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Original article

Association between Variables of Eating Habits and Gastric Trophic Changes in a Gastroenterology Institution in Medellín, Colombia

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Abstract

Aim: To establish the relationship between consuming foods considered risk factors for gastric cancer and trophic changes in gastric mucosa. **Materials and methods:** Cross-sectional study. We included patients older than 18 admitted for upper GI endoscopy with biopsies who adequately answered a survey of personal history and eating habits. Those with a history of gastric cancer or gastric surgical resection for any reason were excluded. The association between feeding variables and trophic changes in the gastric mucosa was estimated. **Results:** In a population of 1,096 patients, the average age was 51 years (standard deviation [SD]: 15.5), and 59% were women. Trophic changes in the gastric mucosa were identified in 173 patients (15.8%). No statistical association was found between the independent variables of eating habits, obesity, and positive *Helicobacter pylori* versus the variable "trophic changes," unlike the variable "family history of gastric cancer" (odds ratio [OR]: 1.49 95% confidence interval [CI]: 1.03-2.17, $p = 0.036$). One case of high-grade dysplasia was detected in the study population (0.91 cases in 1,000 patients). **Conclusions:** No association was established between eating habits and trophic changes in the gastric mucosa in the studied population. A family history of gastric cancer is a statistically significant risk factor for developing atrophy, metaplasia, or dysplasia changes.

Keywords

Eating habits, atrophy, metaplasia, dysplasia.

INTRODUCTION

Nitrates are nitrogenous compounds present in nature, which can be acquired by eating certain foods. When nitrates are reduced to nitrites by bacteria or macrophages, they can react with other nitrogenous substances to form N-nitroso compounds known as mitogens and carcinogens[1].

Nitrosamines are N-nitroso compounds found in preserved meats such as bacon, sausages and other cold meats, and salt-cured or smoked fish. Another source of nitrosamines is found in alcohol. Salt can also act as an inflammatory agent of the stomach mucosa, which is why diets high in salt are associated with a higher risk of gastric cancer, as is the consumption of ultra-processed foods[2,3].

Gastric adenocarcinoma represents one of the leading causes of death from cancer worldwide, and *Helicobacter pylori* infection is considered the most important known risk factor for this disease[4,5]. This infection triggers inflammatory phenomena in the gastric mucosa[6], resulting in a progressive decrease in hydrochloric acid and favoring

bacterial colonization of the gastric mucosa[7], which could reduce nitrates to nitrites. Therefore, there is an interaction between *H. pylori* infection and dietary risk factors for gastric cancer.

There are other risk factors for changes at the level of the gastric mucosa (atrophy, intestinal metaplasia, dysplasia, and gastric cancer) in addition to the nutritional factors already mentioned, such as smoking[8], high salt intake in food and alcohol[9,10], genetic factors (history of family gastric cancer in the first degree of consanguinity)[11], obesity (a risk factor defined as a body mass index [BMI] greater than 30)[12] and chronic use of anti-inflammatory drugs[13], lifestyle habits such as cooking food over firewood and eating food at high temperatures.

The present study seeks to establish the association between the reference population's eating habits and gastric trophic changes.

MATERIALS AND METHODS

A cross-sectional study was conducted in a Gastroenterology institution in Medellín, Colombia, between December 2021 and February 2022. It consecutively included all outpatients over 18 years of age admitted for upper gastrointestinal endoscopy with biopsies for histology and those who adequately responded to a personal history and eating habits survey. Patients with a history of gastric cancer or gastric surgical resection for any reason were excluded.

A database was built from the completed questionnaires, including the following variables: indication for the examination, age, sex, weight, height, history of *H. pylori* infection, family history of gastric cancer, smoking, consumption of non-steroidal anti-inflammatory drugs (NSAIDs), canned foods, fruits, cold meats, smoked meat, alcohol, snacks, sweets or ice cream, food preparation over charcoal or wood, added salt to prepared meals and extremely hot food. Subsequently, the histopathology results were reviewed, creating the variables for *H. pylori*, atrophy, metaplasia, or dysplasia.

One thousand ninety-six endoscopies were performed during the study period in patients who met the inclusion criteria and completed the survey appropriately. The examinations were performed by a team of nine gastroenterologists and interpreted by three pathologists with training in gastrointestinal histopathology.

The endoscopes used are high definition, with different light filters such as linked color imaging (LCI), blue laser imaging (BLI), narrow-band imaging (NBI), and magnification for mucosal characterization. The findings in the endoscopic report describe the thickness of the mucosa, type of surface, chronicity of inflammation and type of activity, type of cells, presence of *H. pylori,* atrophy, metaplasia, percentage of sample involvement, dysplasia, and degree of involvement.

To diagnose *H. pylori*, which generally infects the mucosa of the gastric antrum and body, 70% of the cases were diagnosed from gastric biopsies stained with routine staining (hematoxylin and eosin). The remaining percentage required more sensitive special stains such as Giemsa, Diff-Quick, or Warthyn-Starry*[1,2].

Regarding the degree of gastric atrophy, representative fragments of the gastric mucosa were evaluated in the antral portion, angular incisura, and oxyntic mucosa, as determined by the Sydney protocol[14]. Subsequently, indicators of glandular contraction, lamina propria fibrosis, or intestinal metaplasia were searched. These findings were quantified in terms of the percentage of atrophy with a visual analog scale (VAS) that assigns scores from 0 to 3:

- no atrophy (0%): score 0
- mild atrophy (1-30%): score 1
- moderate atrophy (31-60%): score 2
- severe atrophy (greater than 60%): score 3

A global atrophy score containing the independent scores for the antral and corpus mucosa was obtained from these results. The OLGA stage (the Operative Link for Gastritis Assessment staging system)[15] was obtained by combining the total "antrum score" with the "body score."

The dependent variable, "trophic changes in the gastric mucosa," was the positive histopathological result for atrophy, metaplasia, or dysplasia.

The conduct of the study was approved by the institutional ethics committee, which considered it risk-free since there was no intervention or intentional modification of the biological, physiological, psychological, or social variables of the individuals participating in the study. At the same time, it contemplates the fundamental principles of research ethics under the Declaration of Helsinki version 2013[16] and the provisions of Resolution 008430/1993 issued by the Colombian Ministry of Health[17].

Statistical analysis

We performed a descriptive analysis of the sociodemographic variables and eating habits and subsequently sought to establish the association of these variables with trophic changes in the mucosa in histopathology. Absolute and relative frequencies were determined for qualitative variables, and measures of central tendency and dispersion were used for quantitative variables. The chi-square association test was employed for independent samples, and the odds ratio (OR) was estimated with its respective 95% confidence interval (CI), considering a statistically significant

p-value < 0.05. Statistical analysis was completed with Excel version 2010 and Jamovi version 1.6.23.

RESULTS

Data were obtained from the surveys and endoscopy results of 1,096 patients who underwent the examination between December 2021 and February 2022 at a high-complexity gastroenterology institution in Medellín, Colombia. All patients included in the study voluntarily accepted the completion of the eating habits survey and completed the informed consent prior to the procedure.

The average age of the studied population was 51 (standard deviation [SD]: 15.5), with a range between 18 and 97 years, and 59% were women. Demographic information and clinical history of interest are described in **Table 1**.

Four main indications for requesting endoscopy were identified: dyspepsia (26.6%), gastroesophageal reflux disease (22.6%), intestinal metaplasia (8.8%), screening for gastric cancer (7.6%), and other indications (34.4%).

Eating habits variables were included in the survey following what was reported in the literature as risk factors for gastric cancer. Three groups were established to determine consumption frequencies: often, sometimes, or never. The results are described in **Table 2**.

We identified a history of *H. pylori* infection in 417 people (38.2%) and new cases in 223 individuals (20.3%), for a prevalence of 52.5% (n = 575).

Trophic changes in the gastric mucosa were found in the histopathology of 173 patients (15.8%), distributed in atrophy (n = 152; 13.9%), metaplasia (n = 163; 14.9%), and dysplasia. (n = 2; 0.2%).

When running the chi-square test between the independent variables of feeding habits and the dependent variable "trophic changes," no statistically significant values were obtained to accept the association hypothesis ($p < 0.05$).

The analysis of the variables *H. pylori* prevalence, obesity, and family history of gastric cancer and their association with trophic changes in the gastric mucosa is described in **Table 3**.

Two dysplasia cases (one high grade) were in the 1,096 patients studied.

Table 1. Demographic information and medical history

Variable	n (%)
Sex	
- Female	656 (60)
- Male	440 (40)
Age range	
- < 35	185 (16.9)
- 35-39	97 (8.9)
- 40-49	204 (18.6)
- 50-59	224 (20.4)
- 60-69	244 (22.3)
- 70-79	120 (10.9)
- > 80	22 (2)
BMI	
- Underweight	26 (2.4)
- Normal	537 (49.1)
- Overweight	409 (37.4)
- Obesity	122 (11.2)
Family history of GC	
- Yes	225 (20.7)
- No	864 (79.3)
Smoking	
- Yes	62 (5.7)
- No	1032 (94.3)
NSAID use	
- Yes	155 (14.2)
- No	933 (85.8)

GC: gastric cancer. Table prepared by the authors.

DISCUSSION

Both genetic and environmental factors influence the cancer development process. Around 50% of cases can be caused by environmental agents, mainly dietary habits and social behavior[18]. The typical diet in most countries contains nitrates, nitrites, and nitrosamines. Nitrates and nitrites are found naturally in fruits and vegetables, an essential part of a healthy diet. At the same time, nitrates and nitrites are used as additives in processed meats such as ham, bacon, and sausages to preserve the appearance and flavor of meat products and delay their deterioration. High intake of processed meats is linked to an increased risk of gastric cancer. N-nitroso dimethylamine (NDMA) is one of the most common nitrosamines in foods and is a potent carcinogen capable of inducing malignant tumors in the liver, lungs, and stomach[3]. Most gastric

Table 2. Eating habits in the studied population

Variable	n (%)		
Canned foods	6 (0.5) Often	719 (65.7) Sometimes	370 (33.8) Never
Fruit	545 (49.7) Often	535 (48.8) Sometimes	16 (1.5) Never
Cold meats	85 (7.8) Often	840 (77.1) Sometimes	164 (15.1) Never
Food preparation over charcoal or wood	0 (0) Often	188 (17.2) Sometimes	906 (82.8) Never
Smoked meat	11 (1) Often	427 (39) Sometimes	656 (60) Never
Extra salt	89 (8.1) Always	455 (41.6) Sometimes	551 (50.3) Never
Alcohol	77 (7) Often	713 (65.1) Sometimes	305 (27.9) Never
Snacks	46 (4.2) Often	872 (79.7) Sometimes	176 (16.1) Never
Sweets, ice cream	96 (8.8) Often	925 (84.4) Sometimes	75 (6.8) Never
Food temperature	46 (4.2) Very hot	1019 (93.1) Lukewarm	29 (2.7) Cold

Table prepared by the authors.

cancers occur from chronic mucosal inflammation and are preceded by the development of gastric intestinal metaplasia and atrophy. In the US population, *H. pylori* infection, smoking, and a family history of gastric cancer are independent risk factors for intestinal metaplasia[19]. Additionally, other risk factors such as obesity, age, the use of anti-inflammatory drugs (NSAIDs), and gastroesophageal reflux have been related to the development of changes in the gastric mucosa[20].

In our study population, the trend towards a healthy diet was identified, framed in the "frequent consumption" of fruits and vegetables (49.7%) and the low "frequent consumption" of cold meats (7.8%), canned foods (0.5%), smoked meat (17.2%), and sweets (8.8%). Frequent consumption was defined as eating these products more frequently than three times per week. The population surveyed did not report high salt use in their preparations and mostly ate foods at a medium temperature. In the absence of marked trends in the consumption of foods considered risk factors, no statistically significant results were obtained to accept the hypothesis of association with trophic changes in the gastric mucosa.

Table 3. Association of clinical variables with trophic changes in the gastric mucosa

Variable	OR	95% CI	*p*-value
H. pylori	0.979	0.70-1.36	0.89
Obesity	1.19	0.72-1.95	0.48
Family history of GC	1.49	1.03-2.17	0.036

Table prepared by the authors.

The prevalence of trophic changes in the study population was determined, finding a prevalence of atrophy of 13.9%, intestinal metaplasia of 14.9%, and dysplasia of 0.2%. These data contrast with ranges reported in the literature that vary between 9.4% and 63% for the prevalence of atrophy and 7.1% and 42.5% for intestinal metaplasia[21] within the ranges described.

H. pylori infection is deemed an initial step in the cascade of gastric carcinogenesis[22], and its eradication is a protective factor in this sense. However, it is still uncertain whether eradicating the bacteria can reverse the trophic

changes already triggered in the mucosa[23]. It has recently been documented that nutritional factors and the gastrointestinal microbiota can change the balance of *H. pylori* (commensal versus pathogen), and thus, its carcinogenic potential is variable[24]. In our study, there was a history of *H. pylori* infection in 417 people (38.2%) and new cases in 223 individuals (20.3%), for a prevalence of 52.5% (n = 575). However, no statistically significant association was found between *H. pylori* and trophic changes in the gastric mucosa (OR: 0.979; 95% CI: 0.708-1.36; $p = 0.899$).

Another of the main variables of the analysis was obesity (BMI > 30). It is estimated that 4% of all cancers in men and 7% in women can be attributed to obesity. Gastric cancer has also been associated with increased BMI, especially gastric adenocarcinoma of the cardia. Changes in eating habits have been shown to have a protective effect against gastric cancer, such as increasing the intake of fruits and vegetables, beta-carotene, and ascorbic acid, among others, which suggests that the risk of gastric cancer could be modified by diet[12]. In the study population, a prevalence of obesity of 11.2% was found, with no statistically significant association with gastric trophic changes (OR: 1.19; 95% CI: 0.72-1.95; $p =$ 0,48). No patients with heart cancer were identified.

Family history also plays an essential role in the carcinogenesis of gastric cancer, mainly when there are first-degree relatives with this condition[25]. The finding of a family history of gastric cancer was reported by 20.7% of the study population, and a statistically significant association was found with trophic changes in the gastric mucosa (OR: 1.49; 95% CI: 1. 03-2.17; $p = 0.036$), which coincides with reports in the literature describing that gastric cancer can have a familial aggregation rate of up to 10%[25].

Given the low frequency of consumption of foods considered risk factors for gastric cancer in our study population, we could not establish an association between eating habits and gastric trophic changes, which does not rule out that such an association may exist. One hypothesis is possible changes in eating habits due to patients' previous symptoms. According to the findings, it would be necessary to expand the coverage of the study or conduct it on a population group with different demographic, cultural, and environmental characteristics.

CONCLUSIONS

An association was not established between eating habits and trophic changes in the gastric mucosa in the population studied. *H. pylori* infection and obesity did not show a statistically significant association with changes in atrophy, metaplasia, and dysplasia, unlike a family history of gastric cancer, which is shown to be an independent risk factor for the development of these precursor lesions.

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