including Roux-en-Y gastric bypass (RYGB), one-anastomosis gastric bypass (OAGB), and biliopancreatic diversion with duodenal switch (BPD/DS). This review summarizes current knowledge on MU risk factors, screening, and prophylactic strategies.

Objectives: The goal of this review is to examine technical and patient-related risk factors for MU, assess screening strategies, and recommend prophylactic approaches to reduce MU incidence after anastomotic metabolic and bariatric surgery (MBS).

Setting: A comprehensive review was conducted by members of the American Society for Metabolic and Bariatric Surgery (ASMBS) Clinical Issues Committee, based on available literature from 2000 to the present.

Methods: A systematic search was performed using Ovid MEDLINE and PubMed databases. Relevant studies were screened for inclusion. Technical and patient-related factors were evaluated, and recommendations for MU prevention were formulated.

Results: Several risk factors for MU were identified, including large gastric pouch size, circular stapled anastomoses, use of nonabsorbable sutures, smoking, nonsteroidal anti-inflammatory drugs use, and immunosuppression. While prophylactic proton pump inhibitor (PPI) therapy is widely recommended, its optimal duration remains debated. The role of *Helicobacter pylori* in MU development is not clearly defined.

Conclusions: Prophylactic PPI therapy for at least 3 months postsurgery significantly reduces the risk of MU. Risk stratification and individualized treatment plans are essential to minimize postoperative complications. Further research is needed to clarify the role of *H. pylori* and optimize

Preamble: This review is in response to inquiries made to the society regarding marginal ulcers in patients who have had metabolic and bariatric surgery. These recommendations are based on current clinical knowledge, expert opinion, and published peer-reviewed scientific evidence available at this time. The paper is not intended to establish a local, regional, or national standard of care. The paper will be revised in the future as additional evidence becomes available.

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Keywords: Metabolic and bariatric surgery; Ulcer; Marginal ulcer; Complications

Marginal ulcer (MU) is a term used to describe ulcers that form in proximity to an anastomosis created during metabolic and bariatric surgery (MBS). Historically, the term marginal ulcer was used to describe ulcers that formed after Roux-en-Y gastric bypass (RYGB), typically on the jejunal side of the gastrojejunostomy. The term now has expanded to include anastomotic ulcers found after one-anastomosis gastric bypass (OAGB), and, less commonly, after biliopancreatic diversion with duodenal switch (BPD/DS) or single anastomosis duodeno-ileostomy with sleeve gastrectomy (SADI-S). The term marginal ulcer does not apply to ulcerative disease found after sleeve gastrectomy (SG) in the distal esophagus (related to gastroesophageal reflux disease) or along the sleeve staple line. Such ulcerative diseases after SG are a separate phenomenon and are outside the scope of this review.

This review summarizes the current knowledge about MU after MBS and gives practical recommendations for providers who treat them. We first review risk factors for MU. Second, we review screening strategies for MU after MBS. Third, we describe prophylactic strategies to reduce the risk of MU.

Methods

A literature search was conducted using Ovid MEDLINE using the following terms: “bariatrics,” “bariatric surgery,” “gastric bypass,” “gastroplasty,” “jejunoileal bypass,” “sleeve gastrectomy,” “gastric band,” “biliopancreatic diversion,” “duodenal switch,” “gastric balloon,” “intragastric balloon,” “vagal nerve block,” “transoral outlet reduction,” and “peptic ulcer perforation,” “stomach ulcer,” “duodenal ulcer,” “peptic ulcer,” “ulcer,” “peptic ulcer hemorrhage,” “anti-ulcer agents,” “*Helicobacter pylori*,” and “postoperative period,” “postoperative care,” “postoperative complications,” or “postoperative”. To ensure capture of all pertinent articles, a PubMed search for “bariatric surgery” and “postoperative ulcer” was also completed and the results from both search platforms were merged and deduplicated. All searches were limited to human subjects and English language as well as papers published from 2000 till present.

Abstracts were screened by members of the American Society of Metabolic and Bariatric Surgeons (ASMBS) Clinical Issues Committee. Selected studies could be of any design. Authors were free to add additional articles that met relevance to the topic outside of this search if they saw fit. The manuscript was peer-reviewed by the membership of the Clinical Issues Committee and then approved by the ASMBS Board of Directors prior to submission for publication.

Risk factors for marginal ulcer

Technical factors

Several reports have investigated the effects of various technical factors on the performance of RYGB and its subsequent rate of MU. These technical factors include pouch size, technique of gastrojejunal anastomosis (and type of suture, if any, used when creating the gastrojejunal anastomosis). A larger gastric pouch in RYGB has been shown to be associated with a higher incidence of MU. For example, Edholm et al. reported on 14,168 RYGB patients with 1-year follow-up and reported an overall ulcer rate of .9%; the relative risk of ulcer formation increased by 14% [95% confidence interval (CI) 9–20%] for every 1-cm increase in pouch size [1]. This study measured pouch size by counting the total number and length of linear stapled cartridges used to create the gastric pouch. The mean staple line length was 145 mm (3 stapler cartridges) [1]. In a case-control study, Ayuso et al. matched 122 patients with or without MU after RYGB. They reported that larger pouch size, as measured by 3-dimensional CT volumetry, correlated with a higher rate of ulceration [2]. When stratified for pouch size, each 5 cm^3 increase in pouch size resulted in 2.4 times odds increase of MU formation [2]. In summary, the literature supports the concept that a larger gastric pouch may increase the risk of MU after RYGB.

Another technical factor that may affect the risk of MU is the technique of gastrojejunal anastomosis, whether linear stapled, circular stapled, or handsewn [3–9]. Edholm et al. examined 34,284 patients from the Scandinavian Obesity Registry [3]. The study found that a circular stapled gastrojejunostomy had 3.1- fold (95% CI 1.8–5.3) increased odds of MU compared to linear stapled gastrojejunostomy [3]. Similarly, Sundaresan et al. examined 1112 patients after RYGB and compared linear stapled, circular stapled, and handsewn gastrojejunostomies concerning subsequent ulcer formation [10]. Circular stapled anastomoses had an associated 9.3% MU rate, whereas linear stapled had a 4.8% rate, robotic handsewn had a 5.8% rate ($P < .05$) [10]. A recent systematic review by Fakas et al. included eleven studies published between 2015 and 2019 and included 135,899 patients that underwent RYGB; 4 studies reported that a circular stapled anastomosis had statistically significant higher rates of MU when compared to handsewn and linear stapled

techniques [3]. It is worth noting that not all studies have demonstrated increased MU rates associated with circular stapled gastrojejunostomy. Abellan et al. conducted a randomized controlled trial with 2-year follow-up in 238 patients and reported similar rates of MU in circular stapled and handsewn gastrojejunostomy after RYGB [5]. Collectively, the literature concludes a circular stapled gastrojejunostomy may have a higher associated MU rate than other techniques for gastrojejunal anastomosis creation in RYGB.

Another technical variable that may impact the risk of subsequent MU is the use of permanent suture in the creation of the gastrojejunostomy. Sacks et al. examined the incidence of marginal ulcer in 3285 patients after RYGB and reported a 2.6% ulcer rate with nonabsorbable suture versus a 1.3% rate with absorbable suture ($P < .001$) and recommended using absorbable suture for the inner layer of the gastrojejunostomy [6]. Similarly, Vasques et al. found a lower incidence of marginal ulcers after RYGB with absorbable suture (2.3%) versus nonabsorbable suture (13.4%, $P < .05$) [7]. These studies suggest that an absorbable suture may be preferable when performing gastrojejunostomy to decrease MU risk.

Patient factors

Several studies have examined patient factors that may contribute to the risk of MU. These patient factors include the use of prophylactic proton pump inhibitors (PPIs), nonsteroidal anti-inflammatory drugs (NSAIDs), smoking, immunosuppression, and *Helicobacter pylori* infection. For example, Wennerlund et al. evaluated the impact of PPI use on MU after RYGB on 37,301 patients in the Scandinavian Obesity Registry [11]. Patient-related factors associated with MU formation were type 2 diabetes, smoking, immigrant background, large pouch size, and longer operative times [11]. Interestingly, the use of PPIs did not decrease the risk for MU or stricture in this study, and as a result, the authors concluded that smoking cessation was more important than routine use of PPIs after RYGB [11]. The study did not protocolize PPI use or examine the duration of therapy, which may be an important factor with prophylaxis. A recent survey done by the ASMBS research committee that examined PPI use after MBS in the United States showed great variability in PPI use: only 44% of patients were on PPIs for at least 3 months, and only 19% for 6 months after RYGB [12]. More information on PPI use as a prophylactic strategy is written later in this review.

Similar to the report from the Scandinavian Obesity registry by Wennerlund et al., smoking was identified in multiple reports as a risk factor for MU. Spaniolas et al. studied outcomes of 35,075 patients after RYGB from the New York state administrative registry and found that the overall incidence of MU was 11.4% at 8 years of follow-up, and smoking increased the hazard ratio for MU development by 56% [13]. Indeed, 17.8% of smokers developed MU within 8 years of RYGB [13]. Similarly, Dittrich et al., in a small retrospective single institution study, reported that smoking was associated with a 4.6-fold increased risk for MU after RYGB compared to nonsmokers, and even light smokers (<10 cigarettes per day) were not spared from the increased risk [14]. Athanasiadis studied 766 patients' smoking habits after RYGB and found that 65% of recent smokers resumed smoking after RYGB, of whom 51% developed MU, compared to a 15% MU rate in those who did not resume smoking and a 6% rate in those who never smoked [15]. Additionally, a recent systematic review found that smoking within 1 year prior to RYGB was an independent risk factor for complications including MU [16]. Based on current literature, patients with a history of smoking would be advised to undergo smoking cessation prior to considering anastomotic procedures like RYGB or OAGB, or alternatively, consider sleeve gastrectomy as a nonanastomotic option. [16].

Immunosuppression and NSAIDs have been shown to increase MU risk [17]. Di Palma et al. reviewed 2830 patients who underwent RYGB and reported an MU rate of 6.9% over a mean follow-up of 24 months. In this study, immunosuppression use was associated with a 4.6-fold increased risk of ulceration, and NSAIDs were associated with a 3.1 increased risk of ulceration [17]. Similarly, Coblijn et al. studied MU in a population of 350 RYGB patients and found that both corticosteroids and NSAIDs were associated with increased risk [18]. In summary, the literature supports both immunosuppression and NSAIDs as contributors to MU.

H. pylori

The effect of *Helicobacter pylori* infection as a risk factor for MU has been investigated with conflicting results. Supportive literature includes a meta-analysis of 7 studies with more than 255,000 patients that determined *H. pylori* to be the largest independent predictor of MU in patients undergoing MBS, with a tenfold increase compared to *H. pylori*-negative patients [19]. However, 5 of the 7 studies included were retrospective, and only one was in RYGB patients. Similarly, a review of the Nationwide Inpatient Sample administrative database of 253,765 patients with a previous history of MBS reported an MU rate of 3.9% [20]. Of these, 31.2% had *H. pylori* infection and it was considered the strongest predictor for MU with an adjusted odds ratio of 10.9, far stronger than smoking (odds ratio 1.3) in this population [20]. However, the conclusions of this study are to be interpreted with caution because the definition of MBS included sleeve gastrectomy, laparoscopic adjustable gastric banding, and vertical banded gastroplasty – operations without an anastomosis and, therefore, not at risk for MU. In addition, it was not clear how *H. pylori* infection was determined since this was a retrospective review of an administrative database. Furthermore, the study

did not evaluate or factor in gastric pouch size, technique of gastrojejunal anastomosis, or steroid or NSAID use. Finally, the study design started by identifying MU and then evaluated how many were *H. pylori* positive rather than first identifying patients who were *H. pylori* positive and then determining how many went on to develop MU [21]. A recent systematic review and meta-analysis identified fourteen studies with 344,829 patients and showed that *H. pylori* infection (odds ratio = 4.97), smoking (odds ratio = 2.50), and diabetes mellitus (odds ratio = 1.80) were significant predictors of MU, whereas increased age, body mass index, female gender, obstructive sleep apnea, hypertension, and alcohol use were not predictors [22]. Finally, Rasmussen et al. demonstrated that despite *H. pylori* eradication preoperatively, *H. pylori* infection prior to MBS was more than twice as likely among patients who developed MU compared to those who did not (32% versus 12%; P = .02) [23]. Concluding from these large-scale administrative database studies and their meta-analysis is difficult because they may conflate the MU, which requires an anastomosis, whereas peptic ulcers do not.

On the other hand, a number of reports have questioned the role of *H. pylori* in the development of MU after MBS [21,24–27]. Some investigators have noted that MU typically occurs on the jejunal side of the gastrojejunostomy (because the jejunum naturally has little defense against stomach acid), yet *H. pylori* itself cannot infect jejunal mucosa [21]. For example, Kelly et al. performed intraoperative biopsies to look for H. *pylori* in 708 patients undergoing RYGB and did not prescribe *H. pylori* eradication therapy when *H. pylori* was found, but rather, gave 1 year of a histamine-receptor blocker prophylaxis in all patients [21]. The study found that in the *H. pylori* positive group, 8% of patients developed MU, compared to 17% in the *H. pylori* negative group (P = .05) [21]. Another study casting doubt on the role of *H. pylori* in the pathogenesis of MU was published by Rawlins et al., who demonstrated a 30% preoperative *H. pylori* positivity rate in their study population, of which 35% remained positive despite medical therapy [27]. Postoperatively, 5 patients developed MU. There was only one MU derived from the 44 patients who responded to *H. pylori* therapy, and no complication in the cohort that did not respond, leaving 4 MU in patients who did not test positive for *H. pylori* preoperatively. Similarly, an analysis by Yang et al. demonstrated no difference in *H. pylori* seropositivity between symptomatic and asymptomatic patients after MBS (39% versus 39.7%) [25]. Of the symptomatic patients, 27% had evidence of gastric ulcers identified with endoscopy, but within this cohort, there was no difference in *H. pylori* seropositivity (43% versus 27%, P = .21) comparing patients with no ulcer [n = 60] versus ulcer [n = 22], respectively. A systematic review and meta-analysis examined the impact of *H. pylori* on complications after MBS [28]. The review examined 3 prospective and 18 retrospective studies, categorizing them into those that described pre-operative *H. pylori* eradication versus no eradication. The review demonstrated comparable MU rates between the *H. pylori*-positive and *H. pylori* -negative patients in studies describing preoperative eradication and increased MU rates in *H. pylori* positive versus negative patients (31% versus 6%) in studies without eradication. However, meta-analysis of incidence of MU and *H. pylori* infection demonstrated an odds ratio of .508 (95% confidence interval .031–8.346; P = .63). As noted by the authors, this discrepancy may relate to the heterogeneity of study designs and testing methods.

Taken as a whole, the literature is mixed as to whether *H. pylori* infection is indeed a risk factor for MU specifically; large administrative databases and meta-analyses have shown a strong association while smaller trials with a more controlled methodology have shown no association, or even a protective effect. Based on the current literature, no strong recommendation can be made for or against H. pylori testing prior to MBS to prevent MU specifically.

OAGB and BPD/DS

Just as seen after RYGB, patients may develop MU after OAGB, possibly due to a larger gastric pouch, even though anatomically, the gastrojejunostomy of OAGB is bathed with pancreatic alkaline juice that theoretically can neutralize gastric pouch acid and protect the jejunum [11,29,30]. The incidence of MU in OAGB patients has been reported to range from .5 to 8% [31–35]. In comparative trials, OAGB has not been shown to have a lower risk of MU when compared to RYGB [34]. MU has also been reported in patients after BPD/DS. Bekhali and Sundbom reported on the outcomes of 472 patients who underwent BPD/DS and reported a 1.3% risk of MU per year over a 6-year mean follow-up, similar to the risk seen after RYGB at the same center [36]. In summary, MU can occur after both OAGB and BPD/DS and the magnitude of the risk has not been shown to be different from that of RYGB.

Reoperative procedures

Authors have reported a higher incidence of MU after conversional or revisional RYGB compared to primary RYGB. For example, Anderson et al. reported results from 164 patients who were converted to RYGB after a sleeve gastrectomy or gastric band and compared them to 584 patients who underwent primary RYGB. The MU was 14% in patients who converted from sleeve to RYGB, 6% after primary RYGB, and 3% in patients who converted from band to RYGB [37].

Gastrogastric fistula

Gastrogastric fistulas have been shown to be associated with MU [38]. There are 2 possible mechanisms. First, if the gastric pouch was not completely divided from the gastric remnant during the creation of the gastric pouch in RYGB and OAGB, persistent communication between the

2 can increase the acid load into the pouch and promote ulcer formation. Second, a MU of the gastrojejunostomy can perforate into the gastric remnant located adjacent to the gastrojejunostomy, producing a gastrogastric fistula [38]. Since gastrogastric fistulas can be missed during routine upper endoscopy, some surgeons recommend obtaining an upper gastrointestinal series or computed tomography (CT) with oral contrast prior to operating on refractory MU to look for gastrogastric fistula. Such investigations can also assess pouch size as a contributor to MU.

The identified risk factors for MU are summarized in Table 1.

Screening for ulcer disease after MBS

Evidence for screening by history and physical at recommended follow-up intervals

Little evidence exists to support screening for MU using patients' medical history to identify the most at-risk individuals. Bhayani et al., retrospectively analyzed 763 patients and demonstrated a 3% MU rate over a mean of 64 months [39]. Univariate analysis demonstrated a significantly greater proportion of gastroesophageal reflux disease (48% versus 26%; $P = .02$), hyperlipidemia (52% versus 30%; $P = .02$), sleep apnea (39% versus 18%; $P = .01$), and hypertension (91% versus 56%; $P = .001$) in the group that developed MU compared to those without. Multivariate analysis demonstrated only hypertension as independently associated with the development of MU (odds ratio = 7.84, CI: 1.75–35.06; $P = .007$). The value of using hypertension as a possible screen is limited because only symptomatic patients underwent endoscopy, precluding the ability to assess the sensitivity or specificity of this risk factor.

Evidence for routine esophagogastroduodenoscopy in asymptomatic patients

The diagnosis of MU is generally made when a postoperative patient complains of epigastric pain, nausea, vomiting, or food intolerance. However, evidence suggests that symptoms do not necessarily correlate with objective disease.

Table 1
Risk factors for marginal ulcer

Technical factors	Large gastric pouch
	Circular stapled anastomosis
	Nonabsorbable suture in creation of gastrojejunostomy
Patient factors	Smoking
	NSAIDs
	Corticosteroids and other immunosuppressants
	Type 2 diabetes
	Prior metabolic and bariatric surgery
	Occult gastrogastric fistula

NSAIDs = nonsteroidal anti-inflammatory drugs.

Specifically, Huang et al. retrospectively evaluated 49 symptomatic patients between 5 and 703 weeks (median of 77 weeks) post-RYGB to correlate clinical features to endoscopic findings. They determined that the most common findings were normal anatomy (n = 21; 43%) followed by MU (n = 13; 27%). Further, the most common symptom of abdominal pain (n = 26; 53%) was most frequently associated with normal anatomy ($P = .04$), and while only 15% of endoscopies performed ≦6 months postoperative were normal, the number increased to 53% after 6 months postoperative ($P = .02$) [40]. Marano et al. retrospectively evaluated endoscopic findings in symptomatic patients post-RYGB and determined that MU was the most common finding (n = 12/23; 52%), followed by normal anatomy (n = 7/23; 30%) [41]. Boerlage et al. retrospectively examined endoscopic findings of symptomatic patients after RYGB [42]. Of the 250 patients examined, only 39% had relevant pathology, of which MU comprised the majority (n = 46/250; 18%) [42]. Conjointly, these results suggest that symptoms do not correlate with pathology. Indeed, Csendes et al. conducted a prospective study following 550 patients post-MBS with symptom questionnaires and upper endoscopy 1–8 years after surgery to determine the incidence of later MU, and reported a 1% rate (n = 6) between 12–4 months after surgery. Interestingly, analysis of the symptom questionnaire elucidated two types of epigastric pain. Typical peptic ulcer pain, present in 83% of patients, localized to the epigastrium, was persistent and woke patients at night. Conversely, atypical pain, presenting in 4.7% of patients, was experienced as upper abdominal pain without features of peptic ulcer pain. In this group of patients, the upper endoscopy was normal [43]. Based on the existing literature, it seems prudent to perform upper endoscopy when significant symptoms are present, recognizing that many patients will not have an identified source for their symptoms at endoscopy.

Interestingly, MU can be present without clinical symptoms. Garrido et al. conducted a multicenter, prospective, nonrandomized trial to assess the incidence of MU and dyspeptic symptoms within the first 2 months of MBS. Examining 118 patients, the incidence of MU was 8%, and no patient with MU described epigastric pain, heartburn, nausea, vomiting, or abdominal pain [44]. Spinosa et al. conducted a prospective study examining endoscopic findings 1-year after RYGB in 715 asymptomatic patients. While most patients had normal findings, 27% exhibited endoscopic abnormalities including, esophagitis (10%), jejunitis (10%), MU (3.6%), or stenosis (1.2%) resulting in treatment modification [45]. Finally, Csendes et al. prospectively evaluated the incidence of ulcer formation 1-month and 17 months after surgery in 441 patients. After surgery, 6% of patients developed early MU, of which 28% were asymptomatic [46]. Although these studies document the existence of asymptomatic MU, most surgeons do not perform routine upper endoscopy because the reported

prevalence of asymptomatic MU is low and prophylaxis is available (see below), reserving upper endoscopy for symptomatic patients. Hence, the threshold to perform upper endoscopy after RYGB and OAGB may affect the reported incidence of MU.

Prophylaxis against marginal ulcer

The primary strategy to prevent MU after MBS is to prescribe PPIs in the early postoperative period. Overall, early postoperative PPIs have been shown to decrease the odds of MU after RYGB by 50–70% (adjusted odds ratio of .3 to .5) and decrease the overall incidence of MU from 7.3% to 1.2% [47,48]. There is a strong consensus amongst surgeons and experts for PPI prophylaxis; >90% of surgeons routinely prescribe postdischarge PPI after RYGB [12,48]. Postoperative PPI prophylaxis is recommended for all patients after RYGB and may be prudent for patients undergoing other anastomotic MBS procedures where MU are known to occur.

Prophylaxis duration

The risk of MU formation is highest immediately after surgery and decreases over time but never drops to zero [14,46,50–51]. Most ulcers are diagnosed within the first year [14,50,51]. In one meta-analysis, MU presented at a median of 8–14 months after MBS, and the incidence decreased over time [48,52,53]. Based upon this natural history of MU, surgeons generally agree that early prophylaxis is needed, but duration is not universally agreed upon or supported by robust data [12,49]. Two recent surveys of surgeons reported that 18–29% recommended 1 month of PPIs, 37–45% recommended 3 months, and 18–27% recommended 6 months [12,49]. One study compared the impact of 30 versus 90-day PPI prophylaxis on the incidence of MU after RYGB and demonstrated that patients receiving a daily PPI for 90 days had an ulcer rate of 6.5%, whereas a shorter 30-day regimen had a two-fold increase in ulcer rates at 12.4% [47]. Based on the existing literature, we recommend routine postoperative PPI prophylaxis for all patients after RYGB and other anastomotic MBS procedures for at least 3 months.

Patients with identifiable risk factors for MU may warrant more extended PPI therapy. Such patients include those on NSAIDs, active or recent smokers, patients on immunosuppression, and patients with a personal history of MU.

Conclusions

MU represents a significant source of patient morbidity after anastomotic MBS and may be diagnosed early after the operation or years later.

Risk factors for MU include technical factors, such as large pouch size, circular stapled anastomosis, or permanent suture used to create the anastomosis, as well as patient factors, such as smoking, NSAIDs, corticosteroids or other immunosuppressants, diabetes mellitus, prior gastric surgery, and occult gastrogastric fistula. The role of *H. pylori* in the pathogenesis of MU continues to be controversial and is confounded by reports in the literature that conflate MU with other forms of peptic ulcer disease known to be caused by *H. pylori*.

Prophylaxis against MU with PPIs is effective, reduces ulcer rates by 50–70%, and is generally prescribed in the early postoperative period (e.g. the first 3 months) when the risk of ulceration is the greatest.

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