Is *Helicobacter pylori* eradication required after laparoscopic sleeve gastrectomy?

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Abstract

Introduction: Helicobacter pylori is the most common bacterial infection in humans. *H. pylori* is now known to be responsible for chronic gastritis, peptic ulcer, B-cell gastric lymphoma, and gastric adenocarcinoma. Laparoscopic sleeve gastrectomy (LSG) is increasingly preferred among surgical treatment methods in obese patients.

Aim: To discuss the detection and treatment of *H. pylori* in patients undergoing LSG surgery.

Material and methods: Patients who underwent biopsy with upper gastrointestinal endoscopy in the preoperative and postoperative period of LSG between 2014 and 2019 were included in the study, resulting in a sample of 162 patients who underwent preoperative and postoperative endoscopy. Endoscopic biopsies of these patients were collected in accordance with the preoperative Sydney protocol. The patients did not receive *H. pylori*-related eradication treatment. Endoscopy was performed to investigate dyspeptic complaints in the postoperative period. The biopsy results obtained in the endoscopy in the postoperative period were compared to those obtained in the preoperative period.

Results: Of the 162 patients in our study, 39 were male and 123 were female. All patients were assigned to one of two groups in the preoperative period: *H. pylori* (+) and *H. pylori* (−). *H. pylori* was found to be positive in 99 patients in the preoperative period. *H. pylori* was negative in 62 patients in the biopsy results of these patients after the LSG. *H. pylori* was found to be negative in 63 patients in the preoperative period, and 51 patients were *H. pylori*-negative in the biopsy results of these patients following LSG. These changes were found to be statistically significant when the preoperative and postoperative pathology results were evaluated (p < 0.01).

Conclusions: This study showed that LSG reduced the presence of *H. pylori*.

Key words: *Helicobacter pylori*, morbid obesity, laparoscopic sleeve gastrectomy

Introduction

Obesity is a multifaceted disease caused by a combination of genetic, environmental, and metabolic factors that has been a growing problem for public health [1]. Bariatric surgery was found to be highly effective in the treatment of obesity over time [2]. The European Association for Endoscopic Surgery (EAES) and the Society of American Gastrointestinal and Endoscopic Surgeons (SAGES) published guidelines in 2004 and 2008, respectively, following the introduction of laparoscopic bariatric surgery [3, 4]. Laparoscopic surgery became the gold standard for bariatric surgery as a result of these guidelines. Laparoscopic sleeve gastrectomy (LSG) which was first proposed as the first step of multi-step surgical
treatment in superobese patients, was considered the primary treatment method in morbidly obese patients after its efficacy was reported [5, 6].

*H. pylori* infection is one of the most common in humans: it has been estimated that more than half the global population will be infected with *H. pylori* in their lifetime [7]. This bacterium was classified in 1994 as a definitive human carcinogen by the International Agency for Research on Cancer Group of the World Health Organization. Given these considerations, *H. pylori* infection is a serious public health problem that warrants increased research and treatment efforts.

The frequency of *H. pylori* infection in morbidly obese patients is controversial. The prevalence of *H. pylori* in the preoperative period in bariatric surgery candidates was 8.7% in a German cohort study, whereas this rate was 85.5% in a Saudi Arabia cohort study [8, 9]. The reason for the high variability in *H. pylori* prevalence rates may be the diversity in *H. pylori* diagnostic tests and the low sample size used in these studies. Histological examinations among diagnostic tests have excellent sensitivity and specificity for *H. pylori* detection [10]. The sensitivity and specificity of histopathology have been found to be 66–100% and 94–100%, respectively [11].

Many epidemiological studies aiming to elucidate the relationship between *H. pylori* and body mass index (BMI) have failed to find any statistically significant relationship. BMI was found to be slightly higher in *H. pylori*-positive patients in a meta-analysis of 18 observational studies involving 10,000 cases [12].

**Aim**

Our study aims to investigate the effect of LSG – which is increasingly preferred in the treatment of morbid obesity – on *H. pylori* infection.

**Material and methods**

Patients who underwent biopsy with upper gastrointestinal endoscopy in the preoperative and postoperative period of LSG between 2014 and 2019 were included in the study. In our clinic, a total of 1368 LSG operations were performed during the study period, yielding a study sample of 162 (11.8%) patients who underwent preoperative and postoperative endoscopy.

Patients over the age of 18 years who underwent surgery were included in the study. These patients were evaluated by the endocrinology, psychiatry, chest diseases, cardiology, and anesthesia teams in the preoperative period. All abdominal ultrasonographies and upper gastrointestinal tract endoscopies were routinely performed in the preoperative period. According to the updated Sydney system recommendations, five biopsy samples should be obtained: two from the corpus, two from the antrum, and one from the incisura angularis [13]. The same number of biopsies were performed in the postoperative endoscopy. Biopsy samples were sent to the Department of Pathology for histological examination and evaluation of *H. pylori* density in accordance with the Sydney Classification. Their evaluation generated the following classifications: Absence of lesion: 0, mild: 1+, moderate: 2+, severe: 3+

Patients who had a gastric operation history and received *H. pylori* eradication treatment were excluded from the study. Additionally, patients using antibiotics for any indication in the preoperative and postoperative periods were excluded from the study. In the postoperative period, 40 mg of pantoprazole was prescribed for daily use over a one-month period to expedite wound healing.

LSG was performed by three obesity surgeons, who operated using a bougie ranging from 32F to 38F. The stomach was resected up to 2 cm away from the angle of His, using staplers with various thicknesses, starting from a distance of 2 cm from the pylorus. Anastomosis leak testing was performed with intraoperative methylene blue. Drainage extending from the left upper quadrant to the esophagogastric junction was placed in all patients. The Enhanced Recovery After Surgery (ERAS) protocol was applied in the postoperative period. All operations were completed laparoscopically.

Patients who underwent LSG in our clinic were followed regularly in the first month, third month, sixth month, and thereafter annually in the postoperative period. Upper gastrointestinal endoscopy is performed in cases of complaints during follow-up that include nausea, vomiting, epigastric burning, and difficulty swallowing. Biopsies are again taken during upper gastrointestinal system endoscopy, and these samples underwent histological examination and evaluation of *H. pylori* density.

**Statistical analysis**

The software NCSS (Number Cruncher Statistical System) 2007 (Kaysville, Utah, USA) was used.
for statistical analyses. Descriptive statistical methods (mean, standard deviation, median, frequency, ratio, minimum, maximum) were used to evaluate the study data, while Pearson’s χ² test was used to compare qualitative data. The independent samples t-test was used to compare two groups whose data were normally distributed. McNemar’s test was used to compare qualitative data and two-group parameters during follow-up. Statistical significance was set at $p < 0.01$ and $p < 0.05$.

**Results**

Of the 162 patients included in our study, 39 were male and 123 were female, and the mean age of the patients was 41.69 ±10.96 years. BMI was calculated as 47.64 ±6.69, and the time of perform upper gastrointestinal system endoscopy in the postoperative period was calculated as 17.69 ±9.17 months, on average.

Our study has got two experimental groups *H. pylori* (+) and *H. pylori* (–) in the preoperative period. No statistically significant differences were found between these two groups in gender distribution, diabetes and hypertension incidence rates, age, weight, and BMI ($p > 0.05$). Furthermore, no statistically significant differences were found in the time to perform postoperative upper gastrointestinal system endoscopy and time of the follow-up ($p > 0.05$) (Table I).

The presence of *H. pylori* was found to be positive in 99 patients in the preoperative period; of these patients, *H. pylori* was negative in 62 patients in the biopsy results following LSG. *H. pylori* was negative in 63 patients in the preoperative period; of these, 51 patients were found to be negative in the biopsy results after LSG. The changes were found to be statistically significant between the preoperative and postoperative pathology results evaluated ($p < 0.01$). No comparisons were made between other pathology results (Table II).

**Discussion**

LSG is increasingly preferred among the methods currently used in the treatment of obesity. Therefore, future research will benefit from access to a large pool of patients who have undergone extensive LSG operations. Treatment regimens to eradicate *H. pylori* will become even more complex in the future.

**Table I.** *H. pylori* demographic data of negative and positive groups in the preoperative period

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Total</th>
<th>H.P.(–)</th>
<th>H.P.(+)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>39 (24.1%)</td>
<td>11 (17.5%)</td>
<td>28 (28.3%)</td>
<td>*0.116</td>
</tr>
<tr>
<td>Female</td>
<td>123 (75.9%)</td>
<td>52 (82.5%)</td>
<td>71 (71.7%)</td>
<td></td>
</tr>
<tr>
<td>Age [years] (range)</td>
<td>41.69 ±10.96 (18–63)</td>
<td>41.7 ±11.33 (18–63)</td>
<td>41.69 ±10.78 (18–59)</td>
<td>*0.995</td>
</tr>
<tr>
<td>Weight [kg] (range)</td>
<td>126.28 ±18.94 (95–176)</td>
<td>124.25 ±17.02 (95–176)</td>
<td>127.58 ±20.04 (95–176)</td>
<td>*0.278</td>
</tr>
<tr>
<td>BMI [kg/m²] (range)</td>
<td>47.64 ±6.69 (39–70)</td>
<td>47.13 ±6.67 (39–67)</td>
<td>47.96 ±6.71 (40–70)</td>
<td>*0.442</td>
</tr>
<tr>
<td>Diabetes</td>
<td>63 (38.9%)</td>
<td>19 (30.2%)</td>
<td>44 (44.4%)</td>
<td>*0.069</td>
</tr>
<tr>
<td>Hypertension</td>
<td>45 (27.8%)</td>
<td>18 (28.6%)</td>
<td>27 (27.3%)</td>
<td>*0.857</td>
</tr>
<tr>
<td>Postoperative upper gastrointestinal endoscopy time [months] (range)</td>
<td>17.69 ±9.17 (3–52)</td>
<td>18.6 ±10.13 (6–52)</td>
<td>17.1 ±8.51 (3–46)</td>
<td>*0.311</td>
</tr>
<tr>
<td>Time of follow-up [months] (range)</td>
<td>27.33 ±11.77 (4–65)</td>
<td>28.9 ±10.69 (11–61)</td>
<td>26.33 ±12.35 (4–65)</td>
<td>*0.176</td>
</tr>
</tbody>
</table>

*aPearson’s χ², *aIndependent samples t-test.

**Table II.** Evaluation of postoperative pathology results according to preoperative pathology results

<table>
<thead>
<tr>
<th>Preoperative pathology</th>
<th>Postoperative pathology</th>
<th>P-value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>H.P.(–)</td>
<td>H.P.(+)</td>
<td>H.P.(–)</td>
</tr>
<tr>
<td>63 (38.9%)</td>
<td>99 (61.1%)</td>
<td>113 (69.8%)</td>
</tr>
</tbody>
</table>

*aMcNemar test, **p < 0.01.
effect of *H. pylori*’s relationship with gastritis and malignancy on patients undergoing bariatric surgery remains unclear [14].

In the extant literature, the relationship between LSG and *H. pylori* has been broadly examined in terms of postoperative complications. The relationship between LSG and *H. pylori* has not been associated with complications other than atrophic and chronic gastritis [15].

In the literature, there are very few studies investigating the *H. pylori* density in patients with LSG. A 2009 study was the first to report that LSG could contribute to *H. pylori* eradication [16]. The results of 480 patients who underwent LSG were examined in a retrospective study published in 2015, finding that *H. pylori* eradication would be spontaneous secondary to LSG and, therefore, that eradication treatment would not be required [17].

In our opinion, there may be a decrease in *H. pylori* density in patients who have undergone extensive gastric resection. It is known that *H. pylori* tends to colonize and is limited to the antrum due to tissue tropism [18]. Some publications report the proportion of *H. pylori*-positive patients in the surgical specimen to be 59.5%, while the positivity rate decreases to 28% in the average 3-year postoperative follow-up period in studies on *H. pylori* remission after partial gastrectomy [19]. It seems unlikely that a tropism based on antrum-specific and appropriate acidic conditions will remain unaffected after LSG. Presence of *H. pylori* colonization may also change dramatically, due to changes in the stomach environment after bariatric surgery [20].

Our study supports the observation that there is a decrease in *H. pylori* density after LSG. Our study has limitations. The study design is retrospective and observational. Additionally, our study is limited by its small number of cases and by the fact that it is a single-center study. On the other hand, when compared with other studies in the literature, the strength of our study is the pathological examination of *H. pylori*.

The effect of LSG on *H. pylori* infection can be better understood with multicenter, prospective studies with large case series.

Conclusions

The treatment of *H. pylori* after LSG is an uncertain issue. However, our study showed that LSG can contribute to the eradication of *H. pylori*.

Conflict of interest

The authors declare no conflict of interest.

References


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