ORIGINAL CONTRIBUTIONS



The Impact of *Helicobacter pylori* on the Complications of Laparoscopic Sleeve Gastrectomy

Sulaiman Almazeedi • Salman Al-Sabah • Dheidan Alshammari • Shamlan Alqinai • Ahmed Al-Mulla • Ahmed Al-Murad • Khaled Al-Enezi • Talib Jumaa

Published online: 24 October 2013 © Springer Science+Business Media New York 2013

Abstract Helicobacter pylori (H. pylori) is a very common bacterium present in the gastric tissue of up to 50 % of people, and the mucosal damage it causes can predispose to multiple comorbid conditions. This study aims to observe the prevalence of H. pylori infection in patients undergoing laparoscopic sleeve gastrectomy (LSG) and its correlation with postoperative complications. A retrospective study was done on the gastric pathology specimen results of 682 patients who underwent LSG at Amiri Hospital from 2008 to 2012. Symptomatic patients had preoperative upper gastrointestinal endoscopies (UGIEs) based on the decision of the treating surgeon, along with campylobacter-like organism test (CLO test) for H. pylori detection. The intraoperatively excised gastric specimen was sent for histopathological assessment of H. pylori, and the patients were followed up for complications. Of the 682 patients, 629 (92.2 %) were found to be *H. pylori* negative intraoperatively, while 53 (7.8 %) were positive. A total of 32 (4.7 %) patients were found to have postoperative complications, of which 2 (6.3 %) had H. pylori intraoperatively. No statistical significance (p=0.71), however, was seen between the overall complication rate and H. pylori. Specifically, there were five (0.7 %) cases of leak and eight (1.2 %) cases of neuropathy, both of which were not significantly associated with H. pylori (p = 0.33 and p = 0.12, respectively). All the other complications had no evidence of H. pylori. There appears to be no association between H. pylori infection and post-LSG complications.

S. Almazeedi · S. Al-Sabah (⊠) · S. Alqinai · A. Al-Mulla · A. Al-Murad · K. Al-Enezi · T. Jumaa Department of Surgery, Amiri Hospital, Kuwait Ministry of Health, Kuwait, Kuwait e-mail: salman.k.alsabah@gmail.com

D. Alshammari Department of Surgery, Farwaniya Hospital, Kuwait Ministry of Health, Kuwait, Kuwait **Keywords** Biopsy · Cytopathology · Histopathology · Laparoscopic surgery · Gastrectomy · Sleeve gastrectomy · Bariatric surgery

Introduction

Being obese or overweight is defined on the basis of body mass index (BMI) as recommended by the World Health Organization (WHO). WHO defines those with a BMI between 25.0 and 29.9 kg/m² as overweight and those with a BMI above 30.0 kg/m² as obese [1]. The prevalence of obesity worldwide is monitored by the Global Database of BMI, which revealed in 2005 that at least 400 million adults worldwide were obese and projected that in 2015 at least 700 million will be obese [1]. A study in Kuwait, the country we used as a model for this paper, revealed that 80.4 % of the population was overweight and 47.5 % was obese, with a higher prevalence in women [2]. Alongside the rise in obesity rates over the previous two decades, there has been an exponential increase in the demand for definite solutions to weight loss, i.e., laparoscopic bariatric surgery. For instance, at Amiri Hospital in Kuwait, one of the five major public hospitals in Kuwait, 10-15 laparoscopic sleeve gastrectomies (LSGs) have been done on a weekly basis since 2008. As with any surgery, the sleeve gastrectomy procedure has welldocumented potential complications, the most feared being gastric leak [3].

Helicobacter pylori (H. pylori), a common bacterium, currently infects approximately 50 % of the world population [4]. Even though studies relating H. pylori infection and obesity have been inconclusive, a few recent publications suggest that H. pylori might potentiate obesity through its effects on ghrelin and leptin hormone secretions by gastric mucosal cells [5, 6].

H. pylori is highly equipped in stomach colonization, multiplying in an area of the body that proves treacherous to other bacteria. After successful implantation, *H. pylori* usually becomes the most dominant organism numerically [7]. Surface epithelial degeneration is a probable result of direct tissue injury by bacterial products. Auto-destructive products, produced by neutrophil and monocyte activation such as reactive oxygen metabolites and proteases, result in local tissue atrophy. This is followed by pangastritis, multifocal atrophy, and intestinal metaplasia. The latter changes weaken the mucosa and its defenses, predisposing an individual to future peptic ulceration [8]. With the previous statement in mind, the proposed theory behind a number of leak cases post-LSG may be attributed to *H. pylori*-induced mucosal structural impairment.

H. pylori has also been observed to have effects on vitamin B_{12} absorption. Because of the superficial gastritis it causes and subsequent glandular atrophy, it has been labeled as the main culprit in patients with cobalamin deficiency [9, 10]. This fact also raises the question of whether post-LSG vitamin B_{12} deficiency and subsequent neuropathy might be related to *H. pylori* infection. The aim of this study, hence, was to observe the prevalence of *H. pylori* among obese patients undergoing LSG and, more importantly, observe its possible association with post-LSG complications.

Materials and Methods

All the patients undergoing LSG at Amiri Hospital from the time the first procedure was done in October 2008 to December 2012 had their intraoperative excised gastric tissue sent for histopathological assessment. In the pathology department, the tissue was then prepared by consecutive cuts at 5 µm, with subsequent staining with hematoxylin and eosin. Two cuts of the specimen were taken at random, and the presence of H. pylori was detected microscopically. Based on the presence or absence of preoperative symptoms of the patients, the decision on preoperative upper gastrointestinal endoscopy (UGIE) screening with campylobacter-like organism test (CLO test) was made at the discretion of the treating surgeon. In the event of CLO test positivity, a standard regimen of triple therapy comprising of amoxicillin and clarithromycin for 2 weeks and a proton pump inhibitor for 2 months was administered.

For the purpose of this study, the gastric histopathology reports of the patients who underwent LSG were collected from the department of pathology database. Their medical files were also retrieved and checked for preoperative UGIE and CLO test, postoperative recovery course, documented complications, outpatient clinic follow-up, and readmissions, if any. Statistical analysis of the data was carried out using SPSS software version 17.0.

Results

The data from a total of 682 patients was retrieved and analyzed. The mean age of patients undergoing the procedure was 33.5 ± 10.4 years, with the youngest patient being 12 years old and the eldest 65 years. The mean BMI of the study group was 46 ± 8.1 . In terms of gender differences, the number of females, 498 (73.0 %), exceeded that of males, 184 (27.0 %). Regarding the presence of *H. pylori* in the gastric specimen at the time of the operation, 629 (92.2 %) of patients were found to be negative, while 53 (7.8 %) were positive.

A total of 32 (4.7 %) patients was found to have postoperative complications (Table 1), with 2 (3.8 %) being among those in the H. pylori-positive group and 30 (4.8 %) among those in the H. pylori negative. Of the 32 patients with complications, only 2 (6.3 %) had concurrent H. pylori infections at the time of surgery, with no significant association (p=0.71). Similarly, from the 53 patients with H. pylori intraoperatively, 51 (96.2 %) had uneventful postoperative recoveries, with only 2 (3.8 %) complicated cases. Among all 682 patients, there were five (0.7 %) cases of leak, with one having H. pylori at the time of surgery. H. pylori infection was not significantly associated with increased chances of leak (p =(0.33). In addition, there were eight (1.2%) cases of neuropathy, with one case also found to have H. pylori. Likewise, H. pylori was not found to be significantly associated with neuropathy (p=0.12). There were five (0.7 %) cases of postoperative bleeding, including one involving the spleen, two involving the liver, one at the angle of His, and one of unknown source. All cases of bleeding had no H. Pvlori. In addition, there were five (0.7 %) cases of postoperative abdominal collection, four of which were left subphrenic and one was subhepatic. None of the cases of collection, however, had H. pylori. There were two (0.3 %) cases of hair loss, two (0.3 %) cases of respiratory failure, and one (0.1 %) case of each of the following: fatigue, dehydration, port-site hernia, and pulmonary embolism, all of which had no evidence of H. pylori infection.

Table 1 LSG complications and presence of H. pylori intraop

Complications	Number (%)	Cases with H. pylori
Leak	5 (0.8)	1
Neuropathy	8 (1.2)	1
Collection	5 (0.8)	0
Bleed	5 (0.8)	0
Respiratory failure	2 (0.3)	0
Hair loss	2 (0.3)	0
Fatigue	1 (0.2)	0
Dehydration	1 (0.2)	0
Pulmonary embolism	1 (0.2)	0
Port-site hernia	1 (0.2)	0

Out of the 682 patients in this study, 396 (58.0 %) patients had no preoperative UGIE, compared to 286 patients (42.0 %) who underwent the procedure. Of the 392 patients who had no UGIE, 362 (92.3 %) were negative for *H. pvlori* at the time of surgery, while 30 (7.7 %) were positive (Fig. 1). From those 286 patients who had UGIE, all had H. pylori CLO test results. Of the 286 patients, 146 (51.0 %) were H. pylori negative, while 140 (49.0 %) were positive at the time of the preoperative CLO test. By the time of surgery, however, 260 (90.0 %) were negative, while 26 (9 %) remained positive.

Of the 140 preoperative H. pylori-positive patients, 109 (77.9 %) received triple therapy regimen and 31 (22.1 %) did not. Of those who were treated, 102 (93.6 %) were negative at the time of surgery and 7 (6.4 %) remained positive. From those patients who were H. pvlori positive preop and did not receive triple therapy, 15 (48.4 %) were negative at the time of surgery and 16 (51.6 %) remained positive. Finally, there were 146 patients who were preop H. pylori negative, of which 143 (97.9 %) remained negative at the time of surgery while 3 (2.1 %) became positive.

Discussion

From the results of this study, it can be deduced that H. pylori infection has no association with the main complications of LSG. The vast majority (93.7 %) of patients with postoperative complications had no evidence of H. pylori in the gastric specimen, and the small fraction (6.3 %) of patients who were infected showed no statistically significant association. Even

Fig. 1 H. pylori infection based on preop UGIE CLO test and intraop histopathology

though the results of this study are limited by the small sample size of patients with complications, it is evident that the cases of leak and neuropathy were not significantly associated with H. pvlori presence. In addition, the fact that most (96.2 %) of the patients with H. pvlori at the time of surgery had uneventful postoperative periods further points to the fact that this association is unlikely.

Based on the literature review at the time this study was written, there seems to be only one other article published addressing the issue of H. pylori effect on post-LSG complications. Even though there were only 89 patients included in the recently published article by Albawardi et al., the authors also conclude that there seems to be no significant association between H. pylori and LSG complications [11].

The initiative was taken to compare H. pylori presence preand postoperatively, observe the prevalence of H. pylori in the population undergoing bariatric surgery, and study the impact of H. pylori screening and eradication carried out by some surgeons. When observing the trend of H. pylori infections before and after LSG, it was clear that its presence in symptomatic patients who underwent UGIE was high (49.7 %). Furthermore, since by the time of surgery H. pylori was only seen in 7.8 % of patients, this indicates that preoperative UGIE does help in eradicating the infection, especially in symptomatic patients. This is shown when it is revealed that most (93.6 %) of the H. pylori-positive patients who took triple therapy were rendered negative by the time of surgery. In contrast, more than half of the patients who were positive preoperatively and did not take triple therapy remained positive at the time LSG was performed.



In conclusion, even though H. *pylori* infection is very prevalent and predisposes to multiple morbid conditions, it does not seem to have an effect on post-LSG complications. The decision, however, of performing preoperative UGIE and CLO testing for symptomatic patients is effective and vital in ensuring H. *pylori* eradication by the time of surgery. Further studies with a larger sample size are warranted to provide a better analysis of this finding.

Acknowledgments The authors would like to convey their humble thanks to all those who made this study possible, most notably Dr. John Patrick, head of the Pathology Department at Amiri Hospital, Ms. Reena Thomas, and Dr. Mumtaz.

Conflict of interest All authors declare that there was no conflict of interest in this study.

References

- 1. Nguyen DM, El-Serag HB. The epidemiology of obesity. Gastroenterol Clin North Am. 2010;39(1):1–7.
- 2. Al Rashdan I, Al Nesef Y. Prevalence of overweight, obesity, and metabolic syndrome among adult Kuwaitis:

results from community-based national survey. Angiology. 2010;61(1):42-8.

- Frezza EE, Reddy S, Gee LL, et al. Complications after sleeve gastrectomy for morbid obesity. Obes Surg. 2009;19(6):684–7.
- Brown LM. *Helicobacter pylori*: epidemiology and routes of transmission. Epidemiol Rev. 2000;22(2):283–97.
- Weigt J, Malfertheiner P. Influence of *Helicobacter pylori* on gastric regulation of food intake. Curr Opin Clin Nutr Metab Care. 2009;12: 522–5.
- Tatsuguchi A, Miyake K, Gudis K, et al. Effect of *Helicobacter* pylori infection on ghrelin expression in human gastric mucosa. Am J Gastroenterol. 2004;99:2121–7.
- Alazmi WM, Buhaimed W, Al-Mekhaizeem K, et al. Efficacy of standard triple therapy in the treatment of *Helicobacter pylori* infection: experience from Kuwait. Dig Dis Sci. 2010;55(11):3120–3.
- Dixon MF. Pathophysiology of *Helicobacter pylori* infection. Scand J Gastroenterol Suppl. 1994;201:7–10.
- Serin E, Gumurdulu Y, Kayaselcuk F, et al. Impact of *Helicobacter pylori* on the development of vitamin B12 deficiency in the absence of gastric atrophy. Helicobacter. 2002;7(6):337–41.
- Kaptan K, Beyan C, Ural AU, et al. *Helicobacter pylori*—is it a novel causative agent in vitamin B12 deficiency? Arch Intern Med. 2000;160(9):1349–53.
- Albawardi A, Almarzooqi S, Torab F. *Helicobacter pylori* in sleeve gastrectomies: prevalence and rate of complications. Int J Clin Exp Med. 2013;6(2):140–3.