ORIGINAL ARTICLE

Endoscopic mucosal phenotypes and endoscopic Sydney system gastritis assessment in relation to *Helicobacter pylori* infection and upper digestive clinical signs: A 2-year study among patients with gastroduodenal disorders in Cameroon

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Abstract

Background and Aim: *Helicobacter pylori* represents the major pathogen in the pathophysiology of diverse gastrointestinal conditions. This study sought to determine the endoscopic aspect of the gastric mucosa in relation to *H. pylori* infection in Cameroon.

Methods: This study was conducted in three reference health facilities in Cameroon from October 2020 to October 2022. The study enrolled 494 consecutive volunteer dyspeptic patients attending to the gastroenterology department of the selected health facilities. A description of the aspect of gastric mucosa of all participants was performed during endoscopy examination, and biopsies were collected for *H. pylori* detection using rapid urease tests.

Results: Gastritis, ulcerated lesions, duodenitis, esophagitis, normal mucosa aspect, bulbitis, and gastric neoplastic lesions were found in 40.1, 22.3, 10.9, 10.3, 9.7, 6.3, and 0.40% of biopsy samples, respectively. Erythematous/exudative (45.9%) and enterogastric reflux (12.2%) were the main gastritis types recorded. *H. pylori* was present in 58.1, 46.3, 87.1, 66.7, and 61.8% in gastritis, duodenitis, bulbitis, esophagitis, and ulcerated lesions, respectively. A positive relationship was noticed between the presence of *H. pylori* and gastritis (1.037 [0.720–1.493]; $P = 0.845$), bulbitis (4.237 [1.602–11.235]; $P = 0.004$), esophagitis (1.515 [0.822–2.793]; $P = 0.183$), ulcerated lesions (1.233 [0.798–1.904]; $P = 0.345$), erythematous/exudative gastritis (1.354 [0.768–2.389]; $P = 0.295$), and enterogastric reflux gastritis (1.159 [0.492–2.733]; $P = 0.736$).

Conclusion: Gastritis and erythematous/exudative gastritis are the most frequent gastrointestinal pathophysiology conditions in dyspeptic patient in our milieu. *H. pylori* infection is responsible for 94.8% of the gastrointestinal pathophysiology conditions with bulbitis as the condition is significantly associated with this bacterium infection.

Introduction

Long-term infection with *Helicobacter pylori* represents a serious health burden, with more than the half of the world's population being victims.[1] *H. pylori* infection is a communicable disease although the main route of transmission has not yet elucidated.[2] Several factors such as lack of respect for basic hygiene rules, socioeconomic status, age, blood group O, tobacco consumption, overweight/obesity, use of nonsteroidal anti-inflammatory drugs (NSAIDs), promiscuity/overcrowded living conditions, and family history of gastric disease have been incriminated having a role in the acquisition of this infection.[3,4] The prevalence of this infection is variable in different regions of the world; it is estimated at 15.5% in developed countries, while in underdeveloped countries, it is around 93.6%.[5]

Symptomatic patients usually present epigastric burning and pressure, epigastric pain predominantly associated with other upper gastrointestinal symptom such as pain after meal, nausea,

vomiting, bloating and belching or frequent burping, pain on empty stomach, gastric fullness, and anorexia.[6] Even though the vast majority of infected individuals remains asymptomatic or displays rather minor unspecific symptoms, this pathogen is mainly implicated in the pathophysiology of diverse gastrointestinal conditions including gastritis, peptic ulcer disease, gastric adenocarcinoma, and mucosa-associated lymphoma.[7–9] Hence, accurate diagnosis of *H. pylori* infection is crucial in the management strategy to minimize the *H. pylori*-related gastropathologies and gastric cancer.[10] Several invasive and non-invasive diagnostic tests are available for the detection of infection with this pathogen. Invasive tests, which require endoscopic surgery and biopsy specimens, offer the possibility to explore gastric mucosa during endoscopic examination. Endoscopic examination is a macroscopic assessment of the aspect of the gastric mucosa lining as well as their topographical distribution. Assessment of gastric mucosa in relation to *H pylori* infection is of great significance in evaluating the evolution of the disease and to elucidate the burden of the infection with this pathogen. The Sydney system is used for the assessment of gastritis in the stomach. This system takes into account the topographical distribution of lesions (gastric corpus, antrum, and the entire stomach) and the categories of endoscopic images, which may indicate erythematous/exudative gastritis, flat erosive gastritis, raised erosive gastritis, reflux gastritis, hemorrhagic gastritis, atrophic gastritis, and rugal hyperplastic gastritis.[11–14]

As revealed in previous study, *H. pylori* infection is common in Cameroon.[4] In addition, data from a 5-year retrospective study on 1290 dyspeptic patients for whom histological features of the gastric mucosa were investigated revealed the prevalence rates of 75.35, 8.2, 7.7, 2.8, 9.3, 1.55, and 0.8% for chronic gastritis, atrophic gastritis, intestinal metaplasia, dysplasia, carcinoma, hyperplastic polyps, and MALT lymphoma, respectively.[15] Moreover, *H. pylori*-infected participants were at a high risk to develop gastric lesions with an odds ratio of 1.1775, 1.4866, 1.4415, and 1.2088 for gastritis, atrophic gastric, dysplasia, and carcinoma, respectively.[15] Despite this high prevalence of *H. pylori* infection and disease burden related to this infection in Cameroon, there are a paucity of studies on the epidemiology of this infection in relation with endoscopic aspects of the gastric mucosa in the country. Therefore, this study assesses *H. pylori* infection and endoscopic pathological features among dyspeptic patients in the West and Centre Region of Cameroon in order to fill the gap of knowledge on the correlation between *H. pylori* infection and lesions in the gastric mucosa. This knowledge will provide useful insights into the prevention and management strategies of this infection in the country.

Methods

Study area. This cross-sectional study was conducted in Bafoussam and Yaoundé metropolis located in the West and Centre Regions of Cameroon, respectively, for a period of 16 months.

In Bafoussam (capital of the Western Region of Cameroon), the study was carried out at the Regional Hospital Bafoussam from October 2020 to October 2022. The Regional Hospital Bafoussam is the reference medical structure in the Cameroon health system located in the Western Region of the country. It receives a wide variety of patients from around

20 district hospitals, 26 medical centers, and 240 health centers within this region of the country.

In Yaoundé (capital of Cameroon), the study was conducted in two health facilities: the General Hospital Yaoundé and the Centre Médicale la Cathédrale from December 2021 to October 2022. General Hospital Yaoundé is a tertiary hospital in Yaoundé, receiving a wide variety of patients not only from other hospitals in Yaoundé itself but also from other hospitals nationwide. The Centre Médicale la Cathédrale of Yaoundé is private secondary care facility within Yaoundé, catering for a wide variety of patients as well, both rural and urban.

Selection of participants. The participants were patients with signs and symptoms of gastroduodenal disorders aged from 15 years and above who attended the gastroenterology unit of the selected health facilities and who accepted to partake in the study. Patients who took antibiotics for 6 to 8 weeks or proton pump inhibitors or bismuth salts 6 weeks before the present consultation were excluded in the study. We also excluded patients with misidentification or those who had errors during analysis.

Data collection. A structural questionnaire was used to collect sociodemographic (age and sex) and lifestyle data (income level, smoking and alcohol consumption, and family history of gastric cancer), history of drugs consumption such as antibiotics, proton pump inhibitors or bismuth salts, and nonsteroidal anti-inflammatory drugs (NSAIDs) from participants.

For all participants, direct inquiry about dyspeptic symptoms: upper abdominal pains (epigastric pain, nausea and/or vomiting, burning feeling, frequent burping, and postprandial abdominal discomfort) was performed by a resident gastroenterologist, and the clinical characteristics were recorded. Esophagogastroduodenoscopy (EGD) was also performed for all participants by a resident gastroenterologist. During EGD session, endoscopic findings were recorded and biopsy samples were collected for *H. pylori* detection.

Endoscopy examination. Endoscopic examination was carried out for each eligible dyspeptic patient by a gastroenterologist. The endoscope was inserted through the patient's mouth, down the esophagus, and into the stomach. Once in the stomach, a complete visual observation of the stomach mucosal lining was performed and the different features of the mucosal were recorded.

The endoscopic aspects of the gastric mucosa lining encountered among participants were classified as normal, inflammatory lesions (gastritis, duodenitis, bulbitis, esophagitis, etc.), ulcerated lesions and their topography (antrum, fundus, duodenum, pylorus-bulbar, esophagus, or mixed location), and neoplastic lesions (gastrointestinal stromal tumors, leiomyoma, and mass lesions). The types of gastritis were also noticed according to the Sydney classification.[11–14] In this classification, the following gastritis types were defined: erythematous/exudative gastritis, papular-erosive gastritis, macular-erosive gastritis, hemorrhagic gastritis, enterogastric reflux gastritis, atrophic gastritis, and gastritis with hypertrophic folds. After the exploration of the stomach lining, gastric biopsies were collected in

three topographical areas of the stomach (one from fundus, one from angulus, and one from antrum) for *H. pylori* detection.

H. pylori *detection.* *H. pylori* detection from the gastric biopsy specimens was performed by rapid urease test (RUT) using *H. pylori* AMA Rapid Urease Test 1 kit (AMAT RUT 1, Association of Medicine and Analytics, Saint Petersburg, Russia). In this kit, one test slide is separated from the row and the protective cover is opened. The gastric biopsy was carefully placed on the white reactive element, and the protective cover was resealed gently. The test slide was then inserted into the slot of the AMA RUT Reader, and the button was pressed until the display showed "RUN." The results were read 5 min later. The presence of a color spot on one side of the indicator disk was indicative of urease activity in the biopsy specimen or a positive result. The result was negative in case of the absence of color on the indicator disk.

Statistical analysis. The data obtained were analyzed using the SPSS software version 20. Results were expressed as means ± standard deviation and median for quantitative variables, numbers, ratios, or percentages for qualitative variables. Student *t*-test was used to compare groups of continuous variables, and the Fisher exact test or Chi-square test was used for categorical variables. The strength of the association between *H. pylori* status and endoscopic findings was assessed by means of odd risks (ORs) with 95% confidence intervals (CIs) using univariable and multivariable logistic regression analysis. The differences were considered significant for $P < 0.05$.

All experiments were performed in accordance with the Declaration of Helsinki of 1975 and its later amendments or comparable ethical standards. This study was approved by the local Ethical Committee of Medical Sciences from every selected health facility: Regional Hospital Bafoussam (Ref: 2330/L/MIN SANTE/ SG/DRSPO/HRB/D), General Hospital Yaoundé (Ref: 07-19/ HGY/DG/DPM/NC-TR), and Centre Médicale la Cathédrale (Ref: 01-19CMC/TSM/LMS/AutoRech/2019/10/03), and from the National Ethical Committee on Human Health Research in Cameroon (1476/CE/CNERSH/SP). Informed consent was obtained from all subjects and their legal guardian(s) before including him or her into the study.

Results

Characteristics of the study population. This study included 532 consecutive dyspeptic subjects attending the gastroenterology department of the selected health facilities. Among these patients, those having upper endoscopy and colonoscopy contraindication ($n = 38$) were excluded, resulting in a final sample size of 494 subjects. Of the 494 subjects, 261 (52.8%) were women and 233 (47.2%) were men. Their age ranged from 15 to 79 years with 48.32 ± 17.17 years as mean age, 48.00 years as median, and group 45 to 54 years as the most represented (103: 20.9%), followed by age groups 55 to 64 (97: 19.6%) and older than 65 years (90: 18.2%) (Table 1). By cumulating these three age groups, which are at the top of the graphic (older than 45 years old), they represented more than half (290: 58.7%) of patients who had an indication for upper digestive endoscopy (Table 1).

Table 1 Sociodemographic, clinical, and endoscopic findings and gastritis types among the population

Variable	Number (%)
Sex	
Male	233 (47.2)
Female	261 (52.8)
Age (years)	Mean: 48.32 ± 17.17, range: 15 to 79, median: 48.00, Q1: 34, Q3: 62
≤24	51 (10.3)
25–34	73 (14.8)
35–44	80 (16.2)
45–54	103 (20.9)
55–64	97 (19.6)
>65	90 (18.2)
Clinical signs	
Epigastric pain	389 (78.7)
Burping	301 (60.9)
Burning	301 (60.9)
Vomiting	109 (22.1)
Nausea	133 (26.9)
Postprandial discomfort	214 (43.3)
Risk factors	
Income level	
Low	258 (52.2)
Middle	198 (40.1)
High	38 (7.7)
Smoking	
Yes	27 (5.5)
No	467 (94.5)
Alcohol	
Yes	118 (23.9)
No	376 (76.1)
History of gastric cancer	
Yes	37 (7.5)
No	457 (92.5)
Endoscopic indications	
Normal gastric mucosa	48 (9.7)
Gastritis	198 (40.1)
Duodenitis	54 (10.9)
Bulbitis	31 (6.3)
Esophagitis	51 (10.3)
Ulcerated lesions	110 (22.3)
Neoplastic lesion	2 (0.4)
Gastritis site	
Antrum	130 (65.6)
Fundus	10 (5.1)
Pangastritis	58 (29.3)
Ulcerated lesions site	
Antrum	47 (42.7)
Fundus	6 (5.5)
Duodenum	15 (13.6)
Pylori-bulbar	22 (20.0)
Esophagus	6 (5.5)
Mixed location	14 (12.7)
Gastritis type	
Normal gastritis	74 (37.4)
Erythematous/exudative gastritis	91 (45.9)
Macular-erosive gastritis	3 (1.5)
Papular-erosive gastritis	3 (1.5)
Hemorrhagic gastritis	3 (1.5)
Enterogastric reflux gastritis	24 (12.2)

Regarding factors that may be associated with dyspepsia among the sample population, more than half of our sample population were from the low income level (258: 52.2%), few of them were smokers (27: 5.5%), alcohol consumers (118: 23.9%), or had family history of gastric cancer (37: 7.5%) (Table 1).

Prevalence rate of gastrointestinal abnormalities among the sample population

Frequency of endoscopic lesions. Among patients who were examined, we have observed specified endoscopic aspects in 90.3% (446) of cases and normal endoscopic aspect in just 48 out of the 494 patients (Table 1). Regarding the variation of these, the highest percentage of pathological endoscopic aspects was 40.1% for gastritis, followed by ulcerated lesions in 22.3% of cases, duodenitis in 10.9%, esophagitis in 10.3%, normal mucosa aspect in 9.7%, and bulbitis in 6.3%. Only two cases of neoplastic lesions were detected among the general population examined endoscopically (0.4%: 2/494): one adenocarcinoma and one precancerous condition (intestinal metaplasia).

Frequency of endoscopic lesions according to the topography. 65.6% (130/198) of gastritis were located in the antrum, 5.5% (10/198) in fundus, and 29.3% (58/198) in pangastritis. The majority of ulcerated lesions were also located in the antrum (42.7, 47/110), followed by those located in the pylori-bulbar (20.0%), duodenum (13.6%), and mixed location (12.7%); the least encounter ulcerated lesion sites were esophagus (5.5%, 6/110) and fundus (5.5%, 6/110) (Table 1).

Frequency of endoscopic lesions according to sociodemographic factors. The distribution of endoscopic lesions in relation to participant's age, gender, lifestyle, and clinical sign was evaluated (Table 2). From this table, we noticed a significant difference in the frequency of endoscopic findings according to the gender of participants ($\chi^2 = 16.278$, $P = 0.012$). Females were with a higher rate of inflammatory lesions (gastritis: 42.1 *vs* 37.8%, duodenitis: 12.6 *vs* 9.0%, bulbitis: 8.0 *vs* 4.3%, and esophagitis: 11.5 *vs* 9%) than males, while higher rate of ulcerated lesions was recorded in males than in females (29.6 *vs* 15.7%) (Table 2).

The distribution of endoscopic lesions according to the age group of participants did not show any significant difference ($\chi^2 = 37.582$, $P = 0.161$). However, we noticed an inverse tendency regarding gastritis and ulcerated lesions as age of participants is concerned; the lower rate of gastritis was recorded among participants aged 45–54 years old (30.0%), while a pic of ulcerated lesions was observed at the same age group (35.6%) (Table 2). Moreover, a marginal *t*-value was recorded regarding mean age of patients with ulcerated lesions compared to those with gastritis ($t = 1.674$, $P = 0.0950$).

Frequency of endoscopic lesions according to clinical signs. The common complaints among our sample population were epigastric pain (78.7%), frequent burping (60.9%), gastric burning (60.9%), and postprandial discomfort (43.3) (Table 1). Clinical sign varied from one endoscopic lesion to another. Epigastritis (49.5%), burning (43.5%), and burping (40.9%) were mostly accounted in gastritis; both nausea (15.0%) and burping (13.0%) in duodenitis, nausea (11.9%), vomiting (13.5%), and postprandial abdominal discomfort (12.7%) in esophagitis; and vomiting/nausea (26.6%), epigastritis (22.9%), and burping (22.8%) in ulceration (Table 2). However, *P*-values greater than 0.05 were noticed regarding the distribution of each endoscopic aspect with respect to the clinical signs (Table 2).

Frequency of endoscopic lesions according to lifestyle of the participants. Income level was found to have an impact on the distribution of endoscopic aspects with a significant *P*-value ($P = 0.035$). In fact, participants with higher income levels were more subjected to duodenitis (14.6 *vs* 9.7%), esophagitis (14.4 *vs* 8.9%), and ulcerated lesions (31.8 *vs* 18.2%), whereas an opposite tendency was noticed for bulbitis (5.6 *vs* 7.8%). The difference was significant ($\chi^2 = 22.226$, $P = 0.035$) (Table 2).

A positive relationship with significant *P*-value was noticed between ulcerated lesions and alcohol consumption: 1.792 (1.123–2.858, $P = 0.014$) (Fig. 1e), being male: 2.258 (1.460–3.492, $P = 0.000$) (Fig. 1a), and having high income level: 1.6339 (1.0660–2.506), $P = 0.024$ (Fig. 1c). This link persisted even after adjustment with confounders with significant *P*-value regarding gender (OR: 2.1429 [1.3531–3.3938], $P = 0.006$) (Fig. 1a) and income level (OR: 1.404 [0.888–2.217], $P = 0.046$) (Fig. 1c).

Prevalence rate of gastritis type and distribution in the study population. Erythematous/exudative gastritis, macular-erosive gastritis, papular-erosive gastritis, hemorrhagic gastritis, and enterogastric reflux gastritis were the gastritis types observed in our sample population (Table 1). Erythematous/exudative gastritis was the most common gastritis type with a percentage of 45.9% (91/198). The next was enterogastric reflux gastritis with a percentage of 12.2% (24/198). Gastritis types such as hemorrhagic gastritis, papular-erosive gastritis, and macular-erosive gastritis were not representative in our sample population (1.5%) (Table 1). Regarding the severity of neoplastic lesions, the only adenocarcinoma detected was found to be associated with esophagite, whereas intestinal mataplasia was associated with erythematous mucosa.

The distribution of the gastritis type did not show a significant difference according to the gender, age, income level, alcohol consumption, and smoking status of participants. However, our data showed that females were more affected by erythematous/exudative gastritis than males (51.4 *vs* 38.6%), while males were more affected by enterogastric reflux gastritis than females (14.8 *vs* 10.1%). As age is concerned, participants in age group 35 to 44 years old were less affected by erythematous/exudative gastritis (39.1%) but more subjected to enterogastric reflux gastritis (24.1%) (Table 3).

Prevalence rate of H. pylori *infection in the study population.* Among the 494 dyspeptic subjects recruited, 286 were *H. pylori*-infected, giving a prevalence of 57.9% in our sample population.

The rates of *H. pylori* infection related to sociodemographic and lifestyle factors are presented in Table 4. The prevalence of infection according to age showed a peak of infection among participants aged above 65 years (74.4%) and those aged less than 24 years (66.7%) compared to the other age groups. This difference was significant ($\chi^2 = 21.614$, $P = 0.001$).

Table 2 Frequency of endoscopic lesions in relation to sociodemographic factors, potential risk factors, and clinical signs

Independent variable	N	Normal (%) n = 48	Gastritis (%) n = 198	Duodenitis (%) n = 54	Bulbitis (%) n = 31	Esophagitis (%) n = 51	Ulcer (%) n = 110	Neoplastic (%) n = 2	χ^2 (P value)
Sex									
Male	233	23 (9.9)	88 (37.8)	21 (9.0)	10 (4.3)	21 (9.0)	69 (29.6)	1 (0.4)	16.278 (**0.012**)*
Female	261	25 (9.6)	110 (42.1)	33 (12.6)	21 (8.0)	30 (11.5)	41 (15.7)	1 (0.4)	
Age (years)		46.05 ± 16.72	46.47 ± 15.78	47.28 ± 16.88	52.13 ± 19.89	45.20 ± 16.62	53.15 ± 16.66	83.50 ± 11.50	F = 2.938, (0.078)
≤24	51	4 (7.8)	21 (41.2)	4 (7.8)	4 (7.8)	7 (13.7)	10 (19.6)	1 (2.0)	37.582 (0.161)
25–34	73	9 (12.3)	32 (43.8)	11 (15.1)	4 (5.5)	8 (11.0)	9 (12.3)	0 (0.0)	
35–44	80	12 (15.0)	29 (36.3)	11 (13.8)	3 (3.8)	10 (12.5)	15 (18.8)	0 (0.0)	
45–54	103	5 (5.6)	27 (30.0)	10 (11.1)	8 (8.9)	7 (7.8)	32 (35.6)	1 (1.1)	
55–64	97	10 (10.3)	43 (44.3)	8 (8.2)	3 (3.1)	7 (7.2)	26 (26.8)	0 (0.0)	
>65	90	8 (7.8)	46 (44.7)	10 (9.7)	9 (8.7)	12 (11.7)	18 (17.5)	0 (0.0)	
Clinical signs									
Epigastric pain									
Yes	389	9 (8.6)	52 (49.5)	6 (5.7)	7 (6.7)	6 (5.7)	24 (22.9)	1 (1.0)	10.251 (0.114)
No	105	39 (10.0)	146 (37.5)	48 (12.3)	24 (6.2)	45 (11.6)	86 (22.1)	1 (0.3)	
Burping									
Yes	301	18 (9.3)	79 (40.9)	25 (13.0)	7 (3.6)	19 (9.8)	44 (22.8)	1 (0.5)	5.043 (0.538)
No	193	30 (10.0)	119 (39.5)	29 (9.6)	24 (8.0)	32 (10.6)	66 (21.9)	1 (0.3)	
Burning									
Yes	301	16 (8.3)	84 (43.5)	15 (7.8)	11 (5.7)	16 (8.3)	50 (16.61)	1 (0.5)	7.913 (0.245)
No	193	32 (10.6)	114 (37.9)	39 (13.0)	20 (6.6)	35 (11.6)	60 (19.9)	1 (0.3)	
Vomiting									
Yes	109	10 (9.2)	42 (38.5)	10 (9.2)	5 (4.6)	13 (11.9)	29 (26.6)	0 (0.0)	3.252 (0.777)
No	385	38 (9.9)	156 (40.5)	44 (11.4)	26 (6.7)	38 (9.9)	81 (21.0)	2 (0.5)	
Nausea									
Yes	133	13 (9.8)	45 (33.8)	20 (15.0)	7 (5.3)	18 (13.5)	30 (22.6)	0 (0.0)	7.437 (0.282)
No	361	35 (9.7)	153 (42.4)	34 (9.4)	24 (6.6)	33 (9.1)	80 (22.2)	2 (0.6)	
Postprandial discomfort									
Yes	214	27 (12.6)	78 (33.6)	21 (9.8)	16 (7.5)	27 (12.6)	44 (20.6)	1 (0.5)	8.264 (0.219)
No	280	21 (7.5)	120 (42.9)	33 (11.8)	15 (5.4)	24 (8.6)	66 (23.6)	1 (0.4)	
Risk factors									
Income level									
Low	258	30 (11.6)	111 (43.0)	25 (9.7)	20 (7.8)	23 (8.9)	47 (18.2)	2 (0.8)	22.226 (**0.035**)*
Middle	198	16 (8.1)	71 (35.9)	25 (12.6)	10 (5.1)	19 (9.6)	57 (28.8)	0 (0.0)	
High	38	2 (5.3)	16 (42.1)	4 (10.5)	1 (2.6)	9 (23.7)	6 (15.8)	0 (0.0)	
Smoking									
Yes	27	4 (14.8)	11 (40.7)	2 (7.4)	0 (0.0)	2 (7.4)	7 (25.9)	1 (3.7)	10.968 (0.089)
No	467	44 (9.4)	187 (40.0)	52 (11.1)	31 (6.6)	49 (10.5)	103 (22.1)	1 (0.2)	
Alcohol									
Yes	118	7 (5.9)	48 (40.7)	13 (11.0)	5 (4.2)	9 (7.6)	36 (30.5)	1 (0.8)	10.052 (0.122)
No	376	41 (10.9)	150 (39.9)	41 (10.9)	26 (6.9)	42 (11.2)	75 (19.9)	1 (0.3)	
History of gastric cancer									
Yes	37	2 (5.4)	21 (56.8)	3 (8.1)	2 (5.4)	1 (2.7)	8 (21.6)	0 (0.0)	6.299 (0.391)
No	457	46 (10.1)	177 (38.7)	51 (11.2)	29 (6.3)	50 (10.9)	102 (22.3)	2 (0.4)	
***Helicobacter pylori* status**									
Yes	286	15 (5.2)	115 (40.2)	25 (8.7)	27 (9.4)	34 (11.9)	68 (23.8)	2 (0.7)	27.588 (**0.000**)*
No	208	33 (15.9)	83 (39.9)	29 (13.9)	4 (1.9)	17 (8.2)	42 (20.2)	0 (0.0)	

P-value comparing different modalities (in row) of a specific independent categorical variable on the distribution of endoscopic findings. Bold value with *: significant.
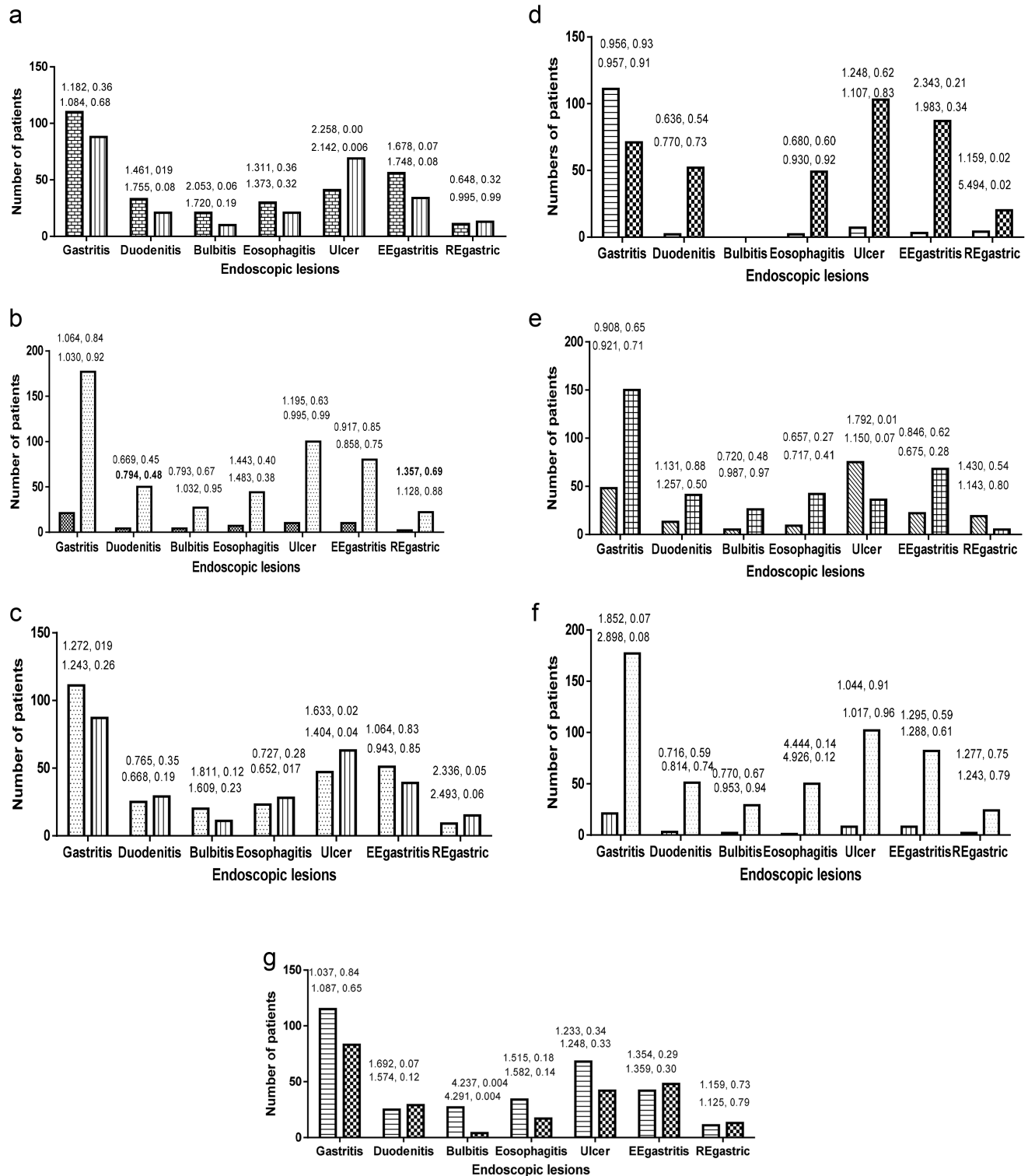
Figure 1 Frequency of endoscopic lesions and gastritis types in relation to socioeconomic and potential risk factors using univariate and multivariate logistic regression analysis. EEgastritis, erythematous/exudative gastritis; REgastric, reflux enterogastric. Number above columns in fist row: odds ratio, *P*-value for univariate analysis, numbers above column in second row: odds ratio, *P*-value for multivariate analysis. (a) Gender: (▨), Female; (▥), male. (b) Age: (▨), Age less than 24 years; (▱), age greater than 24 years. (c) Income level: (▱), Low income; (▥), high income. (d) Smoking: (▱), Smoking; (▩), no-smoking. (e) Alcohol consumption: (◰), Alcohol; (▦), no-alcohol. (f) Family history of gastric cancer (GC): (▥), History of GC; (▱), No history of GC. (g) *Helicobacter pylori* status: (▭), Hp positive; (▩), Hp negative.

Table 3 Frequency of gastritis types according to sociodemographic factors and potential risk factors

Independent variable	N	Norm	Eryth	Macul	Papul	Hem	Reflux	χ^2 (P-value)
Sex								
Male	88	36 (40.9)	34 (38.6)	1 (1.1)	2 (2.3)	2 (2.3)	13 (14.8)	4.410 (0.492)
Female	110	38 (34.9)	56 (51.4)	2 (1.8)	1 (0.9)	1 (0.9)	11 (10.1)	
Age (years)								
≤24	21	7 (33.3)	10 (47.6)	0 (0.0)	1 (4.8)	1 (4.8)	2 (9.5)	18.161 (0.835)
25–34	32	12 (38.7)	17 (54.8)	0 (0.0)	0 (0.0)	0 (0.0)	2 (6.5)	
35–44	29	10 (34.5)	10 (34.5)	1 (3.4)	0 (0.0)	1 (3.4)	7 (24.1)	
45–54	27	21 (45.7)	18 (39.1)	1 (2.2)	1 (2.2)	0 (0.0)	5 (10.9)	
55–64	43	13 (30.2)	23 (53.5)	1 (2.3)	1 (2.3)	1 (2.3)	4 (9.3)	
>65	46	11 (40.7)	12 (44.4)	0 (0.0)	0 (0.0)	0 (0.0)	4 (14.8)	
Risk factors								
Smoking								
Yes	111	4 (36.4)	3 (27.3)	0 (0.0)	0 (0.0)	0 (0.0)	4 (36.4)	6.993 (0.221)
No	71	70 (37.6)	87 (46.8)	3 (1.6)	3 (1.6)	3 (1.6)	20 (10.8)	
Alcohol								
Yes	48	15 (33.3)	22 (48.9)	1 (2.2)	1 (2.2)	1 (2.2)	5 (11.1)	1.026 (0.960)
No	150	59 (38.8)	68 (44.7)	2 (1.3)	2 (1.3)	2 (1.3)	19 (12.5)	
History of gastric cancer								
Yes	21	7 (35.0)	8 (40.0)	1 (5.0)	1 (5.0)	1 (5.0)	2 (10.0)	4.439 (0.488)
No	177	67 (37.9)	82 (46.3)	2 (1.1)	2 (1.1)	2 (1.1)	22 (12.4)	
Income level								
Low	111	45 (40.9)	51 (46.4)	2 (1.8)	1 (0.9)	2 (1.8)	9 (8.2)	16.346 (0.090)
Middle	71	20 (28.2)	37 (52.1)	1 (1.4)	2 (2.8)	1 (1.4)	10 (14.1)	
High	16	9 (56.3)	2 (12.5)	0 (0.0)	0 (0.0)	0 (0.0)	5 (31.3)	
Helicobacter pylori status								
Yes	83	28 (33.3)	42 (50.0)	0 (0.0)	1 (1.2)	2 (2.4)	11 (13.1)	5.585 (0.349)
No	32	46 (40.7)	48 (42.5)	3 (2.7)	2 (1.8)	1 (0.9)	13 (11.5)	

P-value comparing different modalities (in row) of a specific independent categorical variable on the distribution of gastritis type. Bold value with *: significant.

Eryth, erythematous/exudative gastritis; Hem, hemorrhagic gastritis; Macul, macular-erosive gastritis; Norm, normal gastritis; Papul, papular-erosive gastritis; Reflux, enterogastric reflux gastritis.

Regarding gender of participants, the rate of infection among males (57.9%) and females (57.9%) was the same ($\chi^2 = 0.0004$, $P = 0.985$) (Table 4).

Regarding potential risk factors evaluated, participants with family history of gastric cancer were significantly more subjected to *H. pylori* infection ($\chi^2 = 5.187$, $P = 0.023$) (Table 4).

Frequency of endoscopic lesions and gastritis type linked to H. pylori *infection*

Frequency of endoscopic lesions linked to H. pylori *infection.* *H. pylori* infection was found to have a significant impact on the distribution of endoscopic aspect ($\chi^2 = 27.588$, $P = 0.000$). Approximately 95% (94.8%, 271/286) of *H. pylori*-infected patients were with pathological gastric mucosa aspects. *H. pylori* was present in 87.1, 66.7, 61.8, 58.1, and 46.3% cases of bulbitis, esophagitis, ulcerated lesions, gastritis, and duodenitis, respectively. All the patients with gastric cancer were infected (Table 5).

A positive relationship was noticed between *H. pylori* infection and all types of endoscopic pathological aspect excepted duodenitis. ORs of 1.037 (0.720–1.493, $P = 0.845$), 4.237 (1.602–11.235, $P = 0.004$), 1.515 (0.822–2.793, $P = 0.183$), and 1.233 (0.798–1.904, $P = 0.345$) were found for the relationship between *H. pylori* infection and gastritis, bulbitis, esophagitis, and

ulcerated lesions, respectively, but with a significant P-value only for bulbitis ($P = 0.004$). This link persisted even after adjustment with confounders (Fig. 1g).

In contrast, a positive relationship was noticed between duodenitis and being *H. pylori*-non-infected (OR: 1.692 [0.9596–2.9850]), but with marginal P-value ($P = 0.07$) (Fig. 1g).

Regarding site of lesions, *H. pylori* was present in 58.5, 60.0, and 56.98% of gastritis from antrum, fundus, and pangastritis, respectively. This difference was not significant ($\chi^2 = 0.334$, $P = 0.846$) (Table 5). *H. pylori* was present in 59.6% of ulcerated lesions located in the antrum, 66.7% of those in the fundus or esophagus, 72.7% of those located in pylorobulbar, 33.3% of those from the duodenum, and 71.4% of those in mixed locations. This difference was not significant ($\chi^2 = 8.150$, $P = 0.148$) (Table 5).

Frequency of gastritis type linked to H. pylori *infection.* Our findings showed that *H. pylori* was present in 33.3% of hemorrhagic gastritis, 53.8% of erythematous/exudative gastritis, 54.2% of enterogastric reflux gastritis, 66.7% of papular-erosive gastritis, and 100% of macular-erosive gastritis cases. The highest rate of *H. pylori* infection (43.0%) was noticed among patients with erythematous/exudative gastritis, followed by those with normal gastritis (40.4%) and enterogastric reflux gastritis

Table 4 Prevalence of *Helicobacter pylori* infection and distribution in the study population

Independent variable	N	*H. pylori* positive $n = 286$	*H. pylori* negative $n = 208$
Sex			
Male	233	135 (57.9)	98 (42.1)
Female	261	151 (57.9)	110 (42.1)
χ^2 (*P* value)		0.0004 (0.985)	
Age (years)			
≤24	51	34 (66.7)	17 (33.3)
25–34	73	40 (54.8)	33 (45.2)
35–44	80	35 (43.8)	45 (56.3)
45–54	103	62 (60.2)	41 (39.8)
55–64	97	48 (49.5)	49 (50.5)
>65	90	67 (74.4)	23 (25.6)
χ^2 (*P* value)		21.614 (**0.001**)*	
Risk factors			
Income level			
Low	258	104 (40.3)	154 (59.7)
Middle	198	86 (43.4)	112 (56.6)
High	38	18 (47.4)	20 (52.6)
χ^2 (*P* value)		0.916 (0.632)	
Smoking			
Yes	27	16 (59.3)	11 (40.7)
No	467	270 (57.8)	197 (42.2)
χ^2 (*P* value)		0.022 (0.883)	
Alcohol			
Yes	118	65 (55.1)	53 (44.9)
No	376	221 (58.8)	155 (41.2)
χ^2 (*P* value)		0.502 (0.479)	
History of gastric cancer			
Yes	37	28 (75.7)	9 (24.3)
No	457	258 (56.5)	199 (43.5)
χ^2 (*P* value)		5.187 (**0.023**)*	

P-value comparing frequency of *H. pylori* infection according to a specific independent variable. Bold value with *: significant.

(11.4%), but the difference was not significant ($\chi^2 = 4.439$, $P = 0.488$) (Table 5).

An OR value higher than 1 was noticed between *H. pylori* infection and erythematous/exudative gastritis (OR: 1.354 [0.768–2.389]) and enterogastric reflux gastritis (OR: 1.159 [0.492–2.733]), but with non-significant *P*-values ($P = 0.295$ and 0.736, respectively) (Fig. 1g).

For the other risk factors, a positive relationship was noticed between enterogastric reflux gastritis and non-smoking (OR: 4.739 [1.275–17.543], $P = 0.020$) (Fig. 1d), between erythematous/exudative gastritis and being female (OR: 1.678 [0.949–3.968], $P = 0.075$) (Fig. 1a), and between enterogastric reflux gastritis and high income level (OR: 2.336 [0.9699–5.649], $P = 0.059$) (Fig. 1c). This relationship persists after adjustment with confounders.

Discussion

In this study, 532 patients with gastrointestinal complaints attending reference health facilities in the West and Centre Regions of Cameroon were examined for *H. pylori* infection in correlation with endoscopic aspects of their gastric mucosa.

None of the evaluated clinical signs was evocative to a specific endoscopic aspect, suggesting that one cannot get a clear idea on the type and severity of gastric mucosa injury from the clinical signs alone.

The prevalence of *H. pylori* infection was 57.9% in the current sample population. Analysis of demographic data showed that males (57.9%) and females (57.9%) were affected at the same rate ($\chi^2 = 0.0004$, $P = 0.985$). Some studies reported that males were associated with a higher risk of acquiring *H. pylori* infection than the females[4] because males are less hygienic than females, taking in view that the prevalence of *H. pylori* infection and sanitation condition are inversely related. The prevalence of infection according to age showed a peak of infection among participants aged above 65 years (74.4%) and those aged less than 24 years (66.7%) with a significant *P*-value ($P = 0.001$). A similar age association has been reported in prior studies in Cameroon,[4] Tanzania,[16] and Ethiopia.[17] The highest prevalence observed among our younger population correlated with findings showing that in developing countries, contamination occurs early in childhood,[18] and before 10 years, more than 50% of children are already infected,[19–21] while in developed countries, the highest prevalence of infection is among older people (≥60 years old).[7,22] One suggests family transmission as the main reason for the higher rate of infection among younger people in developing countries because of overcrowded household and poor personal and environmental hygiene. Participants with family history of

Table 5 Frequency of endoscopic lesions and gastritis types in relation to *Helicobacter pylori* infection

Independent variable	Number	*H. pylori* positive *n* = 286	*H. pylori* negative *n* = 208
Endoscopic indications			
Normal gastric mucosa	48	15 (31.3)	33 (68.7)
Gastritis	198	115 (58.1)	83 (41.9)
Duodenitis	54	25 (46.3)	29 (53.7)
Bulbitis	31	27 (87.1)	4 (12.9)
Esophagitis	51	34 (66.7)	17 (33.3)
Ulcerated lesions	110	68 (61.8)	42 (38.2)
Neoplastic lesion	2	2 (100.0)	0 (0.0)
χ^2 (*P* value)		27.588 (**0.000**)*	
Gastritis site			
Antrum	130	76 (58.5)	54 (41.5)
Fundus	10	6 (60.0)	3 (30.0)
Pangastritis	58	33 (56.9)	25 (43.1)
χ^2 (*P* value)		0.334 (0.846)	
Ulcerated lesions site			
Antrum	47	28 (59.6)	19 (40.4)
Fundus	6	4 (66.7)	2 (33.3)
Duodenum	15	5 (33.3)	10 (66.7)
Pylori-bulbar	22	17 (72.7)	5 (22.7)
Esophagus	6	4 (66.7)	2 (33.3)
Mixed location	14	10 (71.4)	4 (28.6)
χ^2 (*P* value)		8.150 (0.148)	
Gastritis type			
Normal gastritis	74	46 (62.2)	28 (37.8)
Erythematous/exudative gastritis	91	49 (53.8)	42 (46.2)
Macular-erosive gastritis	3	3 (100.0)	0 (0.0)
Papular-erosive gastritis	3	2 (66.7)	1 (33.3)
Hemorrhagic gastritis	3	1 (33.3)	2 (66.7)
Enterogastric reflux gastritis	24	13 (54.2)	11 (45.8)
χ^2 (*P*-value)		4.439 (0.488)	

P-value comparing the distribution of endoscopic findings and gastritis types according to *H. pylori* status. Bold value with *: significant.

gastric cancer were significantly more subjected to *H. pylori* infection (*P* = 0.023) (Table 4). A similar finding was reported in a previous study performed in Cameroon,[4] Italy,[23] and Saudi Arabia.[24]

Pathological endoscopic aspects were recorded in 90.3% of cases *versus* 9.7% of cases with normal endoscopic aspects. Such observation reinforces the idea that dyspepsia is indicative of gastric mucosa injuries. Among gastric lesions detected, gastritis was the most common one (40.1%), followed by ulcerated lesions in 22.3% of cases and duodenitis in 10.9%. Our result is in accordance with those reported by Geanina *et al.* who encounter gastritis in the highest percentage in their dyspeptic population examined endoscopically.[25] *H. pylori* infection was found to have an impact on the occurrence of mucosal injury with a significant *P*-value (*P* = 0.000). In fact, a pathological aspect was observed in 94.8% of *H. pylori*-infected cases with gastritis as the most frequent (42.4% of cases). Moreover, OR values greater than 1 were recorded between inflammatory lesions (gastritis, bulbitis, and esophagitis), ulcerated lesions, and *H. pylori* infection before or after adjustment with confounders, with a significant *P*-value for bulbitis (*P* = 0.004). Such observation highlights the fact that this infection is a major cause of the endoscopic inflammatory and ulcerated lesions. The current finding is in agreement with reports revealing that the rate of *H. pylori*

infection in patients with gastric lesions is significantly higher than the rate among *H. pylori*-negative patients (*P* < 0.01).[26] About 75% of patients with chronic gastritis have been found to have *H. pylori* infection compared to 10% in those without gastritis.[27,28] Since its discovery, a close relationship between chronic gastritis and *H. pylori* infection has been reported throughout the whole world. *H. pylori* has been found to be strongly associated with inflammation and patchy gastric atrophy, called type B gastritis or chronic non-autoimmune gastritis.[27–29] *H. pylori* is an organism that mediates a compromise of the gastric mucosal barrier by stimulating increased gastric acid secretion, causing alteration of certain immune factors, penetration of the mucosal layer, and provoking persistent inflammation even without invading the mucus membrane.

According to the topography, 65.7% (130/198) of gastritis were confined to the antrum with 84.5% of them being *H. pylori*-colonized, indicating antrum as the preferable site of *H. pylori* colonization in gastritis. Similarly, some authors found antrum as the preferable location of *H. pylori* organism among individuals with gastritis.[30] In fact, it has been reported that antrum is the most prominent site for gastritis compared to upper stomach (fundus) or whole stomach lining (pangastritis) and that the antrum is usually the most common site of inflammation with its submucosal layer frequently colonized by *H. pylori*.[31] The

different expressions of gastritis in antrum and body are suggested to be due to increased reactivity of the antral mucosa to the infection, possibly on the basis of an enhanced immunologic response to *H. pylori* in this region.[32]

This may be due to the absence of parietal cells (acid producers) in the antrum, which favors the bacterial colonization.[33]

The majority of ulcerated lesions were located in the antrum (42.7%), followed by those from the pyloro-bulbar (20.0%), suggesting antrum also as the prominent site for gastric ulceration in our dyspeptic population. Our findings revealed that *H. pylori* was present mostly in ulcerated lesions from the pyloro-bulbar (72.7%), indicating that this pathogen is the main contributor of ulcer in the gastric proximity part to the duodenum. This finding is close to those revealing that *H. pylori* infection is common in patients with duodenal ulcers[34–36] and that more than 90% of duodenal ulcers *versus* 60% of gastric ulcers are associated with *H. pylori* infection in endoscopy.[36,37] *H. pylori* contributes to ulcer formation by increasing acid production, interfering with the stomach's normal defenses against stomach acid and producing toxins.[38]

Our findings also revealed that factors other than *H. pylori* infection might be associated with the outcome of endoscopic aspects. In fact, the strength of the association of mucosa injury in relation to gender showed that females were 1.461: 0.819–2.604 ($P = 0.199$) and 1.692: 0.9596–2.9850 ($P = 0.070$) times more subjected to duodenitis and bulbitis, respectively than males, while males were 2.258: 1.460–3.492 ($P = 0.000$) times more subjected to ulcerated lesions than females. Our findings also showed that alcohol consumers ($P = 0.014$ and 0.077) (Fig. 1e) and participants with high income level were more prone to develop ulcerated lesions ($P = 0.024$, 0.046) (Fig. 1c) and that patients aged less than 24 years old were more subjected to gastritis (1.182: 0.823–1.696), whereas those aged more than 24 years old were mostly affected by ulcerated lesions (1.195: 0.578–2.471) (Fig. 1b).

Regarding gastritis types according to the Sydney classification, erythematous/exudative gastritis was the most common gastritis type (45.9%) in our sample population. This distribution is in accordance with that of a previous study reporting erythematous/exudative gastritis as the most common gastritis type.[25] The distribution of the gastritis type with respect to *H. pylori* status showed that *H. pylori* was present in more than 50% of each form of gastritis detected excepted hemorrhagic gastritis. The strength of the association between gastritis type and *H. pylori* status showed that *H. pylori*-infected patients were 1.354 (0.768–2.389) and 1.159 (0.492–2.733) more prone to develop erythematous/exudative gastritis and enterogastric reflux gastritis, respectively (Fig. 1g). This relationship, which persists after adjustment with confounders, suggests that *H. pylori* infection is a risk factor for these forms of gastritis and corroborates with reports revealing erythematous/exudative gastritis as the major type of gastritis encountered among *H. pylori*-positive patients.[25]

Our findings showed that enterogastric reflux gastritis and ulcerated lesions were affected in a similar way regarding risk factors. Regarding age and gender, enterogastric reflux gastritis and ulcerated lesions were commonly seen among older participants instead of younger ones and among males instead of females. In addition, non-smokers and alcohol consumers were mostly affected by enterogastric reflux gastritis and ulcerated

lesions. Such observation suggested that enterogastric reflux gastritis might lead to ulcerated lesions. It had been reported that bilious reflux might be an important factor in the etiology of gastric ulcer and that excessive enterogastric reflux may be seen in patients with gastric ulcers.[39] When patients with reflux gastritis were compared with normal controls, significant increases in associated peptic ulceration were found in the group with reflux.[40] In fact, bile and stomach acid can reflux into the esophagus when the lower esophageal sphincter does not work properly and may damage the gastric mucosa.

In conclusion, *H. pylori* was present in 57.9% of the general population with upper digestive symptomatology. This infection was mostly frequent among participants aged above 65 years and those aged less than 24 years. In 94.8% of *H. pylori*-infected patients, a pathological aspect was observed by endoscopy, and the most frequent was gastritis. The antrum was the preferable site of *H. pylori* colonization for gastritis, while it was the pyloro-bulbar for ulcerative lesions. A positive relationship between gastritis, bulbitis, and esophagitis or ulceration and *H. pylori* was noticed, but with a significant *P*-value only for bulbitis. The most common gastritis type recorded among *H. pylori*-infected patients was the erythematous/exudative gastritis.

Acknowledgments

Patient consent

Participation was voluntary, and a written informed consent was obtained from all subjects and their legal guardian(s) before including him or her into the study. The written informed consent of participants enrolled in the current study are available from the corresponding author on reasonnable request.

Data availability statement. The datasets used and/or analyzed during the current study are not publicly available because they are confidential but are available from the corresponding author on reasonable request.

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