Received: 5 October 2023 | Accepted: 24 November 2023

DOI: 10.1097/CLD.0000000000000139

REVIEW

The impact of alcohol on patients after bariatric surgery

Rahul Grover[1] | Brett E. Fortune[2] | Clara Y. Tow[2]

[1]Department of Medicine, Albert Einstein College of Medicine/Montefiore Medical Center, Bronx, New York, USA

[2]Department of Medicine, Division of Hepatology, Albert Einstein College of Medicine/Montefiore Medical Center, Bronx, New York, USA

Correspondence
Clara Y. Tow, Department of Medicine, Division of Hepatology, Albert Einstein College of Medicine/Montefiore Medical Center, 1575 Blondell Avenue, Suite 100, Bronx, NY 10461, USA.
Email: ctow@montefiore.org

INTRODUCTION

The prevalence of obesity and metabolic dysfunction continues to rise in the United States. Bariatric or weight loss surgery (WLS) is a highly effective therapeutic option for sustained weight loss in patients with severe obesity or obesity with concomitant metabolic disease.[1] However, a growing body of literature suggests an increased risk of alcohol use disorder (AUD) and alcohol-associated liver disease (ALD) in patients who have undergone bariatric procedures. In this review, we discuss the current literature on this phenomenon, theorized mechanisms, and potential risk mitigation strategies for bariatric surgery candidates.

THE FUNDAMENTALS OF BARIATRIC SURGERY

Weight loss in bariatric surgery is primarily achieved through a combination of restricting gastric capacity, disrupting nutrient absorption, and altering neurohormonal regulation. Sleeve gastrectomy (SG) and the Roux-en-Y gastric bypass (RYGB) are the 2 most commonly performed bariatric procedures worldwide.[1] SG involves the creation of a small, tubular gastric reservoir, or "sleeve," by resecting nearly 80% of the greater curvature of the stomach (Figure 1A). In contrast, RYGB requires more complex anatomical disruptions. First, the stomach is divided into a large distal portion and a significantly smaller proximal pouch. The small intestine is then divided ~50–150 cm from the Ligament of Treitz. The distal portion of the divided jejunum is anastomosed to the gastric pouch to create an alimentary or Roux limb through gastrojejunostomy. Meanwhile, the distal stomach remains connected to the duodenum and proximal jejunum which is then anastomosed through a jejunojejunostomy distally to the gastrojejunostomy creating a common channel where biliary and pancreatic enzymes can continue to facilitate digestion (Figure 1B).

Vertical banded gastroplasty was a historically common but now rarely performed procedure that involved the vertical partitioning of the proximal stomach into a smaller pouch with a restricted outlet bound by mesh or a prosthetic band (Figure 1C).[2] Open or laparoscopic adjustable gastric banding (AGB) is another now rarely conducted procedure that involves the placement of a prosthetic band at the entrance of the stomach to create a restricted compartment (Figure 1D).

THEORIZED MECHANISMS OF INCREASED AUD AND ALD RISK AFTER BARIATRIC SURGERY

Several anatomic, physiologic, and psychosocial mechanisms have been proposed to explain the observed association between bariatric surgery and subsequent development of AUD and ALD (Figure 2). The "addiction transfer model" posits that one behavior, overeating, is replaced by another, alcohol use, as compensation for the mechanical inability to indulge in large volumes of food after bariatric surgery.[3] While the construct of food addiction remains controversial, the proponents of this model suggest that a similar neurophysiological profile of the dopamine-associated reward pathways is triggered in both food-seeking and alcohol-seeking behavior as

Abbreviations: AGB, adjustable gastric banding; AH, alcohol-associated hepatitis; ALD, alcohol-associated liver disease; ASMBS, American Society for Metabolic and Bariatric Surgery; AUD, alcohol use disorder; RYGB, Roux-en-Y gastric bypass; SG, sleeve gastrectomy; WLS, weight loss surgery.

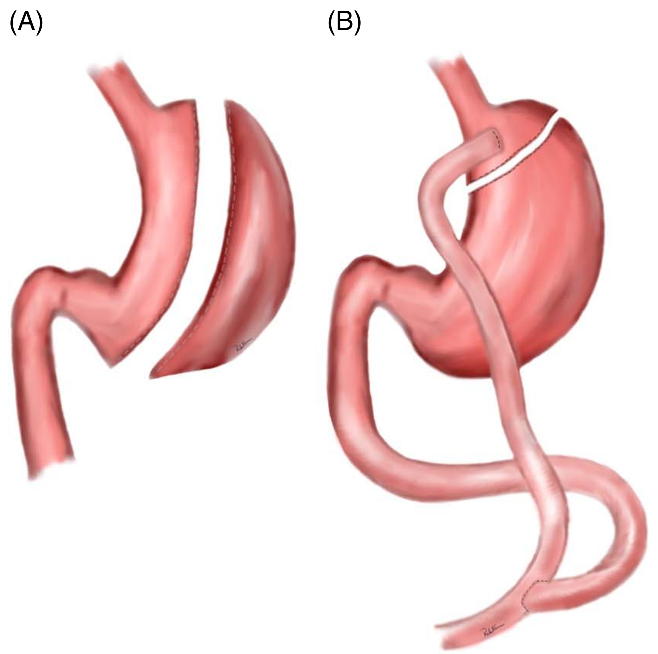

FIGURE 1 Anatomical depictions of various bariatric procedures. (A) Sleeve gastrectomy (B) Roux-en-Y gastric bypass (C) Vertical banded gastroplasty (D) Adjustable gastric banding.

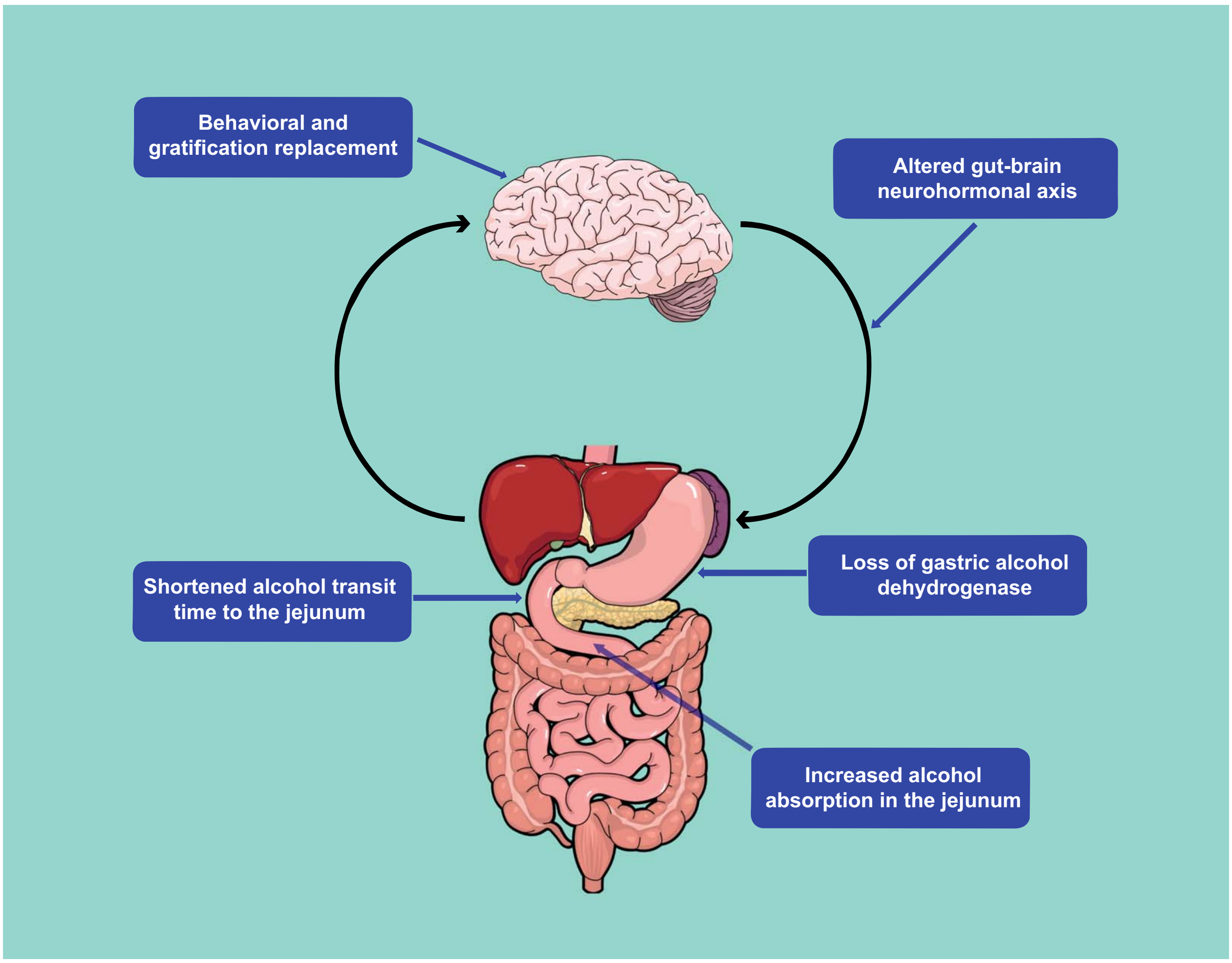

FIGURE 2 Theorized mechanisms of increased AUD and ALD risk after bariatric surgery.

shown in various animal studies.[4] Changes in the gut-brain neurohormonal axis due to anatomical disruption and subsequent downstream dysregulation of digestive hormones such as ghrelin, leptin, glucagon-like peptide-1, and peptide YY have also been proposed as a potential basis for this phenomenon.[5]

Changes in the pharmacokinetic processing of alcohol after bariatric procedures may further augment these behavioral changes by simulating surreptitious binge drinking and exposing patients to higher-than-expected doses of alcohol over a shorter time span. For example, several small physiologic studies have shown that subjects with a history of gastric bypass achieve a higher and faster peak blood alcohol concentration, often in as little as 2–10 minutes of consumption.[6,7] Furthermore, it takes longer for the blood alcohol concentration to reach baseline in subjects with a history of bariatric surgery as compared to nonsurgical controls.[8] Although direct evidence is lacking, these changes in alcohol metabolism are likely due to a dramatically reduced transit time from ingestion to alcohol absorption in the jejunum. Alcohol degradation may also be partially diminished by the loss of gastric alcohol dehydrogenase. Combined, the increased bioavailability of alcohol, altered neurohormonal reward pathways, and a psychosocial predisposition for harmful behavior are thought to elevate the risk of AUD and ALD after bariatric surgery.

EVIDENCE OF ELEVATED AUD RISK AFTER BARIATRIC SURGERY

An anecdotal rise in AUD risk after bariatric surgery has long been suggested. Although robust evidence is lacking, data from several observational studies over the past decade now support this theorized association (Table 1).

RYGB, in particular, appears to confer the greatest risk for AUD. The Longitudinal Assessment of Bariatric Surgery-2 study was one of the first and largest studies to examine alcohol consumption and frequency in patients who primarily underwent RYGB or AGB procedures. Patients with a history of RYGB reported an initial decrease or no change in drinks consumed per day, frequency of use, or prevalence of AUD during the first postoperative year followed by a significant increase by the second postoperative year as compared to a preoperative baseline (AUD prevalence: 7.0% preoperatively, 7.9% at 1-year postop, and 10.7% at 2 years postop; $p < 0.01$). No statistically significant changes in behavior or prevalence of AUD were noted in patients who underwent banding (AUD prevalence: 9.3% preoperatively, 5.6% at 1-year postop, and 7.0% at 2 years postop; $p = 0.24$). Those with a history of RYGB were twice as likely to develop AUD than those who underwent AGB (OR, 2.1; 95% CI, 1.4–3.1; $p < 0.001$).[9] Subsequent, longer-term studies have shown that this disproportionate increase in volume and frequency of alcohol consumption persists for 5–10 years and beyond after RYGB.[10,11,13] The risk of AUD and AUD-related hospitalizations also increases in the years following RYGB when compared to medical controls (HR, 1.7; 95% CI, 1.20–2.41; $p = 0.003$) and SG (HR, 1.98; 95% CI, 1.55–2.53; $p < 0.001$).[12,18]

Over the past decade, SG has now replaced RYGB as the predominantly conducted bariatric procedure in the United States. While initial data on AUD after SG were mixed due to inadequate representation in studied populations, newer studies show a similarly increased risk of unhealthy alcohol use among American veterans, American teens, and adults in Norway who have undergone either RYGB or SG.[14,15,17] Head-to-head comparisons between SG and AGB or vertical banded gastroplasty are limited due to the relative obsolescence of the latter.

EVIDENCE OF ELEVATED ALD RISK AFTER BARIATRIC SURGERY

While the benefits of WLS in the regression of steatotic liver disease are well established, data on the effect of WLS on ALD are mixed and limited by study size, design, and heterogeneity of studied outcomes.[20] Several recent studies, however, suggest that patients who undergo certain weight loss procedures are at greater risk of adverse outcomes related to ALD, specifically alcohol-associated hepatitis (AH) and alcohol-associated cirrhosis (Table 2).

A systematic review of studies exploring ALD and cirrhosis in obese patients noted a significantly lower risk of liver cancer and metabolic dysfunction–associated cirrhosis but an elevated risk of alcohol-associated cirrhosis in patients with a history of bariatric surgery compared to nonsurgical controls (HR, 1.32, 95% CI, 1.35–1.59).[24] This increased risk of cirrhosis after bariatric surgery is further supported by 2 large population-based cross-sectional studies utilizing the National Inpatient Sample and MarketScan insurance claims databases.[16,19] Outcomes may differ by type of bariatric procedure given lower hazard ratios of alcohol-associated cirrhosis in patients who underwent SG or banding.[16] Among patients admitted for alcohol-associated cirrhosis, those with a history of bariatric surgery, specifically RYGB, may have a higher risk of HE and concomitant AH.[22]

Data on alcohol-associated hepatitis outcomes remain sparse. Two single-center cohort studies suggest that short-term mortality, disease severity, or response to steroids are not impacted by a history of WLS.[23,25] However, a history of WLS in those admitted for AH experienced an increased risk of overall mortality (31.4% vs. 24%, $p = 0.03$), 30-day readmission (20.3%

TABLE 1 Summary of literature on alcohol use and alcohol use disorder after bariatric surgery

Year	Group	Study type	Intervention (n)	Main outcomes	Alcohol use disorder–related findings
2012	King et al[9]	Prospective cohort	RYGB (1360) AGB (490) RYGB + Band (30) SG (50) BPD/DS (15)	Drinks per day and frequency of alcohol consumption by AUDIT score Evidence of AUD by AUDIT score	• Drinks per day significantly greater preop and at 2 y postop than at 1-y postop in the RYGB group • Frequency of consumption and AUD significantly greater 2 y postop compared to preop or 1-y postop in the RYGB group • No significant difference in the frequency of consumption or AUD noted in the AGB group
2013	Conason et al[10]	Prospective cohort	RYGB (100) AGB (55)	Frequency of substance use and self-reported problems by CBQ	• Composite substance use initially decreased from preop to 1-mo postop, but significantly increased from preop to 24 mo postop • Frequency of alcohol consumption significantly lower at 1 and 3 mo postop compared to preop but significantly higher 24 mo postop than preop in the RYGB group • No significant difference of alcohol consumption noted in the AGB group
2013	Svensson et al[11]	Prospective controlled cohort	VBG (1369) Banding (376) GB (265) Matched controls (2037)	Alcohol consumption and self-reported alcohol problems by SOS dietary questionnaire	• Percentage of individuals reporting at least WHO medium-risk[a] alcohol consumption was highest among the GB group at all time points • Adjusted risk of at least WHO medium-risk alcohol consumption[a] and self-reported alcohol problems as compared to controls was highest in the GB and VBG groups but not in the banding group
2013	Östlund et al[12]	Retrospective population-based cohort	GB (4161) VBG or banding (6954)	Number of hospitalizations for depression, alcohol abuse, other substance abuse, and attempted suicide	• Approximately double the risk of alcohol abuse–related hospitalizations in the GB group
2017	King et al[13]	Prospective cohort	RYGB (1481) AGB (522)	Drinks per day and frequency of alcohol consumption by AUDIT score Evidence of AUD by AUDIT score Illicit substance use and treatment	• Nearly doubled the prevalence of “regular drinking” in both the RYGB and AGB groups from preop to 7 y postop • Prevalence of incident AUD more than doubled in the RYGB group but not the AGB group
2019	Strømmen et al[14]	Retrospective population-based cohort	RYGB (8196) SG (2012)	Hospitalizations for alcohol-associated diagnoses	• No significant difference seen in the incidence rate of alcohol-associated diagnoses between the RYGB and SG groups
2020	Maciejewski et al[15]	Retrospective cohort study	RYGB (924) SG (1684) RYGB matched control (8038) SG matched controls (1455)	Alcohol use by AUDIT-C	• In patients without unhealthy alcohol use at baseline, the probability of unhealthy alcohol use increased at both 2 and 8 y postop after both SG and RYGB compared to nonsurgical controls • In patients with unhealthy alcohol use at baseline, the probability of unhealthy alcohol use increased in only the RYGB group

2021	Mellinger et al[16]	Population-based cross-sectional study using the MarketScan insurance claims database	RYGB (102,385) SG (64,687) AGB (27,058) Unclassified (209,058)	Alcoholic cirrhosis and alcohol misuse	• Significantly elevated the risk of alcohol misuse after RYGB but not AGB
2022	White et al[17]	Prospective cohort	RYGB (155) SG (62)	Frequency of alcohol use, AUD, risk of alcohol use, and alcohol-associated problems by AUDIT and AUDIT-C	• Similar increase in quantity and frequency of alcohol consumption in both RYGB and SG from preop to postop
2023	Mahmud et al[18]	Retrospective controlled cohort	RYGB (1854) SG (4211) GB (265) Weigh management program (1364)	Time to AUD-related hospitalization Time to all-cause mortality	• Significantly elevated adjusted HR of AUD-related hospitalization in the RYGB vs. weight management and SG groups • No risk elevation noted in the SG as compared to the weight management group • Risk noted to be greatest at lower postop BMI • RYGB group had lowest proportion of AUDIT-C score <1 at all time points
2023	Alvarado-Tapias et al[19]	Population-based cross-sectional study using the NIS database	Bariatric surgery[b] (537,757) Abdominal surgery (537,757)[c]	Prevalence of ALD or psychiatric disorders associated with AUD	• Significantly elevated prevalence of AUD in patients in the bariatric surgery group over time as compared to the abdominal surgery group

[a]World Health Organization (WHO) medium-risk alcohol consumption = 40–60 g pure alcohol consumption per day in men or 20–40 grams in women.
[b]Patients only classified as bariatric surgery or abdominal surgery for analysis. However, 46.88% noted to have undergone RYGB, 33.76% SG, 10.66% AGB, and 8.69% an "other" form of bariatric surgery.
[c]Numbers based on propensity matching.
Abbreviations: AGB, adjustable gastric banding; AUD, alcohol use disorder; AUDIT, Alcohol Use Disorders Identification Test; BMI, body mass index; BPD/DS, biliopancreatic diversion with duodenal switch; CBQ, Compulsive Behaviors Questionnaire; GB, gastric bypass (unspecified); NIS, National Inpatient Sample; RYGB, Roux-en-Y gastric bypass; SG, sleeve gastrectomy; SOS, Swedish Obese Subjects study; VBG, vertical banded gastroplasty; WHO, World Health Organization.

TABLE 2 Summary of literature on alcohol-associated liver diseases after bariatric surgery

Year	Group	Study type	Population and intervention (n)	Main outcomes	ALD-related findings
2021	Mellinger et al[16]	Population-based cross-sectional study using the MarketScan insurance claims database	RYGB (102,385) SG (64,687) AGB (27,058) Unclassified (209,058)	Alcohol-associated cirrhosis and alcohol misuse	• Significantly decreased risk of alcohol-associated cirrhosis after SG (HR = 0.40) or AGB (HR = 0.43) but no association with RYGB (HR = 0.98) in the short term • Possibly elevated risk of alcohol-associated cirrhosis in the long term although unable to stratify by procedure type (HR = 1.31)
2021	Fipps et al[21]	Multicenter retrospective clinical cohort	Adults who underwent ALD liver transplantation and bariatric surgery: RYGB (16) AGB (2)	Descriptive characteristics including demographics, anthropometrics, and surgery type	• Patients who underwent ALD liver transplantation and had a history of bariatric surgery were younger, had a higher MELD, female sex, and comorbid mood/anxiety disorders as compared to patients without a history of bariatric surgery
2022	Yarra et al[22]	Population-based cross-sectional using the NIS database	Adults hospitalized for alcohol-associated cirrhosis with prior: RYGB (2542) Controls (7626)[b]	Discharge diagnosis of alcoholic hepatitis, acute liver failure, and mortality	• Increased risk of AH (OR, 1.14) and HE (OR, 1.42) among the RYGB group but not ACLF or in-hospital mortality
2023	Melkebeke et al[23]	Single-center prospective cohort	Adults hospitalized for biopsy-proven severe alcoholic hepatitis with prior: RYGB, BPD, or AGB (28) Controls (130)	Baseline characteristics, response to steroids, measures of disease severity, and mortality	• Patients with a history of bariatric surgery were nearly a decade younger at presentation for sAH as compared to controls • No difference noted among the groups in terms of disease severity, therapy response, or mortality
2023	Wang et al[24]	Systematic review and meta-analysis	18 studies of adults with obesity who underwent various bariatric procedures compared to those who did not Bariatric surgery (16,800,287) Controls (10,595,752)	Adverse liver disease including nonalcohol-associated cirrhosis, alcohol-associated cirrhosis, and liver cancer	• Obese patients who underwent bariatric surgery experienced significantly fewer adverse liver outcomes, including nonalcoholic cirrhosis and liver cancer, than those who did not undergo bariatric surgery (HR = 0.07) • However, in a meta-analysis of studies evaluating alcoholic cirrhosis bariatric surgery was associated with an increased risk of developing alcoholic cirrhosis (HR = 1.32)
2023	Anugwom et al[25]	Single-center retrospective cohort	Adults hospitalized for clinically diagnosed alcohol-associated hepatitis with prior: RYGB (153) Controls (2481)	Inpatient mortality	• No difference in inpatient mortality • Increased risk of 30-day readmission, overall mortality, and development of cirrhosis in RYGB group
2023	Alvarado-Tapias et al[19]	Population-based cross-sectional study using the NIS database	Bariatric surgery[a] (537,757) Abdominal surgery (537,757)[b]	Prevalence of ALD or psychiatric disorders associated with AUD	• Increased risk of ALD noted in the bariatric surgery group vs. the control surgery group (OR, 1.29) • Even greater risk of ALD in those with concomitant AUD (OR, 2.47) • Increased risk of cirrhosis in the bariatric surgery group vs. the control surgery group (OR, 1.39)

[a]Patients only classified as bariatric surgery or abdominal surgery for analysis. However, 46.88% noted to have undergone RYGB, 33.76% SG, 10.66% AGB, and 8.69% an “other” form of bariatric surgery.
[b]Numbers based on propensity matching.
Abbreviations: ACLF, acute liver failure; AGB, adjustable gastric banding; AH, alcohol-associated hepatitis; ALD, alcohol-associated liver disease; AUD, alcohol use disorder; BPD/DS, biliopancreatic diversion with duodenal switch; MELD, Model of End Stage Liver Disease; NIS, National Inpatient Sample; RYGB, Roux-en-Y gastric bypass; sAH, severe alcohol-associated hepatitis; SG, sleeve gastrectomy.

vs. 11.7%, $p<0.01$), and progression to cirrhosis (37.5% vs. 20.9%, $p<0.01$).[25]

Studies on ALD-related liver transplant outcomes after bariatric surgery are also lacking. The sole retrospective cohort study (1416 LT recipients including 18 subjects who had a history of bariatric surgery) published to date showed no impact of WLS on mortality after liver transplantation. However, patients admitted for alcohol-associated liver transplant with a history of bariatric surgery were noted to have higher MELD scores (median MELD of 22 in the bariatric surgery group vs. 18 in the nonbariatric surgery group, $p=0.02$), younger age at transplantation (median age 50 vs. 57 y, $p=0.003$), and comorbid mood/anxiety disorders.[21]

PROPOSED MITIGATION STRATEGIES

Informed by anecdotal evidence of increased substance misuse and psychiatric comorbidity, professional society guidelines have long supported a comprehensive, multidisciplinary approach to preoperative evaluation of patients referred for bariatric procedures. While alcohol screening is typically conducted during this evaluation, only recent guidelines offer additional recommendations on risk mitigation against the development of AUD after bariatric surgery. For example, a 2021 position statement by the American Society for Metabolic and Bariatric Surgery (ASMBS) acknowledges the mounting evidence of increased de novo AUD after bariatric surgery and suggests:

(1) identifying patients at greatest risk of developing postsurgical substance use disorders;
(2) educating patients on these risks, including education on the expected postoperative changes in alcohol metabolism; and
(3) monitoring patients after their procedures.[26]

Furthermore, while most surgical programs already consider active AUD a contraindication for WLS,[27] a 2021 update to the Enhanced Recovery after Surgery Society guidelines adds that a minimum period of 1–2 years of sobriety should be maintained before surgery.[28]

Despite this growing awareness of AUD risk after WLS, the risk of ALD is underacknowledged and specific risk mitigation strategies are absent from official guidelines. Additional research with larger, prospective studies of contemporary bariatric procedures is needed to better identify risk factors for patients who undergo WLS. One example of a general risk evaluation framework suggested by Mendoza et al[29] that providers could consider is:

(1) Patients with obesity and multiple metabolic risk factors should be evaluated for comorbid liver fibrosis by imaging (elastography) or noninvasive testing (eg, fibrosis-4 index for liver fibrosis and/or enhanced liver fibrosis test).
(2) Patients who engage in harmful alcohol consumption should also undergo liver fibrosis testing and be referred to an alcohol cessation program.
(3) In patients with the greatest risk of AUD or ALD, a restrictive bariatric procedure should be offered over bypass surgery.
(4) After bariatric surgery, patients should receive prolonged, multiyear, postop monitoring through alcohol screening tools, biochemical liver tests, and if needed, alcohol metabolite testing.
(5) Patients should also receive long-term psychosocial screening and support during this monitoring period.

CONCLUSIONS

Overall, the preponderance of evidence shows that bariatric surgery is highly beneficial in preventing cardiometabolic and hepatobiliary adverse events in patients with morbid obesity.[20,30] However, there is growing support that gastric bypass and SG may increase the risk of AUD. Although data on the risk of ALD after WLS are less established, sufficient evidence exists to merit caution in evaluating candidates, selecting appropriate procedures, prolonging postop monitoring periods, or forgoing WLS altogether in favor of medical management in the highest risk patients (eg, glucagon-like peptide-1 analogs which are now commonly prescribed for therapeutic weight loss and may also reduce alcohol consumption in obese patients).[31]

FUNDING INFORMATION

Financial support and sponsorship: none

CONFLICTS OF INTEREST

Brett E. Fortune consults for WL Gore and Associates. The remaining authors have no conflicts to report.

ORCID

Rahul Grover https://orcid.org/0000–0002–3143–0228
Brett E. Fortune https://orcid.org/0000–0002–0646–467X
Clara Y. Tow https://orcid.org/0000–0002–5083–7541

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How to cite this article: Grover R, Fortune BE, Tow CY. The impact of alcohol on patients after bariatric surgery. Clin Liver Dis. 2024;23:e0139. https://doi.org/10.1097/CLD.0000000000000139