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The role of histological evaluation of *Helicobacter pylori* infection in obese patients referred to laparoscopic sleeve gastrectomy

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Abstract

Helicobacter pylori (*H. pylori*) is the etiological factor for gastritis in more than half of the worldwide population. *H. pylori* infection increases the risk for gastric pathology, but could also have consequences on cardio-metabolic status. Obesity has as epidemic growth, and the only efficient long-term treatment for morbidly obese patients is currently surgery. Although of vital importance, the preoperative assessment is not standardized, including the aspects related to *H. pylori* infection. The aim of this prospective study was to evaluate the prevalence of *H. pylori* (Hp) infection in a group of patients referred to bariatric surgery and the agreement of two commonly used methods for its diagnosis. We included 70 asymptomatic obese patients consecutively for 14 months, who were evaluated by serology (anti-Hp IgG antibodies) and by histology (gastroscopy with gastric mucosa biopsy). If diagnosed, *H. pylori* infection was standard treated and afterwards, all patients underwent laparoscopic sleeve gastrectomy; the resected stomach was morphologically evaluated. 58.6% of patients were *H. pylori* positive on serology and 51.4% were *H. pylori* positive on histology, agreement coefficient factor *kappa* between the two methods being 0.686, $p<0.001$. The serological diagnosis had a sensibility of 90.3% and a specificity of 77.8%. The prevalence of *H. pylori* infection in the resected stomach was 11.4%, and was associated with more severe degrees of chronic gastritis. In conclusion, as gastroscopy should anyhow be performed in all patients referred to surgery, our data favor the histological evaluation in all patients and the eradication treatment according to its results.

Keywords: *Helicobacter pylori*, chronic gastritis, laparoscopic sleeve gastrectomy, anti-Hp IgG antibodies.

Introduction

Helicobacter pylori (*H. pylori*) infection is worldwide considered the most frequent cause of chronic gastritis (chronic inflammation of gastric mucosa) [1]. *H. pylori* infection is currently perceived as having a causal role in development of gastric and duodenal ulcer [2], gastric adenocarcinoma (being a group I human carcinogen) [3] and mucosa-associated lymphoid tissue (MALT) lymphoma [4, 5]. *H. pylori* infection is in fact considered the most important risk factor for the development of peptic ulcer and its complications, including gastric cancer [6]. Bacterial eradication reduces the risk of gastric cancer [7], dependent however on the presence, severity and extension of atrophic lesions at the time of eradication [8].

H. pylori infection could also have a pathogenic role in the development of numerous other diseases; studies demonstrate the association between *H. pylori* infection and increased risk of metabolic syndrome [9, 10], atherosclerosis [11, 12], cardiovascular disease [13], hepatic disease [14], iron-deficient anemia and idiopathic thrombocytopenic purpura [9], and altered neurological and cognitive status [15–17]. The mechanisms involved are complex and imply metabolic disturbances and endothelial dysfunction, pro-inflammatory and pro-atherogenic status [18].

Obesity is an important risk factor both for digestive pathology and for cardio-metabolic disease, cancer, respiratory, rheumatologic and dermatological pathologies. Superior digestive tract diseases (gastro-esophageal reflux disease, erosive esophagitis, hiatal hernia, esophageal adenocarcinoma and *H. pylori* infection) have been reported as being 2–3 times more frequent in obese persons compared to normal weight [19]. Currently, metabolic surgery (which involves handling and alteration of the digestive tract), is considered the only efficient method for weight loss and maintenance of new weight for morbidly obese patients and it is included in the treatment guidelines for obesity and other metabolic diseases [20]. In this context, the majority of digestive diseases can be clinically relevant and can have a significant impact on postoperative evolution of the patients. Hence, *H. pylori* infection plays a special role in obesity, it can complicate and aggravate the complications and comorbidities associated with obesity *per se,* by supplementary influencing weight status, and, last, but not least, can be a negative factor in limiting access to bariatric surgery [21].

However, the role of routine endoscopy before bariatric surgery remains controversial. The American guidelines do not give clear indications regarding screening and management of *H. pylori* (screening is recommended to symptomatic patients from high prevalence areas and

endoscopy is recommended in selected cases) [22], while the European guidelines recommend endoscopy prior to surgery in all patients, symptomatic or not, and treatment of any lesion that might lead to postoperative complications, including *H. pylori* [23]. The lack of correlation between the endoscopic aspect and patients' symptoms was documented by various authors, who even suggested that routine preoperative endoscopy would still be useful in detecting the lesion and inflammation [24]. Other authors consider that high costs, the fact that the procedure is invasive and the risks associated with sedation are limits that do not justify the routine performance of this procedure in asymptomatic patients [25].

The influence of *H. pylori* infection on the postoperative follow-up of these patients could mean early postoperative complications, like leakage, collections or intra-abdominal abscesses, ulcer with or without perforation, longer length of stay in hospital or higher rate of early postoperative readmission [26, 27], although reported data are contradictory in this field [28–30]. Hence, it is very important to establish an evaluation algorithm to detect *H. pylori* infection pre-operatory in these patients.

Literature data is controversial for several reasons – different characteristics of included subjects (general population, or patients with dyspeptic symptoms – most frequently, or patients already diagnosed with cardiovascular or metabolic pathology). Another reason for the discrepancies in results is the different adjustment of results, for various parameters; however, probably the most important reason for the contradictory results is the different means of diagnosis, influencing both the prevalence of *H. pylori* infection, and its association with cardio-metabolic risk.

The aim of our study was to evaluate the concordance between two methods of detection of *H. pylori* in gastric mucosa among patients with obesity proposed for metabolic surgery and to suggest an algorithm for assessing these patients preoperative.

Patients, Materials and Methods

In this prospective study, we included consecutive patients, who were evaluated in order to undergo laparoscopic sleeve gastrectomy between September 2014 and November 2015, at the Centre for Obesity of the "St. Spiridon" Clinical Emergency Hospital, Iassy, Romania. All the included patients fulfilled the current guideline criteria for the indication for bariatric surgery [31] and were followed using the same algorithm for the complex preoperative multidisciplinary evaluation. Anthropometric parameters were assessed according to the recommendations of the *World Health Organization* (WHO) [32] and allowed the classification of subjects in two weight categories: stage II obesity [body mass index (BMI) of 35–39.9 kg/m^2) and stage III obesity (BMI ≥40 kg/m^2). Excess weight was calculated with the difference between real weight of patients and ideal weight, using the Devine formula [33].

All patients included in the study underwent an upper gastrointestinal (UGI) endoscopy, performed by the same experienced gastroenterologist, who took gastric mucosa biopsies, which were immediately sent to Department of Pathology for morphological evaluation. The macroscopic aspects observed during the UGI endoscopy were divided into four categories: normal, congestion, gastritis, and other lesions (granular aspect, hypertrophic folds, and biliary reflux). The histological examination of the gastric biopsy provided two types of information regarding gastritis: classification and grading the inflammation (which resulted into three categories: normal, superficial chronic gastritis and profound chronic gastritis), and assessment of *H. pylori* infection [34]. All patients with histological diagnosis of *H. pylori* infection received the same eradication treatment regime – the triple therapy and surgery was performed only after completion of the treatment.

The serological diagnosis of *H. pylori* infection was based on the detection of anti-*H. pylori* (Hp) IgG antibodies, for which a 5 mL blood sample was taken from each patient and immediately transported to the immunology laboratory, where the serum was separated and tested right away. IgG antibodies to *H. pylori* were detected in fresh serum using a solid phase chemiluminescent immunometric assay, commercially available IMMULITE® 2000 *H. pylori* IgG EIA (enzyme immunoassay) reagents (Siemens) and an IMMULITE® 2000 immunoassay system (Siemens Healthcare). The presence of IgG antibodies to *H. pylori* is an indication of previous exposure to the organism. Titers higher than or equal to 1.1 U/mL were considered to be "positive" and indicate that *H. pylori* IgG antibodies were detected in the sample. Titers lower than 0.9 U/mL were considered to be "negative" and indicate that *H. pylori* IgG antibodies were not detected in the sample. Negative results do not preclude recent primary infection. Titers higher than or equal to 0.9 U/mL and lower than 1.1 U/mL were considered to be "indeterminate" and were subject to retesting.

All patients underwent laparoscopic sleeve gastrectomy including the greater curvature of fundus and stomach body and the resected gastric sample was subsequently morphologically analyzed. All fresh surgical specimens were measured and the gross appearance was described. Fixation was done for 18–24 hours in 10% neutral buffered formalin, pH 6. From each gastric sample, three fragments were selected from the proximal, middle and distal sites. They were routinely processed for paraffin embedding using a Leica ASP200 tissue processor. To obtain thin 4–6 μm sections, we used a Leica RM2135 manual rotary microtome; sections were stained with Hematoxylin–Eosin (HE) and Giemsa. Microscopic examination and image acquisition were done using a Nikon Eclipse E600 light microscope with Nikon Coolpix 4500 camera and LuciaNet software. For the classification and grading of gastritis, we used the updated Sydney System [35]. Hence, the observed modifications were classified into three categories: normal, superficial chronic gastritis and profound chronic gastritis.

Data was analyzed using Microsoft Office Excel and SPSS *ver.* 17.0. Numerical data were expressed as means and standard deviation (SD), minimum and maximum and significant differences between numerical data were found using Student's *t*-test. For the description of categorical variables, we used frequencies and percents and the significant differences were assessed with the *chi*-square (χ^2) test (or Fisher's test for small samples) with a significance value of <0.05. We also used cross tabulation

to determine specificity and sensibility of a diagnosis test, Cohen' kappa coefficient of agreement and calculated odds ratio for adequate variables.

The Ethics Committee of the University granted approval for the study and all the patients gave their consent to participate.

Results

The study cohort included 70 patients, among which 19 (27.1%) were men. All descriptive data for the study group are included in Table 1.

Considering the macroscopic aspects observed in gastroscopy, in 30% (21 patients), it was normal, 50.8% (37 patients) had congestion, 7.1% (five patients) had gastritis and 10% presented other lesions. All macroscopic, serological and histological description of the study group (whole group and separately by gender) is presented in Table 2.

Analyzing the serological titers of anti-*H. pylori* antibodies, we observed that there were no patients with an "indeterminate" titer, meaning that the patients were divided in just two categories: *H. pylori* positive and *H. pylori* negative in serology. As such, 58.6% of patients were *H. pylori* positive by serology. In the histological examination of the biopsy, we found that 51.4% of patients were *H. pylori* positive. Among those patients who were *H. pylori* positive in serology, 82.4% were also positive in the histological exam. Among those who were *H. pylori* negative in serology, 12.5% were *H. pylori* positive in the histological examination. The Cohen's kappa agreement factor between the serological and histological diagnosis was 0.686 (p<0.001) (Table 2). Considering the histological diagnosis as gold standard, we found that the serological diagnosis of *H. pylori* had a sensibility of 90.3% and a specificity of 77.8%. Patients who were positive for *H. pylori* serologically, had an odds ratio of 32.667 (95% confidence interval – CI 7.311–145.956) of having *H. pylori* infection at the histological examination.

Table 1 – *General characteristics of the study group*

Characteristics	Unit	Total	Women (*N*; %)	Men (*N*; %)	p^*
Age [years]	mean±SD min.; max.	40.4±12.1 18; 63	40.1±11.9 18; 63	41.4±12.8 22; 62	>0.05
Urban environment	*N*; %	49 (70%)	36 (70.6%)	13 (68.4%)	>0.05
Weight [kg]	mean±SD min.; max.	123.9±24 90; 210	114.8±15.8 90; 158	148.3±25.5 110; 210	<0.001
Height [cm]	mean±SD min.; max.	167±0.09 143; 190	163±0.06 143; 177	179±0.05 170; 190	<0.001
BMI [kg/m^2]	mean±SD min.; max.	44.1±6.8 35.1; 66.5	43.3±6.8 35.1; 66.5	46±6.6 37.2; 63.4	>0.05
Grade II obesity	*N*; %	23 (32.9%)	19 (37.3%)	4 (21.1%)	>0.05
Grade III obesity	*N*; %	47 (67.1%)	32 (62.7%)	15 (78.9%)	>0.05
Excess weight [kg]	mean±SD min.; max.	63.5±19.4 37.1; 133.2	59.6±16.5 37.1; 99	74.1±22.9 44.1; 133.2	0.005
WC [cm]	mean±SD min.; max.	126.6±16.1 88; 170	121.4±14 88; 158	140.8±13.1 122; 170	<0.001

BMI: Body mass index; WC: Waist circumference; SD: Standard deviation. *Between genders.

Table 2 – *Serological and histological characteristics of the study population*

Characteristic		Total (*N*=70)	Women (*N*=51)	Men (*N*=19)	*p*
		N (%)	*N (%)*	*N (%)*	
Hp (+) serology		41 (58.6)	29 (56.8)	12 (63.1)	>0.05
Hp (+) histology endobiopsy		36 (51.4)	24 (47.1)	12 (63.1)	>0.05
Hp (+) resected stomach		8 (11.4)	6 (11.8)	2 (10.5)	>0.05
Macroscopic aspect in gastroscopy	normal	21 (30)	17 (33.3)	4 (21)	>0.05
	congestion	37 (50.8)	25 (49)	12 (63.1)	
	gastritis	5 (7.1)	4 (7.8)	1 (5.3)	
	other	7 (10)	5 (9.8)	2 (10.5)	
Histological aspect in gastric biopsy	normal	16 (22.8)	13 (25.5)	3 (15.8)	>0.05
	superficial chronic gastritis	16 (22.8)	12 (23.5)	4 (21)	
	profound chronic gastritis	38 (54.4)	27 (52.9)	11 (57.9)	
Histological aspect of resected stomach	normal	35 (50)	26 (50.9)	9 (47.4)	>0.05
	superficial chronic gastritis	10 (14.3)	8 (15.7)	2 (10.5)	
	profound chronic gastritis	25 (35.7)	17 (33.3)	8 (42.1)	

Hp (+) indicates infection with *H. pylori*.

Microscopic examination revealed that 16 (22.8%) cases presented normal morphology, as no neutrophils were observed and mononuclear inflammatory cells were absent or extremely rare (Figure 1). Minimal edema, congestion and lymphoid aggregates without germinal centers basally located above the muscularis mucosae (Figure 2) were considered to be normal. On Giemsa staining, the presence of *H. pylori* was not observed in none of these cases (Figure 3).

In 16 (22.8%) cases, we identified a reduced inflam-

matory infiltrate with lymphocytes and plasma cells in the upper third of the lamina propria, edema and/or congestion. These cases were classified as chronic superficial gastritis (Figure 4). Six (37.5%) of them were associated with infection with *H. pylori*.

A number of 38 (54.4%) patients were diagnosed with chronic profound gastritis, when the inflammatory infiltrate was present diffusely in the whole thickness of lamina propria. In some cases (11 cases), lymphoid aggregates with germinal center (follicles) were detected (Figure 5). Most of these cases, namely 29 (76.3%) were *H. pylori* – positive on Giemsa staining (Figure 6).

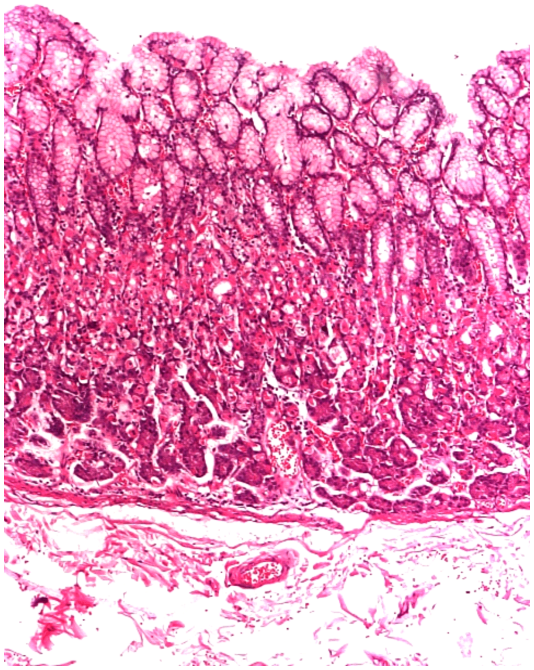

Figure 1 – ***Normal gastric body mucosa with minimal congestion and edema in lamina propria. HE staining, ×40.***

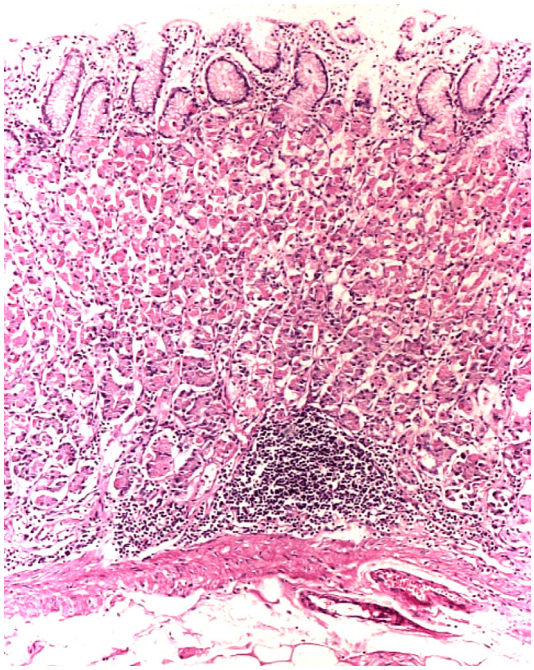

Figure 2 – ***Normal gastric body mucosa with lymphoid aggregates without germinal centers, basally located. HE staining, ×40.***

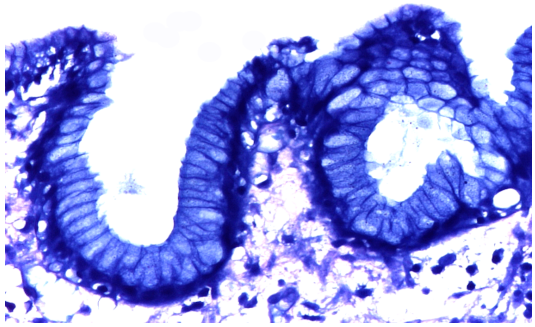

Figure 3 – ***Normal gastric mucosa, no H. pylori infection. Giemsa staining, ×200.***

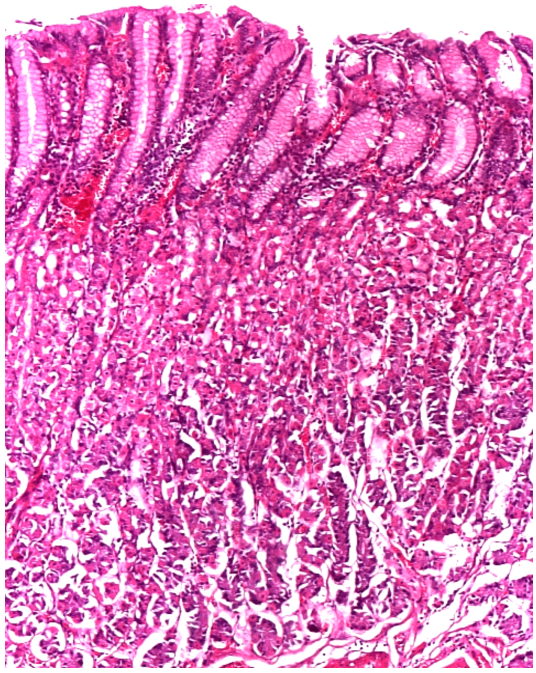

Figure 4 – ***Chronic superficial gastritis: reduced mononuclear inflammatory infiltrate in the upper third of the gastric mucosa. HE staining, ×40.***

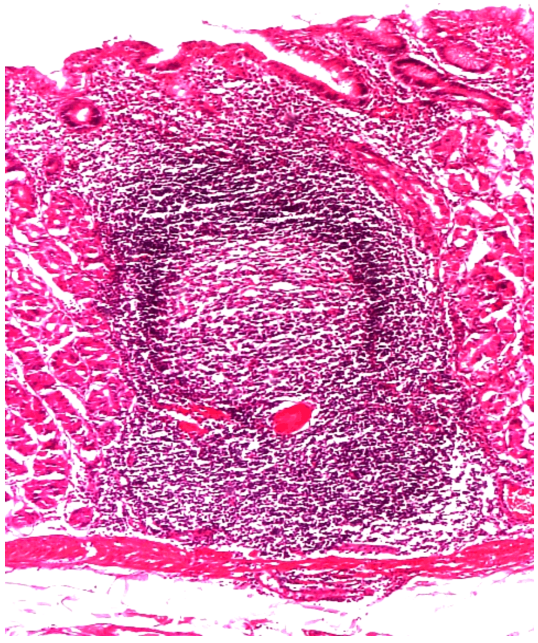

Figure 5 – ***Chronic profound follicular gastritis: abundant mononuclear inflammatory infiltrate with lymphoid follicles. HE staining, ×40.***

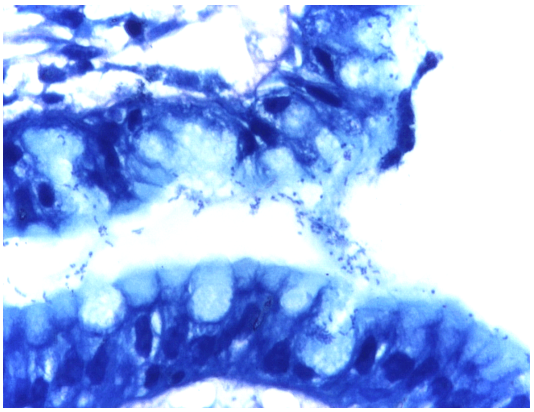

Figure 6 – *Chronic gastritis associated with H. pylori infection. Giemsa staining, ×400.*

Focally, in a reduced number of cases (two cases) with chronic profound gastritis, we observed intestinal metaplasia, the complete type. Complete type of intestinal metaplasia in stomach was diagnosed on the presence of mucin-producing goblet cells, enterocytes with brush border and Paneth cells, sometimes with a villous architecture in the surface of the mucosa (Figure 7).

No patients presented acute gastritis or chronic gastritis in the active phase. No atrophy and no dysplasia were seen in our cases. When present, the inflammatory infiltrate was limited to mucosa. Muscularis propria and subserosa were normal in all our cases.

In the resected gastric specimens, we found *H. pylori* infection in 11.4% of cases (eight cases). Among these, only one patient was also positive for *H. pylori* in the preoperatively histological examination.

The presence of *H. pylori* in the resected stomach was associated with more severe degrees of chronic gastritis in the histological examination of the resected stomach: among those positive for *H. pylori* in the resected stomach, 50% had profound chronic gastritis and 37.5% had superficial chronic gastritis (p=0.037). More information regarding the association of histological diagnosis of *H. pylori* infection with the other parameters investigated in the study can be found in Table 3.

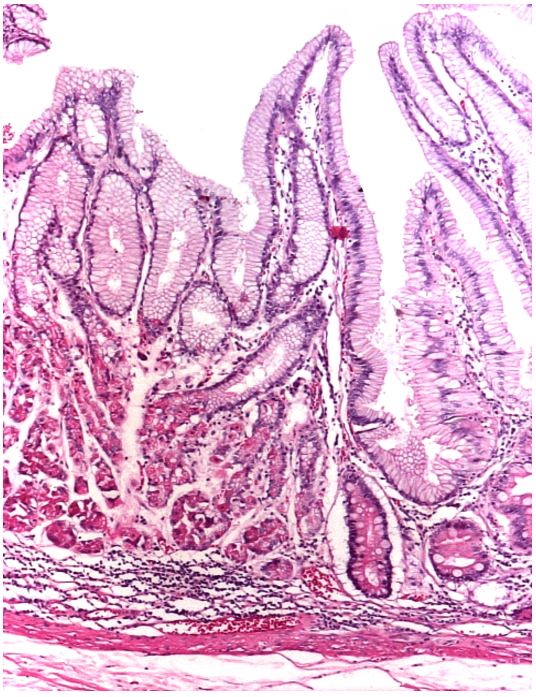

Figure 7 – *Chronic gastritis with complete type intestinal metaplasia (right). HE staining, ×40.*

Table 3 – *Differences in study population according to the histological diagnosis of H. pylori infection*

Characteristic		*H. pylori* in endobiopsy		χ^2; p	κ; p
		+	-		
		N (%)	N (%)		
H. pylori by serology	+	33 (80.5)	8 (19.5)	27.59; <0.001	0.686; <0.001
	-	3 (10.3)	26 (89.6)		
H. pylori in resected stomach	+	1 (12.5)	7 (87.5)	6.343; 0.019	-0.0199; 0.012
	-	37 (59.7)	25 (40.3)		
Histological aspect in gastric biopsy	normal	0	16 (100)	22.439; <0.001	–
	superficial chronic gastritis	6 (37.5)	10 (62.5)		
	profound chronic gastritis	29 (76.3)	9 (23.7)		
Histological aspect in resected stomach	normal	15 (42.9)	20 (57.1)	2.896; >0.05	–
	superficial chronic gastritis	7 (70)	3 (30)		
	profound chronic gastritis	15 (60)	10 (40)		
Macroscopic aspect in gastroscopy	normal	6 (28.6)	15 (71.4)	7.605; 0.055	–
	congestion	21 (56.8)	16 (43.2)		
	gastritis	4 (80)	1 (20)		
	other	5 (71.4)	2 (28.6)		

Discussion

Clinicians need a rapid and cost-efficient algorithm for the detection of *H. pylori* infection. The histological diagnosis is used as the main diagnosis method in symptomatic patients, but also as a screening method in areas with high prevalence of infection. The correct histological diagnosis is also very important in clinical practice to evaluate the efficiency of treatment.

There are several techniques for *H. pylori* infection detection, some of which are non-invasive [for instance the fecal antigen test, the serological diagnosis or the urea breath test (UBT)], other invasive (the histological examination, rapid urease test (RUT), microbiological culture and polymerase chain reaction (PCR)] due to the need of gastric mucosa biopsy taken during gastroscopy [36]. Numerous factors can influence the choice for the test used in the detection of infection, for example the

sensibility and specificity of the test, the clinical circumstances (which could be extremely variable), or the cost-efficiency of the test used. Furthermore, each of these tests has its own limitations [37].

The histological diagnosis is performed on gastric biopsy samples, allowing further for the description and classification of the inflammatory lesions of the gastric mucosa, which is frequently associated with *H. pylori* [38] and will influence the risk of developing gastric cancer through their severity and extension [8, 39]. The endoscopic findings and the microscopic examination of the biopsy sample allow for a complete assessment of the gastric mucosa and also, if the case, for the diagnosis of asymptomatic premalignant gastric lesions [40]. The contribution of an experienced pathologist and the quality of the gastric mucosa biopsy are two essential requirements for the correct histological examination. Incorrect or insufficient biopsies, sampled only from the corpus and not from the antrum, the reduced density of the *H. pylori* colonies, their distribution and type of staining used, previous treatment with proton pump inhibitors, could lead to false negative results [41, 42]. Despite these limitations, the histological examination is generally considered as the gold standard in the direct detection of *H. pylori* infection and it is also the oldest used method [43].

In our study, the histological exam for the detection of *H. pylori* was performed for all included patients, twice: first preoperatively, in the biopsy taken by gastroscopy, and second time in the stomach fragment removed by surgery, therefore after the *H. pylori* eradication treatment, when needed. The results showed that, prior to surgery, 51.4% of patients had *H. pylori* infection and all were treated, and in the resection piece, only 11.4% of patients were positive for *H. pylori*. Among them, only one patient had undergone the eradication treatment. The others had not been previously been diagnosed with *H. pylori* infection, meaning that they were probably infected in the time between preoperative evaluation and surgery or they had paucibacillary forms before surgery and could not be diagnosed on the gastric biopsy. However, in all of these patients, the *H. pylori* colonies were rare or isolated.

In patients who are not subjected for gastroscopy, the most used and easy method for diagnosis is serological, assessing the level of anti-*H. pylori* IgG antibodies. There is currently a wide range of commercially available kits, and the tests are cheap and easy to use. However, this method cannot differentiate between active infection and asymptomatic colonization, or between current or previous infection. The level of antibodies could remain high even a few months after the eradication of infection [44], which makes this test unsuitable for assessing the efficiency of treatment [45, 46]. However, serological tests remain useful in identifying patients at high risk of developing gastric cancer, which depends on the degree of gastric atrophy and intestinal metaplasia [8] and the majority of authors conclude that the serological diagnosis is still useful as screening in epidemiological studies [47].

In our study, the macroscopic aspect of the stomach during gastroscopy was normal in 50% of those without anti-*H. pylori* antibodies and in only 17.6% of those with anti-*H. pylori* antibodies; also, congestion was present in 33.3% of those without antibodies, and in 67.6% of those with antibodies, with a statistical significant difference (p=0.048). We could, therefore, suggest that the presence of congestion at gastroscopy and anti-*H. pylori* antibodies in the same patient are sufficient argument to start eradication treatment, without the need of biopsy, which could be an useful algorithm for those centers with no possibilities for pathological examinations.

Bacterial culture from the gastric biopsy is considered being the definitive proof of *H. pylori* infection, but, due to technical difficulties, the sensibility and specificity of the test could vary extremely, as greatly as to 42% [48] and it is therefore not considered to be the gold standard. The method would allow for the *in vitro* assessment of sensibility/resistance of certain antibiotics, when it is necessary to use the second line of treatment after failure to eradicate the infection first time [49]. However, in clinical practice, other easier and less invasive tests are used. UBT has a higher sensibility and specificity compared to other invasive test, but its specificity declines when other urease-producing bacteria are found in the intestine [50].

The interest for comparing different methods of diagnosis of *H. pylori* infection exists ever since the 1980s. A study published in 1997 compared the serological assessment of anti-*H. pylori* IgG antibodies (through ELISA – enzyme-linked immunosorbent assay) and the histological examination of the gastric biopsy (Giemsa staining) with the microbiological culture (as reference). The results showed that the serological diagnosis, as a non-invasive method, has a high agreement with the positive results from the invasive examinations [51]. One year later, Luthra *et al.* [52] published the results of a similar study, where they compared the serological method for detection of *H. pylori* infection with the histological method, and found statistically significant differences (p<0.001) between the two tests, similar to ours: 63.3% of patients had high levels of antibodies, but only 47.9% had the histological diagnosis of *H. pylori* infection. The authors found that these differences were maintained after adjustment for age, race and gender, and the use of antibiotics was associated with a significant reduction in the prevalence of *H. pylori* infection. The results allowed the authors to conclude that the serological method reports a higher prevalence of *H. pylori* infection compared with the histological method, and suggested the efficiency of antibiotic treatment for the eradication.

In a more recent study, Shin *et al.* [53] were the first to underline the association between metabolic syndrome and *H. pylori* infection, diagnosed through serology and histology. However, focusing on the means of detection of *H. pylori* infection, we noticed that their results were similar to ours: the authors found a *kappa* agreement coefficient of 0.69, whereas in our study found, it was equal to 0.686. Moreover, the limitations of this quoted study were that it was a retrospective study on a large group of patients, but with missing values in some patients (some did not perform gastroscopy, histological data were not available in all), whereas our study is prospective and has data in all patients for both methods for diagnosis.

A study from 2013 published the results from 91 patients referred for routine gastroscopy [54], which showed that the prevalence of *H. pylori* infection was 50.5% in the histological examination, and the serological method showed the poorest specificity and accuracy compared to other tests. However, we should acknowledge the fact that symptomatic patients and those previously treated with antibiotics were excluded from the analysis. The results of this study allowed for a classification of the tests used to detect *H. pylori* infection, according to their accuracy, and considered that the histological method is more suitable than serology. The authors concluded that, although invasive, the biopsy is to be preferred to serology, but even proposed that the two methods be combined in confirming the diagnosis of *H. pylori* infection.

Regarding patients with morbid obesity referred to bariatric surgery, studies report very different prevalence of *H. pylori* infection, between 11% [55] and 85% [56], but higher than in similar non-obese populations. The large differences between prevalence, found in literature, could be partially explained by the different methods used to detect the infection or by the study methodology (prospective *versus* retrospective studies). Our study showed a prevalence of 58.6% of *H. pylori* infection (using the serological method of diagnosis), and 51.4% (using the histological method), which are considered high prevalence for this type of population.

A retrospective study, which included 680 patients who underwent laparoscopic sleeve gastrectomy, reported a 7.8% prevalence of *H. pylori* in the resected stomach pieces [57], lower than our results, where 11.4% of patients presented *H. pylori* in the resected stomach. Furthermore, the authors did not observe an increase in the risk of post-operative complications in patients which presented *H. pylori*, meaning that the presence of infection was not a post-operative risk factor. Similarly, to the conclusions of these authors, we did not observe early post-operative complications in our study group, neither in patients with *H. pylori* infection nor in those without.

Our results showed that patients with anti-*H. pylori* IgG antibodies had an odds ratio of 32.667 (95% CI 7.311–145.956) of having *H. pylori* infection at the histological examination. Also, the serological diagnosis had a sensibility of 90.3% and specificity of 77.8%. Therefore, taking into consideration all above-mentioned data, we could suggest that a reasonable algorithm for assessing patients with morbid obesity prior to bariatric surgery is that the histological examination of the endobiopsy is to be performed only in those with positive *H. pylori* infection at serology. However, gastroscopy should be performed in all patients referred to bariatric surgery, in order to assess other possible gastric pathological findings.

To the best of our knowledge, this is the first study to evaluate the concordance of two diagnostic tests for *H. pylori* infection in asymptomatic morbidly obese patients referred to bariatric surgery in our country. Also, as an absolute novelty, we showed data not only on both methods of diagnosis (serological and histological), but also on the histological aspect of the resected stomach, as a measure of therapeutic efficiency. There are several strengths to our study. First of all, our study is prospective. Secondly, all subjects were asymptomatic, and they all underwent the same common algorithm of preoperative assessment, by the same examiners, including the fact that the two diagnosis tests for *H. pylori* infection were performed in all patients, by the same specialists. Thirdly, all patients who were *H. pylori* positive in the histological test followed the same eradication treatment regime (the triple therapy). This allowed for a thorough and correct interpretation of results.

The limitations of our study consist in the lack of adjustment of the results according to dietary factors, socio-economic status or previous treatment with proton pump inhibitors, which was not possible due to the relatively small number of subjects for this sort of statistical procedures. Also, for the histological diagnosis of *H. pylori* infection, only one method was used.

Conclusions

The discovery of *H. pylori* infection revolutionized previous concepts regarding chronic gastritis and is currently also under attention as a risk factor for cardio-metabolic diseases and cancer. Obese patients, already at high cardio-metabolic risk, have higher prevalence of *H. pylori* infection. This is to be seriously considered when referring patients for bariatric surgery. However, the lack of international agreement in regards to the algorithm for detection of *H. pylori* infection in bariatric patients creates difficulties for the multidisciplinary obesity surgery teams. Our results confirm the high prevalence of *H. pylori* infection in asymptomatic obese patients referred for laparoscopic sleeve gastrectomy. We found a high agreement between the serological and histological detection of *H. pylori* infection. However, as gastroscopy should be performed in all patients referred to surgery, our data favor the histological evaluation of all patients and the eradication treatment according to its results.

Conflict of interests

The authors have no conflict of interest to declare.

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