Spontaneous Elimination of Helicobacter pylori Infection After Bariatric Surgery: Effect of Sleeve Gastrectomy

Serhat Binici [1], *Fırat Aslan*[1], *Veysel Tahiroğlu*[2]

1 Van Yüzüncü Yıl University Department of General Surgery, Van, Türkiye
2 Şırnak University Faculty of Health Sciences Department of Nursing, Şırnak, Türkiye

Abstract

Aim: This study aimed to evaluate the spontaneous postoperative negativization rate of preoperative Helicobacter pylori (H. pylori) positivity in obese patients undergoing sleeve gastrectomy, without any eradication therapy.

Methods: Twenty-three patients who underwent primary laparoscopic sleeve gastrectomy between 2020 and 2024 were included. Preoperative H. pylori positivity was confirmed by histology, rapid urease test, urea breath test, or stool antigen test. H. pylori status was reassessed at 6 and 12 months postoperatively. Demographic, metabolic, and endoscopic data were recorded. Spontaneous elimination was defined as a negative test confirmed by two different diagnostic methods.

Results: At 12 months, 12 patients were H. pylori-negative, with a total spontaneous elimination rate of 52.2%. Transient negativization at 6 months was 45.0%. The mean gastritis score was 2.1 ± 0.6 in patients who became negative versus 3.0 ± 0.7 in those who remained positive. HOMA-IR decreased from 4.9 ± 2.3 to 2.6 ± 1.4. Significant reductions in total cholesterol, LDL, and triglycerides, and an increase in HDL were observed. All three patients who developed ulcers remained H. pylori-positive.

Conclusions: After sleeve gastrectomy, H. pylori may spontaneously disappear in a substantial proportion of patients without eradication therapy, associated with metabolic improvement. Postoperative monitoring and standard test confirmation are important for patient safety and effective antibiotic stewardship.

Keywords: *Sleeve gastrectomy; Helicobacter pylori; spontaneous elimination; obesity; metabolic improvement; bariatric surgery*

1. Introduction

Helicobacter pylori (H. pylori) is a globally prevalent bacterium that primarily colonizes the stomach and is associated with chronic gastritis, peptic ulcer disease, and gastric cancer.[1–3] Obesity, on the other hand, represents a major public health challenge and is strongly associated with increased cardiovascular and metabolic morbidity.[4] Bariatric surgery, particularly sleeve gastrectomy (SG), has become one of the most frequently performed surgical interventions for sustained weight loss and metabolic improvement in patients with obesity.[5,6] Beyond its established metabolic and hormonal effects, growing evidence suggests that SG may also influence gastric physiology and microbial colonization, including the persistence of H. Pylori.[7–9]

Several studies have reported a reduction in H. pylori prevalence following bariatric surgery. This phenomenon has been attributed to postoperative alterations in gastric acid secretion, accelerated gastric emptying, reduction of mucosal surface area, and changes in the composition of the gastric microbiota. In addition, hormonal changes after SG, including alterations in ghrelin, leptin, and glucagon-like peptide-1 (GLP-1), may indirectly contribute to changes in the gastric environment that affect H. pylori survival.[7,10,11] During SG, resection of the fundus and greater curvature transforms the stomach into a narrow tubular structure and significantly accelerates gastric transit, thereby reducing gastric stasis. As a result, both the ecological niche and the mucosal surface available for H. pylori colonization may be substantially reduced, potentially leading to a decrease in bacterial load.[8,12] Furthermore, removal of the fundus, a region frequently reported to harbor high H. pylori density, may exert a so-called “mechanical eradication” effect.[9,12] Nevertheless, an important methodological challenge remains: distinguishing true spontaneous elimination from a reduction of bacterial density below the detection threshold. The timing of sample collection, the diagnostic modality used (histology, rapid urease test, urea breath test, stool antigen test, or serology), and the use of proton pump inhibitors (PPIs) can all significantly influence test performance and interpretation.[13–15]

The prevalence and clinical implications of H. pylori infection in individuals with obesity remain controversial. While some studies report similar or even lower prevalence rates compared with the general population, others have suggested associations with insulin resistance, non-alcoholic fatty liver disease, and systemic

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inflammation.[16–18] Postoperative weight loss, reduction in low-grade inflammation, and improvements in insulin sensitivity after SG may enhance gastric mucosal integrity and alter intragastric pH and mucus barrier function, thereby creating a less favorable environment for H. pylori persistence.[10,11,19] Additionally, postoperative changes in dietary patterns, bile reflux, and duodenogastric flow may further influence bacterial viability and colonization dynamics.[20,21]

Current international guidelines generally recommend routine preoperative screening and eradication of H. pylori before bariatric surgery in order to reduce the risk of postoperative ulceration and staple-line–related complications.[22,23] However, increasing antibiotic resistance, potential adverse effects of eradication regimens, and cost-effectiveness considerations have prompted renewed discussion regarding a universal treatment strategy.[24,25] Notably, several cohort studies have demonstrated spontaneous reduction or clearance of H. pylori following SG in the absence of targeted eradication therapy, raising the question of whether systematic preoperative eradication is always necessary in all patients.[8,9,12,15] Given the heterogeneity of available data, the clinical relevance of spontaneous H. pylori negativization after SG remains incompletely defined.

Therefore, the aim of this study is to evaluate the rate of spontaneous postoperative H. pylori negativization following sleeve gastrectomy, to explore potential biological mechanisms underlying this phenomenon, and to assess its implications for postoperative management and rational antibiotic use in clinical practice.[14,22,24–26]

2. Materials and Methods

Study Design and Ethical Approval

This study was conducted as a single-center retrospective observational cohort study between January 1, 2020, and December 31, 2024. The primary objective was to determine the rate of spontaneous postoperative negativization of preoperatively confirmed Helicobacter pylori infection in patients who underwent sleeve gastrectomy and did not receive any eradication therapy. Secondary objectives included evaluation of diagnostic test concordance, identification of factors associated with spontaneous negativization, and assessment of postoperative complications.

The study protocol was approved by the Şırnak University Non-Interventional Clinical Research Ethics Committee (Decision No: E 94991599 050.99 128821, Date: 26.03.2025). Due to the retrospective and non-interventional design, the requirement for informed consent was waived. All procedures were performed in accordance with the Declaration of Helsinki revised in 2013 and national regulations. Patient data were anonymized prior to analysis.

Patient Selection

Patients aged between 18 and 65 years who underwent primary laparoscopic sleeve gastrectomy during the study period were screened. Only patients with confirmed preoperative Helicobacter pylori positivity were included, as the aim of the study was to evaluate postoperative spontaneous negativization rather than disease prevalence. Patients who were Helicobacter pylori negative before surgery were not included in the study design, which explains the observed one hundred percent preoperative positivity rate.

Inclusion criteria consisted of documented preoperative Helicobacter pylori positivity, availability of postoperative Helicobacter pylori assessment at six and or twelve months, and absence of eradication therapy before or after surgery within the first twelve months. Exclusion criteria included prior upper gastrointestinal surgery, bariatric procedures other than sleeve gastrectomy, incomplete medical records, inappropriate use of antibiotics or proton pump inhibitors before diagnostic testing, or inconclusive test results.

Surgical Procedure

All operations were performed laparoscopically by experienced bariatric surgeons following a standardized institutional protocol. A 36 to 40 French calibration bougie was used. Gastric resection was initiated four to six centimeters proximal to the pylorus, and the fundus and greater curvature were completely resected to create a tubular gastric remnant. Staple line reinforcement with sutures was routinely performed in all patients. Intraoperative leak testing was applied selectively. Postoperative proton pump inhibitor therapy, dietary progression, and micronutrient supplementation were administered according to institutional bariatric follow-up protocols.

Preoperative Assessment of Helicobacter pylori

Preoperative upper gastrointestinal endoscopy was routinely performed. Biopsy specimens were obtained from the antrum, corpus, and incisura angularis, and Helicobacter pylori infection was evaluated histologically. Histopathological examination was considered the reference diagnostic method when available.

Additional diagnostic methods, including rapid urease test, urea breath test, stool antigen test, and serology, were recorded when performed. These tests were used either prior to endoscopy or as complementary diagnostic tools, reflecting routine clinical practice rather than a predefined research protocol.

Postoperative Assessment and Definition of Spontaneous Negativization

Postoperative Helicobacter pylori status was assessed at six and or twelve months after sleeve gastrectomy. Routine postoperative endoscopic biopsy was not performed, and endoscopy was reserved for patients with clinical indications such as persistent symptoms or suspected complications. Therefore, postoperative evaluation was mainly based on non-invasive diagnostic methods, particularly the urea breath test and stool antigen testing.

Spontaneous negativization was defined as the absence of Helicobacter pylori confirmed by at least two different diagnostic methods, provided that proton pump inhibitors and antibiotics had been discontinued for an appropriate period before testing. Patients with transient negativization at six months followed by positivity at twelve months were classified as persistently positive. Cases with ambiguous or single test results were excluded from the final analysis.

Histopathological Evaluation and Gastritis Scoring

Gastric mucosal inflammation was graded according to the Updated Sydney System, which evaluates chronic inflammation, inflammatory activity, atrophy, intestinal metaplasia, and Helicobacter pylori density using a scale ranging from zero to three. A composite gastritis score was derived from inflammatory parameters. Higher scores indicated more severe gastritis and higher bacterial density.

Clinical and Metabolic Data Collection

Demographic characteristics, body mass index, smoking and alcohol consumption, comorbidities, and postoperative complications were recorded. Metabolic parameters, including fasting glucose, insulin levels, homeostasis model assessment of insulin resistance, glycated hemoglobin, and lipid profile, were collected preoperatively and at twelve months postoperatively.

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics for Windows version 29.0 and R software version 4.3.2. Continuous variables were expressed as mean plus or minus standard deviation or as median with interquartile range, and categorical variables as number and percentage. Normality of distribution was assessed

using the Shapiro-Wilk test.

Paired Student t test or Wilcoxon signed rank test was used for preoperative and postoperative comparisons, as appropriate. Categorical variables were analyzed using McNemar or chi-square tests. Multivariable logistic regression analysis was conducted to identify independent factors associated with spontaneous Helicobacter pylori negativization, and results were reported as odds ratios with ninety-five percent confidence intervals. A two-sided p-value below 0.05 was considered statistically significant.

3. Results

A total of 23 patients who underwent primary laparoscopic sleeve gastrectomy were included in the study. The mean age was 38.7 ± 9.6 years, and 73.9% (n = 17) of the patients were female. The mean preoperative body mass index (BMI) was 44.8 ± 6.2 kg/m². The most common comorbidities were type 2 diabetes mellitus (34.8%) and hypertension (26.1%). Obstructive sleep apnea was present in 17.4% of patients, while 30.4% were active smokers and 13.0% reported alcohol consumption. Baseline demographic and clinical characteristics are shown in Table 1.

The study population was intentionally limited to patients with confirmed preoperative *Helicobacter pylori* infection in order to evaluate postoperative spontaneous negativization. Accordingly, all 23 patients were positive for *H. pylori* preoperatively. Diagnosis was established by histopathological examination in 82.6% of cases (n = 19). Additional diagnostic methods included rapid urease testing in 60.8% (n = 14), urea breath testing in 43.4% (n = 10), and stool antigen testing in 26.0% (n = 6). In 65.2% of patients, positivity was confirmed using two or more diagnostic methods.

Postoperative *H. pylori* status was reassessed at 6 and/or 12 months using non-invasive tests, with confirmation by a second diagnostic method when appropriate, according to predefined criteria. At 6 months, 9 of 20 patients (45.0%) tested negative for *H. pylori*. At 12 months, sustained negativization was observed in 12 of 21 patients (57.1%). Based on confirmation with at least two diagnostic methods, the overall spontaneous elimination rate was calculated as 52.2%. Detailed postoperative *H. pylori* status and associated histopathological findings are presented in Table 2.

Patients who achieved spontaneous *H. pylori* negativization had significantly lower baseline gastritis severity compared with those who remained positive. The mean Sydney gastritis score was 2.1 ± 0.6 in patients who became negative and 3.0 ± 0.7 in patients with persistent infection (p = 0.012). Patients with high preoperative *H. pylori* density demonstrated significantly lower rates of spontaneous elimination (p = 0.031).

Agreement between diagnostic methods was evaluated. Concordance between histology and the urea breath test was good (Cohen's κ = 0.74), while agreement between histology and the rapid urease test was moderate to good (κ = 0.69). Agreement between stool antigen testing and other diagnostic modalities was lower (κ = 0.43).

Significant improvements in metabolic parameters were observed at 12 months after surgery. Mean fasting glucose decreased from 116 ± 21 mg/dL to 98 ± 14 mg/dL (p = 0.004), and insulin levels declined from 18.2 ± 7.4 µIU/mL to 11.1 ± 5.0 µIU/mL (p = 0.002). Consequently, HOMA-IR decreased significantly from 4.9 ± 2.3 to 2.6 ± 1.4 (p < 0.001). Total cholesterol, LDL cholesterol, and triglyceride levels decreased significantly, while HDL cholesterol increased (Table 3). Patients with spontaneous *H. pylori* negativization exhibited a significantly greater reduction in HOMA-IR compared with those who remained positive (p = 0.021); this subgroup analysis is not shown in Table 3.

Table 1

Baseline demographic and clinical characteristics of patients with confirmed preoperative Helicobacter pylori infection undergoing sleeve gastrectomy

Variable	Value
Number of cases (n)	23
Age (years, mean ± SD)	38.7 ± 9.6
Female (%)	73.9
BMI (kg/m², mean ± SD)	44.8 ± 6.2
Type 2 diabetes (%)	34.8
Hypertension (%)	26.1
Obstructive sleep apnea (%)	17.4
Smoking (%)	30.4
Alcohol consumption (%)	13.0

Body Mass Index (BMI); Diabetes Mellitus (DM, type 2 diabetes); Hypertension (HT); Obstructive Sleep Apnea Syndrome (OSAS); Homeostasis Model Assessment of Insulin Resistance (HOMA-IR).

Table 2

Postoperative Helicobacter pylori status and baseline histopathological features associated with spontaneous negativization

Parameter	Value
Preoperative *H. pylori* positivity (%)	100
Negative conversion at 6 months (%)	45.0
Sustained negativity at 12 months (%)	57.1
Overall spontaneous elimination (%)	52.2
Mean gastritis score (eliminated cases)	2.1 ± 0.6
Mean gastritis score (persistent cases)	3.0 ± 0.7
p-value	0.012

Sleeve Gastrectomy (SG); Proton Pump Inhibitor (PPI); Length of Stay (LOS); Heart Rate (HR); Systolic Blood Pressure (SBP); Diastolic Blood Pressure (DBP).

Table 3

Changes in metabolic parameters from baseline to 12 months after sleeve gastrectomy

Parameter	Preoperative (mean ± SD)	12 Months (mean ± SD)	p
Glucose (mg/dL)	116 ± 21	98 ± 14	0.004
Insulin (µIU/mL)	18.2 ± 7.4	11.1 ± 5.0	0.002
HOMA-IR	4.9 ± 2.3	2.6 ± 1.4	<0.001
Total cholesterol (mg/dL)	218 ± 38	184 ± 29	0.008
HDL (mg/dL)	43 ± 9	51 ± 10	0.011
LDL (mg/dL)	136 ± 34	108 ± 27	0.007
Triglycerides (mg/dL)	172 ± 58	129 ± 45	0.009

Odds Ratio (OR); Confidence Interval (CI); Body Mass Index (BMI); Proton Pump Inhibitor (PPI); Homeostasis Model Assessment of Insulin Resistance (HOMA-IR).

Postoperatively, gastric ulcers developed in 3 patients (13.0%), transient sleeve stenosis occurred in 2 patients (8.7%), and mild bleeding was observed in 1 patient (4.3%). All patients who developed ulcers remained *H. pylori*-positive during follow-up, and

this association was statistically significant (p = 0.037). No major surgical complications, including staple line leakage or mortality, were observed.

Multivariable logistic regression analysis identified independent factors associated with spontaneous *H. pylori* elimination. Lower baseline gastritis score (OR: 0.41, 95% CI: 0.19–0.89, p = 0.025), greater postoperative reduction in HOMA-IR (OR: 1.72, 95% CI: 1.11–2.69, p = 0.018), and shorter duration of postoperative proton pump inhibitor use (OR: 0.63, 95% CI: 0.41–0.97, p = 0.039) were independently associated with sustained negativization.

4. Discussion

In this retrospective study, approximately half of the patients who were *H. pylori*-positive preoperatively and underwent sleeve gastrectomy (SG) tested negative at 12 months without any eradication therapy. This observation indicates an association between SG and postoperative *H. pylori* negativization rather than a causal eradication effect**.** The global prevalence of *H. pylori* and its management framework are well established.[1,2] Considering the rising burden of obesity and the metabolic benefits of bariatric surgery[4,5], it is plausible that SG could influence *H. pylori* persistence by altering the gastric environment. Previous studies have reported a reduction in *H. pylori* prevalence following SG.[8,9] Our findings support the notion that removal of the upper stomach (fundus), accelerated gastric emptying in the tubular stomach, and changes in acid–bile–pH balance may collectively contribute to a gastric environment that is less favorable for *H. pylori* colonization.[10–12,20,21]

Keren et al. reported a significant reduction in *H. pylori* load after SG[9]; in our cohort, spontaneous negativization reached approximately 50% at 12 months without therapy, providing supportive but not definitive evidence for this observation. Misra et al. demonstrated that *H. pylori* distribution in the stomach is irregular and may concentrate in the fundus[12]; our data suggest that fundus resection could increase the likelihood of negativization, reflecting the potential clinical relevance of this anatomical feature. Additionally, postoperative physiological changes such as pH alteration, bile or reflux exposure, and accelerated gastric emptying are likely to contribute to a less favorable environment for bacterial colonization and proliferation.[10,20,21]

Diagnostic approaches typically outline the test–treatment–confirmation chain.[13,14] In our cohort, agreement between histology and urea breath test (UBT) was good (κ ≈ 0.74), while stool antigen test concordance was weaker (κ ≈ 0.43). These findings suggest that postoperative anatomical and physiological changes after SG may influence the diagnostic performance of certain non-invasive tests. Practically, UBT timed with proton pump inhibitor and antibiotic discontinuation should be considered the first-line test, with a second method (stool antigen testing or targeted biopsy) for confirmation if required.[13–15] Obesity and bariatric follow-up guidelines emphasize the importance of standardized monitoring and confirmation protocols.[6,22] Our results are consistent with previous evidence indicating that postoperative gastric changes may interfere with test accuracy.[10,20,21]

From a metabolic perspective, the relationship between *H. pylori* infection and insulin resistance or metabolic syndrome remains controversial[18], and associations with non-alcoholic fatty liver disease have also been reported.[17] Clinical observations demonstrating postoperative endocrine and metabolic recalibration after SG[11], together with reviews addressing metabolic–microbial interactions[19], provide a biological framework for the association observed in our study between HOMA-IR reduction and spontaneous negativization. However, the observational design precludes determination of whether metabolic improvement facilitates bacterial clearance or represents a parallel consequence of weight loss**.** Studies of microbial communities in the upper gastrointestinal tract show that environmental factors such as pH strongly influence microbial distribution[7] Similarly, in environmental biology, parameters such as pH, temperature, and nutrient availability shape organism distribution.[27] Post-bariatric surgery, a “new balance” in gut microbiota has been reported and may correlate with clinical and metabolic outcomes[28], with potential links to central and peripheral regulatory pathways.[29] The association observed in our cohort should therefore be interpreted within this broader context of post-surgical microbiota–host interaction rather than as a direct mechanistic relationship.[28,29]

Regarding complications, the potential role of *H. pylori* in post-bariatric ulcer development has been previously discussed.[23] In our cohort, all patients who developed ulcers remained *H. pylori*-positive, and this relationship was statistically significant, although the small number of events limits definitive conclusions. Nevertheless, this finding underscores the potential clinical relevance of persistent infection after SG. Accordingly, prompt planning of treatment and confirmation of eradication success in patients with persistent positivity and ulceration or symptoms appears reasonable.[2,14] Globally, macrolide and fluoroquinolone resistance rates are high.[24] Cost-effectiveness analyses support selective treatment strategies rather than routine eradication for all patients, combined with systematic confirmation of treatment success.[25] Practical recommendations for bariatric patients provide a clear framework for screening, treatment, and verification timing.[30] Within this framework, an institutional approach involving preoperative treatment in high-risk patients (e.g., high histological density, active gastritis, ulceration, or significant symptoms), followed by postoperative UBT at 6–12 months with confirmation using a second method in low-risk patients, may represent a balanced strategy for patient safety and rational antibiotic use.[2,12–15,22,30]

From an oncological perspective, the association between *H. pylori* infection and long-term mucosal changes has long been recognized.[3] Choi et al. reported that *H. pylori* eradication after subtotal gastrectomy could contribute to improved survival.[26] Although SG is not an oncologic procedure, persistent postoperative positivity warrants careful consideration and appropriate confirmation of eradication success, particularly in patients with additional risk factors.[2,14,26] Recent global assessments of antibiotic resistance and solution-oriented recommendations support the use of bismuth-based quadruple therapy, rifabutin-containing regimens, or concomitant/sequential approaches, together with meticulous post-eradication confirmation.[31,32]

Overall, our findings indicate that *H. pylori* may spontaneously negativize after SG; however, this phenomenon appears to be closely associated with baseline histological characteristics (bacterial density and inflammatory activity), appropriate timing of diagnostic testing, and the degree of postoperative metabolic improvement rather than guaranteed eradication**.** Future prospective studies with standardized test timing, confirmation using at least two diagnostic methods, and adequate sample sizes are required to clarify whether observed negativization represents true eradication or a reduction of bacterial load below the detection threshold. Moreover, detailed evaluation of intragastric pH changes, histological distribution patterns, and upper gastrointestinal microbial profiling using contemporary techniques may provide further insight into these mechanisms.[7,21,27–30,32]

This study has several limitations. First, the cross-sectional design limits causal inferences between demographic characteristics and knowledge levels regarding early menopause.

Second, the study was conducted in a single university hospital with a relatively small sample size, which may limit the generalizability of the findings. Third, the use of a researcher-developed questionnaire without formal psychometric validation may have affected measurement precision, although content relevance was ensured through expert review.

Additionally, data were based on self-reported responses, which may be subject to response and social desirability bias. Finally, the predominance of female participants may have influenced overall knowledge estimates and limits the ability to evaluate gender-based differences in awareness

Limitations

Several limitations should be considered when interpreting these results. First, the relatively small sample size and single-center design may limit the generalizability of the findings. Second, the study population was intentionally restricted to patients with confirmed preoperative *Helicobacter pylori* infection, which introduces selection bias and precludes comparison with *H. pylori*-negative patients. Third, although postoperative *H. pylori* status was assessed using standardized non-invasive tests and confirmation with multiple methods when possible, endoscopic histological confirmation was not routinely performed during follow-up. In addition, variability in postoperative proton pump inhibitor use may have influenced *H. pylori* detection and spontaneous negativization rates. Finally, the observational design of the study does not allow causal inference regarding the relationship between metabolic improvement and *H. pylori* elimination.

5. Conclusion

In this retrospective study, a substantial proportion of patients who were H. pylori-positive preoperatively and underwent SG tested negative at 12 months without eradication therapy. The likelihood of postoperative negativization was higher in patients with lower initial bacterial density, milder histological activity, appropriately timed diagnostic assessment, and greater postoperative metabolic improvement. These findings suggest that SG, through fundus resection, accelerated gastric emptying, and alterations in the gastric chemical environment, may hinder bacterial persistence rather than directly eradicate infection. Clinically, treatment and confirmation of eradication should be prioritized in patients with persistent positivity accompanied by ulceration or significant symptoms, whereas in asymptomatic, low-risk patients, appropriately timed UBT following drug discontinuation may serve as the primary follow-up modality. Future prospective studies with standardized diagnostic protocols, two-method confirmation, and sufficient sample sizes are needed to determine whether postoperative negativization reflects true elimination or a decrease below detectable levels.

Statement of ethics

This study was approved by the Şırnak University Non-Interventional Clinical Research Ethics Committee (Decision No: E-94991599-050.99-128821, Date: 26.03.2025). The study was conducted in accordance with the principles of the Declaration of Helsinki. Due to the retrospective and non-interventional nature of the study, the requirement for written informed consent was waived by the ethics committee.

genAI

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Conflict of interest statement

The authors declare that they have no conflict of interest.

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Author contributions

All three authors contributed equally to the conception and design of the study. Data collection, analysis, and interpretation were performed collaboratively by all authors. All authors participated in drafting and critically revising the manuscript and approved the final version.

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