Does *Helicobacter pylori* Infection Affect Postoperative Outcomes after Bariatric Surgery?

Kanokkarn Chupisanyarote, M.D.^{1,2}, Siriluk Thaiyanun, M.D.², Pakkavuth Chanswangphuvana, M.D.³

¹Division of Clinical Nutrition, Department of Internal Medicine, Thammasat University Hospital, Prathumtani 12120, Thailand. ²Department of Internal Medicine, Faculty of Medicine, Thammasat University, Prathumtani 12120, Thailand. ³Department of Surgery, Faculty of Medicine, Thammasat University, Prathumtani 12120, Thailand. Received 30 August 2024 • Revised 11 November 2024 • Accepted 18 November 2024 • Published online 18 April 2025

Abstract

Objective: The impact of *Helicobacter pylori* (HP) infection on postoperative outcomes remains controversial. This study aimed to investigate the relationship between HP infection and postoperative outcomes, including the percentage of postoperative excessive weight loss (%EWL) at 3, 6, and 12 months and post-surgical complications.

Material and Methods: This study involved a retrospective cohort study of 173 patients who underwent weight loss surgery at Thammasat University Hospital, Thailand, between 2015 and 2023. Participants met the indications for bariatric surgery and were preoperatively screened for HP infection.

Results: The majority of the participants were female (77.3%). The mean age and preoperative BMI were 35.3±8.1 years old and 44.2±9.4 kg/m² respectively. Seventy-five patients (43.3%) tested positive for HP infection. Baseline characteristics were similar between the HP-positive and non-HP groups. The %EWL of the HP-positive group at 3, 6 and 12 months was 42.72, 62 and 74, respectively. For the non-HP group, the %EWL at 3, 6 and 12 months was 36, 57 and 70.5, respectively. No statistically significant difference of %EWL was observed between the 2 groups. Postoperative adverse effects were reported with no statistically significant difference between the group 7 patients (7.14%) had complications. **Conclusion:** HP infection did not influence postoperative weight loss outcomes or complications after bariatric surgery.

Keywords: bariatric surgery, Helicobacter pylori, H. pylori, morbid obesity, Thailand

Contact: Kanokkarn Chupisanyarote, M.D. Division of Clinical Nutrition, Department of Internal Medicine, Thammasat University Hospital, Prathumtani 12120, Thailand. E-mail: kanokkarn.chu@gmail.com J Health Sci Med Res doi: 10.31584/jhsmr.20251189 www.jhsmr.org

© 2025 JHSMR. Hosted by Prince of Songkla University. All rights reserved. This is an open access article under the CC BY-NC-ND license

 $⁽http://www.jhsmr.org/index.php/jhsmr/about/editorialPolicies {\constraint} open {\cons$

Introduction

Obesity is a significant issue that leads to various health burdens. Obesity-related diseases, including diabetes, hypertension, dyslipidemia, obstructive sleep apnea, and cancer, can increase healthcare costs. The prevalence of obesity has been trending upward in recent years. Bariatric surgery is one of the most effective treatment options for morbid obesity, achieved by reducing the size of the stomach and decreasing the absorption of nutrients. Even though bariatric surgery is highly effective for weight loss, the outcomes and complications can vary due to individual differences, which are influenced by multiple factors.

Helicobacter pylori or *H. pylori* (HP) colonizes the human stomach and causes various gastric pathologies. While most of the infected individuals are asymptomatic, some may develop conditions such as gastritis, gastric ulcers, or gastric malignant lesions¹⁻². The prevalence of HP infection varies among countries, ranging from 9.1% to 88.6%. The overall global prevalence of HP infection is approximately 43.9%³. In Thailand, the infection rate ranges from 34.6% to 45.9%^{3,4}. The prevalence rate at Thammasat University Hospital is 43.8%⁵.

The relationship between HP infection and obesity is not fully understood. HP affects the ghrelin hormone by reducing the production and secretion of ghrelin, leading to hunger suppression⁶. Some studies have reported lower plasma ghrelin levels in HP-infected patients and plasma ghrelin levels increased after HP eradication⁷⁻¹⁰. Conversely, the results differ in obese patients. Morbidly obese patients had a higher density of ghrelin-producing cells in gastric mucosa¹¹. Furthermore, the prevalence of HP infection appears to be higher in obese patients compared to the general population^{12,13}. Currently, there is limited research on the relationship between HP infection and weight loss outcomes following bariatric surgery.

The association between HP infection and postoperative complications also remains unclear. There

have been many studies on this topic. The results indicate that H. pylori infection may either increase postoperative complications¹⁴⁻¹⁸, or may not increase them¹⁹⁻²⁴.

Therefore, this study aimed to investigate the relationship between HP infection and postoperative outcomes after bariatric surgery, including the percentage of excessive weight loss (%EWL) at 3, 6, and 12 months post-surgery, as well as postoperative complications.

Material and Methods Study protocol

The study was approved by the Human Research Ethics Committee of Thammasat University (Medicine). This retrospective study was conducted from 1st November 2015 to 31st December 2023 in the Thammasat University Hospital, Thailand. Patients aged 18–60 years who met the indications for bariatric surgery according to the Thai Society for Metabolic and Bariatric Surgery Consensus Guideline, body mass index (BMI) \geq 37.5 kg/m² or BMI \geq 32.5 kg/m² with obesity-related complications, were enrolled in the study. Patients with a history of previous bariatric surgery or missing data of HP testing before surgery were excluded.

Clinical data were obtained from the electronic medical records, comprising age, gender, weight, height, BMI, type of operation and obesity-related complications, including diabetes, hypertension, dyslipidemia, obstructive sleep apnea (OSA), and metabolic dysfunction-associated steatotic liver disease (MASLD). The procedures for bariatric surgery were laparoscopic sleeve gastrectomy (LSG), Rouxen-Y gastric bypass (RYGB), and laparoscopic sleeve plus bypass procedures (LSG+), including laparoscopic sleeve gastrectomy with proximal jejunal bypass (LSG – PJB) and laparoscopic sleeve gastrectomy with duodenojejunal bypass (LSG-DJB). Participants were assigned to each type of operation based on the clinical criteria and bariatric team consensus. All participants underwent esophagogastroduodenoscopy (EGD) before bariatric surgery and the findings were recorded. The EGD reports were classified as normal or abnormal, with the abnormalities including gastritis, gastric ulcer, and other conditions such as atypia, dysplasia, and metaplasia.

The results of HP infection were obtained through either the rapid urease test or histological examination of a random gastric biopsy taken from the greater curvature of the stomach. We prioritized the rapid urease test; however, if the result was negative, the biopsy was subsequently examined histologically. Participants who tested positive received eradication treatment with standard triple therapy for 2 weeks. The patients did not undergo a repeat rapid urease test to confirm HP eradication preoperatively. Nonetheless, the stomach tissue was sent for histological examination and HP assessment after bariatric surgery.

The primary objective was to investigate the relationship between HP infection and %EWL at 3, 6, and 12 months following bariatric surgery. The secondary objective was to examine the correlation between HP infection and post-surgical complications.

Sample size calculation

The sample size for a study investigating the primary outcome of %EWL between patients with and without HP infection was determined based on data from a pilot study conducted by Dr. Pakkavuth (unpublished data). In this pilot study, 8 out of 67 participants (12%) were identified as HP-positive. The mean %EWL was 80.8±27.8 for HPpositive patients and 69.5±14.1 for non-HP patients.

To compare %EWL between the 2 groups, a Student's t-test with Satterthwaite's correction for unequal variances was selected, given the observed differences in standard deviations. Based on this statistical method and the pilot study data, the estimated minimum sample size required to detect a statistically significant difference in %EWL between HP-positive and non-HP patients was determined to be 128.

Statistical analysis

For descriptive statistics, categorical variables were expressed as frequencies and percentages, or analyzed using Fisher's exact test where appropriate. Continuous variables were reported as means with standard deviations (S.D.) or medians with interquartile ranges (IQR), depending on the context. Comparisons between the groups were conducted using Pearson's chi-squared test for categorical variables and either the Student's t-test or the Wilcoxon rank-sum test for continuous variables, as appropriate. Statistical analyses were performed using SPSS version 22 (SPSS Inc., Chicago, IL, USA), and a p-value of less than 0.05 was considered statistically significant.

Results

A total of 214 patients underwent bariatric surgery. Of these, 41 patients were excluded due to prior bariatric surgery or missing data on HP testing. Thus, 173 patients were included in this study. The majority were female (78%, n=135) and 22% (n=38) were male. The mean age and preoperative BMI were 35.3±8.1 years old and 44.2±9.4 kg/m² respectively. Most of the patients underwent LSG (44.5%), followed by LSG+ (35.2%) and LRYGB (20.2%). Baseline characteristics, summarized in Table 1, were similar between HP-positive and non-HP groups.

Seventy-five patients (43.3%) tested positive for HP infection. Among them, 63 patients had a positive rapid urease test while 12 patients were found to have HP organisms in the histology of their gastric biopsy.

EGD findings, shown in Table 2, indicate that the HP-positive group had a lower percentage of normal EGD results (9.33%) compared to the non-HP group (13.27%).

Moreover, gastritis was more prevalent in the HP-positive group (84%) than in the non-HP group (73.47%), while gastric ulcer was higher in the non-HP group (13.27%) than the HP-positive group (6.67%). However, the difference did not reach statistical significance. There were no reports of atypia, dysplasia, or metaplasia in either group.

The follow-up rate at 3, 6, and 12 months was approximately 80.9%, 71%, and 54.3%, respectively. The details are presented in Table 3. The %EWL for the HPpositive group and the non-HP group at 3 months were 42.72 and 36, respectively (95% CI=-7.47, 4; p-value=0.551). At 6 months, the %EWL for the HP-positive group was 62, while the non-HP group was 57 (95% CI=-13, 4; p-value=0.298). At 12 months, the %EWL for the HP-positive group was 74, compared to 70.5 for the non-HP group (95% CI=-17, 7; p-value=0.363). No statistically significant difference of %EWL was observed between the 2 groups, as presented in Table 4. The %EWL for each type of operation, shown in Table 5, was also examined and there was no statistically significant difference among the groups. In the HP-positive group, pathology samples from 63 patients were sent for histological examination. Most of the patients, 58 patients (92%), achieved complete eradication of HP, while only 5 patients (8%) had persistent HP infection. The %EWL of both groups are displayed in Table 6. The %EWL at 3, 6, and 12 months of the complete eradication group was 42.33±15.87, 62.73±24 and 75.97±29.82, respectively. The %EWL at 3, 6 and 12 months of the persistent infection group was 56.69±17.19, 97.33±35.35 and 89.5±2.12, respectively.

The 30-day postoperative adverse effects, shown in Table 7, were reported with no statistically significant difference between the groups. In the HP-positive group, complications were observed in 5 patients (6.7%), including anastomosis stricture (2 patients), wound infection (2 patients), and persistent vomiting (1 patient). In the non-HP group, 7 patients (7.14%) experienced complications, including anastomosis stricture (3 patients), wound infection (4 patients), persistent vomiting (2 patients). Additionally, 2 patients had both anastomosis stricture and wound infection.

	HP-positive (n=75)	Non-HP (n=98)	p-value
Female, n (%)	58 (77.33)	77 (78.57)	0.845
Age, years, mean±S.D.	34.71±7.80	35.74±8.17	0.400*
BMI, kg/m², median (IQR)	43 (37–49)	42 (37–49)	0.798**
Comorbidity			
Hypertension, n (%)	37 (49.33)	43 (43.88)	0.476
T2DM, n (%)	22 (29.33)	25 (25.51)	0.575
Dyslipidemia, n (%)	38 (50.67)	47 (47.96)	0.724
OSA, n (%)	70 (93.33)	83 (84.69)	0.078
MASLD, n (%)	69 (92.00)	86 (87.76)	0.365
Type of operation			
LRYGB, n (%)	14 (18.67)	21 (21.43)	0.198
LSG, n (%)	32 (42.67)	29 (29.59)	
LSG+, n (%)	29 (38.67)	48 (48.98)	

 Table 1 Baseline characteristics

*Student's t-test, **Wilcoxon rank-sum test, HP=Helicobacter pylori, n=number, S.D.=standard deviation, IQR=interquartile range, BMI=body mass index, T2DM=diabetes mellitus type 2, OSA=obstructive sleep apnea, MASLD=metabolic dysfunction-associated steatotic liver disease, LRYGB=Roux-en-Y gastric bypass, LSG=laparoscopic sleeve gastrectomy, LSG+=laparoscopic sleeve gastrectomy plus bypass

Table 2 Esophagogastroduodenoscopy (EGD) findings

		·	
	HP-positive (n=75)	Non-HP (n=98)	p-value
Normal, n (%)	7 (9.33)	13 (13.27)	0.229*
Abnormal, n (%)			
Gastritis	63 (84.00)	72 (73.47)	
Gastric ulcer	5 (6.67)	13 (13.27)	
Others	0	0	
(dysplasia, atyptia, metaplasia)			

*Pearson's chi-squared test, HP=Helicobacter pylori

Table 3 Follow-up rate during the study period

	HP-positive	Non-HP	Total follow-up rate
Pre-operation	75	98	173
3 months	61 (81.3%)	79 (80.6%)	140 (80.9%)
6 months	50 (66.7%)	73 (74.4%)	123 (71%)
12 months	40 (53.3%)	54 (55%)	94 (54.3%)

HP=Helicobacter pylori

Table 4 Postoperative excessive weight loss between HP-positive and non-HP group

	HP-positive	Non-HP	Difference, % (95% CI)	p-value
%EWL at 3 months, median (IQR)	42.72 (30.91-52.21)	36 (29.67-52)	-7.47 ,4	0.551**
%EWL at 6 months, median (IQR)	62 (50-74)	57 (43–77)	-13 ,4	0.298**
%EWL at 12 months, median (IQR)	74 (57.5–98)	70.5 (50-87)	-17 ,7	0.363**

**Wilcoxon rank-sum test, HP=Helicobacter pylori

Table 5 Postoperative excessive weight loss by type of operation

	LSG		LSG+		LRYGB				
	HP	Non-HP	p-value	HP	Non-HP	p-value	HP	Non-HP	p-value
%EWL at 3 mo, median (IQR)	38.47 (25.84–52)	34.67 (29–52.30)	0.713	44.08 (57–73)	37.70 (29.78–56)	0.252	32.15 (28.44–47.5)	36.78 (27.43–49.73)	0.659*
%EWL at 6 mo, median (IQR)	62 (48–81)	61 (40-80)	0.897	63 (57–73)	56 (46–85.5)	0.419	59 (45–75)	48 (39–63)	0.297*
%EWL at 12 mo, mean±S.D.	73.81±36.46	73.43±29.51	0.970	79±5.10	78.2±22.94	0.919	88.14±46.75	61.89±21.04	0.153**

*Student's t-test, **Wilcoxon rank-sum test, HP=Helicobacter pylori

H. pylori Infection and Bariatric Surgery

	Complete HP eradication (n=58)	Persistent infection (n=5)	Difference, % (95% CI)	p-value
%EWL at 3 months, mean±S.D.	42.33±15.87	56.69±17.19	-31.06, 2.34	0.090*
%EWL at 6 months, mean±S.D.	62.73±24.00	97.33±35.35	-64.36, -4.84	0.024*
%EWL at 12 months, mean±S.D.	75.97±29.82	89.5±2.12	-56.98, 29.92	0.531*

Table 6 Postoperative excessive weight loss between complete HP eradication and persistent infection

*Student's t-test, HP=Helicobacter pylori

Table 7 Postoperative complications

Postoperative complications	HP-positive (n=75)	Non-HP (n=98)	p-value
Anastomosis stricture	3 (4.00)	3 (3.06)	1.000*
Persistent vomiting	1 (1.33)	2 (2.04)	1.000*
Wound infection	2 (2.67)	3 (3.06)	1.000*
Total	5	7**	

*Fisher's exact test, **2 patients had both anastomosis stricture and wound infection, HP=Helicobacter pylori

Discussion

In the present study, the prevalence of HP infection in obese patients undergoing bariatric surgery was 43.3%, which is considered high and close to the general population (43.8%) in Thammasat University Hospital⁵.

To the best of our knowledge, our trial is the first study to assess the impact of HP infection on %EWL and postoperative complications following bariatric surgery in Thailand. Our findings demonstrated that there were no statistically significant differences in %EWL between the HP-positive and non-HP groups at any follow-up time. Although the %EWL was higher in the HP-positive group compared to the non-HP group, the difference was not statistically significant.

Previous studies had similar results to ours, showing no association between HP infection and %EWL at 6 and 12 months²²⁻²⁵. Conversely, some studies reported contrasting results. Farazmand et al. showed that %EWL at 12 months after RYGB was higher in the HP-positive group; there was, however, no difference at other times (24 to 60 months)²⁶. Another study by Wang et al. showed negative results in weight loss outcomes on laparoscopic vertical banded gastroplasty (LVBG). HP infection had a negative impact on %BMI change at 12-36 months after laparoscopic vertical banded gastroplasty. Moreover, a higher HP density was associated with a lower %EWL at 24-48 months' follow-up²⁷. In comparison, our study had a shorter follow-up period, which might not have been long enough to observe that effect.

The mechanism by which HP infection affects obesity is not fully understood. Some potential pathways include hormonal changes and alterations in gut microbiota. HP reduces the production and secretion of ghrelin hormone, leading to hunger suppression⁶. Additionally, HP infection is associated with a reduction in gut microbiome diversity, which is observed in obese patients²⁸. The gut microbiota in the HP-positive group is characterized by a lower abundance of Actinobacteria, Bacteroidetes, and Firmicutes, alongside a higher abundance of Proteobacteria²⁹. Park et al. studied the effect of HP infection on LSG and gut microbiota in obese patients. They found that HP infection was associated with a reduction in the diversity of gut microbiota. Firmicutes and Actinobacteriota decreased while Campylobacterota increased in the HP-positive group. However, there was no significant effect on weight loss outcomes²⁵.

The eradication of HP influences changes in weight. Many studies have reported that HP eradication can lead to an increase in body weight and BMI³⁰⁻³², while others have not^{33,34}. Goday et al. studied the impact of HP eradication on weight changes following bariatric surgery. They discovered a more significant reduction in BMI at 3 months after LSG, but this difference was not statistically significant at other times or in the case of LRYGB³⁵.

While many studies demonstrated no association of HP infection and complications after bariatric surgery¹⁹⁻²⁴, a few reported opposing findings. HP infection could increase the risk of postoperative outcomes, including marginal ulcers, staple line leak, bleeding, or infection¹⁴⁻¹⁹. For that result, preoperative screening for HP infection may help minimize these risks.

Furthermore, HP is well-established as a major risk factor for gastric cancer³⁶. Eradicating HP could reduce the incidence of gastric malignancies, particularly in asymptomatic infected populations^{37,38}. A systematic review has indicated a growing trend in gastric cancer cases following bariatric bypass surgery over the past decade³⁹. Therefore, screening for and eradicating HP before bariatric surgery may be beneficial.

However, the current recommendations regarding screening for HP before undergoing bariatric surgery remain debatable. Some guidelines, such as those from the American Society for Metabolic & Bariatric Surgery (ASMBS), the Canadian guidelines, and Thai guidelines, suggest preoperative screening for HP in specific populations, such as those living in high-prevalence areas or undergoing gastric bypass procedures⁴⁰⁻⁴². Nevertheless, the European Association for Endoscopic Surgery (EAES) does not recommend it for routine HP eradication because there is insufficient evidence showing that HP infection affects postoperative complications⁴³.

The limitations of this study include its study design and short follow-up period. Our study utilized a retrospective design, which can report only associations rather than causations.

Conclusion

HP infection did not influence postoperative weight loss outcomes or complications after bariatric surgery. Further large-scale investigations are needed.

Acknowledgement

The authors would like to express their appreciation to the bariatric staff in Thammasat University Hospital for supporting this research, as well as the patients who participated in this study.

Conflict of interest

The authors declare no conflicts of interest.

References

- FitzGerald R, Smith SM. An Overview of Helicobacter pylori Infection. Methods Mol Biol 2021;2283:1–14.
- De Brito BB, Da Silva FAF, Soares AS, Pereira VA, Santos MLC, Sampaio MM, et al. Pathogenesis and clinical management of Helicobacter pylori gastric infection. World J Gastroenterol 2019;25:5578–89.
- Chen YC, Malfertheiner P, Yu HT, Kuo CL, Chang YY, Meng FT, et al. Global prevalence of Helicobacter pylori Infection and Incidence of gastric cancer between 1980 and 2022. Gastroenterology 2024;166:605–19.

- Uchida T, Miftahussurur M, Pittayanon R, Vilaichone RK, Wisedopas N, Ratanachu-Ek T, et al. Helicobacter pylori infection in Thailand: a nationwide study of the CagA Phenotype. PLoS One 2015;10.
- Aumpan N, Vilaichone RK, Nunanan P, Chonprasertsuk S, Siramolpiwat S, Bhanthumkomol P, et al. Predictors for development of complete and incomplete intestinal metaplasia (IM) associated with H. pylori infection: a large-scale study from low prevalence area of gastric cancer (IM-HP trial). PLoS One 2020;1;15.
- Carabotti M, D'Ercole C, Iossa A, Corazziari E, Silecchia G, Severi C. Helicobacter pylori infection in obesity and its clinical outcome after bariatric surgery. World J Gastroenterol 2014;21:647–53.
- Osawa H, Nakazato M, Date Y, Kita H, Ohnishi H, Ueno H, et al. Impaired production of gastric ghrelin in chronic gastritis associated with Helicobacter pylori. J Clin Endocrinol Metab 2005;90:10–6.
- Isomoto H, Nakazato M, Ueno H, Date Y, Nishi Y, Mukae H, et al. Low plasma ghrelin levels in patients with Helicobacter pylori-associated gastritis. Am J Med 2004;117:429–32.
- Nweneka CV, Prentice AM. Helicobacter pylori infection and circulating ghrelin levels – a systematic review. BMC Gastroenterol 2011;11:7.
- Nwokolo CU, Freshwater DA, O'Hare P, Randeva HS. Plasma ghrelin following cure of Helicobacter pylori. Gut 2003;52:637– 40.
- Maksud FA, Alves JS, Diniz MT, Barbosa AJ. Density of ghrelinproducing cells is higher in the gastric mucosa of morbidly obese patients. Eur J Endocrinol 2011;165:57–62.
- Erim T, Cruz-Correa MR, Szomstein S, Velis E, Rosenthal R. Prevalence of Helicobacter pylori seropositivity among patients undergoing bariatric surgery: a preliminary study. World J Surg 2008;32:2021–5.
- Arslan E, Atilgan H, Yavaşoğlu I. The prevalence of Helicobacter pylori in obese subjects. Eur J Intern Med 2009;20:695–7.
- Rasmussen JJ, Fuller W, Ali MR. Marginal ulceration after laparoscopic gastric bypass: an analysis of predisposing factors in 260 patients. Surg Endosc 2007;21:1090–4.
- Schulman AR, Abougergi MS, Thompson CC. H. Pylori as a predictor of marginal ulceration: a nationwide analysis. Obesity (Silver Spring) 2017;25:522–6.

- Mocanu V, Dang JT, Switzer N, Skubleny D, Shi X, de Gara C, et al. The effect of Helicobacter pylori on postoperative outcomes in patients undergoing bariatric surgery: a systematic review and meta-analysis. Obes Surg 2018;28:567-73.
- Marcolin P, Machado Berleze M, Polettini J, Marchesan Rodrigues MA, Augustin Silveira D. The impact of Helicobacter pylori on laparoscopic sleeve gastrectomy postoperative complications: a systematic review and meta-analysis. Obes Surg 2023;33:3649-57.
- Beran A, Shaear M, Al-Mudares S, Sharma I, Matar R, Al-Haddad M, et al. Predictors of marginal ulcer after gastric bypass: a systematic review and meta-analysis. J Gastrointest Surg 2023;27:1066–77.
- Papasavas PK, Gagné DJ, Donnelly PE, Salgado J, Urbandt JE, Burton KK, et al. Prevalence of Helicobacter pylori infection and value of preoperative testing and treatment in patients undergoing laparoscopic Roux-en-Y gastric bypass. Surg Obes Relat Dis 2008;4:383–8.
- Rawlins L, Rawlins MP, Brown CC, Schumacher DL. Effect of Helicobacter pylori on marginal ulcer and stomal stenosis after Roux-en-Y gastric bypass. Surg Obes Relat Dis 2013;9:760-4.
- Brownlee AR, Bromberg E, Roslin MS. Outcomes in patients with Helicobacter pylori undergoing laparoscopic sleeve gastrectomy. Obes Surg 2015;25:2276–9.
- Rossetti G, Moccia F, Marra T, Buonomo M, Pascotto B, Pezzullo A, et al. Does helicobacter pylori infection have influence on outcome of laparoscopic sleeve gastrectomy for morbid obesity? Int J Surg 2014;12(Suppl 1):S68-71. doi: 10.1016/j.ijsu.2014.05.051.
- Gonzalez-Heredia R, Tirado V, Patel N, Masrur M, Murphey M, Elli E. Is *Helicobacter Pylori* Associated with an Increased complication rate after sleeve gastrectomy? Bariatr Surg Pract Patient Care 2015;10:15–8.
- Shanti H, Almajali N, Al-Shamaileh T, Samarah W, Mismar A, Obeidat F. Helicobacter pylori does not affect postoperative outcomes after sleeve gastrectomy. Obes Surg 2017;27:1298– 1301.
- Park YS, Ahn K, Yun K, Jeong J, Baek KW, Park DJ, et al. Effect of Helicobacter pylori on sleeve gastrectomy and gastric microbiome differences in patients with obesity and diabetes. Int J Obes (Lond) 2024:48;1664–72.
- 26. Farazmand B, Shahsavan M, Eghbali F, Pazouki A,

Kermansaravi M. Comparison of weight loss after Roux-en-Y gastric bypass in Helicobacter pylori-negative and Helicobacter pylori eradicated patients during five years follow-ups. Surg Endosc 2024;38:888-93.

- Wang HH, Lee WJ, Liew PL, Yang CS, Liang RJ, Wang W, et al. The influence of Helicobacter pylori infection and corpus gastritis on the postoperative outcomes of laparoscopic vertical banded gastroplasty. Obes Surg 2006;16:297–307.
- Chanda D, De D. Meta-analysis reveals obesity associated gut microbial alteration patterns and reproducible contributors of functional shift. Gut Microbes 2024;16:2304900.
- Chen CC, Liou JM, Lee YC, Hong TC, El-Omar EM, Wu MS. The interplay between Helicobacter pylori and gastrointestinal microbiota. Gut Microbes 2021;13:1–22.
- Lane JA, Murray LJ, Harvey IM, Donovan JL, Nair P, Harvey RF. Randomised clinical trial: Helicobacter pylori eradication is associated with a significantly increased body mass index in a placebo-controlled study. Aliment Pharmacol Ther 2011;33:922-9.
- 31. Fujiwara Y, Higuchi K, Arafa UA, Uchida T, Tominaga K, Watanabe T, et al. Long-term effect of Helicobacter pylori eradication on quality of life, body mass index, and newly developed diseases in Japanese patients with peptic ulcer disease. Hepatogastroenterology 2002;49:1298–302.
- Kamada T, Hata J, Kusunoki H, Ito M, Tanaka S, Kawamura Y, et al. Eradication of Helicobacter pylori increases the incidence of hyperlipidaemia and obesity in peptic ulcer patients. Dig Liver Dis 2005;37:39–43.
- Alavinejad P, Hajiani E, Parsi A, Satari A, Rezaei MJ, Nayebi M, et al. Effect of Helicobacter pylori eradication on metabolic profile: an international, multicenter, case-control study. BMC Gastroenterol 2022;22:507.
- 34. Kim JW, Baeg MK, Bang CS, Park JK, Oh JH; the Diet, Obesity, and Metabolism Research Study Group of the Korean Society of Neurogastroenterology and Motility. Effect of Helicobacter pylori eradication on body weight: a multicenter propensity score-matched analysis in Korea. J Neurogastroenterol Motil 2023;29:352-9.
- Goday A, Castañer O, Benaiges D, Pou AB, Ramón JM, Iglesias MDM, et al. Can Helicobacter pylori eradication treatment

modify the metabolic response to bariatric surgery? Obes Surg 2018;28:2386-95.

- Watari J, Chen N, Amenta PS, Fukui H, Oshima T, Tomita T, et al. Helicobacter pylori associated chronic gastritis, clinical syndromes, precancerous lesions, and pathogenesis of gastric cancer development. World J Gastroenterol 2014;20:5461–73.
- Lee YC, Chiang TH, Chou CK, Tu YK, Liao WC, Wu MS, et al. Association between Helicobacter pylori eradication and gastric cancer incidence: a systematic review and meta-analysis. Gastroenterology 2016;150:1113–24.
- Ford AC, Yuan Y, Forman D, Hunt R, Moayyedi P. Helicobacter pylori eradication for the prevention of gastric neoplasia. Cochrane Database Syst Rev 2020;7:CD005583.
- Doukas SG, Doukas PG, Vageli DP, Broder A. Gastric cancer after bariatric bypass surgery. Do they relate? (a systematic review). Obes Surg 2023;33:1876–88.
- 40. Mechanick JI, Apovian C, Brethauer S, Garvey WT, Joffe AM, Kim J, et al. Clinical practice guidelines for the perioperative nutrition, metabolic, and nonsurgical support of patients undergoing bariatric procedures – 2019 update: cosponsored By American association of clinical endocrinologists/american college of endocrinology, the obesity society, American society for metabolic & bariatric surgery, obesity medicine association, and American society of anesthesiologists – executive summary. Endocr Pract 2019;25:1346–59.
- Glazer S, Biertho L. Canadian adult obesity clinical practice guidelines: bariatric surgery: selection & pre-operative workup [homepage on the Internet]. Edmonton: Obesity Canada; [cited 2024 Jul 3]. Available from: https://obesitycanada.ca/ guidelines/preop
- 42. Techagumpuch, A, Pantanakul S, Chansaenroj P, Boonyagard N, Wittayapairoch J, Poonthananiwatkul T, et al. Thai society for metabolic and bariatric surgery consensus guideline on bariatric surgery for the treatment of obese patient in Thailand. J Med Assoc Thai 2020;103:300–7.
- 43. Di Lorenzo N, Antoniou SA, Batterham RL, Busetto L, Godoroja D, Iossa A, et al. Clinical practice guidelines of the European Association for Endoscopic Surgery (EAES) on bariatric surgery: update 2020 endorsed by IFSO-EC, EASO and ESPCOP. Surg Endosc 2020;34:2332–58.