

PREVALENCE OF UPPER DIGESTIVE ENDOSCOPY AND GASTRIC HISTOPATHOLOGY FINDINGS IN MORBIDLY OBESE PATIENTS

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ABSTRACT – Context - The prevalence of obesity has been increasing in modern society. Roux-en-y gastric bypass is a bariatric surgery that involves the exclusion of significant part of the stomach. Atrophy, intestinal metaplasia and gastric cancer have been associated with infection by *Helicobacter pylori*. **Objectives** - To evaluate the presence of endoscopy findings and histological changes in morbid obese patients for the presence of inflammatory cells, inflammatory activity, lymphoid hyperplasia, *H. pylori* infection, atrophy and intestinal metaplasia in the gastric mucosa. **Methods** - Upper digestive endoscopy and gastric histopathological were studied in 126 obese patients in the preoperative evaluation for bariatric surgery. **Results** - Upper digestive endoscopy abnormalities were diagnosed in 73/126 (57.9%) patients. In three patients (2.4%) the upper gastrointestinal endoscopy diagnosed gastric ulcer and one patient (0.8%) had duodenal ulcer. The histopathological from gastric biopsies of these obese patients showed 65.1% of mucosa inflammation, inflammatory activity in 50.0%, infection by *H. pylori* in 53.2%, lymphoid hyperplasia in 50.0% and atrophy and/or intestinal metaplasia in 16.7%. **Conclusions** - In present study, with routine preoperative upper gastrointestinal endoscopy and histopathological examination, were detected 57.9% patients with endoscopy abnormalities, high prevalence of infection by *H. pylori* (53%) and 16.7% of gastric atrophy and/or intestinal metaplasia.

HEADINGS – Obesity, morbid. Gastric bypass. Endoscopy gastrointestinal. Helicobacter infections.

INTRODUCTION

Obesity is considered to be a growing problem in modern society and its prevalence has been increasing in both developed and developing countries⁽³⁾.

Most types of bariatric surgery include resection, sutures or partial stomach exclusion. The most frequent bariatric surgery is the Roux-en-Y gastric bypass (RYGBP), which involves partial exclusion of the stomach⁽³⁾.

Mucosal alterations after vertical banded RYGBP have not been clearly evaluated because the excluded stomach is not easily reached by conventional endoscopy^(18, 21, 39).

Infection by the *Helicobacter pylori* (*H. pylori*) causes inflammation of the gastric mucosa, which can develop atrophy, intestinal metaplasia, dysplasia and cancer^(3, 6, 27). The eradication of the *H. pylori* leads to a regression of the inflammatory process of the gastric mucosa, the same does not happen in the more advanced stages, with the presence of atrophy and intestinal metaplasia^(8, 13, 24, 28, 40).

The present study aims to evaluate the prevalence of endoscopic findings and gastric histopathological alterations in morbidly obese patients. The histopathological alterations comprise presence of inflammation cells, inflammatory activity, presence of lymphoid hyperplasia, infection by the *H. pylori*, atrophy and intestinal metaplasia in the gastric mucosa.

METHODS

Consecutive study conducted in outpatient service with 126 morbidly obese patients [body mass index (BMI) = ≥ 40 kg/m²] or obese patients (with BMI between 35-40 kg/m²) with indication for bariatric surgery by significant co-morbidities. Upper gastrointestinal endoscopy (UGI) and gastric biopsies was required in the preoperative period. The patients had not undergone UGI or treatment to eradicate *H. pylori* previously. The study was approved by the Ethics Committee of Hospital Conceição, Porto Alegre, RS, Brazil, and all patients gave informed consent prior to their inclusion in the study. The upper endoscopies were realized by

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the same gastroenterologist with a video-endoscope Fujinon series 400. Four gastric biopsies were carried out, two in the body and two in the antrum, always in the greater gastric curvature. Los Angeles and Sydney classification were used in the endoscopic analysis of esophagitis and gastritis^(1, 8).

Alterations of the gastric mucosa were classified by the pathologist as an inflammatory process through the presence of lymphocytic and plasmocytic cells and inflammatory activity through the presence of neutrophils. Lymphoid hyperplasia, presence of *H. pylori*, epithelial atrophy and intestinal metaplasia were other parameters analyzed.

RESULTS

Female population predominated (104 patients - 82.5%). The mean age was 42.08 years and the mean BMI was 51.2 kg/m² (standard deviation \pm 9.34).

Upper digestive diseases (UGI) were diagnosed in 73 patients (57.9%) through endoscopy, being esophagitis and/or hiatal hernia in 31 cases (24.6%); exsudative/enanthematous gastritis in 6 patients (4.8%); exsudative/enanthematous erosive gastritis in 38 patients (30.2%); gastric ulcer in 3 (2.4%); duodenal ulcer in 1 obese (0.8%). In 53 (42.1%) patients the endoscopy was normal (Table 1). Some patients had more than one lesion at UGI.

TABLE 1. Upper digestive endoscopic findings (n = 126)

Diagnosis	Patients	Percentage
Esophagitis and/or hiatal hernia	31	24.6%
Exudative/enanthematous gastritis	6	4.8%
Erosive gastritis	38	30.2%
Gastric ulcer	3	2.4%
Duodenal ulcer	1	0.8%
Normal endoscopy	53	42.1%

The prevalence of inflammatory cells in the gastric mucosa was found in 82 (65.1%) patients, inflammatory activity in 63 (50.0%), lymphoid hyperplasia in 63 (50.0%), infection by *H. pylori* in 67 (53.2%), epithelial atrophy and/or intestinal metaplasia in 21 (16.7%) and normal gastric histopathological in 33 (26.2%) patients (Table 2).

TABLE 2. Histological analysis of gastric mucosa (n = 126)

Diagnosis	Patients	Percentage
Mucosa inflammation	82	65.1%
Inflammatory activity	63	50.0%
Lymphoid hyperplasia	63	50.0%
<i>Helicobacter pylori</i>	67	53.2%
Epithelial atrophy and/or intestinal metaplasia	21	16.7%
Normal	33	26.2%

Five (8.5%) of the 59 patients with negative results for *H. pylori* had gastric intestinal metaplasia or epithelial atrophy.

In these obese patients, the gastric histopathological parameters studied was not statistical different ($P < 0.05\%$) in the patients with stomach or duodenum abnormalities at

UGI, comparatively with patients without any gastroduodenal abnormalities at UGI (Table 3), except the parameter inflammatory activity (Table 3). The diagnosis of inflammatory activity in gastric mucosa was greater in the group of normal UGI when compared with patients with UGI findings ($P = 0.04$) (Table 3).

DISCUSSION

The prevalence of obese individuals in USA (defined as BMI > 30 kg/m²) has increased from 15.3% to 23.9% in the period of 1995 to 2005 and the prevalence of morbidly obese individuals (BMI > 40 kg/m²) was of 4.8% in 2005⁽⁴⁾.

The most performed bariatric procedure is the RYGBP, which involves the exclusion of a significant part of the stomach. Lesions of the excluded stomach have been reported and the diagnosis of this possibility is difficult, onerous, not always available and, as a result of that, gastric diseases, such as ulcers and malignant neoplasms in the excluded stomach may not be diagnosed^(18, 21, 39).

H. pylori is associated to gastric diseases, such gastritis, ulcer, epithelial atrophy, intestinal metaplasia, lymphoma, adenocarcinoma^(9, 31). The prevalence of infection caused by the *H. pylori* in Latin America is around 60%, varying from 30% to 90%⁽⁵⁾.

H. pylori causes inflammation of the gastric mucosa in all infected individuals⁽¹⁰⁾. This inflammation consists initially in recruitment of neutrophils, followed by lymphocytes, with later epithelial damage⁽¹⁵⁾. The chronic inflammatory process increases the turnover of epithelial cells and apoptosis (cellular death), evolving to epithelial atrophy and intestinal metaplasia⁽⁹⁾.

The development of gastric cancer involves several stages and can start with chronic gastritis, atrophy, intestinal metaplasia, dysplasia and finally invasive cancer⁽⁶⁾. The World Health Organization concluded, in 1994, for the existence of epidemiological and histological evidences to classify the *H. pylori* as carcinogenic, group 1⁽¹⁶⁾. Studies have shown the increased incidence of gastric cancer in the population infected by the *H. pylori*, as well as a positive correlation between intestinal metaplasia and stomach cancer^(27, 31, 37).

Eradication of the *H. pylori* leads to a regression of the inflammation, reduction of cellular turnover, increase in the acid secretion, which are important factors for the prevention of gastric cancer^(9, 14, 22, 24). Some studies have concluded for the irreversibility of the carcinogenic process, even with the eradication of the *H. pylori*, basically when intestinal metaplasia of the stomach was present^(13, 14, 20).

The incidence of stomach cancer is still now a frequent causes in the group of world malignant neoplasm, being an important cause of mortality⁽¹⁷⁾. Few cases of gastric cancer in the bypassed stomach have been described after RYGBP for morbid obesity^(18, 36). Factors such as difficulty of access to the excluded stomach and the reduced number of patients with adequate follow-up after bariatric surgery can interfere in this analysis.

The rationale for performing UGI in the candidates to bariatric surgery is to detect and treat lesions that might

TABLE 3. Obese patients with endoscopic abnormalities in stomach and/or duodenum versus obese with normal stomach and/or duodenum at endoscopy. Gastric histopathological analysis

	Abnormal stomach/duodenum (n = 46)		Normal stomach/duodenum (n = 80)		Odds ratio (95%CI)	P-value
	n	%	n	%		
Mucosal inflammation	48	60.0	34	73.9	0.52 (0.23-1.17)	0.08
Inflammatory activity	35	43.8	28	60.9	0.50 (0.23-1.04)	0.04
Lymphoid hyperplasia	40	50.0	23	50.0	1.00 (0.48-2.06)	0.57
<i>Helicobacter pylori</i>	41	51.3	26	56.5	0.80 (0.38-1.67)	0.35
Epithelial atrophy and/or intestinal metaplasia	15	18.8	6	13.0	1.53 (0.55-4.28)	0.28
Normal	22	27.5	11	23.9	1.20 (0.52-2.78)	0.41

CI = confidence interval

potentially lead to complications in the immediate postoperative period, or result in diseases in the months or years following RYGBP surgery^(25, 26).

Eradication of the *H. pylori* as a routine in the preoperative of bariatric surgery is still debated and scarce researches were performed in regards the histology of gastric mucosa^(2, 7, 11, 23, 25, 26, 30, 33, 34, 35).

Mong et al.⁽²⁵⁾ studied 272 patients who were candidates to bariatric surgery, in which 33 (12%) showed lesions in the upper digestive tract. Endoscopic findings were: esophagitis in 3.7%; Barrett's esophagus in 3.7%; gastric ulcer and/or erosive gastritis in 3.2%; duodenal ulcer in 0.7% and gastric carcinoid tumor in 0.3%.

Muñoz et al.⁽²⁶⁾ diagnosed UGI abnormalities in 46% in the obese patients, one of them with gastric cancer.

Dutta et al.⁽¹¹⁾ found prevalence of gastric ulcer of 2.9%, identified histologically gastritis in 23.7% and *H. pylori* in 6.9% in the obese patients, finding that was not different from the control (non-obese) patients.

Safatle-Ribeiro et al.⁽³³⁾ examined the excluded stomach in 40 patients by enteroscopy in an average of 78 months after surgery. In this study, an inflammatory process in the gastric mucosa was diagnosed in 100% of the cases, atrophy in 14%, intestinal metaplasia in 11.4% and infection by the *H. pylori* in

20%. All cases of *H. pylori* infection in the excluded stomach also had the same infection in the gastric functional pouch.

A review of the literature revealed that the preoperative prevalence of *H. pylori* ranges from 6.9% to 61.3% in obese patients undergoing bariatric surgery^(2, 11, 12, 19, 23, 29, 30, 32, 38).

In the present study, there were 73 (57.9%) cases with abnormalities in upper digestive endoscopy and 53 (42.1%) patients with normal UGI. In 42 cases (33.3%) gastroduodenal abnormalities were diagnosed, like exudative/enanthematous gastritis, erosive gastritis, gastric or duodenal ulcers. Approximately 50% of the obese patients showed inflammatory activity and infection by the *H. pylori* and 16% had atrophy and/or intestinal metaplasia in gastric mucosa. The absence of *H. pylori* infection does not rule out gastric metaplasia or atrophy.

The prevalence of *H. pylori* infection and gastric atrophy and/or intestinal metaplasia in obese patients with gastric or duodenal lesions at endoscopy, comparatively with obese patients with normal stomach or duodenal at UGI, was not statistical different ($P < 0.05$).

The long-term follow-up of patients submitted to RYGBP may probably clarify if histopathological abnormalities of the gastric mucosa diagnosed prior surgery could interfere in the postoperative complications or in risk of gastric cancer.

Dietz J, Ulbrich-Kulczynski, Souto KEP, Meinhardt NG. Prevalência de achados gástricos endoscópicos e histopatológicos em pacientes obesos. Arq Gastroenterol. 2012;49(1):52-5.

RESUMO – Context - A prevalência de obesidade tem aumentado significativamente nos últimos anos. Bypass gástrico em Y-de-Roux, uma das técnicas cirúrgicas realizadas no tratamento da obesidade, envolve exclusão de parte do estômago. Atrofia, metaplasia intestinal e câncer gástrico têm sido associados com infecção pelo *Helicobacter pylori*. **Objetivos** - Avaliar a prevalência de achados endoscópicos e alterações histopatológicas em pacientes obesos mórbidos em relação à presença de células inflamatórias, atividade inflamatória, hiperplasia linfóide, infecção pelo *H. pylori*, atrofia e metaplasia intestinal na mucosa gástrica. **Métodos** - Achados na endoscopia digestiva alta e histopatologia gástrica foram analisados em 126 pacientes obesos na avaliação pré-operatória de cirurgia bariátrica. **Resultados** - Anormalidades endoscópicas do trato digestivo superior foram diagnosticadas em 73/126 pacientes, com três pacientes (2,4%) apresentando úlcera gástrica e um paciente (0,8%) com úlcera duodenal. A histopatologia das biopsias gástricas dos obesos estudados revelou 65,1% de processo inflamatório na mucosa, atividade inflamatória em 50,0%; infecção pelo *H. pylori* em 53,2%, hiperplasia linfóide em 50,0%, atrofia e/ou metaplasia intestinal em 16,7%. **Conclusões** - No presente estudo, através de endoscopia digestiva alta e histopatologia de biopsias gástricas, foram detectados 57,9% de pacientes com anormalidades endoscópicas, prevalência de infecção pelo *H. pylori* em 53% e atrofia e/ou metaplasia intestinal em 16,7%.

DESCRIPTORIOS – Obesidade mórbida. Derivação gástrica. Endoscopia gastrointestinal. Infecções por helicobacter.

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