

Histopathological findings on resected gastric specimens from obese patients undergoing laparoscopic sleeve gastrectomy

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Abstract

Background and aims. This study aimed to investigate the relationship between baseline characteristics of obese patients and histopathological findings on resected gastric specimens resulted after laparoscopic sleeve gastrectomy.

Methods. Seventy-seven patients undergoing laparoscopic sleeve gastrectomy in a university surgical department from Romania were included in the present study. Demographic data, preoperative Body Mass Index values, and their relationship with the histopathological findings of resected gastric specimens were statistically analyzed.

Results. The mean age of patients included was 40.2 ± 11.05 years and the mean Body Mass Index was 43.5 ± 7.8 kg/m2; 71.4% of the patients were female. Active chronic gastritis was the most common gastric pathology (39%) encountered. *Helicobacter pylori* infection was present in 27.2% of the cases. Normal gastric histology was found in 33.7% of the specimens. A strong statistically significant association was noted between *Helicobacter pylori* infection and active chronic gastritis (p<0.0001). Similarly, a statistically significant association was observed between age, Body Mass Index, and intestinal metaplasia (p=0.005 and p=0.009 respectively). No malignancies were found.

Conclusions. Our study results show that the incidence of active chronic gastritis and *Helicobacter pylori* infection is relatively high in obese patients. Considering this, we conclude that it is important to send the resected gastric specimens for histopathological analysis after laparoscopic sleeve gastrectomy.

Keywords: obesity, laparoscopic sleeve gastrectomy, histopathological findings, active chronic gastritis, *Helicobacter pylori*

Background and aims

Obesity was first recognized as a growing public health problem in the 1970s and since then the prevalence has increased worldwide becoming a truly pandemic nowadays. The World Health Organization (WHO) defines obesity as excessive fat accumulation that might impair health and is diagnosed according to a body mass index (BMI) $\geq 30 \text{ kg/m}^2$ [1]. It represents a complex metabolic and endocrine disorder with increasing morbidity and mortality, and it is associated with multiple comorbidities such as type 2 diabetes mellitus, hypertension, obstructive sleep apnea, gastroesophageal reflux disease, and certain types of cancer [2].

Bariatric and metabolic surgery have become the most effective treatment option for obese patients who are struggling with difficulties in achieving sustainable weight loss by diet, physical exercises, and drug therapy alone [3].

Laparoscopic sleeve gastrectomy (LSG) is the most commonly performed bariatric surgery worldwide, as a standalone procedure, after it was initially described as the restrictive component of

DOI: 10.15386/mpr-2180

Manuscript received: 20.04.2021 Accepted: 17.02.2023

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This work is licensed under a Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International License https://creativecommons.org/licenses/ by-nc-nd/4.0/ biliopancreatic diversion with duodenal-switch. In LSG a large amount of the gastric corpus and fundus is resected along the greater curvature and offers the advantage of being easily performed laparoscopically, being associated with short postoperative hospitalization and quick recovery [4]. Being a restrictive procedure LSG reduces caloric intake and also decreases appetite through the removal of ghrelin-producing cells. Long-term results have demonstrated its effectiveness in weight loss and resolution of obesity-related comorbidities [5].

In the published literature, most studies regarding LSG outcomes explore the weight loss process, resolution of obesity-related comorbidities, and postoperative complications [6,7]. Despite being the only procedure that allows for pathological examination from the resected specimens, only several studies describe the histopathological findings encountered in gastric specimens resulted after LSG [8], leading to a scarcity of data about this topic. One reason may be represented by the lack of official guidelines about sending the gastric specimens for a full histopathological examination, and even until the present, the routine application of this practice is still questioned [9,10]. While some published studies found unexpected histopathological results requiring follow-up [11,12], others stated that a routine histopathological examination of LSG specimens was unnecessary because most gastric specimens had no significant histopathological findings [13,14].

In our surgical department, all gastric specimens resulted after LSG, are routinely sent for full histopathological examination.

Therefore, this study aimed to investigate the relationship between demographic data, preoperative BMI, and histopathological findings of LSG specimens, as well as to add more information to the published literature in this regard.

Methods

Patients

Obese patients who underwent LSG in a university surgical department from Târgu Mureş, Romania between January 2019 and November 2020 were included in the study. Inclusion criteria were: $age \ge 18$ years, a BMI ≥ 40 kg/m², or a BMI ≥ 35 kg/m² associated with at least one obesity-related comorbidity.

Preoperative data (age, gender, BMI values) and postoperative histopathological findings were retrospectively obtained from our department database.

The study was approved by the Research Ethics Committee of the "George Emil Palade" University of Medicine, Pharmacy, Science and Technology of Târgu Mureş. The study was conducted in accordance with the principles of the Declaration of Helsinki.

Surgical technique

All procedures were performed laparoscopically in

the same manner with the surgeon standing between the patient's legs (French position). Five trocars were used. The greater curvature of the stomach was dissected starting 2 to 4 cm from the pylorus and extending up to the gastroesophageal junction. A 36-Fr calibration bougie was inserted along the lesser curvature of the stomach. The resection began starting 4 cm from the pylorus and continued toward the angle of His by sequential application of staplers. Any bleeding at the staple line was controlled using metal clips. The bougie was removed and the staple line was checked for any leaks by introducing methylene blue solution through a nasogastric tube. A drainage tube was left in place for the next 48 hours. The resected stomach was extracted through a 12 mm left port site.

Histopathological assessment

As a standardized procedure following the surgery, all resected gastric materials were stored in formalin and sent to our pathology department for histopathological examination. Materials were fully examined macro- and microscopically, and were stained with hematoxylin and eosin, Alcian blue, and Giemsa. Following the histopathological examination, the cases were assessed in terms of histopathological findings, such as chronic and active chronic gastritis, intestinal metaplasia, atrophy, lymphoid aggregates, and *Helicobacter pylori* (*H. pylori*) presence. Pathological reports with the final histopathological diagnosis of all patients were sent back to our surgical department.

Statistical analysis

Kolmogorov-Smirnov test was used to evaluate the assumption of normality in the data. To describe the data, mean \pm standard deviation (SD), range, and frequency (percent) were used. One-way ANOVA/Kruskal-Wallis test and Unpaired *t*-test/Mann-Whitney *U* test were conducted to analyze quantitative variables, in accordance with data distribution. Chi-square test was conducted to analyze qualitative variables. The *p*-value less than 0.05 was considered statistically significant. All statistical analyses were performed by GraphPad Prism Software (version 9, San Diego, CA, USA).

Results

Of the seventy-seven obese patients included in the study, 71.4% were females. The overall mean age of the patients was 40.2 ± 11.05 years. The youngest patient was 19 and the oldest patient was 64 years old. The overall mean BMI was 43.5 ± 7.8 kg/m² (range, 35.1 - 72.1 kg/m²). Table I shows the patient's demographic information distributed by gender.

Histopathological findings of gastric specimens are given in table II. The normal gastric histology was found in 33.7% of the specimens. The most common abnormal histopathological finding was active chronic gastritis, evidenced in 39% of the cases. No malignancies were found.

Gender	N (%)	Age (years old)		BMI (kg/m ²)	
		Mean ± SD	Range	Mean ± SD	Range
Female	55 (71.4)	40 ± 11.7	20 - 64	42.3 ± 7.7	35.1 - 72.1
Male	22 (28.6)	40.9 ± 9.3	19 - 58	46.7 ± 7.1	37.5 - 65.7

BMI - body mass index; SD - standard deviation

Table II. Histopatholog	gical findings o	of resected gastric	specimens.
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Histop	N (%)	
	Normal histology	26 (33.7)
Diagnosis	Chronic gastritis	21 (27.2)
	Active chronic gastritis	30 (39.0)
Intestinal metaplasia	+	9 (11.6)
mestinai metapiasia	-	68 (88.4)
Atrophy	+	6 (7.7)
лиорну	-	71 (92.3)
Lymphoid aggregates	+	20 (25.9)
	-	57 (74.1)
H. pylori	+	21 (27.2)
п. руют	-	56 (72.8)

A significant relationship was found between age, BMI, and intestinal metaplasia (p=0.005 and p=0.009respectively). At the same time, a relationship was found between normal histology, chronic gastritis, active chronic gastritis, and age (p=0.016) and also between atrophy and BMI (p=0.022). The data of the mean age, mean BMI, and histopathological findings are given in table III.

Intestinal metaplasia was more common in older patients and less common in younger patients (49.7 versus 39 years), in contrast with BMI values where intestinal metaplasia was more common in patients with lower BMI mean, and less common in patients with a higher BMI mean (38.6 versus 44.2 kg/m²).

Table III. Relationship between age, BMI, and histopathological findings.

Histopathology		Age (years old)		BMI (kg/m ²)	
		Mean ± SD p		Mean ± SD	р
Normal histology		35.3 ± 9.2		41.7 ± 5.4	0.443 ^{d)}
Chronic gastritis		42.3 ± 12.9	0.016 ^{a)}	43.4 ± 8.1	
Active chronic gastritis		43.1 ± 9.8		45.2 ± 9.07	
Intestinal metanlagia	+	49.7 ± 8.1	0.005 ^{b)}	38.6 ± 5.3	0.009 ^{c)}
Intestinal metaplasia	-	39.01 ± 10.8	0.003*	44.2 ± 7.8	
A 4	+	46.3 ± 10.1	0.163 ^{b)}	39.7 ± 9.6	0.022 ^{c)}
Atrophy	-	39.7 ± 11.04	0.105%	43.8 ± 7.6	
Lymphoid aggregates	+	37.7 ± 10.6	0.228 ^{b)}	44.4 ± 8.6	0.744 ^{c)}
	-	41.1 ± 11.1	0.228	43.2 ± 7.5	0.744°
IIl	+	39.5 ± 10.1	0.651 ^{c)}	45.7 ± 8.4	0.106 ^{c)}
H. pylori	-	40.5 ± 11.4	0.031%	42.7 ± 7.4	0.100 /

BMI - body mass index; SD - standard deviation; a) one-way ANOVA; b) unpaired*t*-test; c) Mann-Whitney U test; d) Kruskal-Wallis test

There was no significant relationship between gender and normal histology (p=0.401), chronic gastritis (p=0.999), active chronic gastritis (p=0.416), intestinal metaplasia (p=0.653), atrophy (p=0.501), lymphoid aggregates (p=0.681) and *H. pylori* infection (p=0.571) (Table IV).

H. pylori infection was detected in 21 (27.2%) patients, whereas 56 (72.8%) patients were found to be not infected. In *H. pylori*-positive patients, normal gastric histology was found in 3.9%, chronic gastritis in 2.6%, active chronic gastritis in 20.7%, intestinal metaplasia in 3.9%, atrophy in 1.3% and lymphoid aggregates in 10.3% of the gastric specimens. A strong significant relationship was found between *H. pylori* infection and active chronic

gastritis (*p*<0.0001) (Table V).

 Table IV. Correlation between gender and histopathological findings.

Historiathalam	Female	Male		
Histopathology	%	%	р	
Normal histology	65.3	34.6	0.401	
Chronic gastritis	71.4	28.5	0.999	
Active chronic gastritis	76.6	23.3	0.416	
Intestinal metaplasia	77.7	22.2	0.653	
Atrophy	83.3	16.6	0.501	
Lymphoid aggregates	75	25	0.681	
H. pylori	66.6	33.3	0.571	

Table V. Correlation between H. pylori and histopathological findings

Histopathology		H. pylori positive	H. pylori negative	р
		N (%)	N (%)	
Normal histology	+	3 (3.9)	23 (29.8)	0.026
Normal histology	-	18 (23.3)	33 (42.8)	0.026
Chronic gastritis	+	2 (2.6)	19 (24.6)	0.032
Chilome gasutus	-	19 (24.6)	37 (48.05)	0.052
Active chronic	+	16 (20.7)	14 (18.1)	0.0001
gastritis	-	5 (6.4)	42 (54.5)	0.0001
Intestinal	+	3 (3.9)	6 (7.7)	0.664
metaplasia	-	18 (23.3)	50 (64.9)	0.004
Atuanlar	+	1 (1.3)	5 (6.4)	0.543
Atrophy	-	20 (25.9)	51 (66.2)	0.545
Lymphoid	+	8 (10.3)	12 (15.5)	0.137
aggregates	-	13 (16.8)	44 (57.1)	0.137

Discussion

The current study reports the histopathological findings of resected gastric specimens from 77 obese patients who underwent LSG in a single surgical department. Female sex was prevalent (71.4%), similar to previously published studies [9-14], and this was noted probably because of the female preference for staying in shape. Our mean preoperative BMI was higher for males (46.7 kg/m²) than females (42.3 kg/m²) and was 43.5 kg/m² for the whole sample.

Histopathological examination of the resected gastric specimens revealed normal histology in 33.7% of the cases, almost equal to those published by Raess et al. (35.2%) [12], but slightly higher than those published by Komaei et al. [14] and Demirbas et al. [15] where they found normal histology in 25.5% and 25.7% of the specimens, respectively. On the other hand, some published studies reported a higher rate of normal histology compared to our results (50.9% and 52%, respectively) [16,17].

As for the abnormal findings, in our study, the most encountered was active chronic gastritis in 39% of the cases, followed by chronic gastritis in 27.2%, lymphoid aggregates in 25%, intestinal metaplasia in 11.6%, and atrophy in 7.7% of the cases, respectively. Similar to our findings, Dogan et al. [18] found active chronic gastritis in 35.05% of the cases, but in terms of chronic gastritis and intestinal metaplasia, they found a lower rate of 19.9% and 2.7% respectively. In another study, Erkinuresin et al. [19] also revealed similar results. They found active chronic gastritis in 37.1% of the cases, but intestinal metaplasia was found only in 9.9% of the cases. On the contrary, in other published studies chronic gastritis was the most encountered abnormal finding [13-15].

An explanation for these discrepancies in pathological results found across the published studies may be represented by the different classifications of abnormal histopathological findings that were used in different countries. For instance, Oner et al. [20] used the same classification as ours, but they found chronic gastritis in 96.9% of the cases, as the most common abnormal finding, followed by lymphoid aggregates and active chronic gastritis in 87.2% and 29.2%, respectively. In another study by Adalı et al. [21], they also used the same classification as ours, but besides that, they had one more item, namely "lymphoid follicles", which was encountered in 59.5% of the cases. Authors like Dogan et al. [18], and Almazeedi et al. [22], used two more items to describe gastritis, in term of "follicular gastritis" and "active follicular gastritis" and contrary to these, Raess et al. [12] grouped all subtypes of gastritis as one category. Such inhomogeneous results lead to a challenge in making a precise comparison between different studies, and because of that, in the future, a more unified and standardized classification of abnormal findings of resected gastric specimens, across different countries, must be proposed.

Our study assessed age, BMI values, gender, and *H. pylori* infection as risk factors for abnormal histopathological findings of resected gastric specimens.

We found a significant correlation between age and histopathological findings, where normal histology was found in younger patients while active chronic gastritis was more common in older patients (35.3 vs. 43.1 years, p=0.016). We also found a significant correlation between age and intestinal metaplasia, where intestinal metaplasia was more common in older patients and less common in younger patients (49.7 vs. 39.01 years, p=0.005).

Regarding BMI, the values were not associated with any of the histopathological findings, except for intestinal metaplasia and atrophy, where both abnormal findings were more common in patients with lower BMI mean, and less common in patients with higher BMI mean (38.6 vs. 44.2 kg/m^2 , p=0.009 and $39.7 \text{ vs.} 43.8 \text{ kg/m}^2$, p=0.022, respectively). However, patients with chronic gastritis, active chronic gastritis, lymphoid aggregates, and *H. pylori* infection had generally higher BMI values compared with patients without these conditions or with normal gastric histology, but the differences were not statistically significant.

In terms of age, Safaan et al. [9] noted that older age was significantly associated with intestinal metaplasia, as we also showed in our study, and these results are in agreement with the literature, that intestinal metaplasia is more prevalent among the older age groups [23]. In terms of BMI values, Adalı et al. [21], published for the first time in literature the existence of a relationship between intestinal metaplasia and BMI values and found a statistically significant correlation between these two (p<0.0001). Like our results, they found that intestinal metaplasia was more common in cases with lower BMI mean.

In our study, we did not find a statistically significant correlation between histopathological findings and gender. Dogan et al. [18], and Öner et al. [20], did not find a significant relationship between gender and histopathological findings either, while Safaan et al. [9] found a significant correlation between active chronic gastritis and female gender.

According to the results of the current study, the rate of *H. pylori* infection was found in 27.2% of the specimens. It is important to know the rate of *H. pylori* infection because it represents a risk factor for gastritis and duodenitis, peptic ulcer, and other benign and also malignant diseases [24]. Our H. pylori infection rate had a strong association with active chronic gastritis (p<0.0001). However, we did not find an association between H. pylori infection and intestinal metaplasia (p=0.664), respectively H. pylori infection and atrophy (p=0.543), conditions considered as premalignant. Also, we did not find a correlation between age, BMI values, gender, and H. pylori infection. On the other hand, in H. pvlori-negative patients, 24.6% of them had chronic gastritis, therefore we could hypothesize that obesity itself could be a risk factor for chronic gastritis. In a study regarding this topic, Yamamoto et al. [25] also presumed that obesity might account for the appearance of chronic gastritis in obese patients and a new category of gastritis has been proposed known as "obesity-related gastritis".

Once again, in published studies, the rate of H. pvlori infection varies from one country to another due to dietary habits, cultural factors, and differences in social and economic status [26]. For instance, in previously published studies, Demirbas et al. [15] found the same rate of H. pylori infection as ours (27%), while Dogan et al. [18], Erkinuresin et al. [19], and Öner et al. [20] reported that they found a higher rate of *H. pylori* infection (43,2%, 41,7%, and 64%, respectively). Contrary to these results, a lower prevalence of H. pylori infection was reported by Di Palma et al. [16], and Canil et al. [27] where they found the rate to be 3.2% and 2.4%, respectively. Like in our study, Safaan et al. [9] also found an association between H. pylori infection and active chronic gastritis, but contrary to our results they also found an association with intestinal metaplasia and lymphoid aggregates. Rossetti et al. [28], similar to our results, found no correlation between H. pylori infection and age, BMI values, and gender.

In our study, no malignancies were found. This is comparable with previously published studies that could also not detect any malignancies in the resected gastric specimens [10,11,15].

This article has some limitations. It was a singledepartment research study and this could be considered a limitation. The second limitation is represented by the relatively small number of patients enrolled. Although, there are published studies in the literature which indicate that the number of included patients may be sufficient to draw conclusions [21,29,30]. In our surgery practice, we do not perform routinely preoperative upper gastrointestinal endoscopy (UGIE) and this could be considered another limitation.

There are still debates in literature whether routine preoperative UGIE should be performed in all LSG patients. Detection of abnormal findings in UGIE before surgery varies between 4.6% and 89.7% [31]. In a study, Ohanessian et al. showed that the results in patients who performed UGIE prior to LSG did not change the operative management. Besides that, the authors stated that in asymptomatic patients the recommendation for preoperative UGIE is little and they recommended the procedure only in patients with clinical indications such as severe reflux or dysphagia. [32] On the other hand, Yardimci et al., recommend preoperative UGIE to all bariatric patients to identify benign (hiatal hernia, ulcers), or premalignant/malignant lesions which could lead to a change in the operative strategy, and also all surgical specimens should undergo histopathological examination following LSG [33].

Conclusions

In conclusion, our study evidences the range of histopathological findings of resected gastric specimens in apparently healthy patients (except obesity as *per se*) undergoing LSG and according to this, routine histopathological analysis of the specimens should be part of "the bariatric and metabolic algorithm" in every obese patient. In the same vein, we suggest that in the absence of a preoperative UGIE plus multiple biopsies, histopathological analysis of the specimens should be mandatory.

Although no malignancy has been encountered, due to metaplastic presence in resected gastric specimens, some patients have to be under close follow-up.

The different classifications of abnormal histopathological findings from resected gastric specimens across literature indicate the need for a more uniform terminology to allow more accurate comparisons between different studies.

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